Developing an Integrated Framework of Eating Behaviour Traits: Evaluating their Associations with Energy Intake and Weight Change.

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Thesis structure

This thesis comprises a series of studies developing a comprehensive framework of eating behaviour traits, and evaluating the framework's ability to predict external outcomes, including energy intake, body mass index and weight change. This thesis is presented in the alternative format. The alternative format refers to a publication-based thesis format. Rather than the traditional chapter style thesis, it allows published manuscripts to be included in this thesis, without the need for the work to be rewritten, and enables the candidate to maximise academic research outputs. This style of thesis includes an overall introduction, as well as a discussion and conclusion section after the included manuscripts, to bind them into a whole. Each chapter is labelled as either a Journal Article or Thesis Sub-section. Where journal articles are presented, the formatting will be consistent with the relevant target journal. This will be clearly labelled at the start of each chapter, with the most up-to-date submission status.

List of publications relating to chapters in this thesis

Dakin, C., Finlayson, G., & Stubbs, R. J. (2024). Exploring the Underlying Psychological Constructs of Self-Report Eating Behaviour Measurements: Towards a Comprehensive Framework. *Psychological Review*. (In Press). (Chapter 2, Paper 1).

Dakin, C., Beaulieu, K., Hopkins, M., Gibbons, C., Finlayson, G., & Stubbs, R. J. (2023). Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis. *Obesity Reviews*, *24*(1), e13515. (Chapter 3, Paper 2).

Dakin, C. A., Finlayson, G., Horgan, G., Palmeira, A. L., Heitmann, B. L., Larsen, S. C., ... & Stubbs, R. J. (2023). Exploratory analysis of reflective, reactive, and homeostatic eating behaviour traits on weight change during the 18-month NoHoW weight maintenance trial. *Appetite*, *189*, 106980. (Chapter 4, Paper 3).

Dakin, C., Finlayson, G., & Stubbs, R. J. (2024). Can eating behaviour traits be explained by underlying, latent factors? An exploratory and confirmatory factor analysis. *Appetite*, *195*, 107202. (Chapter 5, Paper 4).

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Other publications I have contributed to during this thesis.

James Stubbs, R., Horgan, G., Robinson, E., Hopkins, M., **Dakin, C.,** & Finlayson, G. (2023). Diet composition and energy intake in humans. *Philosophical Transactions of the Royal Society B*, *378*(1888), 20220449.

Dakin, C., Beaulieu, K., Halford, J., Finlayson, G., Hodgkins, C. E., Raats, M., … & Harrold, J. A. (2024). Exploratory analysis of the impact of sweeteners on eating behaviours and consumer perceptions during a weight maintenance RCT in adults and children with overweight from the SWEET project. *AJCN*, *Ready to Submit*.

Dakin, C., Finlayson, G., Buckland, N. J., Dalton, M., Katariya, S., Rodriguez, P., Baaij, A., & Stubbs, R. J. (2024). Examining the role of Nutritional, Sensory and Perceptual characteristics in Perceived Satiety of 312 commonly consumed foods in the UK diet. *Ready to Submit*.

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Abstract

Motivations for under or overeating are influential in shaping eating behaviours, and understanding these motivations may help to increase healthy eating and/or prevent dysfunctional eating. Eating behaviour traits (EBT) are self-report measures of eating behaviour constructs, that are used to quantify individual differences in motivations to eat. However, the multitude of EBTs has led to confusion, due to overlap between constructs, theories and proposed mechanisms of action. Research is needed to clarify the current measures of EBTs, how they relate to each other and how they relate to external outcomes. This thesis develops a comprehensive framework of EBTs and examines how it relates to existing theory and its ability to predict external outcomes.

Paper One proposes a provisional framework of EBTs, including three-factors which integrate knowledge from dual-process theory. **Paper Two** identifies existing EBTs and found that EBTs significantly predict short-term energy intake and longer-term energy balance (Body Mass Index; BMI). Susceptibility to hunger, disinhibition and binge eating ('reactive' EBTs) were the strongest predictors. **Paper Three** utilised the strongest predictors along with restraint ('restricted' EBT), and measures of homeostatic eating ('homeostatic' EBTs), in a weight loss maintenance trial. The results found reactive, restricted and homeostatic EBTs were associated with weight change. **Paper Five** and **Paper Six** apply factor analysis to multiple EBT constructs (paper 5) and their individual items (paper 6) and found support for a 6-7-factor model of EBTs, including reactive eating, negative emotional eating, positive emotional eating, restricted eating, homeostatic eating, eating for pleasure and eating for health. Across diverse samples, the framework was significantly associated with BMI and weight change. This thesis supports the use of a framework of EBTs, and highlights the development of a brief EBT domain survey as a measurement tool, to improve eating behaviour tracking and outcomes of research and weight management interventions.

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Abbreviations

AEBQ	Adult Eating Behaviour Questionnaire
BCT	Behaviour Change Technique
BCTTv1	Behaviour Change Taxonomy
BCW	Behaviour Change Wheel
BCTO	Behaviour Change Ontology
BES	Binge Eating Scale
BFCC/IES_BFCC	Body-Food Choice Congruence
BMI	Body Mass Index
BST	Behavioural Susceptibility Theory
CEBQ	Child Eating Behaviour Questionnaire
CFA	Confirmatory Factor Analysis
CFI	Comparative Fit Index
CID	Clinical Investigation Day
CNS	Central Nervous System

COEQ	Control of Eating Questionnaire
COR	Correlation
DEBQ	Dutch Eating Behaviour Questionnaire
DEBQ_EX	External Eating
DEBQ_R	Restraint
DF	Degrees of Freedom
EBT	Eating Behaviour Trait
EDAS	Eating Disorders Assessment Scale
EDDS	Eating Disorders Diagnostics Scale
EDE-Q	Eating Disorder Examination Questionnaire
EDI-2	Eating Disorders Inventory
EF	Enjoyment of Food
EFA	Exploratory Factor Analysis
EI	Energy Intake
EMA	Ecological Momentary Assessment
EOE/AEBQ_EOE	Emotional Over-Eating

EPR/IES_EPR	Eating for Physical Reasons rather than Emotional Reasons
eSatter	Satter Eating Competence Model
EUE/AEBQ_EUE	Emotional Undereating
Facet MAP	Facet-level Multidimensional Assessment of Personality
FCI	Food Craving Inventory
FCQ-T	Food Craving Questionnaire Trait
FF	Food Fussiness
FR/AEBQ_FR	Food Responsiveness
GP	General Population
H/AEBQ_H	Hunger
IES	Intuitive Eating Scale
IES-2	Intuitive Eating Scale 2
IQR	Interquartile Range
IRT	Item Response Theory
LMM	Linear Mixed Model
MEQ	Mindful Eating Questionnaire

ML	Maximum Likelihood
MMAT	Mixed Method Appraisal Tool
NEAT	Non-Exercise Activity Energy Expenditure
PA	Physical Activity
PFC	Prospective Food Consumption
PFS	Power of Food Scale
PNEES	Positive and Negative Emotional Eating Scale
PNEES_N	Negative Emotional Eating
PNEES_P	Positive Emotional Eating
RCT	Randomised Controlled Trial
RED	Reward-Based Eating Drive
RED-9, RED-13, RED-X5	Reward-Based Eating Drive Questionnaire
RHSC/IES_RHSC	Reliance on Hunger and Satiety Cues
RMR	Resting Metabolic Rate
RMSEA	Root Mean Square Error of Approximation
RRS	Revised Restraint Scale

RS	Restraint Scale
SD	Standard Deviation
SE	Standard Error
SE	Slowness in Eating
SES	Socioeconomic Status
SQ	Satiety Quotient
SR/AEBQ_SR	Satiety Responsiveness
SREB	Self-Regulation of Eating Behaviour
SRMR	Standardised Root Mean Square Residual
TDEE	Total Daily Energy Expenditure
TEF	Thermic Effect of Food
TEMS	The Eating Motivation Survey
TFEQ	Three-Factor Eating Questionnaire
TFEQ_D	Disinhibition
TFEQ_H	Susceptibility to Hunger
TFEQ_R	Restraint

UPE/UPTE/IES_UPTE	Unlimited Permission to Eat
WEL	Eating Self-Efficacy
WM	Weight Management
YFAS-2	Yale Food Addiction Scale-2

Chapter 1. Introduction

1.1. Context

The complex relationship between food production, retail and consumption and sociocultural contexts in which these activities occur is constantly evolving over time. Food serves multiple purposes, not only in the fulfilment of nutritional needs, but also in leisure, socialising, health, politics and the media (Poulain, 2017; Warde, 2016). There has been a significant shift in modern eating practices that occurred in the latter half of the twentieth century. Following centuries of intermittent malnutrition, structural transformations in the sectors of food production, distribution and marketing, led to an abundance and increased availability of food, which has now become overabundance (Poulain, 2017; Stubbs et al., 2012). The food system has also become characterised by foods that are on average more energy dense, where an increasing proportion of the calories in the food system come from fat and refined carbohydrates at lower production and consumption cost (Drewnowski & Popkin, 1997). These changes have occurred due to the globalisation of food markets, which has strongly influenced food supply chains, eating behaviours and body weight (Sobal, 1999). These secular trends are believed to be a large driver of the prevalence of obesity around the world (Sobal, 2001). Consequently, governments across the globe are becoming more concerned with what people eat and its impact on weight and health outcomes on the one hand (Warde, 2016) and pressure from food producers and retailers on the other.

Shifts in the food system have led to a ‘culinary revolution’ (Panayi, 2008). In the past, eating was viewed as an instrumental and practical activity. However, more recently, eating has been recognised as an enjoyable process, that can involve communicating with others, a means of forming one’s self-identity, and is involved in the expression of everyday life (Warde, 2016). For many people, food is one of the main pleasures in life and as such, engaging in an activity associated with food is one the main ways people spend their daily lives (Rozin, 1997). Given the above context, understanding why people eat and what motivates them to eat, is essential for understanding what ‘normal’, ‘adaptative’ or ‘healthful’ eating behaviour involves.

This is especially important due to the increasing prevalence of excess adiposity worldwide. Excess adiposity is associated with an increased risk of noncommunicable diseases (NCDs), including cardiovascular disease, diabetes, cancers, neurological disorders, chronic respiratory diseases and digestive disorders (GBD, 2019). In the UK, from 2021-2022, 63.8% of adults were estimated to be living with overweight or obesity (GOV, 2023). The increasing rates of obesity are becoming a rapidly growing public health concern that has significant long-term consequences for quality of life (Agha & Agha, 2017). Accordingly, an understanding of eating behaviours, which encompasses the motivations for both eating and overeating, is needed to better understand how to prevent people from developing dysfunctional eating behaviours which may lead to over or less commonly undernutrition, and to help people develop more adaptive eating behaviour, in an environment that is now radically different from the one in which our eating behaviour systems evolved in.

To better understand eating behaviour, it is important to examine it from a psychological and physiological perspective. Eating is a measurable form of motivated behaviour. Motivations to eat can be biologically and psychologically driven, and can be environmentally influenced (Watts et al., 2022). Taking a physiological approach, eating behaviour can be explained in the context of energy balance, which suggests that energy balance is biologically regulated and is referred to as homeostasis (Cannon, 1929). Homeostasis suggests that the body's internal environment is maintained, within a controlled homeostatic range. There have been numerous theories which propose that various components of the energy balance system function as negative feedback signals, to influence energy intake (EI). Appetite has been proposed to be regulated by adipose tissue (Kennedy, 1953), fat free mass and resting metabolic rate (Blundell et al., 2015), body temperature (Brobeck, 1946), fat (Kennedy, 1953), carbohydrates (Mayer, 1953), amino acids (Mellinkoff et al., 1956), protein leverage (Raubenheimer & Simpson, 2019) and body weight (Hervey, 1969). In the context of energy balance, overeating is viewed as influenced by highly palatable, energy dense foods (Hall et al., 2022). It has been proposed by some that body weight regulation is asymmetric, and has evolved because a positive energy balance is much more adaptive for survival than a negative energy balance (Neel, 1962; Stubbs & Tolkamp, 2006).

As such, it would appear energy balance is regulated, but it is not symmetric or precise. There are 3 basic positions on this. Firstly, that energy balance is symmetrically regulated around set points (Kennedy, 1953), but those points become disturbed or undermined by some aspect of genes or the environment (Farooqi & O'Rahilly, 2008). Secondly, that energy balance is regulated in a symmetrical manner, but that regulation has wide upper and lower limits (Speakman et al., 2011). Thirdly, that balance regulation is asymmetric, and we are designed to protect ourselves against negative energy balances to a far greater extent than positive energy balances (Stubbs et al., 2023). In any of these scenarios, the precision of energy balance regulation seems to be relatively loose, and energy balance regulation should be viewed as occurring over weeks and months rather than hours and days.

From a psychological perspective there several key theories that relate to eating behaviour, which aim to explain reasons and motivations for eating. These are reviewed in further detail in **Chapter 2**. However, in brief, many psychological approaches to eating behaviour draw from dual-process theory (Strack & Deutsch, 2004) and suggest that eating is motivated by reflective and impulsive processes. Indeed, Greaves et al. (2011) highlight the tension between new behaviours (reflective processes) and existing habits, food preferences and learned behaviours which are largely reactive. Overall, the main dimensions that are captured by various psychological theories focus on one or both of two processes involving cognitive and/or automatic behaviours e.g. (Herman & Mack, 1975; Llewellyn & Wardle, 2015; Lowe & Levine, 2005; Schachter, 1967). Many of these psychological theories develop constructs from their central elements, which are used to help explain specific motivations for eating. Externality theory is foundational to the study of EBTs through its core components of external and internal cues (Schachter, 1967). For example, Behavioural Susceptibility theory (Llewellyn & Wardle, 2015) can be seen to draw from externality theory because it proposes the construct of satiety responsiveness and food responsiveness, as motivations for eating. These constructs are developed to measure individual differences in specific dispositional motivations to eat and are termed "eating behaviour traits" (EBTs).

1.2. Eating Behaviour Traits

EBTs are relatively stable psychological characteristics related to eating, which are measured using psychometric questionnaires e.g. the Three Factor Eating Questionnaire (Stunkard & Messick, 1985). They have been developed because they allow researchers to better understand and quantify various aspects of eating behaviours (usually elements of 'disordered' eating or predisposition to overeat), including attitudes towards food, triggers for eating and habitual patterns. For example, EBTs have been used to study changes in fat mass, as well as physical activity, opportunistic eating, tendency to overconsume and food reward (Finlayson et al., 2012). The standard form of construct measurement in psychology has been the use of self-report questionnaires, such as questionnaires used to measure personality (Paulhus & Vazire, 2007). These questionnaires often use scales or items that cluster into traits (constructs), and these constructs can be related to theory (Allport & Allport, 1921). This idea that psychological behaviour such as personality can be classified and measured was first proposed by Allport and Allport (1921), who suggested that personality is too complex to be "trussed up in a single conceptual straight jacket" (Allport, 1955). Accordingly, the structure of personality was developed which separates personality into its major characteristics (Allport, 1955) and attempts to lay the groundwork for the psychology of personality to develop.

One of the key limitations of this approach is that psychological research tends to be abstracted from the behaviours they are trying to explain. It is therefore useful to examine those constructs where possible, in relation to changes in measured behaviours, or indices of long-term behaviour. This is possible in the field of energy balance. Measuring EBT constructs can help to identify eating behaviours that promote prolonged energy imbalances, and hence impact weight and health. In the context of obesity, individual differences in overeating can be explained by individual differences in behaviour (Vainik et al., 2019). For example, research has shown that disinhibition is an important EBT which is associated with higher BMI, obesity and mediating variables including less healthful food choices, all of which contribute to overweight, obesity and poorer health (Bryant et al., 2008). Accordingly, EBTs can be used as indictors of susceptibility to overconsume and develop obesity or to

under consume relative to energy requirements (Bryant et al., 2012). By measuring EBTs, researchers can identify individuals at risk of developing behavioural patterns that lead to health-related problems, such as obesity or eating disorders. This means that EBTs can be used as preventative measures by identifying problematic eating behaviours in individuals early on, which could reduce the risk of developing more serious health disorders/diseases. Early intervention could help individuals to develop healthier eating habits and prevent the onset of obesity. Indeed, there is evidence that engaging in mindful eating has the potential to address problematic eating behaviour and the challenges individuals face when trying to control their food intake (Warren et al., 2017).

Questionnaires that are used to measure EBTs can also be used to help design targeted interventions for individuals who are struggling with specific eating behaviours. Indeed, Teixeira et al. (2005) suggests that treatments should be tailored to meet individual needs, by identifying individuals who are less likely to succeed and providing them with additional treatment above general guidance. EBTs are one method of identifying those individuals who may struggle in weight management interventions. For example, a conceptual review found that individuals with higher levels of binge eating, disinhibition, susceptibility to hunger and negative emotional eating, are at a higher risk for weight regain (Elfhag & Rössner, 2005). Therefore, identifying individuals with higher levels of these EBTs and providing them with more support may improve their weight loss outcomes.

However, there are some limitations in the use of EBTs. Several theories of eating behaviour share similar constructs and proposed mechanisms of action, which limits our ability to measure and understand human eating behaviour (Vainik et al., 2015). For example, food responsiveness, hedonic hunger, external eating, and susceptibility to hunger are four constructs that measure overlapping eating behaviours. If EBTs are overlapping, they could be causing confusion because the psychometric and predictive validity of each EBT is unknown. This could mean that although these questionnaires are widely used in research, their results may not accurately reflect an individual's eating behaviours and motivations for eating. This concern has been highlighted before in the bigger field of personality. Various structure models have been built which propose multiple independent personality

factors (Ashton & Lee, 2007; Eysenck & Eysenck, 1975; Goldberg, 1981). However, research has suggested that there are many sources of common variance between personality measures, with evidence of overlapping traits (Ackerman & Heggestad, 1997). Through a meta-analysis of personality-intellectual ability correlations, the authors found evidence for communality across the domains of personality and identified four trait complexes.

Similarly to the field of personality, there is an abundance of different questionnaires that exist to measure multiple EBTs. However, the field of EBTs is currently limited because it is unknown which EBTs are overlapping, and which measure distinct motivational constructs that can explain or predict eating behaviour. This undermines the ability to compare study findings, synthesise evidence, and replicate studies and interventions (Marques et al., 2023). Indeed, in the field of behaviour change, Marques et al. (2023) argue that the varying descriptions of behaviour change interventions are a barrier to accumulating evidence about the effectiveness of interventions and making recommendations for research and policy. This also hinders the development of more effective interventions. Accordingly, the authors identify the need to organise behaviour change techniques into higher-order groups, which led to the development of the behaviour change wheel (Michie et al., 2011) and behaviour change technique ontology (Marques et al., 2023).

In the field of EBTs, there has been very few attempts to meaningfully organise the constructs that currently exist. Whilst some researchers have begun to statistically examine the underlying structure of EBTs e.g. (Price et al., 2015; Racine et al., 2019; Vainik et al., 2015), there is still a lack of understanding about the overarching domains that may account for different dimensions of motivation to eat or not eat. These limitations are to some extent exacerbated by the numerous overlapping theories and constructs that obfuscate rather than clarify our understanding, meaning researchers do not understand exactly what the scale they are using measures, which may be influencing important outcomes of studies. EBTs also tend to focus solely on psychological motivations for eating, when physiology is also an important cue that can influence eating behaviour. Some EBTs do exist to measure physiological eating behaviour, such as satiety responsiveness (Llewellyn & Wardle, 2015), which measures an individual’s sensitivity to their internal levels of satiety.

However, most questionnaires focus on measuring psychological constructs, and there is a lack of research assessing motivations related to putative biological drivers of energy balance behaviours (physiological hunger and homeostatic eating). Indeed, the vast majority of EBT measures include constructs that primarily relate to 'unregulated' eating.

Taken together, there are significant limitations to the current field of EBTs, which need to be addressed to utilise EBTs to their full potential. There is a need to better understand the measures of motivation to eat that currently exist, how they relate to existing theory, how the constructs relate to each other and how they relate to measured eating behaviour and energy balance outcomes. In the field of behaviour change, an approach has been taken to develop a structured theoretical domains framework of behaviour change taxonomies and ontologies (Michie, 2014). Applying similar approaches to the somewhat smaller field of eating behaviour should enable better clarification and standardisation of measurements, better comparisons of studies through meta-analyses, and comparative effect sizes (Marques et al., 2023), greatly enhancing our insight into the dimensions of eating motivation that affect weight and health. Developing a comprehensive framework of EBTs that encompasses the various dimensions of eating behaviour and provides a coherent structure for the research and application of EBT measurements, would greatly advance this field. Consequently, this thesis aims to address the current limitations of EBTs through a series of studies conducted in **Chapters 2-6**.

1.3. Aims of the present thesis

The overall aim of this thesis is to refine and validate a comprehensive framework of EBTs that attempts to integrate psychological, physiological and neurobiological theory, and examine the ability of the framework to predict energy intake (EI), body mass index (BMI) and weight change, and individual variability in weight change, during weight management interventions.

Specifically, the thesis aims to address the following research questions:

i) What are the core constructs underlying EBTs, and how can they be integrated into a comprehensive framework by drawing from key psychological, physiological and neurobiological models?
ii) To what extent can a framework of EBTs be used to predict external, objectively measured energy balance-related outcomes (short-term EI and long-term energy balance as estimated BMI), and individual variability in weight change, during weight management interventions?
iii) Can the framework of EBTs be refined and validated in samples with differing weight management (energy balance management) goals?

To address the first aim of this thesis, key psychological, physiological and neurobiological models will be drawn from as a starting point to develop a comprehensive framework that identifies the core, latent constructs underlying EBTs measurements (**Chapter 2**). This conceptual review first presents key definitions of human appetite and eating behaviour and then explores key psychological theories of eating behaviour, to better understand the theories and constructs, that have been developed to measure motivations for eating. The review then summarises physiological and neurobiological models that have been proposed to explain motivations for eating. The main purpose of the review is to propose a provisional conceptual framework, and examine it in the context of these existing theories. The review also aims to clarify the theories that relate to each EBT, and the relationships between theories and EBT constructs. By aligning existing theory with existing constructs, it is hoped that the framework of EBTs will obtain similar degrees of structure and clarity, to those being developed in the behaviour change field, including standardisation of terminology, and comprehensive classification, that can

be used to better inform and tailor weight management interventions (Marques et al., 2023).

The framework presented in **Chapter 2** is a start to addressing the aims. However, it is important to refine the framework and empirically test it, to examine whether it can be used to predict external outcomes. Research is needed that focuses on the constructs within the framework, as well as better understanding the explanatory power of EBTs. For example, whilst many studies report associations between EBTs and BMI or EI, associations between EBT and directly measured EI have not been extensively reviewed and little is known about how EBTs predict **objectively** measured markers of energy balance. **Chapter 3** addresses this through a systematic review and meta-analysis of EBTs. The primary aim of the review is to examine whether EBTs predict short-term EI, and whether this effect translates into longer term energy balance, as estimated by BMI. The review also examines if some EBT are better predictors of EI and BMI than others, and discusses methodological and conceptual limitations of studies using EBTs. For example, evaluating laboratory-based measures of EI and whether individuals can accurately assess their own eating behaviours.

Chapter 3 identifies several EBTs that are associated with EI and BMI. Both EI and BMI are examined because EI reflects a short-term change, whilst BMI reflects a longer-term change in energy balance. However, the studies included in the analysis measure cross-sectional associations, often at only one time point. This is a limitation because eating behaviour can change over time in response to various factors e.g. changes in lifestyle, and the analysis in **Chapter 3** is unable to capture any changes over time. **Chapter 4** addresses this limitation by utilising a longitudinal study design, which measures EBTs across an 18-month weight maintenance intervention. The aim of **Chapter 4** is to assess the use of the EBTs framework proposed in **Chapters 2 and 3,** which considers reflective, reactive, and homeostatic aspects of goal-orientated and motivated behaviours. Stepwise multiple regression and linear mixed models are used to examine whether EBTs that measure reflective, reactive and homeostatic eating behaviour are associated with weight change, and to examine changes in these EBTs between participants who lost, maintained and re-gained weight over 18-months. The longitudinal study design allows for a more

comprehensive analysis of how EBTs change over time, and in response to weight change.

Another strength of **Chapter 4** is that a better understanding of how EBTs change in response to lifestyle modifications, that lead to weight change, may help to improve the effectiveness of weight management options and interventions (Bray et al., 2017). Lifestyle modification interventions are often limited because there is wide variability in response to these interventions, such that some individuals are more successful in weight loss than others (Blundell et al., 2005; King et al., 2008). **Chapter 4** aims to identify which EBTs are associated with weight gain, and which are associated with weight loss and the size of effects involved. This analysis provides evidence that can support the development of individually tailored interventions to meet individual needs, by identifying individuals who are at risk of weight gain and/or re-gain and these individuals can be given additional support (Teixeira et al., 2005). Overall, the analysis in **Chapter 4** assesses the predictive power of EBTs over time, and evaluates the use of EBTs in weight management interventions.

Chapters 2 and 3 also highlight that current EBT measures do not capture all the relevant theoretical constructs related to eating behaviour, which further increases the need for a comprehensive framework of EBTs. However, it is important to empirically test the framework, to understand if it is robust across various samples and populations. Using exploratory and confirmatory factor analysis, **Chapter 5** measures 18 EBTs in two samples, including the general public and members of a weight management programme, to identify and validate latent factors that underlie EBTs. **Chapter 5** also aims to expand and extend previous work e.g. (Price et al., 2015; Vainik et al., 2015) by including a more diverse sample of EBTs, than previously examined, and also assessing whether the underlying factors can predict external outcomes. The analyses of **Chapter 5** are heavily shaped by the availability of EBT measurements, which means there is an unbalanced number of EBTs tested from each domain of eating behaviour, where there are many more reactive EBTs than EBTs from other domains analysed. The sample is also limited because it includes an uneven ratio of dieters, compared to the general population and females to males.

These limitations are addressed in **Chapter 6**, which builds on all previous chapters to refine and validate the factor structure of the framework for EBTs. This chapter takes an item-based approach to the analysis, similar to Vainik et al. (2015), which deconstructs the scales into their individual items, leading to a more balanced choice of measures. The analysis also includes a more diverse sample, including dieters and a UK representative sample based on age, gender and ethnicity, which improves the generalisability of the framework. Additionally, previous research has highlighted the potential importance of eating for pleasure (Berridge et al., 2010), which is also included in the framework in this chapter. Taken together, **Chapter 6** utilises insights from **Chapters 2-4,** which identify and evaluate psychological theories of eating behaviour in relation to EBTs, and examine the use of EBTs in obesity research and weight management interventions, to propose a comprehensive framework of EBTs. This chapter also uses insights from **Chapter 5**, which begins to empirically test the proposed framework of EBTs, to refine and balance it for improved stability across diverse populations. The aim is to produce a validated framework, that can be used to help identify individuals at risk of weight gain, and can be used to inform tailored interventions.

1.4. My contributions to the research in this thesis

The data used in this thesis are from the SatMap study (ISRCTN67732674) and NoHoW study (ISRCTN88405328). For the duration of my PhD, I was based in the School of Psychology at the University of Leeds. During my PhD I contributed to the design and management of the SatMap database and datasets, recruitment of participants and the day-to-day management of the SatMap study. Data from the SatMap study is used in **Chapters 5 and 6**.

For Paper 1 in **Chapter 2**, I was responsible for the conceptualisation, writing and visualisation of the conceptual review. I submitted the paper for publication in *Psychological Review* and led the process of responding to reviewer comments, with input from my co-authors.

For Paper 2 in **Chapter 3**, I was responsible for the conceptualisation and design of the systematic review and meta-analysis, and the generation of the review search terms. I developed the protocol for the review and pre-registered this on PROSPERO (CRD42021288694). I conducted the literature search, reviewed all articles, and trained my supervisors, as well as Dr Mark Hopkins, to assist with independent title and abstract screening and full text screening. Additionally, JS, GF and MH assisted with data-extraction by cross-checking for errors. I narratively synthesised the extracted data and was responsible for conducting the meta-analysis, using random-effects models. I was responsible for drafting the manuscript in collaboration with the other authors. All authors contributed to critically revising the subsequent version. I submitted the paper for publication in *Obesity Reviews* and led the process of responding to reviewer comments, with input from my co-authors. Upon acceptance, I also worked with the University of Leeds Library services to ensure open access charges were covered.

For Paper 3 in **Chapter 4**, I led the data-analysis, data curation, writing the original draft and visualisation of the data. All co-authors were involved in the review and editing of the paper. I submitted the paper for publication in *Appetite* and led the process of responding to reviewer comments, with input from my co-authors. Upon

acceptance, I also worked with the University of Leeds Library services to ensure open access charges were covered.

For Paper 4 in **Chapter 5**, I worked with my supervisory team, Dr Catherine Gibbons and Dr Mark Hopkins, to develop the SatMap 24 survey. In collaboration with the team, I developed the survey design using Qualtrics, and was involved in the recruitment of the general population and dieting sample. I led the conceptualisation, methodology, software, formal analysis, data curation, data visualisation and writing the original draft of the paper. I worked with my supervisory team to revise the manuscript. I submitted the paper for publication in *Appetite* and led the process of responding to reviewer comments, with input from my co-authors. Upon acceptance, I also worked with the University of Leeds Library services to ensure open access charges were covered.

For Paper 5 in **Chapter 6**, I led the design of the SatMap 300 survey. I was responsible for developing the ternary plot approach used to select the 312 foods used for the study. I developed the procedure for the inclusion criteria of foods to ensure that the food selection process identified a subset of foods, available to UK consumers, which are balanced according to the nutritional properties of the foods, are easily recognisable to UK consumers, and include a balance of single food items (e.g. apple) and meals (e.g. lasagne). I led the development of the standard operating procedures (SOPs) for the SatMap study. This included the food provision and preparation protocol and the food photography protocol. All 312 foods were prepared and photographed according to the SOPs by myself and assisted by another researcher (Heather Spinks).

I also led the selection process for the measures used in the SatMap 300 survey, alongside my supervisory team. I was responsible for the design of the survey using Qualtrics, including the recruitment material, participant information sheet, consent form and the survey itself. My supervisory team, Dr Catherine Gibbons and Dr Mark Hopkins were involved in developing and editing the survey. I led the ethical review process and completed the revisions requested by the University of Leeds, School of Psychology ethics board. I also worked with my supervisory team to recruit the participants who completed the survey. I led the conceptualisation, methodology,

software, formal analysis, data curation, data visualisation and writing the original draft of the paper. I worked with my supervisory team to revise the manuscript. I submitted the paper for publication in *Appetite* and led the process of responding to reviewer comments, with input from my co-authors.

1.5. References

Ackerman, P. L., & Heggestad, E. D. (1997). Intelligence, personality, and interests: evidence for overlapping traits. *Psychological bulletin*, *121*(2), 219.

Agha, M., & Agha, R. (2017). The rising prevalence of obesity: part A: impact on public health. *International journal of surgery. Oncology*, *2*(7), e17.

Allport, F. H., & Allport, G. W. (1921). Personality traits: Their classification and measurement. *The Journal of Abnormal Psychology and Social Psychology*, *16*(1), 6.

Allport, G. W. (1955). *Becoming: Basic considerations for a psychology of personality* (Vol. 20). Yale University Press.

Ashton, M. C., & Lee, K. (2007). Empirical, theoretical, and practical advantages of the HEXACO model of personality structure. *Personality and social psychology review*, *11*(2), 150-166.

Berridge, K. C., Ho, C.-Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: pleasure and desire circuits in obesity and eating disorders. *Brain research*, *1350*, 43-64.

Blundell, J., 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 & Behavior*, *152*, 473-478.

Blundell, J. E., Stubbs, R., Golding, C., Croden, F., Alam, R., Whybrow, S., Le Noury, J., & Lawton, C. (2005). Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. *Physiology & Behavior*, *86*(5), 614-622.

Bray, G., Kim, K., Wilding, J., & Federation, W. O. (2017). Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. *obesity reviews*, *18*(7), 715-723.

Brobeck, J. R. (1946). Mechanism of the development of obesity in animals with hypothalamic lesions. *Physiological Reviews*, *26*(4), 541-559.

Bryant, E. J., Caudwell, P., Blundell, J. E., Hopkins, M. E., & King, N. A. (2012). Psycho-markers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*, *58*(1). https://doi.org/http://dx.doi.org/10.1016/j.appet.2011.09.006

Bryant, E. J., King, N., & Blundell, J. E. (2008). Disinhibition: its effects on appetite and weight regulation. *obesity reviews*, *9*(5), 409-419.

Cannon, W. B. (1929). Organization for physiological homeostasis. *Physiological Reviews*, *9*(3), 399-431.

Drewnowski, A., & Popkin, B. M. (1997). The nutrition transition: new trends in the global diet. *Nutrition reviews*, *55*(2), 31-43.

Elfhag, K., & Rössner, S. (2005). Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *obesity reviews*, *6*(1), 67-85.

Eysenck, H. J., & Eysenck, S. B. G. (1975). *Manual of the Eysenck Personality Questionnaire (junior & adult)*. Hodder and Stoughton Educational.

Farooqi, I. S., & O'Rahilly, S. (2008). Mutations in ligands and receptors of the leptin–melanocortin pathway that lead to obesity. *Nature clinical practice Endocrinology & metabolism*, *4*(10), 569-577.

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.

GBD, R. F. C. (2019). Global Burden of 87 Risk Factors in 204 Countries and Territories, 1990–2019: a systematic analysis for the global burden of disease study 2019. *Lancet*, *2020;396:1223–1249*.

Goldberg, L. R. (1981). Language and individual differences: The search for universals in personality lexicons. *Review of personality and social psychology*, *2*(1), 141-165.

GOV. (2023). *Obesity Profile: short statistical commentary May 2023*. Retrieved 20/03/24 from https://www.gov.uk/government/statistics/obesity-profile-update-may-2023/obesity-profile-short-statistical-commentary-may-2023

Greaves, C. J., Sheppard, K. E., Abraham, C., Hardeman, W., Roden, M., Evans, P. H., & Schwarz, P. (2011). Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions. *BMC Public Health*, *11*(1), 1-12.

Hall, K. D., Farooqi, I. S., Friedman, J. M., Klein, S., Loos, R. J., Mangelsdorf, D. J., O'Rahilly, S., Ravussin, E., Redman, L. M., & Ryan, D. H. (2022). The energy balance model of obesity: beyond calories in, calories out. *The American Journal of Clinical Nutrition*, *115*(5), 1243-1254.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of personality*.

Hervey, G. (1969). Regulation of energy balance. *Nature*, *222*, 629-631.

Kennedy, G. C. (1953). The role of depot fat in the hypothalamic control of food intake in the rat. *Proceedings of the Royal Society of London. Series B-Biological Sciences*, *140*(901), 578-592.

King, N. A., Hopkins, M., Caudwell, P., Stubbs, R., & Blundell, J. (2008). Individual variability following 12 weeks of supervised exercise: identification and characterization of compensation for exercise-induced weight loss. *International Journal of Obesity*, *32*(1), 177-184.

Llewellyn, C., & Wardle, J. (2015). Behavioral susceptibility to obesity: gene–environment interplay in the development of weight. *Physiology & Behavior*, *152*, 494-501.

Lowe, M. R., & Levine, A. S. (2005). Eating motives and the controversy over dieting: eating less than needed versus less than wanted. *Obesity research*, *13*(5), 797-806.

Marques, M. M., Wright, A. J., Corker, E., Johnston, M., West, R., Hastings, J., Zhang, L., & Michie, S. (2023). The behaviour change technique ontology: transforming the behaviour change technique taxonomy v1. *Wellcome open research*, *8*.

Mayer, J. (1953). Glucostatic mechanism of regulation of food intake. *New England Journal of Medicine*, *249*(1), 13-16.

Mellinkoff, S. M., Frankland, M., Boyle, D., & Greipel, M. (1956). Relationship between serum amino acid concentration and fluctuations in appetite. *Journal of applied physiology*, *8*(5), 535-538.

Michie, S. (2014). ABC of behaviour change theories. *(No Title)*.

Michie, S., Van Stralen, M. M., & West, R. (2011). The behaviour change wheel: a new method for characterising and designing behaviour change interventions. *Implementation science*, *6*(1), 1-12.

Neel, J. V. (1962). Diabetes mellitus: a “thrifty” genotype rendered detrimental by “progress”? *American journal of human genetics*, *14*(4), 353.

Panayi, P. (2008). *Spicing up Britain: The multicultural history of British food*. Reaktion books.

Paulhus, D. L., & Vazire, S. (2007). The self-report method. *Handbook of research methods in personality psychology*, *1*(2007), 224-239.

Poulain, J.-P. (2017). *The sociology of food: eating and the place of food in society*. Bloomsbury Publishing.

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.

Racine, S. E., Hagan, K. E., & Schell, S. E. (2019). Is all nonhomeostatic eating the same? Examining the latent structure of nonhomeostatic eating processes in women and men. *Psychological Assessment*, *31*(10), 1220.

Raubenheimer, D., & Simpson, S. J. (2019). Protein leverage: theoretical foundations and ten points of clarification. *Obesity*, *27*(8), 1225-1238.

Rozin, P. (1997). Why we eat what we eat, and why we worry about it. *Bulletin of the American Academy of Arts and Sciences*, *50*(5), 26-48.

Schachter, S. (1967). Cognitive effects on bodily functioning: Studies of obesity and eating. *Glass, D.(Ed.): Neurophysiology and Emotion, New York (Rockefeller University Press), pp. 117-159.*

Sobal, J. (1999). Food system globalization, eating transformations, and nutrition transitions. *Food in global history*, 171-193.

Sobal, J. (2001). Social and cultural influences on obesity. *International textbook of obesity*, 305-322.

Speakman, J. R., Levitsky, D. A., Allison, D. B., Bray, M. S., De Castro, J. M., Clegg, D. J., Clapham, J. C., Dulloo, A. G., Gruer, L., & Haw, S. (2011). Set points, settling points and some alternative models: theoretical options to understand how genes and environments combine to regulate body adiposity. *Disease models & mechanisms*, *4*(6), 733-745.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and social psychology review*, *8*(3), 220-247.

Stubbs, J. R., Horgan, G., Robinson, E., Hopkins, M., Dakin, C., & Finlayson, G. (2023). Diet composition and energy intake in humans. *Philosophical Transactions of the Royal Society B*, *378*(1888), 20220449.

Stubbs, R., Gail, C., Whybrow, S., & Gilbert, P. (2012). The evolutionary inevitability of obesity in modern society: implications for behavioral solutions to weight control in the general population. In *Obesity and weight management:*

Challenges, practices and health implications (pp. 29-50). Nova Science Publishers Inc.

Stubbs, R. J., & Tolkamp, B. J. (2006). Control of energy balance in relation to energy intake and energy expenditure in animals and man: an ecological perspective. *Br J Nutr*, *95*(4), 657-676. https://doi.org/10.1079/bjn20041361

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Teixeira, P., Going, S. B., Sardinha, L., & Lohman, T. (2005). A review of psychosocial pre-treatment predictors of weight control. *obesity reviews*, *6*(1), 43-65.

Vainik, U., Garcia-Garcia, I., & Dagher, A. (2019). Uncontrolled eating: a unifying heritable trait linked with obesity, overeating, personality and the brain. *The European journal of neuroscience*, *50*(3). https://doi.org/https://dx.doi.org/10.1111/ejn.14352

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229-239.

Warde, A. (2016). *The practice of eating*. John Wiley & Sons.

Warren, J. M., Smith, N., & Ashwell, M. (2017). A structured literature review on the role of mindfulness, mindful eating and intuitive eating in changing eating behaviours: Effectiveness and associated potential mechanisms. *Nutrition Research Reviews*, *30*(2). https://doi.org/http://dx.doi.org/10.1017/S0954422417000154

Watts, A. G., Kanoski, S. E., Sanchez-Watts, G., & Langhans, W. (2022). The physiological control of eating: signals, neurons, and networks. *Physiological Reviews*, *102*(2), 689-813.

Chapter 2. Exploring the Underlying Psychological Constructs of Self-Report Eating Behaviour Measurements: Towards a Comprehensive Framework.

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2.1. Abstract

Food and eating are fundamental for survival, but also have significant impacts on health, psychology, sociology, and economics. Understanding what motivates people to eat can provide insights into ‘adaptive’ eating behaviour, which is especially important due to the increasing prevalence of health-related conditions such as obesity. There has been considerable interest in developing theoretical models and associated constructs that explain individual differences in eating behaviour. However, many of these models contain overlapping theories and shared theoretical mechanisms of action. Currently, there is no recognised standard framework that integrates psychological, physiological, and neurobiological theory to help explain human eating behaviour. The aim of the current paper was to review key psychological theories in relation to energy balance homeostasis, energy intake and motivation to eat and begin to develop a comprehensive framework of relevant factors that drive eating behaviour.

The key findings from this review suggest that eating behaviour is conceptualised by elements of dual process models which include conscious processing (reflective factors), and automatic responses to desires, environmental cues, habits, and associative learning. These processes are mediated by neurobiology and physiological signalling (homeostatic feedback) of energy balance, which is more tolerant of positive than negative energy balances. From a synthesis of available evidence, it is suggested that eating behaviour constructs (traits) can be explained by three latent constructs: reflective, reactive, and homeostatic eating. By understanding the interplay between reflective, reactive, and homeostatic processes, interventions can be developed that tailor treatments to target key aspects of eating behaviour.

2.2. Introduction

Eating behaviour encompasses a complex array of physiological, psychological, environmental, and cultural factors. Food and eating are central to everyday life (Warde, 2016), whereby their meaning and usages extend far beyond nutritional maintenance (Rozin, 1999). Food is a social vehicle, it involves symbolic functions, it takes on moral significance and is a medium for aesthetic expression (Rozin, 2005). Shifts in the food system have led to growing interest in food, eating and body weight across the globe, partly due to the increasing prevalence of health-related diseases that are attributable to foods and diets (Warde, 2016). Part of the complexity in understanding human eating behaviour involves the psychology of why we eat. Thus, understanding what motivates people to eat can help to shed light on what adaptive eating behaviour involves, as well as, what motivates people to under or overeat.

Part of the complexity involved in understanding the causes of obesity is the psychology of eating behaviour. Researchers have developed numerous theoretical models and generated a panoply of constructs that explain the factors that motivate eating behaviour and facilitate overeating. These 'eating behaviour traits' (EBT), quantified by psychometric, self-report instruments, relate aspects of eating behaviour to overeating and obesity (French et al., 2012) and are considered to be reliable indices of food-related behaviours (Llewellyn & Wardle, 2015). EBTs can also be used to phenotype individuals based on their endorsement of specific eating behaviour motivations, allowing for the examination of the moderators and mechanisms that contribute to overeating and obesity. However, several theories of eating behaviour share similar constructs and proposed mechanisms of action. This is a source of confusion since the psychometric and predictive validity of specific EBTs are unknown. Further, researchers use different measures for the same purposes as well as the same measures for different purposes which limits comparability of studies, meta-analyses, and scientific insight. For example, questionnaires that measure emotional eating are used to assess both emotional eating and have been used as proxy measures of food intake (Devonport et al., 2019). This use of different questionnaires which measure the same constructs is termed a jangle fallacy (Kelley, 1927) and previously it has been suggested that

jangle fallacy is common in obesity research (Vainik et al., 2015). The use of varying questionnaires also makes it challenging to compare studies that use different questionnaires for the same or similar EBTs. These numerous scales could be adding unnecessary heterogeneity in cause-effect relationships and adding unnecessary burden on participants who are required to fill in multiple, time-consuming measures.

Currently there is no agreed framework or standardised set of measures that describe motivations for eating. Thus, the aim of this conceptual paper is to draw from key psychological, physiological, and neurobiological models, as a starting point to develop a comprehensive framework that identifies the core, latent constructs underlying EBTs. In a previous systematic review and meta-analysis conducted by our research team, we identified several constructs that measure EBTs and tested the extent to which these EBTs were associated with BMI and energy intake (Dakin et al., 2023a). Having examined these EBTs, we identified that not all the concepts related to motivations for eating are covered in many of the current EBTs measures. Therefore, the purpose of this review is to propose a provisional conceptual framework and examine it in the context of existing psychological theories, which covers some of the areas missed by the previous systematic review.

It is important to clarify the theories that relate to each EBT, and the relationships between theories and EBT constructs. Thus, while the field of EBTs may not need any new theories, there is a need to better understand the measures that currently exist, how they relate to each other, how they relate to physiology and neurobiology, and as trait measures, longer term indices of energy balance. An assumption we make is that most longer-term changes in energy balance are driven by behaviour (Stubbs et al., 2023). This framework examines the underlying common constructs that are shared between numerous overlapping EBT measurements and attempts to align existing theory with existing EBT constructs.

The approach of aligning these empirically derived constructs with existing theory allows for clarification and standardisation of measurements which will enable better comparisons of studies, analyses and effect sizes which cannot currently be determined because the field is overwhelmed with overlapping measurements

(Michie, 2014). The measurement of physiological variables and the integration of objective tracking of eating behaviour with self-report measures of eating behaviour is important (Stubbs et al., 2021). However, the current paper is constrained to a discussion of self-report measures of EBTs. This does not diminish the importance of other types of measures, but these are not the focus of the current paper.

This paper begins with some key definitions of human appetite and eating behaviour to establish the discussion parameters (see section 2.2.1). The next sections summarise and evaluate key psychological theories (section 2.3), physiological (section 2.4) and neurobiological models that have been proposed to explain motivations for eating (section 2.5). Lastly, the constructs from these scientific domains will be synthesised to propose a framework that can be used to describe and quantify human EBT measurements (section 2.6).

2.2.1. Human appetite and eating behaviour

Human appetite is often used rather loosely to describe quantitative and qualitative aspects of motivation to eat and ingestive behaviour. In its broadest definition, appetite covers the whole field of food intake, food selection, motivation, and preference. This includes qualitative aspects of eating, sensory aspects or responsiveness to environmental stimulation and eating in response to physiological stimuli and energy deficit. It is also common to see appetite sub-divided into: 'Homeostatic' – that is regulated and links the physiological needs for energy and nutrients with the behaviour that satisfies these needs (eating), shaped by physiological **excitatory** and **inhibitory** signals; and 'hedonic' – that is reward-driven signals and links pleasant thoughts and cravings about food and sensory appreciation of certain food attributes with the expression of food preference and choice. Hedonic appetite reflects both the liking and wanting for food. Eating is a measurable form of motivated behaviour and motivations to eat can be biologically driven, as well as environmentally influenced e.g., social motives to eat (Watts et al., 2022). The psychobiology of motivation to eat, as with other goal-oriented motivated behaviours follows cycles of anticipation, consummation and cessation which can be influenced by various mechanisms, described as follows:

Hunger (motivation to eat)

Hunger is defined as the experienced subjective motivation to consume which includes provoking and sustaining a behavioural response of eating, often believed to be in response to a biological need. Hunger functions as an intervening variable in the stimulus-response sequence that is responsible for initiating eating (Watts et al., 2022). While hunger initiates eating, there is no single mechanism of hunger.

Satiation (termination of eating)

The processes that occur during a meal that generate negative feedback which lead to the termination of a meal is termed satiation (within-meal inhibition).

Satiety (lack of motivation to eat)

Satiety is defined as between-meal inhibition. Satiety further supresses the drive to consume and determines the length of the inter-meal interval (supresses post-meal intake).

Energy balance

Energy balance is the difference between energy intake and energy expended and excreted over a given period of time (Blaxter, 1989). Therefore, energy storage is equal to intake minus expenditure:

$$\text{Energy Intake} - \text{Energy of Faeces} - \text{Energy of Urine} - \text{Energy of Combustible Gas} = \text{Energy Produced}$$

This equation is frequently simplified to give:

$$\Delta\text{Energy Storage} = \Delta\text{Energy Intake (EI)} - \Delta\text{Energy Expenditure (EE)}$$

Dietary macronutrients summate to determine energy intake and influence energy balance regulation in humans through physiological effects and voluntary food intake (Stubbs et al., 2023). Macronutrients can act as powerful unconditioned stimuli, modifying feeding responses and acting as cues for learned food preferences related to pleasure (Berridge & Kringelbach, 2008; Kringelbach et al., 2012). Energy expenditure (total daily energy expenditure, TDEE) is the heat released by the body through resting metabolism (resting metabolic rate, RMR), the thermic effect of food (TEF), physical activity (PA), and non-exercise activity energy expenditure (NEAT)

(Kleiber, 1961). Energy storage is the potential chemical energy which is mostly stored as fat, but can also be stored as glycogen, protein, and heat energy due to body temperature changes (Kenny et al., 2008). The pathways to, and from obesity appear to be largely behavioural, involving multiple mechanisms (Stubbs et al., 2023). Indeed, there is consensus that increased population-level obesity development is primarily caused by excessive energy intake (Prentice et al., 1989). This means that the key intervention targets by which eating behaviour and energy balance can be altered appear to be largely related to energy intake rather than energy expenditure (Stubbs et al., 2023). As such, several theories have been developed to explain how several different factors of the act as mechanisms to influence food and energy intake (Blundell & Stubbs, 1999; Blundell & Tremblay, 1995; Prentice et al., 1989; Stubbs, 1998).

Eating behaviour traits

Eating behaviour traits (EBT) are constructs that have been theoretically developed to explain a defined eating style or disposition to eat. They are measured using self-report, psychometric questionnaires such as the Three-Factor-Eating Questionnaire, which measures three EBTs: dietary restraint, eating disinhibition and susceptibility to hunger (Stunkard & Messick, 1985). While they are not direct measures of eating behaviour, EBTs are considered to be reliable indices of individual differences in motivation to eat or not eat for different reasons (Llewellyn & Wardle, 2015). For example, the child eating behaviour questionnaire (CEBQ) which measures several EBTs including responsiveness and satiety responsiveness has been validated using behavioural measures (Carnell & Wardle, 2007) and has been shown to be stable over time (Ashcroft et al., 2008). Llewellyn and Wardle (2015) have also shown that EBTs play a causal role in excess weight gain. For example, a population-based paediatric birth cohort of twins has been used to prospectively test the hypothesis that appetite plays a casual role in the development of obesity. The results found that high food responsiveness and low satiety responsiveness predisposes children to obesity. The use of longitudinal data made it possible to test the causal direction between appetite and adiposity. Furthermore, EBTs have also been used to characterise eating behaviour in relation to disordered eating patterns and predisposition to obesity (French et al., 2012).

2.3. Psychological accounts of eating behaviour

In this section, key psychological theories are discussed in relation to eating behaviour. Their central elements, key constructs and mechanisms of action are presented in Table 2.1.

2.3.1. Psychosomatic theory

Kaplan and Kaplan (1957) argue that the development of obesity is attributed to overeating, caused by emotional disturbances that serve to increase food intake, and this is termed a psychosomatic disorder. The underlying assumptions of psychosomatic theory originate from learning theory (Dollard & Miller, 1950; Mowrer, 1950), whereby overeating is viewed as a learned behaviour which is acquired as a strategy for reducing anxiety. The authors proposed two types of abnormal overeating: one which is characterised by feelings of hunger and another that is not associated with excessive hunger and is instead attributed to avoidance of anxiety (Kaplan & Kaplan, 1957). A normal response to negative emotions such as anxiety, fear and anger is loss of appetite (Cannon, 1953). However, some individuals respond to these emotions by excessively overeating.

Compulsive overeating can be understood from the perspective of drive reduction because eating can reduce a drive (e.g., hunger) and is rewarded and therefore learned. Emotions can also become drive states, meaning that eating can be associated with a reduction of negative emotions such as anxiety, and thus the act of eating is rewarded. This means an individual can learn to eat in response to hunger and anxiety, which could lead to overconsumption and obesity. Bruch (1964) further suggested that hunger cues can be confused with emotional states which can result in excessive overeating. She also highlighted the importance of early learning experiences that play a role in associating hunger with a pattern of cues that lead to eating behaviour. For example, if an individual comes from a home where their mother uses food as a means of control, it may increase their chance of using food to reduce anxiety (Bruch & Touraine, 1940).

While Kaplan and Kaplan (1957) argue that overeating reduces negative emotions, and there is evidence that individuals may use food as means of reducing anxiety/stress e.g. (Bruch & Touraine, 1940), there are limitations to this theory. One issue with the anxiety-reduction explanation of obesity is that if overeating is driven by an aversive state and also acts to reduce an aversive state, it is unclear what is maintaining and reinforcing the behaviour. Robbins and Fray (1980) argue that overeating in individuals with obesity cannot be maintained by anxiety reduction and instead, the overeating behaviour acquires its own positive reinforcing properties, termed food reward. Furthermore, psychosomatic explanations cannot account for evidence that some individuals reduce their eating when experiencing high levels of stress. The authors conclude that overeating can be provoked by stress, but that eating itself does not act to reduce stress which disagrees with psychosomatic theory. Overall, the idea that an individual can learn to overeat as a means of anxiety reduction can explain the eating behaviour of some individuals with obesity. However, there are flaws and limited generalisability with this explanation of obesity, which has led to other theories emerging in the literature.

2.3.2. Externality theory

Externality theory suggests that eating behaviour in individuals with obesity is influenced by external cues (which are unrelated to nutritional need states) and less influenced by internal cues. In contrast, normal weight individuals are influenced by both internal and external cues (Schachter, 1967). Responsiveness to external cues was proposed as a direct function of weight, with responsiveness to internal cues as an inverse function of weight (Nisbett, 1968b). Evidence supports the internal hypothesis, showing weaker correlations between gastric mobility and hunger in individuals with obesity (Stunkard & Koch, 1964). Furthermore, individuals with obesity did not increase food intake after deprivation, while normal weight individuals did (Schachter et al., 1968), suggesting that they are less able to respond to their internal/physiological cues of hunger. However, some studies contradict this theory, finding no difference in internal regulation between weight groups (Wooley, 1972) and poor regulation of food intake in normal weight individuals as well (Spiegel, 1973).

Nisbett (1968a) tested the external hypothesis and found that participants with obesity ate more when presented with more food, while normal and underweight participants did not change their intake depending on the amount of food presented. Similarly, manipulating a clock time determined the amount eaten in individuals with obesity but had no effect for normal weight participants (Schachter & Gross, 1968). This suggests that individuals with obesity are more susceptible to external cues and immediate food stimuli. Externality theory has been criticised for being too simplistic (Rodin, 1981). For example, externality can be found in individuals in all weight categories which can lead to overeating but only under specific conditions. Indeed, Meyers and Stunkard (1980) found no evidence to support the hypothesis that individuals with overweight or obesity are more responsive to food cues than individuals who do not live with overweight or obesity. Additionally, internal sensitivity is not a unique characteristic of individuals within a normal weight category. This means that degree of weight gain and obesity is dependent on several factors, not just externality. Rodin (1981) also suggests that the simplistic internal-external dichotomy is not empirically supported. Additionally, the key studies in support of externality theory used relatively small samples sizes and short study durations (one test day). This has led to the development of other theories that address its limitations.

2.3.3. Set-Point Theory

Extending from externality theory, Nisbett (1972) argued that some individuals with obesity are below their biological obese “set-point”, and are hungry because they are attempting to keep their weight below this set-point. From a physiological perspective, the set point model mainly focuses on the importance of fat mass for the feedback loop. This is supported by the discovery of leptin and the associated pathways that provide the link between adipose tissue and the central nervous system (CNS) (Campfield et al., 1996). Set-point theory is based on the belief that hypothalamic centres defend adipose tissue set-points and these set-points have different baselines in each individual. This suggests that an individual could be living with obesity but be below their biological set point which the CNS is trying to defend. If an individual without obesity is below their set-point, their eating behaviour should reflect that of an individual with obesity, assuming the person with obesity is below

their own “very-obese set point”. However, if an individual with obesity is at their set-point, their eating behaviour should reflect normal eating. Nisbett (1972) suggests that an individual with obesity who is below their set-point, is in a state of deprivation and this is what generates increased responsiveness to external cues.

Set-point theory gained attention from researchers, however, since the 1970’s the proportion of the population who are obese has risen dramatically, and this rise is projected to continue (Agha & Agha, 2017). Applying set-point theory, this would indicate that a significant proportion of the population has a set point in the obese range. Set-point theory does not account for why there is an increasing number of individuals with an “obese set-point.” Furthermore, there is a lack of evidence to support the main components of this theory and limited capacity for the theory to identify an individuals purported set point. In a study testing responsiveness to external food cues before and after weight loss through dieting, Rodin et al. (1977) found that across three experiments, weight loss was unrelated to changes in responsiveness to external cues. Experiment one included 85 females with overweight who completed 8 weeks of daily exercise and restriction of food intake. The intervention led to a mean weight loss of 12.55kg in participants with obesity, 8.82kg in participants with overweight and 2.55kg in normal weight participants. The results showed that externality scores change very little before and after weight loss and that any changes that did occur were not related to the amount of weight lost. Overall, in contrast to Nisbett’s hypothesis, the authors conclude that external responsiveness is not a function of degree of obesity.

More recently, other models have been presented to address the limitations of a strict set-point model. The settling point model assumes there is no active metabolic regulation of body weight but behaves as if body weight is being regulated, due to passive regulation of body weight “at a point defined by the level of the unregulated parameter (either inflow or outflow)” (Speakman et al., 2011). The thrifty gene hypothesis suggests that there is an adaptative usefulness of increased energy storage (Neel, 1962; Wendorf & Goldfine, 1991). Furthermore, the dual intervention point model (Speakman, 2007) suggests there are upper and lower boundaries that distinguish the points where active physiological regulation occurs. Between these points, there is weak or no physiological regulation of weight. Lastly, the general

model of intake regulation (de Castro & Plunkett, 2002), proposes that intake is influenced by uncompensated factors that are not influenced by intake and compensated factors that are. This model does not assume there are any set points for body weight or intake and instead assumes that the level of any factor that is defended is flexible. A change in one or more factors can produce a new defended level. These models also assume an asymmetric defence of the body weight set-point such that negative energy balance is defended more aggressively than positive energy balance (Levitsky, 2005).

2.3.4. Restrained Eating

Herman and Mack (1975) expanded Nisbett's set-point theory to explain the eating behaviour of normal weight individuals. They proposed that many normal weight individuals are “biologically underweight” (below their biological set point), meaning they should overeat, because they are below their set point. However, societal pressures lead them to restrain their eating. Restraint theory suggests that individuals who differ in their concern about weight will react differently to restraint manipulation. In an initial experiment, participants with high levels of restrained eating (measured using the restraint scale) consumed more ice cream after a milkshake preload, while those with low levels of restrained eating showed internal regulation, consuming less ice cream with a larger preload. This study indicates that highly restrained individuals exhibit external eating behaviour when restrained eating is discarded. The results suggest that it is high levels of restrained eating that leads to external eating, meaning individuals with and without obesity can be externally responsive. Therefore, eating patterns like restrained eating may be better predictors of eating behaviour than body weight.

Herman and Mack’s restraint theory sparked interest in using self-report measures of eating patterns (EBT) as a way of explaining and predicting eating behaviour. While the concept of restrained eating does have supporting evidence, this construct is only a descriptive term and does not provide a mechanism for eating behaviour. Overall, the predictions of restraint theory are not fully supported by the literature. For example, restraint theory suggests that engaging in restrained eating stimulates counter-regulatory responses which can cause disinhibited eating (overeating in

response to different stimuli) and binge eating behaviour (Herman & Mack, 1975; Herman & Polivy, 1980). Restraint theory proposes that individuals who engage in restrained eating may feel deprived (perceived deprivation) and this could make them vulnerable to overconsumption and weight gain. But it does not elaborate on the nature of the deprivation e.g. physiological deprivation, deprivation of rewards or failure to fulfil other psychological needs. However, other evidence suggests this effect is in the opposite direction, meaning that high levels of restrained eating is not a cause of overeating. Instead, it is the observed consequence of unsuccessful weight management attempts (Schaumberg et al., 2016). There is also evidence against the proposal that restrained eating is associated with overeating and obesity. For example, engaging in a sustained effort to control and monitor food intake (restrained eating) is associated with successful weight maintenance (Johnson et al., 2012). It has since been suggested that inconsistent restrained eating (periods of successful restrained eating followed by disinhibition) can predict the onset of eating disorders and overeating. However, restrained eating that is characterised by consistent and sustainable energy restriction is associated with weight loss and weight maintenance, without eating disorder risk (McGuire et al., 1999; Phelan et al., 2009).

More recently, Polivy et al. (2020) highlight that defining restrained eating as a construct is a complicated endeavour because research suggests that different questionnaires used to identify restrained eaters may be assessing different constructs (Williamson et al., 2007). The initial restrained eating studies attempted to determine how chronic dieters behaved in various challenging situations. The original concept of restrained eating as measured by the restraint scale (Herman & Polivy, 1980), was proposed as a way of identifying individual differences in the motivation to cognitively control body weight, with initial suppression of food intake which then succumbs to temptation, overeating and emotional responses. This means restrained eating was defined as a cycle of restriction and indulgence as a reaction to the demands of chronic dieting (Polivy & Herman, 1985). Consequently, restrained eating was identified as a construct (EBT).

Since the development of the restraint scale (Herman & Mack, 1975), other questionnaires have been developed to measure restrained eating such as dietary

restraint as measured by the TFEQ (Stunkard & Messick, 1985) and restrained eating as measured by the DEBQ (Van Strien et al., 1986). However, these questionnaires use somewhat different defining measures. TFEQ restraint primarily aims to identify successful dieters who can control their intake (restraint is weighted positively), and DEBQ restraint identifies attempts to control intake and the types of cues that hinder restraint (Polivy et al., 2020, 2023). These scales measure successful restriction over time without measuring the other dimensions (e.g. disinhibition when inhibition fails) that the restraint scale measures that were seen as critical to restraint theory.

Polivy et al. (2023) suggest that because the TFEQ and DEBQ remove disinhibition, emotional or externally driven eating out of the measurement of restrained eating, they are measuring a different construct from that of restrained eating as measured by the restraint scale. Consequently, these questionnaires may be measuring a construct that could be better termed as restrictive eating (Polivy et al., 2020). In support of this, research has found that the restraint scale is better at identifying individuals who struggle to control their food intake (Adams et al., 2019), and only the restraint scale can consistently identify dieters who overeat in response to emotions (Polivy et al., 2020). In contrast, the TFEQ is more consistently able to predict dietary success over time (Keller & Hartmann, 2016). The difficulty in defining the construct of restrained eating suggests that while dieters share the same goal of losing or maintaining a lower body weight, they differ in personality, motivation, ability, behaviours, attitudes, and self-image.

This conceptual ambiguity needs to be considered when researchers decide which questionnaire to use. It appears that the TFEQ or DEBQ are more appropriate for understanding successful caloric restriction. Whereas assessing cycles of restriction followed by disinhibited behaviour and overconsumption are better predicted by the restraint scale and eating disorder examination questionnaire (EDE-Q) (Fairburn & Beglin, 1994). Linking back to restraint theory, the evidence now highlights that dieting does not necessarily lead to an individual becoming a restrained eater (Mills et al., 2021). This means that dieting and restrained eating should not be treated as the same construct. The construct of restrained eating as measured by the restraint scale does have supporting evidence that engaging in this type of restrained eating

can lead to overconsumption and obesity. However, other constructs, which are not the same, do not support restraint theory. However, this could be because questionnaires such as the TFEQ and DEBQ are not measuring restrained eating and are instead measuring restrictive eating.

2.3.5. EBT developed from theory - Restraint, Disinhibition and Susceptibility to Hunger (Three Factor Eating Questionnaire)

Since the development of the restraint scale, other questionnaires have been developed that aim to measure different dimensions of eating behaviour. The Three Factor Eating Questionnaire (Stunkard & Messick, 1985) was heavily influenced by the concept of restrained eating and was constructed to measure three dimensions of eating behaviour. As previously described, individuals with high levels of restrained eating (as measured by the restraint scale) consumed more of a test meal after consuming a preload than individuals with low levels of restrained eating (Herman & Mack, 1975; Herman & Polivy, 1980), and this behaviour was termed 'counter-regulation.' Herman and Polivy (1983) proposed the boundary model of eating behaviour which can help to explain why high levels of restrained eating, as measured by the restraint scale, is associated with counter-regulation after consumption of a preload.

The boundary model suggests that eating is controlled by two boundaries along a continuum: the hunger zone and satiety zone. The diet boundary (which lies between the hunger and satiety boundary) represents a self-imposed, cognitive limit on food intake, designed to stop food intake before an individual reaches satiety. This model suggests that restrained eating requires cognitive motivation to limit food intake to reduce or control body weight. This definition of restrained eating also includes feelings of guilt after episodes of overeating. The initial restrained eating studies attempted to examine how restrained eaters behaved when presented with a disinhibitor, for example, alcohol (Polivy & Herman, 1976b), dysphoric emotions e.g., depression (Polivy & Herman, 1976a) and anxiety (Herman & Polivy, 1975). The results showed that these restrained eaters initially supressed their intake through cognitive efforts, but then surrendered to temptation and overeating, meaning restrained eating and disinhibition are both key concepts to restrained eating.

As mentioned in section 2.3.4, there are issues with the construct of restrained eating. However, Stunkard and Messick (1985) argue these limitations are with the scale used to measure restraint, not the concept itself, which led them to develop a new instrument to measure restrained eating and related issues which was initially derived from the revised restraint scale (Herman & Polivy, 1980) and the Latent Obesity Questionnaire (Pudel et al., 1975). Additionally, 17 new items were created based on clinical experience. In their analysis, three factors were discovered and were interpreted as: cognitive control of eating behaviour, disinhibition of control, and susceptibility to hunger. Later analysis of the TFEQ by Westenhoefer et al. (1999) identified two dimensions of restrained eating which were named flexible and rigid restraint. Rigid control was associated with higher disinhibition scores and BMI and more severe binge eating episodes, while flexible control was associated with lower disinhibition scores and BMI and less severe binge eating episodes. More recently, Westenhoefer et al. (2013) found that flexible restraint was associated with better weight loss maintenance, while rigid restraint was associated with less weight loss. However, other studies have failed to replicate these associations, which questions the utility of the distinction between flexible and rigid restraint. For example, Masheb and Grilo (2002) found that flexible and rigid restraint were significantly correlated with each other and both negatively correlated with BMI in binge eating disorder patients, which suggests that this distinction may not be useful for these patients.

There is evidence that the three EBTs measured in the TFEQ are associated with body weight and obesity. A literature review found consistent evidence that disinhibition is positively associated with BMI and fat mass (Bryant et al., 2019). However, the role of restraint using the TFEQ is also conflicting, whereby, studies have found both positive and negative relationships with BMI. In a meta-analysis of the association between EBT, BMI and energy intake, susceptibility to hunger and disinhibition were significantly and positively correlated with BMI and EI, with moderate effect sizes. The role of restrained eating using all available measures was again conflicting, as restrained eating was positively associated with BMI but negatively associated with energy intake (Dakin et al., 2023a). Consequently, the mechanisms that underlie why there are differences in body weight and composition in individuals with different restrained eating and disinhibition scores are not clear but could be partially explained by the interaction between disinhibition and restrained

eating and other factors such as eating patterns, diet quality and eating responses to prompts and cues not measured by these scales (Bryant et al., 2019).

2.3.6. Restrained Eating, Emotional Eating, and External Eating (Dutch Eating Behaviour Questionnaire)

The Dutch Eating Behaviour Questionnaire (DEBQ), similarly to the TFEQ, was developed as another attempt to integrate constructs across different theories of eating behaviour, including Psychosomatic theory (see section 2.3.1), Externality theory (see section 2.3.2), and Restraint theory (see section 2.3.4). Both Psychosomatic and Externality theory attribute the internal state (either emotional eating or a high degree of externality) as a causal factor in developing obesity. However, a major limitation of both theories is that a tendency to emotionally eat or a high degree of externality does not always lead to weight gain and high levels of emotional and external eating can be found in all weight categories (Rodin, 1975; Rodin, 1978). Restraint theory tried to account for these limitations (Herman & Mack, 1975; Herman & Polivy, 1980) and also suggested that external and emotional eating are consequences of dieting (Herman & Mack, 1975). All three theories have been tested, however, as previously described there are limitations with each theory including conflicting and inconsistent findings and lack of replications.

The aim of the DEBQ was to develop a homogenous scale of restrained eating, emotional and external eating to improve understanding of eating patterns in individuals with obesity. The authors identified that the TFEQ was a major improvement on previous scales. However, they note that at the time of their study, the TFEQ was not available. The DEBQ has been widely used since its development, and in general, the theoretical structure of this questionnaire has been confirmed (Cebolla et al., 2014). However, some items in the scales have been found to be problematic including cross-loadings and some negligible loadings onto their proposed factors (Barrada et al., 2016; Cebolla et al., 2014). The implication of using the DEBQ with these problematic items is that they could be adding noise when using this scale for research or clinical analysis. In both studies, the authors suggest that deleting problematic items could increase the reliability of the scale and better distinguish between different dimensions such as boredom and emotional

eating. Additionally, although the DEBQ has been widely used to assess self-reported emotional eating, multiple laboratory studies have found that emotional eating as measured by the DEBQ is unrelated to eating in response to emotional stimuli (Braden et al., 2020; Domoff et al., 2014). These findings suggest possible concerns with the validity of the DEBQ to measure the construct of emotional eating. Therefore, as previously suggested with the construct of restrained eating, the type of questionnaire used to measure emotional eating may be an important consideration for researchers.

2.3.7. Hedonic Hunger (Power of Food Scale)

Another development of an EBT from restraint theory is the concept of hedonic hunger (Lowe & Levine, 2005). In the literature, there is often a distinction made between physiological (the result of short-term energy reduction) and psychological hunger (driven by psychological rather than energy needs e.g., eating when not hungry because of environmental cues, to avoid negative emotions or simple pleasure). This dual-factor perspective is referred to as the standard model of hunger (Lowe & Levine, 2005). There is also a distinction in the brain between the homeostatic system which is activated by energy deficits and the hedonic system which is activated by palatable food cues. Homeostatic appetite control is part of a psychobiological system that evolved to maintain a sufficient supply of nutrients for growth and maintenance. It includes both excitatory and inhibitory signals that influence appetite and food intake via tonic and episodic control mechanisms (Hopkins et al., 2017). In contrast, hedonic hunger is based on the availability and palatability of foods in the environment (Blundell & Finlayson, 2004). Food intake is dependent on the interaction between homeostatic appetite control and hedonic pleasure which involve higher order processes including learning, memory, planning and prediction. These processes generate the conscious experience of the sensory properties of food but also the hedonic pleasure that is acquired from the food (Kringelbach, 2004).

In the current obesogenic environment, there is a constant presence of palatable food cues, and this may chronically activate the hedonic appetite system. This further highlights that dietary restraint does not necessarily create counter-regulatory

responses solely induced by an energy deficit and exercising restraint may be necessary to prevent weight gain and to promote weight maintenance (Schaumberg et al., 2016; Yeomans et al., 2004). Thus, as restraint theory suggests that restricting food intake below homeostatic needs produces deprivation (Herman & Mack, 1975), restricting palatable food intake could induce “perceived deprivation” even when an individual is in energy balance or in a positive energy balance (Lowe & Levine, 2005). The authors argue that motivation to eat more than needed is as powerful as the motivation to eat when deprived.

Evolutionary accounts suggest that it is adaptive to consume food in the absence of physiological hunger because it can prevent the onset of physiological hunger and increase storage of body fat which protects the body from periods of food scarcity (Peters et al., 2002; Stubbs et al., 2012). In resource limiting environments, over the long-term, hedonic needs safeguard the individual from unexpected changes in the environmental supply of energy nutrients. This concept indicates that food consumption can be driven by homeostatic needs (energy deprivation) or hedonic needs (presence of food, specifically palatable foods). The implication is that the rising rates of obesity are a consequence of passive overconsumption (Blundell & Gillett, 2001). However, eating for pleasure of other affective functions could also lead to active overconsumption. Thus, Lowe et al. (2009) developed the power of food scale (PFS), a psychometric questionnaire which aimed to measure hedonic hunger. The PFS was designed to measure individual differences in appetite-related motivations, thoughts, and feelings in environments where palatable foods are available.

If the PFS measures a predisposition for overconsumption, a significant relationship between PFS and BMI would be expected. However, a clinical study found no statistically significant relationship between PFS and BMI (Cappelleri et al., 2009), and a meta-analysis of the PFS found no significant association between PFS and BMI (Dakin et al., 2023a). In contrast, Vainik et al. (2015) did find a significant correlation between PFS and BMI, and suggest this association may be explained by an empirical 2-factor solution of the PFS. The items that relate to uncontrolled eating have a positive association with BMI, however, the items that relate to food liking either have no association or a negative association with BMI. Potentially, the lack of

associated between PFS and BMI could be because studies assess total PFS responses, meaning they do not split PFS items into its 2-factor solution.

2.3.8. Intuitive Eating (Intuitive Eating Scale)

Another EBT to be discussed that has been developed from psychological theory is intuitive eating. From a psychological perspective, the study of eating behaviour has focused on disordered rather than healthful types of eating. However, there is also a need to identify and study healthful eating behaviours that help to maintain psychological health (Seligman & Csikszentmihalyi, 2000). One example of a healthful EBT could be restrained eating. However, as previously described, there is mixed evidence about whether high levels of restrained eating lead to positive or negative health outcomes. Restraint theory argues that counter-regulatory responses can lead to disinhibited and binge eating behaviour (Herman & Mack, 1975; Herman & Polivy, 1980), whilst other researchers suggest that demonstrating flexible control when eating is associated with weight loss and weight loss maintenance (McGuire et al., 1999; Phelan et al., 2009). Furthermore, healthful eating has been discussed within other disciplines, but is often considered regarding guidelines for intakes of specific foods (Ogden, 2011). As a result, there is less known about healthful eating behaviours (Tylka, 2006).

Healthful eating is important to study because it is more than just the absence of disordered eating behaviours; internal cues can be used to determine behaviour. For example, healthful eating involves using physiological hunger and satiety cues as a guide to determine when, what and how much to eat (Tribole & Resch, 1995). Consequently, healthful eating is likely to be negatively correlated with an absence of eating disorder symptoms, but it not only defined by this absence. This led Tylka (2006) to develop a questionnaire to assess the psychometric properties of potential variables that can serve to protect against developing disordered eating behaviours. The focus of this questionnaire was to describe and measure intuitive eating which is defined as “eating based on physiological hunger and satiety cues, rather than external and emotional cues” (Tribole & Resch, 1995). Intuitive eating can be viewed as a healthful eating behaviour because it is strongly related to understanding and responding to internal physiological needs of hunger and satiety in addition to a lack

of concern with food. In the literature, three central dimensions of intuitive eating have been identified and in the initial intuitive eating questionnaire (IES), these three dimensions were found and given the following labels: unconditional permission to eat, eating for physical rather than emotional reasons, and reliance on hunger and satiety cues.

Research has found support for the construct validity of the original IES, including finding negative correlations with total intuitive eating scores and disordered eating symptoms (Shouse & Nilsson, 2011) and BMI (Augustus-Horvath & Tylka, 2011). However, the original scale omits one of the components of intuitive eating proposed by Tribole and Resch (2012) which involves honouring health or engaging in gentle nutrition (choosing foods that promote health, energy, stamina, and body performance). If intuitive eating is a healthful eating behaviour, this construct should also reflect this dimension of choosing nutritious foods that help the body to perform well. Therefore, the Body-Food Choice Congruence subscale (B-FCC) was added to the intuitive eating scale-2 (Tylka & Kroon Van Diest, 2013). The new IES-2 was found to improve upon the original IES, and the B-FCC was found to be a distinct factor within the IES-2. More specifically, B-FCC was significantly correlated with specific body related measures and all psychological well-being measures. Additionally, B-FCC predicted unique variance in self-esteem, positive affect, negative affect, and life satisfaction above and beyond the variance accounted for by eating disorder symptomology. Lastly, the addition of B-FCC supports the interpretation that components of intuitive eating are associated with listening to and appreciating the body.

Furthermore, another similar model to intuitive eating is the Satter Eating Competence Model (eSatter) (Satter, 2007). Although this model examines a wider spectrum of eating attitudes and behaviours than the intuitive eating scale, eSatter highlights the importance of intuitive eating. According to the model, a key construct of a competent eater is “having internal regulation skills that allow intuitively consuming enough food to give energy and stamina and to support stable body weight.” The author also proposes similarly to Tylka (2006) that it is important to study this healthful eating behaviour because it will not only allow for more targeting

interventions, but also help individuals to trust their own capabilities to learn and develop a more adaptive eating style.

2.3.9. Dual Process Model (Social Behaviour Theory)

Separate from psychological explanations of eating behaviour, Strack and Deutsch (2004) argue that social behaviour is controlled by two systems: reflective and impulsive processes, that can operate in accord or compete with each other. While not specifically aimed to explain eating behaviour, this theory can be applied to eating. For example, Finlayson et al. (2007) discussed the implications of a dual-process perspective of food reward for weight gain and obesity. Behaviour in the reflective system occurs as a consequence of a decision process, requiring high cognitive capacity to enable cognitive reasoning. Distraction and low levels of arousal will interfere with the reflective system. In contrast, the impulsive system operates through spreading activation, it is fast, inflexible and needs no attentional resources. Consequently, processes in the reflective system are interrupted more easily than processes in the impulsive system.

Both systems lead to the activation of behavioural schemata, but differ in how they activate a behavioural schema (Strack & Deutsch, 2004). Conflicts can arise if the activated behaviour schemata are incompatible. These conflicts can be shown in eating behaviour. For example, the sight of food, which acts as a food cue, could activate the impulsive system which may trigger a desire to eat, or habitual food intake (Schüz et al., 2015). Indeed, it is well documented that eating behaviour is often driven by the response to food-related cues (Lowe & Butryn, 2007). However, the reflective system could also be activated and depending on the individual's current state (e.g. level of hunger/satiety, motivation to eat) (Cheon et al., 2019) or traits (e.g. whether the individual engages in restrained eating or mindful eating) (Polivy et al., 2020; Warren et al., 2017), or motivations, the individual may consciously decide they do not want to engage in an eating episode.

This means that the impulsive system is in conflict with the reflective system about whether or not to engage in eating. According to the dual-process model, the resolution of the conflict between the reflective and impulsive system depends on the

strength of the activation for each schema. For example, if the food is not well liked by the individual, then the eating schema may not be highly activated as the individual may not strongly desire the food. Whereas, if an individual is distracted or under high levels of stress, they may not have the cognitive capacity available to highly activate the reflective system and therefore the impulsive system may prevail.

Strack and Deutsch (2004) also note that while both systems contribute to behaviour, the impulsive system will assume primary control if operating conditions for the reflective system (e.g., cognitive capacity) are not fulfilled. This means that eating behaviour is less likely to be determined by conscious control over food intake (assessing the valence and probability of future consequences of the behaviour) and more likely to be determined by immediate associations which result in hedonic qualities. Indeed, stress has been shown to interfere with cognition (Tomiyama, 2019) and lead to unhealthy eating (Raio et al., 2013). Some individuals who report high levels of perceived stress, show increased disinhibition, binge eating, and emotional eating compared to individuals reporting lower levels of perceived stress (Diggins et al., 2015; Groesz et al., 2012). These eating behaviours could reflect the impulsive system taking control, as disinhibition, binge eating and emotional eating focus on central features of the impulsive system, including emotions, desires and habits controlling eating behaviour.

The connection between stress and overeating may not always be automatic, as some people may use food as a deliberate coping strategy for stress. Researchers do not often ask participants directly about their use of health behaviours e.g. eating as coping responses to stress (Park & Iacocca, 2014). However, a poll of 1420 American adults asked participants how they cope with stress, and among the top answers was eating (American Psychological Association, 2012). Additionally, a focus group of low socioeconomic status (SES) individuals found that some individuals reported eating a poor diet in response to stress to “self-medicate” (Kaplan et al., 2013). This indicates that in some cases, eating in response to stress is deliberate, meaning stress may influence both the reflective and impulsive system. However, in this focus group, participants also reported that their willpower to resist health behaviours (e.g. eating) was reduced after a long and stressful day. These findings indicate that whilst eating in response to stress can be a deliberate coping

strategy, it is also an automatic response, especially when control/reflective resources are low (e.g. after a long and stressful day). Indeed, although many individuals report eating more than usual to cope with stress (Kaplan et al., 2013), research also demonstrates that many individuals are not aware of their motives for engaging in health behaviours (e.g. eating) (Sheeran et al., 2013). DeSteno et al. (2013) suggest that eating serves an "implicit" emotion-regulation strategy which means that engaging in these behaviours does not meet the commonly used definition of coping as deliberate.

Overall, dual-process theory was developed to explain social behaviour, however, many researchers have applied this approach to eating behaviour e.g. (Kemps et al., 2020; Price et al., 2016) and it is now well recognised that motivations for eating are influenced by two major systems: conscious and automatic processes. Behavioural research supports the concept of a dual-process model of obesity e.g. (Rothman et al., 2009), which suggests that a discrepancy between conscious and automatic processes influences eating behaviour which can promote a positive energy balance and obesity (Cohen & Babey, 2012; Strack & Deutsch, 2004). The field of behaviour change also supports the concept of conscious and automatic components which influence weight management behaviours (Greaves et al., 2011). Furthermore, a previous analyses of EBTs on weight change during an 18-month weight maintenance trial found that increases in reactive eating (automatic processes) and decreases in reflective eating (conscious processes) were significantly and independently associated with concomitant weight change (Dakin et al., 2023b).

2.3.10. Other Psychological theories related to Dual-Process Theory

Since the development of the dual-process model of social behaviour, other theories of eating behaviour have emerged that attempt to explain motivations for eating, overeating and obesity as well as other types of eating patterns e.g., restrained eating and binge eating. Similar to dual-process models, many of these theories regard eating patterns as the results of two competing processes. For example, in the dual pathway model of overeating, restrained eating and negative affect compete to control eating behaviour and are thought to lead to bulimia nervosa (Ouwens et al., 2009). The restrained eating pathway can be seen as a form of reflective process

as it is a conscious attempt to restrict eating which involves deliberate cognitive reasoning. Conversely the second pathway, negative affect, suggests that people with bulimia use purging and bingeing as a means of regulating negative emotions and mood states. Individuals who eat in response to distress, could be confusing emotional distress with hunger, due to a lack of interoceptive awareness, or they may learn to use eating as a means of comforting negative affect (Van Strien et al., 2005). The measures used to assess negative affect include emotional eating and drive for thinness which involve more implicit and automatic processes and therefore, can be seen as similar to the impulsive system.

Another later developed model of eating behaviour, the goal conflict model (Stroebe et al., 2013) suggests that the difficulties restrained eaters have in regulating their food intake occurs due to a conflict between two incompatible goals: eating enjoyment and weight control. This model proposes that individuals fail to regulate their intake due to palatable food cues in food-rich environments that strongly prime the goal of eating enjoyment. Subtle reminders of food/eating trigger processes that activate this goal outside of conscious awareness and leads to inhibition of the weight control goal, which hinders healthy eating (Kavanagh et al., 2005). On the other hand, extended priming of the weight control goal, which is defined as reaching and maintaining a target weight, leads to inhibition of the eating enjoyment goal through deliberate eating behaviour such as dieting.

In the current food-rich environment, it is likely that the eating enjoyment goal is more strongly activated than the weight control goal because individuals are constantly surrounded by palatable food cues. This can often lead to dieting failure and weight relapse in many people engaged in restrained eating for the purposes of weight management. Evidently there are similarities between the goal conflict model and dual-process models. Both theories suggest that resources are important, proposing that individuals are successful in maintaining behaviour if they have enough psychological and physical resources. Dual-process models hypothesise that behaviour can be controlled by the resource intensive reflective system or the automated impulsive system. Resources can act as moderators and determine which system generates a response (Kwasnicka et al., 2016). Studies have found that when control resources were available to regulate the goal of dieting/weight

control, explicit measures, such as the participant's intention to diet, predicted consumption. However, when resources were low, implicit measures, related to the hedonic relevance of food stimuli, were better predictors of consumption (Friese et al., 2008). However, and importantly, many of the empirical findings related to dual-process models were tested and published before the replication crisis (Bem, 2011). Recently, Sommet et al. (2023) conducted a meta-study of 159 studies from influential psychology journals and found that the median power to detect interactions of a typical sample size was 0.18. Given that typical psychological studies including those described above have only 0.18 power to detect these findings, caution must be taken when interpreting results and when considering the validity of dual-process findings.

2.3.11. Behavioural Susceptibility theory

Behavioural Susceptibility theory (BST) combines evidence from both genetics and the role of the environment to better understand how appetite influences obesity onset (Llewellyn & Wardle, 2015). This section describes the theory as it relates to the psychology of eating behaviour. BST argues that individual differences in appetite play a causal role in the development of obesity, suggesting that those who inherit lower sensitivity to satiety, or an increased avid appetite are more likely to overeat in response to the food environment. The theory suggests that the two important components of appetite are responsiveness to hunger cues and responsiveness to satiety cues. Those who are more responsive to food cues are more likely to eat in response to an attractive food opportunity. Additionally, those who have weaker internal satiety cues or are less aware of their satiety cues are more likely to eat for longer. Where this theory differs from previous similar theories, e.g. externality theory, is the suggestion that genetic risk operates through these two appetitive traits, and this results in differences in susceptibility to the food environment. This means that obesity genes determine each person's responsiveness to food opportunities and as opportunities to eat increase, genes for high food responsiveness and low satiety responsiveness are expressed in terms of behaviour. Consequently, BST aims to provide a better explanation for how genetics, the environment, and appetite interact.

The two new constructs that BST presents are food responsiveness (FR) and satiety responsiveness (SR), which can be measured using the adult eating behaviour questionnaire (AEBQ) (Hunot et al., 2016) or the child eating behaviour questionnaire (CEBQ) (Wardle et al., 2001). In support of BST, research has found that increased FR and decreased SR are EBTs that are associated with greater adiposity (Carnell & Wardle, 2008). Additionally, both EBTs are linearly associated with weight across the entire weight spectrum (from underweight to obesity in children) (Croker et al., 2011). Furthermore, a longitudinal study has found that these EBTs are more likely to drive early weight gain, than early weight gain driving changes in EBTs (van Jaarsveld et al., 2011). Overall, BST has built on previous theories that indicate the important role of appetite in obesity, which addresses how appetite, genetics and the environment all play a causal role in obesity development.

2.3.12. Frameworks that attempt to integrate constructs from psychological theory

The suggestion that many EBTs share similar underlying constructs has been previously proposed by Vainik et al. (2015). It is logical to recognise that if EBTs share underlying constructs, then there should be an overarching theoretical basis to which those underlying constructs may belong. The authors suggest that EBTs could be capturing the same underlying construct, termed “uncontrolled eating,” which is defined as increased appetite and decreased self-control. EBTs could also be measuring different levels of severity along this construct. Vainik et al. (2015) adapted a continuum model of uncontrolled eating proposed by Davis (2013) to incorporate 5 EBTs into this model. They suggest that EBTs including eating impulsivity, hedonic hunger, emotional eating, disinhibition, and binge eating measure different ranges of values along a broader continuum of uncontrolled eating.

The continuum begins at ‘homeostatic eating,’ where an individual’s energy intake matches their energy expenditure. Early stages of uncontrolled eating are triggered by responsiveness to food cues in the environment, resulting in ‘passive overeating.’ This reflects a limited capacity to recognise a positive energy balance and therefore an individual exerts little control over their eating behaviour (Prentice & Jebb, 2003).

More severe stages of uncontrolled eating then follow, which includes emotional eating, disinhibition, and binge eating. Using bifactor analysis and item response theory (IRT), the authors found evidence to support the uncontrolled eating model, which suggested that these 5 EBTs capture the same common construct of uncontrolled eating and each focus on different severities of uncontrolled eating. This novel research provides a starting point to develop a comprehensive perspective of the current literature, which suggests that EBTs are measuring different degrees of lack of control of eating. However, only women were included in the analysis and a limited number of EBTs were assessed which were all constructs that measure moderate to severe stages along the continuum of uncontrolled eating.

Racine et al. (2019) examined a wider variety of non-homeostatic EBTs which included 7 questionnaires (Dutch Eating Behaviour Questionnaire, Eating Pathology Symptoms Inventory, Loss of Control over Eating Scale, Binge Eating Scale, Eating Disorder Examination Questionnaire, Power of Food Scale, and Yale Food Addiction Scale). Their results supported a 7-factor solution, suggesting that the items in these questionnaires measure 7 multiple but related constructs termed: emotional eating, external eating, loss of control over eating, overeating, distress over nonhomeostatic eating, hedonic hunger, and food addiction. These results are in contrast to Vainik et al. (2015) and suggest that the questionnaires used are measuring multiple constructs. Although, there is still a lack of research that examines the less severe end of the uncontrolled eating spectrum or “homeostatic eating” constructs. Consequently, there is a need to examine whether a framework can be developed that explains more varied EBTs including EBTs from both ends of the homeostatic eating/uncontrolled eating spectrum.

Furthermore, components of behaviour change models and cognitive theories can also be used to explain how individual differences in eating behaviours can help or hinder weight management attempts. These models also draw similarities with dual-process models. For example, models that explain behaviour change during weight management attempts also include reflective and impulsive components that work against each other to influence changes in eating behaviour which influence weight change (Dunton et al., 2021; Greaves et al., 2011; Kwasnicka et al., 2016). Greaves et al. (2011) suggest that longer-term weight management involves tension between

existing habits (EBTs) and incompatibility of new weight management behaviours. Research has found that engaging in reflective and impulsive processes can influence weight management success. Engaging in and or developing reflective processes such as self-regulation, motivation and managing external influences (Dombrowski et al., 2014; Teixeira et al., 2005; Varkevisser et al., 2019) has been found to aid weight management. In contrast, reactive/automatic processes (e.g. emotions and desires) are the result of associative learning and physiological resistance to weight loss which can undermine longer-term weight loss (Berthoud, 2011; Blundell & Finlayson, 2004).

Additionally, the COM-B model (Michie et al., 2011) was developed as a framework of behaviour change which involves three essential conditions: capability, opportunity, and motivation. The framework took a systematic approach to behaviour change, with the aim of capturing a range of mechanisms including the internal and external environment. Capability is defined as an individual's physical and psychological capacity to undertake a specific activity. Motivation is defined as any brain process that directs behaviour. This involves goals, conscious decision-making, habitual processes, and emotional responding. Lastly, opportunity is defined as any factor outside of an individual that makes a behaviour possible. The COM-B model differentiates between reflective processes and automatic processes, which further highlights similarities with dual-process models. The COM-B model also draws concepts from dual-process models (Strack & Deutsch, 2004) to help with linking interventions to different components in the behaviour system. For example, the COM-B model also suggests that automatic processing is central to the behaviour system and should be considered equally to reflective, systematic, and cognitive processes when designing interventions. Overall, COM-B makes reference to many EBTs, and dimensions covered by psychological theory and attempts to integrate them to predict behaviour. However, the COM-B model refers to behaviour change not EBTs specifically. Therefore, it would be useful to develop a comprehensive framework of EBTs to understand the dimensions/constructs that underlie eating behaviours.

2.3.13. Limitations of current EBTs and frameworks

The current state of EBTs presents some notable critiques. Firstly, there is an abundance of EBTs, which has led to a lack of clarity in understanding the underlying mechanisms of an EBT. The extensive developing of new traits has resulted in a convoluted array of measures without meaningful organisation. This means there is a lack of understanding about which overarching domains of eating behaviour, EBTs specifically belong to or are measuring. Many EBT constructs appear to be closely related or redundant, which has also led to conceptual overlap and confusion. This is defined as a jangle fallacy (constructs are captured with different questionnaires, that measure the same underlying mechanism) (Kelley, 1927).

Secondly, there are some scales which could be causing a jingle fallacy (an incorrect assumption that tests assess the same construct, because they share similar names) (Thorndike, 1904). For example, there are more than four different scales used to measure restrained eating, e.g. the EDE-Q, restraint scale, TFEQ, and DEBQ (Fairburn & Beglin, 1994; Herman & Mack, 1975; Stunkard & Messick, 1985; Van Strien et al., 1986). Each of the above scales are proposed to measure restraint; however, they may or may not be measuring the same construct. The restraint scale, appears to measure excessive overconsumption, weight fluctuations and cognitions and emotions in addition to dietary restraint (Polivy et al., 2023). In contrast the TFEQ primarily identifies successful dieters who can restrict their intake. This has also been found in the personality literature, where although some evidence suggests overlap between individual traits and values, the current body of evidence indicates that personality traits and values are distinct constructs (Higgs & Lichtenstein, 2010). Furthermore, different scales used to measure restrained eating have found conflicting evidence about the role of restraint in overeating and obesity. This indicates how vital it is for researchers to understand what exactly the scale they are using measures because important outcomes of a study could be influenced by the EBT measure used. Similarities between constructs not only adds to the complexity of the field but also limits the ability to distinguish unique contributions from each EBT.

A comprehensive framework that encompasses the various dimensions of eating behaviour and provides a coherent structure for the research and application of EBT measurements is currently lacking. To advance the field and provide more meaningful insights into eating behaviour, researchers should aim to consolidate the description of EBTs, identify meaningful relationships among EBTs, align EBT measures with existing theory and work towards developing a comprehensive framework that identifies the underlying common constructs that are shared between overlapping EBT measurements.

2.3.14. Summary of psychological and cognitive theories

In this section key psychological and cognitive theories, EBTs developed from theory, and attempts to develop frameworks that integrate constructs were reviewed. Their contribution to understanding eating behaviour and obesity were evaluated (see Table 2.1 for a summary). Overall, many of these theories draw on concepts from dual-process models which suggest that eating behaviour is driven by two competing processes. Additionally, many theories draw on approach vs avoidance-based traits, internal vs external traits and implicit vs explicit traits. The main dimensions that seem to be captured by many theories and often EBT questionnaires seem to focus on one or two of the two competing processes that drive eating behaviour. One process involves cognitive, reflective, and decision-making abilities, which includes sensitivity and responsivity to internal cues. The other process involves automatic and reactive, behaviour, which includes reacting to external cues in the environment, eating for pleasure/comfort and overeating to reduce negative or enhance positive emotions.

Although these theories and models are presented as separate, it is important to highlight that there is considerable overlap between each theory/model and many of the newer theories incorporate concepts from earlier theories to develop a more comprehensive understanding of eating behaviour. Consequently, it is suggested that there is an overarching theoretical umbrella to explain human eating behaviour, whereby many EBTs fall under two competing processes. The next section now reviews physiology and neurobiology with the aim of integrating these psychological

and psychometric aspects of behaviour into the current understanding of the biology of eating behaviour.

Table 2.1. This table summarises the key theoretical constructs and where relevant psychological theories that can be applied to eating behaviour, including the central elements, main constructs, and mechanisms of action of each theory

Eating Behaviour Theory	**Central elements to theory**	**Main constructs**	**Mechanism of action**
Psychosomatic Theory	Overeating is viewed as a learned behaviour which is used as a means of reducing anxiety	Emotional Eating	Emotions become drive states, meaning that eating is associated with a reduction of emotions such as anxiety.
Externality Theory	Eating behaviour of individuals with obesity is defined by increased responsiveness to external cues and decreased responsiveness to internal/physiological signals	Internal Control External Control	Responsivity to environmental prompts and cues
Set-point Theory	Individuals with obesity are in a state of chronic energy deficit because they are attempting to keep their body weight below their biological set-point	None	Hypothalamic centres defend different baselines in different individuals

Restrained Eating	Individuals with high levels of restraint behave externally once restraint has been discarded and this triggers further eating. The degree of deprivation (restraint) predicts eating behaviour	Dietary Restraint	Conflict between cognitive control and 'homeostatic' signalling
Hedonic Hunger	Hedonic hunger is the desire to consume palatable foods in the absence of physiological hunger	Hedonic Hunger	Hedonic hunger acts to increase storage of body fat which protects the body from food scarcity
Dual Process Theory	Social behaviour is controlled by reflective (cognitive reasoning) and impulsive systems (fast, inflexible, no attentional resources required)	Impulsive Reflective	Reflective behaviour is the consequence of a decision process. Impulsive behaviour is elicited through spreading activation.
Dual Pathway Model	Restrained eating and negative affect compete to control eating behaviour	Restraint Negative affect	A lack of interoceptive awareness means individuals confuse distress with hunger
Goal Conflict Model	Restrained eaters cannot regulate their food intake due to conflict between two incompatible goals	Eating enjoyment goal Weight control goal	Palatable food cues prime the eating enjoyment goal. Deliberate eating behaviour e.g., dieting primes the weight control goal
Behavioural Susceptibility Theory	Increased food responsiveness and decreased satiety	Food Responsiveness	Genetic risk operates through these FR and SR and this results in

	responsiveness leads to overeating	Satiety Responsiveness	differences in susceptibility to the food environment
COM-B Model	A framework of behaviour change that involves three essential conditions	Capability Opportunity Motivation	A range of mechanisms are involved in this framework including the internal and external environment

2.4. Physiology of eating behaviour

Human appetite or motivation to eat is an example of a phenomenon that is biopsychological in nature. As discussed above, there are psychological and cognitive influences on eating behaviour and many models describe elements of dual-process theory, whereby decisions are motivated by separate cognitive (slow and reflective) and affective (fast and reactive) networks. However, many of these theories and models oversimplify the interplay between cognitive, affective, and metabolic systems as well as the neuroanatomical circuitry that links these systems into patterns of goal-oriented motivated behaviours (Keren & Schul, 2009). Consequently, integrating knowledge of physiology and neurobiological processes that signal motivation to eat (food seeking), appetite behaviours (consummation) and satiation/satiety (cessation of eating) within the current energy balance framework could offer a more detailed and comprehensive understanding of the role of EBTs in appetite research.

There are short (episodic) and long-term (tonic) physiological circuits influencing of motivation to eat and eating behaviour (Blundell et al., 2001). Episodic processes occur periodically, are organised around bouts of eating (e.g., meals or snacks) and typically refer to internal signals from the gut, mediating satiation, and satiety. These signals act via the gut-brain axis and feed into the same hypothalamic circuity as tonic signals. In contrast, tonic processes change over longer-periods of time more relevant to energy balance regulation and signal changes in body size and composition. This long-term appetite control involves the secretion of insulin by the pancreas and the secretion of leptin by adipocytes in proportion to fat mass (Wynne et al., 2005). Both insulin and leptin act on receptors in the hypothalamus which excite inhibitory neurons that decrease food intake and inhibit excitatory neuros that increase food intake. The overall net balance of these signals is believed to control the drive to eat.

Traditional models of homeostatic appetite control argue that adipose tissue is central to appetite control and is the main driver of food intake, with leptin also playing a key role in appetite control (Woods & Ramsay, 2011). However, Blundell et

al. (2015) demonstrated that fat mass is weakly and negatively associated with food intake in people with obesity, whereas, levels of fat free mass (FFM) and resting metabolic rate (RMR) are positively correlated with hunger and energy intake, and could be tonic signals of energy need and motivation to eat. Indeed, Blundell et al. (2020) consider FFM and RMR to be major determinants of energy intake in humans, and since 2011, the relationship between FFM, RMR and EI has been substantially replicated in more than 12 studies from more than 7 different countries. Integrated signals from changes in body size and composition influence the leptin/insulin and related neuropeptide axes to defend against significant weight loss. However, in most individuals, these signals are less responsive to weight gain under modern environmental circumstances.

One of the key theories in the field of human appetite, suggests that energy balance is biologically regulated. The concept of energy balance regulation is referred to as homeostasis. Cannon (1929) first defined homeostasis in the context of appetite and suggested that under normal circumstances, variations from the mean of a regulated variables would impair the functions of a cell or organism and therefore, before any extremes are reached, responses are automatically produced which act to bring the disturbed state back to the mean. This definition implies that the body's internal environment is maintained within a controlled homeostatic range. Since the late 20th century, numerous theories have been developed which propose that almost all components of the energy balance system can function as a negative feedback homeostasis signal to influence energy intake. These theories include the previously mentioned models that propose that appetite is regulated by adipose tissue (Woods & Ramsay, 2011) or FFM and RMR (Blundell et al., 2015). In addition, temperature regulation (Brobeck, 1946), fat (Kennedy, 1953), carbohydrates (Mayer, 1953), amino acids (Mellinkoff et al., 1956), protein leverage (Raubenheimer & Simpson, 2019) and body weight (Hervey, 1969) have all been suggested as homeostatic signals that influence energy intake.

Observational evidence for homeostatic regulation of body weight comes from the finding that a large proportion of the population is able to keep a stable body weight over time, which implies the existence of some degree of active regulation or defence (Shin et al., 2009). It has also been argued that such apparent stability could

simply be a function of the complexity of multiple redundant systems. This is evidenced in human and rodent under and over feeding studies where weight loss or gain is rapidly corrected by compensatory increases and decreases or calorie intake (Hall, 2006; Keesey & Corbett, 1990; Siervo et al., 2008). However, many of these studies test participants over durations of less than a few weeks and involve extreme overfeeding regimes. Furthermore, the prevalence of obesity, has called into question whether body weight is regulated, because 'if body fatness is under physiological control, then how come we have an obesity epidemic?' (Speakman, 2014). Additionally, Watts et al. (2022) argue that the concept of set-point for body weight is not consistent with the majority of experimental findings. To account for this, Wirtshafter and Davis (1977) argue for a simple feed-back control model that does not include a set-point but can account for evidence that does support body weight-set point existence. They coined the term 'settling point' which suggests that body weight is regulated but this regulation can vary considerably. An alternative proposal is that regulation of body weight is asymmetric (Blundell & Hill, 1985). The balance of evidence suggests that energy balance is regulated, but the regulation is not precise or symmetric. Whilst there are physiological changes that oppose weight gain or loss, excess energy intake is tolerated (weak episodic inhibitory control and no tonic negative feedback for weight gain), whereas energy deficit is strongly opposed (strong periodic and tonic negative feedback signals for energy need) (Stubbs & Tolkamp, 2006; Stubbs & Turicchi, 2021). This means that satiety can easily be overridden which allows for positive energy balance (Blundell & MacDiarmid, 1997).

In the energy balance model, overconsumption is viewed as being predominantly influenced by highly palatable, energy dense foods (Hall et al., 2022). The asymmetry of body weight regulation is likely to have evolved because a positive energy balance is much more adaptive for survival then a negative energy balance (Speakman, 2018). Additionally, evidence suggests that the level of feedback is not linear. This means that small deviations from current body weight will induce virtually no or weak compensatory responses. As deviations increase, there will be far greater compensatory responses. In particular, compensatory responses are more severe in response to large negative rather than positive energy balances over time (Stubbs et al., 2004). Taken together, energy balance is regulated but this regulation

is asymmetric and imprecise. Furthermore, physiological and psychological responses to weight loss occur on a continuum and this is influenced by the amount of energy deficit, the duration of energy deficit, body composition at the beginning of the deficit and the psychosocial environment of which the energy deficit occurs (Stubbs & Turicchi, 2021).

Taking a physiological perspective of appetite control and energy balance, it becomes apparent that there are homeostatic processes manifesting in physiological hunger that also mediate the strength and duration of satiety responses. These processes can help to better explain eating behaviour and therefore constitute traits that should be captured in a framework of eating behaviour. There already exists some EBTs that could be used to measure physiological hunger. For example, the hunger subscale of the Adult Eating Behaviour Questionnaire (AEBQ) (Hunot et al., 2016) is a measure of physical hunger which includes items such as “I often notice my stomach rumbling.” Evidence shows that physiological signals including levels of FFM and RMR are positively correlated with hunger and energy intake and act to motivate eating (Blundell et al., 2015). Therefore, it is important for EBTs to distinguish between physiological hunger and hedonic hunger as measured by the PFS. Indeed, another subscale in the AEBQ measures responsiveness to food which can be seen as a proxy of hedonic hunger, and although these scales were strongly related to each other, a confirmatory factor analysis revealed a better model fit when these scales were kept separated. Furthermore, there is good evidence in the literature that these scales measure distinct dimensions of eating (Schachter, 1968; Stunkard & Fox, 1971).

There are also EBTs that could be considered to measure homeostatic processes. For example, the satiety responsiveness scale from the AEBQ (Hunot et al., 2016) measures an individual’s sensitivity to their internal levels of satiety. The concept of satiety responsiveness is also represented in the internal hypothesis of externality theory (Schachter, 1967). The internality hypothesis suggests that eating behaviour is influenced by internal physiological cues that are associated with food deprivation. Taking an energy balance perspective, internality, or satiety responsiveness measures how well an individual responds to internal feedback so that energy intake and energy expenditure are in balance. Clearly, physiological hunger and

homeostatic processes are related; hunger is influenced by differences in interoceptive awareness, the strength of satiety signals and the drive of energy requirements. Consequently, it is important to develop a framework of EBTs that can measure both sides of the energy balance equation, as well as other psychological traits that influence eating behaviour. EBTs that are currently available are limited in that they measure more of certain types of eating behaviour and less of others. There are a limited number of EBTs that could be measuring physiological hunger or homeostatic eating. It would be valuable to develop new scales that cover gaps, e.g., more specific measures of physiological aspects of eating behaviour.

2.5. Neurobiology of eating behaviour

Whilst appetite is regulated by processes of hunger and satiety, there are many other drivers of eating including cognitive and psychological drivers (as previously described), and food reward (Berthoud & Morrison, 2008). Neurobiological studies have been able to give a more comprehensive account of how hedonic, reward-based pathways are more important to energy balance homeostasis than has been previously suggested. Eating is a highly salient source of reward and pleasure for animals and humans. Pleasure is a central cue that links food liking and wanting to learned consumption behaviour (Kringelbach et al., 2012). This suggests that human eating behaviour is not governed solely by homeostatic mechanisms and instead, pleasure and reward also play a central role in control or lack of control of energy intake (Kringelbach, 2004). Indeed, consuming palatable foods engages systems that are involved in reward processes including dopamine and opioid systems (Pfefferbaum et al., 1992). Neurobiological studies can also help to explain why eating behaviour so often leads to a positive energy balance in the current environment where several facilitatory cues and a lack of constraints are present concerning what, when and how much to eat.

It is possible that in resource-limiting environments from which we evolved, homeostatic and hedonic systems operated in a co-ordinated manner to facilitate overconsumption during periods of food abundance. Natural selection would favour these behaviours due to environmental uncertainty (Berridge & Kringelbach, 2008; Stubbs & Tolkamp, 2006; Stubbs & Turicchi, 2021). However, modern environments have changed very rapidly compared to the environment that influenced energy balance regulation. The rate of change of the modern food environment is far faster than the rate at which human evolution can adapt. This means that at times, motivation to eat and food reward behaviours conflict with other non-food related motivations and rewards, for example health and wellbeing. Consequently, understanding how both homeostatic and hedonic factors influence food intake in the current environment requires knowledge of physiology, psychological and neurobiology as well as an appreciation of the asymmetry of energy balance regulation.

Motivation and emotion both drive food intake which is supported by reward and hedonic processing (Kringelbach et al., 2012). Motivation to eat can be triggered by many factors including metabolic need and hedonic drives and there are numerous neuro circuits that underlie these processes. Physiological signals can modulate processing of cognitive and reward functions which can affect regulatory processes. On the other hand, cognition and emotions can also influence homeostatic regulation which can lead to energy imbalance. Appetite is therefore determined by the interaction between homeostatic and hedonic pathways (Berthoud, 2011). Using a neurobiological model of eating behaviour that operates in the context of asymmetric energy balance regulation, many factors can largely influence motivation to eat, eating behaviour and energy balance (Berridge & Kringelbach, 2008; Kringelbach et al., 2012; Stubbs & Tolkamp, 2006). These models can help to explain the development of obesity in the current food environment as well as the difficulties people face when trying to lose weight and maintain weight loss.

As neurobiology can give a more comprehensive understanding of eating behaviour, it is important that neurobiological mechanisms are captured by EBTs. Examples of neurobiological mechanisms that could be captured by EBTs include food reward, executive function, affect-related and stress-related eating. Historically, there has been a focus on how emotions can affect eating behaviour. Positive emotions (e.g., happiness) and negative emotions (e.g., stress and anxiety) can motivate eating through positive reinforcement and negative reinforcement of the emotional state (Skinner, 1963). This is also highlighted in psychosomatic theory, whereby, overeating is thought to be motivated by drive reduction. Eating is associated with a reduction of negative emotions and thus eating is rewarded (Kaplan & Kaplan, 1957). Emotional eating has been widely studied and many EBTs have been developed that measure some aspect of emotional eating e.g., emotional eating (DEBQ), positive emotional eating (PNEES), negative emotional eating (PNEES), emotional overeating (AEBQ), and emotional undereating (AEBQ).

Furthermore, as previously described, food reward plays an important role in eating behaviour, with some individuals displaying strong drives to eat in response to the natural reward of highly palatable foods (Beaver et al., 2006). This concept has been referred to as reward-based eating, and there is evidence for reward-based eating in

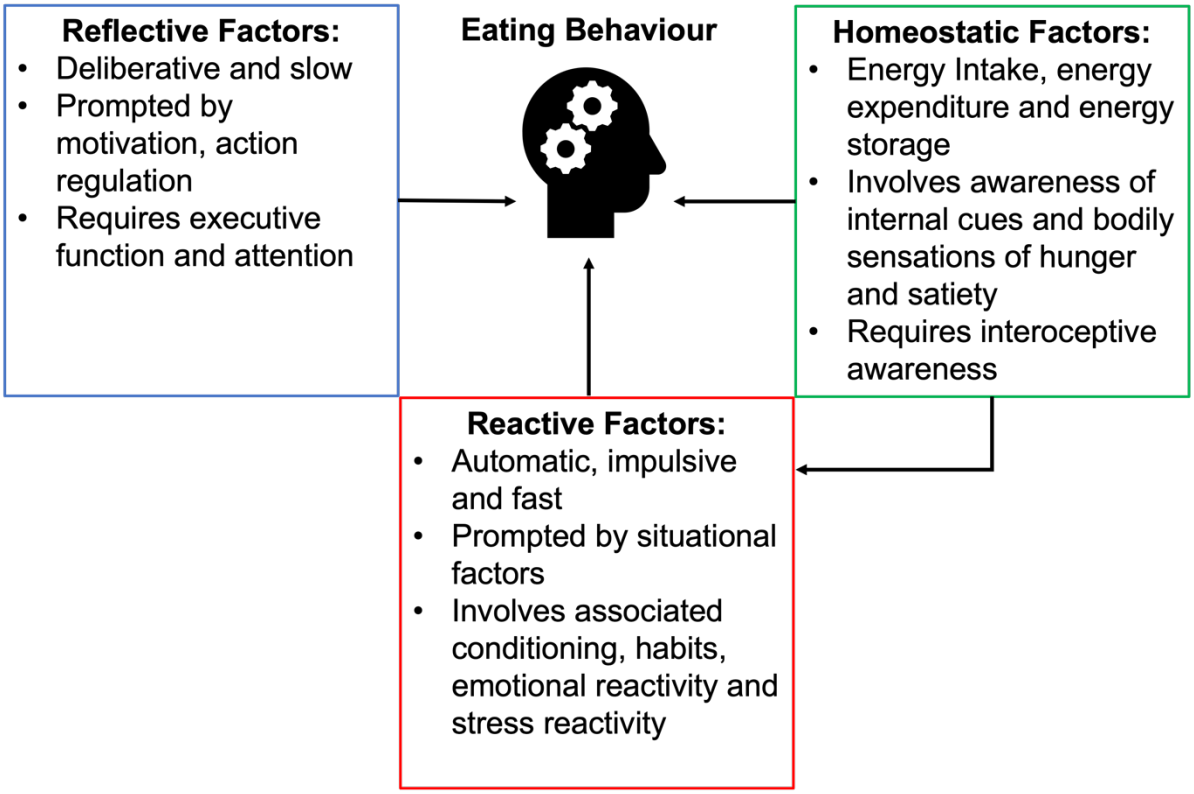
both behavioural and neurobiological studies (Volkow et al., 2012; Volkow et al., 2013). Researchers have proposed that reward-based eating can lead to overconsumption because it overrides satiety signals. The excess energy intake from overconsumption is reinforced overtime through changes in dopaminergic pathways that regulate the neuronal systems related to reward sensitivity (Volkow et al., 2011). Measuring reward-based eating using an EBT would be useful to further understand how food reward influences eating behaviour, because this pattern of eating could lead to obesity (Epel et al., 2014). The PFS does tap into aspects of reward-based eating but focuses more on the impact of the food environment. Consequently, the reward-based eating drive (RED) scale was developed as an EBT to measure reward-based eating (Epel et al., 2014). In support of reward-based eating, the RED scale was found to be positively corelated with BMI. Since its development, the RED scale has been broadened to include additional items to better assess the entire spectrum of reward-related eating. Most recently, the RED-13 has been developed which was also found to be positively correlated with BMI as well as type 2 diabetes and craving for sweet and savoury foods (Mason et al., 2017).

There is also evidence that higher level cognitive functions including learning and memory influence appetite control and that homeostatic signals also influence cognition (Higgs et al., 2017). Decisions around daily eating vary in terms of the cognitive effort that is required to complete them. In general, the automaticity of reward-driven decision-making encourages habits that lead to consumption of energy-dense, highly palatable foods (Iso-Ahola, 2017). Contrastingly, decisions that sustain healthy eating behaviours require more effortful and conscious control, which is termed ‘executive function’ (McGuire & Botvinick, 2010). Executive function is a limited-capacity resource, therefore, depending on the cognitive resources available and the current situation, cognitive or affective neural systems are activated differently to influence eating behaviour decisions. Additionally, research shows that a high-fat, high-sugar diet can damage the hippocampus and decrease learning and memory in rodents (Davidson & Stevenson, 2022). Furthermore, reduced executive function renders individuals more susceptible to reactive processes, which influences food reward-based decision-making and could lead to food seeking and consequently, obesity (Zhou et al., 2023).

It is therefore apparent that both cognition and executive function also influence eating behaviour. At present there are no EBTs that are designed to measure cognitive-related eating or executive function related to eating. There are several food-related executive functioning tasks. For example, a systematic review identified 66 different neurocognitive tasks of which 21 tasks measured executive function, in relation to obesity and eating behaviours (Vainik et al., 2013). The review found that tasks that were sensitive to executive function and food motivation provided the most robust and reliable associations with BMI, BMI change or eating behaviours. However, out of the 66 tasks reviewed, fewer than 11% showed consistent and reliable effects. Additionally, while these tasks are related to food and eating, e.g. the food stroop task, food delay discounting, and relative reinforcing value of food, they are less relevant to self-report measures of eating behaviour. As such, many of these tasks would not be considered EBTs, which are self-report measures of eating behaviour. This discussion highlights areas for methodological development, including the development of EBTs that specifically measure executive functioning related to eating behaviour.

Interestingly, within a neurocognitive model of appetite control, the processes of reflective and impulsive eating can be seen. Indeed, Herman and Polivy (2014) suggest that humans do not consciously consider food decisions all of the time and instead, most of the time, eating engages no mental effort and is ‘mindless.’ The automatic food decisions that are made, mirror the reactive processes within appetite control, which are suggested to be in control especially when cognitive resources are low. Zhou et al. (2023) also argue that motivation to eat and food reward systems are mainly subcortical and reactive, whilst decision-making involves conscious cortical processing and complex motivation. Thus, highlighting the two competing pathways: impulsive and reflective eating. See Figure 2.1 for a graphical representation of an integrated physiological, neurobiological, and psychological model of energy balance regulation.

Figure 2.1. A graphical representation of an integrated physiological, neurobiological, and psychological model of energy balance regulation



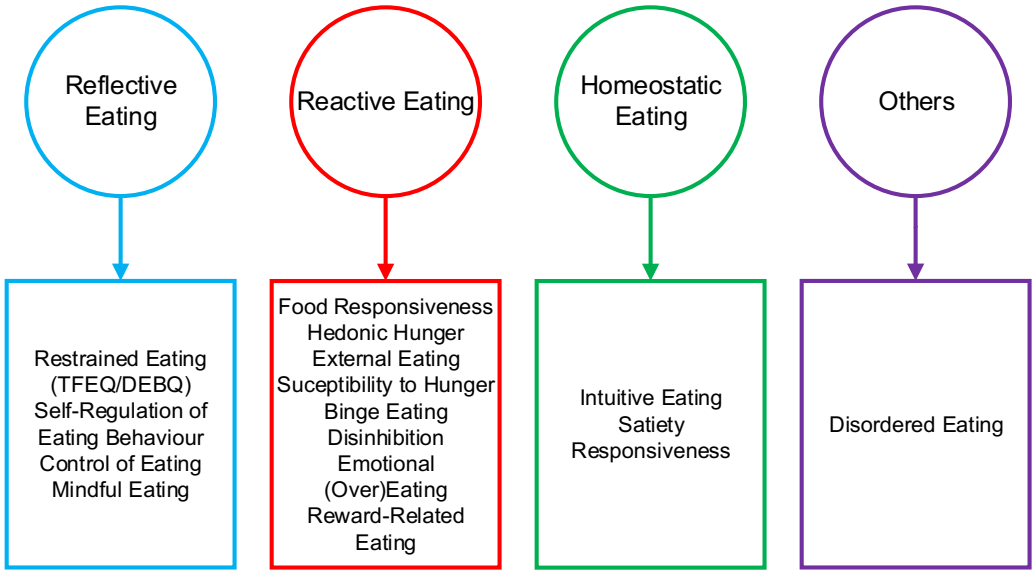
Note. This figure represents eating behaviour as central to energy balance. Directly related to eating behaviour are reactive processes of learned and habitual behaviours that respond to environmental and psychological cues such as food availability and stress respectively. Another domain represented in this diagram is reflective factors which involve deliberative and cognitive modification of attitudes, beliefs and intentions that aim to change eating behaviour. This domain often has less influence on energy balance than other domains and often these strategies conflict with reactive and/or homeostasis and hedonic factors which also renders them less effective. Lastly, the diagram also recognises that elements of the energy balance system influence both reflective eating behaviour and reactive components of eating behaviour which is represented in the homeostatic domain.

2.6. Eating Behaviour Traits Framework

The last aim of this review is to develop a framework of EBTs that explains observed behaviours in the context of key psychological theories, physiology, and neurobiology of appetite. As previously described, many approaches do not highlight the importance of both psychology and physiology of eating behaviour and therefore there is a need to move towards a more integrated approach. Current theories that explain EBTs appear to be made up of theories that can be considered as 'fragments' of dual-process models. Many of these theories regard eating behaviour as comprising of two processes involving automatic and deliberate behaviour. Physiological theories on the other hand, highlight the importance of biological signals influencing behaviour, but also fail to explain how other important non-physiological factors such as environmental cues and emotions drive eating behaviour. Furthermore, newer integrative frameworks such as uncontrolled eating are driven by data on eating behaviour. However, they are not anchored onto an a-priori theory of eating behaviour.

The two major systems that influence motivation to eat are conscious processes (reflective, cognitive factors) and automatic processes (reactive factors). These two domains also interact with physiological signalling which includes homeostatic feedback. Thus, EBTs can be grouped under three higher-order, conceptual domains: reflective, reactive, and homeostatic processes (see Figure 2.2). These domains are labelled. However, other synonymous labels could be used depending on the field of study. For example, cognitive psychologists may refer to reactive eating as automatic eating. Whilst it is acknowledged that different labels could be used for these factors, it would be useful for future research to take a consultative approach with experts to discuss and clarify these labels.

Figure 2.2. A conceptual framework of eating behaviour traits (EBT)



Note. A conceptual framework of eating behaviour traits (EBT) that suggests EBTs can be grouped under three higher-order, conceptual domains: reactive, reflective, and homeostatic eating. This framework also understands that the labels given to these domains can be swapped for many other labels depending on the context for which this framework is being used. Lastly, there are some EBT that this framework does not attempt to explain, and this is recognised in the figure.

Reflective processes involve deliberative cognitive reasoning with some degree of analytical awareness of eating behaviour. This process is governed by slow, logical, and sequential thinking and includes strategies of behaviour change. EBTs influenced by reflective processes refer to conscious control over food intake, involving thought, decisions, or control. See Figure 2.2 for measures that could be influenced by reflective processes. The reflective domain has less influence on energy balance than other domains due to a combination of environmental, physiological, and hedonic factors that work to encourage overconsumption and thus a positive energy balance.

Many weight management interventions use behaviour change techniques as a method of achieving weight loss. Several of these behaviour change techniques can be considered examples of reflective processes. For example, strategies to improve of develop self-efficacy, self-motivation, and self-monitoring involve developing competencies, strategies and skills aimed at resolving challenging situations, reducing emotional stress and cognitive control to exhibit specific behaviours (e.g.

self-weighing or restricting energy intake). Additionally, engaging in consistent exercise requires effort, motivation and determination, which are all examples of reflective processes (Stubbs et al., 2011). Unfortunately, evidence shows that maximum weight loss through behaviour change interventions is achieved at 6 months and after this point, body weight increases gradually to baseline levels (Garcia Ulen et al., 2008). Research also shows that other domains work to oppose the reflective domain comes from an understanding of the barriers to weight loss. Indeed, one study found that the most important perceived barriers to dieting for weight loss were food craving, stress and depression (Sharifi et al., 2013).

Reactive processes involve automatic eating behaviour, which is governed by external stimuli including emotions, impulses, habits, and desires. Evidence suggests that a large amount of human behaviour is reactive and learned and the environment can generate cues, prompts and stimuli that drive learned motivations to eat (Cohen & Babey, 2012). EBTs influenced by reactive processes refer to automatic influences over food intake, which indicate the effect of emotions, desires, prompts, cues, impulses, and habits (see Figure 2.2 for examples). A variety of cues in the environment for example the sight of food can become associated with eating which provokes learned cue-reactivity (Jansen et al., 2016). Studies have shown that food cue reactivity increases the risk or overeating, weight gain and relapse after weight loss (Jansen et al., 2011a; Jansen et al., 2011b).

Reactive processes also include eating behaviours that are driven by habits, which can explain why reactive eating behaviour is pursued even in the absence of a food cue. Habits are behaviours that are frequently repeated over time and are learned sequences that have been reinforced by rewarding experiences, which are largely outside of conscious awareness, because they have become subsumed to automaticity (Neal et al., 2006). van Koningsbruggen et al. (2013) found that tempting food cues increase effortful behaviour towards high-calorie food among unsuccessful, but not successful dieters. Whereas, tempting food cues increased efforts towards low-calorie food in successful dieters, but did not affect unsuccessful dieters.

Additionally, when an individual engages in a weight loss attempt, there are physiological responses which are more pronounced in response to negative rather than positive energy balances. Some of these responses involve active compensation (e.g. decreased TDEE including reductions in RMR and NEAT) and some involve physiological processes that influence behaviour (e.g. decreased physical activity and increased energy intake) (Stubbs & Turicchi, 2021). There are also reactive psychological and behavioural responses to weight loss, including increased motivation to eat and compensatory increases in energy intake, and compensatory decreases in energy expenditure. For example, Polidori et al. (2016), found that appetite increased by ~100kcal/day above baseline per kg of lost weight. This demonstrates how difficult it is to successfully maintain weight loss due to increases in reactive processes such as increased appetite and suppression of energy expenditure that exists partly because of living in an obesogenic environment.

Importantly, the current approach using self-report measures of eating behaviour is limited because reactive eating processes occur automatically. As such, the capability of individuals in being able to answer questions on their automatic eating behaviours can be questioned. For example, in the context of emotions and eating behaviour, Winkielman et al. (2005) argue that “for an emotion to be unconscious, people must not be able to report their emotional reaction at the moment it is caused.” This would suggest that self-report items are unable to access reactive, automatic eating behaviours. However, they also argue that there can be evidence of the emotional reaction in one’s behaviour, physiological responses, or the subjective impression after the event (Winkielman et al., 2005). Thus, items used to measure reactive EBTs measure thoughts, feelings, motivations, and behaviours which can be used to measure automatic EBTs. Though it must be acknowledged there are limitations to this approach, self-report measure of EBTs do allow us to access information about a person that is rich with their eating motivations and contains more information that other people may not be aware of (Paulhus & Vazire, 2007).

Homeostatic eating refers to the conscious awareness of internal cues and bodily sensations of hunger and satiety. It is defined as eating determined by energy deficits (Lowe & Butryn, 2007). This domain therefore reflects the physiological

theories of homeostasis which argue that eating behaviour is driven by physiological signals that respond to a negative energy balance (Watts et al., 2022). No EBT has been specifically designed to measure homeostatic eating, nor indeed types of motivation to eat in response to energy imbalances, but there are scales that measure conscious awareness of sensations that can be considered related to or proxy measures of homeostatic eating (see Figure 2.2). Items on the reliance on hunger and satiety subscales, for example, measure whether an individual can trust and rely on their internal hunger and satiety cues, which are central features of homeostasis.

Homeostatic eating is distinct from reflective eating because it measures the ability to listen and act on internal and biological hunger and satiety signals which is not the same as cognitive control of eating. This may require an individual to have a level of interoceptive awareness to assess attention to internal signals. However, homeostatic eating may also require no interoceptive awareness if an individual is unconsciously compensating for their energy intake over a period of weeks and months (Polidori et al., 2016). An important theoretical consideration of the homeostatic eating domain is that overeating, and obesity can be explained by the asymmetry of body weight regulation.

Overall, there is little evidence that weight gain produces active compensation of behaviour to reduce energy intake. However, there is evidence that weight loss produces physiological and behavioural responses that unconsciously increases energy intake that escalates as weight loss escalates (Stubbs & Turicchi, 2021). The mechanisms involved differ depending on the level of adiposity of an individual. What is likely driving energy intake leading to weight gain is compensatory responses, responses to environmental cues related to hedonics, and habits. The evidence suggests that hunger does not lead to weight gain, but it does influence weight loss such that the extent to which an individual feels hungry, depends on their motivations to eat, including whether they feel deprived of their psychological needs (Stubbs & Turicchi, 2021). In the context of a negative energy balance, it is likely that reactive processes override homeostatic processes to promote increased energy intake, therefore, inhibiting weight loss success. In the context of overeating leading to significant weight gain, there is little evidence that increased body weight or fat mass

exerts homeostatic negative feedback to decrease energy intake, appetite, hunger in order to regulate body weight (Stubbs et al., 2018). This indicates that in individuals with a higher BMI, homeostatic processes fail to compensate for a positive energy balance which can lead to overweight and obesity.

2.7. Discussion

This conceptual paper aimed to begin developing a comprehensive framework to identify the core, latent constructs underlying human EBTs by drawing from key psychological, physiological, and neurobiological theories and models. The paper began by summarising and evaluating influential theories of eating behaviour and EBTs that have been developed from theory. This review has highlighted a lack of an agreed framework or standardised set of measures for describing motivations for eating. Additionally, the current state of EBTs has some limitations. The abundance of EBTs has led to a lack of clarity and conceptual overlap, making it difficult to understand the underlying mechanisms of EBTs. Further, this review has drawn attention to the importance of researchers understanding what exactly a scale is measuring and elucidating why a certain scale is being used to measure a specific outcome. A main outcome of section 2.3 of this review suggests that many theories draw on concepts from dual-process models, which propose that eating behaviour is driven by two competing processes. One process involves cognitive, reflective, and decision-making abilities, which includes sensitivity and responsivity to internal cues. The other process involves automatic, reactive, and impulsive behaviour, which includes reacting to external cues in the environment, eating for pleasure/comfort and overeating to reduce negative or enhance positive emotions. Importantly, there are considerable overlaps between theories of eating behaviour, and many newer theories incorporate concepts from older theories.

It is hoped that EBTs may be better understood using this framework. This is because the framework facilitates insight as to which EBTs overlap and which are distinct. However, it is also highlighted that there are other areas of appetite control and eating behaviour that are not explained by this framework because they are beyond the scope of this paper. For example, eating disorders are not included within this framework of EBT. Some EBTs have been developed to measure abnormal eating behaviours. The Yale Food Addiction Scale (Gearhardt et al., 2009), for example, adapts diagnostic criteria from the DSM to measure the construct of food addiction. There is some evidence to suggest food and drugs of abuse utilise similar pathways in the brain: opiate and dopamine systems (Hoebel et al., 1999;

Nieto et al., 2002). Additionally, the diagnostic criteria for substance abuse is similar to the diagnostic criteria for binge eating disorder (Gold et al., 2003).

There is an ongoing debate about the concept of food addiction, whether highly processed foods are addictive and whether this can help our understanding of overeating and obesity. Evidence for the concept of food addiction includes similarities between highly-processed foods and drugs of abuse such as that both are highly reinforcing and some (but not all) individuals consume them compulsively (Gearhardt & Hebebrand, 2021b). Additionally, highly-processed foods are associated with craving, reduced control over consumption and continued use despite negative consequences which are all behavioural indicators of addiction (Gearhardt & Hebebrand, 2021b).

However, there is also debate against the concept of food addiction. For example, treatment for somatic and mental disorders require exclusion, which is not possible because humans have a physiological need to ingest sufficient calories to maintain body weight (Hebebrand & Gearhardt, 2021). The concept of food addiction also lacks validation and has not led to any new and successful treatments for obesity (Hebebrand & Gearhardt, 2021). Overall, researchers do agree that addictive-like eating exists, however, there are disagreements about whether highly processed foods are addictive and the societal implications for the food addiction concept (Gearhardt & Hebebrand, 2021a). Due to the ongoing debate about food addiction, and because disordered eating goes beyond the scope of the current paper, disordered eating is subsumed into the periphery of the conceptual diagram of EBTs (Figure 2.2), which includes examples of “aberrant” eating behaviour. Disordered eating therefore appears more of an outcome domain than a causal/explanatory domain and for these reasons EBTs that measure disordered eating are not currently included within the framework of EBTs.

One notable framework proposed by Vainik et al. (2015) suggests that many EBTs could be capturing the same underlying construct, termed "uncontrolled eating," which involves increased appetite and decreased self-control. The authors developed a continuum model of uncontrolled eating, incorporating five EBTs: eating impulsivity, hedonic hunger, emotional eating, disinhibition, and binge eating, which

measure different ranges of values along this continuum. This framework has begun to develop a comprehensive perspective of the current literature by suggesting that EBTs are measuring different degrees of lack of control of eating. However, future research is required to understand if this framework can account for a wider variation of EBTs. While there have been some attempts to integrate EBTs into a conceptual framework, much of this research is data driven, meaning it lacks theoretical underpinnings.

The next sections (2.4 and 2.5) of this review, summarised key physiological and neurobiological concepts with the aim of incorporating these concepts with psychological theory. The physiological perspective of appetite control and energy balance reveals the importance of homeostatic processes, manifesting as physiological hunger and satiety responses, in understanding eating behaviour. These processes constitute essential traits that should be included in a framework of eating behaviour. Some existing EBTs already measure physiological hunger, such as the hunger subscale of the Adult Eating Behaviour Questionnaire (AEBQ). Additionally, EBTs like the satiety responsiveness scale from the AEBQ capture an individual's sensitivity to internal levels of satiety, representing homeostatic processes. Neurobiological studies further emphasise that appetite regulation involves not only homeostatic mechanisms but also hedonic and reward-based pathways. Pleasure and reward play a central role in controlling or lack of control over energy intake. Neurobiological mechanisms, including food reward, executive function, affect-related, and stress-related eating, should also be incorporated into EBTs to provide a comprehensive understanding of eating behaviour. Emotional eating, studied extensively, has led to the development of EBTs that measure various aspects of emotional eating.

A critical issue which is emphasised by this review, lies in the lack of integration between physiological and neurobiological approaches to eating behaviour and psychological theories. This disconnect hinders a comprehensive understanding of the complex interplay between physiological processes and psychological factors that influence eating behaviour. To address these limitations, this paper proposed a new framework that categorises EBTs into three higher-order domains: reflective processes, reactive processes, and homeostatic eating. These domains are labelled.

However, several other synonymous labels could also be used depending on the context for which this framework is used.

Reflective processes encompass conscious cognitive reasoning and behaviour change strategies, but their influence on energy balance is limited due to environmental, physiological, and hedonic factors that encourage overconsumption. Weight loss interventions often rely on reflective strategies, but evidence shows that maintaining weight loss is challenging due to increased appetite/motivation to eat and reduced energy expenditure which can lead to weight gain and re-gain. Reactive processes involve automatic eating behaviour triggered by external stimuli, emotions, habits, and desires. These processes can override homeostatic signals, also leading to overeating and weight gain. Environmental cues, learned motivations, and habits play a significant role in reactive eating behaviour, making it difficult to resist overeating. Lastly, homeostatic eating refers to the conscious awareness of internal hunger and satiety cues and is a domain less explored by existing EBTs. Homeostatic eating relies on physiological signals responding to energy deficits, but EBTs targeting this domain specifically do not exist. This therefore provides an avenue for further study.

Future research should aim to develop EBTs that specifically measure awareness of internal hunger and satiety cues. It is noted that homeostatic eating may or may not require interoceptive awareness depending on whether an individual is unconsciously compensating for their energy intake over the long-term. Additionally, the influence of homeostatic processes likely also depends on whether an individual is in a positive or negative energy balance and the strength of an energy imbalance. Consequently, unpicking the complexity of homeostatic processes could provide valuable insights into energy balance regulation and obesity.

2.8. Conclusions and Future Directions

To conclude, this paper proposes a starting point to developing a comprehensive framework of EBTs that encompasses reflective, reactive, and homeostatic processes. This framework has helped to understand EBTs better by identifying the core, latent constructs underlying EBT measurements as well as aligning EBT constructs with existing theory. By considering both psychological and physiological dimensions, this framework aims to bridge existing gaps in our understanding of human eating behaviour.

It is important to highlight that the current review is a starting point to a comprehensive framework of EBTs and not a final framework. Thus, the planned next steps for the work are to take a more inclusive and consultative approach to build on the framework with input from other experts in the field. For future research, we aim to use the Delphi method, and to construct a survey, using the current framework to generate a first round of feedback to try and capture elements that may be missing or under-developed. We hope to use this provisional model as a starting point for discussion with the aim of achieving consensus about the framework, and identifying any constructs that require further development.

A similar and comprehensive approach to the Delphi method has been used in the behaviour change field with the recent development of behaviour change ontology. It has been identified that descriptions of behaviour change interventions vary widely and this hinders the ability to compare or replicate studies and develop and evaluate the effectiveness of interventions (Marques et al., 2023). To address this, Michie et al. (2013) developed the behaviour change taxonomy (BCTTv1). Behaviour change techniques (BCTs) are the smallest elements of a behaviour change intervention and when developing the BCTTv1, a Delphi-like method was used to include the input of 400 experts, which led to 93 BCTs being organised into 16 higher-order groupings. Since its development, the BCTTv1 has been widely cited in more than 5000 published studies and it has enabled a structured method for designing and evaluating behaviour change interventions. The label of ‘v1’ highlights that the taxonomy would need development as experts and users gave feedback and as the

field advanced. This user feedback included responses from surveys, researchers, and interview-based consultations, which improved the labels and definitions and developed the structure of the taxonomy (Corker et al., 2022).

Another necessary step to improve the BCTTv1 was the development of a behaviour change technique ontology (BCTO). Ontologies are structures for expressing knowledge by defining entities and their relationships (Arp et al., 2015). They offer a more comprehensive way of representing information than taxonomies and are designed to be added to with new information. The development of the BCTO was also informed by user feedback which suggested the need for additional BCTs, amendments to labels, definitions and groupings and improvements to improve clarity (Marques et al., 2023). Taking an ontology approach to behaviour change has extended and improved upon the BCTTv1 and the authors note that the BCTO should still be updated and improved using on ongoing and collaborative process. Although the field of EBTs is much smaller than that of the behaviour change field, using similar methods to the BCTO would also benefit the EBT field and improve the current framework in this paper.

Indeed, a Delphi-like approach has also recently been used in the field of personality. Irwing et al. (2023) have developed a comprehensive taxonomy of unique personality facets including 1772 personality items taken from multiple personality inventories. The inventory, termed the Facet-level Multidimensional Assessment of Personality (Facet MAP), version 1, was developed through an iterative, multi-stage, multi-method approach. After identifying 136 potential factors for inclusion within the taxonomy, the authors conducted a panel review whereby each panel member individually evaluated each pair of facets until 10 facet pairs in a row were thought to be unique. The panel members also identified problems with the conceptual coherences of the included scales and systematically compared the item content of each facet with all other facets. Overall, taking a consultative approach such as including a panel review has been found to be an effective procedure (DeMaio & Landreth, 2004). Accordingly, taking inspiration from the personality field and behaviour change, approaches like the Delphi method are needed to advance future research.

Further research and development of new EBT measures within this framework will also enable researchers to gain deeper insights into the underlying motivations for eating and contribute to the development of effective interventions for managing eating behaviour and obesity. Further research is also required to begin refining the number of EBTs used in studies, so that only the most internally and externally valid EBT are used. An important aim is for EBTs to have better explanatory power in human eating behaviour and obesity research. Future research should also test whether this framework is empirically supported through using data analysis techniques such as factor analysis. This will enable validation of the conceptual framework by confirming whether the hypothesised relationships and patterns exist in datasets that measure EBTs in real people. It is also important to note that whist the majority of this paper focuses on EBTs that measure overeating, which is important for the study of obesity, it is important to consider the implications of this framework beyond the field of obesity. Therefore, future research should consider how the current paper and the proposed framework can be used to explain and improve diet choice, health and wellbeing, food insecurity and healthy eating in childhood development. Overall, these next steps are vital to further understand how human eating behaviour is influenced by different domains/latent constructs, and how these domains interact with each other.

2.9. References

Adams, R. C., Chambers, C. D., & Lawrence, N. S. (2019). Do restrained eaters show increased BMI, food craving and disinhibited eating? A comparison of the Restraint Scale and the Restrained Eating scale of the Dutch Eating Behaviour Questionnaire. *Royal Society open science*, *6*(6), 190174.

Agha, M., & Agha, R. (2017). The rising prevalence of obesity: part A: impact on public health. *International journal of surgery. Oncology*, *2*(7), e17.

American Psychological Association. (2012). *Stress by generation*. https://www.apa.org/news/press/releases/stress/2012/generation.pdf

Arp, R., Smith, B., & Spear, A. D. (2015). *Building ontologies with basic formal ontology*. Mit Press.

Ashcroft, J., Semmler, C., Carnell, S., Van Jaarsveld, C., & Wardle, J. (2008). Continuity and stability of eating behaviour traits in children. *European journal of clinical nutrition*, *62*(8), 985-990.

Augustus-Horvath, C. L., & Tylka, T. L. (2011). The acceptance model of intuitive eating: a comparison of women in emerging adulthood, early adulthood, and middle adulthood. *Journal of counseling psychology*, *58*(1), 110.

Barrada, J. R., van Strien, T., & Cebolla, A. (2016). Internal structure and measurement invariance of the Dutch eating behavior questionnaire (DEBQ) in a (nearly) representative Dutch community sample. *European Eating Disorders Review*, *24*(6), 503-509.

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. *Journal of Neuroscience*, *26*(19), 5160-5166.

Bem, D. J. (2011). Feeling the future: experimental evidence for anomalous retroactive influences on cognition and affect. *Journal of personality and social psychology*, *100*(3), 407.

Berridge, K. C., & Kringelbach, M. L. (2008). Affective neuroscience of pleasure: reward in humans and animals. *Psychopharmacology*, *199*(3), 457-480.

Berthoud, H.-R. (2011). Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Current opinion in neurobiology*, *21*(6), 888-896.

Berthoud, H.-R., & Morrison, C. (2008). The brain, appetite, and obesity. *Annu. Rev. Psychol.*, *59*, 55-92.

Blaxter, K. (1989). *Energy metabolism in animals and man*. CUP Archive.

Blundell, J., 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 & Behavior*, *152*, 473-478.

Blundell, J., Goodson, S., & Halford, J. (2001). Regulation of appetite: role of leptin in signalling systems for drive and satiety. *International Journal of Obesity*, *25*(1), S29-S34.

Blundell, J., & Hill, A. (1985). Analysis of hunger: inter-relationships with palatability, nutrient composition and eating. Recent advances in obesity research IV: proceedings of the International Congress on Obesity 5-8 October, 1983 New York, USA/edited by Jules Hirsch, Theodore B. Van Itallie,

Blundell, J., & Stubbs, R. (1999). High and low carbohydrate and fat intakes: limits imposed by appetite and palatability and their implications for energy balance. *European journal of clinical nutrition*, *53*(1), s148-s165.

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

Blundell, J. E., Gibbons, C., Beaulieu, K., Casanova, N., Duarte, C., Finlayson, G., Stubbs, R. J., & Hopkins, M. (2020). The drive to eat in homo sapiens: Energy expenditure drives energy intake. *Physiology & Behavior*, *219*, 112846.

Blundell, J. E., & Gillett, A. (2001). Control of food intake in the obese. *Obesity research*, *9*(S11), 263S-270S.

Blundell, J. E., & MacDiarmid, J. I. (1997). Passive Overconsumption Fat Intake and Short-Term Energy Balance a. *Annals of the New York Academy of Sciences*, *827*(1), 392-407.

Blundell, J. E., & Tremblay, A. (1995). Appetite control and energy (fuel) balance. *Nutrition Research Reviews*, *8*(1), 225-242.

Braden, A., Emley, E., Watford, T., Anderson, L., & Musher-Eizenman, D. (2020). Self-reported emotional eating is not related to greater food intake: results from two laboratory studies. *Psychology & health*, *35*(4), 500-517.

Brobeck, J. R. (1946). Mechanism of the development of obesity in animals with hypothalamic lesions. *Physiological Reviews*, *26*(4), 541-559.

Bruch, H. (1964). Psychological aspects of overeating and obesity. *Psychosomatics*, *5*(5), 269-274.

Bruch, H., & Touraine, G. (1940). Obesity in childhood: V. The family frame of obese children. *Psychosomatic Medicine*, *2*(2), 141-206.

Bryant, E. J., Rehman, J., Pepper, L. B., & Walters, E. R. (2019). Obesity and eating disturbance: the role of TFEQ restraint and disinhibition. *Current Obesity Reports*, *8*, 363-372.

Campfield, L., Smith, F., & Burn, P. (1996). The OB protein (leptin) pathway-a link between adipose tissue mass and central neural networks. *Hormone and Metabolic Research*, *28*(12), 619-632.

Cannon, W. B. (1929). Organization for physiological homeostasis. *Physiological Reviews*, *9*(3), 399-431.

Cannon, W. B. (1953). Bodily changes in pain, hunger, fear, and rage, ed2. *Boston, CH Branford*, 20-36.

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*, *33*(8), 913-922.

Carnell, S., & Wardle, J. (2007). Measuring behavioural susceptibility to obesity: validation of the child eating behaviour questionnaire. *Appetite*, *48*(1), 104-113.

Carnell, S., & Wardle, J. (2008). Appetite and adiposity in children: evidence for a behavioral susceptibility theory of obesity. *The American journal of clinical nutrition*, *88*(1), 22-29.

Cebolla, A., Barrada, J., Van Strien, T., Oliver, E., & Baños, R. (2014). Validation of the Dutch Eating Behavior Questionnaire (DEBQ) in a sample of Spanish women. *Appetite*, *73*, 58-64.

Cheon, B. K., Sim, A., Lee, L., & Forde, C. (2019). Avoiding hunger or attaining fullness? Implicit goals of satiety guide portion selection and food intake patterns. *Appetite*, *138*, 10-16.

Cohen, D. A., & Babey, S. H. (2012). Contextual influences on eating behaviours: heuristic processing and dietary choices. *Obesity Reviews*, *13*(9), 766-779.

Corker, E., Marques, M. M., Johnston, M., West, R., Hastings, J., & Michie, S. (2022). Behaviour change techniques taxonomy v1: Feedback to inform the development of an ontology. *Wellcome open research*, *7*.

Croker, H., Cooke, L., & Wardle, J. (2011). Appetitive behaviours of children attending obesity treatment. *Appetite*, *57*(2), 525-529.

Dakin, C., Beaulieu, K., Hopkins, M., Gibbons, C., Finlayson, G., & Stubbs, R. J. (2023a). Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis. *Obesity Reviews*, *24*(1), e13515.

Dakin, C. A., Finlayson, G., Horgan, G., Palmeira, A. L., Heitmann, B. L., Larsen, S. C., Sniehotta, F. F., & Stubbs, R. J. (2023b). Exploratory analysis of reflective, reactive, and homeostatic eating behaviour traits on weight change during the 18-month NoHoW weight maintenance trial. *Appetite*, *189*, 106980.

Davidson, T. L., & Stevenson, R. J. (2022). Appetitive interoception, the hippocampus and western-style diet. *Reviews in Endocrine and Metabolic Disorders*, 1-15.

Davis, C. (2013). From passive overeating to “food addiction”: a spectrum of compulsion and severity. *International Scholarly Research Notices*, *2013*.

de Castro, J. M., & Plunkett, S. (2002). A general model of intake regulation. *Neuroscience & Biobehavioral Reviews*, *26*(5), 581-595.

DeMaio, T. J., & Landreth, A. (2004). Do different cognitive interview techniques produce different results? *Methods for testing and evaluating survey questionnaires*, 89-108.

DeSteno, D., Gross, J. J., & Kubzansky, L. (2013). Affective science and health: the importance of emotion and emotion regulation. *Health Psychology*, *32*(5), 474.

Devonport, T. J., Nicholls, W., & Fullerton, C. (2019). A systematic review of the association between emotions and eating behaviour in normal and overweight adult populations. *Journal of health psychology*, *24*(1). https://doi.org/http://dx.doi.org/10.1177/1359105317697813

Diggins, A., Woods-Giscombe, C., & Waters, S. (2015). The association of perceived stress, contextualized stress, and emotional eating with body mass index in college-aged Black women. *Eating behaviors*, *19*, 188-192.

Dollard, J., & Miller, N. E. (1950). Personality and psychotherapy; an analysis in terms of learning, thinking, and culture.

Dombrowski, S. U., Knittle, K., Avenell, A., Araújo-Soares, V., & Sniehotta, F. F. (2014). Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. *BMJ*, *348*.

Domoff, S. E., Meers, M. R., Koball, A. M., & Musher-Eizenman, D. (2014). The validity of the Dutch eating behavior questionnaire: some critical remarks. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, *19*, 137-144.

Dunton, G. F., Rothman, A. J., Leventhal, A. M., & Intille, S. S. (2021). How intensive longitudinal data can stimulate advances in health behavior maintenance theories and interventions. *Translational behavioral medicine*, *11*(1), 281-286.

Epel, E. S., Tomiyama, A. J., Mason, A. E., Laraia, B. A., Hartman, W., Ready, K., Acree, M., Adam, T. C., St. Jeor, S., & Kessler, D. (2014). The reward-based eating drive scale: a self-report index of reward-based eating. *PloS one*, *9*(6), e101350.

Fairburn, C. G., & Beglin, S. J. (1994). Assessment of eating disorders: Interview or self-report questionnaire? *International Journal of Eating Disorders*, *16*(4), 363-370.

Finlayson, G., King, N., & Blundell, J. E. (2007). Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews*, *31*(7), 987-1002.

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.

Friese, M., Hofmann, W., & Wänke, M. (2008). When impulses take over: Moderated predictive validity of explicit and implicit attitude measures in predicting food choice and consumption behaviour. *British Journal of Social Psychology*, *47*(3), 397-419.

Garcia Ulen, C., Huizinga, M. M., Beech, B., & Elasy, T. A. (2008). Weight regain prevention. *Clinical Diabetes*, *26*(3), 100-113.

Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale food addiction scale. *Appetite*, *52*(2), 430-436.

Gearhardt, A. N., & Hebebrand, J. (2021a). The concept of “food addiction” helps inform the understanding of overeating and obesity: Debate Consensus. *The American journal of clinical nutrition*, *113*(2), 274-276.

Gearhardt, A. N., & Hebebrand, J. (2021b). The concept of “food addiction” helps inform the understanding of overeating and obesity: YES. *The American journal of clinical nutrition*, *113*(2), 263-267.

Gold, M. S., Frost-Pineda, K., & Jacobs, W. S. (2003). Overeating, binge eating, and eating disorders as addictions. *Psychiatric Annals*, *33(2)*, 117-122.

Greaves, C. J., Sheppard, K. E., Abraham, C., Hardeman, W., Roden, M., Evans, P. H., & Schwarz, P. (2011). Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions. *BMC Public Health*, *11*(1), 1-12.

Groesz, L. M., McCoy, S., Carl, J., Saslow, L., Stewart, J., Adler, N., Laraia, B., & Epel, E. (2012). What is eating you? Stress and the drive to eat. *Appetite*, *58*(2), 717-721.

Hall, K. D. (2006). Computational model of in vivo human energy metabolism during semistarvation and refeeding. *American Journal of Physiology-Endocrinology and Metabolism*, *291*(1), E23-E37.

Hall, K. D., Farooqi, I. S., Friedman, J. M., Klein, S., Loos, R. J., Mangelsdorf, D. J., O'Rahilly, S., Ravussin, E., Redman, L. M., & Ryan, D. H. (2022). The energy balance model of obesity: beyond calories in, calories out. *The American Journal of Clinical Nutrition*, *115*(5), 1243-1254.

Hebebrand, J., & Gearhardt, A. N. (2021). The concept of “food addiction” helps inform the understanding of overeating and obesity: NO. *The American journal of clinical nutrition*, *113*(2), 268-273.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of personality*.

Herman, C. P., & Polivy, J. (1975). Anxiety, restraint, and eating behavior. *Journal of abnormal psychology*, *84*(6), 666.

Herman, C. P., & Polivy, J. (1980). Restrained eating. in A. J. Stunkard (Ed.). *Obesity*, 208-225. Philadelphia: Saunders.

Herman, C. P., & Polivy, J. (1983). A boundary model for the regulation of eating. *Psychiatric Annals*, *13(12)*, 918-927.

Herman, C. P., & Polivy, J. (2014). Models, monitoring, and the mind: Comments on Wansink and Chandon's “Slim by Design”. *Journal of Consumer Psychology*, *24*(3), 432-437.

Hervey, G. (1969). Regulation of energy balance. *Nature*, *222*, 629-631.

Higgs, M., & Lichtenstein, S. (2010). Exploring the ‘jingle fallacy’: a study of personality and values. *Journal of General Management*, *36*(1), 43-61.

Higgs, S., Spetter, M. S., Thomas, J. M., Rotshtein, P., Lee, M., Hallschmid, M., & Dourish, C. T. (2017). Interactions between metabolic, reward and cognitive processes in appetite control: Implications for novel weight management therapies. *Journal of Psychopharmacology*, *31*(11), 1460-1474.

Hoebel, B. G., Rada, P. V., Mark, G. P., & Pothos, E. N. (1999). Neural systems for reinforcement and inhibition of behavior: relevance to eating, addiction, and depression.

Hopkins, M., Beaulieu, K., Myers, A., Gibbons, C., & Blundell, J. E. (2017). Mechanisms responsible for homeostatic appetite control: theoretical advances and practical implications. *Expert Review of Endocrinology & Metabolism*, *12*(6), 401-415.

Hunot, C., Fildes, A., Croker, H., Llewellyn, C. H., Wardle, J., & Beeken, R. J. (2016). Appetitive traits and relationships with BMI in adults: Development of the Adult Eating Behaviour Questionnaire. *Appetite*, *105*, 356-363.

Irwing, P., Hughes, D. J., Tokarev, A., & Booth, T. (2023). Towards a taxonomy of personality facets. *European Journal of Personality*, 08902070231200919.

Iso-Ahola, S. E. (2017). Reproducibility in psychological science: When do psychological phenomena exist? *Frontiers in psychology*, *8*, 879.

Jansen, A., Havermans, R. C., & Nederkoorn, C. (2011a). Cued Overeating. In V. R. Preedy, R. R. Watson, & C. R. Martin (Eds.), *Handbook of Behavior, Food and Nutrition* (pp. 1431-1443). Springer New York. https://doi.org/10.1007/978-0-387-92271-3_92

Jansen, A., Nederkoorn, C., Roefs, A., Bongers, P., Teugels, T., & Havermans, R. (2011b). The proof of the pudding is in the eating: Is the DEBQ-external eating scale a valid measure of external eating? *International Journal of Eating Disorders*, *44*(2), 164-168.

Jansen, A., Schyns, G., Bongers, P., & van den Akker, K. (2016). From lab to clinic: extinction of cued cravings to reduce overeating. *Physiology & Behavior*, *162*, 174-180.

Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*, *36*(5), 665-674.

Kaplan, H. I., & Kaplan, H. S. (1957). The psychosomatic concept of obesity. *Journal of Nervous and Mental Disease*.

Kaplan, S. A., Madden, V. P., Mijanovich, T., & Purcaro, E. (2013). The perception of stress and its impact on health in poor communities. *Journal of community health*, *38*, 142-149.

Kavanagh, D. J., Andrade, J., & May, J. (2005). Imaginary relish and exquisite torture: the elaborated intrusion theory of desire. *Psychological review*, *112*(2), 446.

Keesey, R., & Corbett, S. (1990). Adjustments in daily energy expenditure to caloric restriction and weight loss by adult obese and lean Zucker rats. *International Journal of Obesity*, *14*(12), 1079-1084.

Keller, C., & Hartmann, C. (2016). Not merely a question of self-control: The longitudinal effects of overeating behaviors, diet quality and physical activity on dieters' perceived diet success. *Appetite*, *107*, 213-221.

Kelley, T. L. (1927). Interpretation of educational measurements.

Kemps, E., Goossens, L., Petersen, J., Verbeken, S., Vervoort, L., & Braet, C. (2020). Evidence for enhancing childhood obesity treatment from a dual-process perspective: A systematic literature review. *Clinical psychology review*, *77*, 101840.

Kennedy, G. C. (1953). The role of depot fat in the hypothalamic control of food intake in the rat. *Proceedings of the Royal Society of London. Series B-Biological Sciences*, *140*(901), 578-592.

Kenny, G. P., Webb, P., Ducharme, M. B., Reardon, F. D., & Jay, O. (2008). Calorimetric measurement of postexercise net heat loss and residual body heat storage. *Medicine and Science in Sports and Exercise*, *40*(9), 1629-1636.

Keren, G., & Schul, Y. (2009). Two is not always better than one: A critical evaluation of two-system theories. *Perspectives on psychological science*, *4*(6), 533-550.

Kleiber, M. (1961). The fire of life. An introduction to animal energetics. *The fire of life. An introduction to animal energetics.*

Kringelbach, M. L. (2004). Food for thought: hedonic experience beyond homeostasis in the human brain. *Neuroscience*, *126*(4), 807-819.

Kringelbach, M. L., Stein, A., & van Hartevelt, T. J. (2012). The functional human neuroanatomy of food pleasure cycles. *Physiology & behavior*, *106*(3), 307-316.

Kwasnicka, D., Dombrowski, S. U., White, M., & Sniehotta, F. (2016). Theoretical explanations for maintenance of behaviour change: a systematic review of behaviour theories. *Health Psychology Review*, *10*(3), 277-296.

Levitsky, D. A. (2005). The non-regulation of food intake in humans: hope for reversing the epidemic of obesity. *Physiology & Behavior*, *86*(5), 623-632.

Llewellyn, C., & Wardle, J. (2015). Behavioral susceptibility to obesity: gene–environment interplay in the development of weight. *Physiology & Behavior*, *152*, 494-501.

Lowe, M. R., & Butryn, M. L. (2007). Hedonic hunger: a new dimension of appetite? *Physiology & Behavior*, *91*(4), 432-439.

Lowe, M. R., Butryn, M. L., Didie, E. R., Annunziato, R. A., Thomas, J. G., Crerand, C. E., Ochner, C. N., Coletta, M. C., Bellace, D., & Wallaert, M. (2009). The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*, *53*(1), 114-118.

Lowe, M. R., & Levine, A. S. (2005). Eating motives and the controversy over dieting: eating less than needed versus less than wanted. *Obesity research*, *13*(5), 797-806.

Marques, M. M., Wright, A. J., Corker, E., Johnston, M., West, R., Hastings, J., Zhang, L., & Michie, S. (2023). The behaviour change technique ontology: transforming the behaviour change technique taxonomy v1. *Wellcome open research*, *8*.

Masheb, R. M., & Grilo, C. M. (2002). On the relation of flexible and rigid control of eating to body mass index and overeating in patients with binge eating disorder. *International Journal of Eating Disorders*, *31*(1), 82-91.

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*, 795.

Mayer, J. (1953). Glucostatic mechanism of regulation of food intake. *New England Journal of Medicine*, *249*(1), 13-16.

McGuire, J. T., & Botvinick, M. M. (2010). Prefrontal cortex, cognitive control, and the registration of decision costs. *Proceedings of the National Academy of Sciences*, *107*(17), 7922-7926.

McGuire, M. T., Wing, R. R., Klem, M. L., & Hillf, J. O. (1999). Behavioral strategies of individuals who have maintained long-term weight losses. *Obesity research*, *7*(4), 334-341.

Mellinkoff, S. M., Frankland, M., Boyle, D., & Greipel, M. (1956). Relationship between serum amino acid concentration and fluctuations in appetite. *Journal of applied physiology*, *8*(5), 535-538.

Meyers, A. W., & Stunkard, A. J. (1980). Food accessibility and food choice: A test of Schachter's externality hypothesis. *Archives of general psychiatry*, *37*(10), 1133-1135.

Michie, S. (2014). ABC of behaviour change theories. *(No Title)*.

Michie, S., Richardson, M., Johnston, M., Abraham, C., Francis, J., Hardeman, W., Eccles, M. P., Cane, J., & Wood, C. E. (2013). The behavior change technique taxonomy (v1) of 93 hierarchically clustered techniques: building an international consensus for the reporting of behavior change interventions. *Annals of Behavioral Medicine*, *46*(1), 81-95.

Michie, S., Van Stralen, M. M., & West, R. (2011). The behaviour change wheel: a new method for characterising and designing behaviour change interventions. *Implementation science*, *6*(1), 1-12.

Mills, J. S., Polivy, J., & Herman, C. P. (2021). Distinguishing dieting from restrained eating: A rejoinder to Lowe (2021). *Appetite*, *165*, 105295. https://doi.org/https://doi.org/10.1016/j.appet.2021.105295

Mowrer, O. (1950). Learning theory and personality dynamics: selected papers.

Neal, D. T., Wood, W., & Quinn, J. M. (2006). Habits—A repeat performance. *Current directions in psychological science*, *15*(4), 198-202.

Neel, J. V. (1962). Diabetes mellitus: a “thrifty” genotype rendered detrimental by “progress”? *American journal of human genetics*, *14*(4), 353.

Nieto, M. M., Wilson, J., Cupo, A., Roques, B. P., & Noble, F. (2002). Chronic morphine treatment modulates the extracellular levels of endogenous

enkephalins in rat brain structures involved in opiate dependence: a microdialysis study. *Journal of Neuroscience*, *22*(3), 1034-1041.

Nisbett, R. E. (1968a). Determinants of food intake in obesity. *Science*, *159*(3820), 1254-1255.

Nisbett, R. E. (1968b). Taste, deprivation, and weight determinants of eating behavior. *Journal of personality and social psychology*, *10*(2), 107.

Nisbett, R. E. (1972). Hunger, obesity, and the ventromedial hypothalamus. *Psychological review*, *79*(6), 433.

Ogden, J. (2011). *The psychology of eating: From healthy to disordered behavior*. John Wiley & Sons.

Ouwens, M. A., Van Strien, T., Van Leeuwe, J., & Van der Staak, C. (2009). The dual pathway model of overeating. Replication and extension with actual food consumption. *Appetite*, *52*(1), 234-237.

Park, C. L., & Iacocca, M. O. (2014). A stress and coping perspective on health behaviors: theoretical and methodological considerations. *Anxiety, Stress & Coping*, *27*(2), 123-137.

Paulhus, D. L., & Vazire, S. (2007). The self-report method. *Handbook of research methods in personality psychology*, *1*(2007), 224-239.

Peters, J. C., Wyatt, H. R., Donahoo, W. T., & Hill, J. (2002). From instinct to intellect: the challenge of maintaining healthy weight in the modern world. *obesity reviews*, *3*(2), 69-74.

Pfefferbaum, A., Lim, K. O., Zipursky, R. B., Mathalon, D. H., Rosenbloom, M. J., Lane, B., Ha, C. N., & Sullivan, E. V. (1992). Brain gray and white matter volume loss accelerates with aging in chronic alcoholics: a quantitative MRI study. *Alcoholism: Clinical and Experimental Research*, *16*(6), 1078-1089.

Phelan, S., Liu, T., Gorin, A., Lowe, M., Hogan, J., Fava, J., & Wing, R. R. (2009). What distinguishes weight-loss maintainers from the treatment-seeking obese? Analysis of environmental, behavioral, and psychosocial variables in diverse populations. *Annals of Behavioral Medicine*, *38*(2), 94-104.

Polidori, D., Sanghvi, A., Seeley, R. J., & Hall, K. D. (2016). How strongly does appetite counter weight loss? Quantification of the feedback control of human energy intake. *Obesity*, *24*(11), 2289-2295.

Polivy, J., & Herman, C. P. (1976a). Clinical depression and weight change: a complex relation. *Journal of abnormal psychology*, *85*(3), 338.

Polivy, J., & Herman, C. P. (1976b). Effects of alcohol on eating behavior: influence of mood and perceived intoxication. *Journal of abnormal psychology*, *85*(6), 601.

Polivy, J., & Herman, C. P. (1985). Dieting and binging: A causal analysis. *American psychologist*, *40*(2), 193.

Polivy, J., Herman, C. P., & Mills, J. S. (2020). What is restrained eating and how do we identify it? *Appetite*, *155*, 104820.

Polivy, J., Herman, C. P., & Mills, J. S. (2023). Assessment of Restrained Eating and Dieting. *Assessment of Eating Behavior*, *6*, 15.

Prentice, A., Black, A., Murgatroyd, P., Goldberg, G., & Coward, W. (1989). Metabolism or appetite: questions of energy balance with particular reference to obesity. *Journal of human nutrition and dietetics*, *2*(2), 95-104.

Prentice, A. M., & Jebb, S. A. (2003). Fast foods, energy density and obesity: a possible mechanistic link. *obesity reviews*, *4*(4), 187-194.

Price, M., Lee, M., Higgs, S., & Maw, J. (2016). A dual-process approach to exploring the role of delay discounting in obesity. *Physiology and Behavior*, *162*. https://doi.org/http://dx.doi.org/10.1016/j.physbeh.2016.02.020

Pudel, V., Metzdorff, M., & Oetting, M. (1975). Zur Persoenlichkeit adipoeser in psychologischen tests unter beruecksichtigung latent fettsuechtiger. *Zeitschrift für psychosomatische Medizin und Psychoanalyse*, 345-361.

Racine, S. E., Hagan, K. E., & Schell, S. E. (2019). Is all nonhomeostatic eating the same? Examining the latent structure of nonhomeostatic eating processes in women and men. *Psychological Assessment*, *31*(10), 1220.

Raio, C. M., Orederu, T. A., Palazzolo, L., Shurick, A. A., & Phelps, E. A. (2013). Cognitive emotion regulation fails the stress test. *Proceedings of the National Academy of Sciences*, *110*(37), 15139-15144.

Raubenheimer, D., & Simpson, S. J. (2019). Protein leverage: theoretical foundations and ten points of clarification. *Obesity*, *27*(8), 1225-1238.

Robbins, T., & Fray, P. (1980). Stress-induced eating: fact, fiction or misunderstanding? *Appetite*, *1*(2), 103-133.

Rodin, J. (1975). Causes and consequences of time perception differences in overweight and normal weight people. *Journal of personality and social psychology*, *31*(5), 898.

Rodin, J. (1978). Has the distinction between internal versus external control of feeding outlived its usefulness. *Recent advances in obesity research*, *2*(Part 2), 75-85.

Rodin, J. (1981). Current status of the internal–external hypothesis for obesity: What went wrong? *American psychologist*, *36*(4), 361.

Rodin, J., Slochower, J., & Fleming, B. (1977). The effects of degree of obesity, age of onset, and energy deficit on external responsiveness. *Journal of Comparative and Physiological Psychology*, *91*, 586-597.

Rothman, A. J., Sheeran, P., & Wood, W. (2009). Reflective and automatic processes in the initiation and maintenance of dietary change. *Annals of Behavioral Medicine*, *38*(suppl_1), s4-s17.

Rozin, P. (1999). Food is fundamental, fun, frightening, and far-reaching. *Social research*, 9-30.

Rozin, P. (2005). The meaning of food in our lives: a cross-cultural perspective on eating and well-being. *Journal of Nutrition Education and Behavior*, *37*, S107-S112.

Satter, E. (2007). Eating competence: definition and evidence for the Satter Eating Competence model. *Journal of Nutrition Education and Behavior*, *39*(5), S142-S153.

Schachter, S. (1967). Cognitive effects on bodily functioning: Studies of obesity and eating. *Glass, D.(Ed.): Neurophysiology and Emotion, New York (Rockefeller University Press), pp. 117-159.*

Schachter, S. (1968). Obesity and eating: internal and external cues differentially affect the eating behavior of obese and normal subjects. *Science*, *161*(3843), 751-756.

Schachter, S., Goldman, R., & Gordon, A. (1968). Effects of fear, food deprivation, and obesity on eating. *Journal of personality and social psychology*, *10*(2), 91.

Schachter, S., & Gross, L. P. (1968). Manipulated time and eating behavior. *Journal of personality and social psychology*, *10*(2), 98.

Schaumberg, K., Anderson, D., Anderson, L., Reilly, E., & Gorrell, S. (2016). Dietary restraint: what's the harm? A review of the relationship between dietary restraint, weight trajectory and the development of eating pathology. *Clinical Obesity*, *6*(2), 89-100.

Schüz, B., Schüz, N., & Ferguson, S. G. (2015). It's the power of food: individual differences in food cue responsiveness and snacking in everyday life. *International Journal of Behavioral Nutrition and Physical Activity*, *12*(1), 1-8.

Seligman, M. E., & Csikszentmihalyi, M. (2000). *Positive psychology: An introduction* (Vol. 55). American Psychological Association.

Sharifi, N., Mahdavi, R., & Ebrahimi-Mameghani, M. (2013). Perceived barriers to weight loss programs for overweight or obese women. *Health promotion perspectives*, *3*(1), 11.

Sheeran, P., Gollwitzer, P. M., & Bargh, J. A. (2013). Nonconscious processes and health. *Health Psychology*, *32*(5), 460.

Shin, A. C., Zheng, H., & Berthoud, H.-R. (2009). An expanded view of energy homeostasis: neural integration of metabolic, cognitive, and emotional drives to eat. *Physiology & Behavior*, *97*(5), 572-580.

Shouse, S. H., & Nilsson, J. (2011). Self-silencing, emotional awareness, and eating behaviors in college women. *Psychology of women quarterly*, *35*(3), 451-457.

Siervo, M., Fruhbeck, G., Dixon, A., Goldberg, G. R., Coward, W. A., Murgatroyd, P. R., Prentice, A. M., & Jebb, S. A. (2008). Efficiency of autoregulatory homeostatic responses to imposed caloric excess in lean men. *American Journal of Physiology-Endocrinology and Metabolism*, *294*(2), E416-E424.

Skinner, B. F. (1963). Operant behavior. *American psychologist*, *18*(8), 503.

Sommet, N., Weissman, D. L., Cheutin, N., & Elliot, A. J. (2023). How many participants do I need to test an interaction? Conducting an appropriate power analysis and achieving sufficient power to detect an interaction. *Advances in Methods and Practices in Psychological Science*, *6*(3), 25152459231178728.

Speakman, J. R. (2007). A nonadaptive scenario explaining the genetic predisposition to obesity: the "predation release" hypothesis. *Cell metabolism*, *6*(1), 5-12.

Speakman, J. R. (2014). If body fatness is under physiological regulation, then how come we have an obesity epidemic? *Physiology*, *29*(2), 88-98.

Speakman, J. R. (2018). The evolution of body fatness: trading off disease and predation risk. *Journal of Experimental Biology*, *221*(Suppl_1), jeb167254.

Speakman, J. R., Levitsky, D. A., Allison, D. B., Bray, M. S., De Castro, J. M., Clegg, D. J., Clapham, J. C., Dulloo, A. G., Gruer, L., & Haw, S. (2011). Set points, settling points and some alternative models: theoretical options to understand

how genes and environments combine to regulate body adiposity. *Disease models & mechanisms*, *4*(6), 733-745.

Spiegel, T. A. (1973). Caloric regulation of food intake in man. *Journal of Comparative and Physiological Psychology*, *84*(1), 24.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and social psychology review*, *8*(3), 220-247.

Stroebe, W., Van Koningsbruggen, G. M., Papies, E. K., & Aarts, H. (2013). Why most dieters fail but some succeed: a goal conflict model of eating behavior. *Psychological review*, *120*(1), 110.

Stubbs, J., Whybrow, S., Teixeira, P., Blundell, J., Lawton, C., Westenhoefer, J., Engel, D., Shepherd, R., Mcconnon, Á., & Gilbert, P. (2011). Problems in identifying predictors and correlates of weight loss and maintenance: implications for weight control therapies based on behaviour change. *obesity reviews*, *12*(9), 688-708.

Stubbs, R., Gail, C., Whybrow, S., & Gilbert, P. (2012). The evolutionary inevitability of obesity in modern society: implications for behavioral solutions to weight control in the general population. In *Obesity and weight management: Challenges, practices and health implications* (pp. 29-50). Nova Science Publishers Inc.

Stubbs, R., & Tolkamp, B. (2006). Control of energy balance in relation to energy intake and energy expenditure in animals and man: an ecological perspective. *British Journal of Nutrition*, *95*(4), 657-676.

Stubbs, R. J. (1998). Appetite, feeding behaviour and energy balance in human subjects. *Proceedings of the Nutrition Society*, *57*(3), 341-356.

Stubbs, R. J., Duarte, C., O'Driscoll, R., Turicchi, J., Kwasnicka, D., Sniehotta, F. F., Marques, M. M., Horgan, G., Larsen, S., & Palmeira, A. (2021). The H2020 "NoHoW Project": A Position Statement on Behavioural Approaches to Longer-Term Weight Management. *Obesity facts*, *14*(2), 246-258.

Stubbs, R. J., Hopkins, M., Finlayson, G., Duarte, C., Gibbons, C., & Blundell, J. (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*(5), 698-709.

Stubbs, R. J., Horgan, G., Robinson, E., Hopkins, M., Dakin, C., & Finlayson, G. (2023). Diet composition and energy intake in humans. *Philosophical Transactions of the Royal Society B*, *378*(1888), 20220449.

Stubbs, R. J., Hughes, D. A., Johnstone, A. M., Whybrow, S., Horgan, G. W., King, N., & Blundell, J. (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-R358.

Stubbs, R. J., & Turicchi, J. (2021). From famine to therapeutic weight loss: Hunger, psychological responses, and energy balance-related behaviors. *obesity reviews*, *22*, e13191.

Stunkard, A., & Koch, C. (1964). The interpretation of gastric motility: I. Apparent bias in the reports of hunger by obese persons. *Archives of general psychiatry*, *11*(1), 74-82.

Stunkard, A. J., & Fox, S. (1971). The relationship of gastric motility and hunger: A summary of the evidence. *Psychosomatic Medicine*, *33*(2), 123-134.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Teixeira, P., Going, S. B., Sardinha, L., & Lohman, T. (2005). A review of psychosocial pre-treatment predictors of weight control. *obesity reviews*, *6*(1), 43-65.

Thorndike, E. L. (1904). Introduction to the theory of mental and social measurements. *New York: Science Press*.

Tomiyama, A. J. (2019). Stress and obesity. *Annual review of psychology*, *70*, 703-718.

Tribole, E., & Resch, E. (1995). *Intuitive eating: a recovery book for the chronic dieter: rediscover the pleasures of eating and rebuild your body image*.

Tribole, E., & Resch, E. (2012). *Intuitive eating*. Macmillan.

Tylka, T. L. (2006). Development and psychometric evaluation of a measure of intuitive eating. *Journal of counseling psychology*, *53*(2), 226.

Tylka, T. L., & Kroon Van Diest, A. M. (2013). The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men. *Journal of counseling psychology*, *60*(1), 137.

Vainik, U., Dagher, A., Dubé, L., & Fellows, L. K. (2013). Neurobehavioural correlates of body mass index and eating behaviours in adults: a systematic review. *Neuroscience & Biobehavioral Reviews*, *37*(3), 279-299.

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229-239.

van Jaarsveld, C. H., Llewellyn, C. H., Johnson, L., & Wardle, J. (2011). Prospective associations between appetitive traits and weight gain in infancy. *The American journal of clinical nutrition*, *94*(6), 1562-1567.

van Koningsbruggen, G. M., Stroebe, W., & Aarts, H. (2013). The rise and fall of self-control: temptation-elicited goal activation and effortful goal-directed behavior. *Social Psychological and Personality Science*, *4*(5), 546-554.

Van Strien, T., Engels, R. C., Van Leeuwe, J., & Snoek, H. M. (2005). The Stice model of overeating: tests in clinical and non-clinical samples. *Appetite*, *45*(3), 205-213.

Van Strien, T., Frijters, J. E., Bergers, G. P., & Defares, P. B. (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.

Varkevisser, R., Van Stralen, M., Kroeze, W., Ket, J., & Steenhuis, I. (2019). Determinants of weight loss maintenance: a systematic review. *Obesity Reviews*, *20*(2), 171-211.

Volkow, N. D., Wang, G.-J., & Baler, R. D. (2011). Reward, dopamine and the control of food intake: implications for obesity. *Trends in cognitive sciences*, *15*(1), 37-46.

Volkow, N. D., Wang, G.-J., Fowler, J. S., Tomasi, D., & Baler, R. (2012). Food and drug reward: overlapping circuits in human obesity and addiction. *Brain imaging in behavioral neuroscience*, 1-24.

Volkow, N. D., Wang, G. J., Tomasi, D., & Baler, R. D. (2013). Obesity and addiction: neurobiological overlaps. *obesity reviews*, *14*(1), 2-18.

Warde, A. (2016). *The practice of eating*. John Wiley & Sons.

Wardle, J., Guthrie, C. A., Sanderson, S., & Rapoport, L. (2001). Development of the children's eating behaviour questionnaire. *The Journal of Child Psychology and Psychiatry and Allied Disciplines*, *42*(7), 963-970.

Warren, J. M., Smith, N., & Ashwell, M. (2017). A structured literature review on the role of mindfulness, mindful eating and intuitive eating in changing eating behaviours: effectiveness and associated potential mechanisms. *Nutrition Research Reviews*, *30*(2), 272-283.

Watts, A. G., Kanoski, S. E., Sanchez-Watts, G., & Langhans, W. (2022). The physiological control of eating: signals, neurons, and networks. *Physiological Reviews*, *102*(2), 689-813.

Wendorf, M., & Goldfine, I. D. (1991). Archaeology of NIDDM: excavation of the "thrifty" genotype. *Diabetes*, *40*(2), 161-165.

Westenhoefer, J., Engel, D., Holst, C., Lorenz, J., Peacock, M., Stubbs, J., Whybrow, S., & Raats, M. (2013). Cognitive and weight-related correlates of flexible and rigid restrained eating behaviour. *Eating behaviors*, *14*(1), 69-72.

Westenhoefer, J., Stunkard, A. J., & Pudel, V. (1999). Validation of the flexible and rigid control dimensions of dietary restraint. *International Journal of Eating Disorders*, *26*(1), 53-64.

Williamson, D. A., Martin, C. K., York-Crowe, E., Anton, S. D., Redman, L. M., Han, H., & Ravussin, E. (2007). Measurement of dietary restraint: validity tests of four questionnaires. *Appetite*, *48*(2), 183-192.

Winkielman, P., Berridge, K. C., & Wilbarger, J. L. (2005). Unconscious affective reactions to masked happy versus angry faces influence consumption behavior and judgments of value. *Personality and Social Psychology Bulletin*, *31*(1), 121-135.

Wirtshafter, D., & Davis, J. D. (1977). Set points, settling points, and the control of body weight. *Physiology & Behavior*, *19*(1), 75-78.

Woods, S. C., & Ramsay, D. S. (2011). Food intake, metabolism and homeostasis. *Physiology & Behavior*, *104*(1), 4-7.

Wooley, S. C. (1972). Physiologic versus cognitive factors in short term food regulation in the obese and nonobese. *Psychosomatic Medicine*, *34*(1), 62-68.

Wynne, K., Stanley, S., McGowan, B., & Bloom, S. (2005). Appetite control. *Journal of Endocrinology*, *184*(2), 291-318.

Yeomans, M. R., Blundell, J. E., & Leshem, M. (2004). Palatability: response to nutritional need or need-free stimulation of appetite? *British Journal of Nutrition*, *92*(S1), S3-S14.

Zhou, Y., Stubbs, R. J., & Finlayson, G. (2023). A neurocognitive perspective on the relationship between exercise and reward: Implications for weight management and drug addiction. *Appetite*, *182*, 106446.

Chapter 3. Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis

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Note. As this paper is accepted and published, the formatting and layout are consistent with the requirements for Obesity Reviews, references will not follow the APA style and will be placed at the end of the chapter rather than at the end of this thesis.

3.1. Abstract

At present, it is unclear whether eating behavior traits (EBT) predict objectively measured short-term energy intake (EI) and longer-term energy balance as estimated by body mass index (BMI). This systematic review examined the impact of EBTs on BMI and laboratory-based measures of EI in adults (≥18 years) in any BMI category, excluding self-report measures of EI. Articles were searched up until 28th October 2021 using MEDLINE, PsycINFO, EMBASE and Web of Science. Sixteen EBTs were identified and the association between 10 EBTs, EI and BMI were assessed using a random-effects meta-analysis. Other EBT outcomes were synthesized qualitatively. Risk of bias was assessed with the mixed methods appraisal tool. A total of 83 studies were included (mean BMI = 25.20 kg/m^2, mean age = 27 years and mean sample size = 70). Study quality was rated moderately high overall, with some concerns in sampling strategy and statistical analyses. Susceptibility to hunger (n = 6) and binge eating (n = 7) were the strongest predictors of EI. Disinhibition (n = 8) was the strongest predictor of BMI. Overall, EBTs may be useful as phenotypic markers of susceptibility to overconsume or develop obesity (PROSPERO: CRD42021288694).

3.2. Introduction

Obesity is a common, serious, and costly condition with significant health care and societal costs.[1] There is current interest in identifying psychological and physiological markers that characterise individuals who are susceptible to weight gain. Eating behaviours influence amount and types of foods eaten and hence energy intake (EI).[2] Eating behaviour traits (EBT) are considered to be reliable, acquired indices of food-related behaviours.[3] EBTs have been extensively studied in an attempt to identify potential markers that detect tendency to overconsume.[4] This has led to the development of several constructs and psychometric measures, which aim to capture important individual differences in eating behaviour and disordered eating symptomology e.g. the Three Factor Eating Questionnaire (TFEQ) with sub-scales for cognitive restraint, susceptibility to hunger and disinhibition.[5] Many EBTs have been reported to be associated with excess food intake.[6-9] However, 'overconsumption' is often assessed by other self-report measures which are prone to misreporting and not truly representative of actual food intake.[10] For example, in a systematic review on the associations between emotions and eating behaviour, the majority of studies associated self-reported emotions with another self-reported EBTs as a proxy of food intake.[11] Associations between EBTs and EI have yet to be extensively reviewed and little is known about which EBTs influence objectively measured EI.

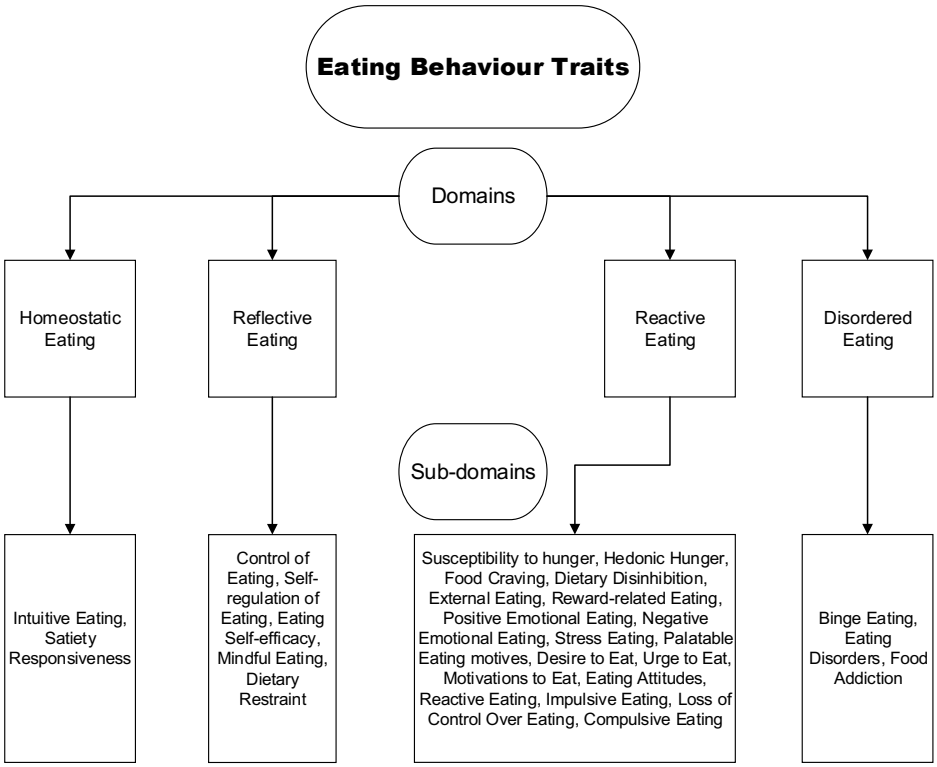
Previous systematic reviews have begun to answer these questions indirectly by examining associations between EBTs and BMI.[9,11,12] BMI is an index of long term energy balance status. Vainik, Dagher, Dubé and Fellows [12] found that several different personality measures were consistently associated with BMI. However, very few measures included were eating-related. French, Epstein, Jeffery, Blundell and Wardle [9] reviewed seven eating behaviour dimensions and found that most of the available data showed positive cross-sectional associations with BMI. However, very few studies reported measures of EI and only four measures were eating-related. Overall, disinhibition had the largest empirical support to link it prospectively with weight gain and a narrative review also found that disinhibition plays a significant role in obesity, diet quality and uncontrolled eating patterns.[13] As few measures

associated with psychological eating behaviour have been reviewed, this systematic review aimed to assess all possible psychological EBTs.

EBTs are theoretical constructs operationalised through eating-related measures. There is currently no database of EBTs, and studies often refer to these traits with different names, e.g., overeating measures, eating behaviour dimensions, appetite measures and eating attitudes. Consequently, a conceptual diagram was developed to capture the scales used to assess EBTs and to a-priori allocate all of the collected scales to theoretical domains (see Figure 3.1). Firstly, a scoping exercise was conducted to create EBT domains, then subdomains were identified, which formed the basis for the search strategy. Secondly, this systematic review was conducted to identify studies that included measures of both EI and BMI in the same population. This systematic review included only laboratory-based measures of EI. Use of a controlled environment allows for more standardised, objective measurement of EI.[14] This is more accurate and precise than self-report EI.[15]

The primary aim of this systematic review was to examine whether EBTs predict short-term EI and whether there is evidence that this effect translates into longer term energy balance, as estimated by BMI. The specific research questions of the review were: (1) What measures are used to capture EBTs? (2) How well do these measures predict short term EI? (3) How well do these measures predict BMI? (4) Do some EBTs and questionnaires used to measure EBTs better predict EI or BMI than others?

Figure 3.1. A conceptual diagram of eating behaviour traits



3.3. Methods

3.3.1. Information sources and search strategy

Four electronic databases were searched: MEDLINE (Ovid), PsycINFO (Ovid), EMBASE (Ovid) and Web of Science (core collection). The MEDLINE, Embase and PsychInfo strategies were run simultaneously as a multi-file search in Ovid on 28th October 2021. Searches for all databases are included in Table A.1. The conceptual diagram, alongside the identification of key databases formed the basis for the search strategy (see Figure 3.1). All proposed EBTs and their domains were included in the search. Key databases such as the UCL database for eating behaviour questionnaires,[16] NIH obesity measures,[17] and Arab psychology scales[18] were used to find relevant eating-related questionnaires to include in the search. Because many studies use EBTs but not in relation to measured eating behaviour, the search also required studies to include key words related to eating behaviour or eating traits (see Table A.1). The two outcome measures were EI and BMI (and their related terms, see Table A.1). Limits were set to include articles published in English (as translating studies into English could lead to relevant information being mistranslated) and humans. The search strategies were peer reviewed by three experts in the field (KB, JS, GF) prior to execution using the PRESS checklist.[19] Duplicates were removed using Screenatron's 'deduplication' tool and were checked to ensure all duplicates found were actual duplications. This systematic review followed PRISMA guidelines[20] and was pre-registered with PROSPERO (registration number = CRD42021288694).

3.3.2. Study selection, inclusion, and exclusion

Articles were included if they recruited adults (≥18 years including older adults) in any BMI category. The presence of the following was not excluded: type 2 diabetes, pre-menopausal/post-menopausal women, cardiovascular diseases. Those with the following comorbidities were excluded: Parkinson's disease, anorexia, bulimia nervosa, binge eating disorder, cancers, inflammatory bowel diseases, intellectual deficiency, psychiatric conditions, sleep disorders, those having undergone weight loss surgery (gastric bypass or bariatric surgery) and those who were pregnant. As binge eating was identified as an EBT in the scoping exercise, any study that used a binge eating scale (e.g. binge eating scale[21]) to classify binge eaters was included.

However, if studies identified participants with binge eating disorder using a version of the DSM e.g. DSM-5 [22], this was not recognised as an EBT and these studies were excluded. Studies that measured psychological variables e.g., stress or mood, were excluded unless an EBT was also measured e.g., emotional eating. In cases where studies measured other variables alongside EBTs, the reviewers discussed whether these variables would be considered potential contaminates of the outcomes. For example, an alcohol preload or a food craving protocol were considered potential contaminates and were excluded if no data was given for a control condition. Self-report measures of EI were excluded as this systematic review focused on laboratory-based measures of EI but were saved for a future review. Laboratory-based measures of EI also included studies where EI was measured in a laboratory and weighed food was given to participants to take home and bring back to be re-weighed, as this measure of EI is considered in the literature to be *ad-libitum* EI.

All titles and abstracts were assessed for eligibility by the main author (CD) and were also independently screened by three researchers (MH, JS, GF), with uncertainty regarding eligibility discussed among the researchers. Reference lists from the resulting reviews were also screened to identify additional articles. Full texts were retrieved and assessed for eligibility by CD and were also independently assessed by JS and GF. Intervention studies that included results for a control condition or baseline measures were included. Studies that only measured one of the outcomes variables (EI or BMI) were excluded. If a study measured both outcome variables but the results for one of the outcome measures was missing, the study authors were contacted for the missing outcome results. In this case, the study was included for data extraction as both outcome variables were measured. Thirty-three authors were contacted about missing data. Five authors responded with the necessary data.

3.3.3. Data extraction and synthesis

Data was extracted by one author (CD) and was cross-checked for data extraction errors by JS and GF. Data was extracted using the data collection form for intervention reviews (randomised control trials and non-randomised control trials) from the Cochrane group.[23] The characteristics of each included article consisted of

title, study ID, publication type, study design, aim of the study, ethical information, number of participants, population characteristics (age, BMI, % female, ethnicity, population description, method of recruitment, inclusion/exclusion criteria), description of EBTs, description of outcomes (method, definition, unit of measurement and validation information for the collection and measurement of BMI and EI), study funding sources, possible conflicts of interests, data and analysis (results, including number of participants, means and standard deviations where possible, unit of analysis, statistical methods used and appropriateness of these), key conclusions of study authors and references to other available studies. Where means and standard deviations were not reported, correlations, F-values or t-values were extracted, and standard errors were converted to standard deviations. Effects on EI and BMI were examined using a random-effects meta-analysis because it was anticipated that there would be considerable between-study heterogeneity. All analyses were carried out in the R environment, version 2021.09.0.[24] The following R packages were used: dmetar,[25] esc,[26] tidyverse,[27] and meta.[28]

Meta-analysis of the effect of each EBT on BMI and EI was performed when ≥2 effect sizes were available for each trait. For each analysis, effect sizes were pooled using the r value from correlations and were transformed to Fisher's Z.[29] Where correlations were not present, the available raw data were transformed into Fisher's Z. Where one study provided data for more than one questionnaire (e.g., all participants completed the DEBQ and TFEQ), the sample size was halved to prevent 'double counting,' which can artificially inflate effect sizes and distort results. [30,31] If studies provided data for multiple types of EI outcome e.g., sweet, and savoury EI, or provided data for multiple preloads, the selected outcomes were pooled to provide a single 'total EI' correlation, to prevent overpowering of a single study. Effect sizes based on Fisher's Z were interpreted as very small (≤0.05), small (>0.05-<0.10), moderate (>0.10-<0.30), large (>0.30-<0.40), and very large (≥0.40).[32] The restricted maximum likelihood estimator was used to calculate heterogeneity variance and heterogeneity was also evaluated using the I-squared statistic (I^2), with values inferred as low (<25%), moderate (25%–75%), and high (>75%). However, the I^2 statistic was interpreted cautiously when the number of studies in each meta-analysis was <7. In this case, it is advised that confidence intervals should supplement the I^2 statistic, therefore, confidence intervals were also provided for

each meta-analysis.[33] Knapp-Hartung adjustments[34] were also used to calculate the confidence interval around the pooled effect. The Knapp-Hartung adjustment can reduce the chance of false positives, especially when the number of studies is small.[35] The results of the meta-analyses are presented as forest plots, which include the authors, standard error (SE), questionnaire type, correlation (COR), 95% confidence intervals, weight and Hedges G[36] that is converted from Fisher's Z.

Further analysis of outliers and influential cases was conducted using a leave-one-out analysis in addition to identifying influential cases, and results are reported for both the main meta-analyses and further analyses where outliers or influential cases were removed. Publication bias was assessed by visually inspecting the funnel plot and Egger's regression test when the number of included studies was <10. Duval and Tweedie's trim-and-fill method was also used. Two subgroup analyses were conducted to test whether the effect of restraint on BMI and EI was influenced by the type of questionnaire used because the questionnaires used to measure restraint varied across studies. Additionally, a subgroup analysis tested whether the use of a preload influenced the correlation between restraint and EI. Studies were split into 'no preload' and 'preload' categories. For the preload studies, effect sizes were conducted for both no preload and preload groups, and sample size was halved to prevent double counting. [30,31] Where studies included multiple preload conditions, the largest preload condition was used to calculate the effect size. Subgroup analyses were also conducted to test the influence of meal type (1 test meal vs multiple test meals) where there were ≥10 effect sizes available for each EBT and ≥2 per category.

3.3.4. Quality assessment

To assess risk of bias, the excel version of the mixed method appraisal tool (MMAT) for appraising studies was used.[37] It is a critical appraisal tool designed for systematic mixed studies reviews (including qualitative, quantitative, and mixed methods studies). The MMAT has been pilot tested and has high content validity.[38] The following questions were answered to determine the risk of bias: is the sampling strategy relevant to address the research question, is the sample representative of the target population, are the measurements appropriate, is the risk of nonresponse

bias low and is the statistical analysis appropriate to answer the research question? The four assessment items represented flaws if answered “No/can’t tell.” It is discouraged to calculate an overall score from the ratings of each criterion; therefore, a detailed discussion of the ratings follows. Quality assessment was completed by the main reviewer (CD) and was cross-checked by JS and GF.

3.4. Results

2760 articles were retrieved from the four databases. After deduplication, 1955 articles remained for title and abstract screening. The reasons for excluding any studies that did not meet the inclusion criteria during full text screening are shown in the flow diagram in figure 3.2. Seventy-seven papers met the inclusion criteria and were included in this systematic review, with some papers consisting of several studies. In total, 83 studies were included. Several studies could not be synthesised for each EBT meta-analysis, therefore the number of studies included in each meta-analysis varies. Where meta-analysis was not possible, associations between the EBTs and outcome variables are qualitatively summarised. Table 3.1 gives the included questionnaires used to measure EBTs and their abbreviations.

Figure 3.2. Flow diagram of study selection

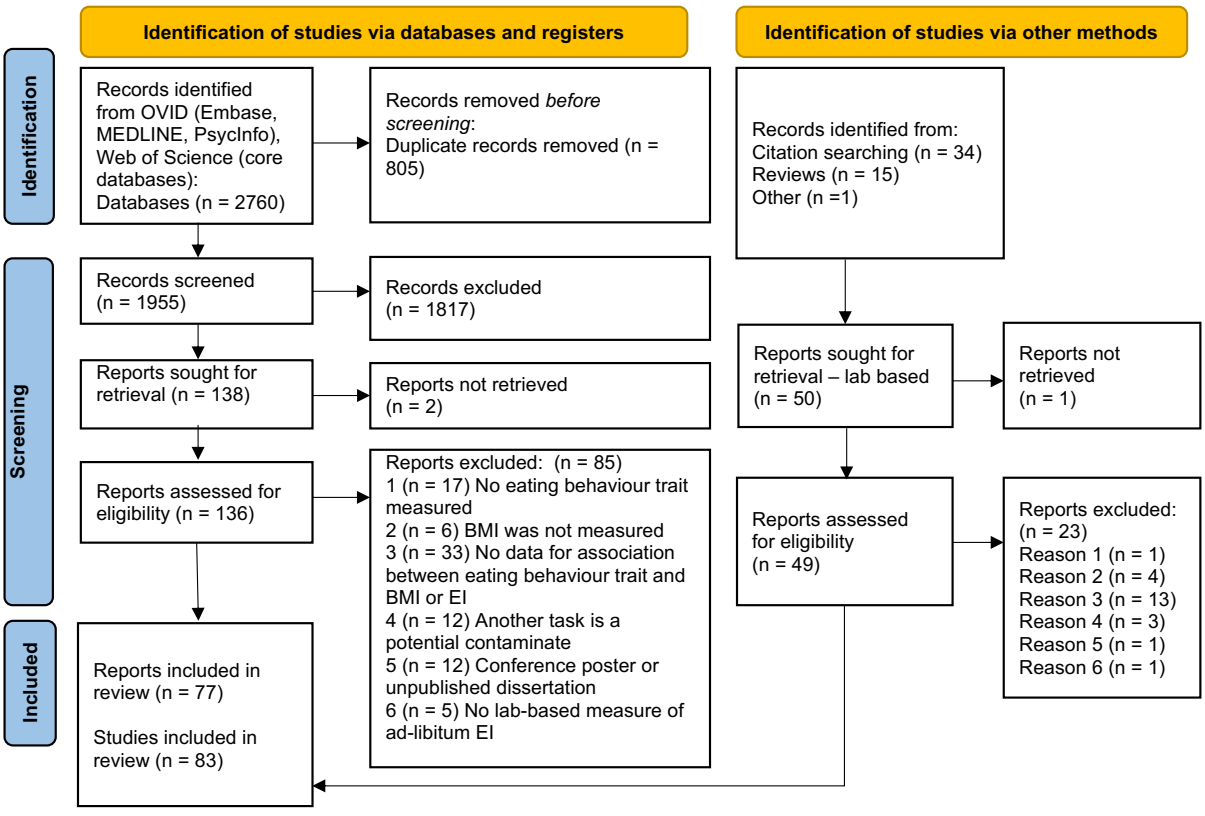


Table 3.1. Table of Questionnaires included and their abbreviations

Questionnaire	Abbreviation	Subscales	Reference Paper
Intuitive Eating Scale	IES	Reliance On Hunger and Satiety (RHSC), Unlimited Permission to Eat (UPE), Eating for Physical Rather Than Emotional Reasons (EPR)	Tylka [39]
Intuitive Eating Scale-2	IES-2	RHSC, UPE, EPR, Body-Food Choice Congruence (B-FCC)	Tylka and Kroon Van Diest [40]
Mindful Eating Questionnaire	MEQ	Disinhibition, Awareness, External Cues, Emotional Response, Distraction	Framson, Kristal, Schenk, Littman, Zeliadt and Benitez [41]
Satiety Quotient	SQ	Desire To Eat, Hunger, Fullness, Satiety, Prospective Food Consumption (PFC)	Green, Delargy, Joanes and Blundell [42]
Adult Eating Behaviour Questionnaire	AEBQ	Hunger (H), Food Responsiveness (FR), Emotional Over-Eating (EOE), Enjoyment of Food (EF), Satiety Responsiveness (SR), Emotional Under-Eating (EUE), Food Fussiness (FF), Slowness in Eating (SE)	Hunot, Fildes, Croker, Llewellyn, Wardle and Beeken [43]
Three Factor Eating Questionnaire	TFEQ	Disinhibition, Dietary Restraint, Susceptibility to Hunger	Stunkard and Messick [5]
Restraint Scale	RS		Herman and Mack [44]

Revised Restraint Scale	RRS		Herman, Polivy and Silver [45]
Dutch Eating Behaviour Questionnaire	DEBQ	Restraint Eating, Emotional Eating, External Eating	van Strien, Frijters, Bergers and Defares [46]
Control of Eating Questionnaire	COEQ	Craving Control, Positive Mood, Craving for Sweet, Craving for Savoury	Dalton, Finlayson, Blundell and Hill [47]
Power of Food Scale	PFS	Food Available, Food Present, Food Tasted	Lowe, Butryn, Didie, Annunziato, Thomas, Crerand, Ochner, Coletta, Bellace and Wallaert [48]
Food Craving Inventory	FCI	High fats, Sweets, Carbohydrates, Fast-Food Fats and Subjective, Behavioural	White, Whisenhunt, Williamson, Greenway and Netemeyer [49]
Binge Eating Scale	BES		Gormally, Black, Daston and Rardin [21]
Eating Disorders Examination Questionnaire	EDE-Q	Restraint, Eating Concern, Shape Concern, Weight Concern	Fairburn, Cooper and O'Connor [50]
Eating Disorders Assessment Scale	EDAS	Restrained Eating, Binge Eating, Purging, Preoccupation with Body Image, and Body Weight.	Akkermann, Herik, Aluoja and Järv [51]
Eating Disorders Diagnostics Scale	EDDS	Anorexia Nervosa, Bulimia Nervosa, Binge Eating Disorder, Noneating Disordered	Stice, Telch and Rizvi [52]
Eating Disorders Inventory	EDI-2	EBT related subscales: Drive for Thinness, Bulimia, Body Dissatisfaction	Garner [53]
Eating Self-Efficacy	WEL	Negative Emotions, Availability, Social Pressure, Physical Discomfort, Positive Activities	Clark, Abrams, Niaura, Eaton and Rossi [54]

Reward-based Eating Drive Questionnaire	RED-9, RED-13, RED-X5	Loss of Control Over Eating, Lack of Satiety, Preoccupation with Food	Vainik, Eun Han, Epel, Janet Tomiyama, Dagher and Mason [55]
Yale Food Addiction Scale-2	YFAS-2		Gearhardt, Corbin and Brownell [56]

3.4.1. Study characteristics

Study characteristics are summarised in Table 3.2 (see Table A.2 for more detailed characteristics). The studies were published between 1975 and 2021. All data from the studies are cross-sectional. The following EBTs were identified, restraint (n = 50), disinhibition (n = 17), emotional eating (n = 12), binge eating (n = 8), external eating (n = 7), susceptibility to hunger (n = 6), hedonic hunger (n = 4), satiety responsiveness (n = 3 via Satiety Quotient, SQ) and n = 1 via AEBQ), intuitive eating (n = 3), food craving (n = 1), mindful eating (n = 1), eating disorders (n = 1), food addiction (n = 1), eating self-efficacy (n = 1), control of eating (n= 1), reward-related eating (n = 1). The mean (range) of the total sample size was 70 (18-273). The mean (range) age of participants was 27 years (19 – 59 years); BMI was 25.20 kg/m^2 (21.0 – 36.8 kg/m^2). Males and females were included in 25 studies, females only in 56 studies, males only in 1 study and no data was given for 1 study. BMI was objectively measured in 52 studies, self-reported in 10 studies, and in 21 studies, there was no information for how BMI was measured. Measures of EI varied widely across studies. EI was measured over one test meal in 69 studies and across multiple test meals in 14 studies. Studies also used various types of food to measure EI. Overall, snack foods were the most popular EI measure (n = 29). Multiple food items (n = 21), pasta (n = 8), cookies (n = 5), ice cream (n = 5), popcorn (n = 3), sandwiches (n = 3), vending machines (n = 2), beef casserole (n = 2), risotto (n = 1), pizza (n = 1), milkshake (n = 1), Boost (a nutritionally complete liquid supplement, n = 1) and breakfast foods (n = 1) were also used.

Table 3.2. Eating behaviour traits: summary of included studies

Eating behaviour trait	Questionnaire used to assess eating behaviour trait	Cross-sectional study reference	Associations with	
			EI	**BMI**
Intuitive eating (IE)	IES	Anderson, Schaumberg, Anderson and Reilly [57]	NS and ↑	↓
	IES	Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58]	NS	NS
	IES-2	Ruzanska and Warschburger [59]	NS and ↑	↓
		Total	3 NS 2 ↑	1 NS 2 ↓
Mindful eating	MEQ	Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58]	NS	NS
		Total	1 NS	1 NS
Satiety Responsiveness	SQ	Dalton, Hollingworth, Blundell and Finlayson [60]	↓	NS
	SQ desire to eat	Drapeau, Blundell, Therrien, Lawton, Richard and Tremblay [61]	NS	NS
	SQ for PFC		NS	NS
	SQ for fullness		↓	NS
	SQ for hunger		NS	NS
	SQ	Drapeau, Jacob, Panahi and Tremblay [62]	NS	NS
	AEBQ	Zuraikat, Roe, Smethers, Reihart and Rolls [63]	↓	NS*
		Total	4 NS 3 ↓	7 NS
Dietary restraint	TFEQ	Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58]	NS	↑
	TFEQ	Ard, Desmond, Allison and Conway [64]	↓	
	TFEQ	Bellisle, Dalix, Airinei, Hercberg and Péneau [65]	NS	NS
	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King [66]	NS	
	TFEQ	Chambers and Yeomans [67]	NS	NS
	RS	Coelho, Polivy, Herman and Pliner [68]	↑	↑

RRS	Cools, Schotte and McNally [69]	↓	
RS	de Witt Huberts, Evers and de Ridder [70] study 1	NS	↑
RS	de Witt Huberts, Evers and de Ridder [70] study 2	NS	↑
RS	de Witt Huberts, Evers and de Ridder [70] study 3	NS	NS
DEBQ	Dweck, Jenkins and Nolan [71]		↑
RRS	Fedoroff, Polivy and Herman [72]	↓	
TFEQ	Finlayson, Blundell,	NS	
DEBQ	Bordes, Griffioen-Roose and de Graaf [73] Finlayson et a. (2012)	NS and ↑	NS
RS	Guerrieri, Nederkoorn, Schrooten, Martijn and Jansen [74]	↑	↑
TFEQ	Haynes, Lee and Yeomans [75]	↓	NS
DEBQ	Herhaus and Petrowski [76]	NS	NS
RS	Herman and Mack [44]	↑ and ↓	NS
RS	Herman, Polivy and Silver [45]	↑	
DEBQ	Higgs, Williamson and Attwood [77]	NS	NS
TFEQ	Hofmann and Friese [78]	↓	
DEBQ	Hopkins et al. (2021)	↓	NS
DEBQ	Jansen [79] study 1	↑	NS
	Jansen [79] study 2	↑	↑
RS	Jansen, Merckelbach, Oosterlaan, Tuiten and Van Den Hout [80]	NS	↑
RS	Lattimore and Maxwell [81]	NS	
TFEQ	Martin, Williamson, Geiselman, Walden, Smeets, Morales and Redmann Jr [82]	NS	NS
TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet [83]	NS	
RRS	Myhre, Buchwald, Kratz, Goldberg, Polivy, Melhorn, Schur and Cummings [84]	NS	NS
DEBQ	Oliver, Wardle and Gibson [85]	NS	

DEBQ	Ouwens, van Strien and van der Staak [86]	NS	
TFEQ	Ouwens, van Strien and van der Staak [86]	NS	
RS	Ouwens, van Strien and van der Staak [86]	NS	
TFEQ	Peluso [87]	NS	NS
TFEQ	Rideout, McLean and Barr [88]	↓	NS
DEBQ	Robinson and Haynes (2021)	NS	NS
TFEQ	Rolls, Castellanos, Shide, Miller, Pelkman, Thorwart and Peters [89]	NS	NS
TFEQ	Ruddock, Field and Hardman [90]	↓	NS
TFEQ	Schoch and Raynor [91]	↓	↑
RRS	Schotte, Cools and McNally [92]	NS	↑
RS	Shapiro and Anderson [93]	NS	
DEBQ	Sim, Lee and Cheon [94]	NS	
TFEQ	Smith, Geiselman, Williamson, Champagne, Bray and Ryan [95]	NS	↑
RS and TFEQ	Stice, Fisher and Lowe [96]	NS	NS
TFEQ and EDEQ-R	study 1 Stice, Fisher and Lowe [96] study 2	NS	
TFEQ	Stice, Sysko, Roberto and Allison [97]	NS	
TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez [98]	↓	NS
DEBQ	van Strien and Ouwens [99]	NS	
TFEQ	Visona and George [100]	NS	NS
DEBQ	Wallis and Hetherington [101]	NS	NS
DEBQ	Wallis and Hetherington [102]	↓	↑
DEBQ	Wardle and Beales [103]	↑	NS
TFEQ	Westenhoefer, Broeckmann, Münch and Pudel [104]	NS	↑
RRS	Westenhoefer, Broeckmann, Münch and Pudel [104]	↑	↑
TFEQ	Yeomans and Coughlan [105]	NS	NS
TFEQ	Yeomans, Tovey, Tinley and Haynes [106]		NS

	TFEQ EDE-2	Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass [107] Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass [107]	↓ NS	
		Total	36 NS 9 ↑ 13 ↓	24 NS 14 ↑
Control of eating	COEQ Craving control Positive mood Lack of Craving for sweet[(a)]	Dalton, Finlayson, Blundell and Hill [47]	 ↓ ↓ ↓	 ↓ ↓ ↓
		Total	3 ↓	3 ↓
Hedonic hunger/food reward sensitivity	PFS	Appelhans, Liebman, Woolf, Pagoto, Schneider and Whited [108]	NS	NS
	PFS	Ely, Howard and Lowe [109]	NS and ↑	↑
	PFS	Finlayson et al. (2012)	NS	NS
	PFS	Nolan-Poupart, Veldhuizen, Geha and Small [110]	NS	
		Total	4 NS 1 ↑	2 NS 1 ↑
Food craving	FCI	Martin, O'Neil, Tollefson, Greenway and White [111]	↑	
		Total	1 ↑	
Dietary disinhibition	TFEQ	Ard, Desmond, Allison and Conway [64]	NS	
	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King [66]	NS	
	TFEQ	Chambers and Yeomans [67]	NS and ↑	
	TFEQ	Epstein, Lin, Carr and Fletcher [112]	↑	↑
	TFEQ	Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf [73]	NS and ↑	↑

	TFEQ	Haynes, Lee and Yeomans [75]	↑	NS
	TFEQ	Higgs, Williamson and Attwood [77]	NS	NS
	TFEQ	Martin, Williamson, Geiselman, Walden, Smeets, Morales and Redmann Jr [82]		NS
	TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet [83]	NS	
	DEBQ	Ouwens, van Strien and	↑	
	TFEQ	van der Staak [86]	↑	
		Ouwens, van Strien and van der Staak [86]		
	TFEQ	Ruddock, Field and Hardman [90]	↑	NS
	TFEQ	Smith, Geiselman, Williamson, Champagne, Bray and Ryan [95]	↑	↑
	TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez [98]	NS	↑
	TFEQ	Westenhoefer, Broeckmann, Münch and Pudel [104]	↑	↑
	TFEQ	Yeomans and Coughlan [105]	NS	↑
	TFEQ	Yeomans, Tovey, Tinley	↑	↑
	TFEQ	and Haynes [106]	NS	
		Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass [107]		
		Total	9 NS 10 ↑	4 NS 7 ↑
Susceptibility to Hunger	TFEQ	Bryant, Caudwell, Blundell, Hopkins and King [66]	↑	
	TFEQ	Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf [73]	NS	NS
	TFEQ	McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet [83]	↑	

	TFEQ	Stinson, Votruba, Venti, Krakoff, Gluck and Perez [98]	NS	↑
	TFEQ	Westenhoefer,	↑	↑
	TFEQ	Broeckmann, Münch and Pudel [104]	NS	
		Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass [107]		
		Total	3 NS 3 ↑	1 NS 2 ↑
External eating	DEBQ	Dweck, Jenkins and Nolan [71]		NS
	DEBQ	Finlayson et al. (2012)	↑	NS
	DEBQ	Hopkins, Michalowska, Whybrow, Horgan and Stubbs [113]	NS	NS
	DEBQ	Kakoschke, Kemps and Tiggemann [114]	↑	
	DEBQ	Robinson and Haynes [115]	NS	NS
	DEBQ	van Strien, Donker and Ouwens [116]	↑	
	DEBQ	van Strien and Ouwens [99]	NS	
		Total	3 NS 3 ↑	4 NS
Emotional eating	DEBQ	Dweck, Jenkins and Nolan [71]	NS	NS
	DEBQ	Evers, de Ridder and Adriaanse [117] study 3	NS	NS
	DEBQ	Evers, de Ridder and Adriaanse [117] study 4	NS	NS
	DEBQ	Evers, de Ridder and	NS	NS
	DEBQ	Adriaanse [117] study 5	NS and ↑	↑
	DEBQ	Finlayson et al. (2012) Hopkins, Michalowska, Whybrow, Horgan and Stubbs [113]	NS	NS
	DEBQ	Oliver, Wardle and Gibson [85]	NS	
	DEBQ	Raspopow, Abizaid, Matheson and Anisman [118]	NS	NS*
	DEBQ	Robinson and Haynes [115]	NS	NS
	DEBQ	van Strien, Donker and Ouwens [116]	↑	

	DEBQ	van Strien, Herman, Anschutz, Engels and de Weerth [119]		↑
	DEBQ	van Strien and Ouwens [99]	↑	
	DEBQ	Wallis and Hetherington [101]	NS	NS
	DEBQ	Wallis and Hetherington [102]		NS
		Total	10 NS 3 ↑	9 NS 2 ↑
Binge eating	BES	Alger, Seagle and Ravussin [120]		
	EDAS	Arumae, Kreegipuu and Vainik [121]	↑	NS
	BES	Dalton, Blundell and Finlayson [122]	↑	NS*
	BES	Dalton, Blundell and Finlayson [123]	↑	NS*
	BES	Finlayson, Arlotti, Dalton,	↑	↑
	BES	King and Blundell [4] Finlayson et al. (2012)	NS	↑
	BES	Nasser, Gluck and Geliebter [124]	NS	NS
	BES	Stinson, Votruba, Venti, Krakoff, Gluck and Perez [98]	NS	↑
		Total	3 NS 4 ↑	4 NS 3 ↑
Eating disorders	EDDS	Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58]	NS	↑
	EDI-2	Long, Meyer, Leung and Wallis [125]	NS	
		Total	2 NS	1 ↑
Food addiction	YFAS 2.0	Schulte, Sonneville and Gearhardt [126]	NS	NS
		Total	1 NS	1 NS
Eating self-efficacy	WEL	Vijayvargiya, Chedid, Wang, Atieh, Maselli, Burton, Clark, Acosta and Camilleri [127]	↓	
		Total	1 ↓	

Reward-related eating	RED-X5 RED-9 RED-13	Vainik, Eun Han, Epel, Janet Tomiyama, Dagher and Mason [55] **Total**	↑ 1 ↑	↑ 1 ↑
Total scores	TFEQ DEBQ TFEQ	Bellisle and Dalix [128] Bellisle and Dalix [128] Nolan-Poupart, Veldhuizen, Geha and Small [110] **Total**	NS NS NS 3 NS	NS NS 2 NS

Note: blank = outcome was measured but the data was not available as authors were contacted but did not reply, NS = non-significant, NS* = significant association with body fat or body weight, ↑ = significance positive association, ↓ = significant negative association, [(a)] = reverse coded. If two results are presented for one outcome (e.g., NS and ↑), the effect differed depending on the group or condition tested.

3.4.2. Study quality

Overall, studies scored relatively high on study quality for four out of five quality criteria. Studies scored low for sampling strategies because none used probability sampling and most recruited via opportunity sampling. Studies did define their target population by presenting inclusion and exclusion criteria, and some included a-priori sample size calculations. Respondents and the target population were well matched and the reasons why certain individuals were excluded were made clear. All questionnaires have been validated and in 51 studies, BMI was measured objectively in the laboratory. The measurements of EI were, for most studies, not previously validated and the most frequently used type of food was snacks (n = 28). The extent to which findings of eating behaviour, using energy dense snacks can generalise to wider and more typical eating behaviour is unknown. The majority of studies also attempted to reduce possible confounders by providing participants with a standardised breakfast, testing during normal lunch hours and keeping testing times between participants consistent.

The risk of non-response bias in studies included in this systematic review was low, no studies reported high drop-out rates. Lastly, study quality was high for the appropriateness of statistical analysis used in studies. However, in some studies, the method used to split participants into groups was a limitation. Using a median split to identify low or high scorers could have misclassified individuals and lead to null

findings.[11] Indeed, van Strien, Herman, Anschutz, Engels and de Weerth [119] found that if a median split was used, their results would have become non-significant. Additionally, in Dalton, Hollingworth, Blundell and Finlayson [60], 40% of the participants were unclassified based on their SQ score. Therefore, any findings cannot be generalised to a wider population if almost half of the sample could not be classified into a level of satiety responsiveness.

Study Findings

3.4.3. Intuitive eating

A meta-analysis of 2 studies (n = 262) found no significant correlation between intuitive eating and EI (*r* = 0.32 [-0.30, 2.00], p = 0.096). A meta-analysis of 3 studies (n = 317) found that intuitive eating was negatively correlated with BMI (*r* = -0.26 [-0.45, -0.04], p = 0.036, Figure A.3) and this represents a moderate effect size. Heterogeneity was low (I^2 = 0.0%, Q = 1.66, p = 0.436). One study was identified as an influential case.[57] With this study removed from the meta-analysis, the overall effect size increased, however, the effect became non-significant (*r* = -0.30 [-0.86, 0.58], p = 0.156). The trim-and-fill method suggested no missing studies.

3.4.4. Mindful eating

Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58] found no significant association between mindful eating and EI (r = 0.15) or mindful eating and BMI (r = 0.01)

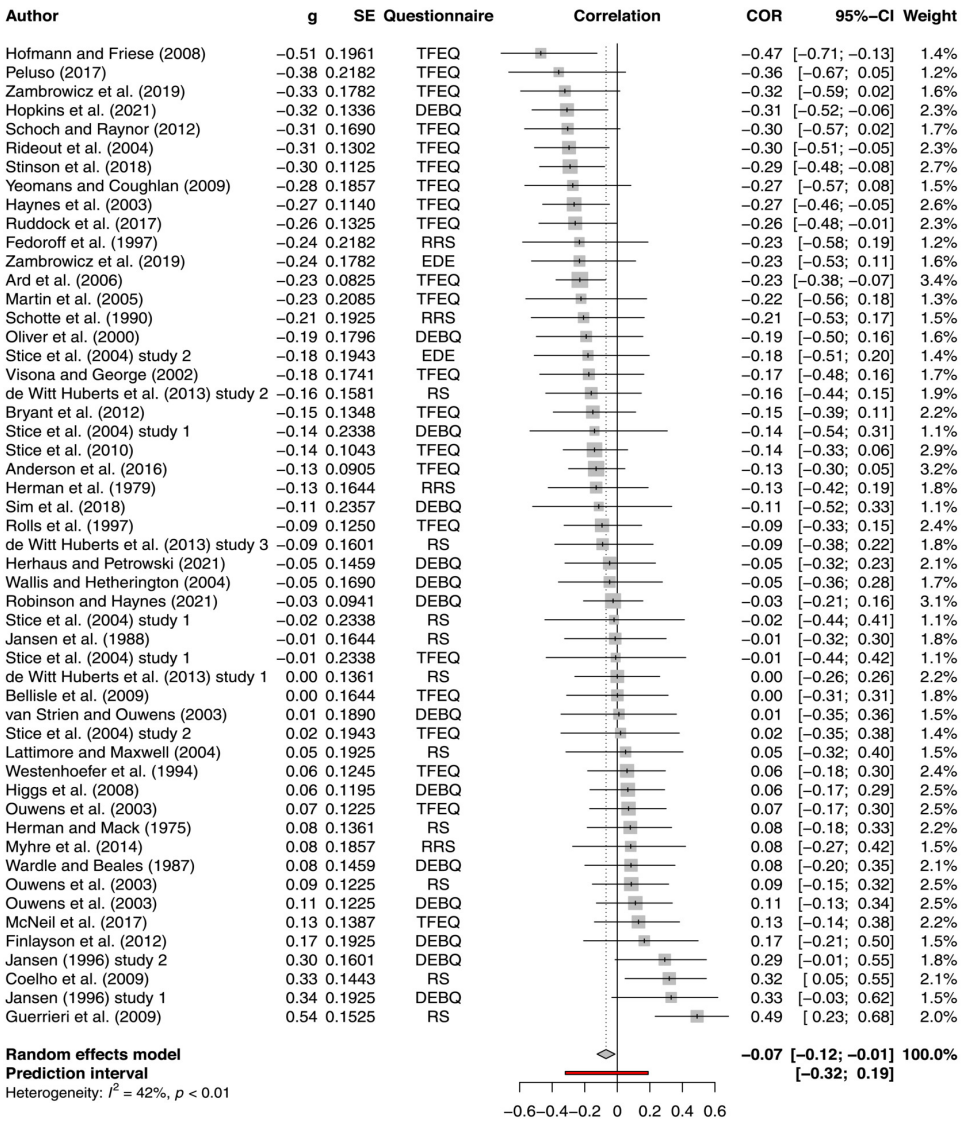
3.4.5. Satiety Responsiveness

Only one effect size was available; therefore, meta-analysis could not be conducted. However, of the four studies that measured satiety responsiveness, three reported that higher satiety responsiveness as associated with reduced EI[60,62,63] and one reported no significant effect.[62] A meta-analysis of 3 studies (n = 197) found a moderate negative correlation between satiety responsiveness and BMI (*r* = -0.18 [-0.31, -0.04], p = 0.031, Figure A.4). Heterogeneity was low (I^2 = 0.0%, Q = 0.41, p = 0.815). No studies were identified as outliers or influential cases. Two missing studies were suggested to the right (adjusted *r* = -0.15 [-0.24, -0.06], p = 0.009).

3.4.6. Restraint

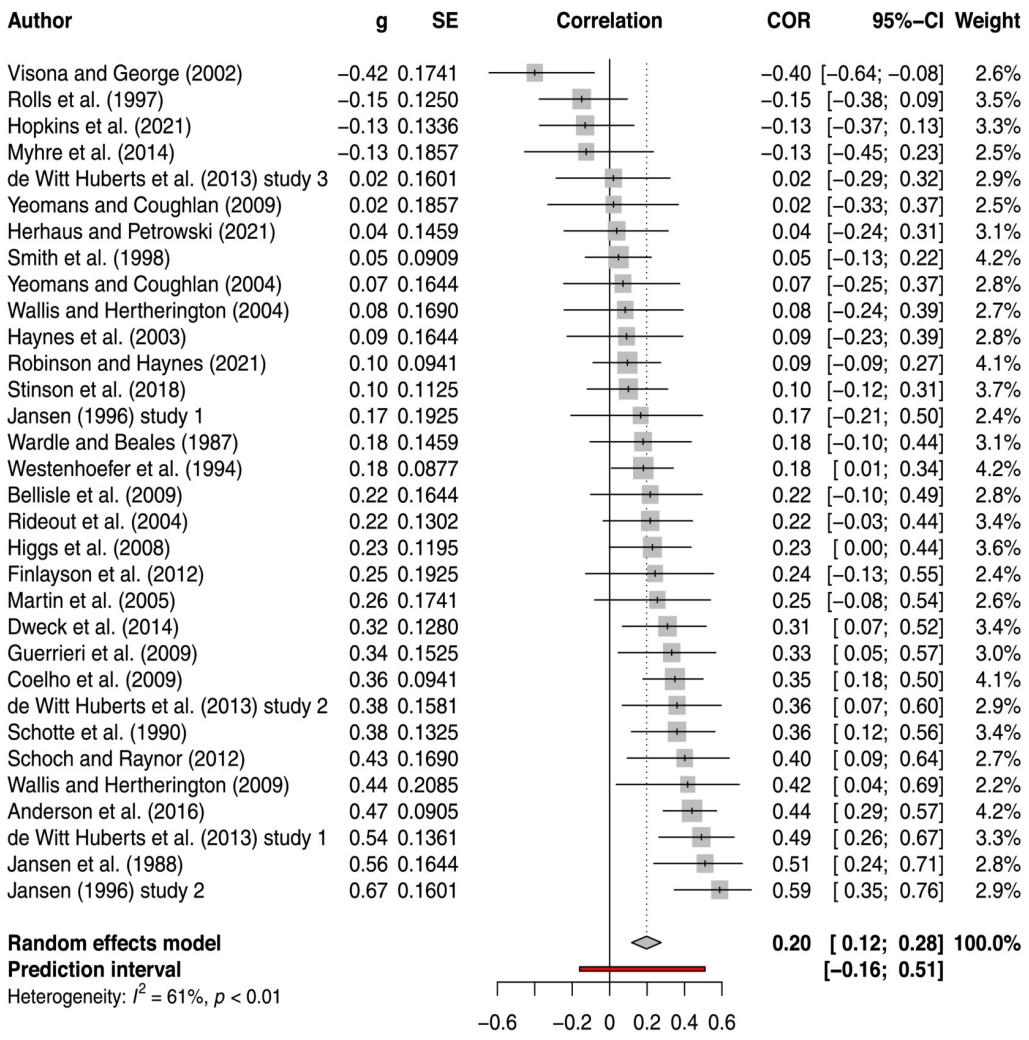
Meta-analysis of 49 studies (n = 2594) showed a small but significant, negative correlation between restraint and EI (52 study arms, r = -0.07, [-0.12, -0.01], p = 0.015, figure 3.3). Heterogeneity was moderate (I^2 = 41.8%, Q = 87.70, p = 0.001). Four outliers were identified[68,74,78,79] and the one-study-removed procedure identified one influential case.[74] With these outliers removed the effect size slightly increased (r = -0.09 [-0.14, -0.05], p < 0.001). Heterogeneity was low (I^2 = 10.2%) and the Q test was non-significant (Q = 52.32, p = 0.275). The funnel plot did not suggest any evidence of publication bias (Figure A.5), no missing studies were suggested, and Egger's test was non-significant (p = 0.680). The association between restraint and EI was influenced by the type of questionnaire used to measure restraint (p < 0.001, Table A.6). For the TFEQ, RRS and EDE-Q, restraint was negatively correlated with EI. Whereas, for the RS and DEBQ, restraint was positively correlated with EI. A random-effects meta-regression was undertaken to test for differences in effect sizes between questionnaires used to measure restraint and EI. The meta-regression was significant (p < 0.05), however TFEQ was the only variable that was significantly associated with EI (p < 0.01). Further subgroup analyses revealed that the effect of restraint on EI was not significantly influenced by the number of test meals (one vs multiple test meals, p = 0.103) or BMI group (healthy, overweight, obese, p = 0.553). Lastly, the association between restraint and EI was influenced by a preload. Consuming a preload was associated with increased EI, while not consuming a preload was associated with decreased EI (p = 0.040, see Table A.7).

Figure 3.3. Forest plot of the effect of restraint on EI



Meta-analysis of 32 studies (n = 1869) showed that restraint was positively correlated with BMI, and this effect was moderate (r = 0.20 [0.12, 0.28], p < 0.001, figure 3.4). Heterogeneity was moderate (I^2 = 61.2%, Q = 79.86, p < 0.001). Four outliers were identified.[58,79,89,100] Without these studies included, there was no change in the overall effect size (r = 0.20 [0.13, 0.26], p < 0.001). However, heterogeneity decreased (I^2 = 34.8%, Q = 41.38, p = 0.038). Using the leave-one-out method, no studies were identified as influential. The funnel plot (Figure A.8) suggested little evidence of publication bias, no missing studies were identified, and Egger's test was non-significant (p = 0.753). The effect of restraint on BMI did not depend on the type of questionnaire used to measure restraint (p = 0.103).

Figure 3.4. Forest plot of the effect of restraint on BMI



3.4.7. Control of eating

Dalton, Finlayson, Blundell and Hill [47] found that craving control and positive mood were negatively associated with EI (r = -0.20, p < 0.05, r = -0.21, p <0.05) and BMI (r = -0.31, p < 0.001, r = -0.23, p < 0.01) respectively, whereas craving for sweet foods was positively associated with EI (r = 0.40, p < 0.001) and BMI (r = 0.23, p < 0.01).

3.4.8. Hedonic hunger

A meta-analysis of 4 studies (n = 183) found no significant correlation between hedonic hunger and EI (*r* = 0.13 [-0.08, 0.33], p = 0.147). Additionally, a meta-analysis of 2 studies (n = 92) also found a non-significant association between hedonic hunger and BMI (*r* = 0.05 [-1.00, 1.00], p = 0.868)

3.4.9. Food craving

Martin et al. (2008) reported that scores on the FCI were associated with increased total EI (r = 0.22, p < 0.05), however there was no data for the effect of FCI on BMI.

3.4.10. Susceptibility to hunger

A meta-analysis of 6 studies (n = 362) found susceptibility to hunger was positively associated with EI, and this effect was moderate-to-large (*r* = 0.27 [0.19, 0.35], p < 0.001, see Figure A.9). Heterogeneity was low (I^2 = 0.0%, Q = 2.05, p = 0.843). No outliers or influential cases were identified. Three missing studies were identified to the left (adjusted *r* = 0.22 [0.13, 0.31], p < 0.001). A meta-analysis of 3 studies (n = 245) found a moderate positive correlation between susceptibility to hunger and BMI (*r* = 0.20 [0.11, 0.28], p = 0.011; Figure A.10). Heterogeneity was small (I^2 = 0.0%, Q = 0.20, p = 0.903). No outliers or influential cases were identified. Two missing studies were identified to the left (adjusted *r* = 0.17 [0.11, 0.23], p = 0.002).

3.4.11. External eating

A meta-analysis of 6 studies (n = 583) found that external eating was positively correlated with EI, and this effect was moderate (*r* = 0.17 [0.07, 0.27], p = 0.007, see Figure A.11). Heterogeneity was low (I^2 = 0.0%, Q = 4.46, p = 0.485). Two influential studies were identified.[115,116] With these studies removed, the effect size slightly increased (*r* = 0.18 [0.01, 0.34], p = 0.044). Heterogeneity was low (I^2 = 0.0%, Q = 2.21, p = 0.531). One missing study was identified to the left (adjusted *r* = 0.16 [0.06, 0.26], p = 0.008). A meta-analysis of 4 studies (n = 269) found no significant correlation between external eating and BMI (r = -0.08 [-0.28, 0.12], p = 0.285).

3.4.12. Emotional eating

A meta-analysis of 10 studies (n = 608) found a small-to-moderate, positive correlation between emotional eating and EI (*r* = 0.12 [0.00, 0.24], p = 0.042, see Figure A.12). Heterogeneity was moderate (I^2 = 32.1%, Q = 13.26, p = 0.151). No influential studies were identified. The funnel plot did not suggest any publication bias (Figure A.13), one missing study was identified to the left (adjusted *r* = 0.11 [-0.02, 0.23], p = 0.088) and the Egger's regression test was non-significant (p = 0.800). The effect of emotional eating on EI was not influenced by the number of test

meals (p = 0.342). A meta-analysis of 10 studies (n = 737) found that emotional eating was positively correlated with BMI (*r* = 0.19 [0.13, 0.25] p < 0.001). This effect was moderate (Figure A.14). Heterogeneity was low (I^2 = 0.0%, Q = 5.17, p = 0.819). No influential cases were identified. The funnel plot did not suggest any publication bias (Figure A.15), No missing studies were suggested, and the Egger's test was non-significant (p = 0.860).

3.4.13. Disinhibition

A meta-analysis of 14 studies (n = 1172) found a moderate positive correlation between disinhibition and EI (*r* = 0.19 [0.14, 0.24], p < 0.001, see Figure A.16). Heterogeneity was low (I^2 = 0.0%, Q = 9.74, p = 0.715). Influence diagnostics identified one influential case.[112] With this study removed, the overall effect size increased (*r* = 0.21 [0.14, 0.27], p < 0.001). Heterogeneity was low (I^2 = 0.0%, Q = 8.88, p = 0.713). The funnel plot showed little evidence of publication bias (Figure A.17), one missing study was suggested to the right (adjusted *r* = 0.20 [0.14, 0.25], p < 0.001), and Egger's test was non-significant (p = 0.973). The effect of disinhibition on EI was not influenced by the number of test meals (p = 0.341). Meta-analysis of 8 studies (n = 618) showed a moderate-to-large, positive correlation between disinhibition and BMI (*r* = 0.28 [0.19, 0.38], p < 0.001, Figure A.18). Heterogeneity was low (I^2 = 13.8%, Q = 8.12, p = 0.322). One influential case was identified.[95] Without this study included, there was a small decrease in the overall effect size (*r* = 0.25 [0.16, 0.33], p < 0.001). Heterogeneity was low (I^2 = 0.0%, Q = 3.68, p = 0.720). No missing studies were suggested.

3.4.14. Binge eating

A meta-analysis of 7 studies (n = 249) found a moderate, positive correlation between binge eating and EI (*r* = 0.26 [0.08, 0.43], p < 0.001, see Figure A.19). Heterogeneity was low (I^2 = 0.0%, Q = 3.68, p = 0.720). Two influential studies were identified.[98,123] With these studies removed, the overall effect size increased slightly (r = 0.28 [0.11, 0.43], p = 0.010). Heterogeneity was low (I^2 = 0.0%, Q = 2.02, p = 0.731). Two missing studies were suggested to the left (adjusted *r* = 0.17 [-0.04, 0.37], p = 0.098). A meta-analysis of 7 studies (n = 277) found a moderate positive correlation between binge eating and BMI (*r* = 0.21 [0.07, 0.34], p = 0.011, see

Figure A.20). Heterogeneity was low (I^2 = 0.0%, Q = 5.30, p = 0.506). No influential studies were identified. One missing study was identified to the right (adjusted *r* = 0.23 [0.09, 0.36], p = 0.006).

3.4.15. Disordered eating

A meta-analysis of 2 studies (n = 152) was conducted to test the correlation between eating disorder symptomology and EI. The analysis was non-significant (r = -0.17 [-0.98, 0.96], p = 0.499). Only one effect size was available to test the effect of eating disorders on BMI, therefore, a meta-analysis could not be conducted. However, Anderson, Reilly, Schaumberg, Dmochowski and Anderson [58] did report that increased scores on the EDDS were associated with increased BMI (r = 0.40, p < 0.05).The YFAS 2.0 was not significantly associated with EI or BMI.[126]

3.4.16. Other EBTs

Eating self-efficacy was negatively associated with EI (r = -0.26, p < 0.05).[127] Three measures of reward-related eating were positively correlated with EI (RED-X5: r = 0.31, p<0.05, RED-9: r = 0.35, p<0.05, RED-13: r = 0.32, p<0.05) and BMI (RED-X5: r = 0.18, p<0.05, RED-9: 0.17, p<0.06, RED-13: r = 0.18, p<0.05).

3.4.17. Summary of meta-analyses findings

Figure 3.5 displays the overall effect sizes for each EBT on EI and BMI. A positive effect size indicates that the EBT is positively correlated with EI or BMI. A negative effect size indicates that the EBT is negatively correlated with EI or BMI. Intuitive eating and eating disorder symptomology are not displayed in Figure 3.5 due to the small number of studies included in these meta-analyses and the large variability in their correlations with EI and BMI. Figure 3.6 displays the effect sizes for the subgroup analysis testing the correlation between restraint and EI influenced by the questionnaire used to measure restraint. Error bars in both figures reflect standard error.

Figure 3.5. Summary of Meta-Analyses Findings

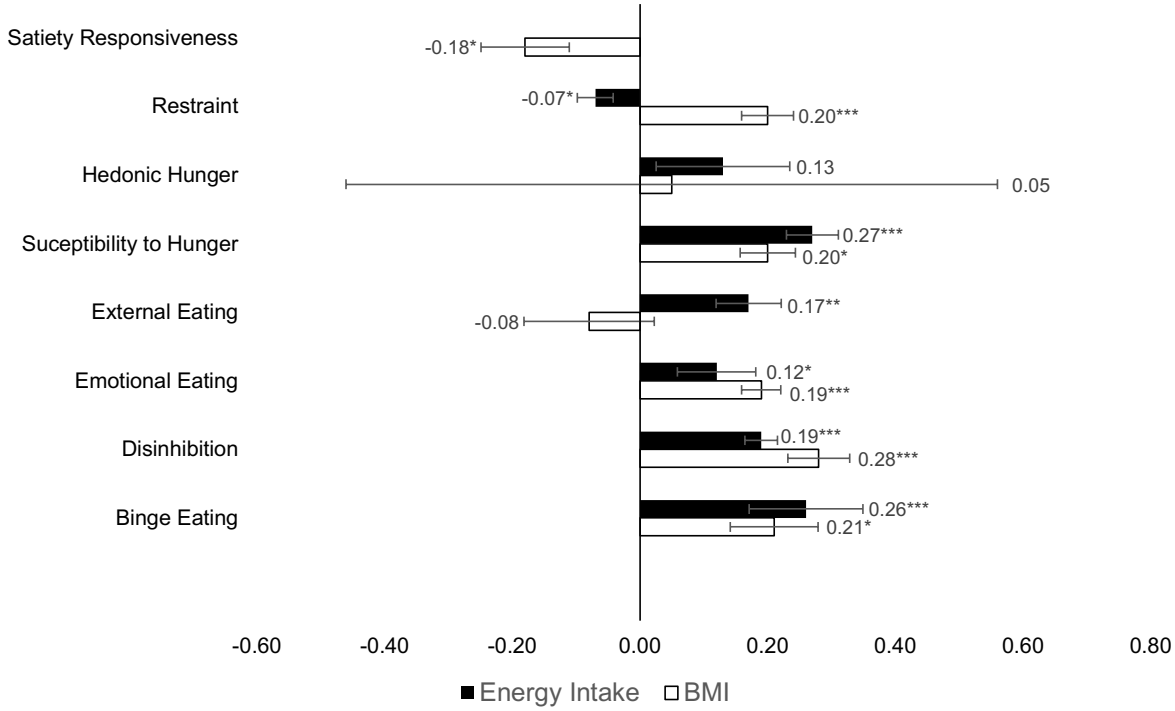
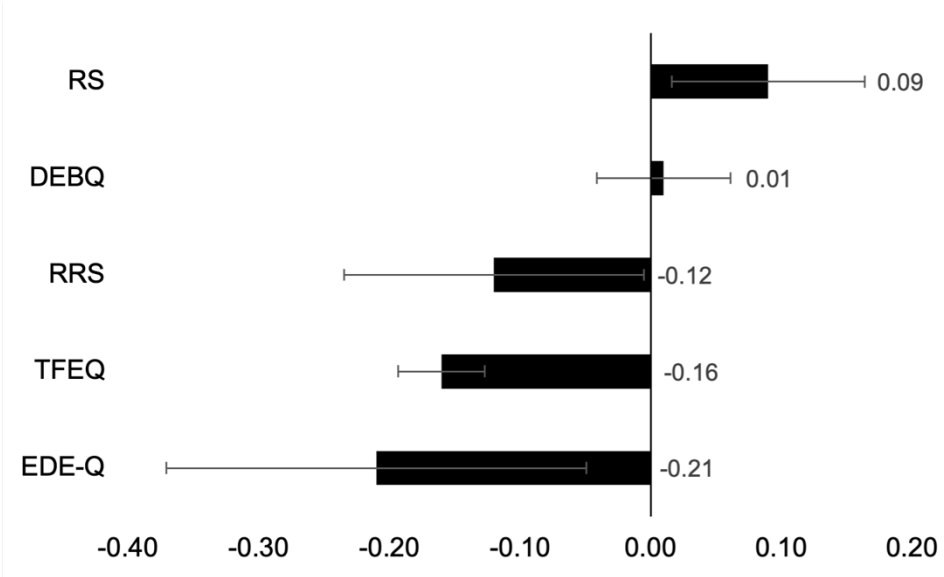


Figure 3.6. Bar graph of the subgroup analysis results, testing the correlation between restraint and EI influenced by the questionnaire used to measure restraint



3.5. Discussion

The aims of this systematic review and meta-analyses were to (1) identify which measures are used to capture EBTs, (2) assess how well these measures associate with short term EI and (3), BMI, (4) and evaluate whether some EBTs and questionnaires used to measure EBTs are better at predicting EI or BMI than others. Sixteen EBTs were identified. However, the majority of EBTs included were restraint, disinhibition, emotional eating, external eating, and binge eating. Considering the >45 years of research on human eating behaviour, this systematic review demonstrates that relatively few studies on EBTs have been conducted in relation to indices of objectively measured short-term EI as well as long term energy balance status (BMI). For most of the EBTs identified (aside from restraint), this research has been accomplished in only a small number of studies. Additionally, the majority of traits were measured using a single questionnaire (see Table 3.1), which were often older measures such as TFEQ or RS. This means that there are additional, typically newer questionnaires with no research that met the inclusion criteria for this systematic review e.g. the Positive and Negative Emotional Eating Scale.[129] Future research should assess the validity of these traits in relation to EI and BMI.

Overall, the meta-analyses demonstrated that EBTs do significantly predict both EI and BMI. Effect sizes were generally moderate, with some analyses reaching larger effects. Susceptibility to hunger and binge eating were the strongest predictors of EI, whilst disinhibition was the strongest predictor of BMI. While intuitive eating was moderately correlated with BMI, this meta-analysis was based on a very small number of studies. If there are not at least two adequately powered studies per meta-analysis, there may not be enough information to contribute an accurate conclusion of evidence about that EBT.[130] Therefore, drawing conclusions about intuitive eating and satiety responsiveness requires caution. Below, the main outcomes of the systematic review are discussed.

3.5.1. Restraint

Restraint as a concept relates to a cognitive intention to restrict food intake, which is not necessarily reflected in actual reduction in EI or adherence to a weight reducing

diet.[103] This meta-analysis found a positive association between restraint and BMI, which is consistent with previous cross-sectional studies.[131,132] However, negative findings between restraint and BMI have also emerged in the literature.[133] The finding of this systematic review could suggest that whilst restrained individuals attempt to restrict their food intake, increased restraint puts them at a higher risk of overeating (disinhibition), leading to a positive energy balance and consequently an increased BMI. However, the results of this systematic review do not agree with this prediction. Increased restraint was associated with decreased short-term EI. This finding is more consistent with the interpretation that people with a higher BMI invoke strategies of dietary restraint to manage their weight. Approximately 40% of the adult population report at least one weight management attempt in the preceding 12 months.[134] It is also evident from the weight management literature that the majority of weight management attempts have limited success and are subject to weight regain.[135]

Previous literature examining associations between restraint and EI have also been inconsistent, which is demonstrated in this systematic review as both positive and negative associations were found between restraint and EI. Research has highlighted that the different measures used to assess restraint could be influencing the different outcomes (research question 4). Heatherton, Herman, Polivy, King and McGree [136] suggest that the TFEQ may be better able to identify individuals who are successful in restricting their food intake, while the RS is better able to identify individuals who are unsuccessful in dieting. Indeed, the subgroup analysis found a significant effect of questionnaire type, such that the TFEQ was associated with decreased EI, whilst the RS was associated with increased EI (see A.6). The EDE-Q and TFEQ had the largest effect size therefore suggesting these measures may be better at predicting EI. This analysis suggests that each questionnaire may not be measuring exactly the same construct and generates questions about which scale is best at measuring restraint. The RS, for example, could be contaminated by other constructs such as disinhibition, as items on this scale refer to overeating e.g. “Do you have feelings of guilt after overeating?” This may explain why the RS was positively associated with EI.[103] Moreover, there is also a potential need to re-evaluate what is meant by the construct of restraint. Is restraint a measure of concern controlling intake and its consequences, or could it be a measure of

motivation or success in restraining intake like the TFEQ appears to measure or perhaps a combination of these considerations? There is a need to clearly define and achieve consensus about what restraint is and what it is not.

A prediction of restraint theory is that the likelihood of a binge/overeating episode should be increased after consuming a preload only in high restraint individuals. This is because dieters who have just broken their diet are predicted to subsequently overeat.[103,137] In the current analysis, the correlation between restraint and EI was influenced by a preload such that the use of a preload was associated with increased EI, whilst no preload was associated with decreased EI (see A.7). Nevertheless, Ouwens, van Strien and van der Staak [86] reported no significant effect of restraint on EI influenced by a preload. The researchers highlight that not all dieters will show this disinhibition effect; but a particular subgroup might. Restrained eaters can therefore be split into two subpopulations: successful dieters and unsuccessful dieters. Successful dieters are characterised by high restraint and low levels of tendency towards overeating. Unsuccessful dieters are characterised by high restraint and are prone to overeating, meaning they are more likely to show a disinhibition effect after a preload. Westenhoefer, Broeckmann, Münch and Pudel [104] provided evidence for this idea and found that overeating only occurred in participants who displayed high scores on both restraint and disinhibition, as measured by the TFEQ.

More recent research such as Bryant, Caudwell, Blundell, Hopkins and King [66] and Chambers and Yeomans [67] have tested this effect by splitting participants into four groups based on their restraint (high vs low; HR vs LR) and disinhibition (high vs low; HD vs LD) scores using the TFEQ (HD/HR, HD/LR, LD/HR, LD/LR). A limitation of the current systematic review is that effect sizes were pooled so that high restraint included high and low disinhibition and low restraint included both high and low disinhibition. By not keeping the four groups separate, the analysis may have been insensitive to the effects of differing disinhibition levels. Consequently, future research could conduct a multi-level meta-analysis of restraint on EI, which can account for the influence of other variables such as disinhibition and emotional eating. Based on these findings, one must consider that restraint itself may not be a homogenous construct. Westenhoefer, Broeckmann, Münch and Pudel [104] proposed

that restraint can be split into rigid and flexible control, with rigid control relating to an all-or-nothing approach. In contrast, flexible control reflects a less stringent approach to restriction of intake. These subscales could reflect successful and unsuccessful dieters and may better account for the differing effects of restraint on EI. Unfortunately, only two studies in this review[66,104] split restraint into flexible and rigid control and therefore testing the validity of this concept is currently not possible. Overall, this systematic raises questions about the current definition of restraint and whether it should be altered. If restraint is associated with increased BMI, then attempting to restrain intake as a dietary approach to weight loss may not be effective and other approaches should be utilised.

3.5.2. Emotional eating

Another interesting finding of this systematic review was that emotional eating was positively correlated with short term EI and BMI, although there were two studies in the meta-analysis that found a negative correlation between emotional eating and EI.[71,101] The small-to-moderate effect size could have been influenced by the inclusion of only control conditions in this systematic review. Conditions that attempted to influence the emotional state of participants were excluded to reduce potential contaminates of the specific emotional eating trait effect. However, studies have shown that emotional eaters increased their food intake after a negative mood induction, compared to a neutral or positive mood induction.[119,138] A limitation of this systematic review was that only one study presented data for the positive and negative subscales of emotional eating,[116] and therefore the influence of positive or negative emotional eating could not be assessed. Further research should look to test systematically whether emotion valence, as well as emotion induction conditions increase EI in high emotional eaters vs low emotional eaters.

3.5.3. Disinhibition

The overall finding that disinhibition was positively associated with short-term EI and BMI supports previous research[6,9] and suggests that associations between uncontrolled eating and food intake are more consistently measured by scales capturing disinhibition, potentially because disinhibition is a ‘pure’ measure of uncontrolled eating. These findings suggest that increased disinhibition is an

indicator of risk for potential weight gain and could be targeted in weight loss interventions characterise participants at risk of weight regain. Disinhibition could influence weight regain [139] and research has found that participants who maintained weight loss were characterised by lower disinhibition scores.[140] Niemeier, Phelan, Fava and Wing [141] found that higher internal disinhibition predicted less successful weight loss and studies have consistently shown that successful weight loss is associated with a decrease in disinhibition.[142-144] If weight loss interventions can successfully target and reduce participants' uncontrolled eating behaviour (disinhibition), this approach may improve weight outcomes.

Furthermore, binge eating and susceptibility to hunger were also consistent predictors of EI and BMI and may be useful constructs to target in weight loss interventions. Assessing whether other traits are associated with behaviour change is another important direction for future research. For example, studies have found that higher baseline emotional eating was associated with more weight gain over time,[145] meaning emotional eating could also be targeted in weight loss interventions. Mindfulness-based interventions have also been used to help participants decrease their tendency to eat in response to negative emotions.[146] Future research should assess which traits are prospectively associated with weight change or weight maintenance, as well as investigating the long-term efficacy of interventions utilising EBTs. Ultimately, disinhibition is a robust EBT that should be used in weight loss interventions and further research should strive to find ways to reduce levels of disinhibition in those scoring high on this subscale.

3.5.4. Methodological comments

This systematic review included only laboratory-based measures of EI because there is substantial evidence that self-report measures are susceptible to misreporting.[10] However, laboratory-based measures of EI are highly susceptible to experimental design and demand characteristics. For example, Long, Meyer, Leung and Wallis [147] highlight that eating in a laboratory is an unnaturalistic setting and could distract and impede a participant's focus on internal signals of hunger and satiety, which could influence their EI. Another concern relates to how well one single eating episode can generalise to usual eating behaviour.[59] Snacking once in a test meal (often involving

unusual foods) may not be an accurate reflection on a participants normal EI. Fourteen studies did test EI over multiple test meals, which helps to reduce this limitation. However, there were still 68 studies that tested a single eating episode and therefore, multiple test meal methods are not commonplace in eating behaviour research. There are also issues relating to the 'observer effect,' whereby participants behaviour is biased by the awareness that they are being observed and that they are participating in a food-related study.[148] Studies rarely attempt to disguise that a meal is being used to measure food consumption and this transparency may alter the way a participant eats.[14] Laboratory measures of food intake may have limited generalisability to normal every day eating behaviour. However, the current meta-analyses generalise over multiple test designs and test meals which helps to overcome the role of specific research designs.

Another limitation of many studies included in this systematic review is the potential for other variables measured to contaminate the EI outcome. While studies that included potential contaminates were excluded, there were still some tasks which may have influenced the outcomes. For example, Coelho, Polivy, Herman and Pliner [68], included a control condition that required participants to complete an arithmetic problem-solving task and a word-recall task. The researchers found that the participants could not complete the test within the time provided and could have exposed the participants to task failure or ego threat which may have affected the participant mood/emotions.

3.5.5. Conceptual comments

An important consideration of EBTs is uncertainty over the extent to which individuals are capable of accurately assessing their own eating behaviour, for example, emotional eating. Evers, de Ridder and Adriaanse [117] suggests a 'triple recall bias,' whereby people are generally unable to perceive their own behaviour, they underreport their EI and personality disturbances, and retrospective emotional ratings are known to be highly sensitive to recall bias. Consequently, questionnaires that measure emotional eating are asking participants to recall their negative or positive emotions, food intake and association between both, all three of which may be biased. It is therefore possible that in some populations e.g., the elderly and

young children, recall methods may not yield accurate findings and as such, should not be used. However, recommendations for specific population where EBTs assessments should not be measured is beyond the scope of this analysis. The triple recall bias can also be extended to other psychological EBTs, which raises concerns over their construct validity and questions whether EBTs are measuring what they were designed to measure? Interestingly, new developments in real time digital tracking of energy balance behaviours,[149-153] digital ecological momentary assessments and mobile video recording[154] could be a new avenue for more ecological assessments of EI to capture EBTs.

Most studies test the validity of EBTs against very short-term EI, often over one test day. In order to relate EBTs to long-term outcomes, EI and BMI need to be measured longitudinally. This also holds true for studies that aim to assess how EBTs change in weight management interventions. In the short term, EBTs are not expected to change because they are long-term processes. Therefore, to examine how EBTs are associated with behaviour change, they also need to be measured over a longer period of time.

3.5.6. Conclusions

The outcome of this review has demonstrated that many EBTs are associated with short-term EI and BMI, with disinhibition, susceptibility to hunger and binge eating having the largest effect sizes for EI and BMI. Dietary restraint is evidently a complex construct and further research with preload conditions and separate subscales are needed to fully understand its effect on EI. The effect of emotional eating may have been limited by not including mood or stress induction conditions. However, this review still indicates it plays a role in EI and BMI. Disinhibition had the strongest positive correlation with EI which highlights its potential use as a phenotypic marker of susceptibility to overconsume, develop obesity, or to influence outcomes in weight management interventions. Importantly, there are methodological and conceptual issues with EBTs that need to be stressed when utilising these traits in eating behaviour research. This does not mean they should not be used but a greater focus on objective indices of what such traits should predict (food intake and EI, BMI or

weight change) might improve their use in explaining eating behaviour and energy balance in the wider population.

3.6. References

1. De Onis M, Blössner M, Borghi E. Global prevalence and trends of overweight and obesity among preschool children. *The American journal of clinical nutrition*. 2010;92(5):1257-1264.
2. Blundell JE, Stubbs R, Golding C, et al. Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. *Physiology & behavior*. 2005;86(5):614-622.
3. Llewellyn C, Wardle J. Behavioral susceptibility to obesity: gene–environment interplay in the development of weight. *Physiology & behavior*. 2015;152:494-501.
4. Finlayson G, Arlotti A, Dalton M, King N, Blundell JE. Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. *APPETITE*. 2011;57(3)doi:10.1016/j.appet.2011.08.012
5. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*. 1985;29(1):71-83.
6. Vainik U, Garcia-Garcia I, Dagher A. Uncontrolled eating: a unifying heritable trait linked with obesity, overeating, personality and the brain. *The European journal of neuroscience*. 2019;50(3)doi:https://dx.doi.org/10.1111/ejn.14352
7. Blundell JE, King NA, Bryant EJ. Disinhibition: Its effects on appetite and weight regulation. *Obesity Reviews*. 2008;9(5)doi:http://dx.doi.org/10.1111/j.1467-789X.2007.00426.x
8. Espel-Huynh HM, Muratore AF, Lowe MR. A narrative review of the construct of hedonic hunger and its measurement by the Power of Food Scale. *Obesity Science and Practice*. 2018;4(3)doi:http://dx.doi.org/10.1002/osp4.161
9. French A, Epstein LH, Jeffery RW, Blundell JE, Wardle J. Eating behavior dimensions. Associations with energy intake and body weight. A review. *Appetite*. 2012;59(2)doi:https://dx.doi.org/10.1016/j.appet.2012.07.001
10. Stubbs RJ, O'Reilly LM, Whybrow S, et al. Measuring the difference between actual and reported food intakes in the context of energy balance under laboratory conditions. *British Journal of Nutrition*. 2014;111(11):2032-2043.
11. Devonport TJ, Nicholls W, Fullerton C. A systematic review of the association between emotions and eating behaviour in normal and overweight adult populations.

Journal of health psychology.
2019;24(1)doi:http://dx.doi.org/10.1177/1359105317697813
12. Vainik U, Dagher A, Dubé L, Fellows LK. Neurobehavioural correlates of body mass index and eating behaviours in adults: a systematic review. *Neuroscience & Biobehavioral Reviews*. 2013;37(3):279-299.
13. Bryant EJ, Rehman J, Pepper LB, Walters ER. Obesity and Eating Disturbance: the Role of TFEQ Restraint and Disinhibition. *Current Obesity Reports*. 2019;8(4)doi:http://dx.doi.org/10.1007/s13679-019-00365-x
14. Robinson E, Haynes A, Hardman CA, Jones A, Kemps E, Higgs S. The bogus taste test: Validity as a measure of laboratory food intake. *Appetite*. 2017;116doi:http://dx.doi.org/10.1016/j.appet.2017.05.002
15. Dhurandhar NV, Schoeller D, Brown AW, et al. Energy balance measurement: when something is not better than nothing. *International journal of obesity*. 2015;39(7):1109-1113.
16. UCL. Eating Behavior Questionnaires. Accessed 21/03/2022, https://www.ucl.ac.uk/epidemiology-health-care/research/behavioural-science-and-health/resources/questionnaires/eating-behaviour-questionnaires
17. Grid-Enabled Measures Database. Obesity. Accessed 21/03/2022, https://www.gem-measures.org/Public/GEMSearchResults.aspx?allm=true&srchterm=obesity
18. Arab Psychology. Psychological Scales. Accessed 21/03/2022, https://scales.arabpsychology.com
19. McGowan J, Sampson M, Salzwedel DM, Cogo E, Foerster V, Lefebvre C. PRESS peer review of electronic search strategies: 2015 guideline statement. *Journal of clinical epidemiology*. 2016;75:40-46.
20. Shamseer L, Moher D, Clarke M, et al. Preferred reporting items for systematic review and meta-analysis protocols (PRISMA-P) 2015: elaboration and explanation. *Bmj*. 2015;349
21. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. *Addictive behaviors*. 1982;7(1):47-55.
22. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders: DSM-5*. vol 5th ed. American Psychiatric Sssociation Washington, DC; 2013.

23. Cochrane. Data Extraction Forms. Accessed 21/03/2022, https://dplp.cochrane.org/data-extraction-forms
24. R Core Team. R: A language and environment for statistical computing. R Foundation for Statistical Computing URL https://www.R-project.org/.
25. Harrer M, Cuijpers P, Furukawa T, Ebert D. Companion R Package For The Guide 'Doing Meta-Analysis in R. 2019;doi:http://dmetar.protectlab.org/
26. Lüdecke D. esc: Effect Size Computation for Meta Analysis (Version 0.5.1). 2019;doi:10.5281/zenodo.1249218, https://CRAN.R-project.org/package=esc.
27. Wickham et al. Welcome to the tidyverse. *Journal of Open Source Software*. 2019;4(43), 1986doi:https://doi.org/10.21105/joss.01686
28. Balduzzi S, Rücker G, Schwarzer G. How to perform a meta-analysis with R: a practical tutorial. *Evidence-Based Mental Health*. 2019;153-160doi:10.1136/ebmental-2019-300117
29. Fisher RA. Statistical methods for research workers. *Breakthroughs in statistics*. Springer; 1992:66-70.
30. Higgins JP, Thomas J, Chandler J, et al. *Cochrane handbook for systematic reviews of interventions*. John Wiley & Sons; 2019.
31. Harrer M, Cuijpers P, Furukawa TA, Ebert DD. *Doing meta-analysis with R: A hands-on guide*. Chapman and Hall/CRC; 2021.
32. Funder DC, Ozer DJ. Evaluating effect size in psychological research: Sense and nonsense. *Advances in Methods and Practices in Psychological Science*. 2019;2(2):156-168.
33. von Hippel PT. The heterogeneity statistic I2 can be biased in small meta-analyses. *BMC medical research methodology*. 2015;15(1):1-8.
34. Knapp G, Hartung J. Improved tests for a random effects meta-regression with a single covariate. *Statistics in medicine*. 2003;22(17):2693-2710.
35. IntHout J, Ioannidis J, Borm GF. The Hartung-Knapp-Sidik-Jonkman method for random effects meta-analysis is straightforward and considerably outperforms the standard DerSimonian-Laird method. *BMC medical research methodology*. 2014;14(1):1-12.
36. Hedges LV. Distribution theory for Glass's estimator of effect size and related estimators. *journal of Educational Statistics*. 1981;6(2):107-128.
37. Hong Q, Pluye P, Fàbregues S, et al. Mixed Methods Appraisal Tool (MMAT) Version 2018. Accessed 21/03/2022,

http://mixedmethodsappraisaltoolpublic.pbworks.com/w/file/fetch/127916259/MMAT_2018_criteria-manual_2018-08-01_ENG.pdf

38. Hong QN, Fàbregues S, Bartlett G, et al. The Mixed Methods Appraisal Tool (MMAT) version 2018 for information professionals and researchers. *Education for information*. 2018;34(4):285-291.

39. Tylka TL. Development and psychometric evaluation of a measure of intuitive eating. *Journal of Counseling Psychology*. 2006;53(2):226.

40. Tylka TL, Kroon Van Diest AM. The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men. *Journal of counseling psychology*. 2013;60(1):137.

41. Framson C, Kristal AR, Schenk JM, Littman AJ, Zeliadt S, Benitez D. Development and validation of the mindful eating questionnaire. *Journal of the American dietetic Association*. 2009;109(8):1439-1444.

42. Green S, Delargy H, Joanes D, Blundell J. A satiety quotient: a formulation to assess the satiating effect of food. *Appetite*. 1997;29(3):291-304.

43. Hunot C, Fildes A, Croker H, Llewellyn CH, Wardle J, Beeken RJ. Appetitive traits and relationships with BMI in adults: Development of the Adult Eating Behaviour Questionnaire. *Appetite*. 2016;105:356-363.

44. Herman CP, Mack D. Restrained and unrestrained eating. *Journal of personality*. 1975;

45. Herman CP, Polivy J, Silver R. Effects of an observer on eating behavior: the induction of "sensible" eating. *Journal of personality*. 1979;47(1)

46. van Strien T, Frijters JE, Bergers GP, Defares PB. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International journal of eating disorders*. 1986;5(2):295-315.

47. Dalton M, Finlayson G, Blundell J, Hill A. Preliminary validation and principal components analysis of the Control of Eating Questionnaire (CoEQ) for the experience of food craving. *European Journal of Clinical Nutrition*. 2015;69(12)doi:http://dx.doi.org/10.1038/ejcn.2015.57

48. Lowe MR, Butryn ML, Didie ER, et al. The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*. 2009;53(1):114-118.

49. White MA, Whisenhunt BL, Williamson DA, Greenway FL, Netemeyer RG. Development and validation of the food-craving inventory. *Obesity research*. 2002;10(2)
50. Fairburn CG, Cooper Z, O'Connor M. The eating disorder examination. *International Journal of Eating Disorders*. 1993;6:1-8.
51. Akkermann K, Herik M, Aluoja A, Järv A. Constructing an assessment scale for eating disorders. *Tartu: Department of Psychology of University of Tartu*. 2010;
52. Stice E, Telch CF, Rizvi SL. Development and validation of the Eating Disorder Diagnostic Scale: a brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological assessment*. 2000;12(2):123.
53. Garner DM. Eating Disorder Inventory-2; Pro-fessional Manual. *Psychological assessment resources*. 1991;
54. Clark MM, Abrams DB, Niaura RS, Eaton CA, Rossi JS. Self-efficacy in weight management. *Journal of consulting and clinical psychology*. 1991;59(5):739.
55. Vainik U, Eun Han J, Epel ES, Janet Tomiyama A, Dagher A, Mason AE. Rapid Assessment of Reward-Related Eating: The RED-X5. *Obesity*. 2019;27(2):325-331.
56. Gearhardt AN, Corbin WR, Brownell KD. Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*. 2016;30(1):113.
57. Anderson DA, Schaumberg K, Anderson LM, Reilly EE. Is level of intuitive eating associated with plate size effects? *Eating behaviors*. 2015;18:125-130.
58. Anderson LM, Reilly EE, Schaumberg K, Dmochowski S, Anderson DA. Contributions of mindful eating, intuitive eating, and restraint to BMI, disordered eating, and meal consumption in college students. *Eating and Weight Disorders*. 2016;21(1)doi:http://dx.doi.org/10.1007/s40519-015-0210-3
59. Ruzanska UA, Warschburger P. How is intuitive eating related to self-reported and laboratory food intake in middle-aged adults? *EATING BEHAVIORS*. 2020;38doi:10.1016/j.eatbeh.2020.101405
60. Dalton M, Hollingworth S, Blundell J, Finlayson G. Weak Satiety Responsiveness Is a Reliable Trait Associated with Hedonic Risk Factors for Overeating among Women. *NUTRIENTS*. 2015;7(9)doi:10.3390/nu7095345
61. Drapeau V, Blundell J, Therrien F, Lawton C, Richard D, Tremblay A. Appetite sensations as a marker of overall intake. *BRITISH JOURNAL OF NUTRITION*. 2005;93(2)doi:10.1079/BJN20041312

62. Drapeau V, Jacob R, Panahi S, Tremblay A. Effect of energy restriction on eating behavior traits and psychobehavioral factors in the low satiety phenotype. *Nutrients*. 2019;11(2)doi:http://dx.doi.org/10.3390/nu11020245
63. Zuraikat FM, Roe LS, Smethers AD, Reihart LW, Rolls BJ. Does the cost of a meal influence the portion size effect? *Appetite*. 2018;127:341-348.
64. Ard JD, Desmond RA, Allison DB, Conway JM. Dietary restraint and disinhibition do not affect accuracy of 24-hour recall in a multiethnic population. *JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION*. 2006;106(3)doi:10.1016/j.jada.2005.12.006
65. Bellisle F, Dalix A-M, Airinei G, Hercberg S, Péneau S. Influence of dietary restraint and environmental factors on meal size in normal-weight women. A laboratory study. *Appetite*. 2009;53(3):309-313.
66. Bryant EJ, Caudwell P, Blundell JE, Hopkins ME, King NA. Psycho-markers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*. 2012;58(1)doi:http://dx.doi.org/10.1016/j.appet.2011.09.006
67. Chambers L, Yeomans MR. Individual differences in satiety response to carbohydrate and fat. Predictions from the Three Factor Eating Questionnaire (TFEQ). *Appetite*. 2011;56(2)doi:https://dx.doi.org/10.1016/j.appet.2011.01.003
68. Coelho JS, Polivy J, Herman CP, Pliner P. Wake up and smell the cookies. Effects of olfactory food-cue exposure in restrained and unrestrained eaters. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.10.008
69. Cools J, Schotte DE, McNally RJ. Emotional arousal and overeating in restrained eaters. *Journal of abnormal psychology*. 1992;101(2):348.
70. de Witt Huberts JC, Evers C, de Ridder DT. Double trouble: restrained eaters do not eat less and feel worse. *Psychology & Health*. 2013;28(6):686-700.
71. Dweck JS, Jenkins SM, Nolan LJ. The role of emotional eating and stress in the influence of short sleep on food consumption. *Appetite*. 2014;72doi:http://dx.doi.org/10.1016/j.appet.2013.10.001
72. Fedoroff ID, Polivy J, Herman CP. The effect of pre-exposure to food cues on the eating behavior of restrained and unrestrained eaters. *Appetite*. 1997;28(1):33-47.
73. Finlayson G, Blundell JE, Bordes I, Griffioen-Roose S, de Graaf C. Susceptibility to overeating affects the impact of savory or sweet drinks on satiation,

reward, and food intake in nonobese women. *Journal of Nutrition*. 2012;142(1)doi:http://dx.doi.org/10.3945/jn.111.148106

74. Guerrieri R, Nederkoorn C, Schrooten M, Martijn C, Jansen A. Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet. *Appetite*. 2009;53(1)doi:https://dx.doi.org/10.1016/j.appet.2009.05.013

75. Haynes C, Lee MD, Yeomans MR. Interactive effects of stress, dietary restraint, and disinhibition on appetite. *Eating behaviors*. 2003;4(4):369-383.

76. Herhaus B, Petrowski K. The effect of restrained eating on acute stress-induced food intake in people with obesity. *Appetite*. 2021;159doi:https://dx.doi.org/10.1016/j.appet.2020.105045

77. Higgs S, Williamson AC, Attwood AS. Recall of recent lunch and its effect on subsequent snack intake. *Physiology & behavior*. 2008;94(3):454-462.

78. Hofmann W, Friese M. Impulses got the better of me: alcohol moderates the influence of implicit attitudes toward food cues on eating behavior. *Journal of abnormal psychology*. 2008;117(2)doi:https://dx.doi.org/10.1037/0021-843X.117.2.420

79. Jansen A. How restrained eaters perceive the amount they eat. *The British journal of clinical psychology*. 1996;35(3)

80. Jansen A, Merckelbach H, Oosterlaan J, Tuiten A, Van Den Hout M. Cognitions and self-talk during food intake of restrained and unrestrained eaters. *Behaviour Research and Therapy*. 1988;26(5):393-398.

81. Lattimore P, Maxwell L. Cognitive load, stress, and disinhibited eating. *Eating Behaviors*. 2004;5(4):315-324.

82. Martin CK, Williamson DA, Geiselman PJ, et al. Consistency of food intake over four eating sessions in the laboratory. *Eating Behaviors*. 2005;6(4):365-372.

83. McNeil J, Lamothe G, Cameron JD, et al. Investigating predictors of eating: is resting metabolic rate really the strongest proxy of energy intake? *The American journal of clinical nutrition*. 2017;106(5)doi:https://dx.doi.org/10.3945/ajcn.117.153718

84. Myhre R, Buchwald D, Kratz M, et al. A twin study of differences in the response of plasma ghrelin to a milkshake preload in restrained eaters. *Physiology and Behavior*. 2014;129doi:http://dx.doi.org/10.1016/j.physbeh.2014.02.008

85. Oliver G, Wardle J, Gibson EL. Stress and food choice: a laboratory study. *Psychosomatic medicine*. 2000;62(6):853-865.
86. Ouwens MA, van Strien T, van der Staak CP. Tendency toward overeating and restraint as predictors of food consumption. *Appetite*. 2003;40(3):291-298.
87. Peluso A. Comparison of mindful and slow eating strategies on acute energy intake. *Dissertation Abstracts International: Section B: The Sciences and Engineering*. 2017;78(5-B(E))
88. Rideout CA, McLean JA, Barr SI. Women with high scores for cognitive dietary restraint choose foods lower in fat and energy. *Journal of the American Dietetic Association*. 2004;104(7):1154-1157.
89. Rolls BJ, Castellanos VH, Shide DJ, et al. Sensory properties of a nonabsorbable fat substitute did not affect regulation of energy intake. *The American journal of clinical nutrition*. 1997;65(5):1375-1383.
90. Ruddock HK, Field M, Hardman CA. Exploring food reward and calorie intake in self-perceived food addicts. *Appetite*. 2017;115doi:http://dx.doi.org/10.1016/j.appet.2016.12.003
91. Schoch AH, Raynor HA. Social desirability, not dietary restraint, is related to accuracy of reported dietary intake of a laboratory meal in females during a 24-hour recall. *Eating behaviors*. 2012;13(1):78-81.
92. Schotte DE, Cools J, McNally RJ. Film-induced negative affect triggers overeating in restrained eaters. *Journal of Abnormal Psychology*. 1990;99(3):317.
93. Shapiro JR, Anderson DA. Counterregulatory eating behavior in multiple item test meals. *Eating behaviors*. 2005;6(2)
94. Sim AY, Lee LL, Cheon BK. When exercise does not pay: Counterproductive effects of impending exercise on energy intake among restrained eaters. *Appetite*. 2018;123doi:https://dx.doi.org/10.1016/j.appet.2017.12.017
95. Smith CF, Geiselman PJ, Williamson DA, Champagne CM, Bray GA, Ryan DH. Association of dietary restraint and disinhibition with eating behavior, body mass, and hunger. *Eating and weight disorders : EWD*. 1998;3(1)
96. Stice E, Fisher M, Lowe MR. Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. *Psychological assessment*. 2004;16(1):51.

97. Stice E, Sysko R, Roberto CA, Allison S. Are dietary restraint scales valid measures of dietary restriction? Additional objective behavioral and biological data suggest not. *Appetite*. 2010;54(2):331-339.
98. Stinson EJ, Votruba SB, Venti C, Krakoff J, Gluck ME, Perez M. Food Insecurity is Associated with Maladaptive Eating Behaviors and Objectively Measured Overeating. *Obesity*. 2018;26(12)doi:http://dx.doi.org/10.1002/oby.22305
99. van Strien T, Ouwens MA. Counterregulation in female obese emotional eaters: Schachter, Goldman, and Gordon's (1968) test of psychosomatic theory revisited. *Eating Behaviors*. 2003;3(4)doi:http://dx.doi.org/10.1016/S1471-0153%2802%2900092-2
100. Visona C, George VA. Impact of dieting status and dietary restraint on postexercise energy intake in overweight women. *OBESITY RESEARCH*. 2002;10(12)doi:10.1038/oby.2002.170
101. Wallis DJ, Hetherington MM. Stress and eating: the effects of ego-threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite*. 2004;43(1):39-46.
102. Wallis DJ, Hetherington MM. Emotions and eating. Self-reported and experimentally induced changes in food intake under stress. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.11.007
103. Wardle J, Beales S. Restraint and food intake: An experimental study of eating patterns in the laboratory and in normal life. *Behaviour Research and Therapy*. 1987;25(3)doi:http://dx.doi.org/10.1016/0005-7967%2887%2990044-1
104. Westenhoefer J, Broeckmann P, Münch A-K, Pudel V. Cognitive control of eating behavior and the disinhibition effect. *Appetite*. 1994;23(1):27-41.
105. Yeomans MR, Coughlan E. Mood-induced eating. Interactive effects of restraint and tendency to overeat. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.10.006
106. Yeomans MR, Tovey H, Tinley E, Haynes C. Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. *International journal of obesity*. 2004;28(1):144-151.
107. Zambrowicz R, Schebendach J, Sysko R, Mayer LES, Walsh BT, Steinglass JE. Relationship between three factor eating questionnaire-restraint subscale and food intake. *International Journal of Eating Disorders*. 2019;52(3)doi:http://dx.doi.org/10.1002/eat.23014

108. Appelhans BM, Liebman R, Woolf K, Pagoto SL, Schneider KL, Whited MC. Inhibiting food reward: Delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*. 2011;19(11)doi:http://dx.doi.org/10.1038/oby.2011.57
109. Ely AV, Howard J, Lowe MR. Delayed discounting and hedonic hunger in the prediction of lab-based eating behavior. *Eating behaviors*. 2015;19doi:https://dx.doi.org/10.1016/j.eatbeh.2015.06.015
110. Nolan-Poupart S, Veldhuizen MG, Geha P, Small DM. Midbrain response to milkshake correlates with ad libitum milkshake intake in the absence of hunger. *Appetite*. 2013;60(1)doi:https://dx.doi.org/10.1016/j.appet.2012.09.032
111. Martin CK, O'Neil PM, Tollefson G, Greenway FL, White MA. The association between food cravings and consumption of specific foods in a laboratory taste test. *Appetite*. 2008;51(2):324-326.
112. Epstein LH, Lin H, Carr KA, Fletcher KD. Food reinforcement and obesity. Psychological moderators. *APPETITE*. 2012;58(1)doi:10.1016/j.appet.2011.09.025
113. Hopkins M, Michalowska J, Whybrow S, Horgan GW, Stubbs RJ. Identification of psychological correlates of dietary misreporting under laboratory and free-living environments. *British Journal of Nutrition*. 2021;126(2)doi:http://dx.doi.org/10.1017/S000711452000389X
114. Kakoschke N, Kemps E, Tiggemann M. External eating mediates the relationship between impulsivity and unhealthy food intake. *Physiology & Behavior*. 2015;147doi:http://dx.doi.org/10.1016/j.physbeh.2015.04.030
115. Robinson E, Haynes A. Individual differences and moderating participant characteristics in the effect of reducing portion size on meal energy intake: Pooled analysis of three randomized controlled trials. *Appetite*. 2021;159doi:http://dx.doi.org/10.1016/j.appet.2020.105047
116. van Strien T, Donker MH, Ouwens MA. Is desire to eat in response to positive emotions an 'obese' eating style: Is Kummerspeck for some people a misnomer? *Appetite*. 2016;100doi:http://dx.doi.org/10.1016/j.appet.2016.02.035
117. Evers C, de Ridder DTD, Adriaanse MA. Assessing yourself as an emotional eater: mission impossible? *Health psychology : official journal of the Division of Health Psychology, American Psychological Association*. 2009;28(6)doi:https://dx.doi.org/10.1037/a0016700

118. Raspopow K, Abizaid A, Matheson K, Anisman H. Anticipation of a psychosocial stressor differentially influences ghrelin, cortisol and food intake among emotional and non-emotional eaters. *Appetite*. 2014;74:35-43.
119. van Strien T, Herman CP, Anschutz DJ, Engels RC, de Weerth C. Moderation of distress-induced eating by emotional eating scores. *Appetite*. 2012;58(1):277-284.
120. Alger S, Seagle H, Ravussin E. Food-intake and Energy Expenditure in Obese Female Bingers and Non-bingers. *INTERNATIONAL JOURNAL OF OBESITY*. 1995;19(1)
121. Arumae K, Kreegipuu K, Vainik U. Assessing the Overlap Between Three Measures of Food Reward. *FRONTIERS IN PSYCHOLOGY*. 2019;10doi:10.3389/fpsyg.2019.00883
122. Dalton M, Blundell J, Finlayson G. Effect of BMI and binge eating on food reward and energy intake: Further evidence for a binge eating subtype of obesity. *Obesity Facts*. 2013;6(4)doi:http://dx.doi.org/10.1159/000354599
123. Dalton M, Blundell J, Finlayson GS. Examination of food reward and energy intake under laboratory and free-living conditions in a trait binge eating subtype of obesity. *Frontiers in Psychology*. 2013;4doi:http://dx.doi.org/10.3389/fpsyg.2013.00757
124. Nasser JA, Gluck ME, Geliebter A. Impulsivity and test meal intake in obese binge eating women. *Appetite*. 2004;43(3)
125. Long S, Meyer C, Leung N, Wallis DJ. Effects of distraction and focused attention on actual and perceived food intake in females with non-clinical eating psychopathology. *Appetite*. 2011;56(2)doi:https://dx.doi.org/10.1016/j.appet.2010.12.018
126. Schulte EM, Sonneville KR, Gearhardt AN. Subjective experiences of highly processed food consumption in individuals with food addiction. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors*. 2019;33(2)doi:https://dx.doi.org/10.1037/adb0000441
127. Vijayvargiya P, Chedid V, Wang XJ, et al. Associations of gastric volumes, ingestive behavior, calorie and volume intake, and fullness in obesity. *American journal of physiology Gastrointestinal and liver physiology*. 2020;319(2)doi:https://dx.doi.org/10.1152/ajpgi.00140.2020

128. Bellisle F, Dalix AM. Cognitive restraint can be offset by distraction, leading to increased meal intake in women. *American Journal of Clinical Nutrition*. 2001;74(2)doi:http://dx.doi.org/10.1093/ajcn/74.2.197
129. Sultson H, Kukk K, Akkermann K. Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. *Appetite*. 2017;116:423-430.
130. Turner RM, Bird SM, Higgins JP. The impact of study size on meta-analyses: examination of underpowered studies in Cochrane reviews. *PloS one*. 2013;8(3):e59202.
131. de Lauzon-Guillain B, Basdevant A, Romon M, et al. Is restrained eating a risk factor for weight gain in a general population? *The American journal of clinical nutrition*. 2006;83(1):132-138.
132. Lluch A, Herbeth B, Mejean L, Siest G. Dietary intakes, eating style and overweight in the Stanislas Family Study. *International journal of Obesity*. 2000;24(11):1493-1499.
133. Johnson F, Pratt M, Wardle J. Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*. 2012;36(5)doi:http://dx.doi.org/10.1038/ijo.2011.156
134. Santos I, Sniehotta FF, Marques MM, Carraça EV, Teixeira PJ. Prevalence of personal weight control attempts in adults: a systematic review and meta-analysis. *Obesity Reviews*. 2017;18(1):32-50.
135. Stubbs RJ, Duarte C, O'Driscoll R, Turicchi J, Michalowska J. Developing evidence-based behavioural strategies to overcome physiological resistance to weight loss in the general population. *Proceedings of the Nutrition Society*. 2019;78(4):576-589.
136. Heatherton TF, Herman CP, Polivy J, King GA, McGree ST. The (mis) measurement of restraint: an analysis of conceptual and psychometric issues. *Journal of abnormal Psychology*. 1988;97(1):19.
137. Mela DJ. Determinants of food choice: relationships with obesity and weight control. *Obesity research*. 2001;9 Suppl 4
138. van Strien T, Cebolla A, Etchemendy E, et al. Emotional eating and food intake after sadness and joy. *Appetite*. 2013;66:20-25.

139. Elfhag K, Rössner S. Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *Obesity reviews*. 2005;6(1):67-85.
140. Karlsson J, Hallgren P, Kral J, Lindroos A-K, Sjöström L, Sullivan M. Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. *Appetite*. 1994;23(1):15-26.
141. Niemeier HM, Phelan S, Fava JL, Wing RR. Internal disinhibition predicts weight regain following weight loss and weight loss maintenance. *Obesity*. 2007;15(10):2485-2494.
142. Chaput J-P, Drapeau V, Hetherington M, Lemieux S, Provencher V, Tremblay A. Psychobiological impact of a progressive weight loss program in obese men. *Physiology & behavior*. 2005;86(1-2):224-232.
143. Foster G, Wadden T, Swain R, Stunkard A, Platte P, Vogt R. The Eating Inventory in obese women: clinical correlates and relationship to weight loss. *International journal of obesity*. 1998;22(8):778-785.
144. Pekkarinen T, Takala I, Mustajoki P. Two year maintenance of weight loss after a VLCD and behavioural therapy for obesity: correlation to the scores of questionnaires measuring eating behaviour. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*. 1996;20(4):332-337.
145. Konttinen H. Emotional eating and obesity in adults: The role of depression, sleep and genes. *Proceedings of the Nutrition Society*. 2020;79(3)doi:http://dx.doi.org/10.1017/S0029665120000166
146. Mantzios M, Wilson JC. Mindfulness, Eating Behaviours, and Obesity: A Review and Reflection on Current Findings. *Current obesity reports*. 2015;4(1)doi:https://dx.doi.org/10.1007/s13679-014-0131-x
147. Long S, Meyer C, Leung N, Wallis DJ. Effects of distraction and focused attention on actual and perceived food intake in females with non-clinical eating psychopathology. *Appetite*. 2011;56(2):350-356.
148. Nichols AL, Maner JK. The good-subject effect: Investigating participant demand characteristics. *The Journal of general psychology*. 2008;135(2):151-166.
149. O'Driscoll R, Turicchi J, Hopkins M, et al. Comparison of the Validity and Generalizability of Machine Learning Algorithms for the Prediction of Energy Expenditure: Validation Study. *JMIR mHealth and uHealth*. 2021;9(8):e23938.

150. O'Driscoll R, Turicchi J, Hopkins M, Horgan GW, Finlayson G, Stubbs JR. Improving energy expenditure estimates from wearable devices: A machine learning approach. *Journal of Sports Sciences*. 2020;38(13):1496-1505.
151. Sanghvi A, Redman LM, Martin CK, Ravussin E, Hall KD. Validation of an inexpensive and accurate mathematical method to measure long-term changes in free-living energy intake. *The American journal of clinical nutrition*. 2015;102(2):353-358.
152. Shook RP, Hand GA, O'Connor DP, et al. Energy intake derived from an energy balance equation, validated activity monitors, and dual X-ray absorptiometry can provide acceptable caloric intake data among young adults. *The Journal of nutrition*. 2018;148(3):490-496.
153. Peng J, Shi P, Qiu J, et al. Clustering Egocentric Images in Passive Dietary Monitoring with Self-Supervised Learning. *arXiv preprint arXiv:220812160*. 2022;
154. Lo FPW, Jobarteh ML, Sun Y, et al. An Intelligent Passive Food Intake Assessment System with Egocentric Cameras. *arXiv preprint arXiv:210503142*. 2021;

Chapter 4. Exploratory analysis of reflective, reactive, and homeostatic eating behaviour traits on weight change during the 18-month NoHoW weight maintenance trial

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4.1. Abstract

Behaviour change interventions for weight management have found varied effect sizes and frequent weight regain after weight loss. There is interest in exploring whether differences in eating behaviour can be used to develop tailored weight management programs. This secondary analysis of an 18-month weight maintenance randomised controlled trial (RCT) aimed to investigate the association between individual variability in weight maintenance success and change in eating behaviour traits (EBT). Data was analysed from the NoHoW trial (Scott et al., 2019), which was designed to measure processes of change after weight loss of ≥5% body weight in the previous year. The sample included 1627 participants (mean age = 44.0 years, SD = 11.9, mean body mass index (BMI) = 29.7 kg/m2, SD = 5.4, gender = 68.7% women/31.3% men). Measurements of weight (kg) and 7 EBTs belonging to domains of reflective, reactive, or homeostatic eating were taken at 4 time points up to 18-months. Increases in measures of 'reactive eating' (binge eating, $p < .001$), decreases in 'reflective eating' (restraint, $p < .001$) and changes in 'homeostatic eating' (unlimited permission to eat, $p < .001$ and reliance on hunger and satiety cues, $p < .05$) were significantly and independently associated with concomitant weight change. Differences in EBT change were observed between participants who lost, maintained, or re-gained weight for all EBTs ($p < .001$) except for one subscale of intuitive eating (eating for physical reasons, $p = .715$). Participants who lost weight ($n = 322$) exhibited lower levels of reactive eating and higher levels of reflective eating than participants who re-gained weight ($n = 668$). EBT domains can identify individuals who need greater support to progress in weight management interventions. Increasing reflective eating and reducing reactive eating may enhance weight management success.

4.2. Introduction

Due to the rising prevalence of obesity, there is a need for more effective weight management options and interventions (Bray et al., 2017). While there are several complex behavioural, biological, cultural, economic, environmental, psychological, and social factors that influence an individual's behaviour and body weight; there has been a large focus on lifestyle modification as a means to address body weight and these complex factors (Wadden & Bray, 2018). Lifestyle modification aims to provide cognitive and behavioural techniques for controlling body weight (Wadden et al., 2020). Obesity Guidelines (Jensen et al., 2014), recommend that comprehensive interventions should last at least 6 months, with programs being delivered by trained health professionals. Interventionists should educate participants in behavioural techniques that are designed to modify physical activity and dietary intake (Jensen et al., 2014).

A systematic review of lifestyle interventions found that face-to-face individual or group treatment leads to an average weight loss of up to 8kg in 6 months (Jensen et al., 2014). Unfortunately, participants involved in lifestyle modification interventions usually re-gain one-third of their lost weight when there is no further intervention (Butryn et al., 2011). Further, only 20% of individuals who are overweight are able to achieve successful long-term weight loss (Wing & Phelan, 2005). However, comprehensive weight maintenance programs have been shown to significantly reduce weight regain. The same systematic review found that 35-60% of participants who received a weight maintenance program sustained a weight loss of ≥5% for more than 2 years at follow-up (Jensen et al., 2014). Another problem with lifestyle modification interventions is the wide variability in response to interventions. Susceptibility to weight gain and success in weight loss differ between individuals and treatments appear to be more successful for certain individuals (Blundell et al., 2005; King et al., 2008). Therefore, the identification of weight loss maintenance and barriers to weight loss could improve the results of long-term weight management interventions and aid in the development of more individualised and tailored programmes. By tailoring treatments to meet individual needs, those who are identified as less likely to succeed could be given more specific or additional treatments over and above the general guidance (Teixeira et al., 2005).

Behavioural research supports the concept of a dual-process model of obesity (Strack & Deutsch, 2004), which suggests that an imbalance of deliberative/reflective and impulsive/reactive processes contribute to eating behaviours promoting a positive energy balance and obesity. Similarly, models of behaviour change also include reactive and reflective components (Dunton et al., 2021; Greaves et al., 2011; Kwasnicka et al., 2016). For example, Greaves et al. (2011) suggest that longer-term weight management involves tension between existing habits and incompatibility of new weight management behaviours. Reflective processes which aid weight management include self-regulation, motivation and managing external influences (Dombrowski et al., 2014; Teixeira et al., 2005; Varkevisser et al., 2019). Reactive processes which involve impulses, emotions and desires that are the result of associative learning and physiological resistance to weight loss are powerful forces that undermine longer-term weight loss and are outside of conscious control (Berthoud, 2011; Blundell & Finlayson, 2004). These processes can be measured using questionnaires that have been designed to measure individual differences in eating behaviour which include constructs that are referred to as eating behaviour traits (EBT). Many EBTs have been developed, but the purpose of the current paper was to assess the use of EBTs with an overarching theoretical framework that considers both reflective and reactive aspects of goal oriented and motivated behaviours. Consequently, dual-process models were used to identify EBTs that measure both reflective and reactive eating behaviours.

The Three Factor Eating Questionnaire (TFEQ) (Stunkard & Messick, 1985), measures three EBTs (dietary restraint, disinhibition, and susceptibility to hunger). Restraint refers to the tendency to restrict food intake to control body weight and shape. Disinhibition measures opportunistic eating and hunger refers to motivation to eat and the degree to which motivation to eat stimulates food intake. The TFEQ has been widely used to measure eating behaviour and all three subscales have emerged as related to body mass index (BMI) and energy intake (Dakin et al., 2023), with disinhibition and restraint, in particular, being recognised as being associated with weight gain, loss and maintenance (Bryant et al., 2012). Successful weight loss is associated with decreased disinhibition (Bryant et al., 2012; Filiatrault et al., 2014; Foster et al., 1998), increased restraint (Chaput et al., 2005; Filiatrault et al., 2014; Foster et al., 1998; Keränen et al., 2009) and decreased susceptibility to hunger

(Karlsson et al., 1994; McGuire et al., 1999). However, some studies have found that increased restraint was associated with weight gain (Hays & Roberts, 2008; Pliner & Saunders, 2008) and hunger was not significantly related to weight loss (Bryant et al., 2012; Foster et al., 1998). The evidence does support a more influential role of disinhibition, restraint and hunger in weight re-gain following weight loss more so than initial weight change. Higher levels of disinhibition and hunger and lower levels of restraint are associated with weight re-gain (Bryant et al., 2012; Cuntz et al., 2001; Fogelholm et al., 1999; McGuire et al., 1999). Overall, disinhibition, restraint and hunger may be indicative markers of adherence to weight management intervention components which are required for weight loss.

Trait binge eating, as measured by the binge eating scale (BES) (Gormally et al., 1982), assesses two components of binge eating: behavioural manifestations of bingeing (e.g. overeating and eating quickly) and the cognitions and emotions that precede or follow a binge (e.g. loss of control and guilt respectively) (Hood et al., 2013). Other studies have found significant decreases in binge eating behaviour after a weight loss programme (Pekkarinen et al., 1996; Wadden et al., 1994). A third of the participants sustained a lower level of binge eating for two years, which was associated with maintained weight loss. Binge eating scores of participants with poor long-term weight control returned to pre-treatment levels after two years (Pekkarinen et al., 1996). These findings imply that assessing binge eating throughout treatment could identify individuals at risk of weight-regain following weight loss.

While the focus of much research on individual variability in eating behaviour during weight loss interventions has been on ‘loss of control’ EBTs, there are also potential EBTs that could be associated with control of eating and weight stability. Intuitive eating, is one such EBT that involves a rejection of calorie restriction diets, avoids labelling food as ‘bad’ and aims to improve gratification with food (Warren et al., 2017). The intuitive eating scale (IES) measures a flexible pattern of eating which involves four subscales: trust and reliance on internal cues of hunger and satiety, eating for physical (rather than emotional) reasons, unlimited permission to eat and choosing foods that support body functions and health (Tylka & Kroon Van Diest, 2013). In a study of 382 participants, those who self-reported that they maintained weight over the past year, had significantly higher total intuitive eating scores

compared to those who gained weight and those who weight-cycled (Tylka et al., 2020). These findings imply that developing strategies of intuitive eating may be useful for individuals who have previously lost weight and are trying to maintain weight loss.

Using knowledge from dual-process models, disinhibition, susceptibility to hunger and binge eating can be viewed as similar to impulsive/reactive processes because they measure implicit and automatic eating behaviour. In contrast, dietary restraint can be viewed as similar to reflective processes because it measures deliberate cognitive reasoning of eating behaviour. Previous literature suggests that higher levels of reactive EBTs and lower levels of reflective EBTs are associated with increased weight gain, weight re-gain and problems with maintaining weight loss (Stubbs et al., 2021; Teixeira et al., 2005; Varkevisser et al., 2019). Intuitive eating, however, is not easily labelled as a reactive or reflective process. The concept of intuitive eating is linked to physiological signals and measures awareness of internal cues and bodily sensations of hunger and satiety. This EBT could be measuring a process that is related to homeostatic processes. Evidence suggests that energy balance is regulated, but this regulation is asymmetric (Stubbs et al., 2023). Therefore, if eating behaviour is part of energy balance regulation, there will be a tendency to overeat more than undereat (Stubbs et al., 2023). Consequently, if intuitive eating is a homeostatic process, it would be expected that individuals showing higher levels of intuitive eating are better able to assess their physiological signals and internal cues, but this does not mean they are less likely to become overweight, due to the nature of this asymmetric regulation. However, research has shown that intuitive eating is associated with weight maintenance and stability. Therefore, engaging in intuitive eating to target homeostatic processes in combination with techniques that engage reactive and/or reflective processes to reduce the effect of an asymmetric energy balance, could be advantageous after intentional weight loss with the aim of improving long-term weight maintenance success (Tylka et al., 2020).

Given the apparent importance of reactive, reflective, and homeostatic processes influencing energy balance behaviour, it is important to study EBTs to understand how they change during weight management attempts and whether any changes are

related to individual variability in weight outcomes. This paper focuses on trait measures of eating behaviour, which are relevant because most of the behaviour change that occurs in weight management attempts relates to eating. Most individuals who lose weight will regain that weight (Elfhag & Rössner, 2005), therefore, there is a need to better understand how weight maintenance can be accomplished. Identifying factors that are associated with weight loss maintenance has important consequences for what strategies should be used in treatment, including specific measures that may be able to detect individuals at risk of weight re-gain (Elfhag & Rössner, 2005).

This study is an EBT focused analysis that uses data which was collected as part of a larger RCT focusing on processes of change. The specific aim of this study was to investigate individual variability in weight change after prior weight loss during an 18-month weight management intervention, and EBT changes across the same 18-month study period. It was hypothesised that changes in EBTs would be associated with weight change such that increased reactive and decreased reflective eating would associate with weight re-gain, whilst decreased reactive eating and increased reflective eating would associate with weight loss. Additionally, it was hypothesised that participants who lost weight during the 18-month study period, would have significantly lower levels of reactive eating (disinhibition, susceptibility to hunger and binge eating) and significantly higher levels of reflective eating (dietary restraint) between 6-18-months than participants who re-gained weight. No specific hypotheses were made for homeostatic eating (intuitive eating), or how change in EBTs would be associated with participants who maintained weight due to a lack of conclusive evidence in previous literature.

4.3. Methods

4.3.1. Participants

Data analysed in the current study were collected as part of the NoHoW RCT (Scott et al., 2019), which took place between March 2017 and September 2019, including 1627 participants. A participant flow diagram can be found in the appendix (B.2). Inclusion criteria were: aged 18 years or older, having a BMI (prior to weight loss) of ≥25kg/m^2, verification of ≥5% of weight loss in the last 12 months and remain 5% below their highest weight, able to use a smartphone and have access to smart phone, computer or tablet with internet access and Wi-Fi, and able to use standing scales for weight measurements and be under the scale weight limit (150kg). Participants were excluded if they were unable to give informed consent, lost weight due to illness or surgery, were pregnant or breastfeeding, were involved in another study that confounded the aims of NoHoW, unable to follow written material, diagnosed with an eating disorder or any condition that may interfere with physical activity, recent diagnosis of type 1 diabetes, had planned extensive travel (> 4 weeks) and were living in the same household as an existing participant in the trial. Participants were recruited from the UK, Denmark and Portugal and centre-specific recruitment strategies were used for 12 months which included commercial and other weight management services. All participants were guided to the country-specific recruitment websites (http://uk.nohow.eu; http://dk.nohow.eu; http://nohow.fmh.ulisboa.pt) and completed an online eligibility survey using Qualtrics. Eligible participants were contacted for a telephone screening interview and were provided with further study information. The participants were also asked to provide documented verification that they had achieved significant weight loss of ≥5% during the previous 12 months. The eligible participants were then invited to a clinical investigation day (CID) where the research staff obtained informed consent before the participants were randomised to an intervention arm.

4.3.2. Ethics

The NoHoW trial was registered with the ISRCTN registry (ISRCTN88405328, see B.1). Ethical approval was granted by each institutional ethics committee before study commencement at each centre. The protocol complied with relevant EC

legislation, international conventions and declarations relating to ethical research practices (World Medical Association, 2001).

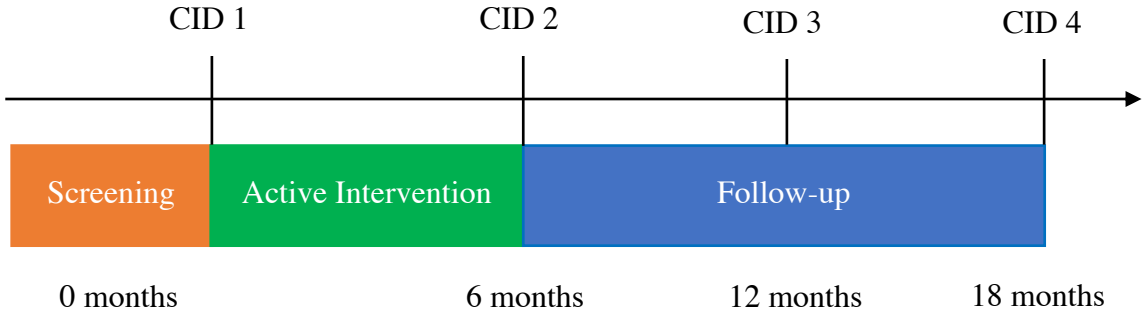
4.3.3. Design

The current study was a secondary analysis using data from the NoHoW trial, an EU Horizon 2020 funded research and innovation programme. The NoHoW trial was a three-centre (University of Leeds (UK), Frederiksberg and Bispebjerg Hospital (Denmark) and University of Lisbon (Portugal)) 2×2 factorial, randomised, single-blind, controlled trial testing the proof-of-concept of a digital toolkit for weight loss maintenance. The intervention design was grounded on a logic model based on dual process models and underpinned by behaviour change theories (Self-Determination Theory and Self-Regulation Theory). For a more detailed description of the logic models and for more information on the main NoHoW trial protocol, see Marques et al. (2021), Scott et al. (2019), and Stubbs et al. (2021). The trial took place over an 18-month period, with measures taken at baseline (CID 1), 6 months (CID 2), 12 months (CID 3) and 18 months (CID 4, see Figure 4.1). Participants were randomised into one of 4 intervention arms (1) active control arm (involving a generic toolkit content, self-weighing, and activity trackers, only); (2) self-regulation and motivation arm, self-weighing, and activity trackers; (3) emotion regulation arm, self-weighing, and activity trackers; and (4) combined motivation/self-regulation and emotion regulation arm, self-weighing, and activity trackers.

4.3.4. Power Calculations and Sample Size Estimation

Power calculations were based on the primary outcome of weight change. To detect a difference between the intervention arms of >1.5kg bodyweight, this results in a Cohen's D value of 0.25 (Dombrowski et al., 2014). Comparing more than 2 groups means a sample size of 250 participants per study arm is needed for 80% power. A 38% drop-out rate was assumed (Larsen et al., 2010a), meaning a sample size 1600 (533 per centre) was needed to achieve a sample of 1002 (334 per centre, ~250 per study arm) participants at 12 months.

Figure 4.1. Trial Design



4.3.5. Measures and Materials

Age, gender, and height (cm) were recorded at baseline. Weight (kg) was measured on calibrated scales at 4 timepoints (CID1-4). All physical measurements were taken with participants wearing light clothing and after an overnight fast by trained research staff. Percentage weight change was calculated as (CID 4 kg – CID 1 kg) / CID 1 kg x 100. Participants were assigned to one of three weight categories according to percentage weight change attained using known criteria (Donnelly et al., 2009; Stevens et al., 2006): >-3.0% weight loss (participants who lost weight), between -2.9% and +2.9% (participants who maintained weight) and >+3.0% weight re-gain (participants who regained weight). Psychometric questionnaires of eating behaviour were used to measure seven EBTs, which were measured at all four timepoints (CID1-4). The questionnaires included in this study are presented in Table 4.1. English, Danish, and Portuguese versions of all questionnaires were used as required in the appropriate countries. Cronbach's alphas were calculated at CID1 and were all >0.6, (binge eating = 0.87, intuitive eating = 0.78, disinhibition = 0.78, susceptibility to hunger = 0.79, restraint = 0.74) demonstrating acceptable internal reliability of scales (Tavakol & Dennick, 2011).

Table 4.1. List of domains and Questionnaires used in the study

Domain	Questionnaire	Subscales	Reference Paper
Reactive Eating	Three Factor Eating Questionnaire (TFEQ)	Disinhibition, Susceptibility to Hunger	Stunkard and Messick (1985)

	Binge Eating Scale (BES)		Gormally et al. (1982)
Reflective Eating	Three Factor Eating Questionnaire (TFEQ)	Dietary Restraint	Stunkard and Messick (1985)
Homeostatic Eating	Intuitive Eating Scale (IES)	Reliance on Hunger and Satiety Cues (RHSC), Unlimited Permission to eat (UPTE), Eating for Physical Rather Than Emotional Reasons (EPR)	Tylka and Kroon Van Diest (2013)

4.3.6. Procedure

Detailed information on the procedure of the NoHoW trial are described in Scott et al. (2019), so are presented here only in brief. At each CID, the participant's weight (kg) was measured by a researcher after an overnight fast in light clothing. The participants were also required to complete the EBT questionnaires shown in Table 4.1 and other questionnaires not relevant to this study at each CID. Participants completed the questionnaires at home using Qualtrics (Qualtrics, Provo, UT), and were given 1 week to complete the survey. All questionnaires took approximately 1.5 hours to complete, however, the measures relevant to this study took approximately 20 minutes to complete. The questionnaires were presented to the participants in a random order.

4.4. Results

4.4.1. Data Analysis

Statistical analyses were performed with SPSS version 27 (IBM Corp, 2020). Raw descriptives, reference data and an additional comparison for all outcome variables are presented in Table 4.2. EBT variables and BMI were visually inspected for normality using histograms and skewness and kurtosis for all measures were within an acceptable range (Kline, 2015). Extreme outliers were identified as above 3rd quartile + 3*interquartile range [IQR] and below 1st quartile – 3*IQR). Using this approach, no extreme outliers were identified. Missing values were imputed using multiple imputation with 5 imputations as part of an intention-to-treat analysis. Pearson correlations were used to test for associations between baseline EBT scores and weight change (CID1 to CID4). To examine whether EBT change between CID1-4 were associated with percent weight change between CID1-4, a stepwise multiple regression analyses were performed. Baseline BMI was entered at step 1, followed by each EBT change score (calculated as CID4 – CID1), together to predict the outcome (percentage weight change). As a stepwise model was used, non-significant associations were removed from the model so that only significant predictors were retained and are presented in Table 4.4.

To examine changes in EBTs between participants who lost, maintained, and re-gained weight, linear mixed models (LMM) for repeated measures were utilised. Firstly, data were entered into separate unadjusted LMM models for each EBT and weight category with time (CID 1, 2, 3, 4) as the within-subject factor and weight category (participants who lost, maintained, or re-gained weight between CID1-4) as the between-subject factor. Next, an adjusted LMM model was performed, with age, baseline BMI and prior weight loss as covariates included in the model. The model also included study arm (control, stress/emotion, self-regulation/emotion, stress/emotion + self-regulation + motivation), gender, and country of residence (UK, Denmark, or Portugal) as between-subjects categorical factors because the differences between the categories were of interest. The model specified was full factorial, with polynomial contrasts and the sum of squares was type III. As there were only 4 timepoints, far apart and with variation in the intervention content

between them, the model was fitted with an unstructured covariance matrix. Adjusted models are reported in the appendix (B.4 – B.10). Significant effects ($p < .05$) were examined with Bonferroni corrected post hoc tests. Mauchly's test of Sphericity was used to indicate whether the assumption of sphericity had been violated. Where data violated the assumption of sphericity ($p < .05$ and $\varepsilon > .75$), the Huynh-Feldt correction was applied to the residual degrees of freedom.

Table 4.2. Descriptives, normative and reference data (given as means and standard deviations) as reported in population and/or validation studies for EBTs and BMI

		Reference	**Other comparison**	**DIOGENES**	**Current sample (at CID1)**		
Scale	Subscale	Mean (SD)	Mean (SD)	Mean (SD)	Mean (SD)	Skew	Kurtosis
IES	RHSC	3.62 (0.71)[a]	3.32 (0.82)[e]	-	**2.84 (0.87)**	-0.08	-0.37
	EPR	3.48 (0.88)[a]	3.35 (0.71)[e]	-	**2.99 (0.59)**	-0.01	0.33
	UPTE	3.58 (0.78)[a]	3.34 (0.92)[e]	-	**3.05 (0.61)**	-0.20	0.32
TFEQ	Disinhibition	10.00 (5.90)[b]	9.90 (3.60)[f]	8.77 (3.63)[g]	**8.75 (3.43)**	0.68	-0.81
	Restraint	10.50 (6.20)[b]	11.20 (3.40)[f]	10.91 (3.20)[g]	**11.07 (3.43)**	-0.30	-0.24
	Susceptibility to hunger	7.10 (4.10)[b]	6.90 (3.60)[f]	6.10 (3.58)[g]	**5.78 (3.47)**	0.40	-0.63
BES		20.80 (8.40)[c]	14.90 (8.90)[f]	-	**12.07 (7.46)**	0.64	0.18
BMI		27.60[d]	31.00 (6.40)[f]	30.54 (6.11)[g]	**29.66 (5.35)**	1.24	2.51

Note: [a] = taken from study 1 (Tylka & Kroon Van Diest, 2013), involving 878 participants (487 women, 391 men). [b] = 78 members of a weight reduction program (60 women, 18 men), [c] = taken from sample 1 (Gormally et al., 1982); a population with overweight seeking obesity treatment (65 women), [d] = taken from (Health Survey for England, 2019), [e] = taken from a sample of 259 undergraduate students and the general public (unpublished data), [f] = 548 participants involved in a multicomponent commercial weight management programme (Slimming World ™), randomly allocated to the control condition of an intervention, [g] = true baseline measures of 956 participants involved in a (Larsen et al., 2010b). IES = Intuitive Eating Scale, RHSC = Reliance on hunger and satiety, EPR = Eating for physical reasons, UPTE = Unlimited permission to eat, TFEQ = Three Factor Eating Questionnaire, BES = Binge Eating Scale, BMI = Body Mass Inde

4.4.2. Preliminary analysis

The sample consisted of healthy women and men with a gender split of 68.7% women/ 31.3% men. Mean (SD) age of the participants was 44.0 (11.9) years. Mean (SD) BMI at baseline was 29.7 (5.4) kg/m^2. Details on sociodemographic characteristics (country of residence, educational, marital and employment status) can be found in Table 4.3. Sample characteristics at baseline for each study arm are presented in the appendix (B.3). There were no significant differences at baseline between study arm conditions for age, BMI, or any of the EBT variables except for susceptibility to hunger. At baseline, there were significant differences in susceptibility to hunger scores between study arm conditions ($F(3,1610) = 3.37$, $p = .018$). Post-hoc analyses revealed that the stress/emotion condition ($M = 6.2$, $SD = 3.5$) had significantly higher hunger scores at baseline compared to the stress/emotion, self-regulation, and motivation condition ($M = 5.4$, $SD = 3.4$). Additionally, the self-regulation/emotion condition ($M = 6.0$, $SD = 3.5$) also had higher susceptibility to hunger scores at baseline compared to stress/emotion, self-regulation, and motivation condition ($M = 5.4$, $SD = 3.4$).

Table 4.3. Sample Description (n = 1627)

Descriptive	**(SD)**
Gender:	
Women (n)	1117
Men (n)	510
Age in years	44 (11.9)
Country of residence:	
UK	34.1%
Denmark	32.9%
Portugal	32.9%
Self-reported ethnicity:	
White European	87.8%
Asian	1.8%
Black African & Black other	1.1%
Other Ethnic groups	0.3%
Unspecified	9.0%
Education:	
Further Education	36.4%
Degree	27.5%
Secondary School Education	16.8%
Masters'	8.8%
PhD	3.1%
Other	5.4%

Employment:	
Full-time/Part-time	74.7%
Retired	7.9%
Student	6.1%
Unemployed	2.8%
Other	6.6%
Weight at baseline	84.8kg (17.3)
Height	168.7cm (15.9)
BMI at baseline	29.7kg/m^2 (5.4)
Weight Category:	
Weight Loss	19.8%
Weight Maintenance	38.0%
Weight Re-gain	41.1%

4.4.3. Association between baseline EBT scores and weight change

For the whole sample, there were no significant correlations observed between all 7 baseline EBTs and subsequent 6-month, 12-month or 18-month weight change.

4.4.4. Changes in EBT (from CID1-4) scores as predictors of weight change

Changes in six out of the seven EBTs were significantly correlated with concomitant weight change. Change in disinhibition ($r = 0.20$, $p < 0.01$), susceptibility to hunger ($r = 0.18$, $p < 0.01$), binge eating, ($r = 0.34$, $p < 0.01$) and unlimited permission to eat ($r = 0.17$, $p < 0.01$) were associated with weight increase. Change in restraint ($r = -0.21$, $p < 0.01$) and reliance on hunger and satiety cues ($r = -0.17$, $p < 0.01$) were associated with weight loss. An increase in disinhibition, binge eating and unlimited permission to eat, and a decrease in restraint and reliance on hunger and satiety cues were associated with weight re-gain.

Changes in EBT scores were entered into a stepwise multiple regression to determine the individual association between each of the EBTs and weight change when accounting for all other EBTs as predictors of weight change. The analysis revealed that binge eating, restraint, unlimited permission to eat and reliance on hunger and satiety cues were all significantly and independently associated with weight change. A decrease in restraint and reliance on hunger and satiety cues and an increase in binge eating and unlimited permission to eat were associated with weight re-gain, whilst an increase in restraint and reliance on hunger and satiety

cues and a decrease in binge eating and unlimited permission to eat and were associated with weight loss (see Table 4.4).

Table 4.4. Stepwise multiple regression predicted change in weight from CID1-4 with change in EBT scores from CID1-4

		B	SE	β	t	*p*	95% CI (Lower)	95% CI (Upper)	F	R^2	ΔR^2
1	(Constant)	5.73	.99		5.81	<.001	3.79	7.66	16.94***	.01	.01
	CID1 BMI	-.13	.03	-.10	-4.12	<.001	-.20	-.07			
2	(Constant)	5.08	.93		5.46	<.001	3.25	6.90	110.51***	.12	.12
	CID1 BMI	-.11	.03	-.08	-3.52	<.001	-.17	-.05			
	BES change	.39	.03	.33	14.21	<.001	.33	.44			
3	(Constant)	4.70	.92		5.12	<.001	2.90	6.50	93.55***	.15	.15
	CID1 BMI	-.10	.03	-.08	-3.33	.001	-.16	-.04			
	BES change	.36	.03	.31	13.40	<.001	.31	.42			
	TFEQR change	-.38	.05	-.17	-7.25	<.001	-.48	-.28			
4	(Constant)	4.59	.91		5.03	<.001	2.80	6.38	75.41***	.16	.16
	CID1 BMI	-.10	.03	-.08	-3.33	.001	-.16	-.04			
	BES change	.35	.03	.30	13.02	<.001	.30	.40			
	TFEQR change	-.33	.05	-.15	-6.28	<.001	-.44	-.23			
	IES_UPTE change	1.10	.26	.10	4.25	<.001	.59	1.61			
5	(Constant)	4.64	.91		5.09	<.001	2.85	6.43	61.47***	.16	.16
	CID BMI	-.10	.03	-.08	-3.34	.001	-.16	-.04			
	BES change	.33	.03	.28	11.54	<.001	.27	.39			
	TFEQR change	-.32	.05	-.14	-6.12	<.001	-.43	-.22			
	IES_UPTE change	1.13	.26	.10	4.37	<.001	.62	1.64			
	IES_RHSC change	-.44	.20	-.05	-2.23	.026	-.83	-.05			

Note: *** p < .001, IES = Intuitive Eating Scale, RHSC = Reliance on hunger and satiety, UPTE = Unlimited permission to eat, TFEQR = Restraint, BES = Binge Eating Scale, BMI = Body Mass Index

4.4.5. Individual variability in weight change

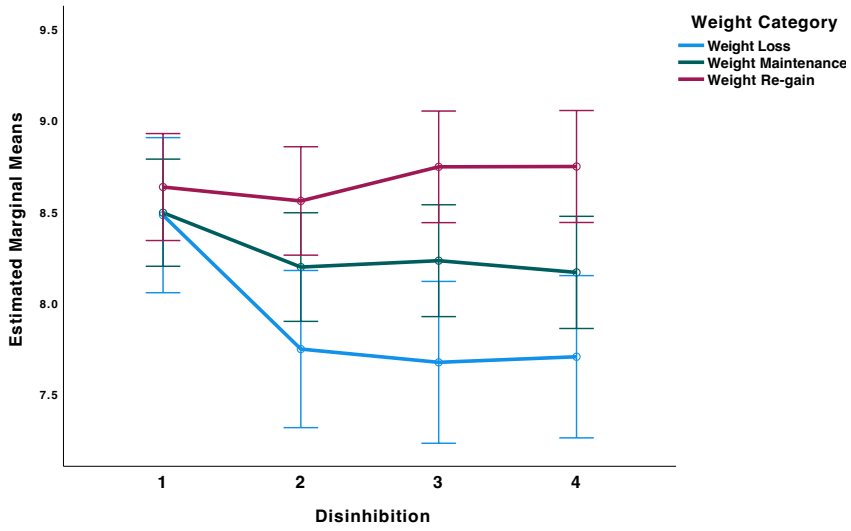
The mean weight change for all participants in the sample was +1.4kg (SD = 6.2). For participants who lost weight, mean weight loss was 6.9kg (SD = 5.0), while mean weight change for participants who maintained weight was +0.2kg (SD = 1.6) and

mean weight gain for participants who re-gained weight was 6.4kg (SD = 4.2). Among the 3 weight groups there were no statistically significant differences at baseline for measures of reactive, reflective, or homeostatic eating (p's all > .05). There were no main effects of study arm for any EBT and there were no significant study arm x time interactions for any EBT (p's all > .05).

4.4.6. Reactive eating

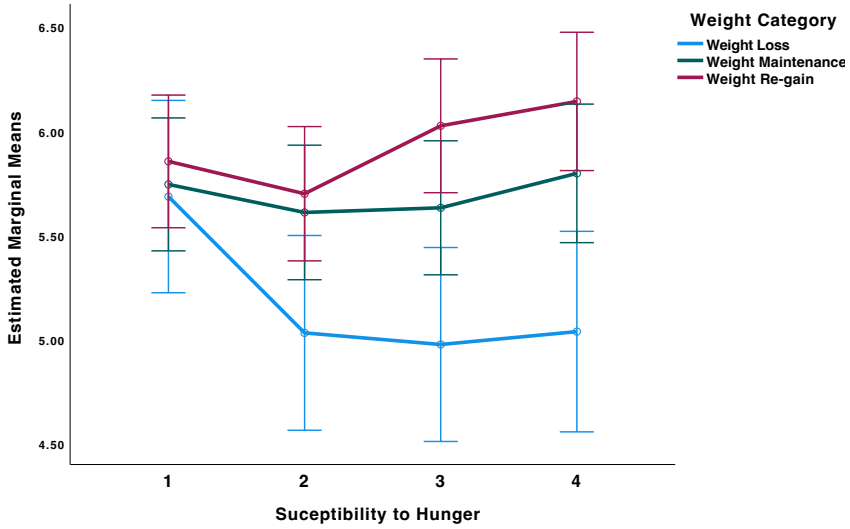
There was no overall effect of time for disinhibition ($F(2.99, 4565.21) = 0.34$, $p = .795$). There was an overall effect of weight category ($F(2, 1526) = 5.55$, $p = .004$) and there was a significant time x weight category interaction ($F(5.98, 4565.21) = 4.54$, $p < .001$, $\eta^2 = 0.01$) for disinhibition. Pairwise comparisons showed that at CID2, participants who lost weight over 18-months had significantly lower disinhibition scores than participants who re-gained weight, and this continued to CID4 (see Figure 4.2 and B.4). There was no overall effect of time for hunger ($F(3.00, 4561.89) = 0.34$, $p = .800$). There was a main effect of weight category ($F(2, 1526) = 4.49$, $p = .011$) and a significant time x weight category interaction ($F(5.98, 4561.89) = 3.96$ $p < .001$, $\eta^2 = 0.01$) for hunger. At CID3 and CID4, participants who lost weight at 18-months had significantly lower hunger levels than participants who re-gained weight (See Figure 4.3 and B.5). There was no overall effect of time ($F(3.00, 4578.00) = 0.56$, $p = .643$) for binge eating. There was a significant effect of weight category ($F(2, 1526) = 12.31$, $p < .001$) and a significant time x by weight category interaction for binge eating ($F(6.00, 4578.00) = 13.89$, $p < .001$, $\eta^2 = 0.02$). At CID 2, 3 and 4, participants who lost weight over 18-months had significantly lower levels of binge eating than participants who re-gained weight. Participants who lost weight over 18-months also had significantly lower levels of binge eating at CID 3 and 4 than participants who maintained weight (see Figure 4.4 and B.6).

Figure 4.2. Change in disinhibition estimated marginal means according to weight category



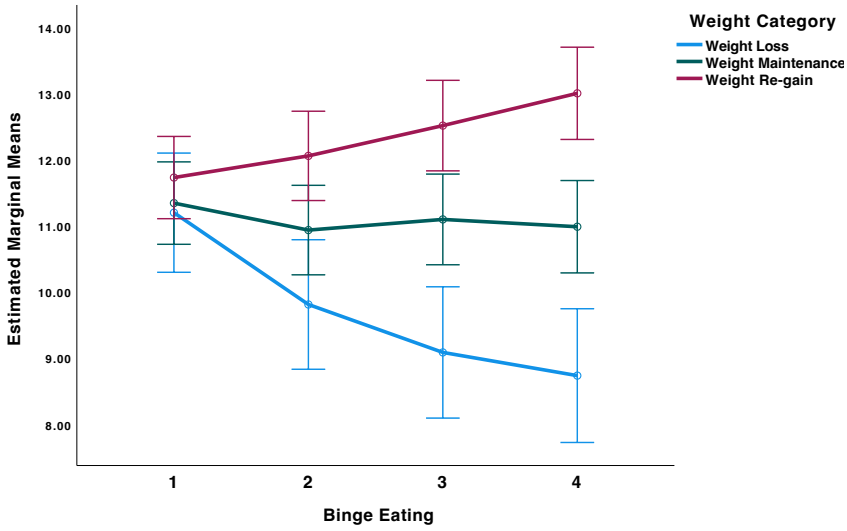
Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

Figure. 4.3. Change in susceptibility to hunger estimated marginal means according to weight category



Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

Figure 4.4. Change in binge eating estimated marginal means according to weight category

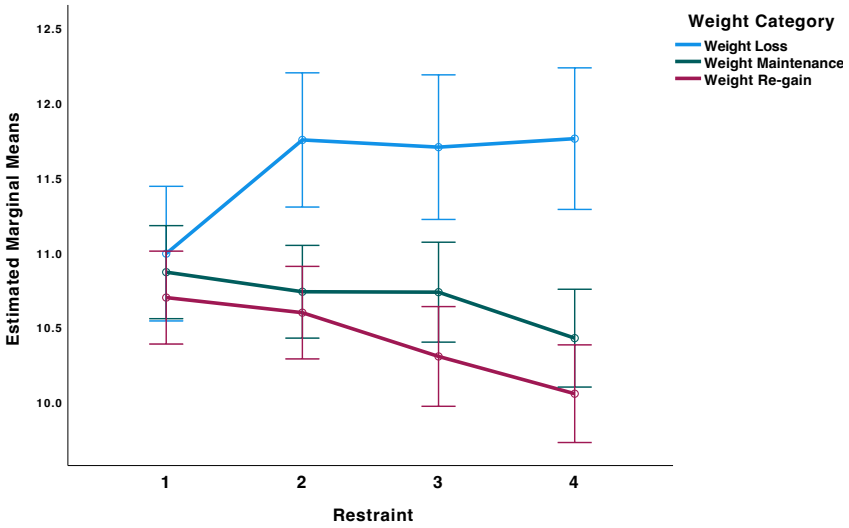


Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

4.4.7. Reflective Eating

There was an overall effect of time for restraint ($F(3.00, 4578.00) = 3.79$, $p = .010$), an overall effect of weight category ($F(2, 1526) = 11.37$, $p < .001$) and a significant time x weight category interaction ($F(6.00, 4578.00) = 6.94$, $p < .001$, $\eta^2 = 0.01$) for restraint. At CID 2, 3 and 4, participants who lost weight over 18-months had significantly higher restraint scores than participants who maintained and re-gained weight (See Figure 4.5 and B.7).

Figure 4.5. Change in restraint estimated marginal means according to weight category

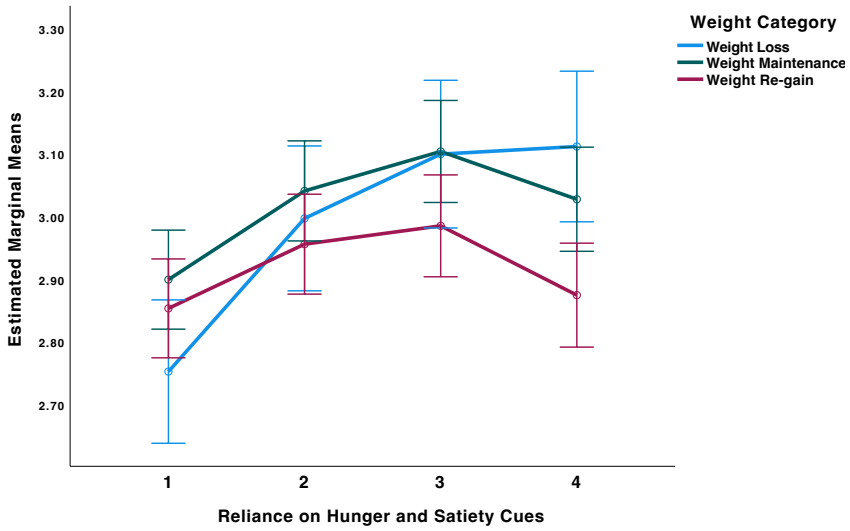


Note. Error bars indicate SE. Covariates appearing in the model are evaluated in the model include baseline BMI, prior weight loss and age. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

4.4.8. Homeostatic Eating

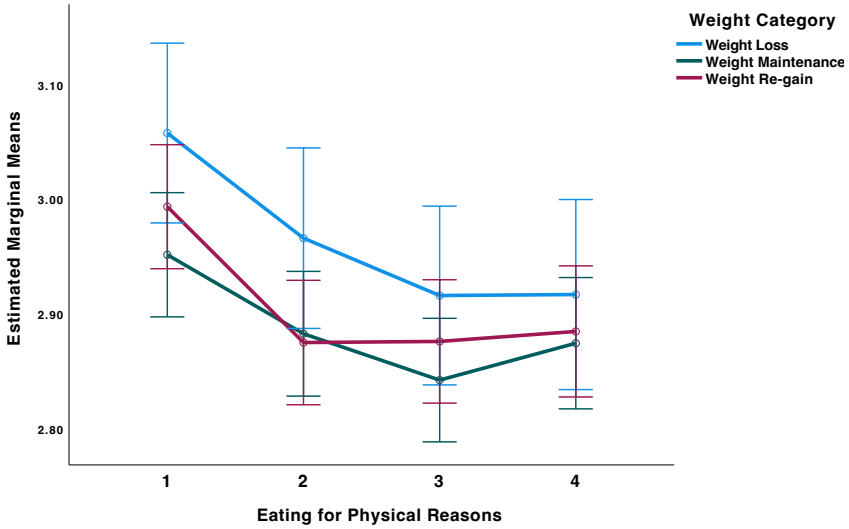
There was no overall effect of time ($F(3.00, 4578.00) = 0.61$, $p = .611$) or weight category ($F(2, 1526) = 2.38$, $p = .093$) for reliance on hunger and satiety cues. There was a significant time x weight category interaction ($F(6.00, 4578.00) = 4.60$, $p < .001$, $\eta^2 = 0.01$) for reliance on hunger and satiety cues. At CID 4, participants who lost and maintained weight over 18-months had significantly higher levels of reliance on hunger and satiety cues than participants who re-gained weight (See Figure 4.6 and B.8). There was no overall effect of time ($F(3, 4578) = 2.04$, $p = .105$), weight category, ($F(2, 1526) = 2.04$, $p = .130$) or time x weight category interaction ($F(6, 4578) = 0.62$, $p = .715$) for eating for physical reasons (See Figure 4.7 and B.9). There was no overall effect of time for unlimited permission to eat ($F(3.00, 4578.00) = 2.00$, $p = .112$). There was an overall effect of weight category ($F(2, 1526) = 5.99$, $p = .003$) and a significant time x weight category interaction ($F(6.00, 4578.00) = 3.72$, $p = .001$, $\eta^2 = 0.01$) for unlimited permission to eat. At CID 3 and 4, participants who lost weight at 18-months had significantly lower unlimited permission to eat levels than participants who maintained and re-gained weight (See Figure 4.8 and B.10).

Figure 4.6. Reliance on hunger and satiety cues estimated marginal means according to weight category



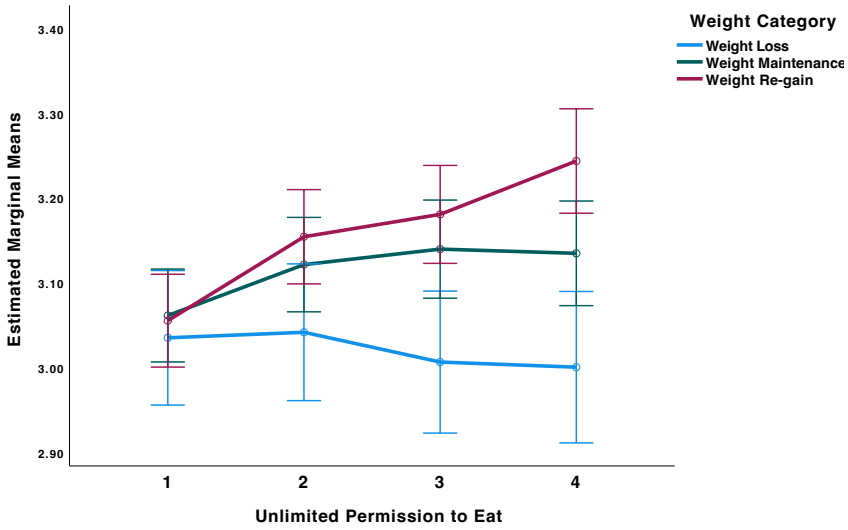
Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

Figure 4.7. Eating for physical reasons estimated marginal means according to weight category



Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

Figure 4.8. Unlimited permission to eat estimated marginal means according to weight category



Note. Error bars indicate SE. Covariates appearing in the model include age, prior weight loss and baseline BMI. Country of Residence, Study Arm and Gender are included in the model as fixed factors.

4.5. Discussion

The current study investigated the association between individual variability in weight change and concomitant changes in EBTs (reflective, reactive, or homeostatic) over an 18-month weight loss maintenance intervention. It was hypothesised that changes in EBTs would associate with weight change such that increased reactive and decreased reflective eating would associate with weight re-gain. It was also hypothesised that participants who lost weight would have significantly lower levels of reactive eating (disinhibition, susceptibility to hunger and binge eating) and significantly higher levels of reflective eating (dietary restraint) than participants who re-gained weight. The results supported the hypotheses, whereby, 6 out of 7 EBTs were significantly correlated with weight change. The results also showed that changes measures of reactive eating (binge eating), reflective eating (restraint) and homeostatic eating (unlimited permission to eat and reliance on hunger and satiety cues) were significantly and independently associated with weight change over 18-months, where the associations with reactive and reflective eating were in the expected direction. Additionally, the LMM's showed that there were differences in EBTs scores across CID2-4 between participants who lost, maintained, and re-gained weight in all EBTs except eating for physical reasons. Participants who lost weight had significantly lower levels of reactive EBTs and significantly higher levels of reflective EBTs from CID2 and continuing to CID4 than participants who re-gained weight. All effect sizes were small, with binge eating having the largest effect size for the interaction between EBTs and weight group. These results suggest that EBTs are associated with weight change and monitoring EBT changes could be advantageous during weight management interventions with the aim of improving weight loss maintenance.

Regarding homeostatic EBTs, there were no differences between weight groups at any CID for eating for physical reasons. However, the results showed that at CID 4, participants who lost and maintained weight had significantly higher levels of reliance on hunger and satiety cues than participants who re-gained weight. This finding is consistent with Tylka et al. (2020) and may suggest that individuals who are successful in maintaining weight loss and those who lost further weight, were able to increase their ability to rely on their body's internal cues, thus, promoting intuitive

eating could be useful in weight management. Interestingly, also consistent with previous findings (Tylka et al., 2020), participants who lost weight had significantly lower levels of unlimited permission to eat between CID3-4 than participants who maintained weight. Unlimited permission to eat appears to be behaving as the reverse of dietary restraint, which could be expected of a homeostatic EBT under conditions of weight loss and maintenance. These results indicate that relying on bodily internal or intuitive signals during a period of weight loss may not be effective because this is a period where physiological hunger may be elevated compared to weight maintenance. However, during weight maintenance, intuitive eating could be promoted.

The finding that participants who lost weight tended to show reductions in reactive EBTs and increases in reflective EBTs, whereas participants who maintained or re-gained weight tended to show increases in reactive and decreases in reflective EBTs are consistent with previous studies that have examined individual variability in weight change (Bryant et al., 2012; Karlsson et al., 1994; Keränen et al., 2011; McGuire et al., 1999; Pekkarinen et al., 1996; Teixeira et al., 2005; Varkevisser et al., 2019). Studies have found that participants who lost or maintained weight had significantly higher levels of restraint than participants who re-gained weight (Bryant et al., 2012; Karlsson et al., 1994; Keränen et al., 2011; McGuire et al., 1999). The construct of restraint can be split into two dimensions, flexible and rigid restraint. Rigid restraint is associated with an all-or-nothing approach to dieting, whilst flexible restraint is characterised by a more graduated approach to dieting where no foods are avoided (Westenhoefer et al., 2013). Research has found that flexible restraint is associated with greater weight loss and better weight maintenance than rigid restraint (Westenhoefer et al., 2013). In the current study, the analysis did not distinguish between flexible and rigid restraint, and therefore it is suggested that these dimensions should be considered in future studies.

There is also evidence that disinhibition, hunger, and binge eating are significantly higher in people who re-gain weight compared to those who maintain or lose weight (McGuire et al., 1999; Pekkarinen et al., 1996). However, no significant interactions for disinhibition and hunger have been found (Bryant et al., 2012) and one study found a trend for TFEQ scores, especially disinhibition, returning to pre-treatment

levels after 24 months of dieting (Karlsson et al., 1994). Taken together, there is good agreement from previous literature that EBTs are associated with weight change and weight re-gain (Elfhag & Rössner, 2005; Teixeira et al., 2005; Varkevisser et al., 2019). The findings from the current study offer a unique contribution to the literature due to the analysis of longitudinal change scores for EBTs related to reflective and reactive constructs which have not always been shown to be important in influencing changes in energy balance status. However, further research is required to better understand how EBT change in individuals who lose, maintain, or re-gain weight and whether participants who lose weight can maintain reduced reactive EBT levels and increased reflective EBT levels post 18-months.

A limitation of many previous studies has been the absence of a control group (Bryant et al., 2012; Chaput et al., 2005; Foster et al., 1998; Karlsson et al., 1994; Pekkarinen et al., 1996), which has led researchers to attribute the changes in EBTs to the effect of a specific intervention. The current study did include a control arm and found no evidence of an intervention effect (no significant main effects of study arm or time x study arm interactions), meaning EBT changes were not significantly different across any of the 4 study arms in the NoHoW trial. This lack of intervention effect raises questions about whether the intervention was simply ineffective and as such the results demonstrate processes of change that are independent of the intervention content. Possibly, the intervention did not target the NoHoW logic models (self-regulation vs emotion regulation in long-term weight management) sufficiently. There are also questions about whether these changes are casual or corollaries of weight outcomes. However, understanding the direction of this association is complex and limited by the study design. It is possible that inferences can be made about direction of effect using cross-sectional data. For example, a previous study tested bidirectional longitudinal associations between the Five Factor Model of personality domains and BMI with casual inferences using within-person correlations (Arumäe et al., 2022). However, the authors identify that their results cannot rule out the possibility of unmeasured time-dependent factors or unknown third variables influencing the associations instead of the causal process. It would still be useful to attempt an approach like the one mentioned above; therefore, a future paper will explore the direction of EBT x weight change effects.

Baseline EBT scores were not correlated with subsequent 18-month weight change in this sample. This is consistent with previous findings (Björvell et al., 1994; Bryant et al., 2012; Filiatrault et al., 2014; Foster et al., 1998; Karlsson et al., 1994; Pekkarinen et al., 1996), but not all (McGuire et al., 1999), suggesting that baseline EBT levels are not useful predictors of weight change. In contrast, it is the processes of change during weight management interventions that are important. Therefore, EBTs are more useful for monitoring progress in behavioural treatments. During weight maintenance, measures of reactive, reflective, and homeostatic eating could be used as indicative measures of likelihood of adhering or discontinuing behavioural treatments. This may help identify individuals at risk of relapse and help triage participants for individually relevant programme components (Björvell et al., 1986). Importantly, it must be considered whether the current study has true baseline measures. It is possible that prior weight loss within 12-months influenced the outcomes of this study. Participants may have entered the study with lower-than-normal reactive EBTs and higher-than-normal reflective EBTs compared to other weight loss interventions, due to their previous weight loss success (all ≥5% body weight). This could then enable them to be more equipped for another journey of weight maintenance and weight loss. It is also possible that the results are reflecting a rebound effect where lower/higher than average EBT levels are returning to normal during the study. The current study showed that EBT levels were not significantly different at baseline between weight losers, maintainers, and re-gainers. Overall, the evidence suggests that in this sample, participants who lost weight do not seem to be any different to participants who re-gained weight, in terms of their ability to cope with another weight loss journey.

To understand whether the current results are displaying a rebound effect, baseline EBT levels can be compared to normative and reference data. The current sample's baseline binge eating, disinhibition and hunger levels were lower than that of reference data and restraint scores were slightly higher than reference data (see Table 4.2). This suggests that participants are starting the study with lower-than-average reactive EBT levels and could indicate that participants may rebound back to average levels after the intervention. However, when comparing to other data including a control condition of a weight management programme (see other comparison, Table 4.2) and a sample which does have a true baseline measure (a

measure before initial weight loss, see DIOGENES, Table 2), reactive and reflective EBTs were more equivalent. Furthermore, restraint levels were higher in the comparison study compared to the current study. Baseline levels of intuitive eating were all below reference data (see Table 4.2). However, these values are taken from a population of college students, not primarily middle-aged treatment-seeking participants, which means reference data may not be reflective of individuals engaged in a weight management program. Overall, baseline levels of EBTs, particularly reactive and reflective EBTs, are comparable to individuals with overweight and obesity who are seeking weight loss treatment. Therefore, it is unlikely that the preceding events of the study influenced the main outcomes such that a rebound effect occurred.

The effects of behaviour change interventions for weight loss and weight loss maintenance are currently modest (Varkevisser et al., 2019), and there are many factors that hinder self-management of eating and physical activity (Elfhag & Rössner, 2005). These factors encompass physiological resistance to weight loss, gradual compensatory adjustments in eating and physical activity, as well as reactive processes associated with stress, emotions, rewards, and desires that fulfil psychological needs (Elfhag & Rössner, 2005; King et al., 2008; Sumithran & Proietto, 2013). To enhance outcomes, it is crucial to better match evidence-based intervention content with quantitatively tracked energy balance behaviours to the specific requirements of individuals. By improving the objective and longitudinal monitoring of energy intake and expenditure over time, a quantitative framework can be established to comprehend the dynamics of behaviour change, the mechanisms of action of behaviour change interventions, and user engagement with intervention components. This, in turn, has the potential to enhance the design and evaluation of weight management interventions. Based on the findings of the current paper and the main NoHoW trial (Stubbs et al., 2021), it is recommended that sustained weight management interventions should focus more on aligning the mechanisms of behaviour change interventions with the compensatory energy balance behaviours that undermine them. In the context of this paper, compensatory energy balance behaviours are linked to reflective and reactive aspects of goal-oriented motivated behaviour, which can be assessed through measurements of EBTs. Thus, improving

understanding of changes in eating behaviour and individual differences in EBTs may enhance weight management interventions.

Whilst the current paper focused solely on EBTs, previous research using data from three countries identified an association between general-self regulation traits and EBTs that may represent a barrier to weight maintenance (Sainsbury et al., 2019). In this study, emotion regulation difficulties were associated with increased weight re-gain. Attribution to emotional reasons were associated with binge eating and using more self-regulatory strategies in weight loss with fewer dietary strategies in weight maintenance. Consequently, there may be an interaction between self-regulation and EBTs that influences weight loss outcomes. For example, poor emotional regulation could result in overeating in response to emotions (emotional eating) (Evers et al., 2010), which is associated with poorer weight outcomes (Canetti et al., 2009). Therefore, it may be useful for future studies to examine the relationship between self-regulation and EBTs on weight change.

Strengths of the current study include its large sample size, prospective design, good adherence, and long assessment and follow-up period. Previous studies have used much shorter intervention lengths and/or no follow-up period (Bryant et al., 2012; Chaput et al., 2005; Filiatrault et al., 2014; Foster et al., 1998). The current study used a 6-month intervention period and another 12-months of follow-up which enabled a more in-depth examination of long-term changes in EBTs and weight. In all the significant interactions (all EBTs except EPR), differences in EBTs between participants who lost, maintained, and re-gained weight were found after the active intervention period (6 months), but were also maintained and sometimes further increased/decreased in the follow-up period (6-18 months) where no active intervention was used. This suggests that participants can maintain changes in EBTs without the assistance that an active intervention provides. Additionally, EBT change, and weight change were tested across 4 time points, whereas many previous studies test only pre and post intervention (Chaput et al., 2005; Filiatrault et al., 2014; Foster et al., 1998). This enabled a better understanding of the extent to which EBTs can change over time.

The study does have limitations which should be considered. Participants were predominantly White (87.8%) and mainly women (68.7%), which may limit generalisability. This could be considered a convenience sample because it was not representative of the general population. However, this limitation can be seen in many clinical trials. Furthermore, the use of stepwise selection in the data analysis does risk overfitting. However, including all variables while better for prediction, would not answer the question of which specific EBTs explain weight change. Finally, although statistically significant, the effect sizes (η^2) found for the interactions between time and weight category for each of the EBT variables were small. Therefore, the clinical significance of these results is unclear and future research is needed to replicate these findings and to examine the use of EBTs in weight management programs and interventions.

To conclude, the current data shows that EBT changes are associated with concomitant weight change, such that reactive eating is associated with weight re-gain, reflective eating is associated with weight loss and homeostatic eating is associated with weight maintenance. There was large individual variability in weight change, with LMM's showing that participants who lost weight having significantly higher levels of reflective EBTs and significantly lower levels of reactive EBTs and these results were not attributable to any specific effect of the interventions studied here. To understand if these associations are based on causal relationships, intervention research is needed to test if promoting reflective and intuitive eating, whilst managing reactive eating can help improve the effectiveness of longer-term weight management interventions.

4.6. References

Arumäe, K., Mõttus, R., & Vainik, U. (2022). Body mass predicts personality development across 18 years in middle to older adulthood.

Berthoud, H.-R. (2011). Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Current opinion in neurobiology*, *21*(6), 888-896.

Björvell, H., Aly, A., Langius, A., & Nordström, G. (1994). Indicators of changes in weight and eating behaviour in severely obese patients treated in a nursing behavioural program. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*, *18*(8), 521-525.

Björvell, H., Rössner, S., & Stunkard, A. (1986). Obesity, weight loss, and dietary restraint. *International Journal of Eating Disorders*, *5*(4), 727-734.

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

Blundell, J. E., Stubbs, R., Golding, C., Croden, F., Alam, R., Whybrow, S., Le Noury, J., & Lawton, C. (2005). Resistance and susceptibility to weight gain: individual variability in response to a high-fat diet. *Physiology & Behavior*, *86*(5), 614-622.

Bray, G., Kim, K., Wilding, J., & Federation, W. O. (2017). Obesity: a chronic relapsing progressive disease process. A position statement of the World Obesity Federation. *obesity reviews*, *18*(7), 715-723.

Bryant, E. J., Caudwell, P., Hopkins, M. E., King, N., & Blundell, J. (2012). Psycho-markers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*, *58*(1), 234-241.

Butryn, M. L., Webb, V., & Wadden, T. A. (2011). Behavioral treatment of obesity. *Psychiatric Clinics*, *34*(4), 841-859.

Canetti, L., Berry, E. M., & Elizur, Y. (2009). Psychosocial predictors of weight loss and psychological adjustment following bariatric surgery and a weight-loss program: The mediating role of emotional eating. *International Journal of Eating Disorders*, *42*(2), 109-117.

Chaput, J.-P., Drapeau, V., Hetherington, M., Lemieux, S., Provencher, V., & Tremblay, A. (2005). Psychobiological impact of a progressive weight loss program in obese men. *Physiology & Behavior*, *86*(1-2), 224-232.

Cuntz, U., Leibbrand, R., Ehrig, C., Shaw, R., & Fichter, M. (2001). Predictors of post-treatment weight reduction after in-patient behavioral therapy. *International Journal of Obesity*, *25*(1), S99-S101.

Dakin, C., Beaulieu, K., Hopkins, M., Gibbons, C., Finlayson, G., & Stubbs, R. J. (2023). Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis. *obesity reviews*, *24*(1), e13515.

Dombrowski, S. U., Knittle, K., Avenell, A., Araújo-Soares, V., & Sniehotta, F. F. (2014). Long term maintenance of weight loss with non-surgical interventions in obese adults: systematic review and meta-analyses of randomised controlled trials. *BMJ*, *348*.

Donnelly, J. E., Blair, S. N., Jakicic, J. M., Manore, M. M., Rankin, J. W., & Smith, B. K. (2009). Appropriate physical activity intervention strategies for weight loss and prevention of weight regain for adults. *Medicine & Science in Sports & Exercise*, *41*(2), 459-471.

Dunton, G. F., Rothman, A. J., Leventhal, A. M., & Intille, S. S. (2021). How intensive longitudinal data can stimulate advances in health behavior maintenance theories and interventions. *Translational behavioral medicine*, *11*(1), 281-286.

Elfhag, K., & Rössner, S. (2005). Who succeeds in maintaining weight loss? A conceptual review of factors associated with weight loss maintenance and weight regain. *obesity reviews*, *6*(1), 67-85.

Evers, C., Marijn Stok, F., & de Ridder, D. T. D. (2010). Feeding your feelings: emotion regulation strategies and emotional eating. *Personality & social psychology bulletin*, *36*(6). https://doi.org/https://dx.doi.org/10.1177/0146167210371383

Filiatrault, M.-L., Chaput, J.-P., Drapeau, V., & Tremblay, A. (2014). Eating behavior traits and sleep as determinants of weight loss in overweight and obese adults. *Nutrition & Diabetes*, *4*(10), e140-e140.

Fogelholm, M., Kukkonen-Harjula, K., & Oja, P. (1999). Eating control and physical activity as determinants of short-term weight maintenance after a very-low-calorie diet among obese women. *International Journal of Obesity*, *23*(2), 203-210.

Foster, G., Wadden, T., Swain, R., Stunkard, A., Platte, P., & Vogt, R. (1998). The Eating Inventory in obese women: clinical correlates and relationship to weight loss. *International Journal of Obesity*, *22*(8), 778-785.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive behaviors*, *7*(1), 47-55.

Greaves, C. J., Sheppard, K. E., Abraham, C., Hardeman, W., Roden, M., Evans, P. H., & Schwarz, P. (2011). Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions. *BMC Public Health*, *11*(1), 1-12.

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.

Health Survey for England. (2019). *Health Survey for England 2019 Overweight and obesity in adults and children*. https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2019

Hood, M. M., Grupski, A. E., Hall, B. J., Ivan, I., & Corsica, J. (2013). Factor structure and predictive utility of the Binge Eating Scale in bariatric surgery candidates. *Surgery for Obesity and Related Diseases*, *9*(6), 942-948.

IBM Corp. (2020). *(2020). IBM SPSS Statistics for Windows (Version 27.0) [Computer software]. IBM Corp.* In

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Karlsson, J., Hallgren, P., Kral, J., Lindroos, A.-K., Sjöström, L., & Sullivan, M. (1994). Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. *Appetite*, *23*(1), 15-26.

Keränen, A.-M., Savolainen, M. J., Reponen, A. H., Kujari, M.-L., Lindeman, S. M., Bloigu, R. S., & Laitinen, J. H. (2009). The effect of eating behavior on weight loss and maintenance during a lifestyle intervention. *Preventive medicine*, *49*(1), 32-38.

Keränen, A.-M., Strengell, K., Savolainen, M. J., & Laitinen, J. H. (2011). Effect of weight loss intervention on the association between eating behaviour measured by TFEQ-18 and dietary intake in adults. *Appetite*, *56*(1), 156-162.

King, N. A., Hopkins, M., Caudwell, P., Stubbs, R., & Blundell, J. (2008). Individual variability following 12 weeks of supervised exercise: identification and

characterization of compensation for exercise-induced weight loss. *International Journal of Obesity*, *32*(1), 177-184.

Kwasnicka, D., Dombrowski, S. U., White, M., & Sniehotta, F. (2016). Theoretical explanations for maintenance of behaviour change: a systematic review of behaviour theories. *Health Psychology Review*, *10*(3), 277-296.

Larsen, T., Dalskov, S., Van Baak, M., Jebb, S., Kafatos, A., Pfeiffer, A., Martinez, J., Handjieva-Darlenska, T., Kunešová, M., & Holst, C. (2010a). The Diet, Obesity and Genes (Diogenes) Dietary Study in eight European countries–a comprehensive design for long-term intervention. *obesity reviews*, *11*(1), 76-91.

Larsen, T. M., Dalskov, S.-M., van Baak, M., Jebb, S. A., Papadaki, A., Pfeiffer, A. F., Martinez, J. A., Handjieva-Darlenska, T., Kunešová, M., & Pihlsgård, M. (2010b). Diets with high or low protein content and glycemic index for weight-loss maintenance. *New England Journal of Medicine*, *363*(22), 2102-2113.

Marques, M. M., Matos, M., Mattila, E., Encantado, J., Duarte, C., Teixeira, P. J., Stubbs, R. J., Sniehotta, F. F., Ermes, M., & Harjumaa, M. (2021). A theory- and evidence-based digital intervention tool for weight loss maintenance (NoHoW Toolkit): systematic development and refinement study. *Journal of Medical Internet Research*, *23*(12), e25305.

McGuire, M. T., Wing, R. R., Klem, M. L., Lang, W., & Hill, J. O. (1999). What predicts weight regain in a group of successful weight losers? *Journal of Consulting and Clinical Psychology*, *67*(2), 177.

Pekkarinen, T., Takala, I., & Mustajoki, P. (1996). Two year maintenance of weight loss after a VLCD and behavioural therapy for obesity: correlation to the scores of questionnaires measuring eating behaviour. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*, *20*(4), 332-337.

Pliner, P., & Saunders, T. (2008). Vulnerability to freshman weight gain as a function of dietary restraint and residence. *Physiology & Behavior*, *93*(1-2), 76-82.

Qualtrics. (Provo, UT). (Version January 2023) https://www.qualtrics.com

Sainsbury, K., Evans, E. H., Pedersen, S., Marques, M. M., Teixeira, P. J., Lähteenmäki, L., Stubbs, R. J., Heitmann, B. L., & Sniehotta, F. F. (2019). Attribution of weight regain to emotional reasons amongst European adults with overweight and obesity who regained weight following a weight loss

attempt. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, *24*, 351-361.

Scott, S. E., Duarte, C., Encantado, J., Evans, E. H., Harjumaa, M., Heitmann, B. L., Horgan, G. W., Larsen, S. C., Marques, M. M., & Mattila, E. (2019). The NoHoW protocol: a multicentre 2× 2 factorial randomised controlled trial investigating an evidence-based digital toolkit for weight loss maintenance in European adults. *BMJ Open*, *9*(9), e029425.

Stevens, J., Truesdale, K. P., McClain, J. E., & Cai, J. (2006). The definition of weight maintenance. *International Journal of Obesity*, *30*(3), 391-399.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and social psychology review*, *8*(3), 220-247.

Stubbs, R. J., Duarte, C., O'Driscoll, R., Turicchi, J., Kwasnicka, D., Sniehotta, F. F., Marques, M. M., Horgan, G., Larsen, S., & Palmeira, A. (2021). The H2020 "NoHoW Project": A Position Statement on Behavioural Approaches to Longer-Term Weight Management. *Obesity facts*, *14*(2), 246-258.

Stubbs, R. J., Horgan Graham, Robinson Eric, Hopkins Mark, Dakin Clarissa, & Graham, F. (2023). Diet composition and energy intake in humans. *In Press*.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Sumithran, P., & Proietto, J. (2013). The defence of body weight: a physiological basis for weight regain after weight loss. *Clinical Science*, *124*(4), 231-241.

Tavakol, M., & Dennick, R. (2011). Making sense of Cronbach's alpha. *International journal of medical education*, *2*, 53.

Teixeira, P., Going, S. B., Sardinha, L., & Lohman, T. (2005). A review of psychosocial pre-treatment predictors of weight control. *obesity reviews*, *6*(1), 43-65.

Tylka, T. L., Calogero, R. M., & Daníelsdóttir, S. (2020). Intuitive eating is connected to self-reported weight stability in community women and men. *Eating Disorders*, *28*(3), 256-264.

Tylka, T. L., & Kroon Van Diest, A. M. (2013). The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men. *Journal of counseling psychology*, *60*(1), 137.

Varkevisser, R., Van Stralen, M., Kroeze, W., Ket, J., & Steenhuis, I. (2019). Determinants of weight loss maintenance: a systematic review. *obesity reviews*, *20*(2), 171-211.

Wadden, T. A., & Bray, G. A. (2018). *Handbook of obesity treatment*. Guilford Publications.

Wadden, T. A., Foster, G. D., & Letizia, K. A. (1994). One-year behavioral treatment of obesity: comparison of moderate and severe caloric restriction and the effects of weight maintenance therapy. *Journal of Consulting and Clinical Psychology*, *62*(1), 165.

Wadden, T. A., Tronieri, J. S., & Butryn, M. L. (2020). Lifestyle modification approaches for the treatment of obesity in adults. *American psychologist*, *75*(2), 235.

Warren, J. M., Smith, N., & Ashwell, M. (2017). A structured literature review on the role of mindfulness, mindful eating and intuitive eating in changing eating behaviours: effectiveness and associated potential mechanisms. *Nutrition Research Reviews*, *30*(2), 272-283.

Westenhoefer, J., Engel, D., Holst, C., Lorenz, J., Peacock, M., Stubbs, J., Whybrow, S., & Raats, M. (2013). Cognitive and weight-related correlates of flexible and rigid restrained eating behaviour. *Eating behaviors*, *14*(1), 69-72.

Wing, R. R., & Phelan, S. (2005). Long-term weight loss maintenance–. *The American journal of clinical nutrition*, *82*(1), 222S-225S.

World Medical Association. (2001). World Medical Association Declaration of Helsinki. Ethical principles for medical research involving human subjects. *Bulletin of the World Health Organization*, *79*(4), 373.

Chapter 5. Can eating behaviour traits be explained by underlying, latent factors? An exploratory and confirmatory factor analysis

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5.1. Abstract

Eating Behaviour Traits (EBTs) are psychological constructs developed to explain patterns of eating behaviour, including factors that motivate people to (over or under) eat. There is a need to align and clarify their unique contributions and harmonise the understanding they offer for human eating behaviour. Therefore, the current study examined whether 18 commonly cited EBTs could be explained by underlying, latent factors (domains of eating behaviour). An exploratory factor analysis (EFA) was used to identify latent factors, and these factors were validated using a confirmatory factor analysis (CFA). 1279 participants including the general public and members of a weight management programme were included in the analysis (957 females, 317 males, 3 others, 2 prefer not to say), with a mean age of 54 years (median = 57 years, SD = 12.03) and a mean BMI of 31.93 kg/m2 (median = 30.86, SD = 6.00). The participants completed 8 questionnaires which included 18 commonly cited EBTs and the dataset was split at random with a 70/30 ratio to conduct the EFA (n = 893) and CFA (n = 383). The results supported a four-factor model which indicated that EBTs can be organised into four domains: reactive, restricted, emotional, and homeostatic eating. The four-factor model also significantly predicted self reported BMI and weight change. Future research should test whether this factor structure is replicated in more diverse populations, and including other EBTs, to advance these domains of eating as a unifying framework for studying individual differences in human eating behaviour.

5.2. Introduction

Obesity has become a pressing public health concern, with a significant proportion of the population in many countries, including the UK, now classified as overweight or obese. Recent statistics reveal that 64% of the UK population falls into these categories (ONS, 2020a). The consequences of obesity are far-reaching, including reduced quality of life, increased risk of serious diseases, and higher mortality rates (Allison et al., 2008). As such, understanding the factors that contribute to the development of obesity and identifying strategies to improve weight loss interventions have become critical areas of scientific inquiry. Eating behaviour is believed to be a major cause for weight gain and an intervention target for weight management. There has therefore, been considerable interest in identifying individual differences in psychological factors that influence overeating and obesity.

Over the last 40 years, a multitude of theoretical models and associated constructs have been developed to explain variations in eating behaviour, including factors that motivate people to (over)eat. These constructs, collectively referred to as Eating Behaviour Traits (EBTs), aim to explain a defined, reliable eating style or disposition to eat. EBTs are measured using self-report questionnaires and have been widely studied and often implicated as causal factors of overeating and obesity in both research (French et al., 2012) and media discourse. They have also been shown as reliable indices of motivated behaviours related to food (Llewellyn & Wardle, 2015).

Some concern arises when evaluating the multitude of EBT questionnaires available. With over 20 questionnaires designed to measure EBTs, there is a need to ascertain if each one contributes uniquely to our understanding of motivations to eat. The large number of scales could be adding to unnecessary heterogeneity in cause-effect relationships that are found using these scales. Many of these EBTs appear to measure overlapping theories and constructs and share similar mechanisms of action (Vainik et al., 2015). For example, the EBTs food responsiveness, external eating and disinhibition all include items that refer to opportunistic eating and the influence of environmental cues that drive eating. Similarly, binge eating, disinhibition and emotional eating all include items that refer to eating in response to emotions. This redundancy is referred to as a jangle fallacy (Kelley, 1927). It appears that

jangle fallacy is common in obesity research (Vainik et al., 2015). The use of different questionnaires for similar purposes is leading to potential confusion in the field since the validity of each EBT is unclear. A jangle fallacy also constrains comparisons between studies and interpretations of research findings. At present there is no standardised set of measures that explains motivations for eating, which also complicates comparisons between studies and interpretations of research findings.

Previously, Vainik et al. (2015) developed a model that attempted to integrate several EBT constructs under one underlying construct. This latent construct was termed uncontrolled eating, which encompasses increased appetite and decreased self-control. The uncontrolled eating model was adapted from a review that proposed a continuum model of uncontrolled eating (Davis, 2013). This model suggests that eating behaviour constructs increase in severity of uncontrolled eating. The continuum begins at “homeostatic eating”, where energy intake matches energy expended, then increases to passive overeating, and finally up to severe bingeing and possibly “food addiction” (Vainik et al., 2015). This continuum model provided an analytical framework which was tested to understand whether EBTs measure different stages of the same dimension (uncontrolled eating). The researchers tested five EBTs (eating impulsivity, power of food, emotional eating, disinhibition, and binge eating) and suggested that eating impulsivity would focus on milder uncontrolled eating, then hedonic hunger should measure passive overeating. Emotional eating, disinhibition and binge eating were proposed to measure increasingly severe degrees of uncontrolled eating. The analysis found support for the existence of a single underlying construct, relating to these 5 EBT measures, and this construct was labelled uncontrolled eating (Vainik et al., 2015). Overall, each of the EBTs included in this study appeared to measure some aspect of increased appetite and decreased self-control. The findings suggested that EBTs are potentially measuring the same latent construct and could be used interchangeably. However, the uncontrolled eating model examined a limited number of EBTs and did not include some common EBTs that do not purport to measure overeating such as intuitive eating. Furthermore, if uncontrolled eating encompasses both increased appetite and decreased self-control, this construct may not be unidimensional.

Another study has also examined commonly cited EBTs including the Emotional Eating Scale, Three-factor Eating Questionnaire, Dutch Eating Behaviour Questionnaire, Power of Food Scale and Yale Food Addiction Scale using factor analysis techniques with the aim of uncovering the underlying structure of these questionnaires (Price et al., 2015). Principal components analysis found support for two underlying components, which were suggested to reflect food reward responsivity ("reduced control over eating in response to external food cues and internal emotional states") and dietary restriction ("the tendency to restrict intake in order to control weight"). Similarly to Vainik's uncontrolled eating model, Price et al. (2015) found support for a single factor that underlies many EBTs measuring food reward responsivity and also extended this model to include food addiction and dietary restraint, which indicated another independent factor "dietary restriction." The authors suggest that future research should examine whether food reward responsivity and uncontrolled eating are overlapping factors.

In light of these considerations, the current study sought to further the work of Vainik et al. (2015) and Price et al. (2015) by examining the underlying structure of a broad range of commonly cited EBTs. The current study aimed to conceptually replicate and extend previous research by including a sample of more diverse EBTs than previously examined. This study combines data from one survey given to two samples which included the public and members of a weight management program (Slimming World, UK) to create one dataset. The analysis of both the general public and weight management programme members was deemed important because these samples may have very different eating behaviours. For example, research has found that current and past dieters have higher scores for restraint and disinhibition (women only) than non-dieters (Provencher et al., 2004). Additionally, some studies have found that eating behaviours change before and after dieting, which suggests that dieting may provoke significant changes in EBTs (Foster et al., 1998; Karlsson et al., 1994). These studies indicate that dieting status may influence EBTs. Therefore, it is important to investigate eating behaviour in people who are actively trying to manage their weight and those who may or may not be actively trying to manage their weight. By conducting an exploratory factor analysis (EFA), we aimed to uncover whether a wide range of commonly cited EBTs can be organised under latent factors. The study then conducted a confirmatory factor

analysis (CFA), to examine the proposed model fit and to confirm whether any identified eating behaviour domains exist in a different sample. Lastly, the final model was asked to predict two external outcomes (self-reported BMI and weight change over 12 months) to assess whether any of the proposed factors could predict objective indices of long-term energy balance status (BMI) and short-term changes in energy balance (weight change). These external outcomes were included to examine the capability of the factor model in predicting real-world, measurable outcomes. We hypothesised that 18 EBT can be organised into underlying latent factors.

5.3. Methods

5.3.1. Participants

The sample consisted of 1677 members of the public (23%) and members involved in a weight management program (77%) (Slimming World, UK), who completed an online survey. The survey included demographic questions and 8 psychometric questionnaires covering 18 EBT constructs. Participants were recruited via opportunity sampling from a range of online sources managed by the University of Leeds and Slimming World. Participants were excluded if they self-reported they were pregnant or breastfeeding in the prior 6 months, had a history of previous eating disorder, weight loss surgery, any medical condition that affected body weight or appetite, and individuals with insufficient English language skills. Ethical approval was granted from the School of Psychology Research Ethics Committee at the University of Leeds (reference number: PSYC-338/904, see C.1). Cases with missing data were removed to leave a complete dataset.

This resulted in a final sample of 1660 participants (Slimming World members, n = 1276, general public, n = 384, 1256 females, 398 males, 3 others, 3 prefer not to say), with a mean age of 50.41 years (SD = 16.05). The mean BMI of the sample was 30.63 kg/m^2 (SD = 6.40). 95.2% of the sample were white, with the remainder being 2.2% Asian, 1.3% mixed race, 0.5% prefer not to say, 0.4% Black, 0.4% other. The highest level of education achieved was for 27.4%, University education, with the remainder achieving as follows: 32.0% high vocational; 14.4% sixth form; 17.0% secondary school; 8.5% other and 0.7% no formal education. This sample, compared to UK population statistics was 14.2 years older (ONS, 2020b) and had an 3.03kg/m^2 higher average BMI (NHS Digital, 2019). The sample had a similar level of education to average census statistics (GOV.UK, 2014), while the percentage of White people was higher than government data (GOV.UK, 2011). Comparing the weight management sample to the general public sample, there were no significant differences in gender ($p = .053$). However, the weight management sample were older (M = 55.55 years, SD = 12.00) than the general public (M = 35.73 years, SD = 17.80), $p < .001$. They had a larger BMI (M = 31.93 kg/m^2, SD = 6.00) than the general public (M = 24.14, SD = 4.32), $p < .001$. Additionally, there was also a

significant difference in dieting status between the weight management sample and the general public sample ($p < .001$). 87.7% of the weight management sample reported actively trying to lose weight, whilst only 48.3% of the general public sample reported actively trying to lose weight.

5.3.2. Questionnaires

Eight EBT questionnaires were used with the aim of measuring a wide range of EBT constructs. The following criteria were used to select EBTs to include in the study: the EBT must (1) measure a defined eating behaviour trait, (2) measure a motivation for eating, (3) have been formally validated in a peer-reviewed publication, (4) have been cited at least 50 times, (5) not be a diagnostic instrument for an eating disorder. Additionally, questionnaires were also chosen with the aim of including a range of extremes of under/overeating from restricted eating styles to extreme measures of overeating. It was also considered important to include older (e.g., 1980's) and newer measures (e.g., 2010's). Using these criteria, 18 EBT were selected from 8 questionnaires (see Table 5.1). The questionnaire was piloted, and it was found that 8 questionnaires was the limit of participant tolerance. Overall, the approach taken to select EBTs was as systematic as possible within the constraints of the survey length. Whilst a multitude of EBTs exist in the literature, only this limited number of measures were included due to the risk of over burdening participants.

Table 5.1. Questionnaires measured in this study

EBT	Questionnaire	Reference
Disinhibition (TFEQ_D) Restraint (TFEQ_R) Susceptibility to hunger (TFEQ_H)	Three Factor Eating Questionnaire (TFEQ)	Stunkard and Messick (1985)
SREB	Self-regulation of Eating Behaviour (SREB)	Kliemann et al. (2016)
Reliance on hunger and satiety (IES_RHSC), Eating for physical rather than emotional reasons (IES_EFPR), Body-food choice congruence (IES_BFCC), Unlimited permission to eat (IES_UPTE)	Intuitive Eating Scale-2 (IES)	Tylka and Kroon Van Diest (2013)
Restraint (DEBQ_R) External Eating (DEBQ_EX)	Dutch Eating Behaviour Questionnaire (DEBQ)	Van Strien et al. (1986)
Food Responsiveness (AEBQ_FR), Satiety Responsiveness (AEBQ_SR), Emotional Overeating (AEBQ_EOE) Emotional Undereating (AEBQ_EUE),	Adult Eating Behaviour Questionnaire (AEBQ)	Hunot et al. (2016)
Positive Emotional Eating (PNEES_P) Negative Emotional Eating (PNEES_N)	Positive and Negative Emotional Eating Scale (PNEES)	Sultson et al. (2017)
PFS	Power of Food Scale (PFS)	Lowe et al. (2009)
BES	Binge Eating Scale (BES)	Gormally et al. (1982)

Intuitive Eating Scale-2 (IES)

The reliance on hunger and satiety subscale (IES_RHSC, 6 items, α = 0.85, example item = “I trust my body to tell me when to eat”) captures an individual’s trust and reliance on their internal satiety and hunger cues to guide their behaviour. Eating for

physical rather than emotional reasons (IES_EPR, 8 items, α = 0.92, example item = “When I am lonely, I do not turn to food for comfort”) measures individuals’ patterns of eating, whether they eat because they are physically hungry or to cope with distress. Unconditional permission to eat (IES_UPE, 6 items, α = 0.52, example item = “If I am craving a certain food, I allow myself to have it”) measures an individual’s willingness to eat when hungry and someone who rejects labelling certain foods as forbidden. Lastly, the body-food choice congruence (IES_BFCC, 3 items, α = 0.86, example item = “Most of the time, I desire to eat nutritious foods”) assesses how well an individual matches their food choices with their body’s needs.

Self-regulation of Eating Behaviour (SREB)

The SREB (5 items, α = 0.79, example item = “I’m good at resisting tempting foods”), measures self-regulatory eating capacity in adults. All 5 items were used in this study.

Dutch Eating Behaviour Questionnaire

The full questionnaire measures restrained, emotional and external eating. Emotional eating was not measured in both samples because multiple other emotional eating subscales were included (e.g., PNEES and AEBQ emotional over and undereating) and we did not want to over burden the participants with the number of questionnaires they were required to complete. The external eating subscale (DEBQ_EX, 10 items, α = 0.88, example item = “If a food tastes good to you, do you eat more than usual?”) captures eating in response to food-related stimuli, regardless of any internal satiety or hunger. The restraint subscale (DEBQ_R, 10 items, α = 0.80, example item = “Do you watch exactly what you eat?”) measures intention to restrict food intake to control body weight.

Adult Eating Behaviour Questionnaire (AEBQ)

The full questionnaire aims to measure appetite traits in adults. Four scales were not used in this study, because they do not measure motivations for eating (enjoyment of food, food fussiness, slowness in eating, and hunger). These scales measure traits associated with eating but not specific motivations or reasons for over or under eating. For example, “I often notice my stomach rumbling” (hunger), “I refuse new foods at first” (food fussiness), “I love food” (enjoyment of food), and “I eat slowly”

(slowness in eating). Satiety responsiveness (AEBQ_SR, 4 items, α = 0.63, example item = “I often get full before my meal is finished”) measures an individuals’ sensitivity to their internal satiety signals. Emotional undereating (AEBQ_EUE, 5 items, α = 0.92, “I eat less when I’m worried”) measures the extent to which individuals undereat when emotional. Emotional overeating (AEBQ_EOE, 5 items, α = 0.90, example item = “I eat more when I’m annoyed”) measures the extent to which individuals overeat when emotional. Food responsiveness (AEBQ_FR, 4 items, α = 0.77, example item = “I often feel hungry when I am with someone who is eating”) measures the level of responsivity to food and was included in both samples.

Power of Food Scale (PFS)

The PFS assesses hedonic hunger, which is defined as an individual’s hedonic appetite drive to intake palatable foods that are beyond homeostatic need, in readily available food environments (15 items, α = 0.92, example item = “I find myself thinking about food even when I’m not physically hungry”). This questionnaire can be split into three subscales; however, these subscales were not used because they were highly correlated with each other and were not thought to contribute any unique offerings separately.

Three Factor Eating Questionnaire (TFEQ)

The full 51-item questionnaire was used as a measure of three behavioural and cognitive dimensions of eating. The restraint subscale (TFEQ_R, 21 items, α = 0.63, example item = “I deliberately take small helpings as a means of controlling my weight”) measures the tendency to restrict food intake to control body weight. Disinhibition (TFEQ_D, 16 items, α = 0.84, example item = “I usually eat too much as social occasions, like parties and picnics”) measures tendency towards overeating. While disinhibition has been shown to have three sub-dimensions, these dimensions are not commonly used in research, therefore, for the purpose of capturing latent constructs in commonly used EBT, the one-factor scale was used in this study. The last subscale of the TFEQ is susceptibility to hunger (TFEQ_H, 14 items, α = 0.86, example item = “I am usually so hungry that I eat more than three times a day”).

Positive and Negative Emotional Eating Scale

The positive emotional eating subscale (PNEES_P, 7 items, α = 0.76, “I have a desire to eat when I am joyful”) captures eating in response to positive emotions, whilst the negative emotional eating subscale (PNEES_N, 12 items, α = 0.94, example item = “I tend to eat when I am grumpy”) captures eating in response to negative emotions.

Binge Eating Scale (BES)

The binge eating scale (BES, 16 items, α = 0.90) measures specific binge eating behaviours. Each question on the BES contains 3-5 statements that the participant is required to choose from based on which statement applies most to them. Within an item, the statements increase in binge eating severity. For example, item 6 includes three statements: “I don’t feel any guilt or self-hate after I overeat,” “after I overeat, occasionally I feel guilt or self-hate,” and “almost all the time I experience strong guilt or self-hate after I overeat.”

5.3.3. Measures of External Outcomes

BMI

BMI was calculated based on participants’’ self-reported height and weight using the standard formula (kg/m^2).

Weight Change

Participants were asked “has your weight changed at all over the previous 6 months?” If the participants answered “Yes,” they were required to input their weight loss or weight gain. Self-reported weight change (kg) was then calculated using this information. The mean weight change of the total sample was -9.13kg (SD = 10.80). The mean weight change of the weight management group sample was -10.18kg (SD = 10.76), whilst the mean weight change of the public sample was 0.02kg (SD = 5.65).

5.3.4. Procedure

Participants were given ethical and study-related information, including data confidentiality, risks, and benefits of taking part and contact information of the

research team. After completing informed consent, the participants entered a unique ID and completed demographic measures. Once finished, the participants completed the questionnaires (Table 5.1), which were presented in a random order, and took approximately 40 minutes to complete. Participants were then debriefed and if they wished, entered their email to be entered into a prize draw for £1,000.

5.4. Results

5.4.1. Data analysis

Total scores were created for the scales and subscales based on the scoring key of each reference questionnaire. All subscales were used except for the PFS where the total scale score was used because the subscales were found to be highly related. The parcelled data (total scores) was used because previous research has shown that parcelling can reduce the effects of nonnormality and can increase validity and reliability of results (Holt, 2004). Outliers were identified via boxplot and extreme outliers (above 3rd quartile + 3*interquartile range [IQR] and below 1st quartile – 3*IQR). Extreme outliers were removed because they were deemed erroneous or unrepresentative values. After removing outliers there were 1658 cases included. The Shapiro-Wilk test for normality indicated that all variables were not normally distributed. However, all variables, except for PNEES_P were within normal ranges for skewness and kurtosis (Kline, 2015) and appeared normally distributed according to the QQ plots. PNEES_P was log transformed to push the data towards a normal distribution. There were reasonably high correlations between EBTs for the whole dataset (n = 1658, see table 5.2).

The dataset was then split at random into test and train datasets with a split of 70/30 respectively. The train dataset (n = 1161) was used to conduct an exploratory factor analysis (EFA) on the data to uncover the number of underlying factors and to give an indication as to which EBT could be subsumed under a given number of factors. The test dataset (n = 497) was then used to conduct a confirmatory factor analysis (CFA) to test the factor models proposed by the EFA. The percentage split of weight management programme participants compared to general public participants was similar between the train and test dataset. For the train dataset, the sample comprised of 77.4% weight management participants and whilst for the test dataset, the sample was comprised of 75.3% weight management participants. There were no significant differences between the train and test dataset for age (p = .333), BMI (p = .518) or gender (p = .340). The full sample (n = 1658) was used to examine whether the proposed factors could predict external outcomes. The following packages were used: ‘psych’ (Revelle, 2022) and ‘dplyr’ (Wickham et al., 2022).

Table 5.2. Correlations between all EBT for the whole sample (n = 1658)

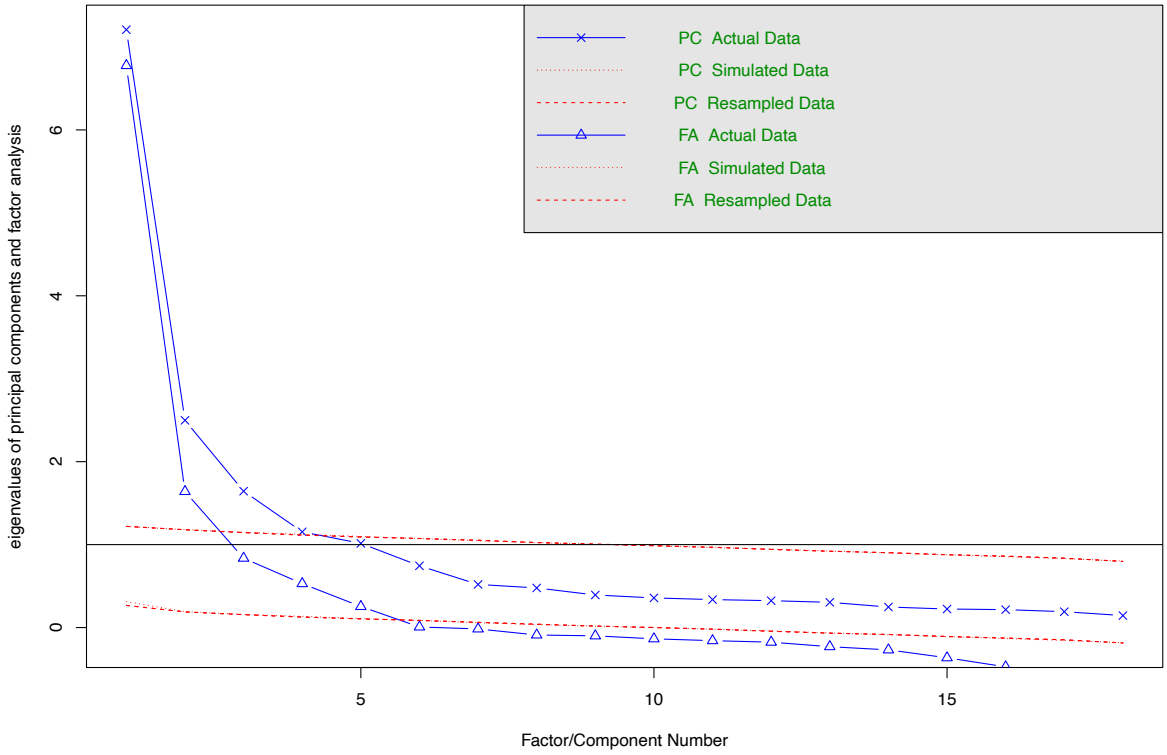
	1	2	3	4	5	6	7	8	9	10	11	12	13	14	15	16	17	18
SREB																		
DEBQ_R	.10**																	
DEBQ_EX	-.54**	.04																
PFS_TOTAL	-.49**	.11**	.74**															
LGPNEES_P	-.23**	-.03	.34**	.33**														
PNEES_N	-.50**	.09**	.55**	.59**	.29**													
AEBQ_EOE	-.42**	.13**	.44**	.47**	.14**	.79**												
AEBQ_SR	.11**	.01	-.27**	-.27**	-.07**	-.18**	-.13**											
AEBQ_EUE	.14**	.01	-.11**	-.12**	.12**	-.38**	-.43**	.26**										
AEBQ_FR	-.43**	.10**	.71**	.75**	.33**	.53**	.50**	-.28**	-.07**									
IES_EPR	.50**	-.14**	-.51**	-.53**	-.14**	-.77**	-.74**	.18**	.31**	-.51**								
IES_UPE	-.10**	-.52**	.06*	-.04	.12**	-.13**	-.19**	.07**	.17**	-.04	.26**							
IES_RHSC	.25**	-.26**	-.23**	-.29**	.06*	-.35**	-.33**	.30**	.28**	-.25**	.47**	.54**						
IES_BFCC	.32**	.11**	-.17**	-.19**	.02	-.30**	-.27**	.13**	.22**	-.17**	.39**	.40**	.61**					
BES	-.58**	.19**	.59**	.67**	.26**	.68**	.55**	-.20**	-.19**	.60**	-.65**	-.20**	-.46**	-.39**				
TFEQ_D	-.57**	.15**	.65**	.67**	.21**	.74**	.67**	-.31**	-.28**	.63**	-.74**	-.19**	-.49**	-.38**	.77**			
TFEQ_H	-.49**	.01	.64**	.67**	.32**	.53**	.44**	-.34**	-.15**	.69**	-.47**	.00	-.28**	-.20**	.62**	.66**		
TFEQ_R	.26**	.64**	-.14**	-.06*	-.07**	-.09**	-.00	.14**	.09**	-.03	.05	-.43**	-.08**	.05	.00	-.06*	-.14**	

Note: AEBQ_FR = Food Responsiveness, PFS = Power of Food, TFEQ_H = Susceptibility to Hunger, DEBQ_EX = External Eating, TFEQ_D = Disinhibition, BES = Binge Eating, AEBQ_EOE: Emotional Overeating, PNEES_N = Negative Emotional Eating, IES_EPR: Eating for Physical Reasons, IES_RHSC = Reliance on Hunger and Satiety, IES_BFCC = Body-Food Choice Congruence, IES_UPE = Unlimited Permission to Eat, TFEQ_R = Restraint, DEBQ_R = Restraint.

5.4.2. Exploratory Factor Analysis

Global diagnostic indicators indicated strong factorability of the correlation matrix for all EBTs in the train dataset (Kaiser–Meyer Olkin = .90, Bartlett's test of sphericity x^2 = 13291.62, p < .001). The eigenvalues and parallel analysis scree plot (Figure 5.1) for all EBT's suggested 5 factors.

Figure 5.1. Scree plot, n = 1161



Note: PC = Principal components, FA = factor analysis.

The EFA was set to 5 factors based on the eigen values and parallel analysis. The factoring method used was maximum likelihood analysis (ML) because ML enables the computation of a wide range of indices of goodness of fit and allowed statistical significance testing of factor loadings (Fabrigar et al., 1999). An oblique rotation method was used because factors were expected to correlate and this type of rotation theoretically creates a more accurate and reproducible solution (Costello & Osborne, 2005). Since there is no widely preferred method of oblique rotation with all tending to produce the same results, direct oblimin was used (Fabrigar et al., 1999). Finally, items attaining a loading of 0.32 or higher on any factor were retained

(Tabachnick et al., 2013). Table 5.3 shows the factor loadings for all EBT that loaded above 0.32 onto a factor.

Table 5.3. EFA result table displaying factor loadings (>0.32), commonality, uniqueness, complexity, and correlations between factors

EBT	ML1	ML3	ML4	ML2	ML5	Commonality	Uniqueness	Complexity
Food responsiveness	.86					0.74	0.26	1.04
Hedonic hunger	.85					0.77	0.24	1.01
External eating	.79					0.71	0.29	1.07
Susceptibility to hunger	.77					0.65	0.35	1.04
Satiety responsiveness	-.46				.39	0.34	0.66	2.89
Binge eating	.44					0.75	0.26	3.17
Disinhibition	.42	.40				0.80	0.20	2.74
Positive emotional eating	.40					0.22	0.78	1.85
Emotional overeating		.93				0.80	0.20	1.03
Negative emotional eating		.82				0.83	0.17	1.07
Eating for physical rather than emotional reasons		-.64				0.75	0.25	1.37
Emotional undereating		-.58			.32	0.37	0.63	2.05
Reliance on hunger and satiety cues			.80			0.73	0.27	1.09

Body-food choice congruence		.75			0.55	0.45	1.12
Restraint (TFEQ)			.84		0.70	0.30	1.02
Restraint (DEBQ)			.77		0.63	0.37	1.06
Unlimited permission to eat		.49	-.53		0.63	0.37	2.27
Self-regulation of eating				-.35	0.58	0.42	4.09
Factor correlation							
ML1							
ML3	.60						
ML4	-.29	-.43					
ML2	-.08	.02	-.14				
ML5	.31	.23	.01	-.05			

Note. TFEQ = Three-Factor Eating Questionnaire, DEBQ = Dutch Eating Behaviour Questionnaire.

The 5 factor EFA goodness of fit indices were: RMSR = 0.02, TLI = 0.95, RMSEA = 0.06, CI = 0.056 – 0.068. Total variance explained was 64%, with individual factors explaining the following variance: Factor 1 (32%), factor 2 (10%), factor 3 (30%), factor 4 (18%), factor 5 (8%).

5.4.3. EFA discussion

The EFA aimed to uncover whether a wide range of commonly cited EBTs could be organised under latent factors. The analyses indicated that the EBTs included in this study could be explained by 5 factors and in total, the model explained 64% of variance in the data. The amount of variance explained is typical for psychological research where total variance explained is generally between 50%-60% (Hair, 2009; Pett et al., 2003). At this stage of the analysis, it is important to examine the loadings of EBTs onto the 5 factors to understand what the similarities are between the EBTs

that load together and to tentatively suggest labels for each factor. The research team reviewed and agreed the proposed labels before testing the proposed model with CFA. This was important to improve the face validity of the framework.

Factor 1: Reactive Eating

Factor 1 included food responsiveness, hedonic hunger, external eating, susceptibility to hunger, binge eating, disinhibition, and positive emotional eating, which all loaded positively onto the factor. Additionally, satiety responsiveness loaded negatively onto factor 1. The similarities between these EBT's are that they refer to automatic, impulsive, or unconscious motivators of (over)eating. For example "I often feel hungry when I am with someone who is eating" (AEBQ_FR), "If I see or small a food I like, I get a powerful urge to have some" (PFS), "when I see a real delicacy, I often get so hungry that I have to eat if right away," (TFEQ_H), "if you see or small something delicious, do you have a desire to eat it?" (DEBQ_EX), "sometimes when I start eating, I just can't seem to stop," (TFEQ_D), "I have a desire to eat when I am joyful" (PNEES_P), and "I feel incapable of controlling my urges to eat" (BES). These scales refer to motivations to eat that are either not conscious or deliberative and instead where eating is stimulated by some automatic process such as recognition of the food itself, a cue associated with food, reward associated with eating or hedonic-driven eating. In the case of positive emotional eating, whilst the emotion is positive, whereas other EBTs appear more negative or unwanted, the motivation to eat is still something unconscious as indicated within the term "desire" to eat.

It is interesting that satiety responsiveness loaded negatively onto factor 1. Example items from this scale include "I often leave food on my plate at the end of a meal," and "I often get full before my meal is finished." Although the loading of AEBQ_SR was not overly strong onto this factor, the small-moderate loading suggests that some part of this EBT is similar to the above mentioned EBTs. The similarities could refer to undereating that is associated with these items e.g., "get full up" and "leave food." If this scale was reversed, it could refer to being unable to "get full up" or overeating and always finishing a plate of food. This does not exclusively refer to unconscious motivations to eat, but does relate to aspects of the other scales that ask about experiences with overeating e.g. "I usually eat too much…" (TFEQ_D) and

“do you eat more than usual when…” (DEBQ_EX). Consequently, we propose that this factor explains eating styles that are *reactive* in nature and therefore we labelled this domain of eating “reactive eating,” This domain, could be given a different label depending on the context it is being used. For example, behavioural theories, such as dual-process theory (Strack & Deutsch, 2004), may refer to this domain as impulsive, whilst cognitive theories may refer to this domain as automatic or implicit (Hermans et al., 1998; Williamson et al., 2004). Therefore, it is important to emphasise that this domain is label is tentative and context dependent.

Factor 2: Restricted Eating

Factor 2 included positive loadings of TFEQ restraint and DEBQ restraint, as well as unlimited permission to eat, which loaded negatively onto this factor. Both restraint EBTs refer to cognitive and executive reasoning which involves eating behaviour as a consequence of a deliberate, decision process. For example, “I consciously hold back at meals in order not to gain weight,” (TFEQ_R), and “do you deliberately eat less in order not to become heavier?” (DEBQ_R). However, they also refer to a dieting style where food is consciously restricted to maintain a certain weight. Many of the items refer to this restriction of eating, with phrases such as “watch portion size,” “consciously eat less,” “deliberately take small helpings,” “deliberately eat foods that are slimming,” and “try to eat less.” When these EBTs are viewed as referring to a controlled eating style where specific foods and portion sizes are limited, it becomes clear how unconditional permission to eat loaded negatively onto this factor. For example, “I have forbidden foods that I don’t allow myself to eat,” and “I try to avoid certain foods…” are items that reversed coded to form the IES_UPE scale. The reverse of the IES_UPE scale, therefore, also refers to “limited permission to eat” which is very similar to the core behaviours associated with restrained eating. To reflect the high level of control and restriction of food, this factor was consequently labelled “restricted eating.” This domain of eating can be seen as most similar to the reflective system of dual-process theory, whereby behaviour is the consequence of a decision process. Therefore, this domain could be labelled “reflective eating,” however, we chose the term “restricted eating” because these EBTs specifically try to limit food intake, which is not necessarily what general reflective eating would aim to do.

Factor 3: Negative Emotional Overeating

This domain included emotional overeating, negative emotional eating, and disinhibition with positive loadings onto factor 3. Eating for physical rather than emotional reasons and emotional undereating loaded negatively onto this factor. When considering the reverse of IES_EPR and AEBQ_EUE, these EBTs all refer to negative emotion driven overeating. For example, “I eat more when I am annoyed,” (AEBQ_EOE), “I tend to eat when I am grumpy,” (PNEES_N), “when I feel blue, I often overeat” (TFEQ_D), “I find myself eating when I’m, feeling emotional” (reverse coded, IES_EPR), and “I eat less when I’m worried,” (AEBQ_EUE). These scales all refer to emotional motivations for eating, and specifically negative emotions and overeating, rather than undereating. Consequently, this domain is labelled “negative emotional overeating.” The fact that positive emotional eating did not load with these EBTs suggests that this domain specifically measures negative emotions that motivate eating and therefore, the research team agreed on the label ‘negative emotional overeating.’ However, aside from positive emotional eating, none of the EBTs included in this analysis refer to positive emotions that drive eating. This could be the reason why positive emotional eating did not load with these other emotional EBTs. This is a limitation of the model, but also a limitation of available EBTs. Perhaps if these EBTs included both positive and negative emotions within their scales, this domain could be extended to include more diverse emotions including positive emotions.

The addition of disinhibition in this domain could be seen as surprising, because disinhibition aims to measure loss of control over eating and opportunistic eating, which would theoretically fit better under reactive eating. However, previous research has found an independent dimension within disinhibition that reflects emotional eating. For example, Ganley (1988) found that the disinhibition subscale split into two independent factors, one of which was labelled emotional eating. Bond et al. (2001) found three disinhibition factors, of which one was associated with negative affective states and was labelled emotional susceptibility. In another analysis of the TFEQ, the highest loadings of items in the disinhibition scale were with the emergent emotional eating factor (Karlsson et al., 2000). The authors suggested that “clearly, items on emotional eating were the backbone of the Disinhibition scale,” but also that emotional eating items are a “unique factor.” Consequently, our findings are

consistent with these previous findings. Disinhibition does have a cross-loading loading of 0.37 onto the reactive factor, which indicates that this EBT refers to automatic motivations to eat as well as negative emotional driven overeating. Therefore, because of the cross-loadings of disinhibition onto factor 1 and 3, this EBT is included in both factors. Furthermore, our findings that negative emotional eating exists as a separate domain to reactive eating is also consistent with previous research that suggests there are strong psychometric reasons that emotional eating should be regarded as a "freestanding construct" (Ganley, 1988; Hyland et al., 1989; Karlsson et al., 2000). Again, however, the EBTs used in these studies refer only to negative emotions that drive eating. Although the authors refer to emotional eating in general, these analyses can only refer to negative emotional eating. More research that includes positive emotions is needed to understand if positive emotional eating also exists as a unique factor.

Factor 4: Homeostatic Eating

The fourth factor identified by the EFA includes three subscales of intuitive eating. These scales refer to an ability to listen and act on the body's internal cues. More specifically, the RHSC subscale measures sensitivity to internal hunger and satiety cues with items such as "I trust my body to tell me when to eat." Items from the BFCC subscale measures the congruency between an individual's food choice and intake and their bodies' needs, e.g. "I mostly eat food that give my body energy and stamina" (Tylka & Kroon Van Diest, 2013). The UPE subscale reflects an individual's willingness to eat when hungry and to not stave off hunger e.g. "If I am craving a certain food, I allow myself to have it."

These scales appear to be measuring what could be considered a proxy of homeostatic eating. Homeostatic eating is defined as eating determined by energy deficits, in contrast with hedonic eating which is defined as eating influenced by palatability (Lowe & Butryn, 2007). While no EBT has been purposely designed to measure homeostatic eating, these scales do appear to be measuring conscious awareness of sensations that can be considered as related to homeostatic eating. This type of eating differs from restricted or reflective eating because it measures an ability to listen and act on internal cues which requires a level of interoceptive awareness and is not the same as and can be quite distinct from cognitive control of

eating. Although there are some EBTs that could be considered related to homeostatic eating, it would be useful to develop trait measures of homeostatic eating and then test whether a specifically designed measure of homeostatic eating loads with these proxy measures.

Factor 5:

The last factor included satiety responsiveness, emotional undereating, and self-regulation of eating. Satiety responsiveness and emotional undereating loading positively onto this factor, whilst self-regulation of eating behaviour loaded negatively onto the factor. Regarding the positive loadings, both subscales focus on "eating less", "getting full up easily" and leaving food for different reasons. The satiety responsiveness scale includes items that refer to "get full up," and "often leave food," which aims to measure the individual's ability to respond to their satiety cues. Emotional undereating also measures "eating less", but this not due to satiety responsiveness and instead due to negative emotions, e.g. "I eat less when I'm worried/anxious/upset/angry." Overall, these subscales could be measuring dimensions of undereating.

Of interest, self-regulation of eating behaviour, loaded negatively onto this factor. The SREB was designed to measure self-regulatory capacity which refers to multiple processes in goal-directed behaviour and the ability to bridge the intention-behaviour gap (Kliemann et al., 2016). Theoretically, this EBT fits with dietary restraint due to the similarities between cognitive control of eating (restraint) and goal-directed behaviour (SREB). Indeed, the authors who created the SREB suggest that dietary restraint overlaps with self-regulation (Herman & Mack, 1975; Johnson et al., 2012), but they also highlight a key difference that restraint also measures a range of personality traits and eating tendencies such as weight fluctuation, self-efficacy, and food choices that the SREB does not (Laessle et al., 1989; Williamson et al., 2007). On examining the EFA more closely, SREB did also have a small cross-loading of 0.28 onto Factor 2, which indicates that it shares some similarities with the restricted eating domain. However, because this was a relatively small loading, there are differences between these EBTs.

Interestingly, SREB also had a moderate negative loading onto the reactive factor (-0.30). This would suggest that items in the SREB are negatively related to reactive eating and could measure deliberate, controlled and conscious measures of (normal/under) eating (the reverse of reactive EBTs). In support of this idea, SREB was significantly and negatively correlated with all reactive traits and positively correlated with satiety responsiveness and emotional undereating. It is therefore surprising that SREB negatively loaded onto factor 5, when it was positively associated with satiety responsiveness and emotional undereating. The negative loading, means that items that are reverse coded to give the SREB scale are items that load positively onto factor 5, for example “I give up too easily on my eating intentions,” and “I easily get distracted from the way I intend to eat.” It is unclear how these items relate to satiety responsiveness and emotional undereating, and therefore, what this factor is measuring. Due to the inconsistency of the direction of effect of the factor loadings and correlations between EBTs, we hesitate to give this factor a label for the current study. Possibly, conducting the CFA with this fifth factor may elucidate how these EBTs are related.

Summary of EFA findings:

The objective of the EFA was to categorise EBTs into latent factors. The analysis revealed that these EBTs could be effectively grouped into five latent factors, explaining 64% of the data's variance, a typical level in psychological research, but also indicating there is potentially more remaining variance to be explained. To enhance the framework's validity, the research team reviewed and agreed upon the proposed labels before confirming the model's accuracy through CFA. Factor 1, labelled "Reactive Eating," included EBTs reflecting automatic and impulsive motivations for eating, driven by cues, food, rewards, and emotions. Factor 2, termed "Restricted Eating," comprised EBTs indicating cognitive control of eating, with specific emphasis on restricted eating. Factor 3, "Negative Emotional Overeating," captured EBTs linked to eating driven by negative emotions. Factor 4, "Homeostatic Eating," encompassed EBTs related to intuitive eating, signifying an ability to listen and act on internal hunger and satiety cues, which is distinct from cognitive control. Factor 5 has not been given a label and thus further research is essential to uncover if this factor exists and what this factor is measuring. While these findings offer valuable insights into motivations for eating, certain EBTs exhibited cross-loadings,

which indicates additional research will be required to refine and validate the model. The assigned labels are provisional and context-sensitive, acknowledging the intricate nature of eating behaviour. A key element of 'context' in this discussion is the set of EBT measures used in the first place. The next section of this paper utilised CFA to validate the domains identified by the EFA.

5.4.4. Data Preparation (CFA):

Global diagnostic indicators indicated strong factorability of the correlation matrix for all EBT in the test dataset (Kaiser–Meyer Olkin = 0.88, Bartlett's test of sphericity x^2 = 5695.84, $p < .001$). EBTs were entered into the CFA based on the rotation matrix suggested by the EFA. The ML estimation method was used. The CFA was analysed from the covariance matrix and the latent variables were allowed to correlate. Lastly, the full sample (n = 1658) was used to examine whether the final factor model could predict external outcomes (BMI and weight change). The final model was first set to predict the full sample, and then this model was set to predict both BMI and weight change. The CFA was conducted using the package 'lavaan' (Rosseel, 2012). Figures were plotted using the 'semPlot' package (Epskamp & Stuber, 2019).

5.4.5. Results: CFA

The first model tested did not meet the criteria for goodness of fit and was unable to find a solution. The CFA indicated that some observed variances were a factor 1000 times larger than others. On examining the EBT loadings, satiety responsiveness had very low loadings onto factor 1 (-245.25) and factor 5 (-245.08). Additionally positive emotional eating had a low loading onto factor 1 (0.34). Satiety responsiveness and positive emotional eating were removed from the model. This meant that the fifth factor included only 2 EBT's and because CFA requires 3 or more EBT's, the fifth factor could not be tested for the second model. The second model was able to find a solution and had a better fit than the first model (χ^2= 599.61, df = 82, $p < 0.001$, CFI = 0.90, RMSEA = 0.11, SRMR = 0.08, good fit criteria, CFI > 0.90, RMSEA < 0.06, SRMR < 0.08, (Bentler, 1990; Kenny, 2014; Kline, 2015)). However, emotional undereating had a low loading onto factor 3 (loading = -0.43) and could be a problematic variable since the items for emotional

undereating are identical to emotional overeating, except for the direction of effect (“I eat more” AEBQ_EOE, “I eat less” AEBQ_EUE), which makes emotional undereating redundant. Consequently, emotional undereating was removed from the model. The third model had a much stronger fit and met the good criteria of fit indices for CFI and SRMR (χ^2= 495.18, df = 69, $p < 0.001$, CFI = 0.91, RMSEA = 0.11, SRMR = 0.07, see Table 5.4 and Figure 5.2).

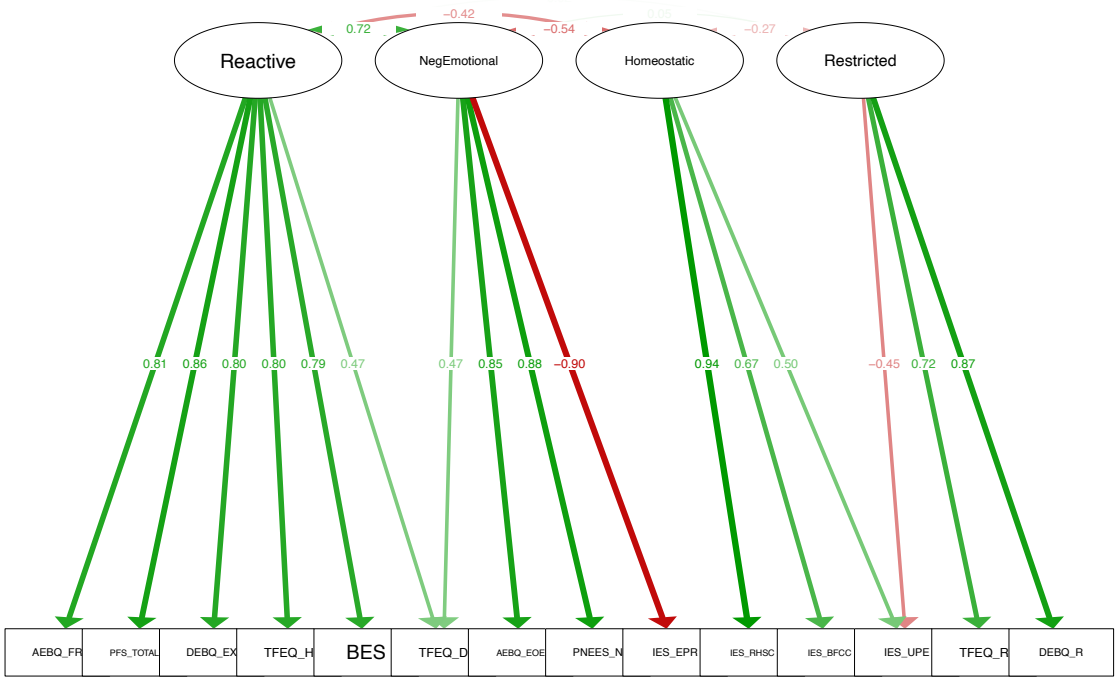
Lastly, due to previous research suggesting that reactive and emotional EBT’s can be subsumed under one latent factor (Price et al., 2015; Vainik et al., 2015), a fourth model was tested which included three factors. The first factor included both reactive and negative emotional EBTs, with factor 2 including homeostatic EBTs and factor 3 including restricted EBTs. However, this three-factor model combing reactive eating with negative emotional overeating, did not meet the criteria of fit and had lower fit indices than model 3 (χ^2= 958.60, df = 73, $p < 0.001$, CFI = 0.82, RMSEA = 0.16, SRMR = 0.09). Consequently, the third model was retained and used as the final model to predict external outcomes.

Table 5.4. Loadings of EBTs onto their proposed latent variables for CFA model 3

Latent Variables	Model 3
Reactive eating	
Food responsiveness	0.81
Hedonic hunger	0.86
External eating	0.80
Susceptibility to hunger	0.80
Binge eating	0.79
Disinhibition	0.47
Negative Emotional Overeating	
Emotional overeating	0.85
Negative emotional eating	0.88
Eating for physical rather than emotional reasons	-0.90
Disinhibition	0.47
Homeostatic eating	
Reliance on hunger and satiety cues	0.94
Body-food choice congruence	0.67
Unlimited permission to eat	0.50
Restricted eating	
Restraint (TFEQ)	0.72
Restraint (DEBQ)	0.87
Unlimited permission to eat	-0.45

Note. TFEQ = Three-Factor Eating Questionnaire, DEBQ = Dutch Eating Behaviour Questionnaire.

Figure 5.2. CFA model 3



Note. Thicker lines represent stronger loadings onto the proposed factor. Reactive = Reactive Eating, NegEmotional = Negative Emotional Overeating, Homeostatic = Homeostatic Eating, Restricted = Restricted Eating, AEBQ_FR = Food Responsiveness, PFS = Power of Food, TFEQ_H = Susceptibility to Hunger, DEBQ_EX = External Eating, TFEQ_D = Disinhibition, BES = Binge Eating, AEBQ_EOE: Emotional Overeating, PNEES_N = Negative Emotional Eating, IES_EPR: Eating for Physical Reasons, IES_RHSC = Reliance on Hunger and Satiety, IES_BFCC = Body-Food Choice Congruence, IES_UPE = Unlimited Permission to Eat, TFEQ_R = Restraint, DEBQ_R = Restraint.

5.4.6. CFA and external outcomes

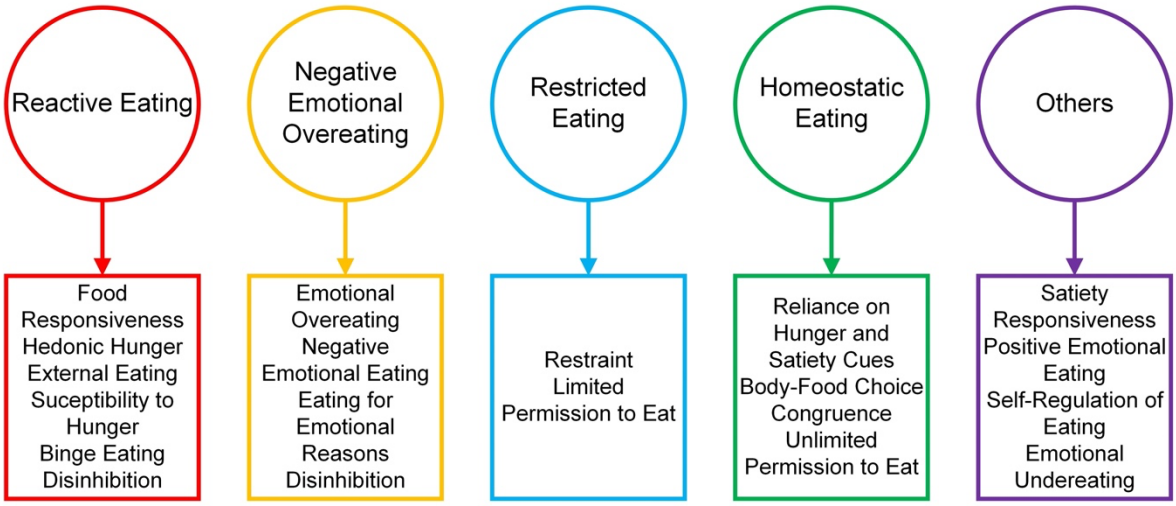
The full sample was used to predict BMI and weight change. First, the final model (model 3) was set to predict the full sample and a good fit was found (χ^2= 1421.25, df = 69, p < 0.001, CFI = 0.92, RMSEA = 0.11, SRMR = 0.07). This model was then asked to predict BMI (χ^2= 1295.96, df = 79, p < 0.001, CFI = 0.92, RMSEA = 0.10, SRMR = 0.07), and all factors significantly predicted BMI with each factor explaining the following variance (reactive = 12%, p <.001, negative emotional = 18%, p <.001, homeostatic = -41%, p <.001, and restricted = 21%, p < .001). There was a positive effect of reactive, negative emotional and restrictive eating on BMI suggesting that individuals with a higher BMI show higher reactive, negative emotional and restricted eating. Whereas there was a negative effect of homeostatic eating on BMI, suggesting that individuals with a higher BMI show lower homeostatic eating.

When the model was asked to predict self-reported weight change (χ^2= 993.53, df = 70, p < 0.001, CFI = 0.93, RMSEA = 0.09, SRMR = 0.06), only homeostatic (17%, p <.001) and restricted eating (-9%, p = .005) predicted significant variance in weight change. There was a positive effect of homeostatic eating which suggests that individuals who gained weight show higher homeostatic eating, whereas individuals who lost weight showed higher restricted eating.

5.5. General Discussion

The aim of the current paper was to investigate the organisational structure of commonly cited EBTs within underlying, latent factors, followed by evaluating the fitness of the proposed EBT model using hold-out data previously unseen by the EFA. The EFA revealed support for a five-factor structure of EBTs, affirming the study's objective and indicating that distinct domains can be used to categorise EBTs. Subsequently, the CFA indicated support for the adequacy of a four-factor model, suggesting that EBTs within each domain can be used interchangeably. For a visual representation, refer to Figure 5.3. This study marks the beginning of an emerging framework of EBTs, and therefore, it is important for future research to continue developing the model by adding more EBTs and testing the domain structure, which may result in branching away from the original framework.

Figure 5.3. A graphical representation of the CFA and framework of EBTs



Previous research has suggested that EBTs can be grouped into one (Vainik et al., 2015) or two (Price et al., 2015) underlying factors. The current study supports the previous finding of one factor measuring uncontrolled eating or food reward responsivity. The reactive factor of the current analysis conceptually overlaps with these factors as the EBTs included in the reactive factor contain various items that pertain to food cue responsiveness and loss of control over eating. Furthermore, the current analysis identified another factor “restricted eating” which also supports the dietary restriction factor that emerged in the two-component model (Price et al.,

2015). Additionally, whilst previous research suggests that reactive and emotional EBTs can be subsumed under one factor, the current study's findings suggest that reactive and negative emotional EBTs are distinct and should form two separate factors. We also extended previous research by including EBTs that have not been examined previously, for example, intuitive eating and additional emotional eating measures. The inclusion of these EBTs led to additional factors "homeostatic eating" and "negative emotional eating" emerging from the analysis. Consequently, the current study has been able to conceptually replicate and extend previous research to identify four factors that underlie commonly cited EBTs.

Through attempting to understand the current state of EBTs, parallels can be drawn with personality psychology. The Big Five (Goldberg, 1990), Five-Factor Model (McCrae & John, 1992) and HEXACO domains (Ashton & Lee, 2020) are instrumental personality constructs that have become the default way of measuring individual differences in personality. Whilst they are useful for summarising the ways in which people can differ in terms of personality, there is a lack of evidence that they can explain behaviour, the psychological processes underling personality, or that they can predict real-world outcomes (Mõttus et al., 2020). Another problem with these personality domains is that the processes that specifically contribute to variance in each dimension is unknown. Furthermore, the domains overlap, we do not know what sets each domain apart, and the domains can be broken down into broader (DeYoung, 2006) and more specific traits (McCrae & Sutin, 2018). These limitations can all be used to critique our current knowledge of eating behaviour. EBTs also overlap and can be broken down into broader and more specific traits. There is also a lack of strong evidence for how EBTs explain the psychological processes the underlie eating behaviour and real-world outcomes such as short-term indices of energy balance like energy intake, or longer-term indices like BMI and weight change. Consequently, the current study sought to address some of these critiques by examining the underlying factor structure of commonly cited EBTs and then testing whether these factors can predict real-world outcomes.

However, the analyses of the current study were heavily shaped by the availability of EBT measurements. Regrettably, there were very few potentially restricted or homeostatic EBTs analysed. The inclusion criteria for EBTs were developed with the

aim of identifying a wide range of EBTs, but there is still an unbalanced number of commonly cited and validated EBTs from each domain available in the literature. Therefore, there needs to be a consideration as to why so many scales focus on reactive and emotional eating. Indeed, Vainik et al. (2019) highlight that newly developed EBTs tend to correlate >0.5 with already existing EBTs, even though they are proposed to measure something new. The authors suggest that the incremental validity of new EBT questionnaires should be rigorously tested and not assumed just because they have a different 'new' name. This would help crystalise and clarify whether there are additional factors and help resolve the jangle fallacy that limits eating behaviour research. Indeed, this approach has been taken to characterising and designing behaviour change interventions, where there exists a large number of frameworks. Michie et al. (2011) identified 19 frameworks and evaluated them according to three criteria. From this, the behaviour change wheel (BCW) was developed to address the limitations of previous frameworks and improve the design and effectiveness of behaviour change interventions.

Overall, the conclusion drawn from these analyses prompts consideration of whether any of these models provide the ideal fit. This raises questions about the adequacy of the current measurement tools. Essentially, there is a need for more diverse and refined measures, particularly those capable of better distinguishing between items. Additionally, it could be that the current study did not utilise a diverse population of participants which could be contributing to the model fit. For example, the sample is limited by an uneven gender ratio of participants and an uneven ratio of dieters (Slimming World members) and the public. The subsequent discussion delves into some of the potential challenges and considerations of the model, with the intention of gaining a more comprehensive understanding of the current state of EBT research and identifying directions for future investigations.

5.5.1. Interchangeability of EBTs

Overall, the CFA identified that EBTs appear to be measuring four distinct patterns of eating (See Figure 5.2 and 5.3). Within these domains, EBTs all seem to be measuring much of the same thing. However, while this suggests that many of these scales are interchangeable, this does not mean that the actual constructs are

interchangeable, rather, this study could be highlighting flaws with the design of these commonly cited scales. The analysis suggests that the scales used to measure EBTs are not measuring distinct factors. For example, food responsiveness is interchangeable with external eating, however, there are two ways to interpret these findings. The constructs could be measuring the same one underlying factor, which suggests the constructs are not different from one another. If this is the case, research could design questions that try to capture reactive eating. On the other hand, the items that are trying to measure food responsiveness and external eating may not be designed well enough to measure these specific constructs and are too similar, so are asking the same questions. This would mean that these constructs are conceptually distinct. If the latter is true, it is important to understand how the design of these questionnaires could be improved to measure the constructs concerned. Whilst this analysis is not able to define what these EBT are measuring, it can provide some insights about which scales are measuring similar factors and which scales are unique. One can then translate this back to the theoretical domain to understand which constructs are important in describing eating behaviours, and which are not well captured by current measurements.

5.5.2. CFA and external outcomes

After conducting the CFA on the test dataset, we used the full sample to test whether the final model could predict various external outcomes that were measured in the sample. The results showed that the four-factor model was able to significantly predict BMI and weight change. All four factors significantly predicted BMI, whilst only homeostatic and restricted eating predicted self-reported weight change. The finding that negative emotional eating and restricted eating positively predicted BMI, supports previous research that found half of participants attributed weight regain to at least one emotional reason, and using self-regulatory strategies in a previous weight loss attempt was associated with emotional reasons for regain (Sainsbury et al., 2019). This indicates the tension of weight management referred to by Greaves et al. (2017).

Homeostatic eating was the only factor that negatively predicted BMI, however, it was positively correlated with weight change. This finding suggests that homeostatic

eating may not be useful for weight loss but could be useful during weight maintenance. Indeed, previous research has shown that higher levels of intuitive eating (included under homeostatic eating as per the CFA) was associated with weight maintenance compared to weight cycling (Tylka et al., 2020). Individuals with higher levels of homeostatic eating should be better able to assess their physiological signals and internal cues but this does not mean engaging in homeostatic eating is useful for weight loss. This is because evidence suggests that energy balance in regulated but the regulation is asymmetric, meaning there is a tendency to overeat more than undereat, which may become pronounced in response to prolonged negative energy balances (Stubbs et al., 2023). The current findings support this idea which suggests that whilst homeostatic eating may not lead to a negative energy balance (weight loss), it is associated with a lower BMI and therefore could be used to improve long-term weight maintenance. In contrast to homeostatic eating, restricted eating was found to positively predict BMI, but negatively predict weight change. This could suggest that engaging in restricted eating is useful for short term (6 month) weight loss, but over the longer-term, it is associated with having a higher BMI. In support of this finding, a previous meta-analysis found that restraint (using all available measures) was negatively correlated with short-term energy intake, but positively correlated with BMI (Dakin et al., 2023). Consequently, individuals with higher level of restricted eating, are more likely to be "unsuccessful dieters" who are prone to overeating (disinhibition), leading to increased BMI (Ouwens et al., 2003).

The CFA showed that the reactive and negative emotional eating factors have high correlations with each other which could suggest that one-factor could be formed. However, when the model was asked to predict external outcomes, negative emotional eating does appear to be a useful factor because it was able to independently predict variance in BMI and, and even predicted more variance in BMI than the reactive factor. Overall, using the model to predict external outcomes has shown that each factor is valuable and independently contributes to both BMI and weight change. Conducting these analyses has also shown that engaging in reactive eating, negative emotional eating and restricted eating is associated with negative health outcomes including higher BMI, and weight gain (for reactive and negative emotional eating). It would therefore be useful for future research to examine

whether interventions that target reducing reactive eating, negative emotional eating and restricted eating can lead to improved health outcomes.

5.5.3. Where do positive emotions fit in the framework?

Another surprising finding from this analysis, was that positive emotional eating did not load onto the emotional domain, and instead loaded onto the reactive domain in the EFA. A-priori, we might expect positive emotional eating to group with other emotional eating EBTs and therefore this domain would measure general emotional eating. However, due to the small negative loading onto this domain, we argue that this domain measures specifically negative emotional eating. When examining the CFA, positive emotional eating had a relatively weak loading onto the reactive factor, which it was proposed to underlie based on the EFA. This suggests that there are some items in this scale that measure reactive, unconscious, and automatic eating, though, in general, positive emotional eating does not fit well under reactive eating. This could also be because generally, reactive eating EBTs measure more negative aspects of overeating. Positive emotional eating may also be diffusely related to hedonic eating, assuming that pleasure is associated in human experience with positive emotions. Due to its lack of loading onto any factors, positive emotional eating was removed from the analysis entirely. We add this EBT to the “other” section, alongside measures which the framework does not try to explain such as disordered eating (see inclusion criteria for selecting questionnaires).

Whilst the current model was not able to include positive emotional eating, this does not mean positive emotional eating is not a useful EBT. Rather, this EBT is a unique construct which has been overlooked by other questionnaire developers. The factor analysis approaches used in the current analysis are limited because as they are data reduction techniques, they can only reduce data if there are redundancies. Due to their being only one EBT that includes items related to positive emotions, there are no redundancies available. The current state of affairs of EBTs means there are a lot of questionnaires that cover common themes such as lack of control and negative emotional eating, whereas there is a lack of original questionnaires such as positive emotional eating and eating for pleasure. In order to have a comprehensive

map of eating behaviour, the sampling of EBTs needs to be wider and more balanced, which means that highly repeated EBTs should be eliminated.

5.5.4. How can we interpret “other” EBTs that do not fit into this model?

While the current factor model does account for a wide range of EBTs, the “others” category shown in figure 5.3 depicts EBTs that do not fit into this model. The EFA indicated that a 5th factor existed in the train dataset, however, the CFA indicated that satiety responsiveness did not load onto this factor and thus it was removed from the model leaving only two EBT’s able to load onto the factor. Consequently, the 5th factor could not be tested using CFA, meaning satiety responsiveness, self-regulation of eating, and emotional undereating were removed from the model. We have reason to believe that these EBTs are important and therefore, highlight that the current model is not final. If new EBTs were to be analysed, this could change the model structure and fit. For example, as explained previously, positive emotional eating may fit into this model if more EBTs that measure positive emotions were developed and included. Additionally, further research is needed to identify whether a 5th factor does exist. Similarly, if a specific measure of homeostatic eating were to be developed, it would be important to test this EBT within the current model to examine whether satiety responsiveness can be included and where it would fit.

Furthermore, a limitation of the current paper is that a measure of food craving was not included. Food craving is defined as an “intense desire to consume a particular food or food type that is difficult to resist” (Weingarten & Elston, 1990). Cravings are hedonic responses to food, which can be intense and specific to certain food types (Hill, 2007). Research has shown that measures of trait food craving are positively associated with external eating, restrained eating, emotional eating, binge eating, weight cycling and disordered eating (Hill, 2007; Taylor, 2019). Additionally, in participants with and without eating disorders, food craving has been positively associated with consumption of craved foods and ad libitum energy intake (Kemps & Tiggemann, 2013; Taylor, 2019). Consequently, trait food craving could be an important construct that measures motivations for eating. The food craving questionnaire trait (FCQ-T) (Cepeda-Benito et al., 2000) measures how cravings are

usually manifested in a specific individual or population (e.g., individuals with obesity). The trait measure of food craving has been shown to be more stable than the state measure and includes 9 factors such as intention and planning to consume food, anticipation of relief from negative states and craving as a physiological state. Future research should aim to include a measure of trait food craving to understand whether it can fit into a model of EBTs and how it relates to other motivations for eating constructs.

Another potentially important construct is eating for pleasure. A considerable driver of obesity has often been suggested to come from greater pleasure from eating (De Graaf, 2005; Nasser, 2001). Indeed, Berridge et al. (2010) argue that trends of increasing body weight are the result of increased availability of foods interacting with a brain reward system that generate pleasure for foods. However, remarkably an EBT that specifically measures eating for pleasure does not exist. The most similar EBT that currently exists, would be positive emotional eating. These EBTs may covary, and for some people positive emotional eating would be a source of eating for pleasure. Yet, these EBTs are not identical. As such, it would be useful to specifically design an EBT that measures the extent to which individuals eat for pleasure.

5.5.5. Conclusion

To summarise, the current paper has demonstrated that various EBTs share underlying common constructs, allowing them to be organised into a single framework. The analyses support a four-factor structure which suggests that most EBTs can be explained by four constructs: reactive, emotional, restricted, and homeostatic eating. This suggests that within these domains, EBTs can be used interchangeably. Additionally, all four factors were able to significantly predict external outcomes, meaning each factor is useful for predicting real-world outcomes. There are, however, EBTs that cannot be explained by this factor structure and were removed from the analysis. It is important to replicate this factor structure, particularly in the general population, to understand whether the excluded EBTs are unique and distinct from the four proposed domains of eating. Additionally, the label of homeostatic eating for factor 4 may not be the best label for what this domain is

measuring. These insights provide a framework to better understand EBTs including what underlying domains of motivation to eat they are measuring.

5.5.6. Recommendations and Future Directions

To further enhance our understanding of EBTs and refine the proposed ongoing model, several recommendations and avenues for future research are suggested. A critical issue of this framework is the imbalanced distribution of commonly cited and validated EBTs across different domains. The current paper used the sum-score factor analysis approach (using subscales) rather than an item-based approach. This approach was taken because the aim was to examine currently existing EBTs. Using an item-based approach would likely deconstruct some scales, most likely the binge eating scale, due to this scale contributing to four separate factors. Moving forward, taking an item-based approach to the factor analysis would lead to a more balanced choice of measures. Using this approach would eliminate highly correlated items which should lead to a more even model. Consequently, we aim to extend the current analysis by using the same questionnaires but analysing the data at single item level. Both models should then be compared and examined to confirm whether these same four factors emerge.

Furthermore, researchers should continue developing this model by exploring and validating a wider array of EBTs and rigorously testing the domain structure. This iterative process might lead to the expansion or refinement of the framework to better capture the nuanced aspects of eating behaviours. Future research should also focus on developing more refined measures, capable of better discerning between items. Improved measurement tools could aid in capturing the unique nuances of various eating behaviours, contributing to a better model fit. For example, researchers should consider whether satiety responsiveness represents its own unique domain or whether intuitive eating subscales are measuring additional eating behaviours that are not related to homeostatic eating. Developing targeted EBTs specifically focused on homeostatic eating might help clarify the existence of homeostatic eating within this framework. Developing a EBT related to eating for pleasure should be a priority. The closest EBT to eating for pleasure could be the pleasure subscale from The Eating Motivation Survey (TEMS) (Renner et al., 2012).

Future research should examine the TEMS pleasure subscale to understand if it can be incorporated into the current model.

There is also the potential existence of a fifth (or more as yet unmeasured) factor that indicates lack of self-regulation that requires further examination. Future studies could focus on designing EBTs that capture these aspects, allowing for a comprehensive assessment of this potential domain. Given the potential impact of participant demographics on model fit, future studies should aim to include a more diverse range of participants. A balanced representation of diverse populations might lead to a more accurate reflection of the domains of eating behaviour. In conclusion, while this study has paved the way for an initial understanding of the organisation of EBTs, there remain numerous avenues for exploration and refinement. We offer recommendations for future research, aimed at enriching our understanding of motivations for eating and advancing the development of an accurate and comprehensive framework for categorising EBTs.

5.6. References

Allison, D. B., Downey, M., Atkinson, R. L., Billington, C. J., Bray, G. A., Eckel, R. H., Finkelstein, E. A., Jensen, M. D., & Tremblay, A. (2008). Obesity as a disease: a white paper on evidence and arguments commissioned by the Council of the Obesity Society. *Obesity*, *16*(6), 1161.

Ashton, M. C., & Lee, K. (2020). Objections to the HEXACO model of personality structure—And why those objections fail. *European Journal of Personality*, *34*(4), 492-510.

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological bulletin*, *107*(2), 238.

Berridge, K. C., Ho, C.-Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: pleasure and desire circuits in obesity and eating disorders. *Brain research*, *1350*, 43-64.

Bond, M., McDowell, A., & Wilkinson, J. (2001). The measurement of dietary restraint, disinhibition and hunger: an examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). *International Journal of Obesity*, *25*(6), 900-906.

Cepeda-Benito, A., Gleaves, D. H., Williams, T. L., & Erath, S. A. (2000). The development and validation of the state and trait food-cravings questionnaires. *Behavior Therapy*, *31*(1), 151-173.

Costello, A., & Osborne, J. (2005). Best pr Best practices in explor actices in exploratory factor analysis: Four or analysis: Four recommendations for getting the most from your analysis. *Pract. Assess. Res. Eval*, *10*, 1-10.

Dakin, C., Beaulieu, K., Hopkins, M., Gibbons, C., Finlayson, G., & Stubbs, R. J. (2023). Do eating behavior traits predict energy intake and body mass index? A systematic review and meta-analysis. *obesity reviews*, *24*(1), e13515.

Davis, C. (2013). From passive overeating to “food addiction”: a spectrum of compulsion and severity. *International Scholarly Research Notices*, *2013*.

De Graaf, C. (2005). Sensory responses, food intake and obesity. *Food, diet and obesity*, 137-159.

DeYoung, C. G. (2006). Higher-order factors of the Big Five in a multi-informant sample. *Journal of personality and social psychology*, *91*(6), 1138.

Epskamp, S., & Stuber, S. (2019). semPlot: Path diagrams and visual analysis of various SEM packages' output. *R package version*, *1*(2).

Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological methods*, *4*(3), 272.

Foster, G., Wadden, T., Swain, R., Stunkard, A., Platte, P., & Vogt, R. (1998). The Eating Inventory in obese women: clinical correlates and relationship to weight loss. *International Journal of Obesity*, *22*(8), 778-785.

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.

Ganley, R. M. (1988). Emotional eating and how it relates to dietary restraint, disinhibition, and perceived hunger. *International Journal of Eating Disorders*, *7*(5), 635-647.

Goldberg, L. R. (1990). An alternative" description of personality": the big-five factor structure. *Journal of personality and social psychology*, *59*(6), 1216.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive behaviors*, *7*(1), 47-55.

GOV.UK. (2011). *Ethnicity Facts and Figures*. Retrieved 31/03/2022 from https://www.ethnicity-facts-figures.service.gov.uk/

GOV.UK. (2014). *Qualifications in the Population*. Retrieved 31/03/2022 from https://www.gov.uk/government/statistical-data-sets/fe-data-library-qualifications-in-the-population-based-on-the-labour-force-survey

Greaves, C., Poltawski, L., Garside, R., & Briscoe, S. (2017). Understanding the challenge of weight loss maintenance: a systematic review and synthesis of qualitative research on weight loss maintenance. *Health Psychology Review*, *11*(2), 145-163.

Hair, J. F. (2009). Multivariate data analysis.

Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of personality*.

Hermans, D., Pieters, G., & Eelen, P. (1998). Implicit and explicit memory for shape, body weight, and food-related words in patients with anorexia nervosa and nondieting controls. *Journal of abnormal psychology*, *107*(2), 193.

Hill, A. J. (2007). The psychology of food craving*: Symposium on 'Molecular mechanisms and psychology of food intake'. *Proceedings of the Nutrition Society*, *66*(2), 277-285.

Holt, J. K. (2004). Item parceling in structural equation models for optimum solutions. annual meeting of the mid-western educational research association,

Hunot, C., Fildes, A., Croker, H., Llewellyn, C. H., Wardle, J., & Beeken, R. J. (2016). Appetitive traits and relationships with BMI in adults: Development of the Adult Eating Behaviour Questionnaire. *Appetite*, *105*, 356-363.

Hyland, M. E., Irvine, S. H., Thacker, C., Dann, P. L., & Dennis, I. (1989). Psychometric analysis of the Stunkard-Messick Eating Questionnaire (SMEQ)

and comparison with the Dutch Eating Behavior Questionnaire (DEBQ). *Current Psychology*, *8*(3), 228-233.

Johnson, F., Pratt, M., & Wardle, J. (2012). Dietary restraint and self-regulation in eating behavior. *International Journal of Obesity*, *36*(5), 665-674.

Karlsson, J., Hallgren, P., Kral, J., Lindroos, A.-K., Sjöström, L., & Sullivan, M. (1994). Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. *Appetite*, *23*(1), 15-26.

Karlsson, J., Persson, L.-O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International Journal of Obesity*, *24*(12), 1715-1725.

Kelley, T. L. (1927). Interpretation of educational measurements.

Kemps, E., & Tiggemann, M. (2013). Olfactory stimulation curbs food cravings. *Addictive behaviors*, *38*(2), 1550-1554.

Kenny, D. (2014). Measuring model fit (http://davidakenny. net/cm/fit. htm). *Accessed January*.

Kliemann, N., Beeken, R. J., Wardle, J., & Johnson, F. (2016). Development and validation of the self-regulation of eating behaviour questionnaire for adults. *International Journal of Behavioral Nutrition and Physical Activity*, *13*(1), 1-11.

Kline, R. B. (2015). *Principles and practice of structural equation modeling*. Guilford publications.

Laessle, R. G., Tuschl, R. J., Kotthaus, B. C., & Prike, K. M. (1989). A comparison of the validity of three scales for the assessment of dietary restraint. *Journal of abnormal psychology*, *98*(4), 504.

Llewellyn, C., & Wardle, J. (2015). Behavioral susceptibility to obesity: gene–environment interplay in the development of weight. *Physiology & Behavior*, *152*, 494-501.

Lowe, M. R., & Butryn, M. L. (2007). Hedonic hunger: a new dimension of appetite? *Physiology & Behavior*, *91*(4), 432-439.

Lowe, M. R., Butryn, M. L., Didie, E. R., Annunziato, R. A., Thomas, J. G., Crerand, C. E., Ochner, C. N., Coletta, M. C., Bellace, D., Wallaert, M., & Halford, J. (2009). The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*, *53*(1). https://doi.org/https://dx.doi.org/10.1016/j.appet.2009.05.016

McCrae, R. R., & John, O. P. (1992). An introduction to the five-factor model and its applications. *Journal of personality*, *60*(2), 175-215.

McCrae, R. R., & Sutin, A. R. (2018). A five–factor theory perspective on causal analysis. *European Journal of Personality*, *32*(3), 151-166.

Michie, S., Van Stralen, M. M., & West, R. (2011). The behaviour change wheel: a new method for characterising and designing behaviour change interventions. *Implementation science*, *6*(1), 1-12.

Mõttus, R., Wood, D., Condon, D. M., Back, M. D., Baumert, A., Costantini, G., Epskamp, S., Greiff, S., Johnson, W., & Lukaszewski, A. (2020). Descriptive, predictive and explanatory personality research: Different goals, different approaches, but a shared need to move beyond the Big Few traits. *European Journal of Personality*, *34*(6), 1175-1201.

Nasser, J. (2001). Taste, food intake and obesity. *obesity reviews*, *2*(4), 213-218.

NHS Digital. (2019). *Weight*. Retrieved 31/03/2022 from http://healthsurvey.hscic.gov.uk/data-visualisation/data-visualisation/explore-the-

trends/weight.aspx#:~:text=Adults'%20average%20BMI&text=In%202019%20the%20mean%20BMI,(kg%2Fm2).

ONS. (2020a). *Health Survey for England 2019*. Retrieved 14/06/2022 from https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2019

ONS. (2020b). *Population estimates for the UK, England and Wales, Scotland and Northern Ireland: mid-2020*. Retrieved 31/03/2022 from https://www.ons.gov.uk/peoplepopulationandcommunity/populationandmigration/populationestimates/bulletins/annualmidyearpopulationestimates/latest#age-structure-of-the-uk-population

Ouwens, M. A., van Strien, T., & van der Staak, C. P. (2003). Tendency toward overeating and restraint as predictors of food consumption. *Appetite*, *40*(3), 291-298. https://www.sciencedirect.com/science/article/abs/pii/S0195666303000060?via%3Dihub

Pett, M. A., Lackey, N. R., & Sullivan, J. J. (2003). *Making sense of factor analysis: The use of factor analysis for instrument development in health care research*. sage.

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., Despres, J.-P., Bouchard, C., & Lemieux, S. (2004). Eating behaviours, dietary profile and body composition according to dieting history in men and women of the Quebec Family Study. *British Journal of Nutrition*, *91*(6), 997-1004.

Renner, B., Sproesser, G., Strohbach, S., & Schupp, H. T. (2012). Why we eat what we eat. The Eating Motivation Survey (TEMS). *Appetite*, *59*(1), 117-128.

Revelle, W. (2022). *psych: Procedures for Psychological, Psychometric, and Personality Research.* In (Version R package version 2.2.5) https://CRAN.R-project.org/package=psych.

Rosseel, Y. (2012). *lavaan: An R Package for Structural Equation Modeling.* In

Sainsbury, K., Evans, E. H., Pedersen, S., Marques, M. M., Teixeira, P. J., Lähteenmäki, L., Stubbs, R. J., Heitmann, B. L., & Sniehotta, F. F. (2019). Attribution of weight regain to emotional reasons amongst European adults with overweight and obesity who regained weight following a weight loss attempt. *Eating and Weight Disorders-Studies on Anorexia, Bulimia and Obesity*, *24*, 351-361.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and social psychology review*, *8*(3), 220-247.

Stubbs, R. J., Horgan Graham, Robinson Eric, Hopkins Mark, Dakin Clarissa, & Graham, F. (2023). Diet composition and energy intake in humans. *In Press*.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Sultson, H., Kukk, K., & Akkermann, K. (2017). Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. *Appetite*, *116*, 423-430.

Tabachnick, B. G., Fidell, L. S., & Ullman, J. B. (2013). *Using multivariate statistics* (Vol. 6). pearson Boston, MA.

Taylor, M. (2019). A review of food craving measures. *Eating behaviors*, *32*, 101-110.

Tylka, T. L., Calogero, R. M., & Danielsdottir, S. (2020). Intuitive eating is connected to self-reported weight stability in community women and men. *Eating Disorders*, *28*(3). https://doi.org/http://dx.doi.org/10.1080/10640266.2019.1580126

Tylka, T. L., & Kroon Van Diest, A. M. (2013). The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men. *Journal of counseling psychology*, *60*(1), 137.

Vainik, U., Garcia-Garcia, I., & Dagher, A. (2019). Uncontrolled eating: a unifying heritable trait linked with obesity, overeating, personality and the brain. *The European journal of neuroscience*, *50*(3). https://doi.org/https://dx.doi.org/10.1111/ejn.14352

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229-239.

Van Strien, T., Frijters, J. E., Bergers, G. P., & Defares, P. B. (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.

Weingarten, H. P., & Elston, D. (1990). The phenomenology of food cravings. *Appetite*, *15*(3), 231-246.

Wickham, H., François, R., Henry, L., & Müller, K. (2022). *dplyr: A Grammar of Data Manipulation*. In https://dplyr.tidyverse.org, https://github.com/tidyverse/dplyr.

Williamson, D. A., Martin, C. K., York-Crowe, E., Anton, S. D., Redman, L. M., Han, H., & Ravussin, E. (2007). Measurement of dietary restraint: validity tests of four questionnaires. *Appetite*, *48*(2), 183-192.

Williamson, D. A., White, M. A., York-Crowe, E., & Stewart, T. M. (2004). Cognitive-behavioral theories of eating disorders. *Behavior modification*, *28*(6), 711-738.

Chapter 6. Investigating motivations to eat: Refining and validating a framework of Eating Behaviour Traits in dieters and the general population

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6.1. Abstract

Due to relationships between diet and health including obesity, there is a need to examine the explanatory power of factors that motivate people to (over or under) eat. In a previous investigation, a four-factor subscale-based model of eating behaviour traits (EBT) was developed which identified individual differences in psychological factors influencing motivations to eat and some residual uncertainties. The current study used a data-driven and theory-driven approach, including individual items to refine and extend previous EBT models. The aim was to examine and validate the domain structure of a framework for EBTs. The analysis used two samples including a representative sample of the UK population (n = 2010, 51% female, 49% male, 18 – 88 years), and members of a weight management program (n = 2317, 96.6% female, 2.8% male, 21 – 84 years), who completed 5 questionnaires including 10 EBTs. The results found some support for a 6-factor model, encompassing reactive eating, negative emotional eating, positive emotional eating, restricted eating, homeostatic eating, and body-food choice congruence (data-driven model) or eating for pleasure (theory-driven model). There were differences between the data-driven model and the theory-driven model regarding the 6th factor. Additionally, the data-driven model did not distinguish between eating for pleasure and reactive eating. The models demonstrated that the eating behaviour factors were significantly associated with BMI category. Overall, this research contributes to a more structured understanding of the dimensions of motivation underlying EBTs, emphasising the utility of this framework for identifying at-risk individuals and tailoring interventions to meet specific individual needs.

6.2. Introduction

The rising prevalence of obesity is a significant public health concern with 64% of adults in England in 2021 to 2022 being classified as living with overweight or obesity (GOV.UK, 2023b). This has led to scientific interest in identifying individual differences in psychological factors influencing eating behaviour, including obesity development and management. Over the past four decades, the examination of eating behaviour has generated numerous theoretical models and constructs that measure motivations for (over)eating. These constructs are collectively termed Eating Behaviour Traits (EBT). EBTs are measured through self-report questionnaires and have been implicated as causal factors in overeating and obesity (French et al., 2012; Llewellyn & Wardle, 2015). However, the abundance of questionnaires designed to measure EBTs has raised concerns about potential redundancies and a lack of standardised measures, contributing to a jangle fallacy in obesity research (Vainik et al., 2015).

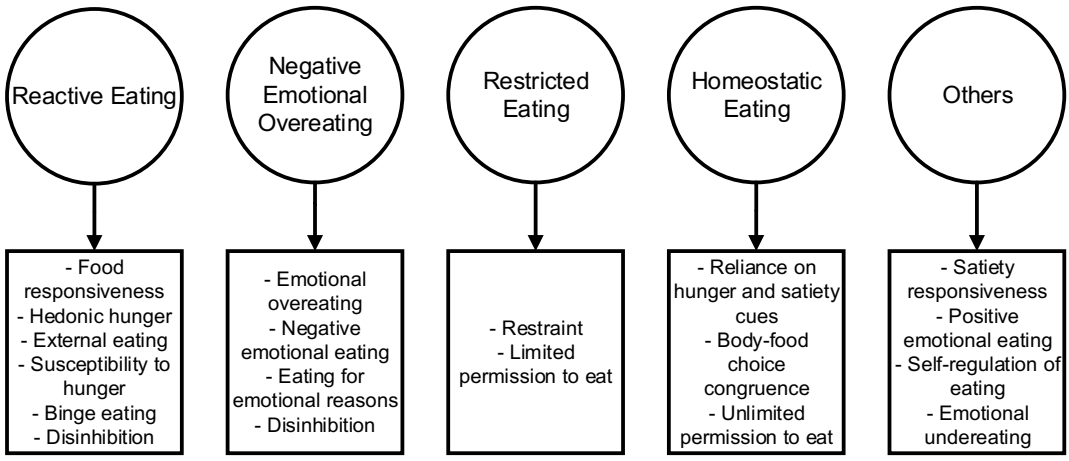
Previous research has examined EBTs and developed models with the aim of examining the similarities and uniqueness between commonly cited traits. Models developed by Vainik et al. (2015) and Price et al. (2015) have laid the groundwork for understanding the underlying structure of these traits. The uncontrolled eating model attempted to integrate various EBT constructs under a single latent factor with EBTs measuring differing severities along a continuum of uncontrolled eating ranging from "homeostatic eating" to severe bingeing (Vainik et al., 2015). Price et al. (2015) identified two underlying components of eating behaviour related to food reward responsivity and dietary restriction.

In a previous study, we used Exploratory Factor Analysis (EFA) with the aim of replicating and extending previously developed models (Dakin et al., 2024). We included EBTs that measure uncontrolled eating, as well as EBTs that measure food reward responsivity and dietary restriction. We also included EBTs that have not been analysed before, such as intuitive eating (Tylka & Kroon Van Diest, 2013) and additional measures of negative and positive emotional eating (Hunot et al., 2016; Sultson et al., 2017). An initial EFA revealed a five-factor structure. However,

conducting Confirmatory Factor Analysis (CFA) using hold-out data, indicated support for a four-factor model, suggesting that EBTs can be organised under four domains (reactive eating, negative emotional overeating, restricted eating, and homeostatic eating, see Figure 6.1). Overall, our results support a factor measuring uncontrolled eating, food reward responsivity or reactive eating, and a factor measuring dietary restriction. The results also indicate the emergence of distinct factors, including homeostatic eating and negative emotional eating.

However, there were some EBTs that the previous model could not account for which were included under the "others" domain (see Figure 6.1). For example, positive emotional eating did not load under any of the identified four factors, which emphasises the need for further exploration and refinement. There were many more negative emotional EBTs than positive emotional EBTs, which also could have influenced the domain factor leading to positive emotional eating being dropped from the analysis. As such, positive emotional eating items are included in the current analysis. Furthermore, other EBTs in the "other" category that did not load under any factor in the previous models should be examined further, however, this is beyond the scope (or capacity) of the current analysis.

Figure 6.1. A graphical representation of a framework of EBTs (Dakin et al., 2024).



The previous analysis was also limited by the availability of EBT measurements, which revealed a bias towards reactive and emotional eating measures. Additionally, a sum-score approach was used, which kept the questionnaires in their original format and loaded the total subscale into the model. Interestingly the results showed

that some subscales had cross loadings onto more than one factor, suggesting that some EBTs could be measuring more than one eating domain. For example, disinhibition from the Three Factor Eating Questionnaire loaded onto both the reactive and negative emotional factors and unlimited permission to eat from the Intuitive Eating Scale-2 loaded onto the restricted and homeostatic eating factor. However, taking a sum-score approach meant that the individual items within the subscale could not be examined to identify the potential causes of the cross-loadings. It was highlighted that future research should take an item-approach which would likely deconstruct the scales and help to understand which items are contributing to specific factors. It is also important to discover whether the current four-factor model can be replicated by analysing the data at the single item level.

This approach has been taken before with different questionnaires measuring EBTs (Racine et al., 2019; Vainik et al., 2015). For example, 7 ‘nonhomeostatic’ questionnaires (Dutch Eating Behaviour Questionnaire, Eating Pathology Symptoms Inventory, Loss of Control over Eating Scale, Binge Eating Scale, Eating Disorder Examination Questionnaire, Power of Food Scale, and Yale Food Addiction Scale), were examined using an item-based approach. Racine et al. (2019) found support for a 7-factor solution in which the items of these questionnaires measured 7 constructs which were labelled: emotional eating, external eating, loss of control over eating, overeating, distress over nonhomeostatic eating, hedonic hunger, and food addiction. These results suggest that nonhomeostatic EBTs are measuring multiple distinct constructs. However, there is still a need to examine a wider variety of EBTs including potential homeostatic EBTs. Another important contributor towards obesity has been suggested to stem from greater pleasure in eating (Berridge et al., 2010; De Graaf, 2005; Nasser, 2001). Finlayson and Dalton (2012) suggest that increases in body weight are the result of increased availability of foods interacting with a brain reward system that generate pleasure for foods. As such, eating for pleasure is a potentially important construct that could motivate eating and shape learned food preferences. However, eating for pleasure has not yet been examined in relation to other EBTs. It is therefore vital to address these previous limitations to better understand the implications of EBT models, their generalisability, and potential limitations of existing questionnaires.

The current study assessed whether commonly cited EBTs capture four or more distinct factors, using EFA and CFA. We took two approaches to the analysis: a data-driven approach and a theory-driven approach. For both approaches, an item-level approach was taken to the analysis. In the data-driven approach, EFA was conducted using a subset of the data, to explore different factor structures and item loadings. In the theory-driven approach, a theoretical model was developed utilising the theory and results from previous studies, with items set to load onto the previously identified four factors of EBTs (reactive eating, emotional eating, restricted eating, and homeostatic eating). In addition, items were also set to load onto two untested factors (positive emotional eating and eating for pleasure). This resulted in a 6-factor theoretical model.

A CFA was then conducted, using hold-out data, previously unseen by the EFA, to test and compare the data-driven model and theory-driven model. Both approaches were taken because evidence suggests that when taking a data-driven approach, it is important to also identify competing theoretical models and compare the fit of each model (Hoyle, 1995; Jackson et al., 2009). Lastly, we aimed to test the extent that EBTs can predict the BMI category of the participants. The analysis was conducted in parallel using two samples of participants involved in a weight management programme and a UK representative sample of the general population. Both samples answered the same set of questionnaires to uncover whether the results were comparable between different samples with differing dietary goals.

6.3. Methods

6.3.1. Participants

The general population (GP) sample included 2,010 participants (51% female, 49% male, 18 – 88 years), whilst the weight management sample (WM) included 2,317 participants (96.6% female, 2.8% male, 21 – 84 years, see Table 6.1). The weight management sample was significantly older ($p < .001$), had a larger BMI ($p < .001$), and included more females ($p < .001$) than the general population. For the general population sample, participants were recruited through Prolific (Prolific, 2023), where Prolific recruited a UK representative sample, based on age, gender and ethnicity. The general population sample were not targeted to be representative of BMI or education. However, they were representative of the UK population with regards to BMI, but overall had achieved a higher level of education. In 2021, the average BMI of the UK population was 27.4kg/m^2 (NHS, 2022), this is comparable to the general population sample's BMI (27.0kg/m^2). In 2023, 48.4% of the UK population (18 – 64 years old) had achieved an NQF level 4 or above (GOV.UK, 2023a), which is the equivalent to an undergraduate degree. In comparison, more of the survey general population sample had achieved a similar level of university education (57.5%). For the weight management sample, participants were recruited from the membership database of a commercial weight management company (Slimming World, UK).

Any participant was excluded if they reported they were pregnant or breastfeeding in the prior 6 months, had a history of previous eating disorder, weight loss surgery, any medical condition that affected body weight or appetite, and individuals with insufficient English language skills. Ethical approval was granted from the School of Psychology Research Ethics Committee at the University of Leeds (reference number: PSCETHS-707, date approved: 05/10/2023, see D.1).

Table 6.1. Descriptives for the general population and weight management sample

Descriptive	GP sample (SD)	WM sample (SD)
Gender:		
Women	50.5%	96.6%
Men	48.5%	2.8%
Non-binary	0.5%	0.2%
Other/Prefer not to say	0.5%	0.3%
Mean Age	45.9 years (15.7)	55.6 years (11.5)
Self-reported ethnicity:		
White	86.9%	98.1%
Asian	7.2%	0.5%
Black African & Black	3.1%	0.2%
other	1.5%	0.8%
Mixed Race		
Other Ethnic groups	1.0%	0.2%
Prefer not to say	0.1%	0.2%
Education:		
University	57.5%	34.5%
High vocational	10.2%	19.6%
Secondary School	15.7%	22.4%
Sixth Form	15.3%	18.0%
Primary School	0.1%	0.2%
No Formal Education	0.1%	1.0%
Other	1.0%	4.4%
Mean BMI	27.0 kg/m^2 (6.1)	31.4 kg/m^2 (6.8)
BMI category		
Underweight	1.9%	0%
Healthy	41.3%	14.1%
Overweight	34.1%	34.2%
Obesity	18.4%	40.5%
Severe Obesity	4.3%	11.2%

Note. GP = General Population, WM = Weight Management sample, BMI category was calculated as follows: BMI < 18.5 “underweight”, BMI ≥ 18.5-24.9 “healthy”, BMI ≥ 25-29.9 “overweight”, BMI ≥ 30-39.9 “obesity”, BMI ≥ 40 “severe obesity.”

6.3.2. Rationale for questionnaire selection

Both samples completed an online survey which included demographic questions and 5 questionnaires covering 10 EBT constructs (see Table 6.2). Our previous analyses of EBTs were limited by an imbalanced distribution of commonly cited and validated EBTs across different domains (Dakin et al., 2024), where there were more reactive and negative emotional EBTs included and few restricted and homeostatic EBTs included. Additionally, there was an imbalance of positive and negative emotional EBTs which could have influenced the domain factor leading to positive emotional eating being removed from the analysis. Additionally, an EBT specifically designed to measure eating for pleasure has not yet been tested to understand how it relates to other EBTs, for example positive emotional eating.

We aimed to address these limitations in the current paper. Questionnaires were chosen to measure one of each potential domain of eating behaviour (reactive, negative emotional eating, positive emotional eating, restricted eating, homeostatic eating, and eating for pleasure). For all EBTs except for eating for pleasure (which has not been previously tested), the questionnaire was chosen because it loaded strongly onto its proposed factor (Dakin et al., 2024), and it has been shown to be significantly associated with objective outcomes e.g., BMI and weight change (Dakin et al., 2023). These questionnaires were thought to measure a balanced range of eating behaviour domains and met the following criteria for (1) measure a defined eating behaviour trait, (2) measure a motivation for eating, (3) formally validated in a peer-reviewed publication, (4) not be a diagnostic instrument for an eating disorder.

Table 6.2. Questionnaires measured in the study

EBT	Predicted Domain Loading	Questionnaire	Reference
Disinhibition (TFEQ_D)	Reactive, Negative Emotional	Three Factor Eating Questionnaire (TFEQ)	Stunkard and Messick (1985)
Restraint (TFEQ_R)	Restricted		
Susceptibility to hunger (TFEQ_H)	Reactive		
Reliance on hunger and satiety (IES_RHSC)	Homeostatic	Intuitive Eating Scale-2 (IES)	Tylka and Kroon Van Diest (2013)
Eating for physical rather than emotional reasons (IES_EFPR)	Negative Emotional		
Body-food choice congruence (IES_BFCC)	Homeostatic		
Unlimited permission to eat (IES_UPTE)	Restricted		
Emotional Overeating (AEBQ_EOE)	Negative Emotional	Adult Eating Behaviour Questionnaire (AEBQ)	Hunot et al. (2016)
Positive Emotional Eating (PNEES_P)	Positive Emotional	Positive and Negative Emotional	Sultson et al. (2017)

		Eating Scale (PNEES)	
Eating for Pleasure (TEMS)	Pleasure	The Eating Motivation Survey	Renner et al. (2012)

Three Factor Eating Questionnaire (TFEQ)

The full 51-item questionnaire was used as a measure of three behavioural and cognitive dimensions of eating. The restraint subscale (TFEQ_R, 21 items, α = 0.84 (GP), α = 0.68 (WM)) measures the tendency to restrict food intake to control body weight. Disinhibition (TFEQ_D, 16 items, α = 0.84 (GP), α = 0.83 (WM)), measures tendency towards overeating. The last subscale of the TFEQ is susceptibility to hunger (TFEQ_H, 14 items, α = 0.85 (GP), α = 0.84 (WM)). See D.2 for information on the recoding and scoring of the TFEQ subscales.

Intuitive Eating Scale-2 (IES)

The reliance on hunger and satiety subscale (IES_RHSC, 6 items, α = 0.88 (GP), α = 0.86 (WM)), captures an individual's trust and reliance on their internal satiety and hunger cues to guide their behaviour. Eating for physical rather than emotional reasons (IES_EPR, 8 items, α = 0.92 (GP), α = 0.92 (WM)), measures individuals' patterns of eating, whether they eat because they are physically hungry or to cope with distress. Unconditional permission to eat (IES_UPE, 6 items, α = 0.73 (GP), α = 0.52 (WM)), measures an individual's willingness to eat when hungry and someone who rejects labelling certain foods as forbidden. Lastly, the body-food choice congruence (IES_BFCC, 3 items, α = 0.85 (GP), α = 0.86 (WM)), assesses how well an individual matches their food choices with their body's needs.

Adult Eating Behaviour Questionnaire (AEBQ)

The full questionnaire aims to measure appetitive traits in adults. Only the emotional overeating subscale was used because other EBTs measured by the AEBQ were either not of interest to the current study, did not measure a specific motivation for eating, or similar/overlapping EBTs were already included in the study (AEBQ_EOE, 5 items, α = 0.93 (GP), α = 0.91 (WM)). This subscale measures the extent to which individuals overeat when emotional. All five items were used in both samples.

Positive and Negative Emotional Eating Scale

The positive emotional eating subscale (PNEES_P, 7 items, α = 0.89 (GP), α = 0.86 (WM)), captures eating in response to positive emotions. All seven items were used in both samples. The negative emotional eating subscale (PNEES_N) was not included in this analysis because the emotional overeating subscale (AEBQ) and eating for physical rather than emotional reasons (IES_EPR) subscale were included, both of which measure negative emotional eating. The rationale to support these choices is that these subscales were also found to have stronger loadings on the negative emotional eating factor than the PNEES_N in the EFA of our previous analysis, and similar factor loadings in the CFA of our previous analysis (Dakin et al., 2024).

The Eating Motivation Survey

The pleasure subscale from the eating motivation survey (TEMS, 5 items, α = 0.80 (GP), α = 0.76 (WM)), measures the motivation to please oneself through food. All five items were used in both samples.

6.3.3. Measures of External Outcomes

BMI was calculated based on participants' self-reported height and weight using the standard formula (kg/m^2). BMI category was calculated as follows: BMI < 18.5 "underweight", BMI ≥ 18.5-24.9 "healthy", BMI ≥ 25-29.9 "overweight", BMI ≥ 30-39.9 "obesity", BMI ≥ 40 "severe obesity."

6.3.4. Procedure

For both samples, participants were presented with an information sheet and consent form that included confirmation of eligibility requirements and were invited to complete a series of further questionnaires, if they consented to take part in the study. Participants were informed that the online survey would take no longer than 35 minutes to complete. After completing informed consent, participants recruited through Prolific entered their Prolific ID, whilst participants recruited from the weight management sample entered their member ID and email. Participants were also asked if they are willing for their email to be used for a prize draw (for WM participants). All participants completed demographics questions, and participants

were randomly counterbalanced to either complete the eating behaviour questionnaires (see Table 6.2) then questions about specific food items or complete the food item questions, and then the eating behaviour questionnaires. All eating behaviour questionnaires were presented in a random order to the participants. The questions about specific food items were not relevant to the current study and will be published elsewhere. At the end of the survey, participants were shown a debrief statement and contact information was provided to allow participants to contact the researchers with any questions. Participants who were recruited through Prolific returned to the Prolific website to approve their submission and once approved, these participants were reimbursed with £4.50 for their time. Participants who were recruited via the weight management program, were entered into a prize draw to win one of five high street shopping vouchers worth £100.

6.3.5. Data analysis

Total scores were created for each EBT by averaging responses. The total scores were checked for normality and the Shapiro-Wilk test for normality indicated that all variables were not normally distributed. However, all variables, except for BMI in both samples were within normal ranges for skewness and kurtosis (Kline, 2015) and appeared normally distributed according to the QQ plots. BMI was log transformed (LG_BMI) in both samples due to its skewed distribution. Outliers were identified via boxplot and extreme outliers (above 3rd quartile + 3*interquartile range [IQR] and below 1st quartile – 3*IQR). In the general population, one extreme outlier was identified for the log transformed BMI variable, and in the weight management sample, one extreme outlier was identified for IES_BFCC. However, these outliers were not thought to be erroneous and were kept in the analysis. Pearson's correlations were conducted between questionnaire total scores and BMI (LG_BMI). Additionally, mean scores for each EBT subscale were compared between the general population and the weight management sample. Firstly, unadjusted univariate general linear models (GLM) examined the impact of the sample (GP vs WM) on each EBT subscale, entering sample as the fixed factor (see D.4 for the unadjusted model results). Then adjusted models were conducted, entering LG_BMI and age as covariates and gender as fixed factors. Only the adjusted models are reported in the main text. Data preparation, correlations and GLM's were conducted

using SPSS version 29 (IBM Corp, 2022). Figures for the GLM's were plotted in R version 4.1.1 using the package 'ggplot2' (Wickham, 2016).

Before conducting EFA or CFA, items from each EBT were examined and the research team discussed each item and agreed whether the item met the criteria for measuring an EBT. Each item had to specifically measure an eating behaviour (1) and the eating behaviour had to measure a specific motivation to eat/overeat or motivation to not eat (2). Any items that did not meet this criterion were removed from the analysis. Table D.3 presents the item labels to be used in the analysis, their description, whether they were included in the analysis and a reason if they were omitted. Overall, 18 items were removed from the analysis, due to not meeting the above criteria. In addition to discussing which items should be included in the analysis, the research team also discussed what factor each item should load onto. This meant that the original subscale was altered by excluding some items (if they did not meet the EBT criteria) and changing the factors some items loaded onto (see Table D.3). This formed the development of the theoretical model. The theoretical model was derived from previous analyses suggesting that at least four factors should exist (reactive eating, negative emotional overeating, restricted eating, and positive eating). The research team also agreed that positive emotional eating and eating for pleasure should be tested as distinct factors, resulting in a 6-factor theoretical model. EFA and CFA were conducted in R version 4.1.1, using the 'psych' (Revelle, 2022), 'dplyr' (Wickham et al., 2022), and 'lavaan' (Rosseel, 2012) packages. Figures were plotted using the 'semPlot' package (Epskamp & Stuber, 2019).

Exploratory Factor Analysis (EFA)

The general population sample was utilised to conduct the EFA. The GP dataset was split at random into test and train datasets with a split of 50/50. The train dataset (n = 1005) was used to conduct EFA on the data to give an indication as to which EBT items could be subsumed under a given number of factors. The EFA was set to 4-10 factors and each model was compared to identify the best fit. The factoring method used was maximum likelihood analysis (ML) because ML enables the computation of a wide range of indices of goodness of fit and allowed statistical significance testing of factor loadings (Fabrigar et al., 1999). An oblique rotation method was used

because factors were expected to correlate and oblique rotation theoretically creates a more accurate and reproducible solution (Costello & Osborne, 2005). Since there is no widely preferred method of oblique rotation, direct oblimin was used (Fabrigar et al., 1999). Items attaining a loading of 0.32 or higher on any factor were retained (Tabachnick et al., 2013). The EFA models were compared and the model with the best fit indices (highest TLI, lowest RMSEA, and highest variance explained), whilst retaining distinct factors (as discussed by the research team) was chosen to be tested using CFA.

Confirmatory Factor Analysis (CFA)

CFA was then used to test the data-driven model developed from the EFA and the theoretical model. The models were compared in the test general population dataset (n = 1005) and then inspected and confirmed in the weight management sample (n = 2317). The data-driven and theory-driven models were used to predict BMI category, with BMI category specified as an ordinal variable. CFA models were built, treating items as categorical variables using the WLSM estimator, and using pairwise deletion to handle missing variables. The CFA was analysed from the covariance matrix and the latent variables were allowed to correlate.

6.4. Results

6.4.1. Correlations between total subscales and BMI

All subscales were related to BMI in at least one of the samples (see Tables 6.3 and 6.4). Most subscales were significantly correlated with BMI in both samples. However, restraint and positive emotional eating was not related to BMI in the general population, and unlimited permission to eat was not related to BMI in the weight management sample.

Table 6.3. Correlations between total subscales and BMI in the general population

	1	2	3	4	5	6	7	8	9	10	11
LG_BMI	--										
TFEQ_D	.39**	--									
TFEQ_R	-.02	.04	--								
TFEQ_H	.24**	.68**	-.13**	--							
IES_RHSC	-.31**	-.51**	-.08**	-.36**	--						
IES_EPR	-.32**	-.77**	.00	-.56**	.44**	--					
IES_BFCC	-.25**	-.38**	.30**	-.35**	.34**	.38**	--				
IES_UPE	-.09**	-.19**	-.66**	.00	.30**	.15**	-.16**	--			
AEBQ_EOE	.27**	.70**	.02	.49**	-.36**	-.80**	-.28**	-.16**	--		
PNEES_P	-.01	.24**	-.06*	.34**	.02	-.25**	-.11**	.08**	.19**	--	
Pleasure	.09**	.36**	-.20**	.43**	-.06**	-.38**	-.20**	.23**	.31**	.43**	--

Note. LG_BMI = log transformed BMI. TFEQ_D = disinhibition, TFEQ_R = restraint, TFEQ_H = susceptibility to hunger, IES_RHSC = reliance on hunger and satiety cues, IES_EPR = eating for physical rather than emotional reasons, IES_BFCC = body-food choice congruence, IES_UPE = unlimited permission to eat, AEBQ_EOE = emotional overeating, PNEES_P = positive emotional eating, Pleasure = eating for pleasure. ** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

Table 6.4. Correlations between total subscales and BMI in the weight management sample

	1	2	3	4	5	6	7	8	9	10	11
LG_BMI	--										
TFEQ_D	.26**	--									
TFEQ_R	-.19**	-.17**	--								
TFEQ_H	.19**	.63**	-.18**	--							
IES_RHSC	-.17**	-.51**	.17**	-.40**	--						
IES_EPR	-.27**	-.75**	.16**	-.49**	.43**	--					
IES_BFCC	-.26**	-.35**	.32**	-.32**	.37**	.34**	--				
IES_UPE	.02	-.02	-.46**	-.02	.06**	.02	-.13**	--			
AEBQ_EOE	.19**	.61**	-.11**	.37**	-.30**	-.71**	-.23**	-.02	--		
PNEES_P	.10**	.29**	-.10**	.36**	-.12**	-.26**	-.16**	.08**	.22**	--	
Pleasure	.10**	.36**	-.14**	.33**	-.13**	-.31**	-.12**	.13**	.25**	.33**	--

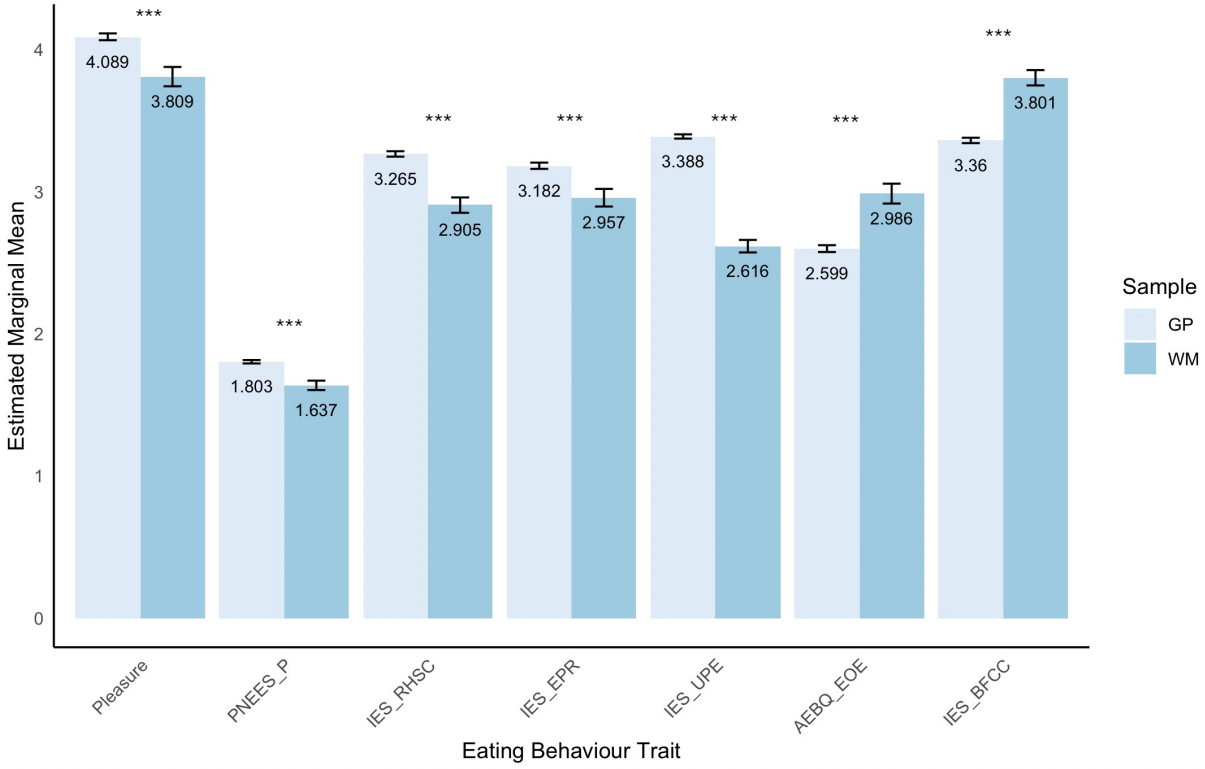
Note. LG_BMI = log transformed BMI. TFEQ_D = disinhibition, TFEQ_R = restraint, TFEQ_H = susceptibility to hunger, IES_RHSC = reliance on hunger and satiety cues, IES_EPR = eating for physical rather than emotional reasons, IES_BFCC = body-food choice congruence, IES_UPE = unlimited permission to eat, AEBQ_EOE = emotional overeating, PNEES_P = positive emotional eating, Pleasure = eating for pleasure. ** Correlation is significant at the 0.01 level (2-tailed). * Correlation is significant at the 0.05 level (2-tailed).

6.4.2. The impact of sample (GP vs WM) on mean EBT scores

When adjusting for age, BMI, and gender, there were significant differences in EBT total scores between the general population and weight management sample for all EBTs (see Table D.4 for unadjusted models). The general population showed significantly higher eating for pleasure ($F(1,3978) = 14.71$, $p < .001$, $\eta^2 = .004$), positive emotional eating ($F(1,3977) = 21.15$, $p < .001$, $\eta^2 = .005$), reliance on hunger and satiety cues ($F(1,3978) = 38.98$, $p < .001$, $\eta^2 = .010$), eating for physical rather than emotional reasons ($F(1,3978) = 11.43$, $p < .001$, $\eta^2 = .003$), unlimited permission to eat ($F(1,3978) = 267.53$, $p < .001$, $\eta^2 = .063$), and susceptibility to hunger ($F(1,3978) = 13.79$, $p < .001$, $\eta^2 = .003$), than the weight management sample. The general population also showed significantly lower disinhibition ($F(1,3978) = 4.66$, $p < .001$, $\eta^2 = .001$), restraint ($F(1,3978) = 102.56$, $p < .001$, $\eta^2 =$

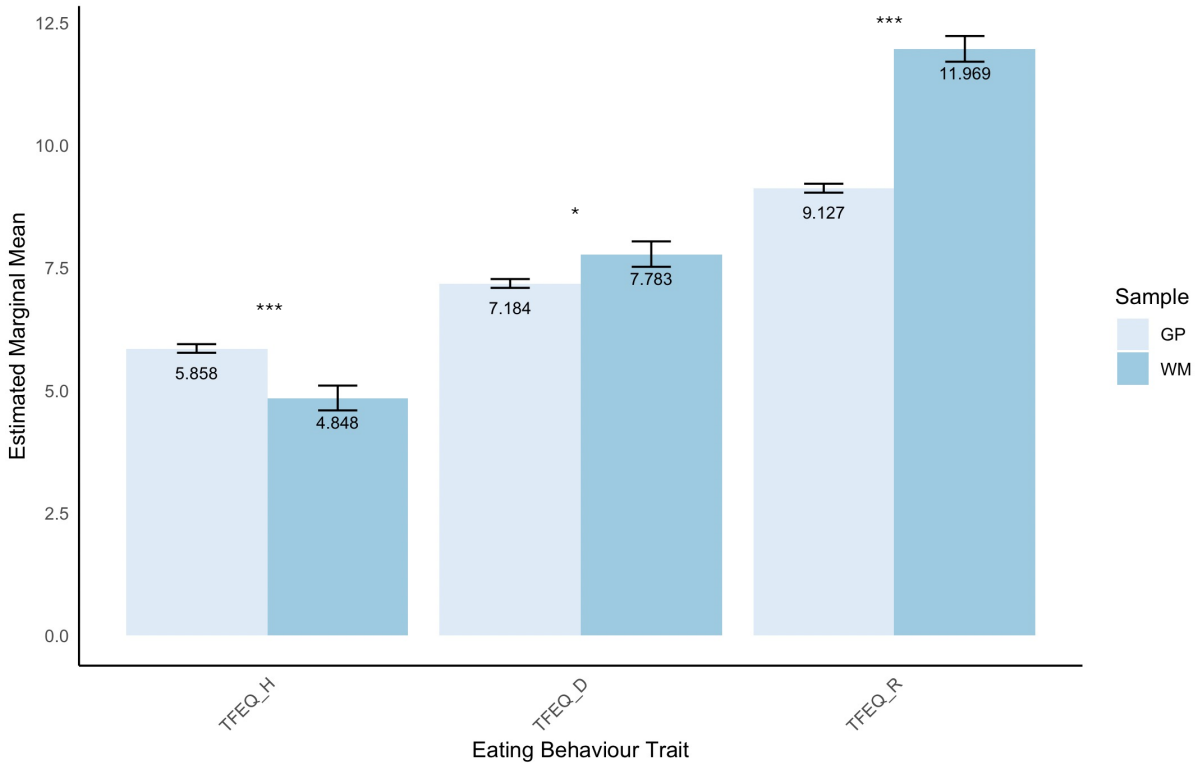
.025), emotional overeating eating (F(1,3978) = 26.78, p < .001, η^2 = .007), and body-food choice congruence (F(1,3978) = 57.781, p < .001, η^2 = .014), than the weight management sample (see Figures 6.2 and 6.3).

Figure 6.2. Estimated marginal means for eating for pleasure, positive emotional eating, intuitive eating subscales, and emotional overeating between the general population and weight management sample



Note. GP = General Population, WM = Weight Management Sample, Pleasure = eating for pleasure, PNEES_P = positive emotional eating, IES_RHSC = reliance on hunger and satiety cues, IES_EPR = eating for physical rather than emotional reasons, IES_UPE = unlimited permission to eat, AEBQ_EOE = emotional overeating, IES_BFCC = body-food choice congruence. Error bars denote standard error of the mean. Covariates appearing in the model include age and BMI. Gender is included in the model as a fixed factor. p*** < .001

Figure 6.3. Estimated marginal means for disinhibition, susceptibility to hunger and restraint between the general population and weight management sample



Note. GP = General Population, WM = Weight Management Sample, TFEQ_H = susceptibility to hunger, TFEQ_D = disinhibition, TFEQ_R = restraint. Error bars denote standard error of the mean. Covariates appearing in the model include age and BMI. Gender is included in the model as a fixed factor. p* < .05, p*** < .001

6.4.3. EFA

Global diagnostic indicators indicated strong factorability of the correlation matrix for the items in the GP train dataset (Kaiser–Meyer Olkin = 0.94, Bartlett's test of sphericity x^2 = 38332.48, p < .001). The EFA results showed that increasing the number of factors in the model increased the model fit and variance explained, with the 10-factor model providing the strongest fit (see Table 6.5). On examination of the models, the first 6 factors were consistent across the models. Factor 1 included emotional eating items from the emotional overeating subscale, eating for physical rather than emotional reasons subscale (negative loadings) and disinhibition subscale. Factor 2 included positive emotional eating items. Factor 3 included items from the body-food choice congruence subscale. Factor 4 included items from the restraint subscale and the unlimited permission to eat subscale (negative loading). Factor 5 included items from the susceptibility to hunger subscale, disinhibition subscale and the eating for pleasure subscale. Factor 6 included items from the

reliance on hunger and satiety cues subscale. The results indicated that 5 out of 6 of the factors are consistent with the theory-driven model and were therefore given matching labels (Factor 1 = negative emotional overeating, Factor 2 = positive emotional eating, Factor 4 = restricted eating, Factor 5 = reactive eating, Factor 6 = homeostatic eating). Factor 3 was not consistent with the theory-driven model and was labelled 'body-food choice congruence.'

Table 6.5. EFA fit indices and variance explained for different factor models

Number of Factors	X^2	df	p	TLI	RMSEA	Variance Explained
4	9654.91	2342	<.001	0.765	0.05	38%
5	8325.47	2273	<.001	0.799	0.04	41%
6	7217.83	2205	<.001	0.829	0.04	43%
7	6215.33	2138	<.001	0.856	0.03	44%
8	5654.97	2072	<.001	0.872	0.03	46%
9	5159.08	2007	<.001	0.884	0.03	47%
10	4717.95	1943	<.001	0.894	0.03	48%

The additional 7-10 factors included various items across the included EBTs. For example, the additional factor in the 7-factor model included some items from the eating for physical rather than emotional reasons subscale and one item from the disinhibition subscale (negative loading). In this 7-factor model, many of the items also loaded onto factor 1 (negative emotional factor). However, they loaded positively onto factor 7, whereas they loaded negatively onto factor 1. These items also had stronger loadings onto factor 1 (loadings onto factor 1 = -0.46 – -0.60) than factor 7 (0.33 – 0.44). An important aim in the development of a framework of EBTs is to understand which EBTs overlap, and which are distinct. It is therefore vital that the framework can explain distinct and different domains of eating behaviour. The additional factors in models 7-10 included items with cross-loadings onto the other 1-6 factors. Often, many of the items that loaded onto these additional factors, had much stronger loadings onto the first 6 factors than the additional 7-10 factors (as explained above). Additionally, some of these extra factors only included 1 item (e.g. one factor from the 10-factor model only included item BFCC_32). Based on the

cross-loadings, and lack of items in the extra factors to test with CFA, the 6-factor model was retained and tested using CFA. This model was chosen because it included distinct factors with no cross-loading items.

6.4.4. CFA: General Population and Weight Management Sample

CFA was conducted to test the data-driven and theory-driven model in the reserved sub-sample of the general population dataset and inspected using the weight management dataset. Both the theory-driven and data-driven model tested 5 factors with matching labels (reactive eating, restricted eating, negative emotional eating, positive emotional eating, and homeostatic eating). The data-driven model included a 6th factor of body-food choice congruence, whilst the theory-driven model included a 6th factor of eating for pleasure. In the data-driven model, the eating for pleasure items were set to load under reactive eating, whereas, in the theory-driven model, the body-food choice congruence items were set to load under the homeostatic eating factor.

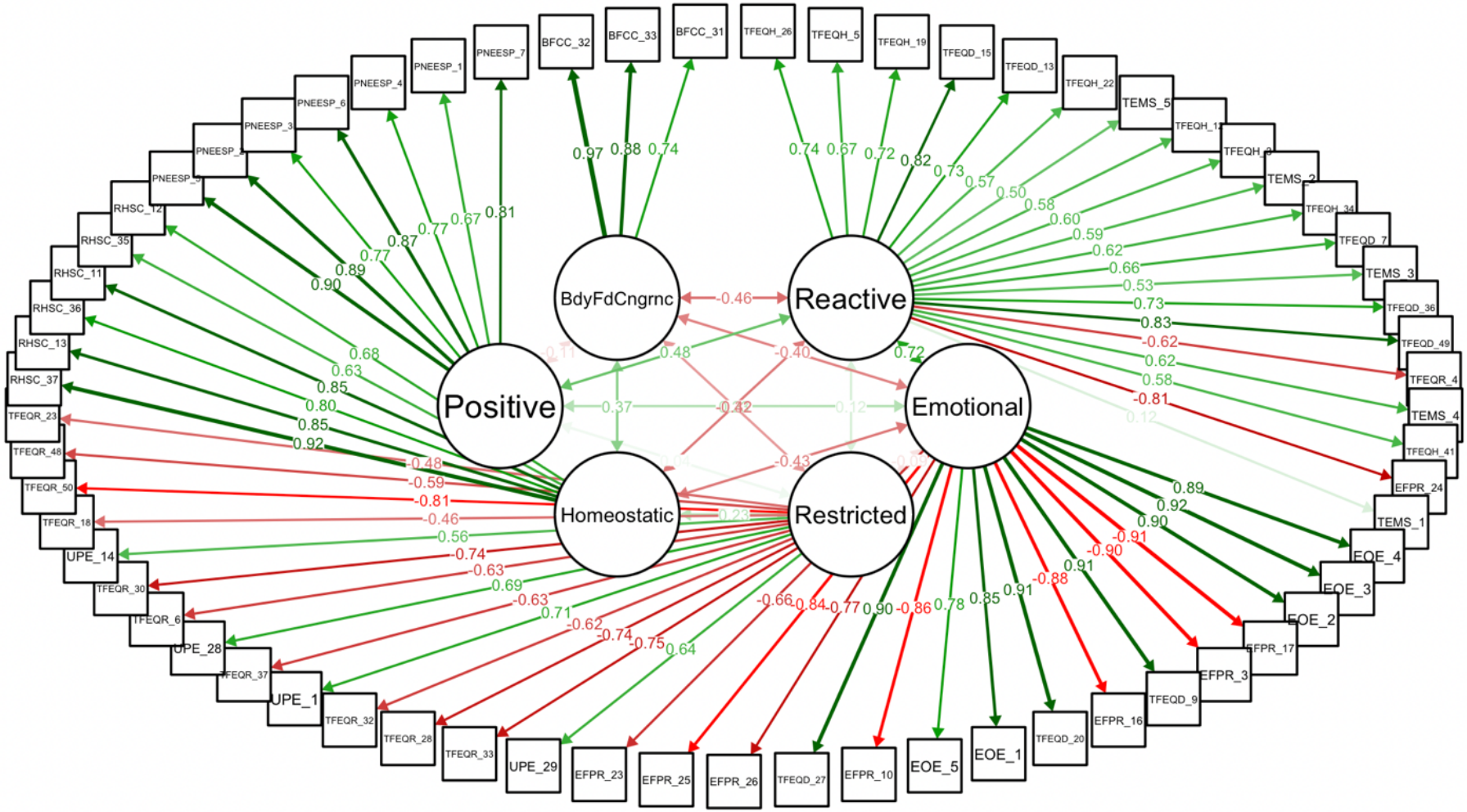
Global diagnostic indicators indicated strong factorability of the correlation matrix for the items in the general population test dataset (Kaiser–Meyer Olkin = 0.94, Bartlett's test of sphericity $x^2 = 37298.38$, $p < .001$), and the weight management dataset (Kaiser–Meyer Olkin = 0.94, Bartlett's test of sphericity $x^2 = 68679.48$, $p < .001$). The results of the data-driven model (see Model 1 and Figure 6.4) and theory-driven model (see Model 2 and Figure 6.5) in the test general population dataset are presented in Table 6.6. The results of these models in the weight management sample are also presented in Table 6.6 (see Model 3, Model 4, and Figure D.5, D.6). The results show that in the general population and weight management sample, the data-driven model had a slightly better fit indices than the theory driven model (good fit criteria = CFI > 0.90, RMSEA < 0.06, SRMR < 0.08, (Bentler, 1990; Kenny, 2014; Kline, 2015)). However, the CFIs were similar across all models and samples, and in the weight management sample, there was less difference in fit between the theory-driven and data-driven model.

Table 6.6. Fit indices for CFA models in the General Population

Model	Sample	χ^2	df	p	CFI	RMSEA	SRMR
Model 1 (data driven)	Test GP	14209.203	1999	<.001	0.971	0.078	0.089
Model 2 (theory driven)	Test GP	3883.248	2540	<.001	0.951	0.091	0.109
Model 3 (data driven)	WM	27073.846	1999	<.001	0.965	0.074	0.075
Model 4 (theory driven)	WM	35730.331	2540	<.001	0.957	0.075	0.082

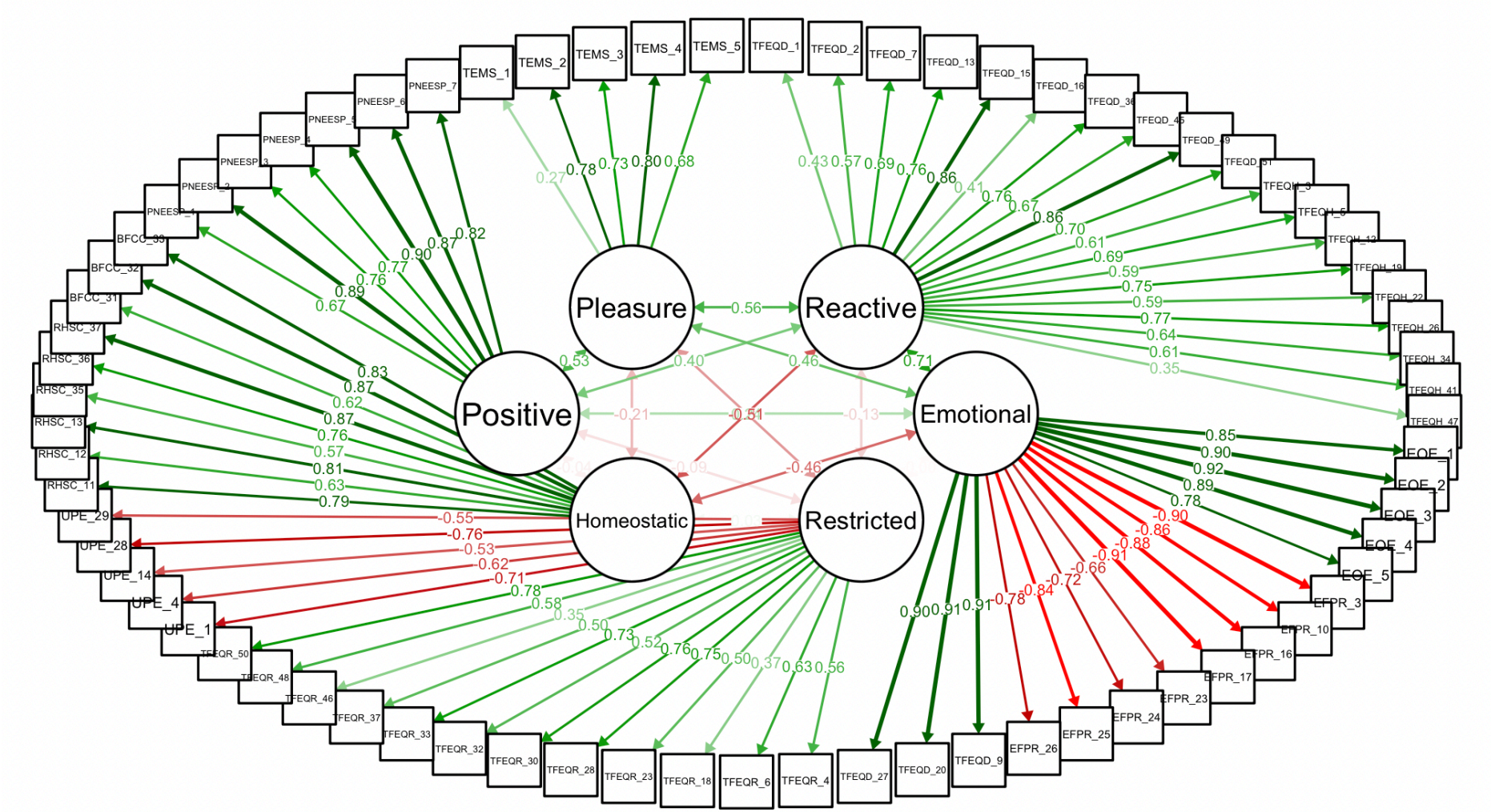
Note. GP = General Population, WM = Weight Mangement Sample, χ^2= chi squared, df=degrees of freedom, p = p-value of chi-squared. CFI = Comparative Fit Index, Root Mean Square Error of Approximation (RMSEA) Standardized Root Mean Square Residual (SRMR).

Figure 6.4. CFA model 1 (data-driven model in the general population)



Note. Green lines represent positive loadings, red lines represent negative loadings. Outer statistics denote factor loadings. Inner statistics denote covariance between factors.

Figure 6.5. CFA model 2 (theory-driven model in the general population)



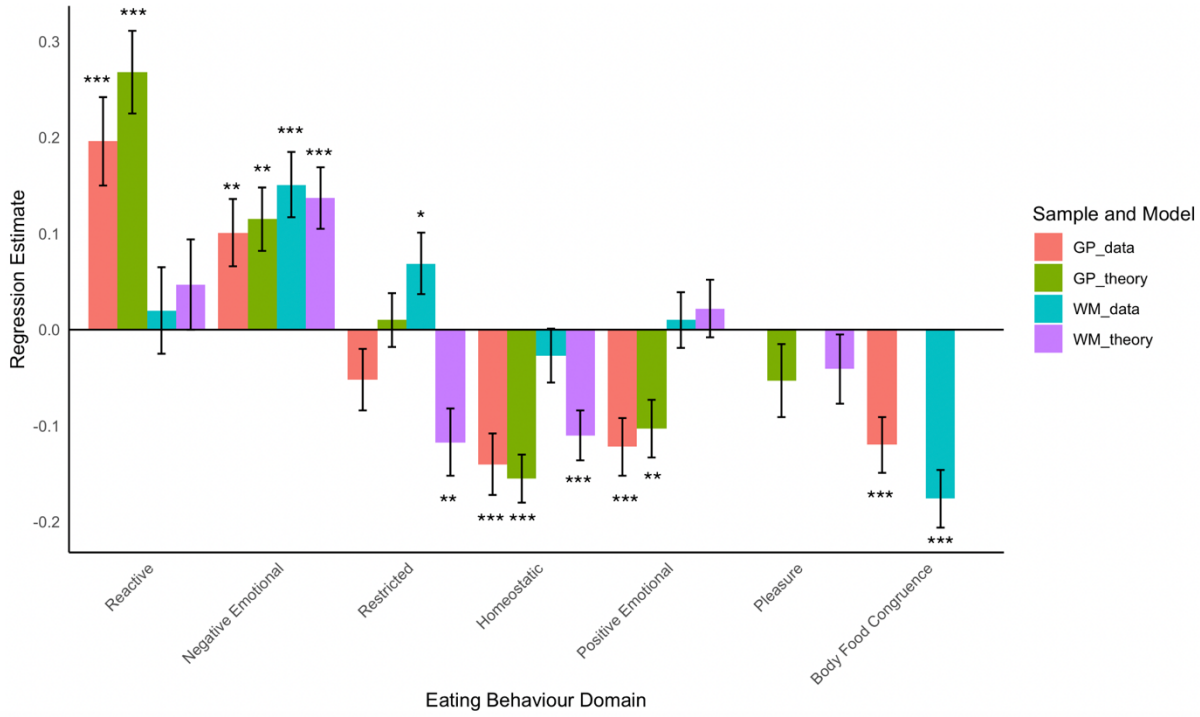
Note. Green lines represent positive loadings, red lines represent negative loadings. Outer statistics denote factor loadings. Inner statistics denote covariance between factors.

6.4.5. Predicting BMI category

The data-driven model and the theory-driven model were both asked to predict BMI category in the general population sample and the weight management sample (see Figure 6.6). In the general population, higher reactive eating and negative emotional eating were significantly associated with a higher BMI category in the data-driven and theory-driven models. Higher homeostatic eating and positive emotional eating were significantly associated with a lower BMI category in both models. In the theory-driven model, there was no significant association between eating for pleasure and BMI category. In the data-driven model, higher body-food choice congruence scores were significantly associated with a lower BMI category. In both models, there was no significant association between restricted eating and BMI category.

In the weight management sample, higher negative emotional eating scores were significantly associated with a higher BMI category in both models. There was no significant association between reactive eating or positive emotional eating and BMI category in both models. In the data-driven model, higher restricted eating scores were significantly associated with a higher BMI category, whereas in the theory-driven model, higher restricted eating scores were associated with a lower BMI category. In the data-driven model, there was no significant association between homeostatic eating and BMI category, whereas for the theory-driven model, higher levels of homeostatic eating were associated with a lower BMI category. In the data-driven model, higher body-food choice congruence scores were associated with a lower BMI category. Lastly, in the theory-driven model, eating for pleasure was not significantly associated with BMI category.

Figure 6.6. Regression estimates for the association between eating behaviour domains and BMI category in the GP and WM sample and theory and data-driven models



Note. GP = General Population, WM = Weight Management Sample, Error bars denote standard error. *p < .05, **p <.01, ***p <.001

6.5. Discussion

The current study examined whether EBT items can be explained by four previously identified factors of motivations to eat and whether the number of factors increase when items measuring eating for pleasure and positive emotional eating were included. A data-driven and theory-driven approach were taken, resulting in two models that were tested and compared. The extent to which the final models could predict BMI category were also tested. In both samples, the results found some support for a 6-factor model (see Figures 6.4, 6.5 and D5, D6) where both models included reactive eating, negative emotional eating, positive emotional eating, restricted eating, homeostatic eating. However, there were some differences between the theory and data-driven model regarding the 6th factor. The data-driven model indicated a separate body-food choice congruence factor, whilst the theory-driven model indicated a separate eating for pleasure factor. Additionally, none of the models met all the good fit criteria, which does cause uncertainty about the fit of the models.

The results supported taking an item-based approach to the analysis, as some items from the subscales (e.g. disinhibition) loaded onto different factors. When age, gender and BMI were accounted for, there were significant differences in mean scores of EBTs between the general population and weight management sample, which indicates that these samples show distinct differences in their eating behaviours. The data-driven and theory-driven models were able to account for significant variance in BMI category in both the general population and weight management sample. Overall, this study supports the use of an EBT framework that separates EBT items into 6 domains of eating behaviour. However, further research is needed to confirm the model fit in various populations, validate the generalisability of the model, and identify potential modifications needed for specific contexts.

Item loadings from the data-driven model compared to the theory-driven model, in most cases, supported the theory-driven model. The EFA supported the existence of a reactive factor (including items from susceptibility to hunger and disinhibition), a negative emotional overeating factor (including items from emotional overeating,

eating for physical rather than emotional reasons, and disinhibition), a restricted eating factor (including items from restraint and unlimited permission to eat), a positive emotional eating factor (including items from positive emotional eating), and a homeostatic eating factor (including items from reliance on hunger and satiety cues). These findings support our previous study (Dakin et al., 2024), and also extend on previous research.

The results support the existence of a reactive factor of EBTs, which overlaps with the Uncontrolled Eating construct (Vainik et al., 2015) and food reward responsivity component (Price et al., 2015). All three factors capture increased appetite and reduced control over eating. Similarly to previous research (Price et al., 2015), we also identified a restricted eating factor. However, our findings differ from that of Vainik et al. (2015) and Price et al. (2015) because in both studies, we found support for the existence of a distinct emotional eating domain. Indeed, Vainik et al. (2015) do highlight that uncontrolled eating "has a strong emotional eating component to it." The current study found that in both models tested, the results support the distinction between positive and negative emotional eating. Yet, the vast majority of literature on emotional eating focuses on negative emotional eating, when positive emotional eating could be an important and distinct construct (Evers et al., 2009). For example, research has found that positive and negative emotional eating have different relationships with overeating and binge eating (Sultson et al., 2017). Future research should therefore measure both positive and negative emotions because both are distinct motivations for eating.

Where the two models differ is with respect to body-food choice congruence and eating for pleasure. The theory-driven model found that body-food choice congruence loads under homeostatic eating, whilst the data-driven model suggests this EBT forms its own factor. The data-driven model found slightly better fit indices, suggesting that body-food choice congruence is distinct from reliance on hunger and satiety cues. Therefore, the body-food choice congruence subscale may not be measuring a form of homeostatic eating. As such, future research should consider if this subscale does measure a different domain of eating behaviour, if this fits in a framework of eating behaviour and if an additional factor can be confirmed. The theory-driven model also suggests that eating for pleasure forms its own factor,

whereas the data-driven model found that all items from the eating for pleasure subscale load with the reactive eating items. These items refer to the context surrounding eating e.g. “I eat what I eat to indulge myself.” Although considerable theoretical research suggests that food reward and reactive eating are distinct, albeit closely linked constructs (Berridge et al., 2010; Berthoud, 2011; Finlayson & Dalton, 2012; Kringelbach et al., 2012), the reference to context and more hedonic eating behaviours could be the reason why, when taking a data-driven approach, these items load under reactive eating.

It is to be expected that eating for pleasure will be strongly associated with reactive eating. There is considerable overlap between reward circuitry for different rewards e.g. food and drugs (Zhou et al., 2023). Pleasure and reward also contain affective and reactive components. Indeed, if one considers that pleasure is the primary sensory/experiential cue, then reward is the learned or conditioned response to that cue, which is encoded by populations of neurones in brain centres associated with the acquisition of learned preferences (Sternson & Eiselt, 2017). A theoretical reason for including a separate construct for eating for pleasure is supported by the scientific literature which highlights the primary importance of pleasure and reward in directing goal oriented motivated behaviours such as eating.

According to Kringelbach et al. (2012), “Food intake is driven by motivation and emotion which are in turn supported by reward and hedonic processing.” Berthoud (2011) argues that “Eating can be triggered by metabolic need, hedonic drive, or an interaction between the two, and there are several neural circuits that represent this interface. Importantly, metabolic signals of energy status can modulate processing of cognitive and reward functions in cortico-limbic systems (bottom-up processing), which influence regulatory processes to restore energy status to the optimal level. Yet the cognitive and emotional brain can also override homeostatic regulation (top-down processing), to yield an energy imbalanced state. Clearly, the emerging obesity epidemic indicates that this top-down processing may be winning the battle of the bulge. “Much more evidence on the mechanisms underlying this process is needed to effectively target this form of overindulgence and halt further increases in obesity” (Berthoud, 2011).

Thus, while there is considerable interface and overlap between reactivity and eating for pleasure we believe and argue that attempting to dissect-out the items that operationalise eating reactivity and eating pleasure is valid from a theoretical and psychometric perspective and important in understanding the factors that motivate human eating behaviour in order to enhance behaviour change interventions for obesity management. In so doing we recognise a key limitation of an item-based approach, that it is only as discriminatory as the scale-items inputted into the analysis. Therefore, we remain open to future refinements and developments of this model.

Regarding model fit, the CFI values of both models indicated a strong model fit in the general population and weight management sample. However, the RMSEA and SRMR values did not support a strong model fit and suggested that the data-driven model provided a better fit than the theory driven model. Furthermore, for both models, there were slightly better fit indices for the weight management sample. Potentially, taking this item-based approach resulted in there being items analysed that do not fit the 6 factors tested and should form additional factors. Whilst the EFA does support this, many of the additional factors included cross-loading items and factors that only included 1 item. The results suggest that the majority of items can be explained by 6-factors. However, there is some additional variance that cannot be explained by these items, which may have reduced the overall fit of the models. Whether these items measure important eating behaviours that are not identified or behaviours that are not relevant to motivations for eating has not been identified in the current analysis. This is an important avenue for future research.

Examining the general population sample (Model 1 and 2) and the weight management sample (Model 3 and 4), the differences in factor loadings suggests that the items driving any differences between these samples come from the restricted eating factor. In the weight management sample, there were weaker loadings of some of the items set to load on the restricted eating factor. It is important to note here that this specific weight management sample focuses on increasing consumption of low energy dense foods, which is a strategy of decreasing energy intake, by increasing the consumption of less energy dense foods without restricting food intake. The lack of loading of these items could suggest that weight

management participants do not perceive they are dieting which could be why some items did not load well onto the restricted eating factor. Indeed, the specific dieting approach taken by the weight management sample does not involve calorie counting and instead encourages the consumption of low energy dense foods, termed ‘Free Foods’ (Buckland et al., 2018; Pallister et al., 2009). This approach aims to be distinct from other dietary approaches, which do focus on restriction of food and energy intake (Slimming World, 2023). Consequently, these participants could be aligning themselves with the weight management program ethos that they are not dieting, which is why in this sample, removing restricted eating from the model improved the fit, whilst in the general population, including restricted eating led to a better model fit. To investigate this phenomenon further, future research should utilise measurement invariance analysis which enables a better examination of differences in the fit of the models, between the general population sample and the weight management sample.

Aside from some items in the restricted eating factor in the weight management sample, items had strong loadings onto their proposed factors in both models. Of interest, unlimited permission to eat was proposed to negatively load onto the restricted factor in the theoretical model. This was because the items were thought to refer more the restricted eating than to homeostatic eating. The CFA showed that these items did load strongly and negatively onto the restricted factor (see Figures 6.4 and 6.5). From a questionnaire development perspective, it is a strength that these items loaded negatively because this helps to capture acquiescence in responding (Hinz et al., 2007). Furthermore, in the data-driven and theory-driven model and across the general population and weight management sample, all items, except for TEMS_1, (and except for some restrained eating items, as explained above), loaded >0.35, indicating that items did load strongly onto their factors and providing support for the 6 factors. In the theory-driven model, the only item that did not load strongly was TEMS_1 onto the pleasure factor, with a loading of 0.268 in the general population and a loading of 0.014 in the weight management sample. Item 1 from TEMS states “I eat what I eat because I enjoy it.” This item seems to refer more to food liking, whereas the other items, “indulge” and “reward myself,” seem to refer more to food reward, as suggested by Sproesser et al. (2018). Additionally, the difference in loadings between “enjoy” and “reward/indulge” could

represent the distinction between liking and wanting of foods (Finlayson & Dalton, 2012; Finlayson et al., 2007).

The lack of loading of TEMS_1, indicates that the pleasure factor measures more reward motivations for eating and captures less of the pleasurable emotions that motivate eating. In support of this, previous research has also found that TEMS_1 has a much weaker loading than other items in this subscale. Sproesser et al. (2018) used the three-item pleasure subscale from the brief TEMS and found that TEMS_1 had a weaker loading than TEMS_2 (“to indulge myself,”) and TEMS_4 (“to reward myself”) in the total sample and in Germany. Interestingly, TEMS_1 had the strongest loading in the Indian and American sample. This could suggest cultural differences in the perception of eating for pleasure. The current UK samples share similar views as Germany, perceiving this factor to be more associated with reward than food enjoyment driving eating. However, in other non-European countries such as India and America, these participants seem to perceive this factor to be more associated with enjoyment as a motivation for eating. In the data-driven models, TEMS_1 also did not load strongly, but in this model, it did not load onto the reactive factor with a loading of 0.094 in the general population and -0.071 in the weight management sample. This finding supports the idea that while food liking is distinct from reactive eating, there is overlap between food reward, pleasure, and reactive eating. Thus, as explained above, theoretically we expect eating for pleasure and reactive eating to be separate constructs, but when taking a data-driven approach, overlap between these factors can also be expected.

The current study also found that both 6-factor models significantly predicted BMI category (see Figure 6.6). There were many similarities between the general population and weight management sample and the data-driven and theory-driven models in the associations with external outcomes. In both models and samples, negative emotional overeating was positively associated with BMI category. This indicates an influential role of negative emotional eating in overeating and obesity, which supports the literature on emotional eating and obesity. A review on negative emotional eating and obesity in adults found that self-reported emotional eating is associated with higher food intake, BMI, waist circumference and body fat

percentage. Higher emotional eating at baseline is also associated with higher weight gain over time (Konttinen, 2020).

Additionally, in the general population and in the theoretical model in the weight management sample, homeostatic eating was negatively associated with BMI category. Interestingly, there was no association with restricted eating in the general population, whereas, in the weight management population, restricted eating was positively associated with BMI category in the data-driven model, but negatively associated with BMI category in the theoretical model. This could be a result of more restraint items in the theoretical model, and restraint as measured by the TFEQ identifies successful dieters who can control their intake (Polivy et al., 2020, 2023).

Interestingly, reactive eating was a large positive predictor of BMI category in the general population, but there was no significant association in the weight management sample. Examining the items that make up the reactive factor, there was a larger correlation between BMI and disinhibition and susceptibility to hunger in the general population compared to the weight management sample. The general population also showed higher variance and standard deviation for disinhibition than the weight management sample. This variability might contribute to a larger positive predictor of BMI category in the general population because this population has a broader spectrum of reactive eating behaviours, which makes it easier to detect an influence of reactive eating on BMI category. Potentially, the decreased variability in disinhibition shown by the weight management sample, may be a result of the individuals in this sample being actively engaged in structured weight management practices, which may mitigate the impact of disinhibition on BMI. Lastly, there was no significant association between eating for pleasure and BMI category in both samples, but there was a significant negative association between body-food choice congruence in both samples.

Overall, the findings of current study indicate a distinct set of predictive factors that have the potential to influence specific populations and weight management contexts. This highlights a need for tailored intervention strategies based on the specific factors that play a significant role in each group. Indeed, Teixeira et al. (2005) suggest that programmes should identify people who may be less likely to

succeed and these individuals should receive tailored treatments to meet their individual needs. Through examining differences in EBTs and how they predict external outcomes in different groups, we may be able to identify certain groups or individuals within groups who could need these tailored treatments.

Strengths of the current study include a more balanced array of EBTs than previously tested. There was an overall good balance of EBTs that captured each of the eating behaviour domains. We also included constructs that have not been previously tested for example, eating for pleasure. Additionally, the number of participants across each sample was more even and the general population sample had a more even gender ratio than our previous study. Furthermore, this sample is representative of the UK population based on age, gender and ethnicity which increases the generalisability of the results. Taking an item-based approach also allowed the subscales to be deconstructed to identify and remove certain items that were measuring multiple factors or items that were not measuring an EBT and therefore reducing the fit of the model. However, there were limitations to the study. The EFA suggested that more factors should be included in the model for a better model fit. We did not include extra factors because the aim was to examine distinct factors in a framework of EBTs. This meant that the overall fit of the models did not meet all the fit criteria, so it is unclear whether more factors should be included in the framework. The fit of the model was also slightly weaker in the general population, so further research is needed to confirm if the model can be generalised to a non-weight management sample.

A potential avenue is the development of a brief and balanced survey that measures the 6 domains of eating behaviour. At present, no survey measures the identified domains of eating behaviour. To test the framework, participants need to complete multiple surveys that may also include items that are not highly relevant to the eating behaviour framework. A short survey that includes the most salient 3-4 of the most relevant items from each domain of eating would be valuable and novel to the field. Including less items, may also lead to stronger support from analysis techniques like CFA. If the model structure of the survey were supported, it could be used as a measurement tool to aid appetite and eating behaviour research and weight management interventions. The survey could then be used prospectively to examine

how eating behaviours change in participants who gain, lose, and maintain a clinically significant amount of weight over time.

In conclusion, this study investigated the explanatory power of previously identified factors of eating behaviour and examined the impact of incorporating items measuring eating for pleasure and positive emotional eating in predicting BMI category. There was some support for a 6-factor model. However, at present, the data-driven model does not distinguish between eating for pleasure and reactive eating. Both models did support the existence of reactive eating, negative emotional eating, positive emotional eating, restricted eating, and homeostatic eating in understanding motivations for eating. Taking an item-level approach meant that subscales could be disaggregated and non-EBT items could be excluded. The 6-factor model demonstrated its predictive validity, significantly predicting BMI category. The findings highlight the utility of an EBT framework, which could be used to identify individuals at risk of weight gain and or those who may struggle with weight loss. In future, by mapping these factors on to changes in eating behaviour states, it may be possible to enhance tailored interventions based on fluctuations in these population-specific factors.

6.6. References

Bentler, P. M. (1990). Comparative fit indexes in structural models. *Psychological bulletin*, *107*(2), 238.

Berridge, K. C., Ho, C.-Y., Richard, J. M., & DiFeliceantonio, A. G. (2010). The tempted brain eats: pleasure and desire circuits in obesity and eating disorders. *Brain research*, *1350*, 43-64.

Berthoud, H.-R. (2011). Metabolic and hedonic drives in the neural control of appetite: who is the boss? *Current opinion in neurobiology*, *21*(6), 888-896.

Buckland, N. J., Camidge, D., Croden, F., Lavin, J. H., Stubbs, R. J., Hetherington, M. M., Blundell, J. E., & Finlayson, G. (2018). A low energy–dense diet in the context of a weight-management program affects appetite control in overweight and obese women. *The Journal of nutrition*, *148*(5), 798-806.

Costello, A., & Osborne, J. (2005). Best practices in in exploratory factor analysis: Four recommendations for getting the most from your analysis. *Practical assessment, research, and evaluation*, *10*, 1-10.

Dakin, C. A., Finlayson, G. S., Horgan, G., Palmeira, A. L., Heitmann, B. L., Larsen, S. C., Sniehotta, F. F., & Stubbs, R. J. (2023). Exploratory analysis of reflective, reactive, and homeostatic eating behaviour traits on weight change during the 18-month NoHoW weight maintenance trial. *Appetite*, 106980. https://doi.org/https://doi.org/10.1016/j.appet.2023.106980

Dakin, C. A., Finlayson, G. S., & Stubbs, R. J. (2024). Can eating behaviour traits be explained by underlying, latent factors? An exploratory and confirmatory factor analysis. *Appetite*, 107202.

De Graaf, C. (2005). Sensory responses, food intake and obesity. *Food, diet and obesity*, 137-159.

Epskamp, S., & Stuber, S. (2019). semPlot: Path diagrams and visual analysis of various SEM packages' output. *R package version*, *1*(2).

Evers, C., de Ridder, D. T. D., & Adriaanse, M. A. (2009). Assessing yourself as an emotional eater: mission impossible? *Health psychology : official journal of the Division of Health Psychology, American Psychological Association*, *28*(6). https://doi.org/https://dx.doi.org/10.1037/a0016700

Fabrigar, L. R., Wegener, D. T., MacCallum, R. C., & Strahan, E. J. (1999). Evaluating the use of exploratory factor analysis in psychological research. *Psychological methods*, *4*(3), 272.

Finlayson, G., & Dalton, M. (2012). Hedonics of food consumption: are food 'liking'and 'wanting'viable targets for appetite control in the obese? *Current Obesity Reports*, *1*, 42-49.

Finlayson, G., King, N., & Blundell, J. E. (2007). Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews*, *31*(7), 987-1002.

French, 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). https://doi.org/https://dx.doi.org/10.1016/j.appet.2012.07.001

GOV.UK. (2023a). *Education and training statistics for the UK*. Retrieved 25/04/2024 from https://explore-education-statistics.service.gov.uk/find-statistics/education-and-training-statistics-for-the-uk

GOV.UK. (2023b). *Obesity Profile: short statistical commentary May 2023*. Retrieved 05/12/2023 from https://www.gov.uk/government/statistics/obesity-profile-update-may-2023/obesity-profile-short-statistical-commentary-may-2023

Hinz, A., Michalski, D., Schwarz, R., & Herzberg, P. Y. (2007). The acquiescence effect in responding to a questionnaire. *GMS Psycho-Social Medicine*, *4*.

Hoyle, R. H. (1995). *Structural equation modeling: Concepts, issues, and applications*. Sage.

Hunot, C., Fildes, A., Croker, H., Llewellyn, C. H., Wardle, J., & Beeken, R. J. (2016). Appetitive traits and relationships with BMI in adults: Development of the Adult Eating Behaviour Questionnaire. *Appetite*, *105*, 356-363.

IBM Corp. (2022). *IBM SPSS Statistics for Windows* In (Version Version 29.0) IBM Corp.

Jackson, D. L., Gillaspy Jr, J. A., & Purc-Stephenson, R. (2009). Reporting practices in confirmatory factor analysis: an overview and some recommendations. *Psychological methods*, *14*(1), 6.

Kenny, D. (2014). Measuring model fit (http://davidakenny. net/cm/fit. htm). *Accessed January*.

Kline, R. B. (2015). *Principles and practice of structural equation modeling*. Guilford publications.

Konttinen, H. (2020). Emotional eating and obesity in adults: The role of depression, sleep and genes. *Proceedings of the Nutrition Society*, *79*(3). https://doi.org/http://dx.doi.org/10.1017/S0029665120000166

Kringelbach, M. L., Stein, A., & van Hartevelt, T. J. (2012). The functional human neuroanatomy of food pleasure cycles. *Physiology & Behavior*, *106*(3), 307-316.

Llewellyn, C., & Wardle, J. (2015). Behavioral susceptibility to obesity: gene–environment interplay in the development of weight. *Physiology & Behavior*, *152*, 494-501.

Nasser, J. (2001). Taste, food intake and obesity. *obesity reviews*, *2*(4), 213-218.

NHS. (2022). *Health Survey for England, 2021: Data tables*. Retrieved 25/04/2024 from https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2021/health-survey-for-england-2021-data-tables

Pallister, C., Avery, A., Stubbs, J., & Lavin, J. (2009). Influence of Slimming World's lifestyle programme on diet, activity behaviour and health of participants and their families. *Journal of human nutrition and dietetics*, *22*(4), 351-358.

Polivy, J., Herman, C. P., & Mills, J. S. (2020). What is restrained eating and how do we identify it? *Appetite*, *155*, 104820.

Polivy, J., Herman, C. P., & Mills, J. S. (2023). Assessment of Restrained Eating and Dieting. *Assessment of Eating Behavior*, *6*, 15.

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.

Prolific. (2023). Retrieved 06/12/2023 from https://www.prolific.com

Racine, S. E., Hagan, K. E., & Schell, S. E. (2019). Is all nonhomeostatic eating the same? Examining the latent structure of nonhomeostatic eating processes in women and men. *Psychological Assessment*, *31*(10), 1220.

Renner, B., Sproesser, G., Strohbach, S., & Schupp, H. T. (2012). Why we eat what we eat. The Eating Motivation Survey (TEMS). *Appetite*, *59*(1), 117-128.

Revelle, W. (2022). *psych: Procedures for Psychological, Psychometric, and Personality Research.* In (Version R package version 2.2.5) https://CRAN.R-project.org/package=psych.

Rosseel, Y. (2012). *lavaan: An R Package for Structural Equation Modeling.* In

Slimming World. (2023). Retrieved 13/12/2023 from https://www.slimmingworld.co.uk/

Sproesser, G., Ruby, M. B., Arbit, N., Rozin, P., Schupp, H. T., & Renner, B. (2018). The eating motivation survey: results from the USA, India and Germany. *Public health nutrition*, *21*(3), 515-525.

Sternson, S. M., & Eiselt, A.-K. (2017). Three pillars for the neural control of appetite. *Annual review of physiology*, *79*, 401-423.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Sultson, H., Kukk, K., & Akkermann, K. (2017). Positive and negative emotional eating have different associations with overeating and binge eating: Construction and validation of the Positive-Negative Emotional Eating Scale. *Appetite*, *116*, 423-430.

Tabachnick, B. G., Fidell, L. S., & Ullman, J. B. (2013). *Using multivariate statistics* (Vol. 6). pearson Boston, MA.

Teixeira, P., Going, S. B., Sardinha, L., & Lohman, T. (2005). A review of psychosocial pre-treatment predictors of weight control. *obesity reviews*, *6*(1), 43-65.

Tylka, T. L., & Kroon Van Diest, A. M. (2013). The Intuitive Eating Scale–2: Item refinement and psychometric evaluation with college women and men. *Journal of counseling psychology*, *60*(1), 137.

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229-239.

Wickham, H. (2016). *Ggplot2*. In *Elegant graphics for data analysis*

Wickham, H., François, R., Henry, L., & Müller, K. (2022). *dplyr: A Grammar of Data Manipulation*. In https://dplyr.tidyverse.org, https://github.com/tidyverse/dplyr.

Zhou, Y., Stubbs, R. J., & Finlayson, G. (2023). A neurocognitive perspective on the relationship between exercise and reward: Implications for weight management and drug addiction. *Appetite*, *182*, 106446.

7. General Discussion

7.1. Introduction

Food and eating are one of the main pleasures in life and are central to the way individuals spend their lives. Shifts in the food production and supply systems have led to an increased availability of energy dense, highly palatable foods which is driving the increased prevalence of obesity around the world (Poulain, 2017; Stubbs et al., 2012). Eating is a goal-oriented, motivated behaviour, central to survival and wellbeing, that fulfils several homeostatic, affective, social and situational needs, including pleasure. Accordingly, motivations for eating and overeating are thought to be influential in shaping one's eating behaviours. Understanding these motivations for eating could help to prevent individuals from developing unhealthy eating behaviours, and/or help individuals to develop more healthy eating behaviours.

Eating behaviour traits (EBT) are psychological characteristics related to eating, which are used to measure and quantify individual differences in eating behaviours and have the potential to exert effects on food intake and energy balance. They are widely used in appetite and obesity research, to quantify individual differences or as predictors of tendency to self-regulate energy intake or overconsume and gain/re-gain weight, and have been tracked throughout weight management interventions as psycho-markers of compensation (Bryant et al., 2012). Additionally, studies have also identified EBTs associated with healthful eating behaviour and successful weight loss, and have developed interventions that aim to increase these EBTs e.g. (Warren et al., 2017). However, there is a lack of understanding about what each EBT is measuring, due to overlaps between constructs, theories and proposed mechanisms of action. There is a need to better understand the measures of motivation to eat that currently exist, how they relate to each other and how they relate to external outcomes e.g. energy intake and energy balance. As such, this thesis sought to address the gaps in the evidence base by developing a comprehensive framework of EBTs, and examining its ability to predict external outcomes.

This thesis had three overall aims: 1) identify the core constructs underlying EBTs, and examine how can they be integrated into a comprehensive framework by drawing from key psychological, physiological, and neurobiological models; 2) use the framework to predict energy balance related outcomes, including short-term EI and longer-term energy balance as estimated BMI, and individual variability in weight change during weight management interventions; and 3) refine and validate the framework, in samples with differing dietary goals related to weight management.

In the first paper (**Chapter 2**) of this thesis, a conceptual review proposed a provisional conceptual framework, and examined the framework in the context of existing psychological, physiological and neurobiological theory. The second paper (**Chapter 3**) conducted a systematic review and meta-analysis, that identified existing EBTs and examined whether EBTs predict short-term EI and longer-term energy balance, as estimated by BMI. Paper three (**Chapter 4**) built on this work by utilising a longitudinal study design, to assess the predictive power of EBTs as possible mechanisms of behaviour change over time, in weight management interventions. Paper four (**Chapter 5**), empirically tested a framework of EBTs, using EFA and CFA, and assessed whether the underlying factors could predict external outcomes. Lastly paper 5 (**Chapter 6**), refined the framework presented in paper four and balanced the framework for improved stability across two different populations. The framework was also validated by assessing its associations with BMI category.

7.2. Summary of findings and contribution to the literature

The findings of this thesis in relation to the three overall aims and corresponding research questions are summarised below.

7.2.1. What are the core constructs underlying EBTs, and how can they be integrated into a comprehensive framework by drawing from key psychological, physiological, and neurobiological models?

Findings from paper 1 (**Chapter 2**) identified 10 eating behaviour theories, 3 frameworks, that integrate constructs from psychological theory and, overall, 25 constructs were identified from these psychological, physiological and neurobiological theories and frameworks (see Table 7.1). However, many of these theories overlap, and draw to greater or lesser extents on concepts from dual-process theory, suggesting that the main dimensions of eating are captured by reflective and impulsive processes. There also appears to be a lack of understanding about the overarching domains of eating behaviour that EBTs are measuring. Whilst most of the EBTs identified in **Chapter 2** are developed from theory, there are some EBTs used in research that are instead data driven.

Indeed, paper 2 (**Chapter 3**) identified 16 EBTs that have been associated with laboratory measured EI and BMI (see Table 7.1). Ten of these identified EBTs matched with constructs identified in **Chapter 2** (see bold constructs in Table 7.1). There are some constructs from **Chapter 2** which do not relate to EBTs specifically. For example, whilst capability, opportunity and motivation can be associated with eating behaviours, they do not directly measure an eating behaviour, so they would not be expected to be identified by the systematic review in **Chapter 3**. However, 2 of the EBTs identified in **Chapter 3** (control of eating and binge eating) were not identified in the conceptual review, and have not been explicitly developed from specified theories directly relating to eating behaviour. For example, binge eating can be measured using the binge eating scale (Gormally et al., 1982). Binge eating is defined as ingesting large amounts of food within a short time, accompanied by a fear of not being able to stop and depressive mood (Gormally et al., 1982). The binge eating scale was developed to measure binge eating because, no research existed which operationalised the diagnostic criteria to assess binge eating. There were also no criteria to assess severity of binge eating problems. While binge eating

is clearly a relevant EBT, the development of this construct was not driven by a theory. Rather, it was driven by the need to design a self-report measure, that assessed binge eating problems.

Table 7.1. A table of constructs identified in Chapter 2 and EBTs identified in Chapter 3

Constructs identified in Chapter 2	EBTs identified in Chapter 3
Emotional Eating	**Intuitive Eating**
Internal control	Mindful Eating
External control	**Satiety Responsiveness**
Dietary Restraint (Restraint Scale)	**Dietary Restraint**
Hedonic Hunger	Control of Eating
Impulsive	**Hedonic Hunger**
Reflective	Food Craving
Restraint (Dual Pathway Model)	**Disinhibition**
Negative affect	**Susceptibility to Hunger**
Eating Enjoyment Goal	**External Eating**
Weight Control Goal	**Emotional Eating**
Food Responsiveness	Binge Eating
Satiety Responsiveness	**Eating Disorders / Food Addiction**
Capability	Eating Self-Efficacy
Opportunity	**Reward-Related Eating**
Motivation	
Disinhibition	
Restraint (TFEQ)	
Susceptibility to hunger	
Restrained Eating (DEBQ)	
Emotional eating (DEBQ)	
External eating (DEBQ)	
Intuitive Eating	
Reward-Based Eating	

Disordered Eating / Food Addiction

Note. Constructs in bold indicate these constructs were identified in both Chapters.

There are some EBTs identified in the systematic review (**Chapter 3**) that have been developed from theory, and were not explored in the conceptual review (**Chapter 2**). For example, mindful eating, as measured by the mindful eating questionnaire (Framson et al., 2009), is an EBT used to describe a non-judgemental awareness of physical and emotional sensations, while eating and engaging in mindful eating is proposed to help individuals recognise and respond to satiety and/or inappropriate cues for eating. Mindful eating has been developed from the concept of mindfulness, which is a construct that is embedded in psychological theory (Brown et al., 2015). Indeed, mindfulness relates to several theories including processing mode theory (Watkins, 2015), self-determination theory (Ryan & Deci, 2017), and cognitive behavioural models, such as acceptance and commitment therapy (Hayes et al., 1999), and mindfulness-based cognitive therapy (Segal et al., 2018).

Furthermore, there are several theories that aim to explain the mechanisms behind the EBT of food craving. These include: physiological theories related to the nutritional and energetic homeostatic role of food cravings e.g. (Wardle, 1987; Wurtman & Wurtman, 1986); psychological affect-based theories, that stress the role of negative emotional traits and triggers for food craving e.g. (Hill et al., 1991; Schuman et al., 1987); and learning theories, that suggest food cravings are conditioned responses to food-related cues e.g. (Rozin et al., 1991). Lastly, the Eating Self-Efficacy Scale was developed based on Bandura's self-efficacy theory, which proposes that behaviour change require the belief that changes will result in a desired outcome, and the belief that one is capable of making a change (Glynn & Ruderman, 1986).

On the other hand, the link between theory and construct for EBTs, measured by the TFEQ and IES, can be questioned. Although restrained eating has clearly been developed from restraint theory, and newer scales have built on restraint theory, the emergence of disinhibition and susceptibility to hunger, as measured by the TFEQ

came from items derived from the revised restraint scale, the latent obesity questionnaire and clinical experience (Stunkard & Messick, 1985). Additionally intuitive eating was developed to assess the psychometric properties of variables, that can protect against developing disordered eating behaviours (Tylka, 2006). Whilst intuitive eating is related to internal physiological needs of hunger and satiety, the link between intuitive eating constructs and eating behaviour theory is quite tenuous. Overall, both **Chapters 2 and 3** highlight that not all EBTs have been developed from theory. Some EBTs have weak links to theory and many EBTs are overlapping, with proposed mechanisms of action that are not easy to distinguish. A lack of theory driven constructs does not mean these EBTs are not useful in research. However, it is important to clarify and align the relationships between existing theories and EBT constructs, and standardise the measures of motivations for eating (Michie et al., 2011).

The findings of **Chapter 2** address these needs and highlight that many EBTs are aligned with aspects of dual-process theory. Specifically, a dual-process perspective was taken to the development of the framework of EBTs, because of its importance in relation to eating behaviour and energy balance. Greaves et al. (2011) describe the tensions between new behaviours (e.g. cognitive strategies) and existing habits (energy balance behaviours), food preferences and learned patterns of behaviour which are largely reactive. They propose that this tension can be managed though self-regulation, motivation, managing external influences to change habits and developing new approaches to meet psychological needs. This management can be seen as a form of reflective process. If one subscribes to the argument of Kringelbach et al. (2012), that motivated behaviours of eating are driven by pleasure and affectivity, then asymmetrical regulation appears to feed into this reactivity. The asymmetry of energy balance regulation means that energy balance behaviours are far less responsive to weight gain than weight loss, and one would expect neurocognitive systems related to feeding behaviour to evolve as adaptations to resource-limiting rather than modern environments (Stubbs et al., 2019; Stubbs et al., 2018).

As such, whilst it is possible to change eating habits, it is likely that reactive processes are powerful forces, that undermine attempts at changing energy balance

behaviours, especially in relation to weight loss (Berthoud, 2011; Blundell & Finlayson, 2004). On the other hand, with repetition and practice, we tend to subsume reflective processes to automatic, reactive processes (e.g. habits). So, reactivity is important to the development of successful behaviour change. Once new, more healthful behaviours become established as habits, then the chance of sustaining those behaviours increases (Stubbs et al., 2021b). Additionally, and perhaps most importantly, dual process models seem to align best with what we understand about the neurobiology of eating, articulated by neurocognitive models (Berthoud, 2012; Berthoud, 2011; Finlayson et al., 2007; Mackey et al., 2019).

Furthermore, physiological and neurobiological theory indicate the importance of homeostatic processes influencing eating behaviour. From a physiological perspective of energy balance, there are homeostatic processes manifesting in physiological motivation to eat or not eat (Stubbs & Turicchi, 2021). These processes help to explain factors that alter motivations for eating, and therefore should be captured in a framework of eating behaviour. The findings from **Chapter 2** also indicate the importance of neurobiological mechanisms that influence eating behaviour. For example, food reward (Kringelbach et al., 2012), cognitive function (Higgs et al., 2017) and executive function (Vainik et al., 2013), all play an important role in influencing eating behaviours. Furthermore, a neurocognitive model of appetite control also supports the concept of reflective and impulsive eating, such that food reward systems are largely (but not entirely) subcortical and reactive, whilst decision-making involves conscious processes and complex motivations (Zhou et al., 2023).

Accordingly, a provisional framework was developed, integrating knowledge from psychological, physiological and neurobiological models. The core constructs of restrained eating, self-regulation of eating behaviour, food responsiveness, hedonic hunger, external eating, susceptibility to hunger, binge eating, disinhibition, emotional overeating, intuitive eating and satiety responsiveness, identified in **Chapters 2 and 3,** were utilised in the development of this framework. The provisional framework suggests that eating behaviour is driven by three underlying domains of eating, reflective eating, reactive eating, and homeostatic eating. Reflective eating involves cognitive reasoning and deliberate control over food intake

(Stubbs et al., 2023). Reactive eating involves automatic eating behaviour, which is driven by emotions, impulses, habits and desires. Both reflective and reactive eating are aligned with dual process theory, which indicates that these two processes can operate in accord, but most often they compete to control eating behaviour (Strack & Deutsch, 2004; Stubbs et al., 2021b). These two domains of eating also interact with physiological signalling, which involves the homeostatic eating domain. Homeostatic eating refers to conscious awareness of or behavioural responsivity to internal cues of hunger and satiety (Dakin et al., 2024; Stubbs et al., 2023).

One of the strengths of this framework is that it accounts for a wider range of EBTs than previous models e.g. (Price et al., 2015; Racine et al., 2019; Vainik et al., 2015). These notable frameworks provide insights into EBTs related to the reactive domain, and the current framework builds on these insights by including EBTs related to reflective and homeostatic domains. However, there are still some areas of eating behaviour that are not explained by the framework. For example, eating disorders are more of an outcome domain than an exploratory domain of motivations for eating (Gearhardt & Hebebrand, 2021). There is ongoing debate about the concept of food addiction, in terms of whether it is an addiction to food, an addiction to eating and whether highly processed foods are addictive (Gearhardt & Hebebrand, 2021). Food addiction has not led to any successful treatments for obesity, and food addiction, as measured by the YFAS, occurs at higher rates in patients with anorexia nervosa, which generates doubts about the validity of this concept, in explaining high levels of BMI (Hebebrand & Gearhardt, 2021). As such, eating disorders and food addiction are not explained in the framework of EBTs and are added to the “other” column in the conceptual framework.

Developing this framework helps to provide insights into the current state of knowledge regarding eating behaviour. At present, behaviour change techniques, many of which can be considered reflective processes, are often used as a method of achieving weight loss (Stubbs et al., 2011). However, evidence shows that whilst many individuals engage in reflective processes, they are often unsuccessful (Garcia Ulen et al., 2008). This may be a result of the reactive system exerting control particularly as weight loss is a long term process that can deplete psychological resources (Greaves et al., 2011). For example, stress, food craving and depression

are important barriers for weight loss (Sharifi et al., 2013). Weight loss attempts are also met with reactive physiological, psychological and behavioural responses that oppose weight loss. In the context of a negative energy balance, it is likely that reactive processes also override homeostatic processes to promote compensatory increases in EI, thereby inhibiting weight loss success. On the other side of the energy balance equation, there is little evidence that overeating, leading to increased body weight, exerts homeostatic feedback to regulate body weight (Stubbs et al., 2018). Taken together, an understanding of these three domains of eating, which integrates knowledge from psychological, physiological and neurobiological theory, can help us to better understand why individuals are motivated to eat, why individuals overconsume, leading to weight gain, and some of the multiple barriers individuals face when attempting to lose weight.

Importantly, the framework developed in **Chapters 2 and 3** is a starting point towards a comprehensive framework of EBTs, and not a final framework. Therefore, the next aim of the thesis was to predict external outcomes using EBTs within the framework, to examine the utility of the framework in eating behaviour research. This analysis was important to improve the explanatory power of EBTs, which should lead to deeper insights into the underlying motivations for eating, and more effective interventions to manage eating behaviour and obesity.

7.2.2. To what extent can a framework of EBTs be used to predict external, objectively measured energy balance related outcomes (short-term EI and long-term energy balance as estimated BMI), and individual variability in weight change during weight management interventions?

Paper 2 (**Chapter 3**) demonstrated that EBTs do significantly predict both EI and BMI. In general, effect sizes were moderate, with some analyses reaching larger effect sizes. Susceptibility to hunger and binge eating were the strongest predictors of EI, whilst disinhibition was the strongest predictor of BMI. These findings are in line with previous research e.g. (French et al., 2012; Konttinen, 2020; Vainik et al., 2019), suggesting that increases in these EBTs are indicators of risk for weight gain, and they may characterise participants at risk of weight re-gain. Building on this, paper 3 (**Chapter 4**) found that participants who re-gained weight, across the 18-

month intervention, had significantly higher levels of reactive EBTs, which included disinhibition, susceptibility to hunger and binge eating, than participants who lost weight. This is interesting given that the theory-driven behaviour change constructs included in the NoHoW intervention did not appear to show strong predictive effects on weight change in the trial.

A major consideration of **Chapter 3** is that while 16 EBTs were identified from the systematic review, there is a lack of research on EBTs in relation to objective outcomes. The majority of research that does exist focuses on restraint. Importantly, as highlighted by paper 1 (**Chapter 2**), there are multiple questionnaires that have been developed to measure a version of restrained eating. However, different questionnaires use different defining measures. This means that some questionnaires e.g. TFEQ, seem to measure more successful restriction over time, whilst others e.g. the restraint scale, identify those who struggle to control their food intake (Polivy et al., 2020, 2023). In support of this, **Chapter 3** found a significant effect of the questionnaire used to measure restraint on EI. Restrained eating as measured by the TFEQ was associated with decreased EI, whilst restrained eating as measured by the restraint scale, was associated with increased EI. As such, it has been proposed that restrained eating, as measured by the TFEQ and DEBQ measure a construct that could be better termed 'restrictive eating' (Polivy et al., 2020). Both **Chapters 2 and 3** highlight the need to consider the conceptual ambiguity of questionnaires used to measured restrained eating in eating behaviour research. It appears that different questionnaires are more appropriate depending on the outcomes being measured in a study (e.g. understanding successful caloric restriction vs cycles of restriction followed by overconsumption). It is therefore vital that researchers are aware of the semantic and predictive differences between questionnaires, because different measures could be influencing the outcomes of studies.

Chapter 3 identified that susceptibility to hunger, binge eating and disinhibition, were the strongest predictors of EI and BMI. Therefore, these EBTs were used to examine individual variability in weight change in **Chapter 4**. This examination was important because the cross-sectional associations identified by the meta-analysis do not provide evidence of how EBTs are associated with weight change, or the long-term

efficacy of interventions that use EBTs. Whereas the longitudinal design of **Chapter 4** can provide these insights, **Chapter 4** also examined reflective and homeostatic EBTs, as proposed by the conceptual framework of EBTs. The results found that change scores in disinhibition, susceptibility to hunger, binge eating, unlimited permission to eat, restraint and reliance on hunger and satiety cues were significantly associated with weight change, such that reactive EBTs were associated with weight gain, and reflective and homeostatic EBTs were associated with weight loss. Regression analysis also found that changes in reactive, reflective and homeostatic EBTs were significantly and independently associated with weight change.

In support of previous research e.g. (Bryant et al., 2012; Karlsson et al., 1994; Keränen et al., 2011; McGuire et al., 1999; Pekkarinen et al., 1996; Teixeira et al., 2005; Varkevisser et al., 2019), participants who lost weight across the intervention showed reductions in reactive EBTs and increases in reflective EBTs, whilst participants who re-gained weight showed increases in reactive EBTs and reductions in reflective EBTs. Interestingly, there were mixed results for the effect of homeostatic eating on weight change. At 18 months, participants who lost and maintained weight had significantly higher levels of intuitive eating (reliance on hunger and satiety cues) than those who re-gained weight. This supports findings that successful weight loss is associated with a better ability to rely on one's internal hunger and satiety cues (Tylka et al., 2020). However, there were no differences in the subscale eating for physical reasons, rather than emotional reasons, between participants who lost, maintained and re-gained weight. Additionally, participants who lost weight had lower levels of unlimited permission to eat between 12 and 18 months, than those who maintained weight. Taken together, the results suggest that promoting some aspects of intuitive or homeostatic eating may not be effective for weight loss because this is a period where hunger may be elevated (Polidori et al., 2016; Stubbs & Turicchi, 2021). However, homeostatic eating could be beneficial for some individuals throughout weight maintenance.

The findings of **Chapter 4** and the main NoHoW trial suggest that weight management interventions should aim to align the mechanisms of behaviour change with the compensatory energy balance behaviours that undermine them (Stubbs et

al., 2021a). In the context of eating behaviour, compensatory energy balance behaviours relate to reflective and reactive aspects, which can be assessed through EBTs. Therefore, improving understanding of individual differences in EBTs, may enhance weight management interventions. As such, the next aim of the thesis was to empirically examine the domain structure of the conceptual framework of EBTs to understand if real life responses to EBT questionnaires support the proposed domain structure. Ensuring the conceptual framework is empirically supported should lead to a more in depth understanding of behaviour change and the mechanisms underlying behaviour change, which can be used to inform weight management interventions.

7.2.3. Can the framework of EBTs be refined and validated in samples with differing dietary goals?

Paper 4 (**Chapter 5**) first examined 8 questionnaires that measured 18 commonly cited EBTs, using EFA to understand if EBTs can be organised under latent factors. The analyses supported a five-factor model, which explained 64% of the variance in the data. After discussion within the research team, the factors were labelled reactive eating, restricted eating, negative emotional overeating, homeostatic eating and factor 5 was not labelled. The EBTs grouped under reactive eating (factor 1) encompassed motivations to eat that are automatic and impulsive, including food cues, reward and hedonic-driven eating. The emergence of this factor supports the conceptual framework and aligns with the impulsive system from dual-process theory.

Factor 2 included EBTs that specifically refer to conscious restriction over food and eating. This domain of eating behaviour was initially hypothesised to measure reflective eating in **Chapters 2-4**. However, after examining the item loadings onto this factor it became clear that this domain of eating refers to a high level of control and restriction, which does not necessarily entirely align with the concept of reflective eating. These findings support previous research that the DEBQ and TFEQ in particular measure more restrictive eating, than restrained eating (Polivy et al., 2020). Overall, the restricted eating domain does support the reflective system from dual-process theory, as eating behaviour is driven by conscious and deliberate

control. However, the label of restricted eating was decided as a better fit for this domain than reflective eating.

Factor 4, labelled homeostatic eating, also supported the conceptual framework proposed in **Chapters 2-4.** The items that loaded under this factor are proposed to measure a proxy of homeostatic eating, whereby, these items reflect conscious awareness of sensations that can be considered related to homeostatic eating. The EFA supported the conceptual framework that distinguishes between restricted and homeostatic eating, such that the ability to listen and act on satiety cues was found to be distinct from cognitive control of eating. Taken together, factors 1,2 and 4 (reactive, restricted, and homeostatic eating) support the conceptual framework, and align with psychological, physiological and neurobiological theory, suggesting that eating behaviour can be explained by reactive, restricted and homeostatic eating.

An interesting finding from the EFA, was the emergence of factor 3, which was labelled negative emotional overeating. This domain encompassed EBTs that measure specifically negative emotional overeating, because positive emotional eating did not load onto this factor. Potentially, this could have been due to a lack of positive emotional eating items in the questionnaires analysed, leading to an unbalanced model, which was a limitation of **Chapter 5**. Disinhibition also loaded onto this factor, as well as the reactive eating factor. Studies have identified that disinhibition measures multiple distinct factors, one being emotional eating/emotional susceptibility (Bond et al., 2001; Ganley, 1988; Karlsson et al., 2000). As such, the EFA finding that disinhibition cross-loaded on the reactive and negative emotional overeating factors, is consistent with previous research and supports the existence of negative emotional overeating as a distinct domain of eating.

Lastly, whilst factor 5 could be measuring dimensions of undereating, this domain was not labelled due to inconsistencies in the direction of loading between EBTs that loaded onto this factor. It was unclear how the items relate to satiety responsiveness and emotional undereating, and therefore what this factor is measuring.

Chapter 5 then used CFA as a method of validating the proposed framework identified by the EFA. A five-factor model was initially tested. However, items did not

load onto the fifth factor, so this factor was removed from the model. The four-factor model was supported by the data, indicating that EBTs within each domain can be used interchangeably. The analysis of **Chapter 5** refined and extended previous work e.g. (Price et al., 2015; Racine et al., 2019; Vainik et al., 2015), and further validates the conceptual framework of EBTs. Indeed, **Chapter 5** adds to the current evidence base through the emergence of novel domains measuring homeostatic eating and negative emotional overeating. The findings also contribute towards a better understanding of the processes that contribute to the variance explained in the data and identified whether the domains are distinct, if they overlap and how the domains can be broken down into more specific EBTs (DeYoung, 2006; McCrae & Sutin, 2018). Taken together, these insights improve our understanding of the eating behaviour field. However, there were some important limitations and considerations of **Chapter 5**, that paper 5 (**Chapter 6**) aimed to address.

The model tested by **Chapter 5** was unbalanced due to a much larger number of reactive EBTs, compared to EBTs from other domains. Additionally, whilst two samples were used (a general population and a weight management sample), there was an uneven ratio of dieters compared to the general population, and there were very few males in both samples. It was therefore important to test the model using a more diverse sample with differing dietary goals. Conceptually, the analysis of **Chapter 5** was also limited by taking a subscale approach to the EFA and CFA. The results highlighted there were cross loadings between some of the factors, and as such some of the EBTs loaded onto multiple factors. However, taking a subscale approach meant the analysis could not break down the scales into items to examine, what items were loading onto which factors and why. Lastly, in **Chapter 5**, positive emotional eating was removed from the CFA and added to the “other” section of the framework, due to its weak loading onto the reactive domain. It is possible that positive emotional eating refers more to hedonic eating, if pleasure is related to positive emotions. However, there were no EBTs included in the **Chapter 5** that measure eating for pleasure, so this hypothesis could not be tested. Accordingly, the results from **Chapter 5** were not able to explain if or how positive emotional eating fits into a framework of EBTs. This means it is important for further research to examine this construct and include EBTs that may be related to positive emotional eating, such as eating for pleasure.

Building on these limitations, **Chapter 6** used an item-level approach to examine a more balanced model of EBTs, in more diverse samples. Importantly, a UK representative sample based on age, gender and ethnicity were included, alongside a weight management sample, to improve the generalisability of the results. Additionally, **Chapter 2** highlighted the importance of neurobiology in understanding eating behaviour, indicating that pleasure and reward are key mechanisms, that ensure individuals seek the rewards that allow for survival and protection (Kringelbach, 2005). Kringelbach et al. (2012) proposed a neurobiological model for the full cycle of eating behaviour which includes the role of reward, including wanting, liking and learning. Furthermore, Castro and Berridge (2014) described the neural basis of food pleasure and highlight the role of hedonic hotspots in the brain which generate food pleasure and appetite. Importantly, it is argued that human eating is not solely governed by homeostatic processes (as evidenced by the rising prevalence of obesity). Instead, pleasure and reward play a central role in the control of food intake (Kringelbach, 2004). Consequently, the neurobiology of eating behaviour suggests that eating for pleasure is an important construct, and motivation for eating and should be tested within a framework of EBTs. Accordingly, trait eating for pleasure was included in **Chapter 6**.

Chapter 6 also took a more strategic approach to the questionnaire selection (see section 6.3.2), which resulted in 10 EBT constructs that measured a balanced range of eating behaviour domains, and all had been formally validated. **Chapter 6** also took two approaches, a data-driven and a theory-driven approach which resulted in the development of two models (data-driven model and theoretical model). Both models found some support for a 6-factor model, suggesting a reactive eating, negative emotional overeating, positive emotional eating, restricted eating and a homeostatic eating factor. However, there were discrepancies regarding the 6th factor. The data-driven model suggested that the body-food choice congruence subscale formed a distinct 6th factor. It also suggested that most items from the eating for pleasure subscale, loaded with the reactive eating items. In contrast, the theoretical model suggested that eating for pleasure formed a distinct 6th factor. It also suggested that the items from the body-food choice congruence subscale loaded with homeostatic eating.

Regarding the discrepancy of the body-food choice congruence subscale, possibly this EBT is related to making decisions about food choice for health rather than homeostasis. Indeed, the definition of the BFCC subscale states that it "assesses how well an individual matches their food choices with their body's needs." It does not necessarily follow that homeostasis and health are one and the same thing. Particularly if one takes that view that energy balance regulation is relatively imprecise and asymmetric e.g. (Stubbs & Turicchi, 2021). Additionally, if one takes that view that a tendency to overconsume in the current nutritional environment is part of the design of our homeostatic signals that evolved in a different environment, which is potentially compromising our health in the current environment. Then, the idea that homeostasis and health are distinct, is also supported. Thus, the data-driven model, suggesting that body-food choice congruence forms its own factor, does not compromise the theoretical model. Instead, it adds an additional factor of choosing to eat for health-related reasons. Accordingly, this factor could be labelled "eating for health," and it is understandable from a semantic perspective, why this EBT may be distinct from the reliance on hunger and satiety cues EBT.

Furthermore, there was discrepancy between the models regarding eating for pleasure. As discussed in **Chapter 6**, because eating for pleasure is strongly associated with reactive eating, it is reasonable that most of the eating for pleasure items loaded strongly onto the reactive factor in the data-driven model. However, it is argued that from a theoretical perspective, it is also valid to differentiate between reactivity and eating for pleasure (Marty et al., 2018; Mela, 2006). As explained above, pleasure is a key driver of eating behaviour. Additionally, Finlayson et al. (2007) argue that processes of liking and wanting have independent roles in characterising susceptibility to weight gain, and are important components of food reward, for appetite control. Therefore, the literature indicates that pleasure is a distinct from reactivity and is a significant motivation for eating.

In support of this, the eating for pleasure subscale did load strongly onto a distinct factor in the theoretical model (aside from TEMS_1, discussed in section 6.4). Importantly, a limitation of taking an item-based approach is that it can only be as discriminatory as the scale-items inputted into the analysis. Thus, providing another reason to analyse and examine both a data-driven model and a theoretical model. It

is also important for future research to refine and develop the model, to better understand where eating for pleasure sits within a framework of EBTs. Overall, the results from **Chapter 6** build on **Chapter 5** to support the existence of reactive eating, negative emotional overeating, restricted eating and homeostatic eating. They also extend the results from **Chapter 5**, by suggesting that emergence of positive emotional eating and potential factor of eating for pleasure.

Relating to both aim 2 and 3 of this thesis, **Chapters 5 and 6** used the EBT framework to predict external outcomes, including BMI and weight change. The findings of **Chapter 5** suggest that reactive eating, negative emotional overeating, restricted eating and homeostatic eating predict significant variance in BMI, whilst only homeostatic eating and restricted eating predicted self-reported weight change. Homeostatic eating was the only EBT that was negatively associated with BMI, and it predicted the largest variance in BMI and weight change. Building on this, the 6-factor data-driven and theory-driven models were used to predict BMI category in **Chapter 6**. Both models found a strong positive association between negative emotional overeating and BMI category, which supports previous literature indicating that negative emotional eating is associated with a higher BMI, fat percentage and food intake (Konttinen, 2020).

There were also similarities between the models and samples, whereby, homeostatic eating was negatively associated with BMI category in all models, except the data-driven model in the weight management sample. In general, the associations were similar between the general population and weight management sample. However, there were more differences in outcomes between the data-driven model and theory-driven model. For example, in the theory-driven model there was a significant positive association between reactive eating and BMI category, but no association for the data-driven model. Additionally, there was a significant negative association between positive emotional eating and BMI category in the theory-driven but not the data-driven model. Lastly, in the weight management sample, there was a significant positive association between restricted eating and BMI category in the data-driven model, but a significant negative association in the theory-driven model. This could be a result of more items being included in the theory-driven model which refer to successful dieters who can control their intake (Polivy et al., 2020, 2023).

These results have identified a distinct set of factors that influence diverse populations and have the potential to influence weight management outcomes. The differences between the samples highlight a need for a more tailored approach to weight management interventions, which target the eating related factors that play a significant role in BMI, weight gain and weight loss depending on the population. For example, it appears that interventions that target reductions in reactive eating may be more useful for the general population than the weight management sample.

Lastly, the results from **Chapter 6** were able to provide important insights into the current field of eating behaviour through examining the relationship between the factors within the framework. Previous research in the field of personality psychology underscores the importance of examining the overlap between domains to identify what sets each domain apart (DeYoung, 2006; McCrae & Sutin, 2018). The framework tested in both **Chapters 5 and 6** can provide theses insights. For example, figures 6.4 and 6.5 depict the covariance between the 6 eating behaviour domains in the general population. The results indicate that most of the domains do covary with each other. In particular, reactive eating and negative emotional overeating have a large positive co-variance, indicating that items within these domains do share similarities. This does support previous research, which suggests that these factors can be subsumed under one factor (Vainik et al., 2015). However, when the models were asked to predict external outcomes, negative emotional overeating was able to independently predict variance in BMI and predicted more variance in BMI than the reactive factor in **Chapter 5**, which supports the usefulness of this factor and keeping these domains distinct.

Interestingly, in the theoretical model, there is no significant co-variance between negative emotional overeating and restricted eating, restricted eating and homeostatic eating, and homeostatic eating and positive emotional eating in the general population. These results support the distinction between these domains, and especially support the existence of positive emotional eating as a separate domain to eating for pleasure or negative emotional overeating. Through analysing the relationships between the domains of eating, it is clear there is some overlap between these motivations. However, it is also clear that these domains should be kept distinct and are all important measures of eating behaviour.

Overall, the results from both **Chapters 5 and 6** support the use of a framework of EBTs in the general population and a weight management sample. However, there are outstanding issues and uncertainties that need to be highlighted. For example, there are uncertainties about the model fit, especially when taking an item-based approach. The results show that taking a subscale-based approach leads to a better model fit than taking an item-based approach. However, as discussed previously this could be a limitation of the items included in the analysis. Potentially, including less items could improve the model fit. There also needs to be further research to identify whether: eating for pleasure should form its own factor; body-food choice congruence ('eating for health') should form its own factor; a 6-factor model (including either eating for pleasure or eating for health), or 7-factor model (including both) is the strongest model to use across diverse samples. When taking an item-based approach, the models did not meet all the good fit criteria. Additionally, there was a lower model fit in the general population in **Chapter 6**, which causes some concern over the generalisability of the model. As such, it is important to highlight that the models developed in the current thesis will need further development and testing.

7.3. Limitations and directions for future research

7.3.1. Taking a consultative approach to the framework development

At present, an inclusive and consultative approach to developing the framework throughout this thesis, including experts in the field beyond the research team, has not been taken. Whilst it is highlighted that the current framework is provisional and a starting point to a comprehensive framework of EBTs, the work in this thesis would be strengthened if an interdisciplinary team had been consulted throughout. There could be theories, models or other EBTs used in sub-areas within psychology or other non-psychology disciplines that may have been missed by the reviews in **Chapters 2 and 3**. This means that the framework may not be as complete in its explanation of motivations for eating as it could be. Thus, to address this limitation, future research should use an improved approach such as the Delphi method (Dalkey & Helmer, 1963; Okoli & Pawlowski, 2004). The Delphi method is a method for structuring a group communication process, which allows for a group of individuals to effectively deal with a complex problem, such as developing a framework of EBTs. This method aims to achieve the most reliable consensus of opinions among experts, through the use of intensive questionnaires and controlled opinion feedback (Dalkey & Helmer, 1963). It has been popular in aiding decision-making, based on the opinions of experts in a variety of fields, including health and social sciences (Landeta, 2006).

Although the field of behaviour change is much larger than the field of EBTs, in terms of theories, related constructs and behaviours studied, it would be valuable to consult experts in the field and experts from other disciplines, and involve these experts in an interdisciplinary, comprehensive and consensus-based approach. As such, we aim to use the Delphi method in future research to construct a brief survey using the EBT framework to measure the domains of eating behaviour. This survey will then be used to collaborate with experts and generate a first round of feedback to identify items and constructs that may be missing or underdeveloped. It is hoped that by consulting other experts, consensus can be achieved, and this approach should support wider update by others.

7.3.2. Methodological and conceptual comments

It is also important to consider the limitations of EBTs and how they are measured. In this thesis, EBTs were measured using self-report questionnaires. As such, there is always some uncertainty about how capable individuals are at assessing their own eating behaviours. For example, some research suggests that individuals are unable to perceive their own behaviour, they often underreport certain outcomes and that retrospective ratings can be sensitive to recall bias (Evers et al., 2009). Indeed, the conceptual issues with the self-report method in various disciplines in psychology is well documented and critiqued in relation to the measurement of EI and expenditure by Dhurandhar et al. (2015). The use of self-reports can lead to response biases, which involve a tendency to respond to the items in a questionnaire on some basis, other than the specific item content (Paulhus, 1991). Some people respond to questionnaires to make them appear more favourable, meaning their responses do not reflect how they actually behave (McDonald, 2008). Other limitations to the way in which individuals respond to self-report measures include acquiescent responding (individuals agree to items without considering the question) and extreme responding (individuals give extreme ratings on scales) (Paulhus & Vazire, 2007). These common response tendencies lead to biased reporting, which reduces the overall credibility and validity of self-report measures.

Furthermore, one can question whether individuals know enough about themselves to be able to accurately portray what the self-report construct is measuring. In relation to EBTs, are individuals able to accurately report whether they can listen and act on their internal hunger and satiety cues as a measure of homeostatic eating? Do they conceptualise motivations to eat as hunger, appetite, satiation and satiety or, do they think more generically in terms of motivation to eat or not eat. Current analyses outside of this thesis suggests the latter. Self-report measures are based on the assumption that an individual can access the construct that a researcher aims to measure, and that the individual is willing to report on that construct (Judd & McClelland, 1998). If individuals cannot access or report on such constructs, there are serious limitations for the methodologies used in this thesis, which attempt to measure EBT constructs. Although these are important concerns, there are steps that can be taken to reduce biases and improve questionnaire construction. For example, balancing the scoring key of a questionnaire can limit the effects of

acquiescent responding (Paulhus & Vazire, 2007). No method of measuring a construct such as EBTs is perfect. However, it is important to understand the limitations of self-report measures of EBTs, and how to improve the validity of EBT measurements.

Self-report measures of EBTs are central to the current research questions because they allow us to access information about a person that is rich with their eating motivations and other introspective information that other people may not be aware of (Paulhus & Vazire, 2007). As a result, the use of EBTs in this thesis are a valid way to provide insights into different eating behaviours and motivations for eating. Though it must be acknowledged there are limitations to this approach, future research could utilise multiple methods which may maximise the validity of EBTs. For example, self-report measures of EBTs could be combined with digital ecological momentary assessments (EMA) and tracking of energy balance behaviours using wearable devices (Stubbs et al., 2021b) to improve the construct validity of EBT measurements. The use of EMA also addresses a common methodological limitation of EBTs that they are often measured cross-sectionally at one time point (Mason et al., 2020). This singular response is then used to represent an individual's level of that construct (e.g. a highly restrained eater). However, the results of **Chapter 4** show that EBTs can change over time, albeit these changes occurred across an 18-month period (consistent with the likely time course of energy balance regulation) (Stubbs & Tolkamp, 2006; Stubbs & Turicchi, 2021), rather than days or weeks.

EMA is a methodological tool that is useful for capturing time-varying behaviours and the experiences of individuals in their typical routines and environments (Hoppmann & Ho, 2015). It involves a real-time, naturalistic assessment methods whereby individuals complete one or more daily reports of certain behaviours and experiences. Whilst it is not expected that EBTs change over the course of days or weeks, as they are traits rather than states, the use of EMA is advantageous because they occur in a participant's natural environment and are temporally close to an actual experience (e.g. an emotional eating episode). These advantages limit retrospective recall biases and can improve ecological validity (Shiffman et al., 2008). Thus, future research should consider combining EMA with self-report EBTs

assessments as an optimal method for studying eating behaviours and motivations for eating.

7.3.3. Interchangeability of EBTs

The results of **Chapters 5 and 6** suggest that EBTs within each domain of eating can be used interchangeably. This means that for example, in the reactive domain, a researcher could measure food responsiveness, hedonic hunger, external eating, susceptibility to hunger, binge eating and some items from the disinhibition subscale and these EBTs all overlap and measure reactive eating. However, there are some limitations to this suggestion. Firstly, if a researcher takes a subscale-based approach, then the disinhibition subscale can be seen as measuring both reactive eating and negative emotional overeating. This means that the domains themselves are not distinct and include overlapping EBTs. If the researcher takes an item-based approach, then different disinhibition items would load onto these domains, meaning the domains are distinct from each other. Taking an item-based approach, is unlikely to be used in studies. This is because, deconstructing the disinhibition subscale into items alters the characterisation of the measure as described by the TFEQ's developers (Slaney & Maraun, 2008). As such, most studies do not alter the subscales they use and instead take and use questionnaire measures in their original form. This means that the use of total subscales, such as disinhibition measures, multiple eating behaviour domains in one EBT.

A further limitation relates to a "jingle fallacy," a term first cited by Thorndike (1904), which describes the incorrect assumption that two tests (or EBTs) assess the same construct because they share similar names. The restricted eating domain of the framework includes dietary restraint as measured by the TFEQ and DEBQ and the unlimited permission to eat subscale. It has previously been discussed that while there are multiple measures of restraint, they are not measuring the same construct. As such, one can question how interchangeable the restraint measures are within the restricted eating domain. However, recent research on this issue has suggested that it is the restraint scale which is a distinct construct from TFEQ and DEBQ restraint. Both TFEQ and DEBQ restraint appear to identify successful dieters who can restrict their intake whilst, the restraint scale measures excessive consumption,

weight fluctuations, cognitions and emotions (Polivy et al., 2023). Consequently, it would not be valid to include the restrained eating, as measured by the restraint scale, under the same domain as TFEQ and DEBQ restraint. Whilst the framework developed throughout this thesis does not attempt to do this, it is possible that researchers may perceive these three EBTs as overlapping, and choose the restraint scale to measure this domain. This is another reason why this domain label was changed to restricted eating in **Chapter 5,** to highlight that this domain is different from restrained eating and reflective eating. However, it is still unknown how restrained eating, as measured by the restraint scale, can be included into a framework of EBTs, or whether there are other EBTs included in the framework developed in this thesis, which also form a jingle fallacy.

Additionally, the framework developed in this thesis is limited because it cannot explain the differences in behaviours captured by different measures underlying each domain e.g. the different measures of dietary restraint. However, the objective of the framework was to identify the core latent constructs underlying EBT measures and consider them in the context of existing theory. This thesis does not attempt to explain the specific nuances between EBTs but does aim to better understand the underlying constructs these EBTs are measuring. This is because there are a vast number of EBTs used in the literature, and very little research about how similar or distinct these measures are. Additionally, the problem of a jingle or jangle fallacy could be obscuring the use of these EBT measures (Vainik et al., 2015). As previously discussed, a similar approach has been taken in the much bigger field of behaviour change, with the recent development of behaviour change ontology (Marques et al., 2023) and a smaller approach within this specific field to the concept of ‘uncontrolled eating’ (Vainik et al., 2015). Taking this approach has developed a standard terminology and comprehensive classification system that can be reliably used to describe interventions and provide evidence for what works in interventions (Marques et al., 2023). Taken together, it is hoped that taking a similar approach will also benefit the EBT field.

7.3.4. Interpretation of EBTs that do not fit into the framework

As discussed in paper 4 (**Chapter 5**), there are some EBTs that the framework developed in this thesis cannot account for. In paper 1 (**Chapter 2**), it was explained that the framework does not attempt to include disordered eating or food addiction. However, there were EBTs analysed in **Chapter 5** that did not load strongly onto their proposed factors, and as a result were removed from the models. For example, satiety responsiveness, self-regulation of eating and emotional undereating, are relevant EBTs that currently are not included in the framework of EBTs, because they did not load strongly onto any of the domains of eating. The items that measure emotional undereating are the reverse of the items that measure emotional overeating. Whilst this EBT could load negatively onto the negative emotional overeating domain, the items could be adding redundancy to the model because they are not distinct from the emotional overeating items.

As previously explained, one could expect self-regulation of eating to overlap with dietary restraint and that satiety responsiveness should overlap with the intuitive eating subscales. Self-regulation of eating did have a small cross-loading with the restricted eating domain, indicating that some shared item content, such as the cognitive control of eating and goal-directed behaviour. On the other hand, a key difference is that self-regulation of eating lacks emphasis on the cognitive control of eating. Whilst it conceptually makes sense that self-regulation of eating does not underlie restricted eating, the framework developed in this thesis is still limited by not accounting for this EBT. Furthermore, as the homeostatic eating domain measures the ability to listen and act on internal hunger and satiety cues, one would expect satiety responsiveness to load under this domain. Consequently, further research is needed to examine whether: satiety responsiveness measures homeostatic eating using a different sample; the satiety responsiveness scale is not a valid measure of homeostatic eating; the current homeostatic eating domain is not a valid label for this domain. Therefore, whilst most EBTs can be explained by these 6 domains of eating, a limitation of this thesis is that there are still some EBTs that do not fit the framework tested in **Chapters 5** and **6**. This suggests that the developed framework is not final, and further research is needed to examine these “other” EBTs, to understand if they are distinct from the other EBT domains, or whether they can be included in this 6-7-factor framework of EBTs.

7.3.5. Developing a brief questionnaire that measures the domains of eating behaviour

The results of paper 4 (**Chapter 5**) mark the beginning of an emerging framework of EBTs and the results of paper 5 (**Chapter 6**) have started to refine and validate the framework across diverse samples with differing dietary goals. However, in its present form, the framework developed in this thesis cannot be used as a measurement tool to aid appetite and eating behaviour research or weight management interventions. Therefore, an essential future direction for the framework is the development of a brief and balanced questionnaire, that measures the identified domains of eating behaviour in this thesis. Due to **Chapter 6** indicating the eating for health (body-food choice congruence items) may be an important and distinct factor, a questionnaire measuring 7 domains of EBTs should be developed.

Importantly, it is not suggested that new items should be developed, but items should be utilised from EBTs within the framework, because these items are commonly cited and highly validated by previous research. A questionnaire measuring 7 domains of EBTs, is both valuable and novel because no questionnaire at present measures all the domains of eating behaviour identified by this thesis. This questionnaire can be used in clinical settings and by weight management companies in multiple ways. For example, to identify individuals at risk of weight gain or re-gain, individuals who require more support through a weight management attempt, individuals who could benefit from engaging in more adaptive eating behaviours, and to track the progress of individuals in weight management interventions.

Key to the usability of a questionnaire measuring 7 domains of EBTs, is that it is brief, meaning it should take less than 15 minutes to complete. Currently, the majority of questionnaires that measure EBTs are overly lengthy, often with 20+ items to measure only one EBT. As such, a 15-minute questionnaire that can measure 7 domains of eating behaviour, is vital to promote a wider update by others, including translating the theory of the eating behaviour framework into a tool that can be easily implemented into research, programmes and interventions. This work is already underway, with latent variable theory and item-response theory (IRT) being used to inform the development of the brief EBT domains questionnaire. Research

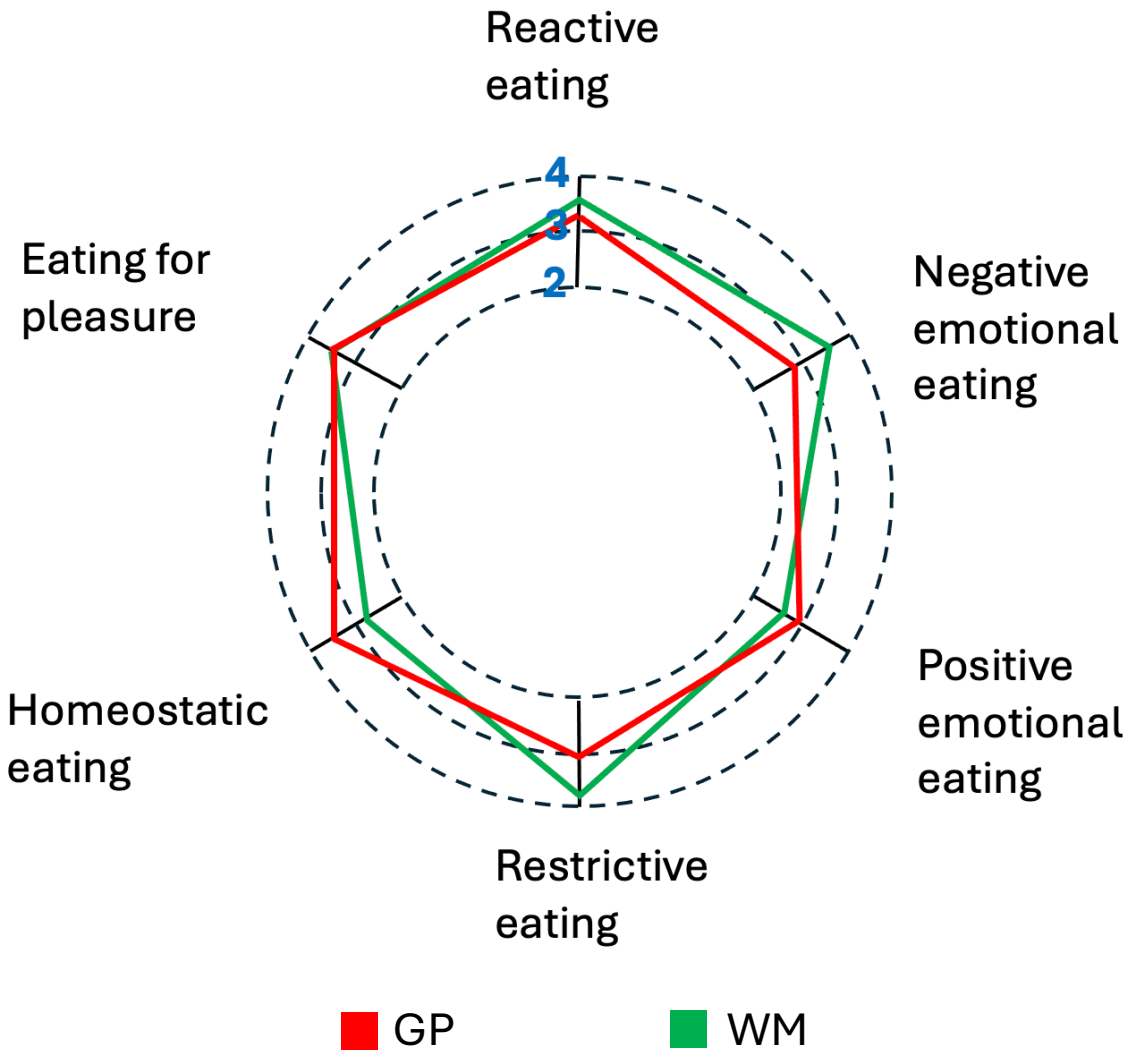
suggests that at least four items should be retained per latent construct (eating behaviour domain) (Maloney et al., 2011). Therefore, the newly developed questionnaire will include a total of 28 items with 4 items per eating behaviour domain.

In a pilot analysis, CFA and IRT were used in combination to select the 4 most appropriate items from all EBTs that load under each domain. The advantage of this approach is that no EBT is being discarded for the questionnaire. Instead, all EBTs that currently fit in the framework developed in this thesis are included in the analysis. At present, this analysis includes 6 eating behaviour domains, and the eating for health domain will be tested, in a future analysis. An in-depth discussion of the statistical analysis conducted is not presented here, because it goes beyond the scope of the current thesis. In brief, CFA and IRT were used to select items that should be retained based on factor loadings, reliability, a-parameters and item information functions. These results give an indication of the variance accounted for in an item by the latent trait, the strength of a relationship between an item and trait, how well an item differentiates between individuals, and the precision and information that is provided by each item (Maloney et al., 2011). Overall, the 4 items with the most information for each domain were selected (see E.1 for the 24-item questionnaire measuring reactive eating, negative emotional eating, positive emotional eating, homeostatic eating, restricted eating and eating for pleasure). This questionnaire (with another 4 items measuring eating for health), will be tested in a UK representative sample and a weight management sample to examine whether its brief form is still a strong fit for the data, and whether the 7 domains of eating emerge. The questionnaire will then be used to cross-sectionally examine the associations between 7 domains of eating behaviour and health and weight outcomes.

Using provisional data, which did not take an item-response theory approach, an 18-item questionnaire was initially developed, that measured 6 eating behaviour domains (with 3 items per domain). This questionnaire was then measured in a general population sample (n = 2505) and a weight management sample (n = 1870). Figure 7.1 depicts a radar chart of the mean scores on the eating behaviour domains between the two samples. The results showed that while eating for pleasure did not

differ between the samples, the weight management sample exhibited higher reactive eating, negative emotional eating and restricted eating. In contrast, the general population sample exhibited higher positive emotional eating and homeostatic eating.

Figure 7.1. A radar chart of the mean scores on the 6 eating behaviour domains between a census sample and weight management sample.



Note. GP = UK representative population, WM = weight management sample taken from members of Slimming World (UK).

Using this approach, we can begin to see how these eating behaviours can be mapped and tracked over time. In future, it is hoped that an EBT domain questionnaire can be utilised in weight management interventions to prospectively examine how the eating behaviour domains change in participants who gain, lose and maintain a clinically significant amount of weight over time. Lastly, the

framework developed in this thesis can be further developed by mapping the EBT domains onto eating behaviour states. EMA could then be used to track individual's EBTs and eating behaviour states longitudinally, across days, weeks and months. These results may lead to important insights which could enhance tailored interventions, based on fluctuations in these population-specific factors.

7.4. Conclusions

In summary, this thesis provides evidence for the importance of developing and utilising a comprehensive framework of EBTs that explains motivations for eating. The findings integrate psychological, physiological and neurobiological theory to identify the core constructs underlying EBTs, and align these constructs with existing theory. The results suggest that EBTs can be explained by 6-7 domains of eating: reactive eating, negative emotional overeating, positive emotional eating, restricted eating homeostatic eating, and eating for pleasure and/or eating for health. The framework developed in this thesis can be used to predict real-world outcomes including EI, BMI and weight change. Taken together, these findings provide support for the use of this comprehensive framework in research and clinical settings to provide deeper insights into individual differences in eating behaviour and motivations to eat. Importantly, future research is essential to resolve and refine outstanding issues (particularly in relation to eating for pleasure and eating for health) and develop a brief EBT domain questionnaire, which can be used as a measurement tool to improve the tracking of eating behaviours and outcomes of eating behaviour research and weight management interventions.

7.5. References:

Berthoud, H.-R. (2012). The neurobiology of food intake in an obesogenic environment. *Proceedings of the Nutrition Society*, *71*(4), 478-487.

Berthoud, H. R. (2011). Metabolic and hedonic drives in the neural control of appetite: Who is the boss? *Curr Opin Neurobiol*, *21*(6), 888-896.

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

Bond, M., McDowell, A., & Wilkinson, J. (2001). The measurement of dietary restraint, disinhibition and hunger: an examination of the factor structure of the Three Factor Eating Questionnaire (TFEQ). *International Journal of Obesity*, *25*(6), 900-906.

Brown, K. W., Creswell, J. D., & Ryan, R. M. (2015). Handbook of mindfulness: Theory, research, and practice.

Bryant, E. J., Caudwell, P., Hopkins, M. E., King, N., & Blundell, J. (2012). Psycho-markers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*, *58*(1), 234-241.

Castro, D., & Berridge, K. C. (2014). Advances in the neurobiological bases for food 'liking'versus 'wanting'. *Physiology & Behavior*, *136*, 22-30.

Dakin, C. A., Finlayson, G. S., & Stubbs, R. J. (2024). Can eating behaviour traits be explained by underlying, latent factors? An exploratory and confirmatory factor analysis. *Appetite*, 107202.

Dalkey, N., & Helmer, O. (1963). An experimental application of the Delphi method to the use of experts. *Management science*, *9*(3), 458-467.

DeYoung, C. G. (2006). Higher-order factors of the Big Five in a multi-informant sample. *Journal of personality and social psychology*, *91*(6), 1138.

Dhurandhar, N. V., Schoeller, D., Brown, A. W., Heymsfield, S. B., Thomas, D., Sørensen, T. I., Speakman, J. R., Jeansonne, M., & Allison, D. B. (2015). Energy balance measurement: when something is not better than nothing. *International Journal of Obesity*, *39*(7), 1109-1113.

Evers, C., de Ridder, D. T. D., & Adriaanse, M. A. (2009). Assessing yourself as an emotional eater: mission impossible? *Health psychology : official journal of the*

Division of Health Psychology, American Psychological Association, *28*(6). https://doi.org/https://dx.doi.org/10.1037/a0016700

Finlayson, G., King, N., & Blundell, J. E. (2007). Liking vs. wanting food: importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews*, *31*(7), 987-1002.

Framson, C., Kristal, A. R., Schenk, J. M., Littman, A. J., Zeliadt, S., & Benitez, D. (2009). Development and validation of the mindful eating questionnaire. *Journal of the American Dietetic Association*, *109*(8), 1439-1444.

French, 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). https://doi.org/https://dx.doi.org/10.1016/j.appet.2012.07.001

Ganley, R. M. (1988). Emotional eating and how it relates to dietary restraint, disinhibition, and perceived hunger. *International Journal of Eating Disorders*, *7*(5), 635-647.

Garcia Ulen, C., Huizinga, M. M., Beech, B., & Elasy, T. A. (2008). Weight regain prevention. *Clinical Diabetes*, *26*(3), 100-113.

Gearhardt, A. N., & Hebebrand, J. (2021). The concept of “food addiction” helps inform the understanding of overeating and obesity: Debate Consensus. *The American journal of clinical nutrition*, *113*(2), 274-276.

Glynn, S. M., & Ruderman, A. J. (1986). The development and validation of an eating self-efficacy scale. *Cognitive therapy and research*, *10*, 403-420.

Gormally, J., Black, S., Daston, S., & Rardin, D. (1982). The assessment of binge eating severity among obese persons. *Addictive behaviors*, *7*(1), 47-55.

Greaves, C. J., Sheppard, K. E., Abraham, C., Hardeman, W., Roden, M., Evans, P. H., & Schwarz, P. (2011). Systematic review of reviews of intervention components associated with increased effectiveness in dietary and physical activity interventions. *BMC Public Health*, *11*(1), 1-12.

Hayes, S. C., Strosahl, K. D., & Wilson, K. G. (1999). *Acceptance and commitment therapy* (Vol. 6). Guilford press New York.

Hebebrand, J., & Gearhardt, A. N. (2021). The concept of “food addiction” helps inform the understanding of overeating and obesity: NO. *The American journal of clinical nutrition*, *113*(2), 268-273.

Higgs, S., Spetter, M. S., Thomas, J. M., Rotshtein, P., Lee, M., Hallschmid, M., & Dourish, C. T. (2017). Interactions between metabolic, reward and cognitive processes in appetite control: Implications for novel weight management therapies. *Journal of Psychopharmacology*, *31*(11), 1460-1474.

Hill, A. J., Weaver, C. F., & Blundell, J. E. (1991). Food craving, dietary restraint and mood. *Appetite*, *17*(3), 187-197.

Hoppmann, C. A., & Ho, A. (2015). Ecological Momentary Assessment.

Judd, C. M., & McClelland, G. J. (1998). Measurement. *Handbook of social psychology*, *1*, 180-232.

Karlsson, J., Hallgren, P., Kral, J., Lindroos, A.-K., Sjöström, L., & Sullivan, M. (1994). Predictors and effects of long-term dieting on mental well-being and weight loss in obese women. *Appetite*, *23*(1), 15-26.

Karlsson, J., Persson, L.-O., Sjöström, L., & Sullivan, M. (2000). Psychometric properties and factor structure of the Three-Factor Eating Questionnaire (TFEQ) in obese men and women. Results from the Swedish Obese Subjects (SOS) study. *International Journal of Obesity*, *24*(12), 1715-1725.

Keränen, A.-M., Strengell, K., Savolainen, M. J., & Laitinen, J. H. (2011). Effect of weight loss intervention on the association between eating behaviour measured by TFEQ-18 and dietary intake in adults. *Appetite*, *56*(1), 156-162.

Konttinen, H. (2020). Emotional eating and obesity in adults: The role of depression, sleep and genes. *Proceedings of the Nutrition Society*, *79*(3). https://doi.org/http://dx.doi.org/10.1017/S0029665120000166

Kringelbach, M. L. (2004). Food for thought: hedonic experience beyond homeostasis in the human brain. *Neuroscience*, *126*(4), 807-819.

Kringelbach, M. L. (2005). The human orbitofrontal cortex: linking reward to hedonic experience. *Nature reviews neuroscience*, *6*(9), 691-702.

Kringelbach, M. L., Stein, A., & van Hartevelt, T. J. (2012). The functional human neuroanatomy of food pleasure cycles. *Physiol Behav*, *106*(3), 307-316.

Landeta, J. (2006). Current validity of the Delphi method in social sciences. *Technological forecasting and social change*, *73*(5), 467-482.

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*, 9-16.

Maloney, P., Grawitch, M. J., & Barber, L. K. (2011). Strategic item selection to reduce survey length: Reduction in validity? *Consulting Psychology Journal: Practice and Research*, *63*(3), 162.

Marques, M. M., Wright, A. J., Corker, E., Johnston, M., West, R., Hastings, J., Zhang, L., & Michie, S. (2023). The behaviour change technique ontology: transforming the behaviour change technique taxonomy v1. *Wellcome open research*, *8*.

Marty, L., Chambaron, S., Nicklaus, S., & Monnery-Patris, S. (2018). Learned pleasure from eating: An opportunity to promote healthy eating in children? *Appetite*, *120*, 265-274.

Mason, T. B., Do, B., Wang, S., & Dunton, G. F. (2020). Ecological momentary assessment of eating and dietary intake behaviors in children and adolescents: A systematic review of the literature. *Appetite*, *144*, 104465.

McCrae, R. R., & Sutin, A. R. (2018). A five–factor theory perspective on causal analysis. *European Journal of Personality*, *32*(3), 151-166.

McDonald, J. D. (2008). Measuring personality constructs: The advantages and disadvantages of self-reports, informant reports and behavioural assessments. *Enquire*, *1*(1), 1-19.

McGuire, M. T., Wing, R. R., Klem, M. L., Lang, W., & Hill, J. O. (1999). What predicts weight regain in a group of successful weight losers? *Journal of Consulting and Clinical Psychology*, *67*(2), 177.

Mela, D. J. (2006). Eating for pleasure or just wanting to eat? Reconsidering sensory hedonic responses as a driver of obesity. *Appetite*, *47*(1), 10-17.

Michie, S., Van Stralen, M. M., & West, R. (2011). The behaviour change wheel: a new method for characterising and designing behaviour change interventions. *Implementation science*, *6*(1), 1-12.

Okoli, C., & Pawlowski, S. D. (2004). The Delphi method as a research tool: an example, design considerations and applications. *Information & management*, *42*(1), 15-29.

Paulhus, D. L. (1991). Measurement and control of response bias.

Paulhus, D. L., & Vazire, S. (2007). The self-report method. *Handbook of research methods in personality psychology*, *1*(2007), 224-239.

Pekkarinen, T., Takala, I., & Mustajoki, P. (1996). Two year maintenance of weight loss after a VLCD and behavioural therapy for obesity: correlation to the

scores of questionnaires measuring eating behaviour. *International journal of obesity and related metabolic disorders: journal of the International Association for the Study of Obesity*, *20*(4), 332-337.

Polidori, D., Sanghvi, A., Seeley, R. J., & Hall, K. D. (2016). How strongly does appetite counter weight loss? Quantification of the feedback control of human energy intake. *Obesity*, *24*(11), 2289-2295.

Polivy, J., Herman, C. P., & Mills, J. S. (2020). What is restrained eating and how do we identify it? *Appetite*, *155*, 104820.

Polivy, J., Herman, C. P., & Mills, J. S. (2023). Assessment of Restrained Eating and Dieting. *Assessment of Eating Behavior*, *6*, 15.

Poulain, J.-P. (2017). *The sociology of food: eating and the place of food in society*. Bloomsbury Publishing.

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.

Racine, S. E., Hagan, K. E., & Schell, S. E. (2019). Is all nonhomeostatic eating the same? Examining the latent structure of nonhomeostatic eating processes in women and men. *Psychological Assessment*, *31*(10), 1220.

Rozin, P., Levine, E., & Stoess, C. (1991). Chocolate craving and liking. *Appetite*, *17*(3), 199-212.

Ryan, R. M., & Deci, E. L. (2017). *Self-determination theory: Basic psychological needs in motivation, development, and wellness*. Guilford publications.

Schuman, M., Gitlin, M. J., & Fairbanks, L. (1987). Sweets, chocolate, and atypical depressive traits. *The Journal of nervous and mental disease*, *175*(8), 491-495.

Segal, Z., Williams, M., & Teasdale, J. (2018). *Mindfulness-based cognitive therapy for depression*. Guilford publications.

Sharifi, N., Mahdavi, R., & Ebrahimi-Mameghani, M. (2013). Perceived barriers to weight loss programs for overweight or obese women. *Health promotion perspectives*, *3*(1), 11.

Shiffman, S., Stone, A. A., & Hufford, M. R. (2008). Ecological momentary assessment. *Annu. Rev. Clin. Psychol.*, *4*, 1-32.

Slaney, K. L., & Maraun, M. D. (2008). A proposed framework for conducting data-based test analysis. *Psychological methods*, *13*(4), 376.

Strack, F., & Deutsch, R. (2004). Reflective and impulsive determinants of social behavior. *Personality and social psychology review*, *8*(3), 220-247.

Stubbs, J., Whybrow, S., Teixeira, P., Blundell, J., Lawton, C., Westenhoefer, J., Engel, D., Shepherd, R., Mcconnon, Á., & Gilbert, P. (2011). Problems in identifying predictors and correlates of weight loss and maintenance: implications for weight control therapies based on behaviour change. *obesity reviews*, *12*(9), 688-708.

Stubbs, R., Gail, C., Whybrow, S., & Gilbert, P. (2012). The evolutionary inevitability of obesity in modern society: implications for behavioral solutions to weight control in the general population. In *Obesity and weight management: Challenges, practices and health implications* (pp. 29-50). Nova Science Publishers Inc.

Stubbs, R., & Tolkamp, B. (2006). Control of energy balance in relation to energy intake and energy expenditure in animals and man: an ecological perspective. *British Journal of Nutrition*, *95*(4), 657-676.

Stubbs, R. J., Duarte, C., O'Driscoll, R., Turicchi, J., Kwasnicka, D., Sniehotta, F. F., Marques, M. M., Horgan, G., Larsen, S., & Palmeira, A. (2021a). The H2020 "NoHoW Project": A Position Statement on Behavioural Approaches to Longer-Term Weight Management. *Obesity facts*, *14*(2), 246-258.

Stubbs, R. J., Duarte, C., O'Driscoll, R., Turicchi, J., Kwasnicka, D., Sniehotta, F. F., Marques, M. M., Horgan, G., Larsen, S., Palmeira, A., Santos, I., Teixeira, P. J., Halford, J., Heitmann, B. L., & The, E. O. M. T. F. (2021b). The H2020 "NoHoW Project": A Position Statement on Behavioural Approaches to Longer-Term Weight Management. *Obes Facts*, *14*(2), 246-258. https://doi.org/10.1159/000513042

Stubbs, R. J., Duarte, C., O'Driscoll, R., Turicchi, J., & Michalowska, J. (2019). Developing evidence-based behavioural strategies to overcome physiological resistance to weight loss in the general population. *Proceedings of the Nutrition Society*, *78*(4), 576-589.

Stubbs, R. J., Hopkins, M., Finlayson, G., Duarte, C., Gibbons, C., & Blundell, J. (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*(5), 698-709.

Stubbs, R. J., Horgan, G., Robinson, E., Hopkins, M., Dakin, C., & Finlayson, G. (2023). Diet composition and energy intake in humans. *Philosophical Transactions of the Royal Society B*, *378*(1888), 20220449.

Stubbs, R. J., & Turicchi, J. (2021). From famine to therapeutic weight loss: Hunger, psychological responses, and energy balance-related behaviors. *obesity reviews*, *22*, e13191.

Stunkard, A. J., & Messick, S. (1985). The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*, *29*(1), 71-83.

Teixeira, P., Going, S. B., Sardinha, L., & Lohman, T. (2005). A review of psychosocial pre-treatment predictors of weight control. *obesity reviews*, *6*(1), 43-65.

Thorndike, E. L. (1904). Introduction to the theory of mental and social measurements. *New York: Science Press*.

Tylka, T. L. (2006). Development and psychometric evaluation of a measure of intuitive eating. *Journal of counseling psychology*, *53*(2), 226.

Tylka, T. L., Calogero, R. M., & Daníelsdóttir, S. (2020). Intuitive eating is connected to self-reported weight stability in community women and men. *Eating Disorders*, *28*(3), 256-264.

Vainik, U., Dagher, A., Dubé, L., & Fellows, L. K. (2013). Neurobehavioural correlates of body mass index and eating behaviours in adults: a systematic review. *Neuroscience & Biobehavioral Reviews*, *37*(3), 279-299.

Vainik, U., Garcia-Garcia, I., & Dagher, A. (2019). Uncontrolled eating: a unifying heritable trait linked with obesity, overeating, personality and the brain. *The European journal of neuroscience*, *50*(3). https://doi.org/https://dx.doi.org/10.1111/ejn.14352

Vainik, U., Neseliler, S., Konstabel, K., Fellows, L. K., & Dagher, A. (2015). Eating traits questionnaires as a continuum of a single concept. Uncontrolled eating. *Appetite*, *90*, 229-239.

Varkevisser, R., Van Stralen, M., Kroeze, W., Ket, J., & Steenhuis, I. (2019). Determinants of weight loss maintenance: a systematic review. *obesity reviews*, *20*(2), 171-211.

Wardle, J. (1987). Compulsive eating and dietary restraint. *British Journal of Clinical Psychology*, *26*(1), 47-55.

Warren, J. M., Smith, N., & Ashwell, M. (2017). A structured literature review on the role of mindfulness, mindful eating and intuitive eating in changing eating behaviours: effectiveness and associated potential mechanisms. *Nutrition Research Reviews*, *30*(2), 272-283.

Watkins, E. R. (2015). Mindfulness in the context of processing mode theory.

Wurtman, R. J., & Wurtman, J. J. (1986). Carbohydrate craving, obesity and brain serotonin. *Appetite*, *7*, 99-103.

Zhou, Y., Stubbs, R. J., & Finlayson, G. (2023). A neurocognitive perspective on the relationship between exercise and reward: Implications for weight management and drug addiction. *Appetite*, *182*, 106446.

Appendix A Supplementary Materials for Chapter Three

A.1 Search Strategy

Population:	NOT “child*”
Intervention (eating-related traits) AND	"overeat*" or "eating trait" or "eating-trait*" or "eating-related trait*" or "eat* behavio* trait*" or "appetit* trait*" or "eat* behavio*" or "eat* attitude*" or "undereat*" or "food approach"
Intervention (specific questionnaires) AND	"emotional eat*" or "PNEES*" or "disinhibition" or "TFEQ-D" or "restrain* eat*" or "TFEQ*" or "TFEQ hunger" or "TFEQ-H" or "binge eat*" or "BES" or "power of food" or "PFS" or "external eat*" or "food addiction" or "food craving" or "LOC-eat*" or "reward-based eating drive" or "hunger" or "intuitive eat*" or "Satiety responsiv*" or "Food responsiv*" or "TFEQ restraint" or "TFEQ-R" or "TFEQ-restraint" or "diet* restraint" or "fullness" or "desire to eat" or "urge to eat" or "reflective eat*" or "reactive eat*" or "homeostat* eat*" or "eating impulsiv*" or "eating-impulsiv*" or "self-regulation of eat*" or "eat* questionnaire*" or "appeti* questionnaire*" or "food* questionnaire*" or "DEBQ" or "mind* eat*" or "MEQ" or "IES" or "SMEQ" or "stunkard-messick eat* questionnaire" or "WREQ" or "weight-related eat* questionnaire" or "adult eat* behavio* questionnaire" or "AEBQ*" or "eat* satiety" or "GNQK" or "general nutrition knowledge questionnaire" or "SREBQ" or "food choice questionnaire" or "FCQ" or "food preference questionnaire*" or "FPQ" or "EAT-26" or "loss of control over eat*" or "leeds food preference questionnaire" or "LFPQ" or "Brownell stress eating questionnaire" or "MIDUS" or "stress eating items" or "PEMS" or "palatable eating motives scale" or "trait food craving questionnaire" or "FCQ-T-r" or "yale food

	addiction scale" or "YFAS" or "compulsive eating scale" or "CES" or "motivations to eat" or "eating attitudes test" or "EAT-40" or "multidimensional psychology of eating questionnaire" or "MPEQ" or "eating disorder diagnostic scale" or "eating disorder examination questionnaire" or "EDE-Q" or "Reward-related eating" or "RED-13"
Comparison	No comparison for systematic review For Energy Intake meta-analysis, compare high scores to low scores (for measures with defined high vs low scores)
Outcome AND	"food intake" or "energy intake" or “calori* intake” or exp energy intake/ or exp food intake/
Outcome AND	“BMI" or "body mass index" or "body composition" or exp body weights and measures/ or exp body weight/ or exp body mass index/
Limits	English language and humans

A.2 Characteristics of included studies

Authors	Study type	Population and Participant characteristics	Inclusion Information	Eating behaviour trait measured	BMI and EI method	Results (BMI)	Results (EI)
Alger, Seagle and Ravussin [1]	Cross-sectional	N = 18 (sex not given). Age (M = 40 years, SD = 3.5) BMI not given.		BES.[2] Binge eaters were defined as scoring > 25 on the BES.	No data for how BMI was calculated. Participants ate ad libitum from two vending machines over 8 days. EI was calculated as intake over the initial 8 days.	Missing data	Mean daily EI was not significantly different between obese binge eaters (M = 2587, SD = 454) and obese non-bingers (M = 2386, SD = 201).
Anderson, Schaumberg, Anderson and Reilly [3]	Cross-sectional	N = 137 college students, 63.5% female. Age (M = 19.3 years, SD = 1.3). BMI (M = 23.0, SD = 3.8). Ethnicity = 65.7% Caucasian.		IES.[4]	Weight and height were self-reported to calculate BMI. Participants were randomly assigned to either a small (8-inch) plate condition, (N = 72), or a large (12-inch) plate condition, (N = 65) of pasta with tomato sauce. Pasta	IES was significantly negatively correlated with self-reported BMI, ($r = -0.21$, $p < 0.05$)	Within the small plate condition, IES and pasta consumption were unrelated ($r = 0.19$). Within the large plate condition, levels of intuitive eating and pasta consumption were significantly related, (r =

					consumption was weighed by digital scale.		0.53, p < 0.001).
Anderson, Reilly, Schaumberg, Dmochowski and Anderson [5]	Cross-sectional	N = 125 university students, 64% female. Age (M = 19.3 years, SD = 1.3). BMI (M = 23.0, SD = 4.0). Ethnicity = 65.4% Caucasian.		IES.[4] MEQ.[6] TFEQ-R.[7] EDDS.[8]	BMI was calculated from self-reported items in the EDDS scale. Pasta/sauce consumption was weighted using a digital food scale.	TFEQ-R (r = 0.44, p < 0.05) and EDDS (r = 0.40, p < 0.05) were significantly correlated with BMI. IES (r = -0.25) and MEQ (r = 0.01) were not significantly correlated with BMI.	No EBT were significantly correlated with EI (IES, r = 0.27 MEQ, r = 0.15 TFEQ-R, r= -0.13 EDDS, r = -0.29). However, controlling for gender, hierarchical multiple regression analyses suggested that IES, MEQ and TFEQ-R total scores accounted for 8% of the total variance in EI. Only IES accounted for a significant amount of unique variance for EI.

Appelhans, Liebman, Woolf, Pagoto, Schneider and Whited [9]	Cross-sectional	N = 62 overweight adults, 100% female. Age (M = 31.0 years, SD = 7.7). BMI (M = 31.5, SD = 3.4). Ethnicity = 37% Non-Hispanic White, 32% Black/African American.		PFS.[10]	Height and weight were measured in light clothing in the laboratory. Participants consumed a preload (oatmeal) and then complete the taste test. The six sample foods included: crisis, peanuts, chocolate, raisins (palatable), crackers and cheerio's (bland). Food was weighed before and after consumption.	BMI and PFS were not significantly correlated (r = -0.17).	PFS was not significantly correlated with palatable food intake (r = 0.12) or bland food intake (r = -0.04).
Ard, Desmond, Allison and Conway [11]	Cross-sectional	N = 150 adults, 47% female. Age (M = 43 years, SD = 12.25). BMI (M = 28.9, SD = 4.90).		TFEQ (restraint, disinhibition and hunger).[7]	Weight and height were measured using standard techniques. Three meals over the course of one day, e.g.,	Missing data	Restraint was negatively correlated with EI. Controlling for sex, race, and BMI, restraint was a significant

		Ethnicity = 8% non-Hispanic African Americans.			bread, bacon, pizza, salad, cookies. All foods consumed were recorded, and uneaten foods were returned and measured		predictor of energy intake (r=-0.23, p < 0.01). Disinhibition was not a significant predictor of total EI.
Arumae, Kreegipuu and Vainik [12]	Cross-sectional	N = 39, 100% female. Age (M = 25.51 years, SD = 5.99). BMI (M = 22.51, SD = 3.58).		Binge eating subscale EDAS.[13]	Information about participant's height and weight were collected via an online form. EI was measured as the intake of snack foods (waffles, peanuts, raisins, and pretzels).	Binge eating was not correlated with BMI (r = 0.03)	Binge eating was significantly correlated with snack food intake (r = 0.40, p < 0.05).
Bellisle, Dalix, Airinei, Hercberg and Péneau [14]	Cross-sectional	N = 40, 100% female. Age (M = 26.15 years, SD = 7.59). BMI (M = 21.95, SD = 2.85)	Condition 1,3 and 5 are included because they act as controls (eating alone, eating alone with a neutral TV program on, eating alone	TFEQ restraint.[7] Participants are categorised into high (>10) and low (≤5) restraint levels.	The laboratory physician measured participants height and weight. Test meals included ground beef and mashed potatoes, fruit	Participants with high levels of restraint did not have significantly different BMI's (M = 22.4, SEM = 0.5) compared to participants with low levels of restraint (M =	There were no differences in EI depending on level of restraint.

			whilst listening to a radio detective story).		sherbets, plain water. Leftovers were weight to calculate EI.	21.5, SEM = 0.4), p = 0.18.	
Bellisle and Dalix [15]	Cross-sectional	N = 41, 100% female. Age (M = 35 years, SD = 9). BMI (M = 21.3, SD = 1.9).		TFEQ.[7] DEBQ.[16]	No information for how BMI was calculated. Four identical lunch meals scheduled on the same day of the week with ≥1 week between tests. Meals consisted of casserole of ground beef and potatoes, fruit sherbet, water. Leftover food was weighed to calculate EI.	Total TFEQ score did not significantly correlate with BMI (r = 0.18)	TFEQ and DEBQ were not significantly correlated with EI.
Bryant, Caudwell, Blundell, Hopkins and King [17]	Cross-sectional	N = 58 overweight and obese adults, 67% female. Age (M = 35.57 years, SD = 9.78). BMI (M = 31.83, SD = 4.46).		TFEQ.[7] Participants were grouped as either high or low TFEQ-D (LD vs. HD) and with high or low TFEQ-R (LR vs. HR), which generated four	Body composition was measured on probe days. Breakfast consisted of cereal, toast, butter and jam and tea or	Missing data	TFEQ-H was significantly correlated with EI (r = 0.38, p < 0.01). TFEQ-R (r = -0.15), TFEQ-D (r = 0.25), TFEQ-rigid (r = -0.26)

				TFEQ-groups. For both TFEQ-R and TFEQ-D, ≥7 denoted a high score and ≤6 a low score.	coffee. Lunch consisted of cheese, salad sandwiches, ready salted crisps, and fruit malt loaf. Dinner consisted of lasagne, peas, and raspberry yoghurt. EI was calculated by weighing food before and after consumption.		and TFEQ-flexible (r = 0.001) were not significantly correlated with EI.
Chambers and Yeomans [18]	Cross-sectional	N = 64 university students, 100% female. Age (M = 21.8 years, SD = 4.8). BMI (M = 23.4, SD = 3.2).		TFEQ restraint and disinhibition.[7]	Participant's height and weight were recorded after the second test session. EI was calculated over two test days. Breakfast provided on both test days included vanilla yogurt, fresh strawberries, and water. The high fat breakfast	BMI did not significantly differ between high and low TFEQ-D groups (p = 0.16) or the TFEQ-R groups (p = 0.70).	Total EI (for snack foods) over the two test days did not differ significantly between high and low TFEQ-D groups (p = 0.15) or TFEQ-R groups (p = 0.88). Intake following the HF breakfast did not differ between the

					contained 61% fat; the high carbohydrate contained 80% carbohydrate content. Four snack foods were presented at the bogus taste test (Mini Cheddars, peanuts, chocolate buttons and grapes). All ingredients were weighed.		high and low TFEQ-D groups (p = 0.57), but after the HC breakfast the high TFEQ-D group consumed, on average, 31% more energy than the LD group (p = 0.04).
Coelho, Polivy, Herman and Pliner [19]	Cross-sectional	N = 116, university students, 100% female. Age not given. BMI not given.	Data is included for the control condition (no-cue) only.	RS.[20] Participants were classified as restrained eaters if they scored ≤15 or (n = 57) and unrestrained eaters if they scored >15 (n = 59).	Height and weight were measured by the experimenter. The test meal included gourmet chocolate chip, oatmeal-raisin, and double-chocolate cookies. Cookies were weighed before	Restrained eaters had a significantly higher BMI (M = 24.71, S.D. = 4.1) than unrestrained eaters (M = 21.71, S.D. = 3.6), p < 0.001.	The EI of restrained eaters in the control condition was significantly higher than unrestrained eaters (p < 0.03).

					and after consumption.		
Cools, Schotte and McNally [21]	Cross-sectional	N = 91, 100% female. Age (M = 28.6 years, SD = 8.9). BMI (M = 23.8, SD = 4.5).	Data is included for the neutral film condition only.	RRS.[22]	Demographic information was obtained. EI was measured as the intake of popcorn during the film.	Missing data	Low restrained eaters ate more popcorn (M = 24.7, SD = 11.70g) than high restrained eaters (M = 14.1, SD = 13.60g).
Dalton, Blundell and Finlayson [23]	Cross-sectional	N = 50 staff and university students, 100% female. Age (M = 26.25 years, SD = 8.6). BMI (M = 26.35, SD = 2.4).		BES.[2] Binge status (binge-type or non-binge type) was determined following a median-split of scores on the BES.	BMI was calculated from measuring standing height without shoes to the nearest 0.5 cm using a stadiometer. Body weight was measured using an electronic balance and recorded to the nearest 0.1 kg. There were two conditions (fasted and fed) In the fed condition, a fixed energy	There was no significant difference in BMI for the obese binge type (M = 32, SD = 1.26) compared to the obese non-binge type participants (M = 29.68, SD = 0.64). There was a difference in waist circumference between obese binge and obese non-binge type participants ($p < 0.05$). There was also no significant	Obese binge-types consumed more energy overall in both conditions (fasted and fed) compared to the obese non-binge and both lean types ($p < 0.01$). There was no significant difference in overall EI between obese non-binge, lean binge, and lean non-binge types.

					lunch meal (cheese sandwich and strawberry yoghurt) was consumed before the ad libitum food intake task. The test meal included six pre-weighed bowls of snack foods (chocolate, biscuits, cookies, crisps, peanuts, and tortilla chips). Each bowl was weighed.	difference in BMI for the lean binge-type (M = 22.44, SD = 0.47) compared to the lean non-binge type participants (M = 21.71, SD = 0.38).	
Dalton, Blundell and Finlayson [24]	Cross-sectional	N = 24 staff and university students, 100% females. Age (M = 25.42 years, SD = 6.42). BMI (M = 30.30, SD = 2.6).		BES.[2] CoEQ (control of eating questionnaire) [25,26].	Standing height without shoes was measured to the nearest 0.5 cm using a stadiometer. Body weight was measured using an electronic balance and	There was no significant difference in BMI between obese binge-types (M = 31.5, SD = 1.3) and obese non-binge types (M = 30.1, SD = 0.4). Obese binge-types had	Obese binge-types consumed more energy overall from the ad libitum snack box compared to obese non-binge-types ($p < 0.02$). Laboratory-based total EI

					recorded to the nearest 0.1 kg. EI was examined at breakfast, lunch, and dinner. Ad libitum test meals included breakfast (cereal, toast, milk, butter, jam), lunch (sandwiched, yogurt, crackers), dinner (pasta, sauce, side salad, garlic bread, chocolate cake rolls). Participants were also given a snack box, which contained four snacks representing high-fat savoury, low-fat savoury, high-fat sweet, low-fat sweet Food was measured to the nearest 0.1g.	significantly greater fat mass (M = 36.3kg, SD = 3.8) than non-binge types (M = 27.4kg, SD = 1.4), $p < 0.05$.	was higher in the obese binge types (M = 3417.5 (SD = 192.2) compared to the obese non-binge-types (M = 2590.7, SD = 143.9).

Dalton, Finlayson, Blundell and Hill [27] study 1	Cross-sectional	N = 80 staff, students, and local residents, 67.5% female. Age (M = 26.5 years, SD = 8.1). BMI (M = 24.2, SD = 4.3).		CoEQ.[25,26]	Standing height without shoes was measured to the nearest 0.5 cm using a stadiometer. Body weight was measured using an electronic balance and recorded to the nearest 0.1 kg. The ad libitum food intake task included six pre-weighed bowls of palatable high-fat snack foods (milk chocolate, chocolate finger biscuits, cookies, ready salted crisps, salted peanuts and flavoured tortilla chips). Each bowl was weighed.	Three CoEQ subscales were correlated with BMI. Craving control ($r = -0.31$, $p < 0.001$), positive mood ($r = -0.23$, $p < 0.01$), craving for sweet ($r = 0.23$, $p < 0.01$).	Three subscales were correlated with total EI. Craving control ($r = -0.20$, $p < 0.05$), positive mood ($r = -0.21$, $p < 0.05$) and craving for sweet ($r = 0.40$, $p < 0.001$).

Dalton, Finlayson, Blundell and Hill [27] study 2	Cross-sectional	N = 50 staff, students, and local residents, 100% female. Age (M = 24.3 years, SD = 5.9). BMI (M = 27.1, SD = 5.4).		“”	“”	“”	“”
Dalton, Hollingworth, Blundell and Finlayson [28]	Cross-sectional	N = 30 staff, students, and local residents, 100% female. Age (M = 28.0 years, SD = 10.6). BMI (M = 23.1, SD = 3.0). Ethnicity = 77% Caucasian.		CoEQ.[25,26] SQ.[29] To determine whether participants were reliably low or high in satiety responsiveness changes in subjective ratings of hunger and fullness were recorded before and following consumption of four, fixed energy breakfasts.	During an initial screening visit, standing height without shoes was measured to the nearest 0.5 cm using a stadiometer (Seca, Birmingham, UK). Body weight was measured using an electronic balance (Seca, Birmingham, UK) and recorded to the nearest 0.1 kg. Tests foods included a fixed energy breakfast (muesli, yoghurt,	There was no significant difference in BMI for the low satiety phenotypes (M = 24.6, SD = 2.6) compared to the high satiety phenotypes (M = 22.7, SD = 3.1).	The low satiety phenotype consumed for energy from the ad libitum lunch in the 25% ($p < 0.02$) and 35% RMR ($p < 0.01$) conditions compared to the high satiety phenotype. There were no differences in EI in the 20% RMR condition or 30% RMR condition.

					semi-skimmed milk, and honey) and ad libitum lunch (tomato and herb risotto, strawberry yoghurt, and garlic bread). Food was measured to the nearest 0.1g.		
de Witt Huberts, Evers and de Ridder [30] study 1	Cross-sectional	N = 57 university students, 100% female. Age (M = 20.91 years, SD = 2.0). BMI (M = 21.81, SD = 2.95).		RS.[20]	No information on how BMI was measured. Food intake was measured as a bogus taste test of four different snack types (two high calorie, two low calorie).	BMI correlated significantly with restraint (r = 0.49, p < 0.01).	Restraint did not correlate significantly with total calorie intake (r = 0.00).
de Witt Huberts, Evers and de Ridder [30] study 2		N = 43, 100% female. Age (M = 22.67, SD = 2.84). BMI (M = 22.58, SD = 3.11).			Identical to study 1 except participants compared different brands of palatable snacks (chips, peanuts, cookies). For each food, participants	BMI correlated significantly with restraint (r = 0.36, p < 0.05).	Restraint did not correlate significantly with total calorie intake (r = -0.16).
de Witt Huberts, Evers and de Ridder [30] study 3		N = 42, 100% female. Age (M = 20.57, SD = 2.70).				BMI did not correlate significantly with restraint (r = 0.02).	Restraint did not correlate significantly with total calorie

		BMI (M = 20.98, SD = 1.98).			were provided with two different brands.		intake (r = -0.09).
Drapeau, Blundell, Therrien, Lawton, Richard and Tremblay [31]	Cross-sectional	N = 51, 45% female. Age (M = 37.80 years, SD = 7.3). BMI (M = 27.65, SD = 5.28).		TFEQ.[7] SQ.[32]	Height (bathing suit, without shoes), waist circumference and percentage body fat were assessed for each participant. There were three test meals (standardized breakfast, ad libitum lunch and ad libitum dinner). Lunch was a buffet style meal (e.g., ham, cheese, bread, salad, biscuits) and dinner was a meal lasagne and granola bar. All foods were weighed before and after consumption	In women, BMI was negatively correlated with SQ for PFC (r = 0.49, p< 0·02). In men, only BMI was positively correlated with SQ for fullness (r = 0.44, p < 0.02).	SQ for fullness was the only subscale that was significantly correlated with total EI (r = -0.42, p < 0.001).

Drapeau, Jacob, Panahi and Tremblay [33]	Cross-sectional	N = 100, 71% female. Age (M = 38.7 years, SD = 8.7). BMI (M = 33.2, SD = 3.6).	Data is included for the control groups only.	SQ.[32] Participants were divided in two satiety responsiveness groups using the SQ median. LSR group = mean SQ < 10.1 mm/100 kcal and HSR group = mean SQ ≥ 10.1 mm/100 kcal).	Body weight was measured to the nearest 0.1 kg using a digital scale, and height to the nearest 0.1 cm using a standard stadiometer. EI was measured using a buffet-type meal which included a variety of cold foods. All foods were weighed to the nearest 0.1g immediately before and after the test meal.	The LSR group did not significantly differ in BMI (33.7, SD = 3.9) from the HSR group (M = 32.6, SD = 3.3), p = 0.11.	EI in the low LSR group was not significantly different to the HSR group (p = 0.74).
Dweck, Jenkins and Nolan [34]	Cross-sectional.	N = 64 university students, 100% female. Age (M = 18.8 years, SD = 0.4). BMI (M = 24.5, SD = 0.6). Ethnicity = 87.5% White.	Data is included for study 2 because a control group is used.	DEBQ.[16] Participants were divided into emotional eating groups with classification of high emotional eating >2.6 and low emotional eaters <1.8. Participants scoring in	Height and weight were measured using a stadiometer and digital scale. After the no-stress (control condition) the participants were presented with a snack tray and water	BMI was not correlated with emotional eating (r = 0.163) or external eating (r = -0.178). BMI was correlated with restraint (r = 0.309, p < 0.05)	There was no significant corelation between emotional eating and EI in the control condition (r = -0.03)

				between were classified as moderate emotional eaters. Only 12 participants scored below 1.8, so they were grouped with the moderate emotional eaters (low/moderate N = 36; high N = 28).	(cookies, cheese, candies, jellybeans, crackers and celery sticks). Each item was weighed before and after consumption.		
Ely, Howard and Lowe [35]	Cross-sectional	N = 79, 100% female. Age (M = 20.70 years, SD = 2.60). BMI (M = 22.45, SD = 2.14).		PFS.[10]	Participant's height and weight were measured to calculate BMI. Participants were given a preload (oatmeal) before the taste test. Food intake was measured as the consumption of snack foods (e.g., cookies and popcorn),	BMI was significantly correlated with PFS scores (p = 0.028).	PFS did not significantly predict snack food intake (p = 0.53) but did significantly predict the oatmeal preload intake (p = 0.02).

Epstein, Lin, Carr and Fletcher [36]	Cross-sectional	N = 273, 50.92% female. Age (M = 34.40 years, SD = 10.70). BMI (M = 29.90, SD = 7.40). Ethnicity = 27% Caucasian.		TFEQ disinhibition.[7] High and low disinhibition levels were calculated as <6 = low disinhibition and ≥6 = high disinhibition.	The participant's weight and height were measured using a digital scale (TANITA Corporation of America Inc., Arlington Heights, IL) and a digital stadiometer (Measurement Concepts & Quick Medical, North Bend, WA). Before ad libitum food intake, participants were given a choice of two energy bar preloads. The taste test included six palatable, high-energy-density snack foods (potato chips, Doritos, M&M's,	High TFEQ-D participants had significantly higher BMI's than low TFEQ-D participants ($p < 0.0001$).	Disinhibition was positively associated with EI.

					KitKat, and Butterfinger).		
Evers, de Ridder and Adriaanse [37] Study 3	Cross-sectional	N = 37 university students, 100% female. Age (M = 22.84 years, SD not given). BMI (M = 22.99, SD = 2.97).	Data is included for studies 3-5 because they use a control condition.	DEBQ.[16]	No information is included for how BMI was calculated. EI was assessed by bogus taste tests. Participants were provided with bowls containing different foods (chocolate, crisps, and cookies).	BMI did not differ between emotional (M = 22.19) and non-emotional eaters (M = 23.75), p = 0.112.	Emotional eating did not predict EI (p = 0.45).
Evers, de Ridder and Adriaanse [37] Study 4		N = 57 university students, 100% female. Age (M = 20.80 years, SD not given). BMI (M = 21.80, SD = 2.46).			No information is included for how BMI was calculated. EI was assessed by bogus taste tests. Participants were provided with bowls containing different foods (chocolate, crisps, cookies and fruit).	BMI did not differ between emotional (M = 21.92) and non-emotional eaters (M = 22.19), p = 0.720.	Emotional eating did not predict EI (p = 0.76).

Evers, de Ridder and Adriaanse [37] Study 5		Study 3 and 4 results combined				BMI did neither differ between emotional (M = 21.71) and non-emotional eaters (M = 22.30) nor between studies, p = 0.134.	Emotional eating did not predict EI (p = 0.73).
Fedoroff, Polivy and Herman [38]	Cross-sectional	N = 91 university students, 100% female. Age (M = 20.86 years, SD = 5.13). BMI not given.	Data is included for control group (no cue and free thoughts condition) only.	RRS.[22] Participants with scores ≤14 were classified as unrestrained eaters and those with scores of ≥15 were classified as restrained eaters.	The participant's height and weight were recorded. After the control condition, participants were given a plate of four individual pizzas.	Missing data	The EI of unrestrained eaters (M = 103.76, SD = 30.91) was larger than that of the restrained eaters (M = 89.06, SD = 29.38).
Finlayson, Arlotti, Dalton, King and Blundell [39]	Cross-sectional	N = 34 non-dieting adults, 100% female. Age (M = 24.10 years, SD = 5.83). BMI (M = 21.90, SD = 2.92).		BES.[2]	No information given for how BMI was calculated. A preload-test meal design was used. The preload consisted of jam on white bread with chocolate milk. The ad	Binge eating was positively correlated with BMI (r = 0.37, p < 0.05).	Before and after adjustment for BMI, binge eating was positively correlated with EI (r = 0.35, p < 0.05 and 0.35, p < 0.05).

					libitum test meal comprised of 8 different foods that were high or low in fat and sweet or non-sweet in taste (crisps, cheese, biscuits, salad, crackers, and fruit salad. Each plate was weighed before and after consumption.		
Finlayson, Blundell, Bordes, Griffioen-Roose and de Graaf [40]	Cross-sectional	N = 30, 100% female. Age (M = 21.90 years, SD = 2.74). BMI (M = 22.7, SD = 2.19).		TFEQ.[7] The authors also sent data for BES, DEBQ and PFS.	BMI was calculated by measuring height, and weight using bioelectrical impedance. A pre-load study design was used (milk-based drinks). The test meal included 8 foods that differed in sweet or savoury taste (see Finlayson, Arlotti, Dalton,	BMI was significantly correlated with binge eating ($r = 0.41$, $p < 0.05$), emotional eating ($r = 0.37$, $p < 0.05$) and disinhibition ($r = 0.44$, $p< 0.05$). BMI was not significantly correlated with DEBQ restraint ($r = 0.24$), hunger ($r = 0.21$), external eating ($r = 0.22$)	Disinhibition was highly correlated with total food intake after consumption of the sweet preload ($r = 0.59$, $p<0.001$). There were no relationships between other TFEQ scales and food intake.

					King and Blundell [39]).	and power of food (r = 0.31).	
Guerrieri, Nederkoorn, Schrooten, Martijn and Jansen [41]	Cross-sectional	N = 46 university students, 100% female. Age (M = 20.4 years, SD = 2.00). BMI (M = 21.99, SD = 2.44).		RS.[20]	Participants self-reported their weight and height. A bogus taste test enabled EI to be tested. Participants were given chocolate, wine gums, marshmallows, and nuts. Food was weighed before and after consumption.	BMI significantly differed between high restrained eaters (M = 22.76, SD = 2.66) and low restrained eaters (M = 21.13, SD = 1.87), $p < 0.05$.	Restrained eaters (M = 443.76, SEM = 30 kcal) consumed significantly more calories than the unrestrained eaters (M = 276.61, SEM = 32 kcal).
Haynes, Lee and Yeomans [42]	Cross-sectional	N = 80 staff and university students, 100% female. Age (M = 23.28 years, SD = 12.17). BMI (M = 22.23, SD = 5.69).	Data is included for the control condition (no-stress).	TFEQ.[7] Women were categorised a priori as high or low on both TFEQ-R and TFEQ-D, according to whether they fell above or below the median on both measures determined from a previous sample	Weight and height were recorded in the laboratory. Breakfast consisted of either yogurt or cereal. The test meal served at lunch comprised of cheese sandwiches, egg sandwiches,	BMI was not significantly different across the four different TFEQ-R and TFEQ-D categories.	LR-HD consumed significantly more food than HR-HD ($p < 0.05$) and marginally more food than HR-LD ($p = 0.08$) and LR-LD ($p = 0.08$.)

				of 150 women from the same population.	cheese, crisps, tomato, cake, chocolate, and cookies. Each food item was weighed in its container to the nearest 0.1 gram before and after the test meal.		
Herhaus and Petrowski [43]	Cross-sectional	N = 50 adults with Obesity, 52% female. Age (M = 37.84 years, SD = 12.65). BMI (M = 33.63, SD = 3.94).	Data is included for the control condition (resting).	DEBQ.[16] Participants were split into high (N = 24) and low restrained eating (N = 26).	No data included for how BMI was calculated. The test food included four cheese sandwich halves, 12 biscuits, a fizzy drink and water. Food and beverages were weighed.	There were no significant differences in BMI between the LR group (M = 33.48, SD = 4.08) and the HR group (M = 33.78, SD = 3.85), p = 0.79.	There were no significant differences in EI between the HR group and the LR group (p = 0.74).
Herman and Mack [20]	Cross-sectional	N = 45 university students, 100% female. Age not given. BMI not given.		RS.[20]	Weight and height were measured by the experimenter to calculate BMI. Participants were assigned	Obese participants showed slightly more overall restraint, but the difference was not significant.	There was a significant positive correlation between restraint and EI in the 2 preload

					to either a no preload condition, 1 preload or 2 preload (milkshakes). The following taste test comprised of 3 contained of ice cream (chocolate, vanilla, and strawberry). The ice creams were weighed before and after consumption.		condition (r = 0.38). There was a weak positive correlation between restraint and EI in the 1 preload condition (r = 0.14). There was a negative correlation between restraint and EI in the no preload condition (r = -0.28). For normal weight participants, the HR group consumed more after the milkshake preload than after no preload. LR subjects consumed decreasing amounts of ice cream as a function of the

							size of the preload. Adding the data of participants with obesity did not substantially alter the results.
Herman, Polivy and Silver [22]	Cross-sectional	N = 80 university students, 100% female. Age not given. BMI not given.	Data is included for the unobserved (control) condition.	RRS.[22] Participants scoring ≤18 on the revised version of the scale were classed as unrestrained, and that subjects scoring >18 were classed as restrained.	The experimenter measured the weight and height of participants. A 5oz or 15oz preload was given to the participants. The ad libitum test meal consisted of four bowls containing cashews, peanuts, almonds and sunflower seeds. The experimenter re-weighed the four bowls.	Missing data	Restrained eaters ate significantly more nuts than unrestrained eaters ($p < 0.01$).
Higgs, Williamson	Cross-sectional	N = 73 students, 100% female.	Data is only included for study 2 because	DEBQ restraint.[16] TFEQ disinhibition.[7]	The participant's weight and height were	BMIs of the participants did not significantly	There was no significant effect of restraint or

and Attwood [44]		Age (M = 20 years, SD = 1.71). BMI (M = 21.00, SD = 1.45).	this study measured EBT and reported data for the outcomes.	Participants were allocated to one of four groups: LR/LD, HR/LD, HR/HD, LR/HD based on cut off scores of 2.3 for the DEBQ restraint scale and 8 for the TFEQ disinhibition scale.	measured. EI was measured as popcorn intake. Three bowls of popcorn were placed on the table. The bowls were weighed after consumption.	differ across conditions	disinhibition on popcorn intake.
Hofmann and Friese [45]	Cross-sectional	N = 63 university students, 100% female. Age (M = 21.6 years, SD = 2.4). BMI (M = 21.80, SD = 2.18).	Data is included for the control condition (no-alcohol)	TFEQ restraint.[7]	No information for how BMI was measured. Participants were given a package of M&Ms to taste test. Candies were weighed to determine consumption.	Missing data	Restraint was negatively correlated with candy consumption ($r = -0.47$, $p < 0.05$).
Hopkins, Michalowska, Whybrow, Horgan and Stubbs [46]	Cross-sectional	N = 59, 49% female. Age (M = 42.7 years, SD = 13.6). BMI (M = 26.1 years, SD = 3.8).	Data from only study 1 is included because study 2 does not include the associations between eating behaviour traits and EI or BMI.	DEBQ.[16]	Height was measured using a portable stadiometer and body weight was measured after voiding to calculate BMI. Food intake was measured using	No subscales of the DEBQ were correlated with BMI. Restraint ($r = -0.13$), External eating ($r = -0.48$), emotional eating ($r = 0.13$).	Restraint was negatively correlated with covert LWI ($r = -0.31$, $p < 0.05$). External eating ($r = 0.06$) and emotional eating ($r = 0.06$) were not

					a laboratory weighed intake method (LWI). A re-analysis of the data used only covert EI as the outcome variable to reduce the potential of an observer contaminating results.		correlated with covert LWI.
Jansen [47] study 1	Cross-sectional	N = 30, 100% female. Age (M = 23.5 years, SD = 5.85). BMI (M = 25.55, SD = 6.30).		DEBQ.[16] The participants were classified as restrained or unrestrained eaters based on the median split score on the DEBQ restraint scale.	Weight and height were measured. The taste test involved 10 large pre-weighed dishes containing: nuts, smarties, peanuts, marshmallows, unsalted peanuts, sugared peanuts, chocolate nuts, liquorice, shanghai nuts and cake. The	The restrained and unrestrained groups did not differ in BMI.	There was a marginally significant difference in EI between the restrained and unrestrained groups (p = 0.06), with restrained individuals eating more than unrestrained individuals.

					remaining food was weighed.		
Jansen [47] study 2	Cross-sectional	N = 42, 100% female. Age (M = 20.6 years, SD = 2.05). BMI (M = 22.2, SD = 1.65).		DEBQ.[16] Participants scoring ≥3.3 were classed as restrained (N = 17), whereas scorers <2.9, (N = 25) were classed as unrestrained.	Weight and height were measured. The taste test consisted of ice cream (chocolate, strawberry, and vanilla).	The restrained sample had a significantly higher BMI than the unrestrained sample ($p < 0.001$)	The restrained eaters ate significantly more ice cream than the unrestrained eaters ($p < 0.03$)
Jansen, Merckelbach, Oosterlaan, Tuiten and Van Den Hout [48]	Cross-sectional	N = 40 staff and university students, 100% female. Age (M = 25.5 years, SD = 8.1). BMI (M = 22.8, SD = 2.7).		RS.[20] Participants were classified as restrained or unrestrained based on the median split score on the RS.	Each participant's weight and height were measured. EI was measured through consumption of ice cream (strawberry, chocolate, and vanilla).	Restrained participants had a significantly larger BMI (M = 24.2, SD = 2.6), than unrestrained participants (M = 21.5, SD = 1.9) $p < 0.001$.	There was no main effect of restraint on EI.
Kakoschke, Kemps and Tiggemann [49]	Cross-sectional	N = 144 university students, 100% female. Age (M = 20.20 years, SD = 2.64). BMI (M = 22.90, SD = 5.11).		DEBQ external eating.[16]	No information for how BMI was measured. Food intake was measured as the amount of snacks consumed. The taste test	Missing data	External eating was significantly correlated with sweet food intake ($r = 0.28$, $p < 0.01$) but not savoury food intake ($r = 0.08$).

					consisted of M&M's, biscuits, crisps, and pretzels.		
Lattimore and Maxwell [50]	Cross-sectional	N = 119 university students, 100% female. Age (M = 23.60 years, SD = 7.70). BMI (M = 23.40, SD = 3.6).	Data is included for the one control condition (low cognitive load, colour name stroop test).	RS.[20]	Participant's self-reported height and weight. The taste test included portions of snack foods (crisps, biscuits, dried fruits). Food was weighed after consumption.	Missing data	There was no significant effect of restraint on EI
Long, Meyer, Leung and Wallis [51]	Cross-sectional	N = 27 university students, 100% female. Age (M = 21.10 years, SD = 3.64). BMI (M = 23.80, SD = 3.33).	Data is included from only the control condition.	EDI-2.[52]	Participants were weighed with digital scales and their height was measured using a stadiometer. The ad libitum test meal consisted of pasta and Dolmio pasta sauce. EI was measured by weighing food	Missing data	No significant correlations were found between EDI-2 and EI (Bulimia r = 0.01, Drive for Thinness, r = 0.09, Body Dissatisfaction, r = 0.06, all p > .05.)

					before and after consumption		
Martin, O'Neil, Tollefson, Greenway and White [53]	Cross-sectional	N = 91 adults with overweight and obesity, 81% female. Age (M = 43.20 years, SD = 10.70). BMI (M = 35.1 years, SD = 2.8). Ethnicity = 73.6% Caucasian.	Data included is from baseline measures.	FCI.[54]	No data given for the measurement of BMI. A laboratory-based taste test consisted of four types of food (baked potato chips, jellybeans, regular potato chips and M&M's). The serving bowls were weighed after consumption.	Missing data	There was a significantly positive correlation between FCI score and total EI ($r = 0.22$, $p < 0.05$). FCI significantly correlated more specifically with intake of regular potato chips ($r = 0.23$, $p < 0.05$) and M&M's ($r = 0.23$, $p < 0.05$).
Martin, Williamson, Geiselman, Walden, Smeets, Morales and Redmann Jr [55]	Cross-sectional	N = 36 staff and university students, 72% female. Age (M = 22.42 years, SD = 6.33). BMI (M = 22.05, SD = 2.15). Ethnicity = 88.9% White.		TFEQ restraint and disinhibition.[7]	Self-reported height and weight were used to calculate BMI for three LR females. All other participants' height and weight were measured at the	There were no significant differences in BMI across restraint or disinhibition conditions.	EI did not differ as a function of restraint ($p = 0.21$).

					PBRC by research staff. Two types of test meal were used, one sandwich type (chicken salad) or three types of sandwich (chicken salad, turkey and ham).		
McNeil, Lamothe, Cameron, Riou, Cadieux, Lafreniere, Goldfield, Willbond, Prud'homme and Doucet [56]	Cross-sectional	N = 246, 86.6% female. Age (M = 31 years, SD = 11). BMI (M = 26.50, SD = 6.00).		TFEQ.[7]	Participants in all studies were weighed to the nearest 0.1 kg with a digital scale. Their standing height without shoes was measured to the nearest centimetre using a wall stadiometer. Acute EI was measured with either a test meal selected from a validated food menu or a buffet in all	Missing data	TFEQ-H was positively correlated with daily EI ($r = 0.34$, $p = 0.01$). TFEQ-R and TFEQ-D were not correlated with daily EI.

					studies. Daily EI was calculated based on EI during the standard breakfast, the ad libitum test meal inside the laboratory, and from containers that were taken home for the remainder of that day. The participants brought back the containers the following day, at which time all remaining food items were weighed.		
Myhre, Buchwald, Kratz, Goldberg, Polivy, Melhorn, Schur and Cummings [57]	Cross-sectional	N = 32 twins, 100% female. Age (M = 31.5 years, SD = 13.6). BMI (M = 23.50, SD = 3.10).		RRS.[22] Restrained eaters scored ≥15 on this scale.	Participants self-reported their weight and height. A standardised breakfast drink was given to the participants. At midday participants	There were no significant differences in BMI between the restrained compared to unrestrained eaters.	There were also no significant differences in EI between the restrained compared to unrestrained eaters (p = 0.83).

					consumed a preload milkshake and were then presented with the ad libitum taste test (turkey sandwich, tortilla chips, fruits, and cookies). At dinner the meal consisted of teriyaki chicken, rice, peas, salad, roll, milk, and cookie.		
Nasser, Gluck and Geliebter [58]	Cross-sectional	N = 22 adults with Obesity, 100% female. Age (M = 31.25 years, SD = 7.50). BMI (M = 34.35, SD = 4.30).	Data is included for binge eaters and controls but not BED patients.	BES.[2] Controls were classified as no binge eating episodes. Binge Eaters were classified as having fewer than two binge episodes/week for 6 months.	No data given for how BMI was measured. The test meal consisted of Boost, a nutritionally complete food with water. Food was weighed before and after consumption	There were no significant differences in BMI between binge eaters and controls.	There were also no significant differences in EI between binge eaters and controls.
Nolan-Poupart, Veldhuizen,	Cross-sectional	N = 20, 50% female.		TFEQ.[7] PFS.[10]	Height and weight were measured after a mock scan.	Missing data.	No scales (TFEQ or PFS) were significantly

Geha and Small [59]		Age (M = 27 years, SD = 6.20). BMI (M = 25.00, SD = 4.00).			Participants first consumed a milkshake preload. After fMRI scans, the participants were offered a full bottle of chocolate milkshake. The amount consumed was weight after.		correlated with milkshake consumption.
Oliver, Wardle and Gibson [60]	Cross-sectional	N = 34 (control group), 61.76% female. Age (M = 26.10 years, SD = 5.45). BMI (M = 22.4, SD = 2.45).	Data is included for the control condition (non-stressful task).	DEBQ.[16] Participants were divided based on a median split into high and low emotional and restrained eaters.	Height and weight were recorded. Participants ate freely from a buffet lunch which included foods such as bread, butter, tomatoes, cheese, crisps, peanuts, fruits, jam, and biscuits. The foods were weighed before and after consumption.	Missing data.	There were no significant differences in EI between restrained and unrestrained eaters. There were also no significant differences between emotional and non-emotional eaters.

Ouwens, van Strien and van der Staak [61]	Cross-sectional	N = 209 university students, 100% female. Age (M = 20.90 years, SD = 2.40). BMI (M = 23.00, SD = 3.18).		DEBQ.[16] RS.[20] TFEQ.[7]	Participant's height and weight were measured. 109 participants were randomly assigned to the preload condition (strawberry milkshake). The other 100 participants were randomly assigned to the no preload condition. After preload or no preload, participants were given three pre-weighed plates of cookies to taste. The plates were weighed after consumption.	Missing data	There were no significant correlations with restraint scales (TFEQ, DEBQ or RS) and cookie consumption. However, there were significance positive correlations between TFEQ disinhibition ($r = 0.20$, $p < 0.001$) and cookie consumption as well as DEBQ disinhibition ($r = 0.25$, $p < 0.001$) and cookie consumption.
Peluso [62]	Cross-sectional	N = 24, 50% female. Age (Median = 24.00).	Data is included for the control condition and measures taken at baseline.	TFEQ.[7]	Height and weight were measured during the baseline	There were no significant differences between BMI and levels of restraint.	Levels of restraint were not significantly correlated with EI

		BMI (Median: = 29.10).			assessments. The control test meal consisted of pre-portioned and packaged macaroni cheese, which was weighed before and after consumption.		
Raspopow, Abizaid, Matheson and Anisman [63]	Cross-sectional	N = 46 university students (control condition), 100% female. Age (M = 20.72 years, SD = 2.43). BMI not given.	Data is included for the control condition (non-stressful event)	DEBQ emotional eating.[16]	The height and weight of participants was measured at the end of the laboratory session. Half of the participants in each condition were provided with 6 pre-weight miniature brownies and were allowed to eat freely.	Emotional eating was not correlated with BMI (r = 0.16) was but positively correlated with body fat (r = 0.23).	Emotional eating was not significantly correlated with food intake (r = 0.33).
Rideout, McLean and Barr [64]	Cross-sectional	N = 62 college students, 100% females. Age (M = 21.60 years, SD = 2.5).		TFEQ restraint.[7]	No information for how BMI was measured. Participants consumed ad libitum breakfast	BMI did not differ significantly between groups.	The EI of restrained eaters was significantly less than that of unrestrained

		BMI range = 18.5 to 25).			in the laboratory and lunch and dinner were also consumed in the laboratory if possible. Any meals that could not be consumed in the laboratory were taken away, consumed, returned, and re-weighed.		eaters (M = 2,095, SD = 569 kcal vs M = 2,423, SD = 475 kcal, $p < 0.05$).
Robinson and Haynes [65]	Cross-sectional.	N = 111, 50.45% female. Age (M = 31.10 years, SD = 11.80). BMI (M = 26.80, SD = 3.60).	Data is pooled from 3 studies.	DEBQ.[16]	Weight and height were measured using a digital scale and stadiometer to calculate BMI. The test foods over the 3 studies included pasta with tomato sauce, chicken curry, desserts, pesto pasta, carbonara and Bolognese. Data from lunchtime intake of the	No subscales of the DEBQ were correlated with BMI: restraint, ($r = 0.095$), emotional eating ($r = 0.074$), external eating ($r = -0.123$).	No subscales of the DEBQ were correlated with total calorie intake: restraint ($r = -0.026$), emotional eating ($r = 0.084$), external eating ($r = 0.058$).

					three studies was combined to create total calorie intake.		
Rolls, Castellanos, Shide, Miller, Pelkman, Thorwart and Peters [66]	Cross-sectional	N = 67, 65.67% female. Age (M = 24.83 years, SD not given). BMI (M = 26.10, SD not given).		TFEQ.[7] A score of ≥9 on the restraint factor of the TFEQ (high restraint) for women and < 9 (low restraint). For men, ≥8 (high restraint) and < 8 (low restraint).	Weight and height measurements were taken in shorts and t-shirt to determine BMI. Participants consumed 3 preloads (three soups that differed in fat) and no preload over 4 test sessions. Energy intake was calculated as the intake from the lunch buffet meal (e.g., turkey, cheese, salad, cookies).	There were no significant differences in BMI across restraint groups.	EI at lunch was not affected by restraint.
Ruddock, Field and Hardman [67]	Cross-sectional	N = 60, 100% female. Age (M = 23.92 years, SD = 9.38).		TFEQ restraint and disinhibition.[7]	Participant's weight and height were assessed to provide BMI.	BMI did not correlate with restraint or disinhibition.	Disinhibition was a significant positive predictor and

		BMI (M = 23.72, SD = 4.57).			Participants completed a tapping task, a lunch meal (sandwiches) and then consumed chocolate and grapes ad libitum. Food intake was measured as the amount of grapes and chocolate consumed.		restraint a significant negative predictor of calorie intake.
Ruzanska and Warschburger [68]	Cross-sectional	Middle-aged adults, N = 55 middle aged adults, 76.4% female. Age (M = 59.29 years, SD = 5.73). BMI (M = 26.88, SD = 5.11).		IES-2.[69]	Height and weight were assessed with validated devices. Food intake was measured using a taste test. The foods consisted of low-calorie foods (apples, carrots) and high calorie foods (chocolate, peanuts). The	IES-2 was negatively correlated with BMI (r = -0.399).	IES-2 was not significantly associated with total food intake. In addition, IES-2 subscales were not associated with total food intake. However, when sex was added as a covariate, the eating for physical reasons

					foods were weighed before and after consumption.		subscale was significantly associated with healthy food intake.
Schoch and Raynor [70]	Cross-sectional	N = 38, 100% female. Age (M = 20.30 years, SD not given). BMI (M = 22.43, SD not given).		TFEQ restraint.[7] Scores of ≤10 were classed as low restraint. Scores of ≥13 were classed as high restraint.	Height and weight measures were taken on an electronic scale with stadiometer to calculate BMI. Food intake was measured as the consumption of sandwich wraps, chips, fruit, and ice cream.	BMI significantly differed across restraint groups with low restraint participants having lower BMI's (M = 21.7, SD = 1.8) than high restraint participants (M = 23.1, SD = 1.4), $p < 0.01$.	The high restraint participants consumed significantly less energy (M = 437, SD = 169 kcal) than the low restraint participants (M = 559, SD = 207 kcal), $p < 0.05$.
Schotte, Cools and McNally [71]	Cross-sectional	N = 60 university students, 100% female. Age (M = 29.60 years, SD = 9.90) BMI (M = 23.90, SD = 0.9).	Data is included for the control condition (neutral film)	RRS.[22] Participants who scored below the sample median (< 17) were classified as low restraint and those who scored above it, as high restraint.	No information given on how BMI was measured. Participants were given a pre-weighed bag of popcorn. The amount of popcorn consumed was the measure of food intake.	There was a significant correlation between restraint and BMI ($r = 0.36$, $p < 0.01$).	Restrained eaters ate less during the neutral film than unrestrained eaters, but this effect was non-significant.

Schulte, Sonneville and Gearhardt [72]	Cross-sectional	N = 44 adults with overweight and obesity, 100% female. Age (M = 30.75 years, SD = 4.20). BMI (M = 33.68, SD = 5.46). Ethnicity = 52.3% White.		YFAS 2.0.[73] Participants were split by YFAS score into a food addiction group (N = 17) and control group (N = 27).	Height and weight were measured in the laboratory to calculate BMI. Participants completed a taste test task which consisted of 14 foods e.g., chocolate, cheese, applies, pizza and rice. After the taste test they were welcome to consume left over food. Foods were weighed before and after consumption.	BMI did not differ between the food addiction group and controls.	There were no differences in food consumption between the food addiction group and controls.
Shapiro and Anderson [74]	Cross-sectional	N = 86 university students (control group). Age (M = 19.30 years, SD = 3.0). BMI (M = 24.00, SD = 4.6). Ethnicity = 69.3% Caucasian.	Data is included for the control condition (no-stress)	RS.[20] Using the customary cut-off of 16, 99 participants were defined as non-restrained, and 54 participants were defined as restrained eaters.	Height and weight were measures using a Detecto scale. The taste test consisted of several foods that encompassed a variety of food	Missing data	Restrained eaters consumed significantly more calories from grapes than non-restrained eaters ($p < 0.05$). Un-

					categories e.g., cookies, grapes, pretzels and carrots. The food was weighed.		restrained eaters consumed significantly more pretzels than restrained eaters ($p < 0.01$). However, overall, there was no main effect of restraint.
Sim, Lee and Cheon [75]	Cross-sectional	N = 21 physically inactive men, 100% male. Age (M = 24 years, SD = 2.00). BMI (M = 26.7, SD = 1.80).	Data is included for the control condition (video watching)	DEBQ restraint.[16] Classification was based on the cut off values of >3 for restrained eaters and <3 for unrestrained eaters.	No information given for how BMI was measured. To assess EI, participants were given two bags of crisps.	Missing data	The EI of unrestrained eaters was slightly higher than restrained eaters, but a re-analysis found this effect failed to reach significance ($p = 0.60$).
Smith, Geiselman, Williamson, Champagne, Bray and Ryan [76]	Cross-sectional	N = 124 university students, 100% female. Age (M = 22.60 years, SD = 6.20). BMI (M = 23.55, SD = 4.45).		TFEQ restraint and disinhibition.[7] Four groups were formed based on restraint and disinhibition scores (LR/LD, HR/LD, LR/HD, HR/HD)	Height and weight were measured to calculate BMI. A pre-load study design was utilised whereby participants in the pre-load	The mean BMI of the LR/HD group was significantly larger than the LR/LD group and the HR/LD group (all p values < 0.05). Furthermore, the	Participants scoring high in disinhibition ate significantly more macaroni and beef than participants scoring low in disinhibition.

		Ethnicity = 86.3% Caucasian.			condition tasted four puddings. After the pre-load or no preload, participants were served a large dish of macaroni and beef.	mean BMI of the HR/HD group was significantly larger than the mean BMI of the LR/LD group ($p < 0.05$)	There was no main effect of restraint on food intake.
Stice, Fisher and Lowe [77] study 1	Cross-sectional	N = 64 university students, 100% female. Age (M = 19.10 years, SD = 3.2). BMI (M = 24.60, SD = 6.0). Ethnicity = 69% White	Studies 1-2 are included because they use lab-based measures of EI. For study 2, data is included for the 59 participants who did not meet the DSM-IV for bulimia or BED.	RS.[20] TFEQ restraint.[7]	A direct reading stadiometer and digital scale were used to measure height and weight. EI was measured as total intake of three types of cookies.	No measures of dietary restraint were significantly correlated with BMI.	Restraint measures were positively correlated with caloric intake (r = 0.30), but this relationship was insignificant.
Stice, Fisher and Lowe [77] study 2		N = 59, 100% female. Age (M = 41.20, SD = 10.90). BMI (M = 31.30, SD = 7.3). Ethnicity = 73% White.		TFEQ restraint.[7] EDEQ-R.[78]	Height and weight were measured. A standard breakfast was served to participants (cereal, fruit, bread roll and decaffeinated tea or coffee). Food items were	Missing data	TFEQ–R and EDEQ–R did not show significant correlations with caloric intake among the participants with no eating disorder (r = 0.02 and -0.18)

					weighed before and after consumption.		
Stice, Sysko, Roberto and Allison [79]	Cross-sectional	N = 94 (control condition), 50% female. Age (M = 30.22 years, SD = 12.54). BMI (M = 25.46, SD = 6.82).	Data is only included for study 1 (no calorie label condition) because this is the only study that fulfilled the inclusion criteria.	TFEQ restraint.[7]	No information for how BMI was measured. In the no calorie condition, participants were presented a menu without calorie labels. The dishes were weighed before and after consumption.	Missing data	Restraint was not significantly correlated with caloric intake in the no calorie label condition (r = -0.14).
Stinson, Votruba, Venti, Krakoff, Gluck and Perez [80]	Cross-sectional	N = 82, 35.37% female. Age (M = 38, SD = 12). BMI (M = 29, SD = 7).		TFEQ.[7] BES.[2]	No information included for how BMI was measured. A vending machine paradigm was used to measure EI.	Disinhibition (r = 0.34, p < 0.01), hunger cues (r = 0.23, p < 0.05) and BES (r = 0.26, p < 0.05) were significantly correlated with BMI. Restraint was not correlated with BMI (r = 0.10).	Restraint was significantly correlated with EI (r = -0.29, p < 0.01). Disinhibition (r = 0.11), hunger cues (r = 0.20) and BES (r = 0.08) were not significantly correlated with EI.
van Strien, Donker and	Cross-sectional	N = 203, 100% female.	Data from Study 1 and 3 are included	DEBQ.[16]	BMI was measured objectively.	Missing data	Food intake was significantly correlated with

Ouwens [81] study 3		Age (M = 20.88, SD = 2.39). BMI (M = 22.93, SD = 3.04).	because they measure BMI and EI.		Food intake was the sum of the grams of chocolate cookies eaten.		DEBQ-positive (r = 0.16, p < 0.01), DEBQ-negative (r = 0.22, p < 0.001) and DEBQ-external (r = 0.23, p < 0.001)
Vainik, Eun Han, Epel, Janet Tomiyama, Dagher and Mason [82]	Cross-sectional	N = 165 total (n = 51 for EI data) university students, 64.24% female. Age not given for Canadian sample. BMI: 22.6 (SD = 3.15).	Data included for Canadian University student sample because this sample measured BMI and EI.	RED-X5, RED-9, RED-13.[82]	BMI was computed from self-reported height and weight. Food intake was measured as the consumption of Lays potato chips, measured in grams by weighing the bowl before and after the session.	BMI was significantly correlated with all RED questionnaires. RED-X5 (r = 0.18, p < 0.05, RED-9 (0.17, p < 0.06), RED-13 (r = 0.18, p < 0.05).	Food intake was significantly correlated with all RED questionnaires. RED-X5 (r = 0.31, p < 0.05), RED-9 (r = 0.35, p < 0.05), RED-13 (r = 0.32, p < 0.05).
van Strien, Herman, Anschutz, Engels and de Weerth [83]	Cross-sectional	N = 45 university students, 100% female. Age (M = 21.80 years, SD = 3.60).	Only data from study 1 are included because they use a control condition.	DEBQ.[16]	Weight and height were measured to calculate BMI. Food intake was measured as the intake of two	A re-analysis of the summary data found that high emotional eaters had a larger BMI than low	Missing data

		BMI (M = 23.30, SD = 3.70).			pre-weighed bowls of crisps and M&M's.	emotional eaters (p = 0.035).	
van Strien and Ouwens [84]	Cross-sectional	N = 31 adults with moderate obesity, 100% female. Age (M = 21.65 years, SD = 3.32). BMI (M = 28.63, SD = 2.86).		DEBQ.[16]	Weight and height were measured. Half of the participants were randomly assigned to a preload condition (milkshake). Food intake was measured as the consumption of chocolate cookies.	Missing data	Emotional eating was positively correlated with food consumption (r = 0.40, p < 0.05). Restraint and external eating were not correlated with food consumption (r = 0.01 and r = 0.19).
Vijayvargiya, Chedid, Wang, Atieh, Maselli, Burton, Clark, Acosta and Camilleri [85]	Cross-sectional	N = 62 adults with obesity, 91.94% female. Age (M = 38 years, SD = 10.10). BMI (M = 36.80, SD = 4.80).		WEL.[86]	No information for how BMI was measured. Participants consumed a liquid preload then were invited to eat an ad libitum buffet meal (lasagne, vanilla pudding, and milk). The amount of food consumed was	Missing data	Buffet meal intake was significantly correlated with total WEL score (r = -0.26, p < 0.05) and the social pressure subscale of WEL (r = -0.44, p < 0.001).

					analysed using validated software.		
Visona and George [87]	Cross-sectional	N = 36 university students who are overweight, 100% female. Age (M = 26 years, SD = 7). BMI (M = 27, SD = 3).	Data is included for the control group (non-exercise condition).	TFEQ restraint.[7] High restraint >10, low restraint ≤10 Three groups were created: dieting high-restraint (D-HR), non-dieting high-restraint (ND-HR) and non-dieting low-restraint (ND-LR).	BMI was based on self-reported weight and height. After the non-exercise condition, participants chose their lunch meal ad libitum from a wide variety of foods e.g., fast food, sandwiches, pastry, chips, and cookies. The foods were weighed and recorded after consumption.	There were no significant differences in BMI between restraint groups.	Re-analysis of the data found that while high restrained eaters ate less than low restrained eaters, the difference was non-significant (p = 0.32).
Wallis and Hetherington [88]	Cross-sectional	N = 38 university students, 100% female. Age (M = 24.38 years, SD not given). BMI (M = 24.10, SD not given).	Data is included for the control condition (neutral words).	DEBQ.[16] Participants were allocated to one of four groups based on the median split score of 2.8 for restraint and emotional eating	Weight and height were measured using a stadiometer and portable scales to calculate BMI. Food intake was measured as the	There were no significant differences in BMI across the four restraint/emotional groups.	In the control condition, the LR/LE group had the greatest food intake (M = 52.1, SD = 23.4). However, a re-analysis combining

				(HR/HE, HR/LE, LR/HE, LR/LE).	consumption of chocolate buttons.		groups into high and low restraint found no significant difference in EI ($p = 0.12$).
Wallis and Hetherington [89]	Cross-sectional.	N = 26, 100% female. Age (M = 27.40 years, SD = 16.83). BMI = 24.25, SD = 5.66).	Results from study 2 are included because a control condition is used.	DEBQ emotional and restraint.[16] Participants were allocated to high and low groups on restrained and emotional eating using a median split of scores (2.6 for restraint and 2.5 for emotional eating).	BMI was based on self-reported weight and height. Two snack foods were presented on a tray (chocolate and dried fruit).	The high and low emotional groups did not differ significantly in BMI. However, the high restraint group had a significantly higher BMI than the low restraint group ($p < 0.05$).	Missing data for effects in the control condition.
Wardle and Beales [90]	Cross-sectional	N = 50 volunteers from a university, 100% female. Age (M = 26.34 years, SD = 9.47). BMI (M = 21.51, SD = 2.42).		DEBQ restraint.[16] Restraint classification was based on the median split of scores.	Weight and height were measured to calculate BMI. Half of the subjects were given a preload (two milkshakes). Food consumption was measured as the amount of	The correlation between restraint and BMI did not reach significance ($r = 0.18$, $p = 0.10$).	The restraint group showed a significant positive correlation with food intake ($r = 0.24$, $p < 0.05$).

					food eaten during the taste test, which consisted of three two-litre containers of ice-cream. The containers were weighed before and after consumption.		
Westenhoefer, Broeckmann, Münch and Pudel [91]	Cross-sectional	N = 133 young adults from a university and hospital, 100% female. Age (M = 25.70 years, SD = 5.73). BMI (M = 21.40, SD = 1.90).		TFEQ.[7] Group Classifications were based according to the medians of a representative sample of the German population. RRS[22] to assess flexible and rigid control of eating behaviour.	No information for how BMI was measured. 65 participants were given a preload (banana milkshake). Then participants were given pre-weighed containers of ice-cream (vanilla, chocolate, and strawberry).	BMI was positively correlated with TFEQ-R ($r = 0.18$, $p < 0.05$), TFEQ-D ($r = 0.23$, $p < 0.01$), TFEQ-H ($r = 0.17$, $p < 0.05$) and rigid control ($r = 0.24$, $p < 0.01$). BMI was not significantly correlated with flexible control ($r = 0.03$, $p = 0.70$).	In the no-preload condition, restraint was not correlated with ice-cream intake ($r = 0.03$). However, disinhibition ($r = 0.30$, $p < 0.01$) and hunger ($r = 0.27$, $p < 0.05$) were correlated with ice-cream intake. In the preload condition, restraint was uncorrelated with ice-cream intake (r =

							0.09). Again, both disinhibition (r = 0.48, p < 0.001) and hunger (r = 0.28, p < 0.05) were correlated with ice-cream intake. There was a significant main effect of rigid restraint on ice-cream intake (p < 0.05) but no significant effect of flexible restraint.
Yeomans and Coughlan [92]	Cross-sectional	N = 96 staff and university students, 100% female. Age (M = 21.60 years, SD = 3.92). BMI (M = 22.40, SD = 3.92).	Data is included for the control condition (neutral mood).	TFEQ restraint and disinhibition.[7] Participants were categorised as high or low on both TFEQ-R and TFEQ-D, based on the median split from a previous sample of 150 women from the same population.	Participant's height and weight were measured to calculate BMI. Food intake was assessed by weighed consumption of two snack foods (popcorn and raisins).	Women in the high TFEQ-D group had a larger BMI than those in the low TFEQ-D group (p < 0.05). There were no significant differences in BMI between the TFEQ-R groups.	The low TFEQ-R groups had larger food intakes than the high TFEQ-R groups, however a re-analysis of the data found the difference to be non-significant (p = 0.12).

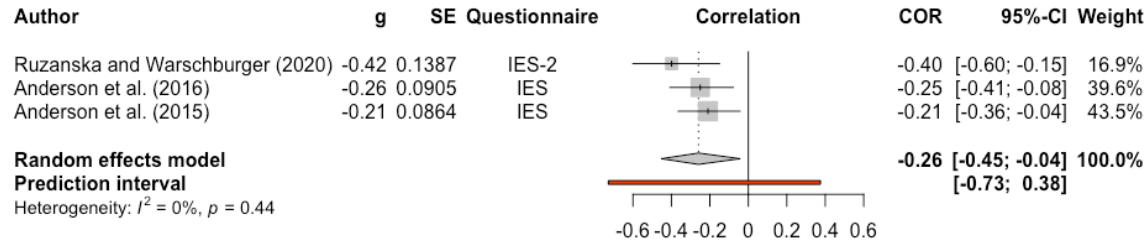
							A re-analysis found no differences in food intake between high and low TFEQ-D groups (p = 0.73)
Yeomans, Tovey, Tinley and Haynes [93]	Cross-sectional	N = 40 staff and university students, 100% female. Age (M = 24.45 years, SD = 10.20). BMI (M = 22.65, SD = 5.38).		TFEQ restraint and disinhibition.[7] Participants were categorised as high (H) or low (L) on both TFEQ-R and TFEQ-D, based on the median split from a previous sample of 150 women from the same population.	Weight and height of all participants were recorded at the end of testing, to calculate BMI. Participants were served breakfast (cereal, milk, and orange juice). The food for the test meal consisted of pasta served with either an unseasoned (bland) or seasoned (palatable) tomato and onion sauce. Food was	A re-analysis was conducted. BMI did not differ between TFEQ restraint groups. (p = 0.65). The BMI of the high disinhibition group was greater than that of the low disinhibition group (p = 0.05)	HD was associated with increased food intake response to palatability, whereas HR was associated with reduced sensitivity to palatability. The LR–HD group was more responsive to palatability than any other group.

					weighed automatically by a digital balance.		
Zambrowicz, Schebendach, Sysko, Mayer, Walsh and Steinglass [94]	Cross-sectional	N = 70 healthy controls with no prior eating disorder, 98.57% female. Age (M = 27.31 years, SD = 9.53). BMI (M = 22.60, SD = 3.00).	Data is only included for the control group.	TFEQ.[7] EDE-Q.[95]	No information as to how BMI was measured. Caloric intake was calculated based on grams consumed from multi-item test meals which included a range of foods (e.g., chicken, salad, cookies, sandwiches and crisps).	Missing data	Caloric intake was only correlated with TFEQ-restraint ($r = -0.32$, $p < 0.05$). Caloric intake was not correlated with TFEQ-hunger ($r = 0.20$, $p = 0.11$), TFEQ-disinhibition ($r = 0.21$, $p = 0.09$) or EDE-Q-restraint ($r = -0.23$, $p = 0.06$).
Zuraikat, Roe, Smethers, Reihart and Rolls [96]	Cross-sectional	N = 79, 69.62% female. Age (M = 33.90, SD = 12.70) BMI (M = 25.60, SD = 5.00). Ethnicity = 79% White.		AEBQ SR.[97]	Participants had their height and weight measured to calculate BMI. The experimental lunch consisted of pasta, salad, bread, and water. Intake was determined	Satiety responsiveness was correlated with BMI ($r = -0.19$) but this trend was not significant ($p = 0.087$). However, satiety responsiveness was significantly correlated with	Participants scoring low on satiety responsiveness increased their intake when they were served larger meals ($p < 0.0001$).

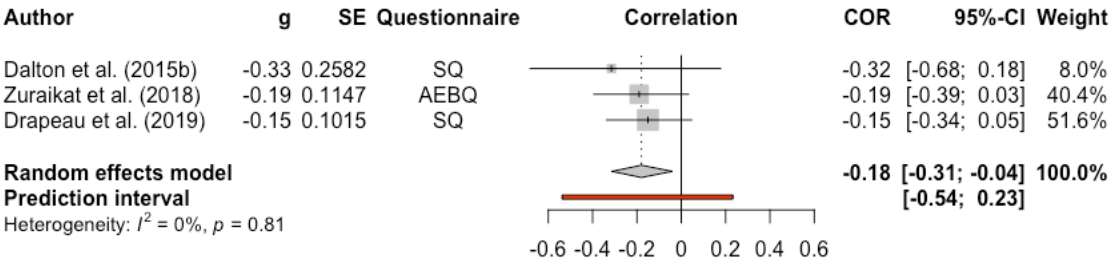
					by weighing food before and after consumption.	body weight (r = -0.35, p < 0.001).	

Note. BMI (body mass index weight (kg/m^2)), SD (standard deviation), BES (Binge Eating Scale), EI (Energy Intake), IES (Intuitive Eating Scale), MEQ (Mindful Eating Scale), TFEQ-R (Three Factor Eating Questionnaire Restraint), TFEQ-H (Three Factor Eating Questionnaire Hunger), TFEQ-D (Three Factor Eating Questionnaire Disinhibition), HD (High Disinhibition), LD (Low Disinhibition), HR (High Restraint), LR (Low Restraint), EDDS (Eating Disorders Diagnostic Scale), PFS (Power of Food Scale), EDAS (Eating Disorders Assessment Scale), DEBQ (Dutch Eating Behaviour Questionnaire), HE (High Emotional Eating), LE (Low Emotional Eating), HF (high fat), LF (low fat), HC (high carbohydrate), LC (low carbohydrate), RS (Restraint Scale), RRS (Revised Restraint Scale), CoEQ (Control of Eating Questionnaire), RMR (resting metabolic rate), SQ (Satiety Quotient), PFC (Prospective Food Consumption), HSR (High Satiety Responsiveness), LSR (Low Satiety Responsiveness), EBT (Eating Behaviour Traits), LWI (Laboratory Weighed Intake Method), EDI-2 (Eating Disorders Inventory). FCI (Food Craving Inventory), IES-2 (Intuitive Eating Scale 2), YFAS 2.0 (Yale Food Addiction Scale). EDEQ-R (Eating Disorder Examination Questionnaire—Restraint subscale), WEL (Eating Self-Efficacy), EDE-Q (Eating Disorders Examination Questionnaire, AEBQ SR (Adult Eating Behaviour Questionnaire Satiety Responsiveness subscale).

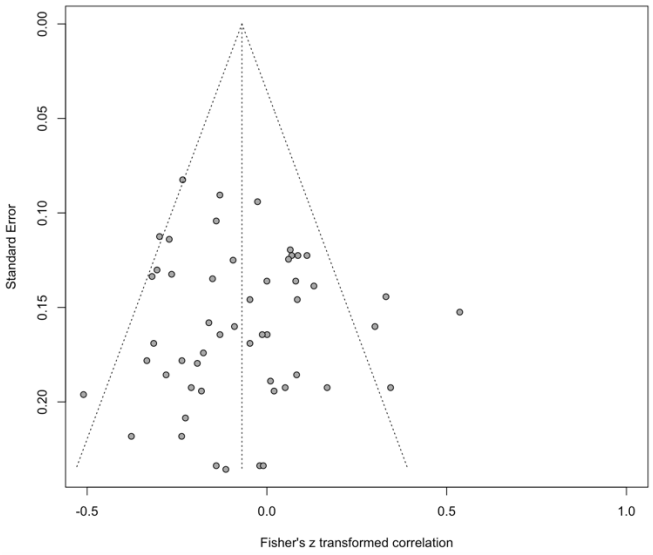
A.3 Forest plot of the effect of IES on BMI



A.4 Forest plot of the effect of satiety responsiveness on BMI



A.5 Funnel plot of the effect of restraint on EI



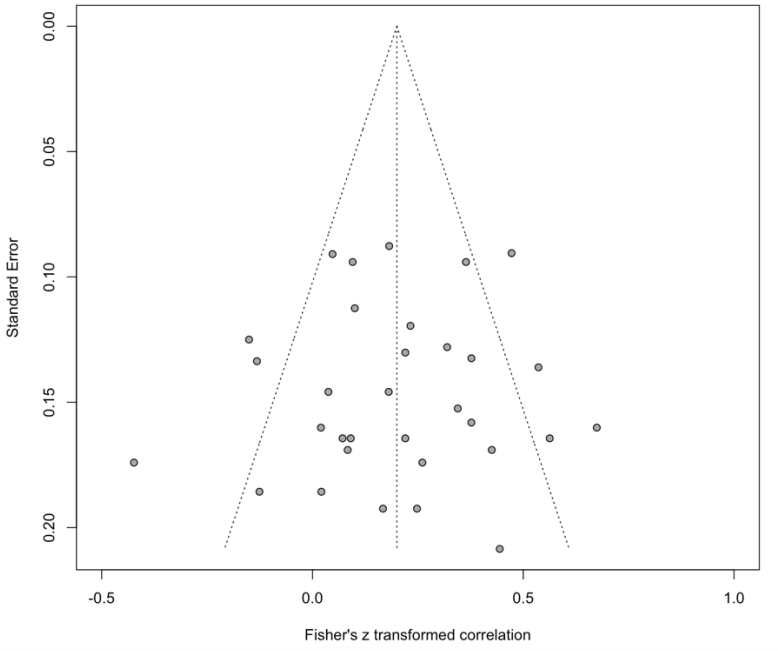
A.6 Results of the subgroup-meta-analysis for the effect of restraint on EI, influenced by questionnaire type

Questionnaire	**k**	***r***	**95% CI**	**I^2**
TFEQ	22	-0.16	-0.23, -0.10	16.9%
RS	10	0.09	-0.06, 0.23	44.6%
RRS	4	-0.12	-0.33, 0.12	0.0%
DEBQ	14	0.01	-0.09, 0.11	21.7%
EDE-Q	2	-0.21	-0.50, 0.13	0.0%

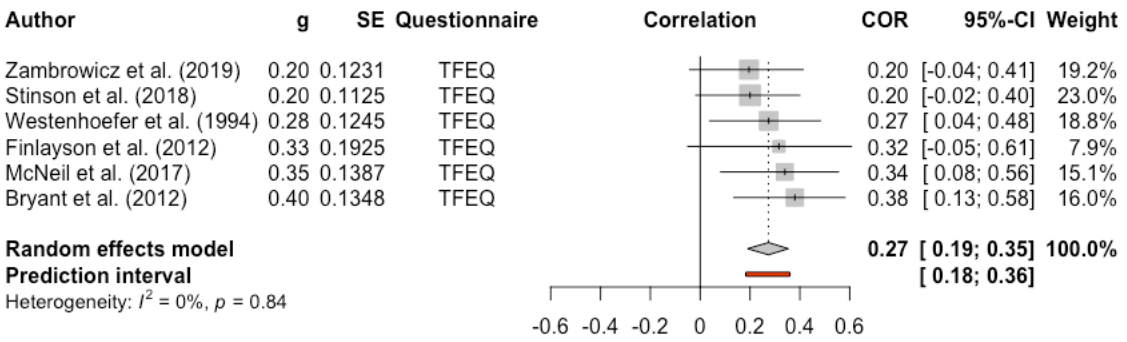
A.7 Results of a subgroup meta-analysis on the effects of restraint on EI influenced by a preload

Preload?	**k**	***r***	**95% CI**	**I^2**
No preload	45	-0.09	-0.15, -0.03	41.0%
Preload	10	0.04	-0.09, 0.16	0.0%

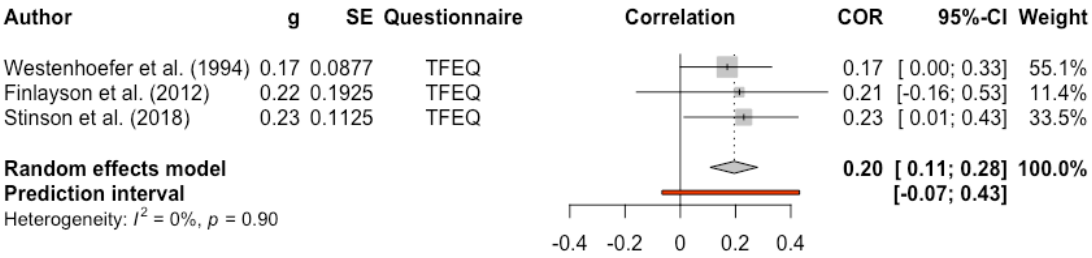
A.8 Funnel plot of the effect of restraint on BMI



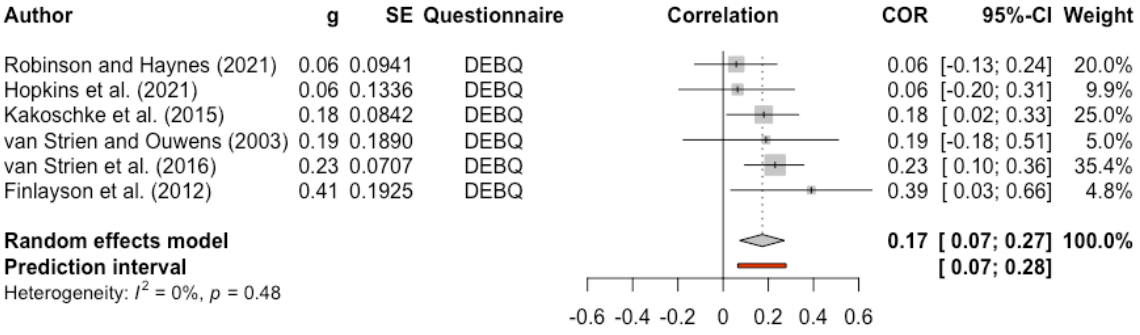
A.9 Forest plot of the effect of susceptibility to hunger on EI



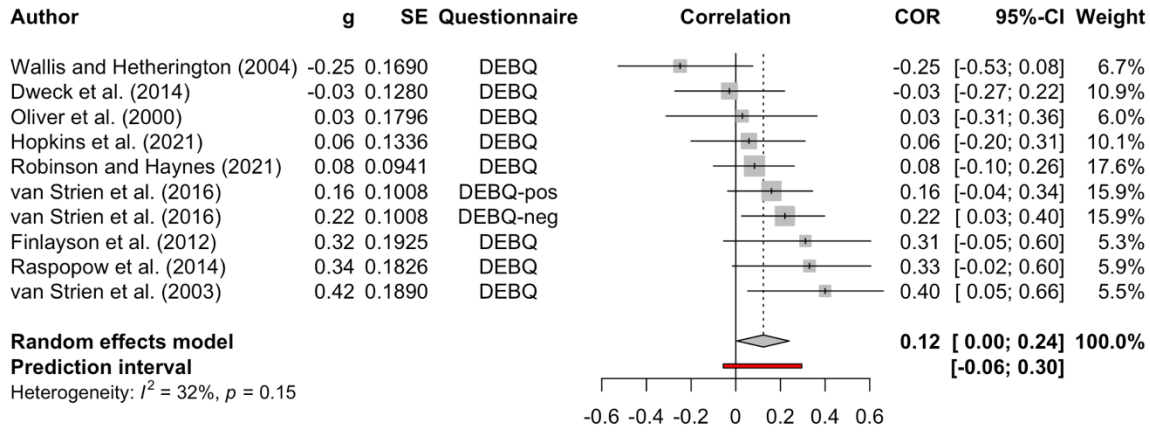
A.10 Forest plot of the effect of susceptibility to hunger on BMI



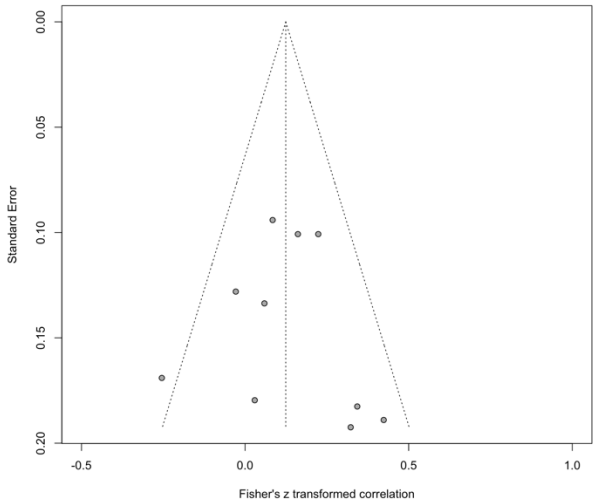
A.11 Forest plot of the effect of external eating on EI



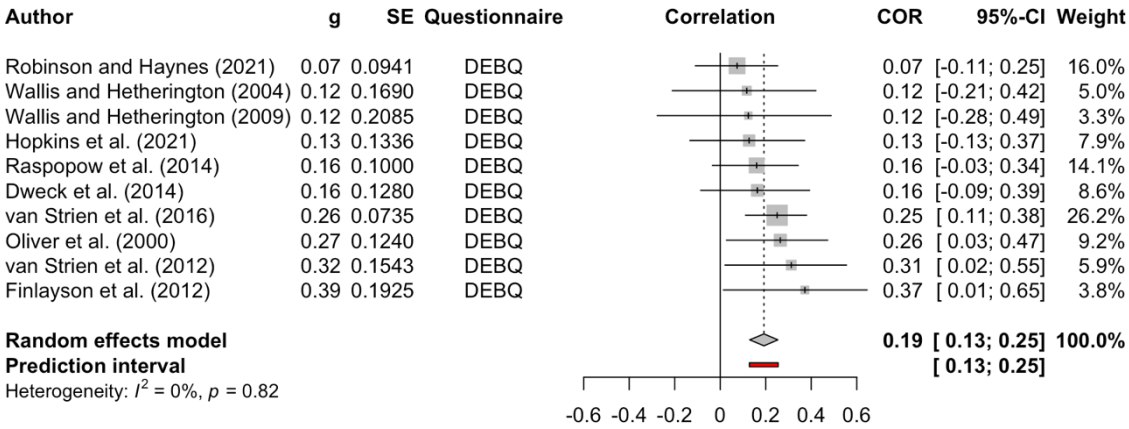
A.12 Forest plot of the effect of emotional eating on EI



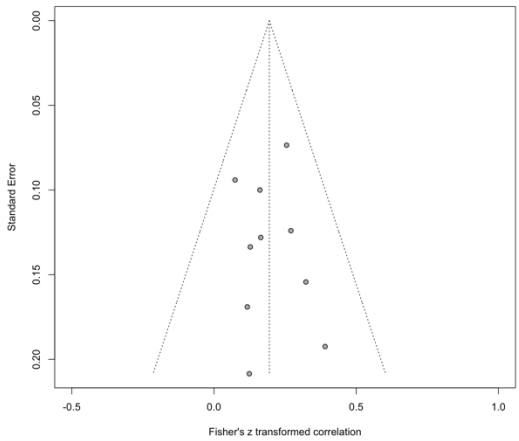
A.13 Funnel plot of the effect of emotional eating on EI



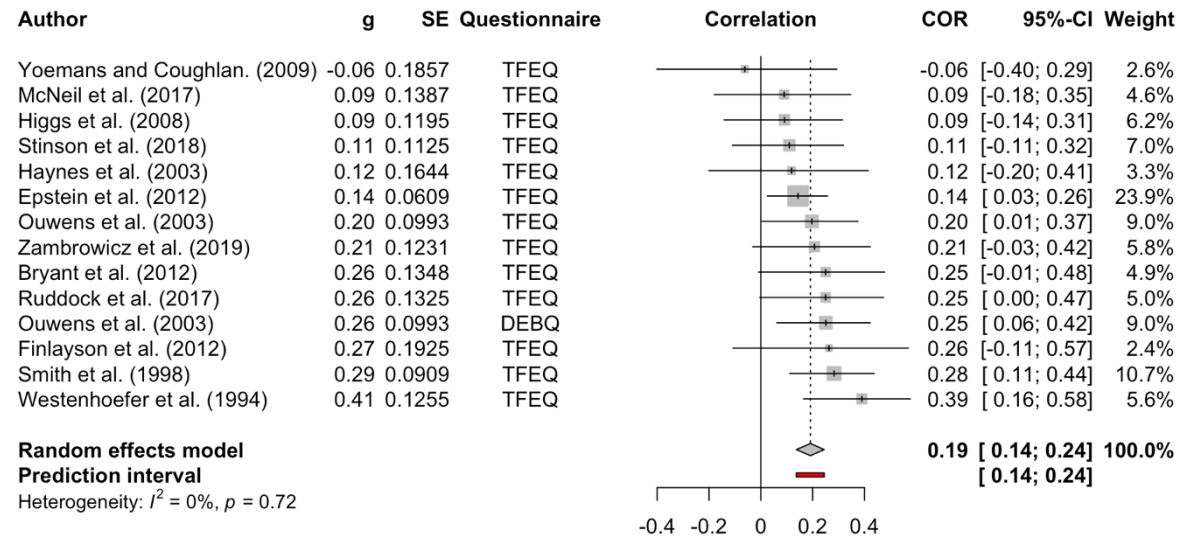
A.14 Forest plot of the effect of emotional eating on BMI



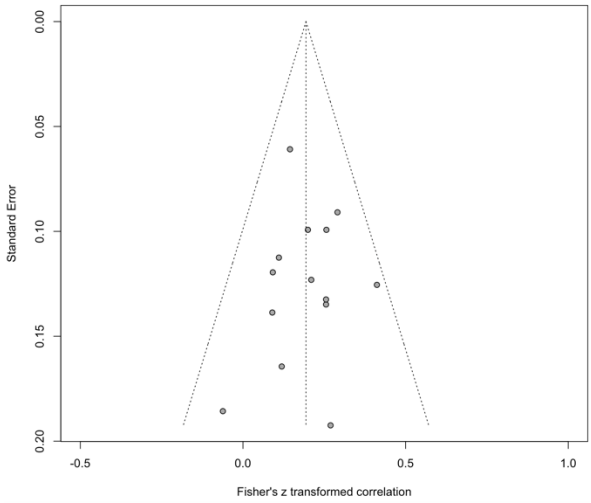
A.15 Funnel plot of the effect of emotional eating on BMI



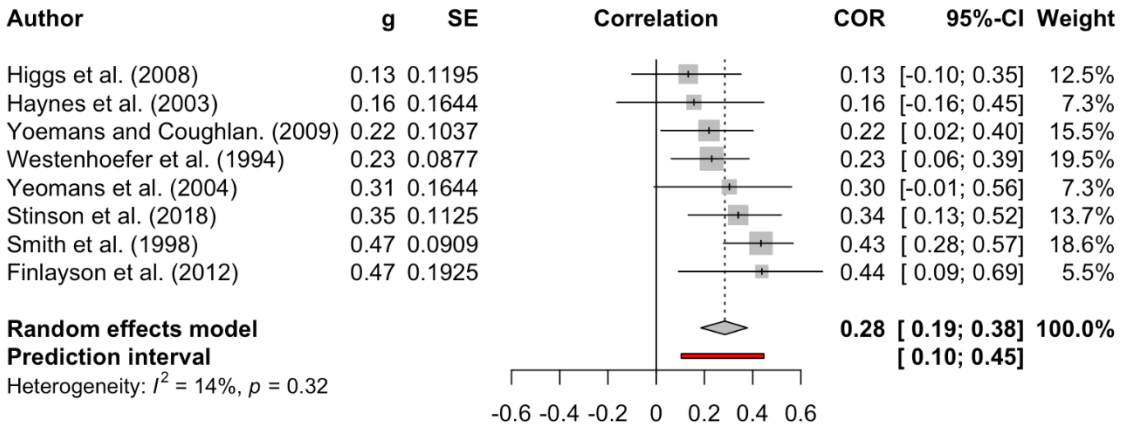
A.16 Forest plot of the effect of disinhibition on EI



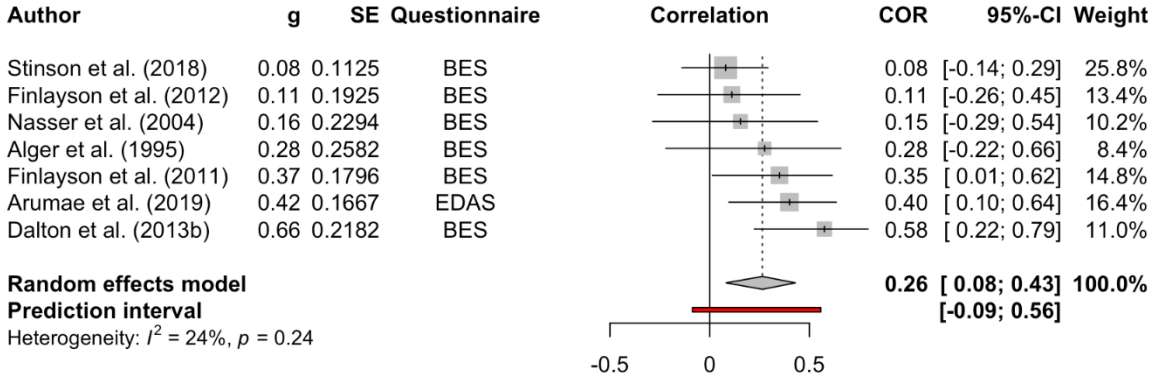
A.17 Funnel plot of the effect of disinhibition on EI



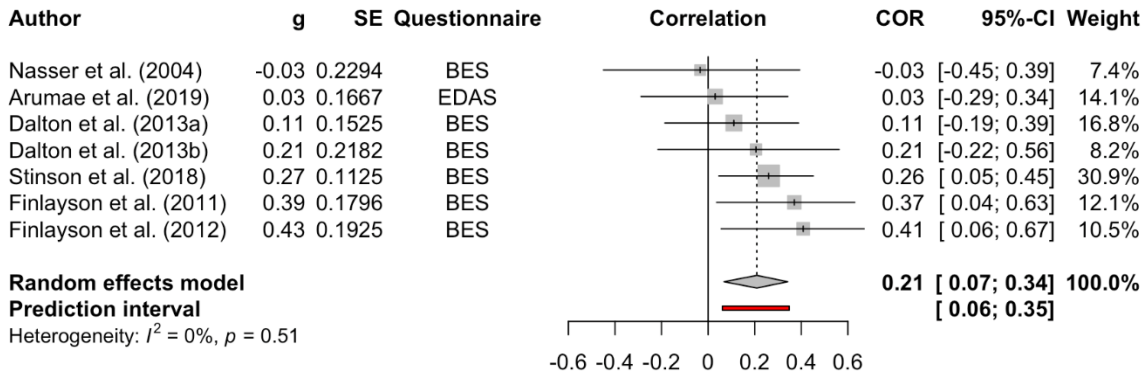
A.18 Forest plot of the effect of disinhibition on BMI



A.19 Forest plot of the effect of binge eating on EI



A.20 Forest plot of the effect of binge eating on BMI



A.21 References for supplementary materials (Chapter 3)

1. Alger S, Seagle H, Ravussin E. Food-intake and Energy Expenditure in Obese Female Bingers and Non-bingers. *INTERNATIONAL JOURNAL OF OBESITY*. 1995;19(1)
2. Gormally J, Black S, Daston S, Rardin D. The assessment of binge eating severity among obese persons. *Addictive behaviors*. 1982;7(1):47-55.
3. Anderson DA, Schaumberg K, Anderson LM, Reilly EE. Is level of intuitive eating associated with plate size effects? *Eating behaviors*. 2015;18:125-130.
4. Tylka TL. Development and psychometric evaluation of a measure of intuitive eating. *Journal of Counseling Psychology*. 2006;53(2):226.
5. Anderson LM, Reilly EE, Schaumberg K, Dmochowski S, Anderson DA. Contributions of mindful eating, intuitive eating, and restraint to BMI, disordered eating, and meal consumption in college students. *Eating and Weight Disorders*. 2016;21(1)doi:http://dx.doi.org/10.1007/s40519-015-0210-3
6. Framson C, Kristal AR, Schenk JM, Littman AJ, Zeliadt S, Benitez D. Development and validation of the mindful eating questionnaire. *Journal of the American dietetic Association*. 2009;109(8):1439-1444.
7. Stunkard AJ, Messick S. The three-factor eating questionnaire to measure dietary restraint, disinhibition and hunger. *Journal of psychosomatic research*. 1985;29(1):71-83.
8. Stice E, Telch CF, Rizvi SL. Development and validation of the Eating Disorder Diagnostic Scale: a brief self-report measure of anorexia, bulimia, and binge-eating disorder. *Psychological assessment*. 2000;12(2):123.
9. Appelhans BM, Liebman R, Woolf K, Pagoto SL, Schneider KL, Whited MC. Inhibiting food reward: Delay discounting, food reward sensitivity, and palatable food intake in overweight and obese women. *Obesity*. 2011;19(11)doi:http://dx.doi.org/10.1038/oby.2011.57
10. Lowe MR, Butryn ML, Didie ER, et al. The Power of Food Scale. A new measure of the psychological influence of the food environment. *Appetite*. 2009;53(1)doi:https://dx.doi.org/10.1016/j.appet.2009.05.016
11. Ard JD, Desmond RA, Allison DB, Conway JM. Dietary restraint and disinhibition do not affect accuracy of 24-hour recall in a multiethnic population.

JOURNAL OF THE AMERICAN DIETETIC ASSOCIATION. 2006;106(3)doi:10.1016/j.jada.2005.12.006

12. Arumae K, Kreegipuu K, Vainik U. Assessing the Overlap Between Three Measures of Food Reward. *FRONTIERS IN PSYCHOLOGY*. 2019;10doi:10.3389/fpsyg.2019.00883

13. Akkermann K, Herik M, Aluoja A, Järv A. Constructing an assessment scale for eating disorders. *Tartu: Department of Psychology of University of Tartu*. 2010;

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

15. Bellisle F, Dalix AM. Cognitive restraint can be offset by distraction, leading to increased meal intake in women. *American Journal of Clinical Nutrition*. 2001;74(2)doi:http://dx.doi.org/10.1093/ajcn/74.2.197

16. van Strien T, Frijters JE, Bergers GP, Defares PB. The Dutch Eating Behavior Questionnaire (DEBQ) for assessment of restrained, emotional, and external eating behavior. *International journal of eating disorders*. 1986;5(2):295-315.

17. Bryant EJ, Caudwell P, Blundell JE, Hopkins ME, King NA. Psycho-markers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*. 2012;58(1)doi:http://dx.doi.org/10.1016/j.appet.2011.09.006

18. Chambers L, Yeomans MR. Individual differences in satiety response to carbohydrate and fat. Predictions from the Three Factor Eating Questionnaire (TFEQ). *Appetite*. 2011;56(2)doi:https://dx.doi.org/10.1016/j.appet.2011.01.003

19. Coelho JS, Polivy J, Herman CP, Pliner P. Wake up and smell the cookies. Effects of olfactory food-cue exposure in restrained and unrestrained eaters. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.10.008

20. Herman CP, Mack D. Restrained and unrestrained eating. *Journal of personality*. 1975;

21. Cools J, Schotte DE, McNally RJ. Emotional arousal and overeating in restrained eaters. *Journal of abnormal psychology*. 1992;101(2):348.

22. Herman CP, Polivy J, Silver R. Effects of an observer on eating behavior: the induction of "sensible" eating. *Journal of personality*. 1979;47(1)

23. Dalton M, Blundell J, Finlayson G. Effect of BMI and binge eating on food reward and energy intake: Further evidence for a binge eating subtype of obesity. *Obesity Facts*. 2013;6(4)doi:http://dx.doi.org/10.1159/000354599
24. Dalton M, Blundell J, Finlayson GS. Examination of food reward and energy intake under laboratory and free-living conditions in a trait binge eating subtype of obesity. *Frontiers in Psychology*. 2013;4doi:http://dx.doi.org/10.3389/fpsyg.2013.00757
25. Hill AJ, Weaver CF, Blundell JE. Food craving, dietary restraint and mood. *Appetite*. 1991;17(3):187-197.
26. Greenway FL, Fujioka K, Plodkowski RA, et al. Effect of naltrexone plus bupropion on weight loss in overweight and obese adults (COR-I): a multicentre, randomised, double-blind, placebo-controlled, phase 3 trial. *The Lancet*. 2010;376(9741):595-605.
27. Dalton M, Finlayson G, Blundell J, Hill A. Preliminary validation and principal components analysis of the Control of Eating Questionnaire (CoEQ) for the experience of food craving. *European Journal of Clinical Nutrition*. 2015;69(12)doi:http://dx.doi.org/10.1038/ejcn.2015.57
28. Dalton M, Hollingworth S, Blundell J, Finlayson G. Weak Satiety Responsiveness Is a Reliable Trait Associated with Hedonic Risk Factors for Overeating among Women. *NUTRIENTS*. 2015;7(9)doi:10.3390/nu7095345
29. Drapeau V, King N, Hetherington M, Doucet E, Blundell J, Tremblay A. Appetite sensations and satiety quotient: predictors of energy intake and weight loss. *Appetite*. 2007;48(2):159-166.
30. de Witt Huberts JC, Evers C, de Ridder DT. Double trouble: restrained eaters do not eat less and feel worse. *Psychology & Health*. 2013;28(6):686-700.
31. Drapeau V, Blundell J, Therrien F, Lawton C, Richard D, Tremblay A. Appetite sensations as a marker of overall intake. *BRITISH JOURNAL OF NUTRITION*. 2005;93(2)doi:10.1079/BJN20041312
32. Green S, Delargy H, Joanes D, Blundell J. A satiety quotient: a formulation to assess the satiating effect of food. *Appetite*. 1997;29(3):291-304.
33. Drapeau V, Jacob R, Panahi S, Tremblay A. Effect of energy restriction on eating behavior traits and psychobehavioral factors in the low satiety phenotype. *Nutrients*. 2019;11(2)doi:http://dx.doi.org/10.3390/nu11020245

34. Dweck JS, Jenkins SM, Nolan LJ. The role of emotional eating and stress in the influence of short sleep on food consumption. *Appetite*. 2014;72doi:http://dx.doi.org/10.1016/j.appet.2013.10.001
35. Ely AV, Howard J, Lowe MR. Delayed discounting and hedonic hunger in the prediction of lab-based eating behavior. *Eating behaviors*. 2015;19doi:https://dx.doi.org/10.1016/j.eatbeh.2015.06.015
36. Epstein LH, Lin H, Carr KA, Fletcher KD. Food reinforcement and obesity. Psychological moderators. *APPETITE*. 2012;58(1)doi:10.1016/j.appet.2011.09.025
37. Evers C, de Ridder DTD, Adriaanse MA. Assessing yourself as an emotional eater: mission impossible? *Health psychology : official journal of the Division of Health Psychology, American Psychological Association*. 2009;28(6)doi:https://dx.doi.org/10.1037/a0016700
38. Fedoroff ID, Polivy J, Herman CP. The effect of pre-exposure to food cues on the eating behavior of restrained and unrestrained eaters. *Appetite*. 1997;28(1):33-47.
39. Finlayson G, Arlotti A, Dalton M, King N, Blundell JE. Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. *APPETITE*. 2011;57(3)doi:10.1016/j.appet.2011.08.012
40. Finlayson G, Blundell JE, Bordes I, Griffioen-Roose S, de Graaf C. Susceptibility to overeating affects the impact of savory or sweet drinks on satiation, reward, and food intake in nonobese women. *Journal of Nutrition*. 2012;142(1)doi:http://dx.doi.org/10.3945/jn.111.148106
41. Guerrieri R, Nederkoorn C, Schrooten M, Martijn C, Jansen A. Inducing impulsivity leads high and low restrained eaters into overeating, whereas current dieters stick to their diet. *Appetite*. 2009;53(1)doi:https://dx.doi.org/10.1016/j.appet.2009.05.013
42. Haynes C, Lee MD, Yeomans MR. Interactive effects of stress, dietary restraint, and disinhibition on appetite. *Eating behaviors*. 2003;4(4):369-383.
43. Herhaus B, Petrowski K. The effect of restrained eating on acute stress-induced food intake in people with obesity. *Appetite*. 2021;159doi:https://dx.doi.org/10.1016/j.appet.2020.105045
44. Higgs S, Williamson AC, Attwood AS. Recall of recent lunch and its effect on subsequent snack intake. *Physiology & behavior*. 2008;94(3):454-462.

45. Hofmann W, Friese M. Impulses got the better of me: alcohol moderates the influence of implicit attitudes toward food cues on eating behavior. *Journal of abnormal psychology*. 2008;117(2)doi:https://dx.doi.org/10.1037/0021-843X.117.2.420
46. Hopkins M, Michalowska J, Whybrow S, Horgan GW, Stubbs RJ. Identification of psychological correlates of dietary misreporting under laboratory and free-living environments. *British Journal of Nutrition*. 2021;126(2)doi:http://dx.doi.org/10.1017/S000711452000389X
47. Jansen A. How restrained eaters perceive the amount they eat. *The British journal of clinical psychology*. 1996;35(3)
48. Jansen A, Merckelbach H, Oosterlaan J, Tuiten A, Van Den Hout M. Cognitions and self-talk during food intake of restrained and unrestrained eaters. *Behaviour Research and Therapy*. 1988;26(5):393-398.
49. Kakoschke N, Kemps E, Tiggemann M. External eating mediates the relationship between impulsivity and unhealthy food intake. *Physiology & Behavior*. 2015;147doi:http://dx.doi.org/10.1016/j.physbeh.2015.04.030
50. Lattimore P, Maxwell L. Cognitive load, stress, and disinhibited eating. *Eating Behaviors*. 2004;5(4):315-324.
51. Long S, Meyer C, Leung N, Wallis DJ. Effects of distraction and focused attention on actual and perceived food intake in females with non-clinical eating psychopathology. *Appetite*. 2011;56(2)doi:https://dx.doi.org/10.1016/j.appet.2010.12.018
52. Garner DM. Eating Disorder Inventory-2; Pro-fessional Manual. *Psychological assessment resources*. 1991;
53. Martin CK, O'Neil PM, Tollefson G, Greenway FL, White MA. The association between food cravings and consumption of specific foods in a laboratory taste test. *Appetite*. 2008;51(2):324-326.
54. White MA, Whisenhunt BL, Williamson DA, Greenway FL, Netemeyer RG. Development and validation of the food-craving inventory. *Obesity Research*. 2002;10(2):107-114.
55. Martin CK, Williamson DA, Geiselman PJ, et al. Consistency of food intake over four eating sessions in the laboratory. *Eating Behaviors*. 2005;6(4):365-372.
56. McNeil J, Lamothe G, Cameron JD, et al. Investigating predictors of eating: is resting metabolic rate really the strongest proxy of energy intake? *The American*

journal of clinical nutrition.
2017;106(5)doi:https://dx.doi.org/10.3945/ajcn.117.153718
57. Myhre R, Buchwald D, Kratz M, et al. A twin study of differences in the response of plasma ghrelin to a milkshake preload in restrained eaters. *Physiology and Behavior*. 2014;129doi:http://dx.doi.org/10.1016/j.physbeh.2014.02.008
58. Nasser JA, Gluck ME, Geliebter A. Impulsivity and test meal intake in obese binge eating women. *Appetite*. 2004;43(3)
59. Nolan-Poupart S, Veldhuizen MG, Geha P, Small DM. Midbrain response to milkshake correlates with ad libitum milkshake intake in the absence of hunger. *Appetite*. 2013;60(1)doi:https://dx.doi.org/10.1016/j.appet.2012.09.032
60. Oliver G, Wardle J, Gibson EL. Stress and food choice: a laboratory study. *Psychosomatic medicine*. 2000;62(6):853-865.
61. Ouwens MA, van Strien T, van der Staak CP. Tendency toward overeating and restraint as predictors of food consumption. *Appetite*. 2003;40(3):291-298.
62. Peluso A. Comparison of mindful and slow eating strategies on acute energy intake. *Dissertation Abstracts International: Section B: The Sciences and Engineering*. 2017;78(5-B(E))
63. Raspopow K, Abizaid A, Matheson K, Anisman H. Anticipation of a psychosocial stressor differentially influences ghrelin, cortisol and food intake among emotional and non-emotional eaters. *Appetite*. 2014;74:35-43.
64. Rideout CA, McLean JA, Barr SI. Women with high scores for cognitive dietary restraint choose foods lower in fat and energy. *Journal of the American Dietetic Association*. 2004;104(7):1154-1157.
65. Robinson E, Haynes A. Individual differences and moderating participant characteristics in the effect of reducing portion size on meal energy intake: Pooled analysis of three randomized controlled trials. *Appetite*.
2021;159doi:http://dx.doi.org/10.1016/j.appet.2020.105047
66. Rolls BJ, Castellanos VH, Shide DJ, et al. Sensory properties of a nonabsorbable fat substitute did not affect regulation of energy intake. *The American journal of clinical nutrition*. 1997;65(5):1375-1383.
67. Ruddock HK, Field M, Hardman CA. Exploring food reward and calorie intake in self-perceived food addicts. *Appetite*.
2017;115doi:http://dx.doi.org/10.1016/j.appet.2016.12.003

68. Ruzanska UA, Warschburger P. How is intuitive eating related to self-reported and laboratory food intake in middle-aged adults? *EATING BEHAVIORS*. 2020;38doi:10.1016/j.eatbeh.2020.101405
69. Ruzanska UA, Warschburger P. Psychometric evaluation of the German version of the Intuitive Eating Scale-2 in a community sample. *Appetite*. 2017;117:126-134.
70. Schoch AH, Raynor HA. Social desirability, not dietary restraint, is related to accuracy of reported dietary intake of a laboratory meal in females during a 24-hour recall. *Eating behaviors*. 2012;13(1):78-81.
71. Schotte DE, Cools J, McNally RJ. Film-induced negative affect triggers overeating in restrained eaters. *Journal of Abnormal Psychology*. 1990;99(3):317.
72. Schulte EM, Sonneville KR, Gearhardt AN. Subjective experiences of highly processed food consumption in individuals with food addiction. *Psychology of addictive behaviors : journal of the Society of Psychologists in Addictive Behaviors*. 2019;33(2)doi:https://dx.doi.org/10.1037/adb0000441
73. Gearhardt AN, Corbin WR, Brownell KD. Development of the Yale Food Addiction Scale Version 2.0. *Psychology of Addictive Behaviors*. 2016;30(1):113.
74. Shapiro JR, Anderson DA. Counterregulatory eating behavior in multiple item test meals. *Eating behaviors*. 2005;6(2)
75. Sim AY, Lee LL, Cheon BK. When exercise does not pay: Counterproductive effects of impending exercise on energy intake among restrained eaters. *Appetite*. 2018;123doi:https://dx.doi.org/10.1016/j.appet.2017.12.017
76. Smith CF, Geiselman PJ, Williamson DA, Champagne CM, Bray GA, Ryan DH. Association of dietary restraint and disinhibition with eating behavior, body mass, and hunger. *Eating and weight disorders : EWD*. 1998;3(1)
77. Stice E, Fisher M, Lowe MR. Are dietary restraint scales valid measures of acute dietary restriction? Unobtrusive observational data suggest not. *Psychological assessment*. 2004;16(1):51.
78. Fairburn CG, Beglin SJ. Assessment of eating disorders: Interview or self-report questionnaire? *International journal of eating disorders*. 1994;16(4):363-370.
79. Stice E, Sysko R, Roberto CA, Allison S. Are dietary restraint scales valid measures of dietary restriction? Additional objective behavioral and biological data suggest not. *Appetite*. 2010;54(2):331-339.

80. Stinson EJ, Votruba SB, Venti C, Krakoff J, Gluck ME, Perez M. Food Insecurity is Associated with Maladaptive Eating Behaviors and Objectively Measured Overeating. *Obesity*. 2018;26(12)doi:http://dx.doi.org/10.1002/oby.22305
81. van Strien T, Donker MH, Ouwens MA. Is desire to eat in response to positive emotions an 'obese' eating style: Is Kummerspeck for some people a misnomer? *Appetite*. 2016;100doi:http://dx.doi.org/10.1016/j.appet.2016.02.035
82. Vainik U, Eun Han J, Epel ES, Janet Tomiyama A, Dagher A, Mason AE. Rapid Assessment of Reward-Related Eating: The RED-X5. *Obesity*. 2019;27(2):325-331.
83. van Strien T, Herman CP, Anschutz DJ, Engels RC, de Weerth C. Moderation of distress-induced eating by emotional eating scores. *Appetite*. 2012;58(1):277-284.
84. van Strien T, Ouwens MA. Counterregulation in female obese emotional eaters: Schachter, Goldman, and Gordon's (1968) test of psychosomatic theory revisited. *Eating Behaviors*. 2003;3(4)doi:http://dx.doi.org/10.1016/S1471-0153%2802%2900092-2
85. Vijayvargiya P, Chedid V, Wang XJ, et al. Associations of gastric volumes, ingestive behavior, calorie and volume intake, and fullness in obesity. *American journal of physiology Gastrointestinal and liver physiology*. 2020;319(2)doi:https://dx.doi.org/10.1152/ajpgi.00140.2020
86. Clark MM, Abrams DB, Niaura RS, Eaton CA, Rossi JS. Self-efficacy in weight management. *Journal of consulting and clinical psychology*. 1991;59(5):739.
87. Visona C, George VA. Impact of dieting status and dietary restraint on postexercise energy intake in overweight women. *OBESITY RESEARCH*. 2002;10(12)doi:10.1038/oby.2002.170
88. Wallis DJ, Hetherington MM. Stress and eating: the effects of ego-threat and cognitive demand on food intake in restrained and emotional eaters. *Appetite*. 2004;43(1):39-
89. Wallis DJ, Hetherington MM. Emotions and eating. Self-reported and experimentally induced changes in food intake under stress. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.11.007
90. Wardle J, Beales S. Restraint and food intake: An experimental study of eating patterns in the laboratory and in normal life. *Behaviour Research and Therapy*. 1987;25(3)doi:http://dx.doi.org/10.1016/0005-7967%2887%2990044-1

91. Westenhoefer J, Broeckmann P, Münch A-K, Pudel V. Cognitive control of eating behavior and the disinhibition effect. *Appetite*. 1994;23(1):27-41.
92. Yeomans MR, Coughlan E. Mood-induced eating. Interactive effects of restraint and tendency to overeat. *Appetite*. 2009;52(2)doi:https://dx.doi.org/10.1016/j.appet.2008.10.006
93. Yeomans MR, Tovey H, Tinley E, Haynes C. Effects of manipulated palatability on appetite depend on restraint and disinhibition scores from the Three-Factor Eating Questionnaire. *International journal of obesity*. 2004;28(1):144-151.
94. Zambrowicz R, Schebendach J, Sysko R, Mayer LES, Walsh BT, Steinglass JE. Relationship between three factor eating questionnaire-restraint subscale and food intake. *International Journal of Eating Disorders*. 2019;52(3)doi:http://dx.doi.org/10.1002/eat.23014
95. Fairburn CG, Cooper Z, O'Connor M. The eating disorder examination. *International Journal of Eating Disorders*. 1993;6:1-8.
96. Zuraikat FM, Roe LS, Smethers AD, Reihart LW, Rolls BJ. Does the cost of a meal influence the portion size effect? *Appetite*. 2018;127:341-348.
97. Hunot C, Fildes A, Croker H, Llewellyn CH, Wardle J, Beeken RJ. Appetitive traits and relationships with BMI in adults: Development of the Adult Eating Behaviour Questionnaire. *Appetite*. 2016;105:356-363.

Appendix B Supplementary Materials for Chapter Four

B.1 Ethical approval for Chapter Four

Title of study: NoHoW: Evidence-based tools for weight loss maintenance

Ethics reference: 16-0275

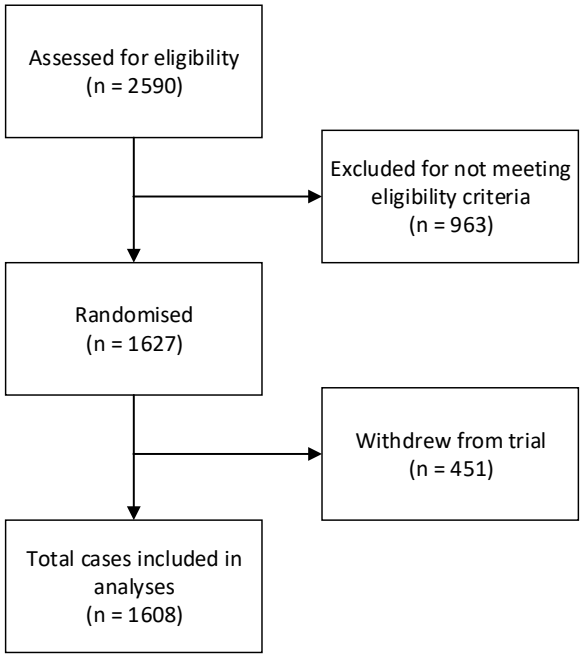
I am pleased to inform you that the above research application has been reviewed by the IPS Research Ethics Committee and has been approved.
Please note that this approval only relates to the particular version of documentation supplied in this specific application (ref no: 16-0275; date approved: 27-Oct-2016). If you wish to make any amendments to the approved documentation, please note that all changes require ethical approval prior to implementation.

Please note: You are expected to keep a record of all your approved documentation, as well as documents such as sample consent forms, and other documents relating to the study. This should be kept in your study file, which should be readily available for audit purposes.
You will be given a two week notice period if your project is to be audited. There is a checklist listing examples of documents to be kept which is available at https://eur03.safelinks.protection.outlook.com/?url=http%3A%2F%2Fris.leeds.ac.uk%2FEthicsAudits&data=05%7C02%7Cps18cad%40leeds.ac.uk%7Ce21122ac855f40fa9c7b08dc69dc1238%7Cbdeaeda8c81d45ce863e5232a535b7cb%7C0%7C0%7C638501641111430237%7CUnknown%7CTWFpbGZsb3d8eyJWIjoiMC4wLjAwMDAiLCJQIjoiV2luMzIiLCJBTiI6Ik1haWwiLCJXVCI6Mn0%3D%7C0%7C%7C%7C&sdata=iXZ%2FwtCYTkZGod6uNa%2FvaNdiPF21676jh6vvsNkFCPQ%3D&reserved=0 <https://eur03.safelinks.protection.outlook.com/?url=http%3A%2F%2Fris.leeds.ac.uk%2FEthicsAudits&data=05%7C02%7Cps18cad%40leeds.ac.uk%7Ce21122ac855f40fa9c7b08dc69dc1238%7Cbdeaeda8c81d45ce863e5232a535b7cb%7C0%7C0%7C638501641111435023%7CUnknown%7CTWFpbGZsb3d8eyJWIjoiMC4wLjAwMDAiLCJQIjoiV2luMzIiLCJBTiI6Ik1haWwiLCJXVCI6Mn0%3D%7C0%7C%7C%7C&sdata=cNhrXtNTJxlDvuRovtdgiizygeLyIHEgHdrW72DP6RQ%3D&reserved=0>

Yours sincerely,

IPS Research Ethics Committee
(Chair: Donna Lloyd)

B.2 Participant flow diagram



Note: Missing values were imputed where possible using multiple imputation as part of an intention-to-treat analysis

B.3 Descriptives table displaying age, BMI, and baseline EBT variables for each study arm condition

Study Arm		N	Mean	SD
Control	Age of respondent	400	43.98	12.07
	cid1_BMI	400	29.76	5.68
	cid1_TFEQ_D	398	8.73	3.34
	cid1_TFEQ_R	398	10.92	3.42
	cid1_TFEQ_H	398	5.71	3.47
	cid1_BES	398	12.07	7.27
	cid1_IES_RHSC	398	2.82	.84
	cid1_IES_EPR	398	3.01	.59
	cid1_IES_UPTE	398	3.02	.61
Stress/Emotion	Age of respondent	403	43.60	11.28
	cid1_BMI	403	29.56	5.20
	cid1_TFEQ_D	399	8.98	3.40
	cid1_TFEQ_R	399	11.03	3.52
	cid1_TFEQ_H	399	6.16	3.42
	cid1_BES	399	12.56	7.76
	cid1_IES_RHSC	399	2.83	.91
	cid1_IES_EPR	399	3.00	.58
	cid1_IES_UPTE	399	3.05	.62
Self-regulation/emotion	Age of respondent	416	43.90	12.30
	cid1_BMI	416	29.35	4.99
	cid1_TFEQ_D	411	8.96	3.61
	cid1_TFEQ_R	411	11.18	3.32
	cid1_TFEQ_H	411	5.99	3.53
	cid1_BES	411	12.15	7.55
	cid1_IES_RHSC	411	2.84	.88
	cid1_IES_EPR	411	2.97	.60
	cid1_IES_UPTE	411	3.07	.62
Stress/emotion, self-regulation	Age of respondent	408	44.58	11.78
and motivation	cid1_BMI	408	29.96	5.49
	cid1_TFEQ_D	403	8.39	3.29

cid1_TFEQ_R	403	11.12	3.47
cid1_TFEQ_H	403	5.44	3.42
cid1_BES	403	11.66	7.30
cid1_IES_RHSC	403	2.86	.85
cid1_IES_EPR	403	2.99	.57
cid1_IES_UPTE	403	3.04	.56

B.4. Disinhibition estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

Weight Category	Time (Disinhibition)	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Weight Loss	1	8.48[a]	.22	8.05	8.90
	2	7.74[a]	.22	7.31	8.18
	3	7.67[a]	.23	7.23	8.12
	4	7.70[a]	.23	7.26	8.15
Weight Maintenance	1	8.49[a]	.15	8.20	8.79
	2	8.19[a]	.15	7.90	8.49
	3	8.23[a]	.16	7.92	8.54
	4	8.16[a]	.16	7.86	8.47
Weight Re-gain	1	8.63[a]	.15	8.34	8.93
	2	8.56[a]	.15	8.26	8.85
	3	8.74[a]	.16	8.44	9.05
	4	8.75[a]	.16	8.44	9.05

a. Covariates appearing in the model are evaluated at the following values: Age of respondent = 44.06, Prior Weight Loss = 11.67, cid1_BMI = 29.62.

B.5. Hunger estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

Weight Category	Time (Hunger)	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Weight Loss	1	5.69[a]	.23	5.23	6.14
	2	5.03[a]	.23	4.57	5.49
	3	4.98[a]	.23	4.52	5.43
	4	5.04[a]	.24	4.57	5.51
Weight Maintenance	1	5.74[a]	.16	5.43	6.06
	2	5.61[a]	.16	5.29	5.93
	3	5.63[a]	.16	5.32	5.95
	4	5.80[a]	.17	5.47	6.12
Weight Re-gain	1	5.86[a]	.16	5.54	6.17
	2	5.70[a]	.16	5.38	6.02
	3	6.03[a]	.16	5.71	6.34
	4	6.14[a]	.17	5.82	6.47

a. Covariates appearing in the model are evaluated at the following values: Age of respondent = 44.06, BMI_cid1 = 29.62, Prior Weight Loss = 11.67.

B.6 Binge eating estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

Weight Category	Time (Binge Eating)	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Weight Loss	1	11.20[a]	.46	10.30	12.11
	2	9.81[a]	.50	8.83	10.79
	3	9.09[a]	.51	8.09	10.08
	4	8.74[a]	.52	7.72	9.75
Weight Maintenance	1	11.35[a]	.32	10.72	11.97
	2	10.94[a]	.35	10.26	11.62
	3	11.10[a]	.35	10.41	11.79

	4	10.99[a]	.36	10.29	11.69
Weight Re-gain	1	11.73[a]	.32	11.11	12.36
	2	12.06[a]	.34	11.39	12.74
	3	12.52[a]	.35	11.84	13.21
	4	13.01[a]	.36	12.31	13.71

a. Covariates appearing in the model are evaluated at the following values: BMI_cid1 = 29.62, Prior Weight Loss = 11.67, Age of respondent = 44.06.

B.7 Restraint estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

Weight Category	Time (Restraint)	Mean	Std. Error	95% Confidence Interval	
				Lower Bound	Upper Bound
Weight Loss	1	10.99[a]	.23	10.55	11.43
	2	11.75[a]	.22	11.31	12.19
	3	11.70[a]	.24	11.23	12.18
	4	11.76[a]	.24	11.30	12.22
Weight Maintenance	1	10.87[a]	.16	10.56	11.17
	2	10.74[a]	.16	10.43	11.04
	3	10.73[a]	.17	10.40	11.06
	4	10.43[a]	.16	10.10	10.75
Weight Re-gain	1	10.70[a]	.16	10.39	11.00
	2	10.60[a]	.15	10.29	10.90
	3	10.30[a]	.17	9.98	10.63
	4	10.05[a]	.16	9.73	10.37

a. Covariates appearing in the model are evaluated at the following values: Age of respondent = 44.06, cid1_BMI = 29.62, Prior Weight Loss = 11.67.

B.8 Reliance on hunger and satiety cues estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

				95% Confidence Interval	
Weight Category	Time (RHSC)	Mean	Std. Error	Lower Bound	Upper Bound
Weight Loss	1	2.75[a]	.06	2.64	2.86
	2	3.00[a]	.06	2.88	3.11
	3	3.10[a]	.06	2.98	3.22
	4	3.11[a]	.06	2.99	3.23
Weight Maintenance	1	2.90[a]	.04	2.82	2.98
	2	3.04[a]	.04	2.96	3.12
	3	3.10[a]	.04	3.02	3.18
	4	3.03[a]	.04	2.95	3.11
Weight Re-gain	1	2.85[a]	.04	2.78	2.93
	2	2.96[a]	.04	2.88	3.03
	3	2.99[a]	.04	2.91	3.06
	4	2.87[a]	.04	2.79	2.96

a. Covariates appearing in the model are evaluated at the following values: BMI_cid1 = 29.62, Prior Weight Loss = 11.67, Age of respondent = 44.06.

B.9 Eating for physical reasons estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

				95% Confidence Interval	
Weight Category	Time (EPR)	Mean	Std. Error	Lower Bound	Upper Bound
Weight Loss	1	3.06[a]	.04	2.98	3.13
	2	2.97[a]	.04	2.89	3.04
	3	2.92[a]	.04	2.84	2.99
	4	2.92[a]	.04	2.84	3.00
Weight Maintenance	1	2.95[a]	.03	2.90	3.00
	2	2.88[a]	.03	2.83	2.94
	3	2.84[a]	.03	2.79	2.89
	4	2.87[a]	.03	2.82	2.93

Weight Re-gain	1	2.99[a]	.03	2.94	3.05
	2	2.87[a]	.03	2.82	2.93
	3	2.88[a]	.03	2.82	2.93
	4	2.88[a]	.03	2.83	2.94

a. Covariates appearing in the model are evaluated at the following values: BMI cid1 = 29.62, Prior Weight Loss = 11.67, Age of respondent = 44.06.

B.10 Unlimited permission to eat estimated marginal means with 95% confidence intervals and effects of the weight category x time interaction from adjusted linear models

				95% Confidence Interval	
Weight Category	Time (UPTE)	Mean	Std. Error	Lower Bound	Upper Bound
Weight Loss	1	3.04[a]	.04	2.96	3.11
	2	3.04[a]	.04	2.96	3.12
	3	3.01[a]	.04	2.92	3.09
	4	3.00[a]	.04	2.91	3.09
Weight Maintenance	1	3.06[a]	.03	3.01	3.12
	2	3.12[a]	.03	3.07	3.18
	3	3.14[a]	.03	3.08	3.20
	4	3.13[a]	.03	3.07	3.20
Weight Re-gain	1	3.06[a]	.03	3.00	3.11
	2	3.15[a]	.03	3.10	3.21
	3	3.18[a]	.03	3.12	3.24
	4	3.24[a]	.03	3.18	3.30

a. Covariates appearing in the model are evaluated at the following values: BMI_cid1 = 29.62, Prior Weight Loss = 11.67, Age of respondent = 44.06.

Appendix C Supplementary Materials for Chapter Five

C.1 Ethical approval for Chapter Five

Dear Alscient Developer

Re your ethics application, Perceived satiety in a sample of foods from different food groups in the UK diet., ethics reference number: PSYC-338.

I am pleased to inform you that the above research application has been reviewed by the School of Psychology Research Ethics Committee and the decision is Approved.

If the reviewers have left any comments they will appear below.
Primary reviewer comments (if applicable) :
Secondary reviewer comments (if applicable) :

Please note that this approval only relates to the particular version of documentation supplied in this specific application (ethics ref no: PSYC-338).

If you wish to make any amendments to the approved documentation, please note that all changes require ethical approval prior to implementation.

Yours sincerely,

School of Psychology Research Ethics Committee

Dear Graham Finlayson,

Re your ethics application, Perceived satiety in a sample of foods from different food groups in the UK diet., ethics reference number: PSC-904.
I am pleased to inform you that the above research application has been reviewed by the School of Psychology Research Ethics Committee and has been approved.

If the reviewers have left any comments they will appear below.

Primary reviewer comments (if applicable) : This seems fine

Secondary reviewer comments (if applicable) :

Please note that this approval only relates to the particular version of documentation supplied in this specific application (ethics ref no: PSC-904).

If you wish to make any amendments to the approved documentation, please note that all changes require ethical approval prior to implementation.

Please note: You are expected to keep a record of all your approved documentation, as well as documents such as sample consent forms, and other documents relating to the study. This should be kept in your study file, which should be readily available for audit purposes.

You will be given a two week notice period if your project is to be audited. There is a checklist listing examples of documents to be kept which is available at http://ris.leeds.ac.uk/EthicsAudits.

Yours sincerely,

School of Psychology Research Ethics Committee

Appendix D Supplementary Materials for Chapter Six

D.1 Ethical approval for Chapter Six

Dear AS_PAFlow_Service_Account_Ethics

Re your ethics application, Perceived satiety in a sample of foods from different food groups in the UK diet., ethics reference number: PSCETHS-707.

I am pleased to inform you that the above research application has been reviewed by the School of Psychology Research Ethics Committee and the desision is Approved.

If the reviewers have left any comments they will appear below.

Primary reviewer comments (if applicable) : all good - thanks for sorting all my previous requests. addition of new measures fine too

Secondary reviewer comments (if applicable) :

Please note that this approval only relates to the particular version of documentation supplied in this specific application (ethics ref no: PSCETHS-707).

If you wish to make any amendments to the approved documentation, please note that all changes require ethical approval prior to implementation.

Please note: You are expected to keep a record of all your approved documentation, as well as documents such as sample consent forms, and other documents relating to the study. This should be kept in your study file, which should be readily available for audit purposes.

You will be given a two week notice period if your project is to be audited. There is a checklist listing examples of documents to be kept which is available at http://ris.leeds.ac.uk/EthicsAudits.

Yours sincerely,
School of Psychology Research Ethics Committee

D.2 Scoring of TFEQ subscales

Recoding of items:

Items 1-36

1, 2, 3 , 4, 5, 6, 7, 8, 9, 11, 12, 13, 14, 15, 17, 18, 19, 20, 22, 23, 24, 26, 27, 28, 29, 32, 33, 34, 35, 36

True =1

False = 0

Items 10, 16, 21, 25, 30, 31,

True =0

False =1

Items 37-51 - 37, 38, 39, 40, 41, 42, 43, 44, 45, 46, 48, 49, 51

Scale 1,2,3,4 recoded to 1=0, 2=0, 3=1, 4=1

47

Scale 1,2,3,4 recoded to 1=1, 2=1, 3=0, 4=0

50

Scale 0,1,2,3,4, recoded to 0=0, 1=0, 2=0, 3=1, 4=1, 5=1

Scoring (r =recoded value):

Disinhibition= 1r + 2r + 7r + 9r + 11r + 13r + 15r + 16r +
20r + 25r + 27r + 31r + 36r + 45r + 49r + 51r.

Susceptibility to Hunger 3r + 5r + 8r + 12r + 17r + 19r + 22r +
24r + 26r + 29r + 34r + 39r + 41r + 47r.

Dietary Restraint == 4r + 6r + 10r + 14r + 18r + 21r + 23r + 28r +
30r + 32r + 33r + 35r + 37r + 38r + 40r + 42r + 43r + 44r + 46r +
48r + 50r.

D.3 Questionnaires and items including in the study, with information relating to if they were included in the analysis, a reason if no and a proposed factor loading for the theoretical model

Questionnaire	Item	Item Included	Reason (if no)	Factor loading theoretical model
The Eating Motivation Survey (TEMS)	I eat what I eat because I enjoy it (TEMS_1).	Yes		All items measure eating for pleasure
	I eat what I eat in order to indulge myself (TEMS_2)	Yes		
	I eat what I eat because it puts me in a good mood (TEMS_3).	Yes		
	I eat what I eat in order to reward myself (TEMS_4).	Yes		
	I eat what I eat because it is fun to eat (TEMS_5).	Yes		
Positive Emotional Eating (PNEES_P)	When I am feeling active and motivated I have a tendency to eat (PNEESP_1).	Yes		All items measure positive emotional eating
	I have a desire to eat when I am joyful (PNEESP_2).	Yes		
	I have a desire to eat when I am full of energy (PNEESP_3).	Yes		
	I feel like eating when I am content with myself (PNEESP_4).	Yes		
	I tend to eat when I get excited about something (PNEESP_5).	Yes		
	Feeling confident drives me to eat (PNEESP_6).	Yes		
	Being fascinated about something drives me to eat (PNEESP_7).	Yes		
Emotional Overeating (AEBQ_EOE)	I eat more when I'm annoyed (EOE_1).	Yes		All items measure negative emotional eating

	154- I eat more when I'm worried (EOE_2).	Yes		
	155 - I eat more when I'm upset (EOE_3).	Yes		
	156 - I eat more when I'm anxious (EOE_4).	Yes		
	157 - I eat more when I'm angry (EOE_5).	Yes		
Body-Food Choice Congruence (BFCC)	Most of the time, I desire to eat nutritious foods (BFCC_31)	Yes		All items measure homeostatic eating
	I mostly eat foods that make my body perform efficiently (well) (BFCC_32).	Yes		
	I mostly eat foods that give my body energy and stamina (BFCC_33).	Yes		
Unlimited Permission to Eat (UPE)	*I try to avoid certain foods high in fat, carbohydrates, or calories (UPE_1)	Yes		All items measure restricted eating
	If I am craving a certain food, I allow myself to have it (UPE_4).	Yes		
	*I get mad at myself for eating something unhealthy (UPE_9).	No	Measures an emotional response to eating not a motivation to eat/not eat.	
	*I have forbidden foods that I don't allow myself to eat (UPE_14)	Yes		
	I allow myself to eat what food I desire at the moment (UPE_28)	Yes		
	I do NOT follow eating rules or dieting plans that dictate what, when, and/or how much to eat (UPE_29)	Yes		
Eating for Physical Rather than Emotional	I find myself eating when I'm feeling emotional (e.g., anxious, depressed, sad), even when I'm not physically hungry (EFPR_3).	Yes		All items measure negative emotional eating
	I find myself eating when I am lonely, even when I'm not physically hungry (EFPR_10)	Yes		

Reasons (EFPR)			
	I use food to help me soothe my negative emotions (EFPR_16)	Yes	
	I find myself eating when I am stressed out, even when I'm not physically hungry (EFPR_17).	Yes	
	I am able to cope with my negative emotions (e.g., anxiety, sadness) without turning to food for comfort (EFPR_23).	Yes	
	When I am bored, I do NOT eat just for something to do (EFPR_24).	Yes	
	When I am lonely, I do NOT turn to food for comfort (EFPR_25).	Yes	
	I find other ways to cope with stress and anxiety than by eating (EFPR_26).	Yes	
Reliance on Hunger and Satiety Cues (RHSC)	I trust my body to tell me when to eat (RHSC_11).	Yes	All items measure homeostatic eating
	I trust my body to tell me what to eat (RHSC_12).	Yes	
	I trust my body to tell me how much to eat (RHSC_13).	Yes	
	349 - I rely on my hunger signals to tell me when to eat (RHSC_35).	Yes	
	I rely on my fullness (satiety) signals to tell me when to stop eating (RHSC_36).	Yes	
	I trust my body to tell me when to stop eating (RHSC_37).	Yes	
Disinhibition (TFEQD)	When I smell a sizzling steak or see a juicy piece of meat, I find it very difficult to keep from eating, even if I have just finished a meal (TFEQD_1).	Yes	Items either measure reactive or negative emotional eating Reactive

I usually eat too much at social occasions, like parties and picnics (TFEQD_2).	Yes		
Sometimes things just taste so good that I keep on eating even when I am no longer hungry (TFEQD_7).	Yes		Reactive
When I feel anxious, I find myself eating (TFEQD_9).	Yes		Emotional
Since my weight goes up and down, I have gone on reducing diets more than once (TFEQD_11).	No	Does not measure a motivation to eat/not eat	
When I am with someone who is overeating, I usually overeat too (TFEQD_13).	Yes		Reactive
Sometimes when I start eating, I just can't seem to stop (TFEQD_15).	Yes		Reactive
It is not difficult for me to leave something on my plate (TFEQD_16).	Yes		Reactive
When I feel blue, I often overeat (TFEQD_20).	Yes		Emotional
My weight has hardly changed at all in the last ten years (TFEQD_25).	No	Does not measure a motivation to eat/not eat	
When I feel lonely, I console myself by eating (TFEQD_27).	Yes		Emotional
Without even thinking about it, I take a long time to eat (TFEQD_31).	No	Slowness in eating is not measure a motivation to eat/not eat	
While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods (TFEQD_36).	Yes		Reactive

	Do you eat sensibly in front of others and splurge alone (TFEQD_45)?	Yes		Reactive
	Do you go on eating binges though you are not hungry (TFEQD_49)?	Yes		Reactive
	To what extent does this statement describe your eating behaviour? 'I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow (TFEQD_51).	Yes		Reactive
Susceptibility to Hunger (TFEQH)	I am usually so hungry that I eat more than three times a day (TFEQH_3).	Yes		All items either measure reactive eating or do not measure an EBT Reactive
	Dieting is so hard for me because I just get too hungry (TFEQH_5).	Yes		Reactive
	Since I am often hungry, I sometimes wish that while I am eating, an expert would tell me that I have had enough or that I can have something more to eat (TFEQH_8).	No	Presupposes someone has a problem with their appetite. The trait is wishing someone else would tell them what to do.	
	I often feel so hungry that I just have to eat something (TFEQH_12).	Yes		Reactive
	At certain times of the day, I get hungry because I have gotten used to eating then (TFEQH_17).	No	Measures temporal conditioning	
	Being with someone who is eating often makes me hungry enough to eat also (TFEQH_19).	Yes		Reactive

	When I see a real delicacy, I often get so hungry that I have to eat right away (TFEQH_22).	Yes		Reactive
	I get so hungry that my stomach often seems like a bottomless pit (TFEQH_24).	No	Measures the sensation of hunger not motivation to eat	
	I am always hungry so it is hard for me to stop eating before I finish the food on my plate (TFEQH_26).	Yes		Reactive
	I sometimes get very hungry late in the evening or at night (TFEQH_29).	No	Measures sensation of hunger	
	I am always hungry enough to eat at any time (TFEQH_34).	Yes		Reactive
	How often do you feel hungry (TFEQH_39)?	No	Measures sensation of hunger	
	How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours (TFEQH_41)?	Yes		Reactive
	How frequently do you skip dessert because you are no longer hungry (TFEQH_47)?	Yes		Reactive
Restraint (TFEQR)	When I have eaten my quota of calories, I am usually good about not eating any more (TFEQR_4)?	Yes		All items either measure restricted eating or do not measure an EBT Restricted
	I deliberately take small helpings as a means of controlling my weight (TFEQR_6).	Yes		Restricted
	Life is too short to worry about dieting (TFEQR_10).	No	Measures an eating philosophy	

I have a pretty good idea of the number of calories in common food (TFEQR_14).	No	Not an eating behaviour	
While on a diet, if I eat food that is not allowed, I consciously eat less for a period of time to make up for it (TFEQR_18).	Yes		Restricted
I enjoy eating too much to spoil it by counting calories or watching my weight (TFEQR_21).	No	Measures reward sensitivity	
I often stop eating when I am not really full as a conscious means of limiting the amount that I eat (TFEQR_23).	Yes		Restricted
I consciously hold back at meals in order not to gain weight (TFEQR_28).	Yes		Restricted
I eat anything I want, any time I want (TFEQR_30).	Yes		Restricted
I count calories as a conscious means of controlling my weight (TFEQR_32).	Yes		Restricted
I do not eat some foods because they make me fat (TFEQR_33).	Yes		Restricted
I pay a great deal of attention to changes in my figure (TFEQR_35).	No	Not an eating behaviour	
How often are you dieting in a conscious effort to control your weight (TFEQR_37)?	Yes		Restricted
Would a weight fluctuation of 5 lbs (2.3 kg) affect the way you live your life (TFEQR_38)?	No	Not an eating behaviour	
Do your feelings of guilt about overeating help you to control your food intake (TFEQR_40)?	No	Measures use of negative emotions to undereat) self-criticism as a means of self-control.	

How conscious are you of what you are eating (TFEQR_42)?	No	Measures conscious awareness of eating behaviour not necessarily restriction	
How frequently do you avoid ‘stocking up’ on tempting foods (TFEQR_43)?	No	Measures changing your food environment. One step removed from the eating behaviour itself.	
How likely are you to shop for low calorie foods (TFEQR_44)?	No	Not an eating behaviour	
How likely are you to consciously eat slowly in order to cut down on how much YOU eat (TFEQR_46)?	Yes		Restricted
How likely are you to consciously eat less than you want (TFEQR_48)?	Yes		Restricted
On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never ‘giving in’), what number would you give yourself (TFEQR_50)?	Yes		Restricted

D.4 Unadjusted ANOVA models

Pleasure

Tests of Between-Subjects Effects						
Dependent Variable: PLEASURE						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	46.163[a]	1	46.163	45.966	<.001	.011
Intercept	68952.645	1	68952.645	68658.790	<.001	.941
SampleType	46.163	1	46.163	45.966	<.001	.011
Error	4311.374	4293	1.004			
Total	73417.155	4295				
Corrected Total	4357.536	4294				
a. R Squared = .011 (Adjusted R Squared = .010)						

Positive Emotional Eating

Tests of Between-Subjects Effects						
Dependent Variable: PNEES_P						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	41.899[a]	1	41.899	175.026	<.001	.039
Intercept	12883.325	1	12883.325	53817.728	<.001	.926
SampleType	41.899	1	41.899	175.026	<.001	.039
Error	1027.454	4292	.239			
Total	13913.943	4294				
Corrected Total	1069.353	4293				
a. R Squared = .039 (Adjusted R Squared = .039)						

Reliance on hunger and satiety cues

Tests of Between-Subjects Effects						
Dependent Variable: IES_RHSC						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	348.206[a]	1	348.206	552.306	<.001	.114
Intercept	40009.118	1	40009.118	63460.265	<.001	.937
SampleType	348.206	1	348.206	552.306	<.001	.114
Error	2706.562	4293	.630			
Total	42732.444	4295				
Corrected Total	3054.768	4294				
a. R Squared = .114 (Adjusted R Squared = .114)						

Eating for physical reasons

Tests of Between-Subjects Effects						
Dependent Variable: IES_EPR						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	301.991[a]	1	301.991	342.557	<.001	.074
Intercept	37847.303	1	37847.303	42931.234	<.001	.909
SampleType	301.991	1	301.991	342.557	<.001	.074
Error	3784.621	4293	.882			
Total	41642.222	4295				
Corrected Total	4086.613	4294				
a. R Squared = .074 (Adjusted R Squared = .074)						

Unlimited permission to eat

Tests of Between-Subjects Effects						
Dependent Variable: IES_UPE						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	602.532[a]	1	602.532	1529.102	<.001	.263
Intercept	39622.179	1	39622.179	100552.932	<.001	.959
SampleType	602.532	1	602.532	1529.102	<.001	.263
Error	1691.627	4293	.394			
Total	41415.154	4295				
Corrected Total	2294.158	4294				
a. R Squared = .263 (Adjusted R Squared = .262)						

Susceptibility to hunger

Tests of Between-Subjects Effects						
Dependent Variable: TFEQ_H						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	348.863[a]	1	348.863	24.558	<.001	.006
Intercept	128610.208	1	128610.208	9053.598	<.001	.678
SampleType	348.863	1	348.863	24.558	<.001	.006
Error	60983.890	4293	14.205			
Total	189650.000	4295				
Corrected Total	61332.753	4294				
a. R Squared = .006 (Adjusted R Squared = .005)						

Disinhibition

Tests of Between-Subjects Effects						
Dependent Variable: TFEQ_D						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	4890.294[a]	1	4890.294	310.714	<.001	.067
Intercept	269457.806	1	269457.806	17120.517	<.001	.800
SampleType	4890.294	1	4890.294	310.714	<.001	.067
Error	67567.024	4293	15.739			
Total	348699.000	4295				
Corrected Total	72457.318	4294				
a. R Squared = .067 (Adjusted R Squared = .067)						

Restraint

Tests of Between-Subjects Effects						
Dependent Variable: TFEQ_R						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	8850.988[a]	1	8850.988	629.284	<.001	.128
Intercept	483540.299	1	483540.299	34378.559	<.001	.889
SampleType	8850.988	1	8850.988	629.284	<.001	.128
Error	60381.777	4293	14.065			
Total	564988.000	4295				
Corrected Total	69232.765	4294				
a. R Squared = .128 (Adjusted R Squared = .128)						

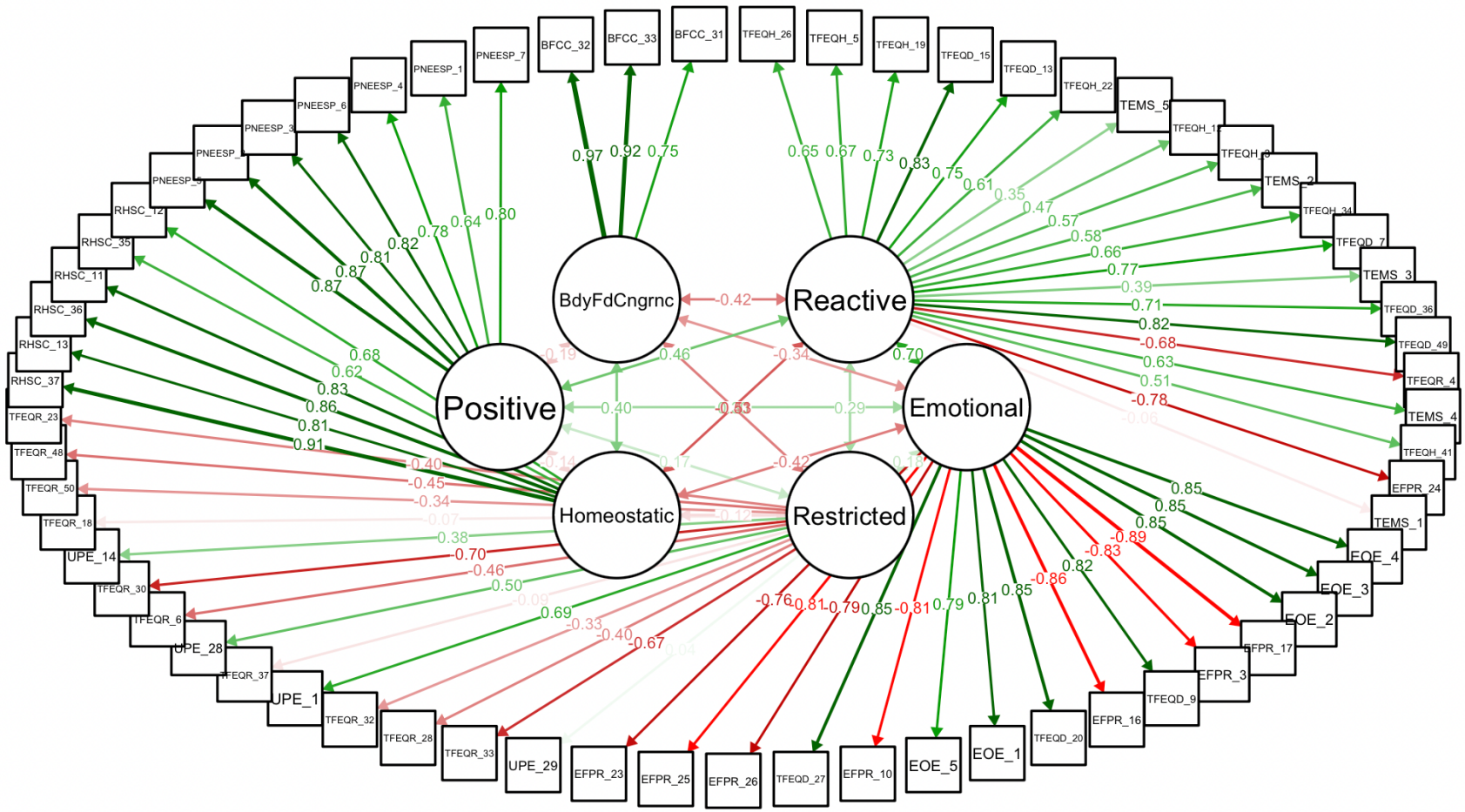
Emotional Overeating

Tests of Between-Subjects Effects						
Dependent Variable: AEBQ_EOE						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	422.033[a]	1	422.033	394.905	<.001	.084
Intercept	35024.828	1	35024.828	32773.501	<.001	.884
SampleType	422.033	1	422.033	394.905	<.001	.084
Error	4587.901	4293	1.069			
Total	40787.378	4295				
Corrected Total	5009.934	4294				
a. R Squared = .084 (Adjusted R Squared = .084)						

Body-food choice congruence

Tests of Between-Subjects Effects						
Dependent Variable: IES_BFCC						
Source	Type III Sum of Squares	df	Mean Square	F	Sig.	Partial Eta Squared
Corrected Model	100.625[a]	1	100.625	155.654	<.001	.035
Intercept	53939.523	1	53939.523	83437.424	<.001	.951
SampleType	100.625	1	100.625	155.654	<.001	.035
Error	2775.282	4293	.646			
Total	57445.333	4295				
Corrected Total	2875.907	4294				
a. R Squared = .035 (Adjusted R Squared = .035)						

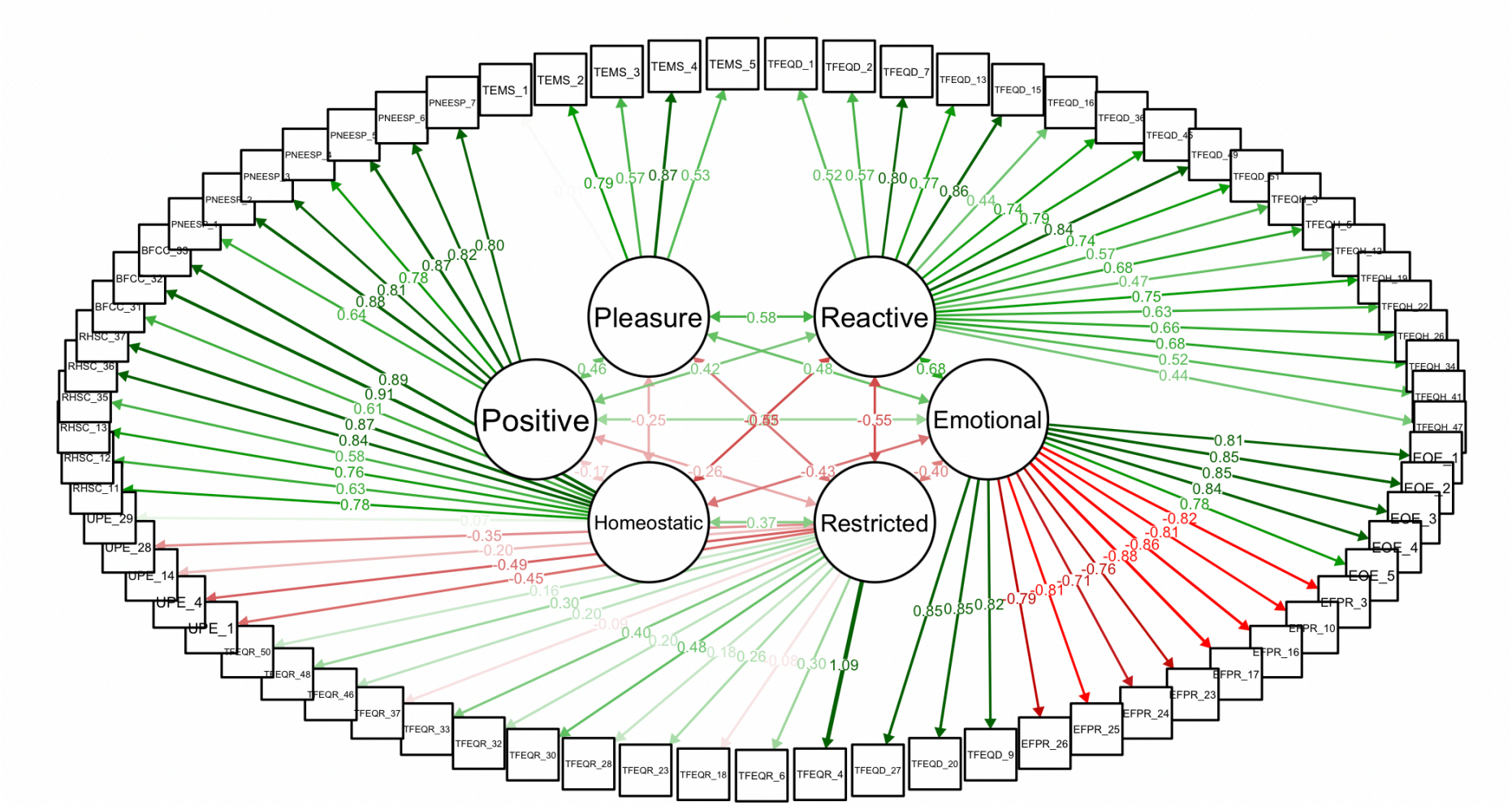
D.5 CFA data-driven model in the weight management sample



Note.

Green lines represent positive loadings, red lines represent negative loadings. Outer statistics denote factor loadings. Inner statistics denote covariance between factors.

D.6 CFA theory-driven model in the weight management sample



Note. Green lines represent positive loadings, red lines represent negative loadings. Outer statistics denote factor loadings. Inner statistics denote covariance between factors.

Appendix E Supplementary Materials for Chapter Seven

E.1. 24 Item Questionnaire to measure 6 domains of EBTs

1. PFS_3 = When I know a delicious food is available, I can't help myself from thinking about having some (reactive).
2. PFS_7 = It seems like I have food on my mind a lot (reactive).
3. TFEQD_15 = "Sometimes when I start eating, I just can't seem to stop" (reactive).
4. PFS_9 = "If I see or smell a food I like, I get a powerful urge to have some." (reactive).
5. PNEES_2 = I have a tendency to eat when I am upset (negative emotional overeating).
6. PNEES_7 = When I am feeling helpless I want to eat (negative emotional overeating).
7. PNEES_3 = I tend to eat when I am disappointed (negative emotional overeating).
8. PNEES_5 = Feeling tense or anxious drives me to eat (negative emotional overeating).
9. PNEESP_6 = Feeling confident drives me to eat. (positive emotional eating).
10. PNEESP_5 = I tend to eat when I get excited about something. (positive emotional eating).
11. PNEESP_3 = I have a desire to eat when I am full of energy. (positive emotional eating).
12. PNEESP_2 = I have a desire to eat when I am joyful (positive emotional eating).
13. RHSC_37 = I trust my body to tell me when to stop eating. (homeostatic eating).
14. RHSC_13 = I trust my body to tell me how much to eat. (homeostatic eating).
15. RHSC_36 = I rely on my fullness (satiety) signals to tell me to stop eating. (homeostatic eating).
16. RHSC_11 = I trust my body to tell me when to eat. (homeostatic eating).

17. TFEQR_28 = I consciously hold back at meals in order not to gain weight. (restricted eating)
18. TFEQR_23 = I often stop eating when I am not really full as a conscious means of limiting the amount that I eat. (restricted eating)
19. DEBQR_7 = Do you deliberately eat less in order not to become heavier? (restricted eating)
20. DEBQR_6 = When you have eaten too much, do you eat less than usual the following days? (restricted eating)
21. TEMS_3 = I eat what I eat because it puts me in a good mood (eating for pleasure).
22. TEMS_4 = I eat what I eat in order to reward myself (eating for pleasure).
23. TEMS_2 = I eat what I eat in order to indulge myself (eating for pleasure).
24. TEMS_5 = I eat what I eat because it is fun to eat (eating for pleasure).