

**Sweet Food Preferences and Associated Appetite Regulatory
Mechanisms**

Dominic James O'Connor

Submitted in accordance with the requirements for the degree of
Doctor of Philosophy

The University of Leeds
School of Psychology

July 2023

The candidate confirms that the work submitted is his own and that appropriate credit has been given where reference has been made to the work of others.

This copy has been supplied on the understanding that it is copyright material and that no quotation from the thesis may be published without proper acknowledgement.

The right Dominic O'Connor to be identified as Author of this work has been asserted by him in accordance with the Copyright, Designs and Patents Act 1988.

© 2022 The University of Leeds and Dominic James O'Connor

Acknowledgements

Thanks is owed to each of my supervisors, Professor Graham Finlayson, Associate Professor Catherine Gibbons, Dr. Mark Hopkins and Dr. Kristine Beaulieu. The passion and high standards brought to their work have shaped and developed me as a researcher as I endeavour to make my mark in the scientific community. I also thank my colleagues within the Human Appetite Research Unit, namely Dr. Beverley O'Hara, Dr. Nuno Martins, Dr Pauline Oustric and Fiona Croden, for their support and dedication during data collection periods.

This PhD was jointly funded by the School of Psychology, University of Leeds, and an EU-project under the acronym "SWEET" from the European Union's Horizon 2020 research and innovation programme under Grant agreement No 774293. I am grateful to colleagues in both the EU SWEET consortium and the School of Psychology at the University of Leeds for providing me with their collaboration and assistance.

I am grateful to my family and friends for their constant support and patience during trying times.

Contents

1	General Introduction and Background.....	2
1.1	The Prevalence and Causes of Obesity.....	2
1.2	The Food Environment	4
1.3	Energy Balance and Appetite Control	4
1.4	Food Reward.....	7
1.4.1	Components of Food Reward	7
1.4.2	Food Cravings	9
1.5	Sweet Food Preferences	10
1.5.1	Sweet Liker Phenotype	12
1.5.2	Body Weight Differences in Sweet Food Preference.....	14
1.5.3	Food Preferences and Physiological Mechanisms.....	16
1.5.4	Effects of Fasting on Sweet Food Preferences.....	17
1.6	The Neurobiology of Sweet Food Preferences	18
1.7	Types of Sweet Stimuli.....	20
1.7.1	Sugar.....	20
1.7.2	Sugar and Fat	22
1.7.3	High-Intensity Sweeteners.....	24
1.8	Thesis Aims	28
1.8.1	Specific Objectives.....	29
2	A systematic review on the differences between high intensity and caloric sweeteners on appetite, food reward and body weight.....	31
2.1	Introduction.....	31
2.1.1	High intensity sweeteners.....	31
2.1.2	Types of low-calorie intense sweeteners	32
2.1.3	The role of the sweet taste in obesity	33
2.1.4	Effects of HIS on appetite-related outcomes	34
2.1.5	Effects of HIS on food reward	34
2.1.6	Effects on body weight-related outcomes	35
2.1.7	Aims of the present review	36
2.2	Methods.....	37
2.2.1	Literature search	37
2.2.2	Inclusion criteria	39
2.2.3	Data extraction	40
2.2.4	Outcomes.....	40
2.2.5	Risk of bias	40
2.3	Results.....	41
2.3.1	Risk of Bias.....	41

2.3.2	Study and Participant Characteristics.....	42
2.3.3	Effects of HIS on Appetite	43
2.3.4	Effects of HIS on Food Reward	61
2.3.5	Effects of HIS on Body Weight	64
2.4	Discussion.....	68
2.4.1	Appetite.....	68
2.4.2	Food Reward.....	73
2.4.3	Body Weight and Composition	74
2.4.4	Limitations.....	76
2.4.5	Future Directions	77
2.4.6	Conclusions.....	79
3	General Methodology	81
3.1	Measures completed in both study one and study two	81
3.1.1	Assessing Appetite and Food Intake	81
3.1.2	Leeds Food Preference Questionnaire Assessment	83
3.1.3	Height, Weight, BMI and Body Composition	86
3.1.4	Sensory Visual Analogue Scales (VAS).....	87
3.1.5	Ethical Considerations	88
3.2	General Methodology: Study One, Diet Induced Variability in Appetite (DIVA) ...	88
3.2.1	Design	88
3.2.2	Participants	89
3.2.3	Procedure.....	91
3.2.4	Dietary Interventions	94
3.2.5	Measures.....	95
3.2.6	Ethical Approval.....	99
3.3	General Methodology: Study Two, Sweeteners & Sweetener Enhancers (SWEET)	100
3.3.1	Design	100
3.3.2	Participants	100
3.3.3	Procedure.....	102
3.3.4	Screening	102
3.3.5	Clinical Investigation Days	103
3.3.6	Substitution Strategy	104
3.3.7	Wash-out Period	105
3.3.8	Measures.....	105
3.3.9	Ethical Approval.....	108
3.3.10	Statistical Analysis	108
4	The stability of sweet food preference within one day and after diet-induced weight loss	109

4.1	Introduction.....	109
4.2	Method	113
4.2.1	Data Analysis	114
4.3	Results.....	115
4.3.1	Differences in Sweet Food Preferences in Fasted, Non-fasted and Fed states	116
4.3.2	Association between Sweet Food Preferences in different states within one day	123
4.3.3	The stability of sweet food preferences within one day	124
4.3.4	Differences in sweet food preference across a dietary weight loss intervention	125
4.3.5	Associations in sweet food preference across a dietary weight loss intervention	128
4.3.6	The stability of sweet food preference across a dietary weight loss intervention	129
4.4	Discussion.....	131
4.4.1	Differences in Food Preferences	131
4.4.2	The Stability of Sweet Food Preferences	133
4.4.3	Conclusion	134
5	The influence of sweet food preferences on eating behaviours in women across a range of BMIs	135
5.1	Introduction.....	135
5.2	Methods.....	139
5.2.1	Sweet Liker Phenotype Categorisation.....	140
5.2.2	Data Analysis	142
5.3	Results.....	144
5.3.1	Descriptive Statistics and Baseline Differences Between BMI Groups	144
5.3.2	Effects of Sweet Food Preferences on Rate of Weight Change	147
5.3.3	Differences in Eating Behaviour traits between Sweet Liker Phenotypes in response to Weight Loss	148
5.4	Discussion.....	155
5.4.1	Differences and Associations between Baseline Sweet Food Preferences and Eating Behaviour Traits	155
5.4.2	Differences in Eating Behaviour Traits between Sweet Liker Phenotypes in Individuals with Overweight and Obesity in Response to Weight Loss	159
5.4.3	Conclusions.....	161
6	A comparison of IER and CER pre- and post- 5% weight loss on sweet food preferences	163
6.1	Introduction.....	163
6.2	Methods.....	166
6.2.1	Data Analysis	168
6.3	Results.....	170

6.3.1	Sweet Food Preferences (LFPQ)	170
6.3.2	<i>Ab Libitum</i> Intake.....	171
6.3.3	Post-Meal Palatability Responses.....	171
6.4	Discussion.....	175
6.4.1	Differences Between Groups in Sweet Food Preferences During and Following Weight Loss	175
6.4.2	Differences in <i>Ad Libitum</i> Intake.....	177
6.4.3	Differences in Post-Meal Palatability Responses.....	177
6.4.4	Conclusions.....	178
7	The Effects of Acute and Repeated Ingestion of Sucrose vs. High-Intensity Sweeteners formulated in biscuits on Sweet Food Preferences and Related Eating Behaviours	179
7.1	Introduction.....	179
7.2	Methods.....	184
7.2.1	Design	185
7.2.2	Materials.....	185
7.2.3	Procedure.....	187
7.2.3.1	Data Analysis	189
7.3	Results.....	191
7.3.1	Participant Descriptive Statistics	191
7.3.2	Co-Primary Outcome.....	193
7.3.2.1	Explicit Liking Sweet Bias	196
7.3.2.2	Explicit Wanting Sweet Bias	197
7.3.2.3	Implicit Wanting Sweet Bias	197
7.3.2.4	Choice Sweet Bias	198
7.3.3	Secondary Outcomes	199
7.3.4	Exploratory Outcomes	208
7.4	Discussion.....	213
7.4.1	Sweet Food Preferences – High-Intensity Sweeteners versus Sugar.....	213
7.4.2	Food Cravings, Appetite for Sweet/Savoury and Subsequent Intake	215
7.4.3	Body Weight and Composition and 24hr Dietary Recall	217
7.4.4	Conclusions.....	218
8	An Exploration into Sweet Phenotype Differences in the Effects of Sucrose and High-Intensity Sweeteners on Subsequent Sweet Food Preferences	220
8.1	Introduction.....	220
8.2	Methods.....	223
8.2.1	Sweet liker phenotype	223
8.2.2	Data Analysis	223
8.3	Results.....	225
8.3.1	Co-Primary Outcomes	225

8.3.2	Secondary Outcomes	231
8.3.3	Body Weight and Composition	233
8.3.4	Diet Recall	236
8.4	Discussion.....	242
8.4.1	Sweet Food Preferences	242
8.4.2	Appetite Sensations and Food Cravings	244
8.4.3	Body Weight and Composition	245
8.4.4	Diet Recall	246
8.4.5	Conclusion	248
9	General Discussion.....	249
9.1	Overview of thesis	249
9.2	Systematic review of RCTs comparing HIS vs. sucrose and/or water controls on appetite, food reward and body weight	250
9.3	Are sweet food preferences stable?.....	251
9.4	Eating behaviour traits and sweet food preferences.....	254
9.5	Sweet liker phenotype.....	255
9.5.1	LFPQ use in the assessment of sweet liker phenotype	256
9.5.2	Dichotomisation of sweet liker phenotype groups	256
9.6	Expression of sweet food preferences	258
9.7	Effect of sweetener type and subsequent sweet food preferences	259
9.8	Issues Regarding Sweetener use in Processed Foods.....	261
9.8.1	NOVA classification system.....	263
9.9	Strengths and limitations of this thesis	264
9.9.1	Protocol Limitations in both studies	264
9.9.2	Protocol strengths in both studies	266
9.9.3	Limitations to Study 1: DIVA.....	267
9.9.4	Limitations to Study 2: SWEET	267
9.10	Concluding Remarks	268
	References.....	270

Table of Figures

Figure 1.1 The energy balance equation taken from (Schoeller, 2009).	5
Figure 1.2. Visualisation of appetite control systems taken from (Higgs et al., 2017).	6
Figure 1.3. Four distinguishable patterns of sweet liking in response to sucrose concentration (taken from Iatridi et al., 2019a, 2019b).	13
Figure 1.4. Schematic of thesis aims divided by chapter and study.....	29
Figure 2.1. Study selection flow chart.	41
Figure 2.2. The number of studies conducted in differing a) BMI groups (left) and b) age (right) groups.....	43
Figure 3.1. Representation of the a) Explicit Liking and b) Explicit Wanting trials in the Leeds Food Preference Questionnaire.	84
Figure 3.2. Representation of the Implicit Wanting trials in the Leeds Food Preference Questionnaire.	85
Figure 3.3. Frequency weighted algorithm used to score IW in the LFPQ.....	85
Figure 3.4. Instructions provided to participants prior Leeds Food Preference Questionnaire completion.....	86
Figure 3.5. Study 1 timeline of diet intervention and measures days for participants with overweight and obesity.	92
Figure 3.6. Study 1 measures day protocol.....	93
Figure 3.7. Image of standardised breakfast presentation.....	93
Figure 3.8. <i>Ad libitum</i> test meal presentation.....	94
Figure 3.9. MyFood24 meal selection and database search bar.....	98
Figure 3.10. MyFood24 example of a food search.	98
Figure 3.11. MyFood24 portion size selection.	98
Figure 3.12. Number of laboratory visits and time commitment per volunteer in the FAST study. Each visit is scheduled in the morning and lasts up to 4 hours.	102
Figure 3.13. Timeline of Clinical Investigation Days during the SWEET protocol.	104
Figure 4.1. Changes in Explicit Liking Sweet Bias across baseline.	117
Figure 4.2. Changes in Explicit Liking High Fat Sweet and Low Fat Sweet across Baseline.	118
Figure 4.3. Change in Explicit Wanting Sweet Bias within a single day.	119
Figure 4.4. Changes in Explicit Wanting High Fat Sweet and Low Fat Sweet within a single day.....	120
Figure 4.5. Change in Implicit Wanting Sweet Bias within a single day.	120
Figure 4.6. Changes in Implicit Wanting High Fat Sweet and Low Fat Sweet within a single day.....	121
Figure 4.7. Change in Choice Sweet Bias within a single day.	122
Figure 4.8. Changes in mean Choice High Fat Sweet and Low Fat Sweet within a single day.	123

Figure 4.9. Changes in Explicit Liking Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention.	126
Figure 4.10. Changes in EW Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention.	126
Figure 4.11. Changes in IW Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention.	127
Figure 4.12. Changes in Choice Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention.	128
Figure 5.1. Mean explicit liking sweet bias for high ($n=25$) and low ($n=11$) sweet likers at baseline ($t(34)=-6.796$, $p<.001$).	148
Figure 5.2. Differences in Three Factor Eating Questionnaire outcomes (A = Restraint, B = Disinhibition, C = Hunger) across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.	151
Figure 5.3. Differences in Binge Eating Scale scores across a dietary induced weight loss intervention in high ($n=24$) and low ($n=11$) sweet liker phenotype groups.	152
Figure 5.4. Differences in Control of Eating Questionnaire outcomes across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.	154
Figure 6.1. Study 1 timeline of diet intervention and measures days for participants with overweight and obesity.	166
Figure 6.2. Leeds Food Preference Questionnaire Sweet Bias scores by group across the weight-loss intervention.	171
Figure 6.3. Bar chart displaying post- <i>ad libitum</i> wanting more sweet VAS ratings across the intervention by group.	173
Figure 7.1. Number of laboratory visits and time commitment per volunteer in the FAST study. Each visit is scheduled in the morning and lasts up to 4 hours.	185
Figure 7.2. Timeline of a probe day during the SWEET trial.	188
Figure 7.3. Presentation of intervention products to participants at each probe day.	188
Figure 7.4. Participant recruitment flow diagram.	192
Figure 7.5. Changes in Leeds Food Preference Explicit Liking Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models.	196
Figure 7.6. Changes in Leeds Food Preference Explicit Wanting Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models.	197
Figure 7.7. Changes in Leeds Food Preference Implicit Wanting Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models.	198
Figure 7.8. Changes in Leeds Food Preference Choice Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models.	199
Figure 7.9. Unadjusted models iAUC Appetite for Sweet subjective VAS rating change from PD1 to PD14.	202

Figure 7.10. Adjusted model iAUC Appetite for Sweet subjective VAS rating change from PD1 to PD14 across conditions.....	202
Figure 7.11. Unadjusted models iAUC Desire to Eat VAS rating change from PD1 to PD14.....	203
Figure 7.12. Adjusted model iAUC Desire to Eat subjective VAS rating change from PD1 to PD14 across conditions.....	204
Figure 7.13. Control of Eating Questionnaire Craving Control as a function of condition and probe day.....	206
Figure 7.14. Control of Eating Questionnaire Craving Control adjusted model condition*probe day interaction effect.....	206
Figure 7.15. Control of Eating Questionnaire Sweet Craving as a function of condition and probe day.....	207
Figure 7.16. Control of Eating Questionnaire Savoury Craving as a function of condition and probe day.....	207
Figure 8.1. Differences in the change from pre- to post-ingestion explicit wanting sweet bias in high ($n=36$) and low ($n=14$) sweet likers.....	229
Figure 8.2. Differences in the change from PD1 to PD14 explicit wanting sweet bias in high ($n=36$) and low ($n=14$) sweet likers.....	229
Figure 8.3. Differences in change in body weight (kg) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers.....	235
Figure 8.4. Differences in changes in fat-free mass (kg) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers.....	236
Figure 8.5. Differences in changes in total energy intake (kcal) between high ($n=35$) and low ($n=13$) sweet likers between PDs.....	238
Figure 8.6. Difference in change in carbohydrate intake (g) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers.....	239
Figure 8.7. Differences in change in fibre intake (g) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers.....	240
Figure 8.8. Differences in change in fat intake (g) from PD1 to PD14 in high ($n=35$) and low ($n=13$) sweet likers.....	241

Table of Tables

Table 2.1. List of approved HIS in Europe from the European Food Safety Authority	33
Table 2.2. Table of search terms by category.	38
Table 2.3. GRADE risk of bias assessment outcomes.	42
Table 2.4. The effects of acute or repeated HIS ingestion, compared to sugar or water controls on subjective appetite ratings, appetite related biomarkers, <i>ad libitum</i> intake and free-living intake.....	44
Table 2.5. Effects of HIS versus sugar and/or water controls on body weight.....	65
Table 3.1. Full inclusion and exclusion criteria for study 1 (DIVA).	91
Table 3.2. Calorie and macronutrient composition of the <i>ad libitum</i> test meal.	97
Table 3.3. Table of inclusion and exclusion criteria for the SWEET study.....	101
Table 4.1. Descriptive statistics for full study sample ($n=94$).	115
Table 4.2. Means and Standard Deviations from Leeds Food Preference Questionnaire for full study sample ($n=94$).....	116
Table 4.3. Correlation coefficients for Bivariate Pearson’s correlations of Leeds Food Preference Scores scores at three times points across baseline in the full sample ($n=94$).	124
Table 4.4. Intraclass correlation coefficients for Leeds Food Preference Questionnaire choice bias at breakfast, pre-lunch and post-lunch across baseline.....	125
Table 4.5. Correlation coefficients for Leeds Food Preference Questionnaire outcomes across a weight loss intervention.	129
Table 4.6. Intraclass correlation coefficients for Leeds Food Preference Questionnaire Sweet Bias at breakfast, pre-lunch and post-lunch across baseline, week2 and post-intervention in participants with overweight or obesity ($n=37$).	130
Table 5.1. Participant baseline descriptive statistics in both participants with overweight and obesity and normal weight by group.....	144
Table 5.2. Correlation matrix displaying baseline correlations between Leeds Food Preference Questionnaire sweet bias outcomes and eating behaviour trait variables ($n=87$).	146
Table 5.3. Table of correlations between Leeds Food Preference Questionnaire sweet bias outcomes and Rate of Weight Change ($n=37$).	147
Table 5.4. Participant baseline descriptive statistics and differences in High ($n=25$) and Low ($n=11$) Sweet Liker Phenotype Groups.....	Error! Bookmark not defined.
Table 5.5. Baseline, Week 2 and Post-Intervention Eating Behaviour Trait Scores for High and Low Sweet Liker Phenotype Groups.	149
Table 6.1. Table of LighterLife food products and energy and macronutrient compositions..	168
Table 6.2. Baseline study characteristics of participants by group.	170
Table 7.1. Energy and macronutrient composition of the intervention products.	187

Table 7.2. Participant descriptive statistics at PD1 $n=50$	191
Table 7.3. Means and Standard Deviations for Leeds Food Preference Questionnaire Sweet Bias Outcomes Pre- and Post-Ingestion across Probe Days and Conditions.	194
Table 7.4. Models of adjusted and unadjusted repeated measures ANOVA results for Leeds Food Preference Questionnaire sweet bias outcomes.	195
Table 7.5. Means and Standard Deviations of iAUC appetite ratings.	200
Table 7.6. Models of adjusted and unadjusted repeated measures ANOVA for iAUC subjective appetite ratings.....	201
Table 7.7. Means and standard deviations for Control of Eating Questionnaire outcomes ($n=50$).....	204
Table 7.8. Models of adjusted and unadjusted repeated measures ANOVA results for Control of Eating Questionnaire outcomes ($n=50$).....	205
Table 7.9. Means and standard deviations for body weight and composition outcomes ($n=50$).	208
Table 7.10. Models of adjusted and unadjusted repeated measures ANOVA results for body weight and composition outcomes ($n = 48$).	209
Table 7.11. Means and standard deviations for 24hr dietary recall outcomes.	211
Table 7.12. Models of adjusted and unadjusted repeated measures ANOVA results for 24hr dietary recall outcomes ($n=48$).	211
Table 8.1. Descriptive statistics of high and low sweet liker phenotype groups, with independent groups t-test values ($n=50$).	225
Table 8.2. Means and standard deviations of Leeds Food Preference Questionnaire outcomes in high ($n=36$) and low ($n=14$) sweet likers both condition, PD and time.....	227
Table 8.3. Models of adjusted and unadjusted mixed ANOVA results for Leeds Food Preference Questionnaire sweet bias outcomes.	228
Table 8.4. Models of unadjusted mixed ANOVA results for iAUC subjective appetite rating outcomes.	231
Table 8.5. Model of unadjusted mixed ANOVA results for Control of Eating Questionnaire outcomes.	233
Table 8.6. Model of unadjusted values for mixed ANOVA results for body weight and composition outcomes.	234
Table 8.7. Model of unadjusted mixed ANOVA results for dietary recall outcomes.....	237

Table of Appendices

Appendix 1 Leeds Food Preference Breakfast and Lunch Versions (Study1).....	310
Appendix 2 Visual Analogue Scale Questions (Study1)	311
Appendix 3 Eating Attitudes Test 26.....	315
Appendix 4 Short sweet Food Frequency Questionnaire.....	316
Appendix 5 Control of Eating Questionnaire	317
Appendix 6 Binge Eating Scale	319
Appendix 7 Three Factor Eating Questionnaire.....	323
Appendix 8 Calculation of composite appetite score and visual analogue scale questions (Study 2).	326
Appendix 9 Leeds Food Preference Questionnaire core images.....	327

Abstract

Obesity rates have increased globally alongside intake of palatable, energy-dense foods. Sweet preferring individuals display increased energy intake, experiencing greater risk of developing obesity. Sweet preferences require study to illuminate potential barriers to successful weight loss and responses to sweet foods reformulated with high intensity sweeteners (HIS).

The following thesis presents exploratory analyses of two randomised clinical trials investigating; i) the stability of sweet preferences, ii) the relationship between baseline sweet preferences and eating behaviour traits, iii) comparison of two differing dietary weight loss protocols on sweet related outcomes, iv) the impact of acute and repeated consumption of HIS and sucrose sweetened products on subsequent sweet preferences and eating behaviours and finally, v) an exploration of the potential effect of sweet liker phenotypes on the impact of acute and repeated consumption of HIS and sucrose sweetened products on subsequent preferences and eating behaviours.

Results showed; i) sweet food preferences were demonstrated to be stable across a period of weight loss, ii) participants with overweight/obesity presented with greater scores on eating behaviour traits involving a loss of control around food, iii) the method of dietary weight loss did not impact outcomes, iv) there was no difference in sucrose and HIS products on subsequent sweet food preferences and v) the reduction in sweet wanting after consuming a sweet food differs between sweet phenotype groups. It is concluded that sweet preferences are a stable trait that does not change during weight loss, and reformulated sweet foods may facilitate a reduction in sugar and energy density whilst maintaining consumer palatability.

1 General Introduction and Background

1.1 The Prevalence and Causes of Obesity

Obesity has been termed a global epidemic and presents a public health challenge (James, 2018). Estimates provided by the World Health Organisation state the worldwide prevalence of obesity to have almost tripled since 1975 (World Health Organization, 2015). Among adults over the age of 16 years in the UK, 68% of men and 60% of women possess an overweight body mass index (BMI) – equal to or greater than 25 kg/m^2 , with obesity rates increasing across age groups up to 75 years (NHS Digital, 2019). Current forecasts predict that maximum levels of obesity will be reached between 2030 and 2052, with the UK being one of the first countries to reach this point (Janssen et al., 2020), whilst other projections estimate that by the year 2035, five million people in England, Scotland and Wales will be living with morbid obesity – a BMI $\geq 40 \text{ kg/m}^2$ (Keaver et al., 2020). The current levels of overweight and obesity and future forecasts are concerning, as increased levels of body fat increases the risk of certain types of cancer (Vucenik & Stains, 2012), lowers overall life expectancy (Peeters et al., 2003) and is associated with type-II diabetes mellitus development (Lazar, 2005), thereby placing a greater strain on health services. Obesity is also a key component of ‘metabolic syndrome’ – a collection of health related issues such as glucose intolerance, insulin resistance, high triglyceride levels, low levels of high-density lipoprotein (HDL) and hypertension (Abete et al., 2010). Most recent estimations regarding the cost to the NHS of obesity and obesity related treatment cited a figure of £6.1 billion, with the wider societal costs estimated at £27 billion – consequently the costs of obesity on UK society is greater than the combined costs of the police, fire service and judicial systems combined (Public Health England, 2017). Therefore, it is economically more viable to prevent the associated metabolic disorders and health ailments, rather than to provide treatment (Lawlor & Chaturvedi, 2006).

It is important to acknowledge that singular explanations of obesity are not sufficient, as obesity is a multifaceted issue with a wide variety of contributing factors including, but not limited to, social, biological, individual psychology and environmental. At an individual level, genetic evidence is partially capable of explaining individual differences in bodyweight,

however at a population level this evidence cannot explain the drastic increase in obesity prevalence (Wardle et al., 2008). Changes to obesity levels have occurred too rapidly to be fully attributed to genetics. This therefore suggests that lifestyle changes are an important influence in the development of global obesity rates. For example, individuals with obesity consistently demonstrate lower levels of physical activity and higher levels of sedentary behaviour (Curran et al., 2023). Additional evidence also highlights increasing in parallel to obesity rates has been a number of environmental changes, in which the modern lifestyle encourages the consumption of energy (Chaput et al., 2011). Foods rich in sugar and saturated fats are the major source of energy in most countries and shifts in the food supply caused by economic development increasing the availability per capita of food, are a major driver of the obesity epidemic (Zobel et al., 2016).

It has been suggested that increases in adiposity should be seen as a normal physiological response to changes in the environment (Zheng et al., 2009). Specifically these changes are an escalation in the availability of so called highly- and ultra-processed foods that are low in nutrients and poorly satiating, but highly palatable, and affordable (Swinburn et al., 2011) (for further discussion see section 1.7.3). There have also been alterations to the amount of food consumed, with notable increases in the portion sizes of foods commonly eaten away from the home (Young & Nestle, 2002) and recent evidence has demonstrated that consumed portion sizes tend to be significantly greater than serving-size recommendations (Rippin et al., 2019). Furthermore, a 2006 review summarised the trends in energy intake and expenditure in children aged 2-19 years from 1970 onwards, citing increases in sugar-sweetened beverage (SSB) as well as fast food consumption as notable contributors to obesity rates (Anderson & Butcher, 2006). Despite there being no currently agreed upon consensus regarding the precise cause of obesity, review of the available literature produces a clear image that eating patterns and energy intake are playing a highly important role (Ross et al., 2016). Therefore, to aid in reducing obesity and improve longevity at a population level, it is imperative to develop an understanding of factors influencing eating behaviour and food selection behaviours in greater detail.

1.2 The Food Environment

Historically, in the UK intake of sugar and saturated fats were restricted by their low availability due to post-war rationing, however their availability, and subsequently their intake, has increased in recent years due to technological advances (Cordain et al., 2002, 2005). Key changes in the food environment have also involved changes to the proximity and density of supermarkets, fast-food outlets, restaurants, convenience stores and bakeries (James et al., 2017; Reardon et al., 2003). There also exists a positive correlation between the density of fast-food outlets in an area and obesity rates in both children (Fraser & Edwards, 2010) and adults (De Vogli et al., 2011). The increasing availability of outlets providing highly-palatable and energy-dense foods is characteristic of a Western diet (Stender et al., 2007). The modern Western style diet is associated with a higher incidence of overweight and obesity (Murtaugh et al., 2007), chronic kidney disease (Odermatt, 2011), type-II diabetes mellitus and cardiometabolic risk (Odegaard et al., 2012). A typical Western style diet is characterised by a high intake of sugars (easily digestible carbohydrates possessing one or two molecular bonds) and saturated fatty acids (Cordain et al., 2005) with a simultaneous lower intake of fibre, fruit and vegetables and complex carbohydrates (Nielsen et al., 2002). It is therefore clear that the food environment, crucially, the availability of energy-dense foods, is in part contributing to increasing body weight and adiposity, with evidence suggesting that changes in the food energy supply are capable of explaining changes in bodyweight in the UK since the 1980s (Scarborough et al., 2011).

1.3 Energy Balance and Appetite Control

Obesity is a consequence of a positive energy balance, as dictated by the First Law of Thermodynamics which relates to the conservation of energy. Specifically, when energy intake exceeds energy expenditure there is a consequential increase in body fat stores (Bray et al., 2004) and when expenditure exceeds intake, there is weight loss (see Figure 1.1). Subsequently, it is generally accepted that overweight and obesity are conditions resulting from the consumption of excess calories, placing an individual in a chronic caloric surplus. Unfortunately, the First Law does not provide consideration to the complexity of food intake

that is nutritional composition, palatability, food preferences, social circumstances, lifestyle or genetic contributions. When applied to human energy balance, the First law is a simplification of a multifaceted issue, focusing on energy regulation from a purely numerical viewpoint, outlining the mechanism of action by which excess energy intake leads to an increase in body fat levels. Due to the nuanced reasons contributing to food selection, it remains important to consider behavioural aspects of appetite regulation, in order to better inform methods to achieve either a calorie deficit or a state of energy balance.

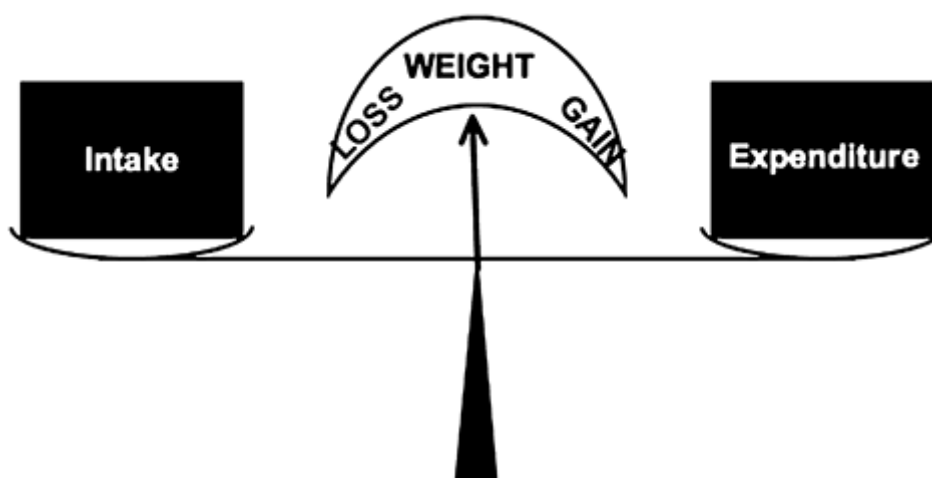


Figure 1.1 The energy balance equation taken from (Schoeller, 2009).

Appetite regulation is the integration of several processes which together form a psychobiological system to signal hunger (drives intake), satiation (inhibits intake) and satiety (between-meal suppression of hunger) (Blundell, 1991). Episodic and tonic signals interact to influence these processes. Episodic signals arise from the gastrointestinal tract and as such occur on a meal-to-meal basis (Berthoud, 2002). Episodic signals can be excitatory although are primarily inhibitory. Tonic signals on the other hand stem from bodily tissues to relay information regarding energy availability and demands (Morton et al., 2006). A number of hormones are secreted by the endocrine pancreas to influence food intake. Insulin and amylin are secreted by B-cells and signal the circulating energy levels (glucose) and stored energy (visceral adipose tissue) (Woods et al., 2006). The overall expression of appetite involves the interaction of these homeostatic processes with hedonic processes (Blundell & Finlayson, 2004).

More recently, the notion of independent homeostatic and hedonic systems has been developed into a framework emphasising the crosstalk between neurochemical substrates within the two systems (Berthoud et al., 2017). The development of this framework is consistent with evidence demonstrating that metabolic state influences eating behaviour through modulation of the hedonic value of foods (Berthoud, 2011). Alliesthesia is the term used to describe the process by which pleasurable sensations are altered by metabolic state (Cabanac, 1971), in that food is more well liked and desired when hungry, but less liked when satiated (Berridge et al., 2010). Further developing this model is the notion that cognitive processes such as learning, attention and memory influence appetite (Higgs et al., 2017). However, the inclusion of cognition within a food reward model does not suggest that individuals constantly consider food decisions at a conscious level, as eating behaviours are influenced by sub-conscious processes the majority of the time (Herman & Polivy, 2014). Underlying cognitive factors influence preferences and eating behaviours via expectations associated with a food (involving memory and attention) as well as the perceived pleasantness and reward whilst eating the meal (involving attention and cognitive control) (Higgs et al., 2017). As displayed in Figure 1.2 homeostatic, cognitive and hedonic elements of appetite control work together to influence eating behaviours.

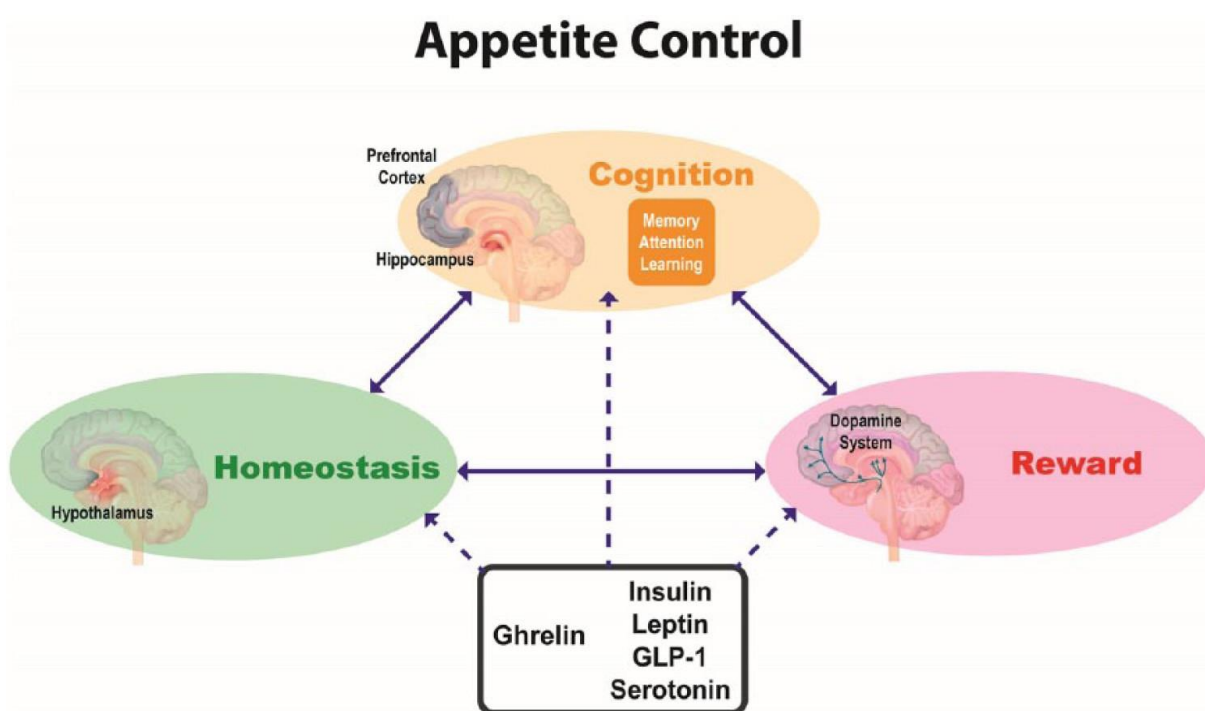


Figure 1.2. Visualisation of appetite control systems taken from (Higgs et al., 2017).

1.4 Food Reward

Whilst homeostatic systems operate due to the necessity to negate energy depletion, driven by nutritional demands, hedonic systems are modulated by external as well as internal factors (Anderson et al., 2006). The key components affecting hedonic systems include palatability, food reward states and eating behaviour traits (Berthoud & Zheng, 2012). Food preferences and reward can be influenced by thoughts relating to sensory appreciation of attributes such as sugar and fat and contribute to determining meal size and eating frequency (Dalton et al., 2013b).

It has been suggested that mechanisms of reward which were originally serving to increase ingestion are no longer an asset in an obesogenic environment defined by a ready availability of palatable and energy dense foods (Olszewski et al., 2019). Mechanisms such as increased attention, motivation and reward towards food when deprived of calories (Stice et al., 2013) are, in certain individuals, a disadvantage. Due to the constant availability of foods within society, these mechanisms now assist in producing weight gain through an encouragement to overconsume. For example, in humans eating in the absence of hunger is associated with increased energy intake and weight gain (Feig et al., 2018). Moreover, evidence provided via comparison of lean females relative to those with obesity has demonstrated greater activation of brain regions involved in anticipation of food in those living with obesity (Stice et al., 2008), highlighting the association between food reward and obesity.

1.4.1 Components of Food Reward

Food reward and by extension, the expression of food preferences, involves two core processes – liking and wanting (Berridge & Robinson, 2003). Within the current thesis, the conceptualisation of liking and wanting as psychological constructs is based upon the definition outlined by Finlayson and colleagues (e.g., Finlayson & Dalton, 2012) in which liking and wanting are dissociable components of reward, expressing both implicit and explicit dimensions. Liking and wanting are derived from the semantics of a shared language to describe human activities, in which liking and wanting have separate meanings. Food liking and wanting are frequently discussed in relation to subjective states or feelings that correspond

to the ordinary understanding of these terms in the context of human appetite as well as being used to denote specific neural processes of reward. To distinguish ordinary use and specific psychological processes, explicit liking refers to conscious pleasure whilst explicit wanting refers to the desire of a food which involves cognitive appraisals mediated by cortically-weighted circuitry (Berridge & Kringelbach, 2015; Morales & Berridge, 2020).

Liking is the perceived hedonic value of a reward – either imagined or experienced, the appreciation of its sensory qualities or the subjective degree of pleasure that consumption elicits. Liking may not be a stable trait, with ratings being influenced when in a satiated (Small et al., 2003) or a fasted state (Cameron et al., 2014). Wanting on the other hand, is defined as the motivational attraction towards a food cue or related food cues and this implies a target food or food type. Wanting demonstrates a greater degree of variation than liking due to factors such as hunger (Small, 2001) or sleep duration and quality (Benedict et al., 2012). It is possible for wanting to be broad, such as when in a food deprived state and increases independently of BMI (Castellanos et al., 2009), or focused, with a drive for a specific macronutrient when in a state of imbalance (Griffioen-Roose et al., 2012).

In this thesis, explicit liking (EL) is defined as the perceived or anticipated hedonic reaction produced by a tasted food, whereas explicit wanting (EW) is the subjective desire for a perceived food. Implicit wanting (IW) is derived from the concept of ‘incentive salience attribution’ and involves the implicit motivation to consume one food over another (Finlayson et al., 2007b). Liking and wanting are viewed as separate as their degradation following food consumption can occur at differing rates. For example, when healthy participants consumed chocolate beyond satiation it resulted in a greater and faster decline in subjective ratings of wanting relative to ratings of liking (Small, 2001). Liking in this manner can be reduced following consumption of a food, but cannot be entirely eliminated, and thus it is possible for liking to remain present even in the absence of wanting. Furthermore, it is possible for liking to activate wanting, but liking does not necessitate wanting (Berridge & Robinson, 2003), whereas wanting without liking can describe a compulsive element to eating.

Cognitive processes become involved via learnt associations. Tastes and smell are cues which enable the learning of the nutritional consequences of food and drinks that are consumed, with positive or negative hedonic responses shaping behaviour to ensure that nutritional needs are met. When a consumed food evokes a hedonic response, the characteristics of that food, such as the sight or smell, become associated with the positive consequences elicited by consumption. Because these food associated cues acquire the ability to be sought after, the association between cues and consumption of pleasurable foods is able to promote food seeking behaviours and intake (Berridge, 1996). Similarly, these cues will evoke expectations around taste, satiation and whether the consumption of the food is consistent with long-term health goals (Brunstrom, 2011; Rangel, 2013). Decisions made about whether to eat, or how much to eat, are informed by these expectations (Rangel & Hare, 2010), however there are several additional factors, such as whether the consequences of behaviour are retrieved from memory and become the focus of attention (Hare et al., 2010; Whitelock et al., 2018), as well as interference from alternative competing cognitive demands, such as watching television (Braude & Stevenson, 2014).

1.4.2 Food Cravings

Food cravings are an intense desire directed towards specific foods (Gendall et al., 1999) and are experienced by as much as 97% of the adult population (Christensen, 2007). As with liking and wanting components of food reward, food cravings are a hypothetical construct and as such are subject to the issues surrounding such constructs in that they are not directly observable nor measurable (Weingarten & Elston, 1990). Cravings are often measured through use of subjective evaluations (Meule, 2020c) and are differentiated from a general hunger – which can be diminished or eliminated by consumption of any type of food (Hormes, 2014; Martin et al., 2006), as cravings represent an intense desire to consume a specific food (Weingarten & Elston, 1990). The distinguishing feature is the intensity of the state and the specificity of the craved food (Pelchat, 2002). Food cravings arise from a range of biological, affective and cognitive triggers (Tiggemann & Kemps, 2005). Although cravings are commonly associated with hunger (Hill et al., 1991) and short-term energy deprivation (Meule, 2020a) these are not

prerequisites (Pelchat & Schaefer, 2000). Negative affect (Christensen, 2007; Lafay et al., 2001), visual imagery (Steel et al., 2006), smell (Fedoroff et al., 2003) and menstrual cycle in women (Dye et al., 1995) are all factors involved in the experience of food cravings.

Food cravings are a normative part of life, survey data highlights the majority of people commonly indulge their cravings, with chocolate being the most commonly and intensely experienced craving in Western cultures (Lafay et al., 2001). Food cravings, although not necessarily pathological, can give rise to negative affect such as guilt or shame (Fletcher et al., 2007). Moreover, cravings are a precursor to binge eating (Gendall et al., 1998) and are associated with eating psychopathology, an association which is stronger in females than males (Chao et al., 2014). Food cravings demand cognitive availability, and operate in a similar fashion to other distractions which interfere with cognitive processes (Green et al., 2000) although the cognitive resources demanded by cravings are limited (Kemps et al., 2008).

Sweet cravings in particular have been linked to hormonal changes within the body (Chao et al., 2014; Tsouristakis et al., 2019). Administration of leptin, a sweet taste moderator, has been shown to reduce sweet cravings (Conroy et al., 2014) whilst conversely, higher ghrelin levels appear to be linked to increased cravings (Chao et al., 2017). However, cravings cannot be fully explained via physiological processes. Sensitivity to reward is positively associated with food cravings (Franken & Muris, 2005) and brain imaging techniques have demonstrated that individual variability in reward sensitivity is highly correlated with activation of brain regions when shown images of food (Beaver, 2006).

1.5 Sweet Food Preferences

Habitual sweet liking, referred to as a 'sweet tooth' in colloquial terms, is defined as a preference for increasing concentrations of sweeteners in food and drinks, in addition to a preference for sweet over savoury tastes (Drewnowski et al., 2012). Every species has developed taste sensitivity and preferences that are co-adapted to its ecological niche, resulting in the genes for sweet taste receptors evolving over 400million years ago (Angotzi et al., 2020). Sweet tastes are registered by the combination of two G-protein coupled receptors – T1R2 and

T1R3 – to create a sweet sensing receptor in mammals (Li et al., 2002; Nelson et al., 2001; Zhao et al., 2003). Sweet taste receptors are located in the mouth, gut and pancreas and do not discriminate between caloric and non-caloric (high-intensity sweeteners (HIS)) sources of sweetness (Drewnowski et al., 2012). These receptors are subject to diurnal variation in their sensitivity, believed to be a consequence of variations in circulating leptin levels within the blood (Nakamura et al., 2008). Leptin serves as a sweet taste moderator, for example, when mice receive an injection of leptin there occurs an observable reduction in sweet taste sensitivity (Kawai et al., 2000). In human trials, it has been suggested that chronically elevated circulating leptin levels in individuals with an elevated level of adiposity are responsible for a reduction in sweet taste sensitivity (Sartor et al., 2011). Similarly, fibroblast growth factor 21 (FGF21) is a liver derived hormone which has been demonstrated to reduce appetite for sugars and HIS in mice (Talukdar et al., 2016). FGF21 is released following consumption of sucrose and sweet disliking individuals have been demonstrated to have elevated levels of the hormone (Søberg et al., 2017).

The sweet taste possesses a powerful hedonic impact due to the association shared with energy-density, for this reason it has been proposed as an important contributor towards excess energy intake and subsequent weight gain (Te Morenga et al., 2012). In rodent models, following a high sugar diet has been shown to reshape sweet preferences and subsequent feeding behaviours (May et al., 2019). This is because repeated exposure conditions flavour preferences (Liem & De Graaf, 2004), consequently, a preference for sweet foods is associated with a greater intake of carbohydrates (Drewnowski, 1999). Evidence provided via a prospective study demonstrated that the hedonic response to sweet tastes predicts weight gain at a 5-year follow up (Salbe et al., 2004). In light of this, an un-sweetening of the diet has been suggested as a possible solution to the current obesity epidemic (Yang, 2010). However, this may not be the most optimal solution, as when in an energy deficit the subjective pleasantness of a sweet taste increases, which may in turn be a driver of increased sweet food intake (Rodin et al., 1976) and may present an explanation for failed weight-loss attempts in chronic dieters.

Willingness to consume sweet nutrients is likely influenced by believed, or known dietary effects encountered personally in popular literature and the media or advised by health professionals (Reed & McDaniel, 2006). Despite UK guidelines recommending a free sugar intake of no more than 5% of total energy intake (Scientific Advisory Committee on Nutrition, 2015), thematic analysis has identified confusion in the public regarding information and recommendations as a barrier to weight-loss that requires addressing (Buchanan & Sheffield, 2017). Subsequently, cross-sectional data available via the National Diet and Nutrition Survey (2008-2014) highlighted that the average intake of free sugars in the UK is 12.4% of total energy intake, with 61.3% of participants consuming >10% of total energy intake from free sugars (Rauber et al., 2019). However, during the months following the March 2020 UK coronavirus lockdown there were large alterations to diet and physical activity levels, with 56% of responders to an online survey reporting an increase in snack food intake (Robinson et al., 2021). Subsequently the figures provided by Rauber and colleagues (2019) may in reality underestimate these values.

Moreover, the time of day is an important variable when considering the intake of sweet foods. Sweet foods tend to be consumed between meals as snacks (Bertéus Forslund et al., 2005; van Langeveld et al., 2018) and snack foods high in sugar and fat are often consumed beyond homeostatic needs (Cleobury & Tapper, 2014). However, not every individual is prone to overconsumption of snack foods. Certain behavioural traits are linked to increased intake of sweet snack foods. For example, online survey data ($n=875$) collected during the coronavirus lockdown reported that lower craving control was the strongest predictor of increased intake of energy dense foods. Similarly, low cognitive restraint predicted greater intake of sweet energy dense snack foods (Buckland et al., 2021).

1.5.1 Sweet Liker Phenotype

Liking of sweet stimuli is considered innate and universal, with evidence demonstrating preferences in infants as young as 23-84hrs old (Desor et al., 1973). Despite being a universal preference, it was first shown in 1970 that the preferred sweetness level differs between individuals (Pangborn, 1970). Pangborn examined individual profiles of rated liking as a

function of sugar concentration, using this to identify three distinct phenotypes – sweet likers, moderate-sweet likers and sweet dislikers. However, due to the innate nature of sweet preferences, there have been recent suggestions for reconsideration of these groups (Iatridi et al., 2019b, 2019a; Yang et al., 2019). More recently, through the use of sucrose solutions, at least four different sweet liker phenotypes have been identified, as shown in Figure 1.3 (Iatridi et al., 2019b, 2019a). These response patterns are characterised by a rise in liking with increasing sucrose concentrations (sweet liker phenotype), an inverted U-shaped hedonic response (inverted U-shaped phenotype), a decline in liking as sucrose concentration increase (sweet disliker phenotype) and an insensitive response to changes in sucrose concentration.

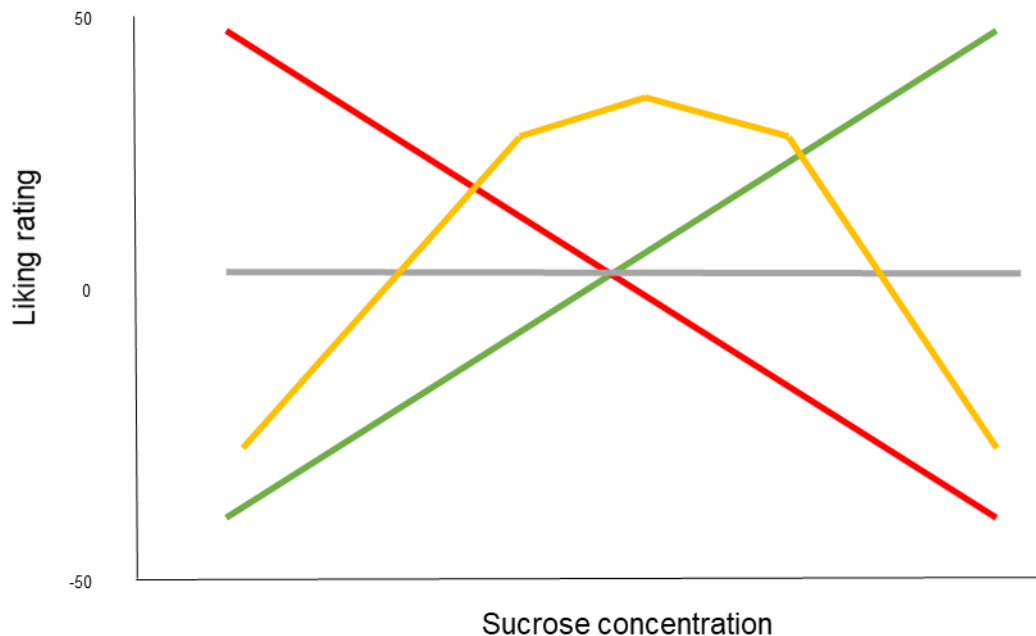


Figure 1.3. Four distinguishable patterns of sweet liking in response to sucrose concentration (taken from Iatridi et al., 2019a, 2019b).

Currently there is no clear explanation as to why these different phenotypes exist, however it was originally argued that the momentary expression of liking directed towards a sweet taste was reflective of an underlying physiologically sensed nutrient deficit (Cabanac, 1971, 1989; Rolls et al., 1983). However, modifications to sweet liking on the basis of hunger state is not always observed (Moskowitz et al., 1974), therefore this is unlikely to be the case. In light of recent evidence which demonstrates higher sensitivity to hunger cues in extreme sweet likers

(Iatridi et al., 2020) it has been suggested that these differences reflect variations in underlying sensitivity to appetite signalling (Armitage et al., 2021). For example, sweet likers tend to score higher on the TFEQ hunger scale (Iatridi et al., 2020), as well as showing higher interoceptive performance (Iatridi et al., 2021). However, although this highlights differences between the phenotypes, it fails to establish why these phenotypes exist in the first instance.

It is possible that sweet phenotypes are a manifestation of differences in exposure to sweetness in the diet (Armitage et al., 2021). A review of 21 studies reported that increased exposure to sweetness lead to a reduction in sweet food preferences (Appleton et al., 2018), although a 40% reduction in sugar intake was found to increase sweetness intensity with no impact on pleasantness ratings (Wise et al., 2016). Furthermore, studies which contrast dietary intake using a dichotomous phenotype approach (liker/disliker) have reported higher intakes of sugars (Holt et al., 2000) and sugar sweetened beverages in sweet likers (Turner-McGrievy et al., 2013). Studies which compare differences across three phenotype groups (including extreme sweet likers) found that sugar sweetened beverage intake was higher in extreme sweet likers (Garneau et al., 2018; Iatridi et al., 2020). Together, these findings suggest that sweet exposure within the diet is an unlikely cause of sweet liker phenotype groups. When examining potential genetic differences, individuals with increased sensitivity to the bitter tastant 6-n-propylthiouracil (PROP) are reported to perceive more sweetness (Looy & Weingarten, 1992). This has been exhibited for the HIS saccharin (Bartoshuk, 1979) as well as sucrose (Gent & Bartoshuk, 1983). These genetic differences may impact preferences and intake, as sweet taste intensity is negatively associated with the frequency of sweet food intake, and hedonic ratings of sweet tastes are positively associated with intake (Jayasinghe et al., 2017). Therefore, those individuals that are most sensitive to sweet tastes may perceive solutions as more intense, resulting in these individuals experiencing the stimuli as less pleasant (Peterson et al., 1999).

1.5.2 Body Weight Differences in Sweet Food Preference

Despite early evidence suggesting that sweet food preference is not influenced by body weight (Wooley et al., 1972), more recent evidence has highlighted differences between sweet food preferences and subsequent intake when differentiating the population by body weight and sex

(van Langeveld et al., 2018). However, the evidence regarding body weight differences in food preferences remains inconclusive, as some studies have demonstrated an association between BMI and liking for dietary fats (Cox et al., 2016; Deglaire et al., 2015), liking for fat and salty foods (Matsushita et al., 2009) or no association present with taste preferences (Cox et al., 1999). However, the incongruence observed within the literature is likely due to methodological variations between studies generating difficulties in drawing conclusions (Cox et al., 2016).

There is however some evidence to suggest differences in food intake and preferences between individuals differing by BMI in regards sweet food preferences. Proxy measures of sugar intake – such as dental cavities – suggest that women with a BMI defined as overweight, consume more sugar than do lean women (Barkeling et al., 2001). Similarly, when measuring salivary counts of mutans streptococci and lactobacilli as an indication of sugar containing sweet food intake, it was found that women with obesity consumed a higher amount of sweet foods than lean controls (Barkeling et al., 2001). However, this evidence fails to establish whether the differences in sugar intake are a cause or a consequence of the increased levels of body fat. It is possible that differences in sweet food preferences occur as a consequence of weight gain. Distortions to taste and smell perception are closely linked to visceral fat levels within the body (Fernandez-Garcia et al., 2017), likely arising from differences in sweet taste thresholds (Donaldson et al., 2009). Through the use of taste strips it has previously been established that a general lowering of taste sensitivity occurs with increases in BMI (Vignini et al., 2019). Lean individuals when compared to individuals with overweight and obesity present an increased sweet taste sensitivity and consequently require weaker concentrations of a stimuli. This leads to a lower intake of carbohydrates, frequency of sweet food intake and a lower total energy intake (Jayasinghe et al., 2017). Subsequently individuals with overweight and obesity present with a lower perceived sweetness intensity (Donaldson et al., 2009). Moreover, there exists a positive association between BMI and preferences for high fat sweet (HFSW) foods, with the effect more pronounced in women than in men (Deglaire et al., 2015). The introduction of fat to a sweet stimulus leaves the sweet taste unaltered, although increases the palatability of the

stimulus (Bolhuis et al., 2018; Drewnowski et al., 1992). In this manner, taste sensitivity may affect food preferences and subsequent intake, potentially resulting in a positive association between BMI and HFSW preferences specifically.

1.5.3 Food Preferences and Physiological Mechanisms

Aspects of taste perception such as sensitivity thresholds are important to consider due to their potential capacity to impact food preferences (Akella et al., 1997), for example, individuals with an increased sweet taste sensitivity consume significantly more non-sweet foods (Han et al., 2017). When considering the causality of this observation, an informative, but extreme population to examine are individuals that have undergone weight reduction surgery, whom demonstrate dramatic changes to food preferences post-surgery (Altun et al., 2016). Preferences prior to surgery tend to be directed towards HFSW foods whereas post-surgery preferences are directed towards fruits and foods lower in energy density (Andriessen et al., 2018). Following laparoscopic sleeve gastrectomy (LSG) 94.8% of patients report an increase in sweet taste sensitivity (Shoar et al., 2019). LSG results in increases in circulating ghrelin levels, and supports the influence of hormones in the regulation of sweet taste sensitivity. Conversely, following Roux-en-Y gastric bypass (RYGB), patients report a decrease in sweet taste sensitivity (Shoar et al., 2019), with RYGB reducing circulating ghrelin levels. Furthermore, following changes to taste sensitivity, patient's food intake patterns are reportedly altered, with HFSW foods causing postprandial discomfort (Nielsen et al., 2019). This population serves to highlight the relationship between physiological elements of appetite regulation and the manner in which they are influential in determining food preferences. It would appear that circulating ghrelin levels exerts influence over the perceived intensity of sweet tastes (Shoar et al., 2019), with a positive association between ghrelin and sweet taste sensitivity likely.

Similarly, utilising rodent models, leptin has been identified as a sweet taste moderator (Kawai et al., 2000; Shigemura et al., 2004). Leptin is produced in adipose tissue within the body and serves to regulate food intake through inhibition of gustatory responses to sweet substances specifically (Nakamura et al., 2008). Leptin is released in direct proportion to the amount of fat mass within the body, creating a negative feedback loop, whereby elevated circulating leptin

levels inhibit ingestive behaviours in order to maintain optimal levels of adiposity within the body (Zhang et al., 1994). This feedback loop is influential in the role of sweet taste thresholds and occurs via the hypothalamus (Berthoud & Zheng, 2012). However, in individuals with elevated levels of adiposity within the body, this results in chronically elevated circulating leptin and results in desensitisation to its sweet taste suppressing effects (Izquierdo et al., 2019), which can be reversed following sufficient weight-loss (Umabiki et al., 2010).

1.5.4 Effects of Fasting on Sweet Food Preferences

Dietary restraint is defined as the intention to purposefully restrict food intake with the aim of controlling energy intake (Herman & Mack, 1975), and as a consequence of dietary energy restriction a compensatory drive to overeat may arise (Melby et al., 2017). Following a period of short-term fasting the resulting energy depletion and increase in homeostatic hunger, coupled with influences on hedonic mechanisms, serve to increase the drive for food intake (Schwartz, 2006; Schwartz et al., 2003; Thivel et al., 2018; Woods et al., 1998). For example, energy depletion produced by a period of fasting for 24hr led to an increase in VAS rated hunger, as well as increased *ab libitum* food intake in a sample of healthy males (Thivel et al., 2018). This effect can also be seen in the hedonic appeal of food. Participants' ratings of liking and wanting, as assessed via the LFPQ, remained elevated following food intake after a 24hr fast in another study (Cameron et al., 2014). Importantly in this study liking scores for sweet foods did not reduce, highlighting the retention of a strong hedonic saliency, whereas preferences for savoury foods reduced once the fast was broken. Therefore, whilst a period of fasting may increase the drive to eat, this effect may not be equivalent across foods differing in their dominant taste (i.e., sweet versus savoury).

Elsewhere, the length of restriction has been highlighted as an influential factor in determining the effects on food reward (Kahathuduwa et al., 2016). An early study highlighted that manipulating the period of energy deprivation can influence palatability. Through a repeated-measures design requiring participants to complete dietary fasts of either 3.5hrs or 12-15hrs it was shown that the lengthier deprivation period increased subsequent palatability (Spiegel et al., 1989). However, more recent evidence has highlighted differences between acute and

prolonged energy restriction, in which short-term energy restriction (<1 day) serves to increase the hedonic appeal of foods (e.g., Cameron et al., 2014), whereas long-term restriction (e.g., Martin et al., 2006) decreases the hedonic appeal of foods.

1.6 The Neurobiology of Sweet Food Preferences

Whilst the hypothalamus is responsible for the regulation of food intake to satisfy energetic needs, the dopamine reward circuitry includes striatal, limbic and cortical areas that affect eating behaviour (Volkow et al., 2017). Other neurotransmitters involved are serotonin, endogenous opioids and endocannabinoids, each involved in the modulation of a food's hedonic properties (Volkow et al., 2011). Mammalian taste recognition occurs via specialized epithelial cells arranged in taste buds on the tongue (Scott, 2004). Dopaminergic pathways within the brain are activated upon detection of a sweet taste with dopamine being released in proportion to the self-reported level of pleasure elicited by consumption (Small et al., 2003). Sweet taste receptors are not only located on the tongue, but are also located within the gut and pancreas (Margolskee et al., 2007; Sclafani, 2007) as well as the hypothalamus (Kohno, 2017). Consumption of foods high in sugar and fat can strongly trigger these reward mechanisms and thereby encourage consumption beyond homeostatic requirements (Hoch et al., 2014).

Sweet preferences tend to be strongest when an individual is young and display a natural decline with age (Desor & Beauchamp, 1987; Yoshinaka et al., 2016) before increasing again in older adulthood (Venditti et al., 2020), although genetic polymorphisms give rise to large inter-individual differences in taste perception and subsequent preferences (Barragán et al., 2018). This is reflective of an elevated energy demand when young as sweetness is typically indicative of carbohydrates, however, the concept of a macronutrient preference is not concordant with human studies, with human samples rarely requesting pure sugar, in part because humans react to the taste and texture of foods and not only their chemical composition (Levine et al., 2003). Sweetness alone is sufficient to produce a hedonic response (Westwater et al., 2016) although the hedonic response can be magnified, with energy load and sweetness intensity interacting to develop a more potent hedonic response (Veldhuizen et al., 2017). For example, sucralose – a HIS – produces a weaker hedonic response relative to sucrose (Frank et

al., 2008). This highlights the existence of two separate variables in sweetness and energy-density. The neural circuitry that is activated in the presence of the sweetness and energy load interaction, overlaps with circuitry activated via drugs of abuse (Drewnowski et al., 1995). Although this highlights the strength of the stimuli's rewarding characteristics, this is not to suggest that a sweet food preference and addictive behaviours are equivalent (Drewnowski, 2007; Finlayson, 2017).

Liking and wanting are associated with different substrates in the brain. Specifically, liking is related to the nucleus accumbens and ventral pallidum where opioid and endocannabinoid signals can increase pleasure, whereas wanting is linked to larger dopamine networks in the nucleus accumbens, striatum and amygdala (Berridge et al., 2010). Liking and wanting have been referred to as 'go' systems, which can be diminished, but cannot be entirely turned off (Lemmens et al., 2009; Small, 2001). These reward systems may become dysfunctional, for example, if incentive salience detaches from hedonic liking, it may result in wanting to eat rising alone (Finlayson et al., 2007b; Mela, 2006). In a study in which 18 individuals with obesity were compared to 18 lean weight controls, there were reported increases in food cue incentive salience in both groups when fasted. However, this remained elevated only in those with obesity following food consumption and a decreased self-reported hunger (Castellanos et al., 2009), thereby demonstrating dysfunction in those with obesity. Moreover, our knowledge of the energy density of a food drives both behavioural and neural valuation of foods. In one study, the true energy density of an item, rather than the estimated energy density, accurately predicted participant's motivation levels towards that item, as well as correlating with the neural response in the ventromedial prefrontal cortex – an area involved in the encoding of stimuli value (Tang et al., 2014).

It remains unknown what causes differences in reward activation between individuals. An inverse relationship between reward sensitivity and BMI has been proposed, in which both ends of the BMI spectrum experiencing low dopamine reactivity, creating under and over-weight individuals (Davis & Fox, 2008). Indeed, individuals with obesity have been shown on numerous occasions to have marked structural and functional alterations in brain-circuitry

(Patriarca et al., 2017; Volkow et al., 2008; Wang et al., 2002, 2004). Specifically, when looking at pictures of high-calorie foods vs. neutral images, individuals with obesity display greater activation in the caudate/putamen (reward/motivation) and anterior insula (taste) amongst other regions (Rothmund et al., 2007). In another study, when provided with an oral glucose load (75g) following a 12-hr fast, men with obesity displayed activation of a midsagittal slice of the hypothalamus (crucial in the homeostatic regulation of food intake), whereas lean men displayed an inhibitory response (Matsuda et al., 1999). It has been demonstrated that individuals with overweight relative to lean counterparts present with a higher prevalence of the TaqI A1 allele of the dopamine D₂ gene (DRD2), which is associated with a lower D₂ receptor availability (Thomas et al., 2001). This evidence displays a system of hypo-responsivity in reward centres, which has been reasoned to arise due to chronic exposure to energy dense food items (Carnell et al., 2012).

1.7 Types of Sweet Stimuli

As stated, throughout human evolution, the sweet taste has been associated with carbohydrates as an ample energy source (Blundell, 2019; Tan & Tucker, 2019). However, there have been claims that due to the variability of the modern diet that this learnt association is no longer reliable as HIS in absence of the post-ingestive consequences that couple a caloric substance, effectively disrupt this learned association (Swithers, 2013). Indeed, neuroimaging studies highlight that brain regions are not activated equally when ingesting a sugar or HIS (Han et al., 2018). All HIS are capable of activating oral sweet receptors, resulting in signals that generate the conscious perception of sweetness (Brown & Rother, 2012). However, evidence has shown that the brain is capable of dissociating between sugars and HIS (Green & Murphy, 2012) despite participants failing to correctly identify the sweetener type in a blind taste test (Delogu et al., 2016). This would suggest that the sweetener type used may be distinguished at an implicit level, if not at an explicit level, within the brain.

1.7.1 Sugar

Carbohydrates can be divided into three groups, mono/disaccharides, oligosaccharides and polysaccharides (Lim & Pullicin, 2019). Sugar is the generic name provided to sweet tasting,

mono and disaccharides occurring naturally in a number of foods produced in nature, but can also be man-made. Simple sugars are also known as monosaccharides and possess one molecule, forming the building blocks for carbohydrates. Monosaccharides include glucose, fructose and galactose. Disaccharides are composed of two sugar molecules linked together through a glycosidic bond and includes, sucrose (glucose + fructose), lactose (glucose + galactose) and maltose (glucose + glucose). Sugar in its simplest form, provides the building blocks for carbohydrates – carbohydrates when eaten and digested by the body are broken down into sugar.

‘Free sugars’ or also known as ‘added sugars’ are defined as those sugars included during the manufacturing process as well as those naturally occurring in syrups, honey and fruit juices (Scientific Advisory Committee on Nutrition, 2015). Free sugars included in food products serve to increase the palatability of the product (Azaïs-Braesco et al., 2017) but this occurs at the cost of an increase in energy-density. Intake of so called ultra-processed foods has in one study, been shown to provide 57.9% of energy intake with 89.7% of this provided via sugars (Martínez Steele et al., 2016). This creates concerns that the intake of highly-palatable, energy-dense foods displace other more nutrient dense foods in the diet as their intake shares an inverse association with fruit and vegetable intake (Schröder et al., 2007).

Sugar intake has been proposed to be a driver of the current obesity epidemic (Bray & Popkin, 2014) due to the positive association shared between sugar intake and BMI (Elliott, Keim, Stern, Teff, & Havel, 2002; Howard & Wylie-Rosett, 2002; Malik & Hu, 2012). On the other hand it has equally been stated that there is a lack of compelling evidence to indicate that sugar is unique relative to any other source of calories within the diet with regards to the development of obesity (Kahn & Sievenpiper, 2014). However, the relationship between SSB intake and obesity has been demonstrated consistently throughout the literature (Chen et al., 2009; Hu, 2013; Tahmassebi & BaniHani, 2019). Given evidence highlighting a weaker satiating effect of energy obtained via liquid relative to solid products (Almiron-Roig et al., 2003) it may be hypothesised that the sources of sugar and type of sweet foods within the diet are important variables to consider. Sugars within the diet may be provided via fruit and vegetables, dairy,

beverages or general sweet products (Azaïs-Braesco et al., 2017). Other studies have attempted to classify sources of sugar within the diet as natural sweetness, added sugar and general sweet products and observed differences in outcomes as a result. For example, a liking for natural sweetness demonstrates a reduced risk of obesity (Lampuré et al., 2016) and diabetes (Lampuré et al., 2019). It would appear that measurements of total sweet intake, and indeed sugar intake, are therefore limited, with the inclusion of sweet foods either as a replacement or as an addition to other nutrients may be a more correct predictor of whether sweet foods will exert any influence on body weight (Reed & McDaniel, 2006). Further, this notion is supported by the fact that when energy intake is held constant, there is no difference in weight change at varying levels of sugar intake (Te Morenga et al., 2012). Therefore, it may not be the inclusion of sugar per se that is responsible for weight gain, but its proclivity to facilitate a calorie surplus.

1.7.2 Sugar and Fat

Fat is the term applied to naturally occurring triglycerides (Liu et al., 2016). Following the observation that individuals with overweight and obesity ingest a larger proportion of their dietary intake from sources high in dietary fats, it has been proposed as a driver for weight gain (Miller et al., 1990). Dietary fats are considered to be the least satiating macronutrient despite containing 9kcal/g and can promote passive overconsumption – the passive form of high consumption rather than eating as actively driven (Blundell & Macdiarmid, 1997). The hedonic response to sweet tastes is potentiated by the fat content of a food and preferences towards fat content are directly linked to an individual's body fat levels (Drewnowski, 1997). Indeed, women with an elevated BMI tend to report a disliking of sweet solutions absent fat with favourable ratings upon its inclusion (Deglaire et al., 2015; Drewnowski et al., 1985). This may suggest that dietary fat preference is a more important driver of obesity rates than sugar and sweet food preference, however, when the two nutrients are combined in a single food, the overriding taste remains sweet, with the inclusion of dietary fat serving to improve textural qualities and overall palatability of the product (Bolhuis et al., 2018; Drewnowski & Greenwood, 1983). This combination creates a poorly satiating yet highly palatable product that is capable of contributing to passive overconsumption (Lucas, 1985).

The current food environment is a complex one, with a number of the commonly consumed sweet foods simultaneously high in fat (Drewnowski et al., 1992). The inclusion of fat to a sweet product serves to increase the palatability without impacting the sweet perception of the product (Drewnowski et al., 1985) but the inclusion of sugar in the item masks the perception of fat (Drewnowski et al., 1992). Therefore individuals with overweight and obesity may identify this as a sweet food preference as the dominant sensation is that of sweetness (Weingarten & Elston, 1990). As evidenced via the use of food frequency questionnaire data, which highlighted that large sources of dietary fat are obtained via consumption of foods that are simultaneously high in sugars (Drewnowski & Greenwood, 1983). In a hospital-based sample, when asked to report their favourite food items, 56.2% of women with obesity reported 'donuts, cookies and cakes' examples of commonly consumed HFSW foods (Drewnowski et al., 1992) and HFSW snack foods tend to be consumed beyond homeostatic requirements (Cleobury & Tapper, 2014). Similarly, consumption of fast food – foods characterised by large levels of both sugar and fat - and BMI are positively associated, whilst simultaneously displaying an inverse association with fruit and vegetable consumption (Schröder et al., 2007). Therefore, fat may be included within the diet via these highly palatable foods that would otherwise not be consumed, and sweetened fat has largely been responsible for the increased ingestion of carbohydrate fat combinations (Bolhuis et al., 2018; Emmett & Heaton, 1995). In addition to producing an elevated energy intake, HFSW foods also displace more favourable foods from the diet, with a typical individual's diet consisting of HFSW snack food, fast food or sugar sweetened beverages (Martínez Steele et al., 2016) and are often eaten in daily life due to their affordability and accessibility (Drewnowski, 2007).

Therefore, excess consumption of both sugar and fat have been proposed as potential drivers of excess energy intake and subsequent weight gain (Field et al., 2007). This may be more likely than either sugar or fat being the primary driver, as a single nutrient focus is flawed, as demonstrated through the Australian-Paradox, which has shown that over a 30 year period sugar intake has decreased by 20% whilst obesity rates have increased by approximately 300% (Barclay & Brand-Miller, 2011). Conversely, in North America over a 10 year period both BMI

and obesity rates have increased, whilst fat intake has decreased (Heini & Weinsier, 1997).

These incongruent findings would not be observed if the presence of either sugar or fat within the diet were responsible for increasing obesity rates. Therefore, it is necessary to consider the diet within the wider context, considering food preferences and their impact on intake.

Common dietary advice provided to individuals attempting to reduce body fat levels has previously consisted of reducing both free sugar and fat levels within the diet (Gibson, 1996). However, data were obtained via dietary surveys has identified an inverse association between sugar and fat intake, with historical evidence highlighting that high sugar consumers are concurrently low fat consumers, and vice versa (Baghurst et al., 1994; Blundell & Macdiarmid, 1997). This has been termed the sugar-fat seesaw (McColl, 1988) and suggests that reduction in both sugar and fat within the diet may not be optimal advice and may not be achievable at a population level (Gibney, 1990). However, it is important to note that despite the above studies utilising self-report techniques, and such methods requiring caution in their interpretation (Schoeller et al., 1997), the preponderance of studies either do not consider under-reporting participants or do not find a significant effect on results from their exclusion (Sadler et al., 2015). Therefore, observations from the sugar-fat seesaw suggest that as energy intake from one macronutrient increases it will occur at the expense of another macronutrient, causing a displacement effect. Therefore, HIS may prove a more viable option for individuals seeking to reduce or control energy intake, as they are capable of maintaining the sweet taste desired by consumers, whilst potentially reducing energy intake.

1.7.3 High-Intensity Sweeteners

HIS are defined as a substance possessing a sweetness profile thirty times greater or more than sucrose (Hutchinson et al., 1999). HIS can take many forms, consisting of artificial sweeteners (e.g., aspartame), natural sweeteners (e.g., stevia), sugar alcohols (e.g., xylitol), sweet proteins (e.g., thaumatin) or chemical compounds (e.g., steviol glycosides). For the purposes of this thesis all forms of low-calorie, artificial and non-caloric or non-nutritive sweeteners will be termed as HIS, for the fact they provide no nutritional benefits in the form of vitamins and minerals and do not sufficiently contribute to total energy intake. Recent evidence has

demonstrated an increase in the number of HIS sweetened products highlighting a lack of concern regarding the exceedance of HIS average daily intakes globally (Martyn et al., 2018). Similarly, consumers have expressed a desire for clean label foods, creating a drive towards natural sweeteners, despite neither natural nor synthetic sweeteners being metabolically inert (Mora & Dando, 2021).

Due to their substantially greater sweetness intensity, much smaller amounts of HIS are required to achieve the same intensity levels as sucrose, although each HIS presents a unique intensity, persistence of taste and aftertaste (Mortensen, 2006), with some reported to leave a metallic aftertaste (Portmann & Kilcast, 1996). Despite this, HIS use is on the increase (Sylvetsky, Welsh, Brown, & Vos, 2012; Sylvetsky & Rother, 2016) particularly in those individuals seeking to control or reduce the energy content of their diets (Catenacci et al., 2014). However, there remains disagreement regarding the precise effects of these sweeteners on subjective states and behaviours that contribute to body weight, including appetite, food intake and food reward. For example, a recent review demonstrated that observational studies tend to report a negative effect of the inclusion of HIS in the diet, where RCTs tend to report a beneficial effect (Normand et al., 2021).

Beverages sweetened through the use of HIS contain more sweeteners by volume and weight than any other product (Appleton & Conner, 2001). Unfortunately, the mode of administration (i.e. liquid or solid) appears to play an additional role in appetite behaviour and so distinguishing between effects on appetite of the HIS and the vehicle of ingestion is required, particularly when providing a comparison of HIS to sucrose controls. For example, in studies which provide sugar as a liquid supplement to diets, there appears to be no, or limited, compensatory behaviours decreasing intake of other food sources for the additional calories ingested (Raben et al., 2002; Sørensen et al., 2005). Compensatory eating describes the adjustment of energy intake as a consequence of consuming a food item, such as a previous meal, snack or beverage (Booth, 1972). Comparatively, there is a decrease in the amount of additional calories consumed when sugar is included in the diet through a solid food (Tordoff & Alleva, 1990). The reason behind this may be due to differences in post-ingestive

consequences between liquid and solid food items, with satiating consequences absent from beverages (Almiron-Roig et al., 2003) and the satiating effects of beverages tends to be more attributable to the volume rather than the sweetener per se (Black et al., 1991, 1993). Equally, the complete removal of sucrose from baked goods is impossible without having a negative impact on the quality of the product (Luo, 2019). Sucrose in a baked product serves a number of functions, namely, to sweeten, retain moisture and extend the product shelf-life.

Consequently, the preponderance of evidence has utilised beverages as the vehicle of administration and evidence regarding the effects of HIS in reformulated solid matrices is limited (O'Connor et al., 2021).

Despite the use of HIS having increased in recent years (Sakurai et al., 2014; Tahmassebi & BaniHani, 2019) there remains much speculation regarding the effects of their ingestion on subsequent food preferences, particularly in light of evidence which suggests an increased risk of overweight and obesity (Azad et al., 2017; Bruyère et al., 2015). There exists a positive association between the intake of HIS and BMI (Fowler, 2016) resulting in speculation that HIS consumption may increase sweet preferences and intake (Yang, 2010). Given that repeated exposure conditions food preferences (Liem & De Graaf, 2004) this may be a valid concern. However, this is not to say that HIS intake directly causes increases in body fatness. Indeed, habitual users of HIS report greater concerns regarding their eating styles and body weight (Appleton & Conner, 2001), suggesting reverse causation, whereby individuals turn to HIS products to control energy intake and weight. Nonetheless, it is warranted to investigate the effects of HIS ingestion on subsequent preferences and food intake in order to inform policies and recommendations for individuals seeking to manage their energy intake.

Moreover, the reward elicited from HIS relative to sugars may be different (Delogu et al., 2016), with sugars potentially providing greater activation to reward related brain regions due to the energy-density of the product, which HIS lacks (Smeets et al., 2011). Moreover, repeated consumption of HIS has been demonstrated to produce a dissociation between energy and sweet taste, despite no difference in pleasantness ratings (Green & Murphy, 2012), generating concerns that habitual HIS may lead to greater energy intake (Appleton et al., 2004).

Conversely, in participants categorised as high or low sweetened beverage consumers, there was no effect of sweetener type on liking ratings, but a significant difference in habitual consumption of sweetened beverages. High consumers of sweetened beverages reported greater liking for increasing sweetness concentrations (Mahar & Duizer, 2007) - thus sweetener type may be of less importance than general consumption of sweet food and drinks, when influencing preferences. Supporting this notion, is evidence provided via a 12-month intervention, in which consumption of HIS beverages or SSB did not result in any change in preferred sweetness concentration, whereas consumption of an unsweetened beverage resulted in a significant decline (Ebbeling et al., 2020). Similarly, in a small sample, following exclusion of all added sugars and HIS for 2 weeks 95% of responders reported that sweet food and drink tasted either sweeter or too sweet (Bartolotto, 2015). Therefore, it is important to understand not only the impact of repeated exposure of HIS foods on sweet preferences, but to also consider the habitual consumption of sweet foods on subsequent sweet preferences.

The most recent recommendations regarding HIS use in weight management has been provided by the World Health Organisation, stating that HIS should not be used as a means of achieving weight control or reducing the risk of noncommunicable diseases (World Health Organization, 2022b). This recommendation is made after a systematic review identified no observable long-term benefits to weight from the inclusion of HIS in the diet, as well as identifying potentially undesirable long-term effects in increased risk of type 2 diabetes mellitus, cardiovascular diseases and mortality in adults (World Health Organization et al., 2022). Noted in this report is that short-term weight loss is not considered a positive outcome, as weight loss must occur over the long-term to improve health outcomes. Similarly, paediatricians express concerns regarding their use in child populations, highlighting potential interferences with gut regulatory mechanisms, mimicking dopaminergic reward pathways and safety of long-term consumption over the lifespan (Baker-Smith et al., 2019).

Moreover, HIS products fall under the category of ultra-processed foods due to the use of HIS as a cosmetic additive (Monteiro et al., 2019). Ultra-processed foods according to the NOVA food classification system are not only modified or processed foods, but are industrial

formulations manufactured from substances derived from foods containing a variety of additives and limited intact food (Monteiro et al., 2019). These foods are specifically designed to be extremely palatable and convenient and are sold in large portions (Moodie et al., 2013). Ultra-processed foods contribute more than half of total dietary energy intake in the UK (Rauber et al., 2019), partly because these foods are high-energy-dense products, with high levels of sugar and fats and simultaneously low levels of fibre, protein or vitamins (Louzada et al., 2018; Moubarac et al., 2017; Rauber et al., 2019). This is problematic as increased intake of ultra-processed foods results in a deterioration of the overall quality of the diet (Julia et al., 2018; Martínez Steele et al., 2016, 2018). However, the reformulation of already energy-dense foods may provide a means of facilitating weight loss (O'Connor et al., 2021).

1.8 Thesis Aims

Currently it remains unknown precisely what causes excess intake and weight gain, although an elevated preference for sweet foods has been postulated as one potential cause. However, there exists a lack of clarity as to how this preference may predispose some individuals and not others to increases in body weight. Furthermore, there exists disagreement in the literature surrounding the use of HIS within the diet as a means of managing weight. The aim of the present thesis is to expand upon the current knowledge on the association between sweet food preferences and factors contributing to appetite control. As outlined, the intake of palatable sweet foods is capable of contributing to an elevated energy intake and subsequently rates of individuals with overweight and obesity are increasing globally. In order to protect against a further increase in body fatness, as well as facilitate a reduction in energy content in the diet, the present thesis will provide an examination of sweet food preferences before a weight loss trial against a lean control group, and during a dietary induced weight loss trial. This will be followed by an independent analysis using data collected via a multisite cross-over trial comparing the effects of three sweetened products using sucrose and two novel HIS blends. The effects of acute and repeated exposure will investigate the impact of three different sweeteners (a natural HIS, an artificial HIS and sucrose) on subsequent reward (liking and wanting) for sweet food, with an exploration into differences in responses between sub-groups.

Displayed in Figure 1.4 is a schematic of the thesis aims by chapter, with the corresponding study that will be utilised to achieve these aims.

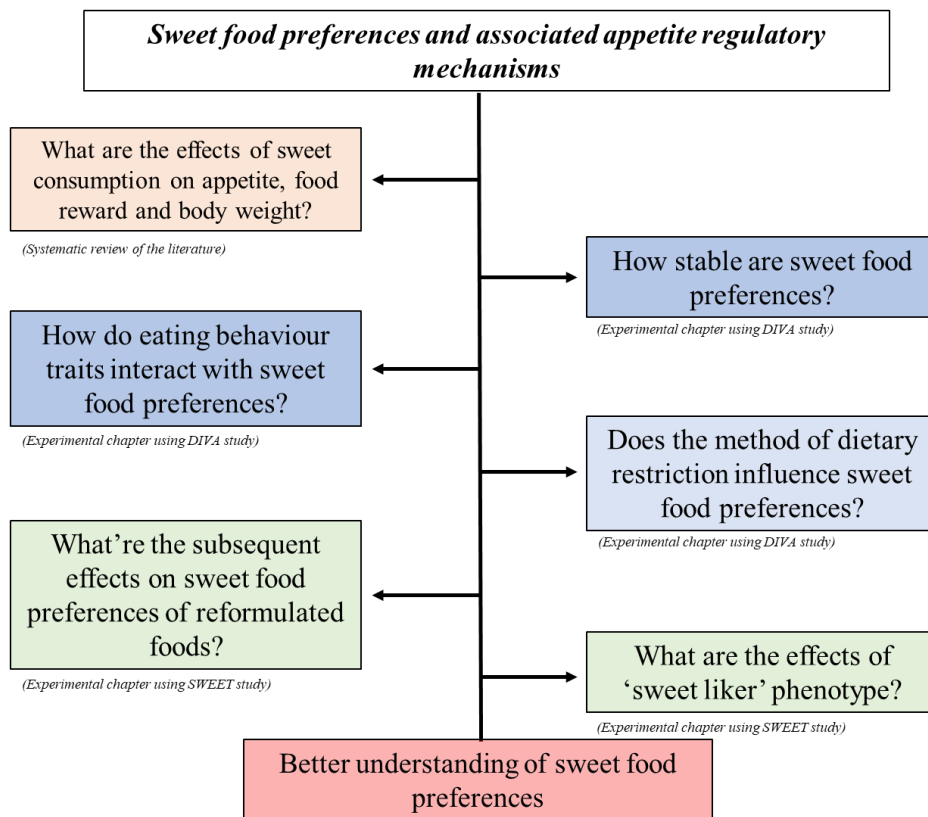


Figure 1.4. Schematic of thesis aims divided by chapter and study.

1.8.1 Specific Objectives

1. Systematically review the literature comparing the effects of HIS against a sucrose and/or water control on appetite, food reward and body weight in randomized controlled trials (Chapter 2).
2. Investigate the stability of sweet food preferences during a single day and across a period of diet induced weight loss (Chapter 4).
3. Investigate the role of sweet food preferences and eating behaviour traits in female participants across a range of BMIs (Chapter 5).
4. Compare the differences in effects on sweet food preferences of alternate day fasting and continual calorie restriction following significant weight loss (Chapter 6).
5. Investigate the effects of acute and repeated exposure to three sweeteners on sweet food preferences and associated eating behaviours (Chapter 7).

6. Explore differences between sweet liker phenotype groups on the effects of acute and repeated exposure to three sweeteners on sweet food preferences and associated eating behaviours (Chapter 8).

2 A systematic review on the differences between high intensity and caloric sweeteners on appetite, food reward and body weight.

Aims: The objective of the current chapter is to establish the effects of sweet consumption on appetite, food reward and body weight related outcomes, through a systematic examination of RCTs comparing high-intensity sweeteners with either, or both, a sucrose or water control comparison, on the aforementioned outcomes. This is a necessary provision to establish potential differences in the effects of different sweetener types, to inform the development of hypotheses for the proceeding experimental chapters and will address the first objective listed in section 1.8.1.

Key findings:

- High-intensity sweeteners (HIS) do not impact subjective appetite, food intake or appetite-related biomarkers.
- Definitions and assessment methods of food reward are inconsistent, requiring standardisation in future work.
- HIS inclusion in the diet facilitates a reduction of sugar, carbohydrates and energy intake in individuals with overweight and obesity, whereas lean individuals demonstrate a degree of compensation and hold net energy intake constant through an increase in protein and fat intake.
- Substitution of a SSB with a HIS beverage at a meal does not affect intake of the meal, but reduces calories ingested via the concomitant beverage.
- Evidence of HIS effects in foods is currently lacking.
- Data remains insufficient to perform a meta-analysis on subjective hunger ratings.

2.1 Introduction

2.1.1 High intensity sweeteners

Public concern surrounding food quality and composition has been increasing (Gregory, 2000) in an attempt to minimise the risk of non-communicable diseases (Rimal et al., 2001; Wandel & Fagerli, 2001). The sugar content of the diet has received particular scrutiny; UK guidelines

recommend an upper limit of no more than 5% of total energy intake to be obtained from free sugars within the diet (those added during manufacturing) (Azaïs-Braesco et al., 2017; Great Britain: Scientific Advisory Committee on Nutrition, 2015). Few individuals currently meet this ambitious target (Bennett et al., 2018) and pressure is being placed onto food manufacturers to provide alternative reduced sugar, or no added sugar, products to lessen the energy density of the diet whilst enabling consumers of sweetened products to maintain a palatable diet.

HIS provide the opportunity to reduce the energy density of a number of products, particularly beverages, whilst maintaining the sweet taste desired by consumers. The use of products sweetened using HIS has increased in recent years (Sakurai et al., 2014; Tahmassebi & BaniHani, 2019) partly motivated by weight monitoring goals (Pielak et al., 2019). It may seem intuitive that HIS use has the potential to facilitate weight loss, or weight maintenance targets, due to a lower energy density (Miller & Perez, 2014; Rogers et al., 2016). Nevertheless, some individual studies suggest an increased risk of overweight and obesity (Azad et al., 2017; Bruyère et al., 2015), others demonstrate a reduction in weight (Maersk, Belza, Stødkilde-Jørgensen, et al., 2012; Tate et al., 2012) whereas others yet suggest no change (Bonnet et al., 2018; Kuzma et al., 2015). The source of the current disagreement surrounding the precise effects of HIS may be due to variations within methodology; type of HIS, dose, length of study, frequency of dosing and study population, which may be capable of influencing the anticipated outcomes and will be examined within the current review.

2.1.2 Types of low-calorie intense sweeteners

Presently there are a number of distinct HIS types approved for human consumption (see Table 2.1). The majority of research has been conducted using beverages for administration, however, it is important to consider alternative modes of ingestion due to differences in post-ingestive consequences between beverages and solid foods (Almiron-Roig et al., 2003) resulting from variations in dietary fibre (Warrilow et al., 2019), macronutrients (Rolls, Hetherington, & Burley, 1988) or texture (Mattes, 1996), which may impact outcomes of interest.

HIS are defined as substances with a sweetness profile at least 30 times greater than that of table sugar (sucrose) (Hutchinson et al., 1999) and subsequently much smaller amounts are required to achieve the desired sweetness level (see Table 2.1). HIS are not all processed in an equivalent manner by the body, some bulk sweetening agents such as isomalt and tagatose, whilst not entirely free from energy content, do not produce a pronounced thermic effect following ingestion and subsequently are not metabolised sufficiently to contribute to net energy intake (Buemann et al., 1998). Due to differences in how HIS and other sweetening agents are processed by the body, it is possible that their effects on the aforementioned outcomes of interest are not equivalent and so consideration must be given to different types of sweeteners and the potential differences they produce.

Table 2.1. List of approved HIS in Europe from the European Food Safety Authority

Name	Food Additive Code	Type of Sweetener	Sweetness Index*
Acesulfame-K	E950	Artificial	200
Aspartame	E951	Artificial	180
Cyclamate	E952	Artificial	40
Saccharin	E954	Artificial	300
Sucralose	E955	Artificial	600
Thaumatococin	E957	Sweet protein	2,000
Neohesperidine DC	E959	Artificial	1,000
Steviol glycosides	E960	Chemical compound	480
Neotame	E961	Artificial	8,000
Aspartame-acesulfame salt	E962	Artificial	
Advantame	E969	Artificial (aspartame analogue)	20,000

*Note: Sweetness Index is a comparison relative to sucrose, e.g., a sweetness index value of 100 would be 100 times sweeter than sucrose.

2.1.3 The role of the sweet taste in obesity

Given that the sweet taste in nature is indicative of an ample energy source (Tan & Tucker, 2019) and possesses a powerful hedonic drive, it has been proposed to be an important contributor towards excess weight gain (Te Morenga et al., 2012). The inclusion of a sweetener

– either caloric or non-caloric – serves to increase the palatability of a food item, consequently, increases in subjective palatability tend to be paralleled by increases in intake through larger portion sizes (de Castro, 2000). Moreover, habitual consumption of a taste increases future preference for that taste, which may result in an increased intake (Appleton & Blundell, 2007) – as demonstrated with sugar use (Jemel et al., 1996), although whether this remains true when the associated energy content is removed remains to be established.

2.1.4 Effects of HIS on appetite-related outcomes

Presently there remains a lack of clarity regarding the specific effects of HIS on appetite (Black et al., 1993; Mattes & Popkin, 2009; Rogers et al., 1990; Tordoff & Alleva, 1990a) although improved assessment techniques present the opportunity to quantify different aspects of appetite such as the satiety cascade. Concerns exist that energy saved by the reduction in sugar could be compensated for by an increase in appetite and lead to increased food or energy intake (Anderson et al., 2012). Early evidence has shown that in normal weight, non-dieting participants, consumption of a low-calorie food product did not result in a significantly lower 24-h energy intake (King et al., 1999; Rolls et al., 1989), which has been confirmed in more recent research (Farhat et al., 2019). This may negate the potential benefits of using HIS instead of sugar when attempting to reduce total energy content of a diet.

Moreover, early evidence suggested that there may be a short-lived suppression of subjective appetite following acute ingestion of a beverage sweetened using a blend of HIS (saccharin, aspartame or acesulfame-K) (Rogers et al., 1988). However, more recently motivation to eat was demonstrated to be unaltered following consumption of a commercially available beverage (aspartame, acesulfame-K and sucralose) (Fantino et al., 2018) and so the influence of HIS on appetite may not be equivalent and examination of different components of appetite may contribute to explaining current incongruence within the literature.

2.1.5 Effects of HIS on food reward

Hedonic processes involved in the ingestion of food interact with the homeostatic appetite system in the regulation of energy intake (Finlayson et al., 2007b). At present, it remains

unclear if this holds true when the associated energy content is removed, as neuroimaging studies have indicated that the human brain is capable of differentiating between caloric (i.e. sugars) and non-caloric (i.e. HIS) sweet tastes (Chambers et al., 2009; Frank et al., 2008; Haase et al., 2009). Moreover, a substantial reduction of simple sugars within the diet, produces an increase in perceived sweetness intensity whilst leaving sweet pleasantness unaltered (Wise et al., 2016). It remains unknown whether this occurs as a function of the reduced sugar intake or reduced sweet perception and this warrants further consideration. On the other hand, as outlined, habitually consumed items become liked and preferred over initially equivalent or initially preferred alternatives (Mela, 1999) which may result in HIS encouraging sweet cravings and sugar intake precisely because of the sweet taste (Bello et al., 2018). For this reason, it remains important to consider the acute effects of HIS on food reward and any subsequent effects later in the day, following both acute and repeated ingestion, as these may not be equivocal. It is also necessary to understand the effects of HIS ingestion on food reward in order to establish whether their inclusion in the diet encourages further energy intake.

2.1.6 Effects on body weight-related outcomes

Despite a wealth of research, evidence for the effectiveness of HIS use on body weight-related outcomes remains unclear – possibly in part because their effects on related outcomes of interest remains uncertain also. Concern exists due to the correlation between HIS use and the incidence of overweight and obesity (Fowler et al., 2008; Mossavar-Rahmani et al., 2018; Sylvetsky et al., 2017). For example, early evidence showed an increased risk of weight gain at one year follow up regardless of baseline weight (Stellman & Garfinkel, 1986). However, it is not possible to infer causal inferences from observational studies (Hill, 1965) and the possibility of reverse causation cannot be ruled out, with individuals turning to products with HIS in attempts to reduce their energy intake and body weight, rather than these products leading to an elevated BMI. Therefore, it is necessary to examine evidence provided via randomised controlled trials (RCTs) in order to identify variables that may explain weight gain in studies such as this.

To understand the effects of HIS on body weight it is necessary to first understand how changes in body weight occur through the consequences of changes to appetite and food reward and their subsequent effects on food intake. For this reason, it is important to understand the potential effects of HIS on outcomes relating to appetite and food reward as presently, disagreement remains surrounding the precise effects of HIS inclusion in the diet.

2.1.7 Aims of the present review

The overarching aim of the present review was to disclose the effects of HIS versus sugar and/or water controls on outcomes related to appetite, food reward and body weight.

Consideration of the various HIS types currently in use in the diet emphasises the requirement for distinguishing between the effects of different HIS types from sugar and water to isolate the effects of sweetener type. No review has to date attempted to review appetite and food reward as mechanisms which could explain the currently high levels of incongruence within the literature surrounding the effectiveness of HIS ingestion to assist weight-based goals. Any changes to body weight or composition must be preceded by alterations to energy balance which may be due to changes in appetite and food reward impacting eating behaviours. It is therefore the aim of the present review to provide a unified examination of appetite and food reward so that the impact on body weight related outcomes may be better understood.

Moreover, the review is novel in its approach to examining HIS types, with the aim of providing an investigation into potential differences in outcomes of distinct sweetener types and distinguishing the influence of sweet taste and energy content. Finally, the review also aimed to provide a meta-analysis of pre- and post-prandial subjective hunger ratings which was not possible due to a lack of unification in timing of subjective ratings, sweetener type and dose as well as study population employed.

2.2 Methods

This systematic review was conducted following the preferred reporting items for systematic reviews and meta-analysis (PRISMA) guidelines and the protocol was registered in the PROSPERO database (Registration Number RD42020176603). Ethical approval was not required for this research.

2.2.1 Literature search

A comprehensive literature search using four databases (Cochrane Library, Medline (Ovid), Embase (Ovid), and PsycInfo (Ovid)) was conducted to identify randomized controlled trials (RCT) published up to November 2019, with no lower date limit set. The search strategy was organised into five categories of terms (sweetener, sugar, appetite, food reward and body weight outcomes) with previous systematic reviews screened to identify and inform these categories and the key words used in each (Beaulieu et al., 2016; Oustric et al., 2018; Toews et al., 2019; Wanders et al., 2011). The complete list of key words can be found in Table 2.2. Limits were set to only include papers published in the English language and in human populations.

Table 2.2. Table of search terms by category.

Sweetener	Sugar	Appetite	Food reward	Body weight outcomes
Aspartame	Monosaccharide	Hunger AND Hungr*	Liking	Fat mass AND FM
Stevia	Disaccharide	Appetite	Wanting	Fat free mass AND FFM
Natural caloric sweeteners	Oligosaccharides	Eating	Palatab*	Body weight
Artificial sweeteners	Dextrose	Eating behaviour	Food preference*	Body mass
Natural non-caloric sweeteners	Fructose	Food intake	Liking for food	Body composition
Acesulfame K	Lactose	Ingestion	Pleasure-giving value of food	Obes*
Advantame	Maltose	Food consumption	Pleasure/food	Overweight
Brazzein	Galactose	Dietary intake	Hedonic value of food	Normal weight
Cyclamate	Soft drinks		Food hedonics	Lean
Neotame	Soda	Prospective food consumption	Affective pleasure	Healthy weight
Saccharin	Fruit drinks	Desire to eat	Sensory perception of food	Weight loss
Sucralose	Sport drinks	Fullness	Food enjoyment	Weight reduction
Steviol glycosides	Sweetened ice tea	Ingestion	Consummatory reward	Weight management
Thaumatococin	Squashes	Caloric intake	Wanting for food	Weight maintain*
Neohesperidin	Lemonade	Eating behav*	Incentive motivation	Weight control
Alitame	Syrup	<i>Ad libitum</i> intake*	Disposition to eat	Prevent* weight regain
Rebaudioside	Honey	Satie*	Drawn to food	Weight maintenance
Rebiana	Lactose	Satiation	Motivational drive to eat	Energy balance
Brazzein	Candy	Gut hormone*	Incentive salience	Energy restriction
Mogrosin	Molasses	Gut peptide*	Motivation for food	Negative energy balance
Non-calorie sweetener	Carbonated beverages	Peptide YY OR PYY	Motivation to eat	BMI
Non-sugar	Carbonated drink	Ghrelin	Drive to eat	Weight manag*
Non-nutritive sweetener	Confectionary	Glucagon-like peptide-1 OR GLP-1	Food craving	

Sweetener	Sugar	Appetite	Food reward	Body weight outcomes
Sugar substitute	Sugar intake	Pancreatic peptide OR PP	Food-related motivation	
High intensity sweetener	Sugar sweetened beverages	Leptin Insulin Cholecystokinin OR CCK	Anticipatory reward Food reward Hedonic Hedonic driven eating Hedonic hunger Food reinforcement Relative reinforcing value Hedonic eating Hedonically driven Reward driven eating Food responses Response to food cues	

Abbreviations: FM – fat mass. FFM – fat-free mass. BMI – body mass index. CCK – cholecystokinin. PP – pancreatic peptide. GLP-1 – glucagon-like peptide-1.

2.2.2 Inclusion criteria

Inclusion and exclusion criteria were predefined and developed based on the study's suitability to address the research question of this review. Studies were included if they comprised of healthy individuals, including both adults (≥ 18 years of age) and children/adolescents (< 18 years).

There was no limit applied to body weight or BMI categorisation. Studies including participants living with overweight and/or obesity undertaking dietary interventions to lose weight were not excluded.

Interventions and exposures of interest consisted of any type of HIS as the experimental condition, with either a caloric sweetener or water as a control comparison. Studies which used a blend of different HIS, or provided reformulated foods with HIS and did not specify the type or dose, were not excluded. No vehicle of administration was specified. RCTs were eligible for inclusion if they met the following pre-specified criteria: 1) study population was apparently healthy (i.e. no metabolic-related disorders and an absence of mental illness or an eating disorder diagnosis); 2) included a HIS and control condition comparison consisting of either a

nutritive sweetener or water; and 3) examination of at least one of the outcomes of interest (appetite, food reward or body weight). Studies consisting of pregnant or lactating women, in vivo or animal studies were excluded. No minimum or maximum study duration was set. Each abstract and full-text article was assessed for eligibility independently by two authors; uncertainty regarding eligibility was discussed between the lead author and co-authors to reach an agreement.

2.2.3 Data extraction

Following abstract and full-text screening, one author extracted the following information from each RCT into an Excel spreadsheet: first author, year of publication, sample size and demographics (i.e. mean age and BMI), type of sweetener, dose and mode of administration for both HIS and nutritive controls, outcome measures, setting, study length and results. Studies that provided more than one experimental condition (i.e. examined multiple HIS types) or multiple control conditions (i.e. multiple nutritive sweeteners and/or water) were noted and entered separately (see Table 2.4).

2.2.4 Outcomes

As the method of assessment for each outcome of interest was not consistent across studies, the results were presented in a qualitative synthesis. Appetite outcomes included subjective appetite ratings, appetite-related biomarkers and food intake – both *ad libitum* and free-living. Food reward outcomes included subjective ratings (e.g., liking, pleasantness, taste intensity), food appeal or preference and neuroimaging. Body weight outcomes included body weight, BMI, waist and hip circumference and body composition.

2.2.5 Risk of bias

Risk of bias was assessed through use of GRADE (Guyatt, 2008) for sequence generation, allocation concealment, blinding of participants, personnel and outcome assessors, incomplete outcome data, selective outcome reporting and other sources of bias. Study inclusion was not influenced by the results of the risk of bias assessment.

2.3 Results

Figure 2.1 details the study selection process. Database searches yielded 1,406 results, application of filters (human participants and English language) produced 752 results, removal of duplicates produced 506 eligible for title and abstract screening. Following abstract and full text screening by independent reviewers, 58 separate studies were identified for inclusion in the review.

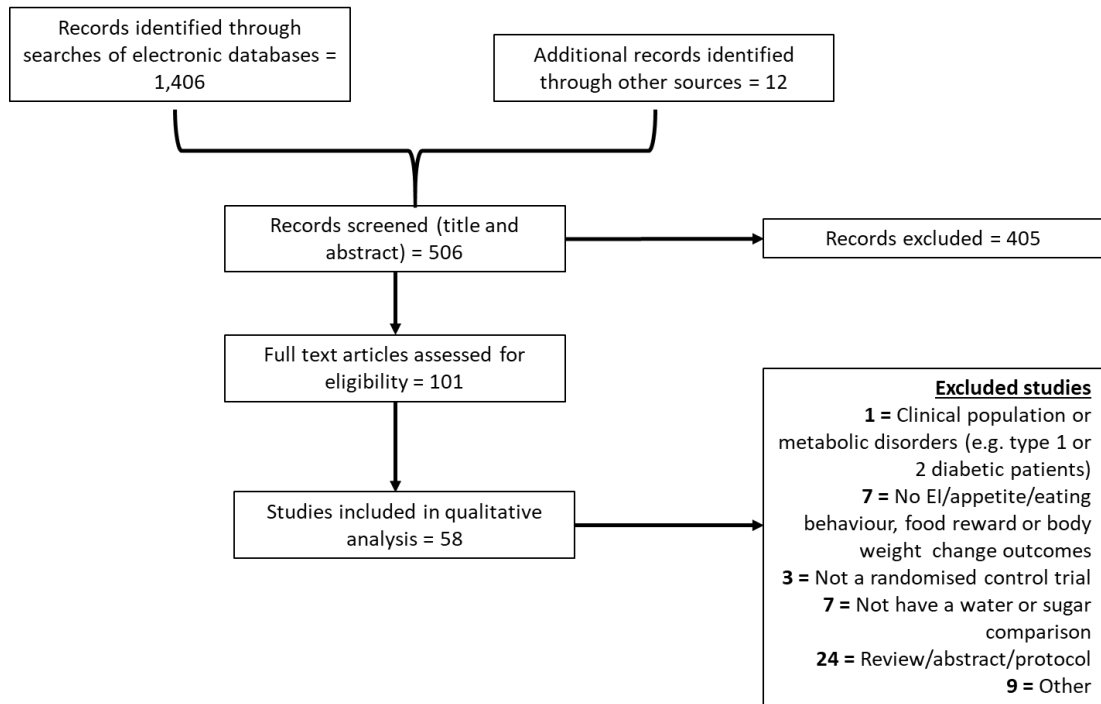


Figure 2.1. Study selection flow chart.

2.3.1 Risk of Bias

Results of the risk of bias assessment can be seen in

Table 2.3. Overall evidence was rated as either high quality ($n=396$) or moderate quality (1,902). A small number of studies ($n=159$) were rated as very-low quality evidence.

Table 2.3. GRADE risk of bias assessment outcomes.

No. of patients (studies)	Design	Limitations	Inconsistency	Indirectness	Imprecision	Publication bias
High quality of evidence						
396	RCT	No serious limitations	Not relevant	No serious indirectness	No serious imprecision	Unlikely
Moderate quality of evidence						
1902	RCT	Minor limitations*	Not relevant	No serious indirectness	No serious imprecision	Unlikely
Low quality of evidence						
304	RCT	More than one minor limitation*	Not relevant	No serious indirectness	No serious imprecision	Unlikely
Very-low quality of evidence						
159	RCT	Numerous limitations*	Not relevant	No serious indirectness	No serious imprecision	Unlikely

1 One limitation in the quality of evidence rating due to i) none-randomised groups, ii) single or unblended procedures or iii) high rate of drop out

2 Two limitations in the quality of evidence rating due to i) none-randomised groups, ii) single or unblended procedures or iii) high rate of drop out

3 Three or more limitations in the quality of evidence rating due to i) none-randomised groups, ii) single or unblended procedures or iii) high rate of drop out

2.3.2 Study and Participant Characteristics

Details of the participant characteristics are displayed in Figure 2.2 and Table 2.4, and the variety of HIS types, comparators and doses utilised in each study in Table 2.4. As the present review provides a focus on differential effects of sweetener types, some studies are presented multiple times. 36 studies followed within-subjects designs and 20 between-subjects designs. 36 studies were acute whereas 19 were repeated ingestion studies (ranging from 7 days to 18 months) – with the notable exception of one study which was acute but also involved repeated ingestion during the study session (Griffioen-Roose et al., 2013).

Underweight participants were examined in one study (mean BMI 17.1 kg/m²), 29 studies utilised healthy weight participants, 17 studies examined participants with overweight and five studies employed a study population with obesity. three studies used children, 42 studies were conducted in adults 11 studies did not provide a mean age, rather providing a range.

Evidence was examined through a comparison of studies utilising the same, or similar HIS types and blends. The HIS types most consistently investigated were acesulfame-K (5 studies), aspartame (25 studies) and sucralose (13 studies); all other HIS types studied were considered under the labels ‘HIS blends’ or ‘other HIS types’.

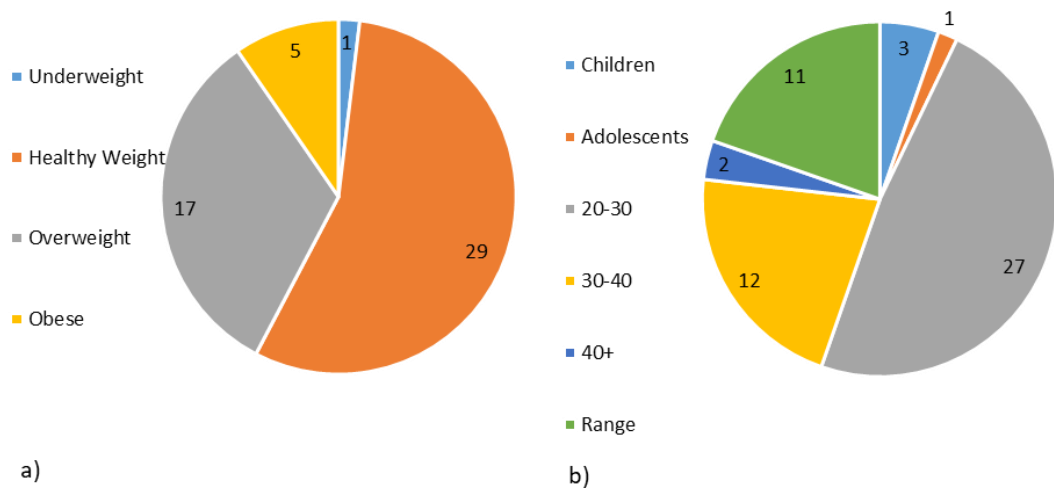


Figure 2.2. The number of studies conducted in differing a) BMI groups (left) and b) age (right) groups.

2.3.3 Effects of HIS on Appetite

42 studies included a relevant appetite-related outcome (subjective appetite ratings, appetite-related biomarkers, *ad libitum* or free-living energy intake), with 35 noting a significant difference in the effects of HIS compared to sugar or water controls and 7 reporting no significant difference in effects. 24 studies included subjective appetite ratings, 17 included laboratory meals (*ad libitum* intake, universal eating monitor, self-selected meals or pre- and post-weighed meals in a metabolic unit), 9 included free-living intake and 13 included measures of biomarkers (e.g., glucose, insulin, ghrelin or PYY). 26 studies were acute and 16 were repeated ingestion (ranging from 7 days to 18 months). Results are presented in Table 2.4.

Table 2.4. The effects of acute or repeated HIS ingestion, compared to sugar or water controls on subjective appetite ratings, appetite related biomarkers, *ad libitum* intake and free-living intake.

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Acesulfame-K								
Acute								
Steinert et al., (2011)	n: 12 Age (years): 23.3 (0.7) BMI (kg/m ²): 23.0 (0.5) % Female: 50	Sucralose (62mg) Aspartame (169mg) Acesulfame K (220mg)	Glucose (25g) Fructose (50g) Water	VAS ratings recorded at 0, 15, 30, 60, 90 and 120minutes GLP-1 measured by ELISA kit PYY, ghrelin, measured by commercially available RIA kit Glucose measured by glucose oxidase method	No difference in appetite ratings across HIS or water conditions	Significant increase GLP-1, PYY and glucose, with reduction in ghrelin in sugar conditions, but no effect in HIS conditions		
Meyer-Gerspach et al. (2018)	n: 12 Age (years): 23.00 (NR) BMI (kg/m ²): 23.00 (NR) % Female: 50	Acesulfame-K (220mg)	Glucose (50g) Fructose (25g)	VAS ratings of hunger and satiety scored every 5minutes for 180minutes	Increase in satiety and decrease in hunger in acesulfame-K condition, with steeper return to baseline values			
Rogers et al. (1988)	n: 12 Age (years): 19-25 BMI (kg/m ²): 21.3 % Female: 66	Saccharin (145mg) Aspartame (162mg) Acesulfame-K (240mg)	Glucose (50g) Water	VAS ratings recorded in 10minute intervals for 60minutes <i>Ad libitum</i> meal consumed 65minutes after preload ingestion	No difference in appetite ratings across conditions		Higher intake acesulfame-k compared to glucose condition	
Aspartame								
Acute								
Rodin et al., (1990)	n: 24 Age (years): 22-50 years BMI (kg/m ²): 18.5- + 29.9 % Female: 50	Aspartame (250mg)	Glucose (50g) Fructose (50g) Water	Pre-weighed lunch buffet ~50minutes after preload ingestion			Higher intake in aspartame condition compared to	

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Rolls et al., (1990)	n: 42 Age (years): 25.0 (4.3) BMI (kg/m²): NR % Female: 0	Aspartame (1,100mg or 2,200mg)	Sucrose (20g or 40g)	VAS ratings recorded at various times across conditions Preloads provided either with <i>ad libitum</i> meal or 30, or 60minutes before meal	No difference in hunger ratings across conditions		fructose condition Increased energy intake when sucrose beverage provided alongside meal	
Tey et al., (2017)a	n: 30 Age (years): 18-50 years BMI: 18.5-25kg/m ² % Female: 0	Aspartame (440mg)	Sucrose (65g) Water	Continuous glucose monitoring for 24hr		No difference in 24hr glucose levels across conditions	Increased intake in aspartame condition compared to sucrose – no test of significance reported	Increased intake in aspartame condition compared to sucrose – no test of significance reported
Tey et al., (2017)b	n: 10 Age (years): 26.2 (3.8) BMI (kg/m²): 21.2 (1.7) % Female: 0	Aspartame (440mg, 630mg or 330mg)	Sucrose (65g) Water	VAS ratings recorded at 15, 30, 45, 60, 90, 120, 150 and 180minutes <i>Ad libitum</i> meal provided 60minutes after preload (fried rice)	Increased ratings of 'desire to eat', 'hunger' and 'prospective consumption' in aspartame condition relative to sucrose condition, with no difference to other HIS types		Increased intake in aspartame condition relative to sucrose condition	No difference in intake across conditions
Black et al., (1991)	n: 20 Age (years): 19-25 years BMI (kg/m²): 22-29 % Female: 0	Aspartame (160-170mg or 320-340mg)	Water	Serial VAS appetite ratings recorded over 3hrs	No effect of sweetener condition – reduction in 'desire to eat' and increase in 'fullness' for larger beverage volume			

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Black et al., (1993)	n: 18 Age (years): 19-25 years BMI (kg/m ²): 21-25 % Female: 0	Aspartame (340mg) beverage or capsule	Water	Serial VAS appetite ratings recorded over 3hrs	No effect of sweetener condition – reduction in ‘desire to eat’ and increase in ‘fullness’ for larger beverage volume			
Rogers et al., (1998)	n: 12 Age (years): 19-25 BMI (kg/m ²): 21.3 (SD NR) % Female: 66	Aspartame (162mg) Saccharin (145mg) Acesulfame-K (240mg)	Glucose (50g) Water	VAS appetite ratings recorded in 10minute intervals for 60minutes <i>Ad libitum</i> meal consumed 65minutes after preload ingestion	Increase in hunger and desire to eat ratings in aspartame condition compared to water condition (no comparison to saccharine or acesulfame-K)		No difference in aspartame intake compared to glucose or water (no comparison to saccharine or acesulfame-K)	
Canty et al., (1991)	n: 20 Age (years): NR BMI (kg/m ²): NR % Female: NR	Aspartame (NR) Saccharin (NR)	Sucrose (NR) Water	VAS appetite ratings in 30min intervals between 0800 and 1400.	Only significant differences were found between sucrose and water conditions			
Steinert et al., (2011)	n: 12 Age (years): 23.3 (0.7) BMI (kg/m ²): 23.0 (0.5) % Female: 50	Sucralose (62mg) Aspartame (169mg) Acesulfame K (220mg)	Glucose (25g) Fructose (50g) Water	VAS ratings recorded at 0, 15, 30, 60, 90 and 120minutes GLP-1 measured by ELISA kit PYY, ghrelin, measured by commercially available RIA kit Glucose measured by glucose oxidase method	No difference in appetite ratings across HIS or water conditions	Significant increase GLP-1, PYY and glucose, with reduction in ghrelin in sugar conditions, but no effect in HIS conditions		
Repeated Ingestion								
Higgins et al., (2018)	n: 93 Age (years): 18-60	Aspartame (350mg or 1,050mg)	Dextrose (680mg)	Free living VAS appetite ratings on hourly basis for waking period of a day	No effect of aspartame on appetite ratings			

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
	BMI (kg/m²): 18.5-25 % Female: 54%							
Kuzma et al. (2015)	n: 10 Age (years): 21.0 (2.0) BMI (kg/m²): 22.7 (1.3) % Female: 55.56	Aspartame*	Glucose (25% EER) Fructose (25% EER)	Food was provided at 125% of estimated energy requirements and consumed <i>ad libitum</i> , with 25% (glucose and fructose conditions) or 4% (aspartame condition) mandatory consumption provided through sweetened beverages			Increased energy consumption in SSB conditions compared to aspartame condition	
Ballantyne et al. (2011)	n: 40 Age (years): 30-55 BMI (kg/m²): 25-35 % Female: 0	Aspartame (varied)	Sucrose (NR)	3 day food diary record over four separate weeks				No effect of aspartame consumption on intake
Reid et al., (2010)	n: 53 Age (years): 32.93 (8.84) BMI (kg/m²): 27.83 (1.83) % Female: 100	Aspartame* ² (4x daily)	Sucrose (105g)	7 day food diary for 5 weeks				Small increase in energy intake in sucrose condition at week 1 which reduced at week 4 Small increase in energy intake in aspartame condition at week 1 was non-significant No difference between

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Tordoff et al., (1990)	n: 30 Age (years): 28.2 (2.7) – females, 22.9 (0.8) - males BMI (kg/m²): 25.4 (1.4) – females, 25.1 (0.5) - males % Female: 30	Aspartame (590mg) (4x daily)	High fructose corn syrup (133g) No beverage	Food diaries completed continuously for 9 week period				conditions at week 4 Reduction in sugar intake in aspartame condition compared to HFCS condition
Porikos et al., (1982)	n: 6 Age (years): 39.8 BMI (kg/m²): NR % Female: 0	Aspartame sweetened foods (NR)	Conventional foods	Energy intake recorded during in-patient stay at metabolic unit Diet energy density was covertly reduced by 40%			Aspartame consumption increased food consumption, but energy intake remained below baseline (85%)	
Reid et al., (2007)	n: 133 Age (years): 31.8 (9.1) BMI (kg/m²): 22.5 (2.8) % Female: 100	Aspartame*	Sucrose (105g)	VAS appetite ratings completed before and after consumption of test drinks at 11:00, 14:00, 18:00 and 20:00	No effect of aspartame on appetite ratings			
Sucralose								
Acute								
Steinert et al., (2011)	n: 12 Age (years): 23.3 (0.7) BMI (kg/m²): 23.0 (0.5) % Female: 50	Sucralose (62mg) Aspartame (169mg) Acesulfame K (220mg)	Glucose (25g) Fructose (50g) Water	VAS ratings recorded at 0, 15, 30, 60, 90 and 120minutes GLP-1 measured by ELISA kit PYY, ghrelin, measured by commercially available RIA kit Glucose measured by glucose oxidase method	No difference in appetite ratings across HIS or water conditions	Significant increase GLP-1, PYY and glucose, with reduction in ghrelin in sugar conditions, but no effect in HIS conditions		

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Griffioen-Roose et al. (2013)	n: 40 Age (years): 21.0 (2.0) BMI (kg/m ²): 21.5 (1.7) % Female: 62.5	Sucralose (0.11g/l) Sucralose + Acesulfame-K (0.008g/l + 0.013g/l)	Sucrose (68.6g/l or 6.8g/l)	Drinks were provided for <i>ad libitum</i> consumption with covert assessment of intake			No difference in intake across sweetener conditions	
Sylvetsky et al., (2016)	n: 30 Age (years): 29.7 (7.6) BMI (kg/m ²): 25.8 (4.2) % Female: NR	Sucralose (68, 170 or 250mg) Sucralose + Acesulfame-K (68 + 41mg) Sucralose + Acesulfame-K + Aspartame (18 + 18 + 57mg)	Water	Hunger and satiety questionnaires completed at 0, 30, 60, 90 and 120mins Blood samples collected at -10, 0, 10, 20, 30, 60, 90 and 120mins	Hunger and satiety comparable across conditions – data not shown	No difference in GLP-1, GIP, glucose, insulin or C-peptide in sucralose condition		
Casperson et al., (2017)	n: 21 Age (years): 24.0 (6.0) BMI (kg/m ²): 23.0 (2.0) % Female: 52	Sucralose (4,000mg)	Sucrose (31g)	Satiety and desire to eat ratings assessed before and every 30mins post-meal for 4hr	No difference between SSB and HIS conditions on satiety or desire to eat AUC			
Chern & Tan (2019)	n: 11 Age (years): 24.9 (6.1) BMI (kg/m ²): 25.0 (4.7) % Female: 27	Sucralose (120mg) Sucralose + Maltodextrin (120mg + 50g)	Sucrose (50g)	VAS appetite ratings pre and post-consumption of test food (09:00) and pre and post- <i>ad libitum</i> meal	No difference between conditions		No difference between conditions	No difference between conditions
Van Opstal et al., (2019)a	n: 20 Age (years): 22.2 (1.3) BMI (kg/m ²): 22.4 (1.1) % Female: 0	Sucralose (50mg)	Glucose (50g) Fructose (25g)	Two VAS hunger, fullness and wanting a meal ratings ~45mins apart	No difference between conditions			
Wu et al., (2012)	n: 10 Age (years): 28.8 (4.0) BMI (kg/m ²): 25.5 (1.5) % Female: 30	Sucralose (60mg) Tagatose + Isomalt (40g)	Glucose (40g) 3-0-Methylglucose (40g)	VAS appetite ratings and blood samples taken at -25, -10, 0, 15, 30, 60, 90 and 120mins	Higher ratings of fullness in glucose, 30MG, and TIM conditions compared to sucralose condition	GIP, GLP-1 and insulin remained unchanged in sucralose condition		

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Gadah et al., (2016)	n: 144 Age (years): 26.2 (9.5) BMI (kg/m²): 22.9 (3.3) % Female: 50	Sucralose (500mg)	Sucrose (105g)	VAS ratings at 0, 5, 10, 15 and 20mins <i>Ad libitum</i> test meal of cheese sandwiches, ham sandwiches and yoghurt dessert provided	No difference in hunger, desire to eat or prospective consumption ratings Hunger increased over time significantly Sucralose condition non-significantly higher than sucrose condition ($p=.07$)		Sucrose condition reduced test meal energy intake compared to sucralose condition	
Maersk et al. (2012)a	n: 24 Age (years): 33.5 (9.2) BMI (k/m²): 31.4 (3.11) % Female: 50	Commercially available diet-cola	Commercially available cola Semi-skimmed milk Water	VAS ratings at 30min intervals for 4hr Blood samples at 0, 30, 60, 120, 180 and 240mins <i>Ad libitum</i> meal served 4hr after test drink	No difference in AUC ratings between HIS condition or water condition	No change in ghrelin, GLP-1, GIP, glucose or insulin in HIS condition or water conditions	No difference between conditions in energy intake	
Temizkhan et al., (2015)	n: 8 Age (years): 45.0 (4.1) BMI (kg/m²): 30.3 (4.5) % Female: 50	Sucralose (24mg) Aspartame (72mg)	Water	Blood samples at -15, 0, 15, 30, 45, 60, 75 90, 105 and 120mins		Earlier blood glucose peak when sucralose ingested alongside energetic load		
HIS Blends								
Acute								
Monsivais et al. (2007)	n: 37 Age (years): 22.6 (4.0) BMI (kg/m²): 23.4 (1.8) % Female: 48	Diet cola 475-525ml	HFCS (NR) Milk	VAS ratings in 20min intervals for 3hr 20min <i>Ad libitum</i> test meal provided 2hr 30min post test beverage	No difference in ratings between HIS condition and no beverage condition		No difference in intake between HIS condition and no beverage condition	
DellaValle et al. (2005)	n: 44 Age (years): 24.6 (0.5)	Diet cola (NR)	Cola (NR) 1% Milk (NR) Orange Juice (NR)	VAS ratings completed pre- and post-consumption of <i>ad libitum</i> test meal	No difference across beverage conditions in fullness ratings,		No difference between conditions in intake	

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
	BMI (kg/m²): 22.7 (0.2) % Female: 50		Water (NR)		but lower for a no-beverage condition			
Sylvetsky et al., (2016)	n: 30 Age (years): 29.7 (7.6) BMI (kg/m²): 25.8 (4.2) % Female: NR	Sucralose (68, 170 or 250mg) Sucralose + Acesulfame-K (68 + 41mg) Sucralose + Acesulfame-K + Aspartame (18 + 18 + 57mg)	Water	Hunger and satiety questionnaires completed at 0, 30, 60, 90 and 120mins Blood samples collected at -10, 0, 10, 20, 30, 60, 90 and 120mins	Hunger and satiety comparable across conditions – data not shown	No difference in GLP-1, GIP, glucose, insulin or C-peptide in sucralose condition		
Chern & Tan (2019)	n: 11 Age (years): 24.9 (6.1) BMI (kg/m²): 25.0 (4.7) % Female: 27	Sucralose (120mg) Sucralose + Maltodextrin (120mg + 50g)	Sucrose (50g)	VAS appetite ratings pre and post-consumption of test food (09:00) and pre and post- <i>ad libitum</i> meal	No difference between conditions		No difference between conditions	No difference between conditions
Smeets et al., (2011)	n: 10 Age (years): 23.3 (2.8)* BMI (kg/m²): 22.4 (2.0) % Female: 0	Aspartame + Acesulfame K + Cyclamate + Saccharin (11 + 5.8 + 1.5mg/100g) Aspartame (880mg)	Sucrose (10.4g) Maltodextrin (78.9g) Glucose (75g)	9-point scale rating fullness, desire to eat and desired amount to be eaten before and after fMRI scan	No difference in the increased fullness ratings across conditions			
Van de Ven et al., (1994)	n: 24 Age: 24-40 BMI (kg/m²): 23.00-29.00 % Female: 100	Cyclamate + Saccharin + Aspartame + Acesulfame K (260 + 11 + 34 + 28mg) Cyclamate + Saccharin (280 + 24mg)	Fructose (25g)	Hunger VAS ratings pre- and post-preload intake, pre- and post-lunch and every hour for 5hr after lunch	Lower hunger ratings in preload conditions than placebo condition at post-preload intake, post-lunch, and 1, 3 and 5hr post-lunch			
Creze et al., (2018)	n: 18 Age: NR BMI (kg/m²): NR % Female: 0	Cyclamate (137.2 + 78.4mg) Acesulfame K (63.35 + 36.2mg) Aspartame (40.6 + 23.2mg)	Sucrose (37.1g + 21.2g)	Blood samples at -60, 0, 30, 60, 90, 150 and 210mins <i>Ad libitum</i> buffet at the end of test day with 12 types of snacks		Higher insulin values and lower ghrelin values in sucrose condition compared to HIS and	Significantly less intake in sucrose than water of HIS conditions	

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Wu et al., (2012)	n: 10 Age (years): 28.8 (4.0) BMI (kg/m²): 25.5 (1.5) % Female: 30	Sucralose (60mg) Tagatose + Isomalt (40g)	Glucose (40g) 3-0-Methylglucose (40g)	VAS appetite ratings and blood samples taken at -25, -10, 0, 15, 30, 60, 90 and 120mins	Higher ratings of fullness in glucose, 30MG, and TIM conditions compared to sucralose condition No difference in hunger, desire to eat or prospective consumption ratings	water conditions GIP, GLP-1 and insulin remained unchanged in sucralose condition		
Repeated ingestion								
Bonnet et al., (2018)	n: 50 Age (years): 31.1 (10.3) BMI (kg/m²): 24.7 (3.2) % Female: 56	Aspartame + Acesulfame-K (129 + 13mg)	Water	Matsuda Insulin Sensitivity Index after an oral glucose load 12 week intervention		No difference between conditions in insulin secretion estimates		
Sorensen et al., (2005)	n: 41 Age (years): 33.4 (9.0) + 37.1 (10.0)*7 BMI (kg/m²): 28.0 (2.3) + 27.6 (2.1) % Female: 85	Sugar reduced foods	Sucrose sweetened foods	Blood samples taken at weeks 0 and 10 7 day food diaries completed 10 week intervention		Haptoglobin, transferrin, and CRP increased in sucrose condition and decreased in HIS condition		No difference between conditions in intake
Raben et al., (2011)	n: 11 + 12 Age (years): 35.5 (3.6) + 35.3 (2.8)*7 BMI (kg/m²): 27.6 (0.8) + 28.7 (0.7) % Female: NR	Sugar reduced foods	Sucrose sweetened foods	Blood samples taken at weeks 0 and 10 7 day food diaries completed 10 week intervention		Higher postprandial glucose and insulin response in sucrose condition than HIS condition		No change in intake in HIS condition, but 161% increase in sucrose in sucrose condition

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
De Ruyter et al., (2013)	n: 103 Age (years): 10.2 (0.8) BMI (kg/m²): 17.1 (2.2) % Female: 52	Sucralose + Acesulfame-K (34 + 12mg)	Sucrose (26g) Water	Satiety measured on 5-point scale at 0, 6, 12 and 18 months 18 month intervention	No difference between conditions on satiety ratings			
Markey et al., (2016)	n: 50 Age (years): 32.0 (9.8) BMI (kg/m²): 23.5 (3.0) % Female: 68	Sugar reduced foods	Sucrose sweetened foods	4 day weighed food diaries week 0 and 8 8 week intervention				Carbohydrate, sugar and non-milk extrinsic sugars reduced, with higher fat and protein intake in HIS condition
Fantino et al., (2018)	n: 166 Age (years): 18-45 BMI (kg/m²): 19.00-28.00 % Female: 48	Acesulfame-K + Aspartame + Sucralose (NR)	Water	<i>Ad libitum</i> food intake using plate waste method 1 day food diaries 5 week intervention			No difference in HIS condition and water condition in intake	No difference in HIS condition and water condition in intake
Other HIS types								
Acute								
Canty et al., (1991)	n: 20 Age (years): NR BMI (kg/m²): NR % Female: NR	Aspartame (NR) Saccharin (NR)	Sucrose (NR)	VAS appetite ratings in 30min intervals between 0800 and 1400.	Hunger ratings significantly lower in saccharin and aspartame condition than water, but higher than sugar condition			
Farhat et al., (2019)	n: 30 Age (years): 26.1 (10.56) BMI (kg/m²): 23.44 (3.42) % Female: 66	Stevia Extract (1,000mg)	Sucrose (60g)	VAS appetite ratings and blood samples collected at 30min intervals until 120min post-lunch	Lower hunger ratings in stevia condition than water, with no difference to sucrose condition	No effect	No effect	No effect

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Rogers et al., (1988)	n: 12 Age (years): 19-25 years BMI (kg/m²): 21.3 (SD NR) % Female: 66	Saccharin (145mg) Aspartame (162mg) Acesulfame-K (240mg)	Glucose (50g)	One day diet diary was collected on each test day VAS ratings recorded in 10minute intervals for 60minutes <i>Ad libitum</i> meal consumed 65minutes after preload ingestion			No difference in saccharin compared to glucose or water conditions	
Steinert et al., (2011)	n: 12 Age (years): 23.3 (0.7) BMI (kg/m²): 23.0 (0.5) % Female: 50	Sucralose (62mg) Aspartame (169mg) Acesulfame K (220mg)	Glucose (25g) Fructose (50g) Water	VAS ratings recorded at 0, 15, 30, 60, 90 and 120minutes GLP-1 measured by ELISA kit PYY, ghrelin, measured by commercially available RIA kit Glucose measured by glucose oxidase method	No difference in appetite ratings across HIS or water conditions	Significant increase GLP-1, PYY and glucose, with reduction in ghrelin in sugar conditions, but no effect in HIS conditions		
Tey et al., (2017)a	n: 30 Age (years): 18-50 years BMI (kg/m²): 18.5-25 % Female: 0	Aspartame (440mg)	Sucrose (65g) Water	Continuous glucose monitoring across 24hrs		No difference in 24hr glucose levels across conditions	Increased intake in aspartame condition compared to sucrose – no test of significance reported	Increased intake in aspartame condition compared to sucrose – no test of significance reported
Tey et al., (2017)b	n: 10 Age (years): 26.2 (3.8) BMI (kg/m²): 21.2 (1.7) % Female: 0	Aspartame (440mg, 630mg or 330mg)	Sucrose (65g) Water	VAS ratings recorded at 15, 30, 45, 60, 90, 120, 150 and 180minutes <i>Ad libitum</i> meal provided 60minutes after preload (fried rice)	Increased ratings of 'desire to eat', 'hunger' and 'prospective consumption' in aspartame condition		Increased intake in aspartame condition relative to sucrose condition	No difference in intake across conditions

Author, Year	Study Population	Sweetener	Comparator	Important methodology details	Subjective Appetite Ratings	Appetite Related Biomarkers	<i>Ad Libitum</i> Intake	Free-living Intake
Solomi et al., (2020)	n: 10 Age (years): 26.9 (3.3) BMI (kg/m²): 24.7 (1.1) % Female: 50	Aspartame + acesulfame-K (NR)	Glucose (25g) Carbonated water	Blood glucose measured using portable glucometers before and 10min after preload consumption and every 15min over 120min period	relative to sucrose condition, with no difference to other HIS types	Lower blood glucose in HIS condition than water at 120min only		

Abbreviations: NR – not reported. BMI – body mass index.

Key: Green – significant difference between HIS condition and comparator condition. Red – no significant difference between HIS condition and comparator condition. Grey – not included.

2.3.3.1 Acesulfame-K

Two acute studies examined the impact of acesulfame-K provided either via intragastric tube (Meyer-Gerspach et al., 2018) or consumed as a preload (Rogers et al., 1988) on subjective appetite ratings. One, a crossover study noted, a greater increase in subjective satiety ratings relative to water (Meyer-Gerspach et al., 2018), whilst one noted no effect on post-prandial subjective ratings compared to water (Rogers et al., 1988). Rogers et al., (1988) also examined *ad libitum* intake, observing a lower intake across all sweet preload conditions (saccharin, 145mg; aspartame, 162mg; acesulfame-K, 240mg), with greater energy intake following acesulfame-K ingestion than glucose.

2.3.3.2 Aspartame

Aspartame was the most frequently studied HIS, included in 25 studies, the sweetener types and doses, study population descriptive statistics and direction of effects are displayed in Table 2.4. Two acute studies noted an increased desire to eat and hunger following aspartame ingestion relative to sucrose ingestion (Rogers et al., 1988; Tey et al., 2017b). Four acute studies (Black et al., 1991, 1993; Canty & Chan, 1991; Steinert et al., 2011) and a 12-week repeated ingestion study (Higgins et al., 2018), reported no effect on subjective appetite relative to a water control, whereas an acute study (Rolls et al., 1990) and a 4-week repeated ingestion study (Reid et al., 2007) reported no difference to sugar controls.

Nine studies examined aspartame ingestion's effects on *ad libitum* intake, seven of which were acute (Black et al., 1991, 1993; Rodin, 1990; Rogers et al., 1988; Rolls et al., 1990; Tey et al., 2017b, 2017a), and two repeated ingestion studies, lasting 8 days (Kuzma et al., 2015) or 24 days (Porikos et al., 1982). Four studies reported no difference in *ad libitum* intake between aspartame and water conditions (Black et al., 1991, 1993; Rodin, 1990; Rogers et al., 1988) with three also reporting no difference between aspartame and sugar controls (Rodin, 1990; Rogers et al., 1988; Rolls et al., 1990) – one of which also reported reduced energy intake in a fructose condition (Rodin, 1990) and one which reported a greater energy intake in the sucrose condition when the energy content of the preload beverages was included (Rolls et al., 1990). One study reported a lower energy intake in an aspartame condition than a sucrose condition

(Rolls et al., 1990) with two studies reporting higher energy intake in aspartame than sucrose or water conditions (Tey et al., 2017b, 2017a) – although one did not report a test of significance (Tey et al., 2017b).

Four studies included free-living intake, two were acute (Tey et al., 2017b, 2017a) and two were repeated ingestion, lasting 3 weeks (Tordoff & Alleva, 1990) and 5 weeks (Reid et al., 2010) respectively. One acute study did not report a test of significance (Tey et al., 2017b), the second acute study reported higher rest of day energy intake in an aspartame condition relative to sucrose (Tey et al., 2017a). In a repeated measures study, a sucrose condition produced a greater energy intake (Reid et al., 2010) whereas consumption of high fructose corn syrup (HFCS) sweetened beverages produced lower energy intake (Tordoff & Alleva, 1990) – although when the energy content of the beverages were included this resulted in an elevated total energy intake.

In a repeated ingestion study, sucrose-sweetened beverages increased energy intake above baseline intake following one week of consumption which reduced by the fourth week of consumption relative to baseline (Reid et al., 2010). One study reported that 3-week ingestion of aspartame and HFCS sweetened beverages significantly reduced energy intake in the diet to the same extent, although when including the energy content of the beverages the HFCS condition resulted in a large increase in total intake (Tordoff & Alleva, 1990). One study reported a higher mean rest of the day energy intake following aspartame ingestion relative to sucrose ingestion (Tey et al., 2017a) and one study reported comparable mean total daily energy intake between aspartame and sucrose conditions (Tey et al., 2017b) – although neither reported tests of significance.

Seven studies reported no effects on appetite related blood biomarkers or peptides (glucose, insulin, ghrelin, GLP-1, GIP, PYY, glucagon) from acute aspartame ingestion (Maersk, Belza, Holst, et al., 2012; Rodin, 1990; Smeets et al., 2005; Steinert et al., 2011; Temizkan et al., 2015; Tey et al., 2017b, 2017a) and one reported no effects on blood glucose following 12-week repeated ingestion (Higgins et al., 2018).

2.3.3.3 Sucralose

Effects of sucralose ingestion on appetite were examined in 11 studies – all of which were acute and are displayed in Table 2.4. One crossover study reported no statistical differences between water, sucralose and sugar sweetened water provided via an intragastric feeding tube on appetite ratings (Steinert et al., 2011), one crossover study reported no differences between sucralose or commercially available diet beverages or water conditions (Sylvetsky et al., 2016) and three crossover studies reported no differences to sugar conditions (Casperson et al., 2017; Chern & Tan, 2019; Van Opstal et al., 2019). One crossover study noted lower fullness ratings following sucralose ingestion than sugar ingestion with no differences in hunger, desire to eat or prospective consumption (Wu et al., 2012) and one between-groups study noted reduced hunger in a sucrose condition relative to a sucralose condition (Gadah et al., 2016).

One between-groups study reported lower *ad libitum* energy intake following sucrose relative to sucralose ingestion (Gadah et al., 2016) and one crossover study reported no difference in *ad libitum* or total daily energy intake between sucralose and sugar conditions (Chern & Tan, 2019). Another crossover study reported a non-significant trend ($p=0.08$) for higher sweet snack food consumption after sucralose relative to a sugar sweetened beverage ingestion (Casperson et al., 2017).

Four studies demonstrated no effect on appetite related biomarkers or peptides. One provided sucralose (GLP-1, PYY, ghrelin, glucagon, glucose or insulin) (Steinert et al., 2011), three provided glucose (Sylvetsky et al., 2016; Temizkan et al., 2015; Wu et al., 2012), with two noting no impact on glucose, insulin, GLP-1, C-Peptide 4 (Sylvetsky et al., 2016; Temizkan et al., 2015), and one reporting no impact on glucose, insulin, GLP-1, GIP, (Wu et al., 2012). One acute crossover study reported an earlier blood glucose peak following sucralose ingestion in response to an oral glucose tolerance test than following a water control condition which did not impact the blood glucose response (Temizkan et al., 2015).

2.3.3.4 HIS Blends

The effects of HIS blends on appetite related outcomes are displayed in Table 2.4. Thirteen studies examined blends of HIS on appetite. An 18-month repeated ingestion study of a HIS

blend (sucralose & acesulfame-K, 34mg & 12mg) in children did not show any impact subjective appetite compared to sucrose ingestion (26g) or water (de Ruyter et al., 2013). In adults, two acute studies reported no difference in effects to water ingestion, one which was an acute crossover study examining insulin levels (Sylvetsky et al., 2016), and another 9-week between-groups study examining *ad libitum* energy intake (Fantino et al., 2018a). Three acute crossover studies reported no difference of HIS blends to sucralose or sucrose in isolation on subjective appetite ratings (Chern & Tan, 2019; Smeets et al., 2011; Wu et al., 2012) and one also noted no effect on *ad libitum* or free-living energy intake (Chern & Tan, 2019). One acute crossover study reported reduced subjective hunger across all conditions with no difference between conditions (cyclamate + saccharin + aspartame + acesulfame-K, 260mg + 11 mg + 34mg + 28mg; cyclamate + saccharin, 380mg + 24mg; fructose, 25g) (Van de Ven et al., 1994). One acute study reported no difference in *ad libitum* energy intake, or hunger and satiety ratings between commercially available diet cola or cola sweetened using HFCS – although there was a significantly higher energy intake for caloric beverages relative to the diet cola condition when the energy content of the beverage was also included (Monsivais et al., 2007).

One acute study noted no effect on *ad libitum* energy intake after HIS blends ingestion (Chern & Tan, 2019). One acute study reported lower *ad libitum* intake at a buffet style meal, as well as elevated insulin and glucose levels following sucrose ingestion relative to a HIS blend, with simultaneously lower ghrelin, as well as reduced hunger ratings (Crézé et al., 2018). One study reported no differences in energy intake between water and HIS blend conditions after acute and repeated ingestion (5 weeks) - although sugar intake decreased following acute ingestion in HIS naïve participants (those not habitually consuming HIS) (Fantino et al., 2018a).

One acute crossover study reported no effect on free-living energy intake following consumption of a HIS blend (Chern & Tan, 2019) as did one 9-week repeated ingestion study (Fantino et al., 2018a). One acute crossover study reported a higher total energy intake in sugar conditions when the energy content of beverages was included (Van de Ven, 1994). One 8-week repeated ingestion, crossover study which provided participants a diet consisting of reformulated products reported no effect on energy intake, as examination of macronutrient

intake highlighted a reduction in carbohydrate and sugar intake but an increase protein and fat intake (Markey et al., 2016). Two 10-week repeated ingestion studies noted increased sugar and carbohydrate intake in sugar conditions relative to HIS blends (Raben et al., 2011; Sørensen et al., 2005).

One acute crossover study reported elevated plasma insulin levels following sucrose ingestion relative to a HIS blend, but no difference between the HIS and water conditions (Crézé et al., 2018). One acute crossover study reported no effect on glucose, insulin, glucagon, PYY, ghrelin or GLP-1 following water or HIS blend ingestion (Steinert et al., 2011). One acute crossover study reported no difference in glucose, insulin or GLP-1 following HIS blend or water consumption (Sylvetsky et al., 2016). One acute crossover study reported no difference in glucose, insulin or GLP-1 following consumption of tagatose + isomalt compared to sucralose ingestion, but following tagatose + isomalt consumption there was a greater release of GIP following a meal (Wu et al., 2012). Two 10-week repeated ingestion studies reported increased post-prandial glycaemia and lipidaemia following sucrose ingestion but not HIS (Raben et al., 2011; Sørensen et al., 2005). One 12-week repeated ingestion study reported no effect on insulin sensitivity (Bonnet et al., 2018).

2.3.3.5 Other HIS Types

The effects of other HIS types (e.g., cyclamate, saccharin, stevia and monk fruit extract) on appetite-related outcomes can be seen in Table 2.4. Six acute studies (Canty & Chan, 1991; Farhat et al., 2019; Rogers et al., 1988; Solomi, 2020; Tey et al., 2017b, 2017a) and one 6-month repeated ingestion study (Tate et al., 2012) examined other forms of HIS. One acute, repeated measures study noted reduced subjective hunger following saccharin ingestion relative to water but not sugar conditions (Canty & Chan, 1991). One acute cross-over study reported no difference following ingestion of water or a saccharin solution (Rogers et al., 1988) and one acute study reported greater desire to eat, prospective consumption and hunger ratings with lower fullness ratings following monk fruit or stevia ingestion compared to sucrose (Tey et al., 2017b). One acute study reported significantly lower hunger and desire to eat ratings following consumption of stevia relative to water (Farhat et al., 2019).

One acute cross-over study reported a decreased *ad libitum* intake following saccharin and glucose ingestion (Rogers et al., 1988). Two acute studies reported higher *ad libitum* intake in monk fruit and stevia conditions relative to sucrose (Tey et al., 2017b, 2017a) – only one of these (Tey et al., 2017b) reported a test of significance.

One acute crossover study reported no difference in free-living energy intake between HIS and sugar conditions (Tey et al., 2017b), whereas another study noted an increased free-living energy intake following stevia ingestion relative to sucrose and monk fruit ingestion – although did not report a test of significance (Tey et al., 2017a). One 6-month repeated ingestion, between-groups study reported significant reductions in energy intake following both caloric and diet beverages with no differences between groups (Tate et al., 2012).

One acute study reported no difference between monk fruit and stevia in AUC glucose responses which were significantly lower than the response to sucrose (Tey et al., 2017a) and one study noted lower fasting blood glucose levels following 6-month daily ingestion of reformulated beverages similar to levels seen following repeated water ingestion (Tate et al., 2012). One acute, crossover study provided aspartame and acesulfame-K (commercially available beverage) or carbonated water 10-min prior to a 25-g glucose load and reported no significant difference in glycaemic response until 120-min where a small but significant drop below baseline values was observed in the commercial beverage condition (Solomi, 2020).

2.3.4 Effects of HIS on Food Reward

18 studies included a food reward-related outcome. Food reward related outcomes included neuroimaging techniques, food appeal or preference, subjective ratings (e.g., liking, wanting or pleasantness, sweetness intensity ratings, a relative reinforcing value task and a lexical decision making task). No results tables were created for the effects on food reward due to differences in methodology used to assess and operationalised definitions on food reward.

2.3.4.1 Acesulfame-K

Three acute studies examined acesulfame-K on food reward. Acesulfame-K displayed a sigmoidal dose-response on sweetness intensity ratings in one study (Wee et al., 2018),

received a lower intensity rating than sucrose in another (Kamerud & Delwiche, 2007) and in a third study, acesulfame-K reduced the number of foods checked in a food preference checklist that were high in protein (Rogers et al., 1988).

2.3.4.2 Aspartame

Five acute studies examined the effect of aspartame on food reward (Black et al., 1991, 1993; Rogers et al., 1988, 1990; Tey et al., 2017a) - one acute repeated measures study noted no difference in pleasantness ratings when comparing aspartame and sucrose solutions (Rogers et al., 1990). Aspartame was rated as less bitter and sweeter than monk fruit or stevia in one study (Tey et al., 2017a). Two studies noted no effect on subjective food appeal following aspartame ingestion relative to water ingestion (Black et al., 1991, 1993) and one study noted an increase in savoury food appeal following aspartame ingestion relative to water or glucose ingestion (Rogers et al., 1988).

2.3.4.3 Sucralose

Six acute studies examined the effect of sucralose on food reward (Casperson et al., 2017; Frank et al., 2008; Gadah et al., 2016; Griffioen-Roose et al., 2013; van Opstal et al., 2019; Van Opstal et al., 2019) and two repeated ingestion studies, 9-weeks (Fantino et al., 2018a) and 18-months in length (de Ruyter et al., 2013) assessed sucralose's impact on food reward. One study reported lower sweetness intensity ratings in response to a sucralose beverage than fructose or sucrose (Wee et al., 2018) and one reported no difference in intensity ratings between sucralose, sucrose and maltodextrin (Chern & Tan, 2019). One study reported a decreased BOLD signalling across all sweet conditions (sucralose, 330mg; glucose, 50g; fructose, 50g; sucrose, 50g), but with delayed responses in sugar conditions relative to sucralose (Van Opstal et al., 2019). Two studies reported no effect of sucralose on subjective liking ratings compared to sucrose (Gadah et al., 2016) or sucrose, glucose or fructose (Kamerud & Delwiche, 2007). One study noted an increased relative reinforcing value (RRV) of sweet relative to savoury snack foods following sucralose ingestion compared with savoury snack foods (Casperson et al., 2017). One study reported sugar-sweetened rather sucralose-sweetened soft drinks were chosen more often, although no significant effect of sweetener type

on liking ratings or neural activation was reported (Griffioen-Roose et al., 2013). One study reported that sucrose sweetened beverages were chosen more often than HIS sweetened beverages, although there was not a main effect on liking, and that sucrose increased activation of the right precuneus (Griffioen-Roose et al., 2013).

2.3.4.4 HIS Blends

Eight acute studies examined HIS blends on food reward (Chern & Tan, 2019; Crézé et al., 2018; Delogu et al., 2016; Griffioen-Roose et al., 2013; Hill et al., 2014; Li et al., 2015; Smeets et al., 2011; Thai et al., 2011). One acute study reported no effect on sweetness intensity ratings (Chern & Tan, 2019) and one study, no effect on craving ratings, but also demonstrated activation of the dorsolateral prefrontal cortex in both sucrose and HIS blend conditions that was elevated relative to a water condition (Crézé et al., 2018). One study noted reduced liking ratings with increasing HIS dose in a blend of monk fruit and sucrose or stevia and sucrose (Li et al., 2015). One acute, repeated measures study reported higher sweetness intensity and lower pleasantness ratings for diet commercial beverages than sugar sweetened commercial beverages (Thai et al., 2011). One acute between-groups study reported more sweet foods chosen in a food selection task following consumption of a HIS blend and these foods were rated as less satisfying than following water or sugar ingestion (Hill et al., 2014). One acute between-groups study noted HIS sweetened beverages were correctly identified more often than those sweetened using sugars, with sugar sweetened beverages also rated more pleasant and more intense, across 14 commercially available beverages (Delogu et al., 2016). One acute crossover study reported deactivation of the amygdala in response to sucrose sweetened beverages but not HIS (Smeets et al., 2011). One acute crossover study reported higher implicit liking for a yoghurt sweetened using sucralose and acesulfame-K (0.008g/l and 0.013g/l, respectively) although no difference in fMRI images when compared to a sucrose condition (Griffioen-Roose et al., 2013).

2.3.4.5 Other HIS Types

Six studies included other HIS types (e.g., cyclamate, saccharin, stevia and monk fruit extract) (Gaudette & Pickering, 2012; Green & Murphy, 2012; Kamerud & Delwiche, 2007; Li et al.,

2015; Tey et al., 2017b; Wee et al., 2018). One acute study reported that RebA did not significantly reduce the bitter taste of a (+)-catechin solution whereas sucralose did significantly reduce bitter taste (Gaudette & Pickering, 2012) and one acute study reported higher bitterness ratings of monk fruit and stevia than aspartame or sucrose (Tey et al., 2017b). One acute crossover study reported lower intensity ratings in allulose, erythritol, sorbitol and mannitol than sucrose (Wee et al., 2018). One acute crossover study reported reduced liking ratings for monk fruit and stevia than sucrose concentrations (Li et al., 2015). One acute between-groups study noted no effect on neuro-activation of sweetener condition (saccharin versus sucralose) but greater right amygdala activation in habitual HIS consumers (Green & Murphy, 2012). One acute repeated measures study reported sucrose sweetened beverages were more well liked than cyclamate, d-tryptophan, thaumatin and saccharin (Kamerud & Delwiche, 2007).

2.3.5 Effects of HIS on Body Weight

The effects of HIS ingestion relative to sugar and/or water controls can be seen in Table 2.5.

Table 2.5. Effects of HIS versus sugar and/or water controls on body weight.

Author	Population	Sweetener	Comparator	Length	Outcomes
Higgins et al. (2018)	n: 93 Age (years): 18-60 years BMI: 18.5-25kg/m ² % Female: 54	Aspartame (35mg or 1,050mg)	Water	12 weeks	No effect of aspartame intake on body weight or composition
Kuzma et al. (2015)	n: 10 Age (years): 21.0 (2.0) BMI (kg/m ²): 22.7 (1.3) % Female: 56	Aspartame (NR)	Fructose (25% EER) Glucose (25% EER)	8 days	No effect of aspartame intake on body weight
Maersk et al. (2012b)	n: 12 Age (years): 39.0 (8.0) BMI (kg/m ²): 32.8 (3.8) % Female: 75	Aspartame (NR)	Glucose + Fructose (106g) Milk (47g) Water	6 months	No effect on body weight or total fat mass in aspartame and water conditions Increase in total fat mass in glucose+fructose condition
Porikos et al. (1982)	n: 6 Age: 39.8 BMI (kg/m ²): NR % Female: 0	Aspartame (sweetened foods)	Sucrose (sweetened foods)	24 days	No effect of aspartame intake on body weight
Reid et al. (2007)	n: 133 Age: 31.8 (9.1) BMI (k/m ²): 22.5 (2.8) % Female: 100	Aspartame (NR)	Sucrose (105g)	3 weeks	No effect of aspartame intake on body weight
Reid et al (2010)	n: 53 Age (years): 32.93 (8.84) BMI (kg/m ²): 27.83 (1.83) % Female: 100	Aspartame (NR)	Sucrose (26.25g)	5 weeks	No effect of aspartame intake on body weight
Reid et al. (2014)	n: 20 Age (years): 35.1 (9.9) BMI (kg/m ²): 32.9 (1.8) % Female: 100	Aspartame (NR)	Sucrose (26.25g)	4 weeks	No effect of aspartame intake on body weight
Tordoff et al. (1990)	n: 30 Age: 28.2 (2.7) – females, 22.9 (0.8) - males BMI (kg/m ²): 25.4 (1.4) – females, 25.1 (0.5) - males % Female: 30	Aspartame (590mg)	High fructose corn syrup (133g)	3 weeks	Decrease in body weight of males but not females (no effect in grouped sample) in aspartame condition Increase in body weight in high fructose corn syrup condition

Author	Population	Sweetener	Comparator	Length	Outcomes
Chern & Tan (2019)	n: 11 Age: 24.9 (6.1) BMI (k/m ²): 25.0 (4.7) % Female: 27	Sucralose (120mg) Sucralose + Maltodextrin (120mg + 50g)	Sucrose (50g)	Acute	Body weight did not change from baseline
Thai et al. (2011)	n: 325 Age: 21.0 (14.5) BMI (k/m ²): NR % Female: 51	Coca-Cola Light	Coca-Cola Regular	Acute	No change in body weight
Bonnet et al. (2018)	n: 50 Age: 31.1 (10.3) BMI (k/m ²): 24.7 (3.2) % Female: 56	Aspartame + Acesulfame-K (129mg + 13mg)	Water	12 weeks	No change in body weight
Markey et al. (2016)	n: 50 Age: 32.0 (9.8) BMI (k/m ²): 23.5 (3.0) % Female: 68	Sugar reduced foods	Sugar sweetened foods	8 weeks	No change in body weight
Raben et al. (2002)	n: 41 Age: 37.1 (2.2) BMI: 27.6 (0.5) % Female: NR* ¹	Sugar reduced foods	Sugar sweetened foods	10 weeks	Increase in body weight, fat mass and blood pressure in sucrose condition, but no effect in HIS condition
Raben et al. (2011)	n: 23 Age: 35.5 (2.8) BMI: 27.6 (0.8) % Female: NG* ¹	Sugar reduced foods	Sugar sweetened foods	10 weeks	Increase in body weight in sucrose condition compared to HIS condition
Sorensen et al. (2005)	n: 41 Age: 33.4 (9.0) BMI: 28.0 (2.3) % Female: 85* ¹	Sugar reduced foods	Sugar sweetened foods	10 weeks	Increase in body weight in sucrose condition and decrease in body weight in HIS condition
Maersk et al. (2012)a	n: 24 Age: 33.5 (9.2) BMI (k/m ²): 31.4 (3.11) % Female: 50	Commercially available diet-cola	Commercially available cola Semi-skimmed milk Water	6 months	Greater relative change in liver fat, skeletal muscle fat and visceral fat in regular cola condition Total fat mass did not differ between conditions

Abbreviations: NR – dose not given. NR – not reported. BMI – body mass index.

¹ Means provided for one experimental group, means for both groups can be found in supplementary materials.

2.3.5.1 Acesulfame-K

No studies examined the effect of acesulfame-K on body weight.

2.3.5.2 Aspartame

Aspartame ingestion had no effect on body weight in eight studies (Higgins et al., 2018; Kuzma et al., 2015; Maersk, Belza, Stødkilde-Jørgensen, et al., 2012; Porikos et al., 1982; Reid et al., 2007, 2010, 2014; Tordoff & Alleva, 1990), the minimum length of trial being 8-days (Kuzma et al., 2015) and the longest trial being 6-months (Maersk, Belza, Stødkilde-Jørgensen, et al., 2012). Three studies were conducted in healthy weight participants (Higgins et al., 2018; Kuzma et al., 2015; Reid et al., 2007) and five were conducted in participants with overweight or obesity (Maersk, Belza, Holst, et al., 2012; Maersk, Belza, Stødkilde-Jørgensen, et al., 2012; Reid et al., 2010, 2014; Tordoff & Alleva, 1990). One study reported a gender difference, with aspartame ingestion increasing body weight in women but not men (Reid et al., 2007), two studies identified increases in body weight following sugar ingestion relative to aspartame (Porikos et al., 1982).

2.3.5.3 Sucralose

One acute study examined sucralose on body weight - reporting no effects (Chern & Tan, 2019). Due to being an acute study (single day) these findings will not be considered further.

2.3.5.4 HIS Blends

Seven studies examined body weight response to HIS blends. Two acute studies reported no effect on body weight (Chern & Tan, 2019; Thai et al., 2011) – which will not be considered further due to their acute (single day) design. Two studies reported no effect on body weight following HIS blends ingestion in healthy weight individuals (Bonnet et al., 2018; Markey et al., 2016) ranging from 8-weeks (Markey et al., 2016) to 12-weeks in length (Bonnet et al., 2018). Two studies reported an increase in body fat levels following sugar ingestion and reduction in body weight following HIS ingestion following 10-weeks of repeated ingestion when provided via commercially available foods (Raben et al., 2002, 2011; Sørensen et al., 2005).

2.3.5.5 Other HIS Types

No studies examined other HIS types on body weight.

2.4 Discussion

The aim of the current systematic review was to examine the effects of different HIS types to sugar (presented in either beverages or food) and/or water controls on appetite and food reward related outcomes as factors influencing changes in body weight. An additional aim was to examine whether distinct HIS types may affect the aforementioned outcomes in distinct ways. The principal findings were that HIS did not impact subjective appetite, or stimulate *ad libitum* or free-living food intake, and instead appear to reduce energy intake through a reduction in the energy content of beverages; with most consistent evidence surrounding aspartame ingestion. Furthermore, HIS ingestion, unlike sugars, does not stimulate appetite-related biomarkers when provided in isolation (i.e. minus an energy-containing load). Moreover, there was a lack of robust evidence and inconsistency in findings surrounding the effects of food reward. Finally, inclusion of HIS in the diet may serve to reduce body weight in individuals living with overweight and obesity following repeated ingestion, although there was no effect on body weight in lean individuals.

2.4.1 Appetite

Aspartame was most frequently examined and the evidence demonstrated that aspartame ingestion did not stimulate or suppress subjective appetite when compared with water controls (Black et al., 1991, 1993; Canty & Chan, 1991; Higgins et al., 2018; Maersk, Belza, Holst, et al., 2012; Steinert et al., 2011) ($n=187$) or sugar controls (Reid et al., 2007; Rolls, Hetherington, & Laster, 1988) ($n=175$). Two studies (Rogers et al., 1988; Tey et al., 2017b) reported an increase in appetite following aspartame ingestion which were smaller in sample size ($n=24$) and were compared relative to a sucrose control. The evidence highlighting an absence of differences produced by either aspartame or water ingestion rejects the notion that acute aspartame ingestion is capable of stimulating appetite. The evidence further suggests that aspartame does not suppress appetite in the same manner to a nutritive sugar, possibly due to a lack of post-ingestive effects as produced by ingestion of an energetic load. Furthermore, when

participants were provided aspartame in capsule form (absent buccal sweet stimulation), they did not report any alteration to subjective appetite (Black et al., 1993). This is a compelling finding highlighting the differential nature of sweet taste and energy density as two distinct variables exerting influences on appetite in their own right. Moreover, when providing participants beverages of varying volumes (280ml vs 560ml) there were observed appetite suppressant effects (Black et al., 1991) – not attributable to the sweetener, but rather produced by the volume of the beverage. It was similarly demonstrated in one study that ratings of fullness were higher in beverage conditions, regardless of beverage type which included commercially available diet and regular cola, milk, water and orange juice, than a no beverage condition (DellaValle et al., 2005). Together these findings further support that aspartame does not influence appetite when presented as an independent sweetener or when included in a blend, and suggests that any appetite-related effects may be due to the volume of the ingested beverage, rather than the sweetener utilised. Further supporting this are two repeated ingestion studies. In Reid et al. (2007) there was a lack of difference in appetite response when compared to baseline following repeated ingestion and in Higgins et al. (2018) there was no difference between appetite at baseline or after 12-weeks repeated ingestion, therefore highlighting that aspartame consumption can be advocated from an appetite stimulation viewpoint.

Similar findings were found regarding acute sucralose ingestion, highlighting a difference in the effects exerted by sucralose and sucrose on appetite. Important to note, are the studies which reported no difference between sucralose and sugars - (Black et al., 1991; Casperson et al., 2017; Chern & Tan, 2019; van Opstal et al., 2019) – Casperson et al. (2017), provided a test beverage which was 360ml, possibly large enough to reduce feelings of hunger and increase feelings of fullness, as previously suggested (Black et al., 1991). One study (van Opstal et al., 2019) recorded a single 30-min post-prandial response which may miss variations in response as a function of time whereas Chern and Tan's study (2019) represented a pilot study and findings from this study must be interpreted with care. The caveats in the four studies may explain the lack of differences observed. Two studies reported significant differences in the means between sucralose and a nutritive sugar. One study reported reduced hunger following

sucrose ingestion relative to sucralose (Gadah et al., 2016) and a second reported lower fullness ratings following sucralose ingestion than a glucose condition (Wu et al., 2012) – findings which are anticipated and attributable to the energy content of the sugar sweetened beverages. From the available evidence, it can be suggested that sucralose ingestion does not exert an effect on subjective appetite, however an awareness of the strength of this evidence must be applied when interpreting these findings.

A consumer concern surrounding the inclusion of HIS in the diet is that they may increase food intake. However, the evidence outlined in the current review does not support this concern. An early study conducted in a metabolic ward provided participants with a diet consisting of sucrose sweetened foods in buffet style meals in which intake was recorded by experimenters (Porikos et al., 1982). When the energy density of the food was covertly manipulated through the use of aspartame, participants compensated for this reduction in energy density by increasing their intake; however, it is important to note that energy intake remained below baseline, suggesting only partial energy compensation (Porikos et al., 1982). This is a compelling finding, emphasising the capability of reducing the energy density of diets with the use of HIS to such an extent that even in the event food intake increases, due to only partial energy compensation, individuals will likely remain in a calorie deficit. However, key to note here is that the reduction in energy content was done covertly, and so these findings demonstrate a lack of complete homeostatic energy compensation – whether the same can be said for instances with overt energy reduction cannot be said (Doucet et al., 2018)

Another historical study which provided aspartame via capsule form or dissolved in water reported a lower *ad libitum* energy intake when ingested in the absence of sweet stimulation (Black et al., 1993). Three separate studies also reported no increase in *ad libitum* energy intake following acute consumption of aspartame and sugar controls (Rodin, 1990; Rolls, Hetherington, & Laster, 1988) or following repeated ingestion of aspartame (Kuzma et al., 2015). However all three reported a higher total energy intake in sugar conditions when including the energy content of beverages. These findings suggest that HIS do not stimulate intake in a single test meal. Rather, they may facilitate a reduction in energy intake via a

reduction in the energy density of beverages provided alongside a meal, whilst leaving the energy intake of the meal unaltered. This represents an important and significant finding, with HIS appearing to not alter food intake whilst being capable of autonomously reducing energy intake.

It would appear that the effects of repeated ingestion are more prominent than acute ingestion. One study reported a decline in energy intake following repeated consumption of an aspartame-sweetened beverage and a higher total energy intake in a HFCS condition, with a decreased sugar intake that could not be attributed to a singular item, but represented a general reduction in all sugar-containing foods in the diet (Tordoff & Alleva, 1990). Similarly, sucrose-sweetened beverages increased energy intake in a more recent study (Reid et al., 2010). Further corroborating this, a 6-month dietary intervention which utilised diet beverages reported equivalent reduced energy intake between water and HIS conditions (Tate et al., 2012). These long-term studies demonstrate that products sweetened using HIS rather than sugars facilitate a reduction in energy intake. This is in line with previous evidence which has suggested that energy-dense liquids (i.e., SSBs) consumed between meals may not result in subsequent adequate energy adjustment (Almiron-Roig et al., 2003) as the majority of evidence surrounding HIS utilises beverages as the vehicle of administration. This finding may have important applications, as although HIS ingestion may not exert an influence on appetite or food intake, due to their lack of contribution to energy intake, it would appear that their inclusion can effectively reduce energy intake through a substitution with poorly satiating SSBs.

The overwhelming evidence is that HIS do not impact appetite-related biomarkers or peptides, such as glucose, insulin, GLP-1, ghrelin, glucagon or PYY with consistent effects demonstrating this. The elicitation of an appetite biomarker response is dependent on more than the detection of sweetness, a conclusion corroborated elsewhere (Steinert et al., 2011). This highlights the role of sweetness and energy-density as two distinct variables, that whilst share a level of synergy when provided in unison (i.e., via nutritive sweeteners), do not produce equivalent post-prandial appetite responses when ingested in isolation (i.e., HIS providing

sweetness minus energy-density). Compelling evidence is offered via long-term repeated ingestion studies, with 12-week repeated ingestion (aspartame + acesulfame-K) not impacting insulin sensitivity (Bonnet et al., 2018) or 10-week repeated ingestion of commercially available beverages also not impacting post-prandial glycaemia (Raben et al., 2011; Sørensen et al., 2003) – both outcomes which are in turn capable of impacting appetite. This evidence highlights that HIS can be ingested repeatedly on a long-term basis in terms of appetite-related biomarkers and post-ingestive responses, and given the evidence that a sucrose-rich diet increased glycaemia, insulinemia and lipidemia after 10-weeks (Raben et al., 2011), HIS may prove safer than sucrose. However, these conclusions have been reached on the basis of evidence in healthy individuals, recommendations for individuals with metabolic related disorders such as hyperinsulinemia (e.g., individuals with type 2 diabetes mellitus) must be made with care and should not be formed on the basis of this evidence alone.

A small number of studies demonstrated small, but possibly clinically important changes to hormone responses. It was highlighted that GLP-1 is released in response to a nutritive sweetener but sweet stimulation absent of energy was not sufficient to elicit this response (Temizkan et al., 2015). A 24-mg sucralose dose 15-mins prior to a 75-g oral glucose tolerance test (~307kcal), did not result in any response in GLP-1 secretion in healthy individuals. However, sucralose enhanced GLP-1 secretion when provided prior to an energetic load – with a higher AUC GLP-1 response than aspartame or water conditions. In this way, sucralose enhanced GLP-1 release in the presence of glucose, reinforcing that GLP-1 release occurs in response to energy, but also suggesting that sucralose provided in conjunction with energy may result in a higher response, a response that is not observed with other HIS types.

Similarly, ingestion of an aspartame and acesulfame-K blend or water prior to an energetic load resulted in comparable glycaemic responses until 120-min post-ingestion (Solomi, 2020). This finding suggests that when HIS are provided prior to an energetic load, the post-prandial responses may be impacted, although this may not necessarily occur immediately. This finding requires further investigation with an emphasis placed on a better understanding of the interaction between HIS when provided absent of energy and prior to an energetic load, and

whether this differs amongst HIS types. Taken together, these findings also reinforce concerns that all HIS should not be considered equivalent in their effects on outcomes of interest and consideration of this should be carried forward in the development of future studies.

2.4.2 Food Reward

An issue surrounding consumer acceptability of products using HIS is that a number include persistent or unpleasant after tastes (Mortensen, 2006). Three studies reported higher subjective liking ratings to sugar rather than HIS conditions (de Ruyter et al., 2013; Delogu et al., 2016; Thai et al., 2011). This may be explained by differences in sweetness intensity, with HIS often rated as more bitter (Gaudette & Pickering, 2012; Tey et al., 2017b, 2017a) or less sweet (Kamerud & Delwiche, 2007; Thai et al., 2011; Wee et al., 2018) than sugar sweetened beverages. Interestingly, one study examined consumer acceptability ratings of beverages sweetened using different ratios of HIS to sugar. As the ratio of monk fruit or stevia to sucrose increased, liking ratings declined, in both adults and children (Li et al., 2015). Highest ratings were provided for a solution consisting of 25% monk fruit or stevia and 75% sucrose and were not significantly different to a 100% sucrose control beverage. This suggests that sugar is more liked and acceptable to consumers than HIS - but additionally, when HIS are combined with sugar, there may be an optimal ratio to maintain liking whilst reducing the energy content of the beverage. Unfortunately, this was only demonstrated utilising beverages and so the findings may not be replicated when applied to solid foods – however given the manufacturing difficulties of substituting sugar for HIS in food products this finding may provide important information for manufacturers (Luo, 2019). Furthermore, an incomplete reduction in the sugar content of popular beverages or foods may enable a greater consumer acceptability, ensuring ‘diet’ or ‘lite’ products remain acceptably palatable.

The extent to which individuals can accurately identify sweeteners in beverages remains unclear, with one study demonstrating a higher accuracy of recognition for sugar (HFCS) sweetened beverages (Tordoff & Alleva, 1990) whereas another demonstrated a higher degree of accuracy for HIS sweetened beverages (Delogu et al., 2016). This may be because Delogu (2016) utilised a variety of commercially available beverages, whereas Tordoff & Alleva,

(1990a) provided an aspartame solution. This may indicate that participants are inaccurate at identifying a sweetener when presented in the absence of other flavourings, but are better able to identify beverage type when presented with commercially-available and therefore ecologically-valid products. If this is true, then the inclusion of additional flavourings may assist in the reduction of unpleasant or unwanted after tastes, which appear to be present following HIS ingestion.

Regarding food appeal, sensory specific satiety effects were observed in an early study, with aspartame and saccharin ingestion both increasing the number of savoury and high protein foods selected in a food preference checklist (Rogers et al., 1988). However, a more recent study identified that participants who ingested a HIS solution were almost 3 times more likely to choose candy than participants who had consumed a SSB or water (Hill et al., 2014), suggesting that HIS may increase the appeal of sweet, not savoury foods. These differences in findings may stem from the use of different measures, with food preference checklists requiring participants to imagine the stimuli, whereas Hill et al. (2014) provided a physical stimulus. Alternatively, this may stem from differences in stimuli used. Rogers and colleagues (1988) provided aspartame or saccharin dissolved in tap water, whereas Hill and colleagues (2014) utilised commercially available beverages, which consisted of a blend of HIS (aspartame and acesulfame-K). This may indicate a synergistic effect of the two sweeteners serving to increase sweet food appeal, which is not observed when sweeteners are provided in isolation.

Interestingly, Hill and colleagues (2014) also reported that participants reported foods to be less satisfying when proceeded by consumption of a HIS sweetened beverage than water or a sucrose sweetened beverage. This thereby reinforces the suggestion that sensory specific satiety is capable of exerting an influence following HIS ingestion, which appears to be a more powerful influence than that exerted on the appeal of sweet foods and so should not result in an increased sweet intake.

2.4.3 Body Weight and Composition

It has previously been highlighted that paradoxically, habitual HIS use is positively associated with BMI (Appleton & Blundell, 2007; Fowler, 2016; Fowler et al., 2015). However, the

findings of the current review indicate it to be an example of reverse causation as long-term interventions did not observe an increase in weight following HIS consumption. In a 6-month trial which examined the effect of 'diet beverages' on body weight, a reduction in body weight was not significantly different to the reduction observed following repeated water ingestion (Tate et al., 2012). Important to note is that this was conducted in participants with overweight or obesity. The majority of studies that investigated body weight outcomes were conducted in lean participants (Bonnet et al., 2018; Chern & Tan, 2019; Higgins et al., 2018; Kuzma et al., 2015; Reid et al., 2007; Thai et al., 2011) - although this should not be interpreted as HIS having no effect on body weight. In addition to the findings provided by Tate and colleagues (2012), three studies were conducted in populations with overweight or obesity, which failed to report a reduction in body weight (Reid et al., 2010, 2014; Tordoff & Alleva, 1990). Although each of these were considerably shorter in length (3-weeks to 5-weeks) which may be interpreted as indicating that HIS only produces reductions in body weight in populations with overweight and obesity when included on a long-term basis (e.g., 6-months). Further supporting this is evidence from two 10-week interventions in which participants with overweight were assigned to consume products sweetened using sucrose or a number of HIS. In Sørensen et al., (2005) the sucrose condition caused an increased sucrose intake of 151% resulting in a 1.6-kg weight gain, and in the HIS condition, sucrose intake decreased by 42% resulting in a 1.2-kg weight loss. Raben and colleagues (2002) highlighted that the increase in body weight following sucrose intake was primarily due to an increase in fat mass, which should be attributed to the increase in energy intake provided from the additional sucrose consumed via the intervention. Finally, in an 8-week crossover study which required healthy weight participants to replace sucrose sweetened products with reformulated HIS products, no differences in body weight or body fat percentage were observed post intervention (Markey et al., 2016). The lack of change in body weight was likely due to a reduction in sugar intake and increase in protein and fat intake. Taken together, these findings may be explained by differences in energy balance regulation, with leaner individuals displaying better energy balance regulation than those with overweight or obesity. These findings would also suggest

that HIS use, from a weight balance viewpoint, are favourable to sugar sweetened products which serve to increase energy intake and subsequently lead to weight gain, whereas HIS ingestion, which may not lead to a reduction in body weight in every instance, will not contribute to an increase in weight.

The studies included within the current review primarily report on body weight rather than body composition. A single study utilised dual x-ray absorptiometry assessment to measure body composition. This study reported no differences in total fat mass (kg) between aspartame or water conditions, whilst total fat mass increased in a commercially available SSB condition (Maersk, Belza, Stødkilde-Jørgensen, et al., 2012). This finding highlights that HIS beverages are comparable in their effects on body weight and composition to water, which is also highlighted in the (Bonnet et al., 2018) study included within the review. Assessment of body composition often requires the use of expensive equipment and is more time consuming than the recording of body weight, which presents both a cheaper and quicker option. This may be a reason for the lack of body composition outcomes included within the literature reviewed here, a potential solution to which may be the use of hip and waist circumference as an outcome. Hip and waist circumference has been recommended to be included in clinical examinations of adults (The Canadian Heart Health Surveys Research Group et al., 2001) due to the high diagnostic precision and simplicity of its determination and is a useful predictor of obesity related comorbidities such as cardiovascular disease (De Koning et al., 2007) or mortality and morbidity (Kartheuser et al., 2013).

2.4.4 Limitations

Various definitions were employed for each of the outcomes of interest, which may contribute to some of the inconsistent patterns observed. This is particularly evident when operationalising food reward, with a number of studies employing subjective VAS responses, which utilise a number of different aspects of food reward, including both liking and wanting, perception and pleasantness. In addition to this, neuroimaging techniques have been employed by a number of studies, offering an insight into areas of the brain which are stimulated by various tasks such as viewing food images. Finally, a small number of standalone studies provided methods such as a

relative reinforcing task or food preference checklists, producing difficulties in drawing conclusions regarding the effects of HIS ingestion on food reward due to challenges in the comparison of study findings.

A large number of studies gathered subjective ratings (appetite and food reward related) through the use of VAS. These may prove to not be sufficiently sensitive enough to capture individual differences in responses, with evidence demonstrating that the intensity labels used in these traditional scales do not denote the same perceived intensities in individuals with obesity and lean individuals (Bartoshuk et al., 2006; Pepino & Mennella, 2012). A general labelled magnitude scale (gLMS) has been proposed as a more sensitive measure that is capable of considering the fact that labels vary as the intensity or hedonic which they represent is based on experience - although few studies in the current review employed this method.

Moreover, a preponderance of evidence has been conducted in healthy individuals, absent metabolic disorder with a BMI ranging between 19.0-24.9 kg/m². Presently, there exists a positive association between BMI and the habitual consumption of HIS products (Fowler et al., 2008; Mossavar-Rahmani et al., 2018; Sylvetsky et al., 2017) and motivation to consume these products originates in weight-related goals (Pielak et al., 2019). In the current review, of the twelve repeated ingestion studies which included body weight as an outcome, six were conducted in participants with overweight or obesity – emphasising the requirement for further studies of this nature.

2.4.5 Future Directions

At present, no study has combined outcomes of appetite and food reward as mechanisms capable of influencing changes in body weight; the current review highlights this requirement. In addition to this, there is a need for greater focus on differential effects of HIS types as there appears to be a number of small, yet important differences (e.g., sucralose provided alongside an energetic load results in an altered ghrelin response not observed following consumption of other HIS types (Temizkan et al., 2015)).

The effects of HIS versus sugar or water on food reward must be interpreted with careful consideration of the various definitions and methodologies used in assessing food reward. Food reward can refer to the expected or anticipated impact of ingesting a food, the acute impact at the precise moment of ingestion, in addition to the subsequent impact on the reward of other foods following HIS ingestion. Studies investigating food reward currently fail to establish this distinction, subsequently it is recommended that future studies address this omission with consideration of measurement timings relative to the time of ingestion and the implications of their findings. Additionally, a distinction between subjective food reward and neuroimaging studies should be made when considering results of studies. Food reward remains a broad topic operationalised in numerous methods – future studies should endeavour to explicitly address the specific components of food reward being investigated.

Furthermore, consideration should be given to sample characteristics, as sweet taste is known to vary as a function of age (Boesveldt et al., 2018; Desor & Beauchamp, 1987), sex (Yoshinaka et al., 2016) and possibly body fatness (Ettinger et al., 2012). It cannot be assumed that the effects of HIS and sugars are equitable in individuals whose characteristics are believed to influence appetite sensitivity, hedonic response and taste preferences. Moreover, habitual consumption is rarely considered, despite one study highlighting greater amygdala activation in response to a sucrose rather than a HIS sweetened beverage in non-habitual consumers compared to habitual consumers of HIS products (Kamerud & Delwiche, 2007) and thus this may represent an important confounding variable in future analyses.

A final recommendation is made regarding the mode of ingestion. The preponderance of evidence is provided through an examination of HIS ingested via beverages and a conclusion drawn from this evidence is that replacement of sugar sweetened beverages with HIS beverages facilitates a reduction in calories obtained via the beverage. However, it remains unclear, in part due to a lack of consistent evidence, what the effect of HIS in foods which simultaneously provide an energetic load will be. For this reason, future work should endeavour to provide HIS via food products rather than beverages, in order to better understand their interaction with

components of solid food products which are not involved in beverage ingestion (e.g., macronutrients, fibre, texture or mastication).

2.4.6 Conclusions

The evidence provided highlighted differential effects of sweetener types, consequently conclusions must be drawn for each specific sweetener type and not for HIS as a homogenous group. An interesting finding was that aspartame and acesulfame-K appeared to impact glycaemia (12- min post-ingestion) (Solomi, 2020) and sucralose impacted GLP-1 release (Temizkan et al., 2015) when provided prior to an energetic load. These two studies serve as an example to demonstrate that different HIS types impact post-ingestive responses differently, or different post-ingestive responses to HIS ingestion. It is clear that further work needs to be done to identify which scenario is the reality of the situation but also reinforcing the notion that HIS effects are not all synonymous.

Effects on appetite were most frequently investigated using aspartame, which does not appear to stimulate subjective appetite, nor energy intake, and HIS on a whole do not appear to stimulate the release of appetite-related biomarkers – with the exception of a minority of studies which suggest small yet clinically significant effects. RCTs examining the influence of food reward remain too inconsistent in their methodology and definitions to enable clear effects to be observed. However, participants tended to provide higher liking ratings to sugar than HIS beverages, and commercially available diet beverages may be more recognisable to participants due in part to varying aftertastes. Finally, the effects of HIS on body weight appear to be dependent on the participant's BMI and study length, with reductions in body weight displayed in individuals with overweight and obesity whilst healthy weight individuals hold weight constant. The inclusion of HIS serves to reduce the energy density of the diet through a reduction of sugars and carbohydrates in the diet.

It is important to consider differences in methodology and future work should endeavour to provide more consistency in methodology and establish the evidence surrounding specific sweetener stimuli, rather than grouping all HIS types into one group. This is important given

the current increase in the consumption of HIS in individuals motivated to lose weight, and the diverse food environment available to these individuals.

3 General Methodology

3.1 Measures completed in both study one and study two

The two studies outlined herein shared a commonality in a number of measures which are discussed below. For specific details regarding their implementation in each study, please see section 3.2 for study 1 (DIVA, ethical approval ref: PSC-238 and PSC-551) and 3.3 for study 2 (SWEET, ethical approval ref: PSYC-127).

3.1.1 Assessing Appetite and Food Intake

It is important when investigating appetite and food intake that a whole diet approach is adopted as a focus on single nutrients or dietary components does not prove fruitful. Similarly, an examination of only specific eating behaviour scenarios, such as only *ad libitum* feeding or free-living behaviours tells only half the story. An *ad libitum* test meal is a commonly used method in laboratory environments, in which food portions are weighed before and after serving to participants who are required to eat until comfortably full, with portions provided in excess of reasonable consumption. Free-living assessment usually involves self-report methods (for example, retrospective food diaries), providing an indication of habitual dietary patterns. Both methods suffer from limitations; laboratories are unfamiliar environments and often involve participants consuming a meal in an isolated cubicle away from external distractions. An advantage however of *ad libitum* meals is the requirement for participants to rely on internal satiety cues to determine a meals cessation point, and this assessment technique has been shown to be reproducible (Gregersen et al., 2008). On the other hand, free-living dietary recall relies on the accuracy of participant recollection (and their truthfulness in reporting) and it has been suggested to be an indicator of the participant's memory of the diet, rather than the diet itself (Krall et al., 1988). Moreover, they are also impacted by participant related factors, for example, female participants tend to be influenced by demand characteristics when reporting (Hebert et al., 1995). However, this method of assessment provides data regarding a participant's natural eating behaviour, in a natural setting away from an artificial laboratory environment. Therefore, when examining food intake in order to provide an accurate

understanding of participant eating behaviours, it is important to examine both types of studies with an awareness of their respective limitations and consideration of what each method is able to accurately represent (Archer et al., 2013).

When assessing appetite, subjective ratings are a commonly utilised measure (for example visual analogue scales (VAS) or general labelled magnitude scales (gLMS) for hunger, fullness and other components of appetite) and/or blood biomarkers (for example glucose, insulin, ghrelin) (Bartoshuk et al., 2006; Flint et al., 2000). Subjective ratings involve an anchored scale in which participants indicate along the scale how they feel at that moment in time. VAS have been shown to be valid when compared to single test meals (Flint et al., 2000) and are capable of assessing a variety of components of subjective appetite. Similarly, gLMS have been shown to be a more accurate and reliable measure as these scales are less prone to suffering from individual variability in responses (Bartoshuk et al., 2006), despite this, the preponderance of appetite related research utilises VAS measures. Blood biomarkers on the other hand can provide an insight into the physiological processes involved in appetite regulation, identifying the underlying physiological responses involved in appetite. Both assessment techniques provide a useful insight into a different domain of appetite, however, as the present thesis forms one component of a wider collaborative study, the blood biomarker assessments utilised are unable to be included due to unavailability of the data at the time of writing.

Food intake may also be utilised as an indication of appetite and hunger and thus is included as an appetite related outcome within the present thesis. It is important to note that an increase in food intake does not guarantee an increase in energy intake (Porikos et al., 1982) – and so the distinction between food and energy intake must be made when considering the results of trials as the energy content of the food consumed is an important variable.

When examining the effects of HIS on the diet a number of studies examine the acceptability of various sweet substances. However, when considering the use of HIS in the diet, it is important to consider the strategy of implementation employed, with key differences in outcomes depending on whether a strategy of substitution or addition is employed. For example, reducing

free sugars by 40% without any replacement over a 5 year period would lead to an average reduction in energy intake of 38.4kcal per day, producing an average reduction in bodyweight of 1.2kg (Ma et al., 2016). At a population level, this reduction would lead to a decline of approximately 0.5million adults with overweight and 1million adults with obesity (Ma et al., 2016).

3.1.2 Leeds Food Preference Questionnaire Assessment

The Leeds Food Preference Questionnaire (LFPQ) (Finlayson et al., 2008) is a computer-based procedure designed to assess two distinct psychological components of food reward – liking and wanting. The test utilises sixteen images of common food stimuli varying in fat content (high [HF] or low [LF]) and taste (sweet [SW] or savoury [SA]), with the combination of fat content and taste producing four categories (HFSW, LFSW, HFSA and LFSA). Two different question formats allow measurement of explicit liking, explicit wanting and implicit wanting, with the two separate procedures preventing cross contamination between the two concepts – frequency of selection is also recorded. In instances of low acceptance of foods (established and confirmed during screening) there were a number of additional images for each category that could be substituted. In study 1 a breakfast and lunch version with appropriate pictures was utilised (Appendix 1), whilst in study 2 a single set of images was employed at each site, with culture specific images being provided that were specific to the site of data collection (Appendix 9).

3.1.2.1 Leeds Food Preference Questionnaire Procedure

Food images are presented in a randomised order individually via a VAS to provide a measure of explicit liking (EL). Participants are required to rate “How pleasant would it be to taste some of this food now?” on 100mm scales with weighted answers at either end “Not at all” and “Extremely”. Explicit wanting (EW) is assessed in a similar manner although participants answer “How much do you want some of this food now?”. Please see Figure 3.1 for a visual representation of the EL and EW trials.

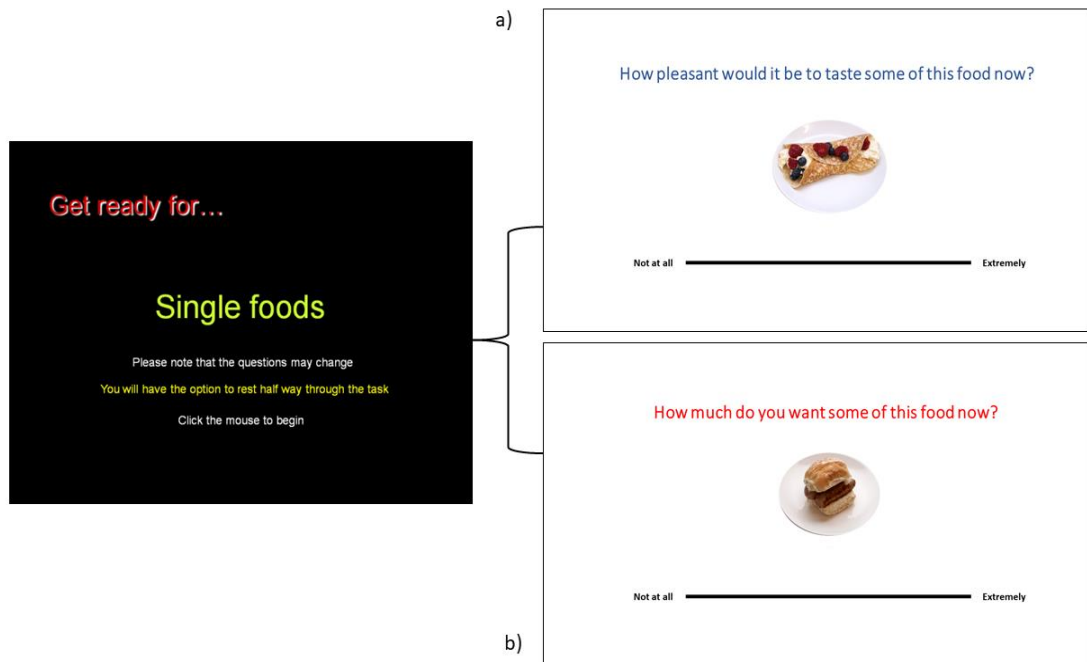


Figure 3.1. Representation of the a) Explicit Liking and b) Explicit Wanting trials in the Leeds Food Preference Questionnaire.

A sweet bias score can be calculated by subtracting the mean sweet scores from the mean savoury scores, providing a sweet versus savoury score for each outcome. Sweet bias scores usually range from 0 to 48. Similarly, a fat bias score can be calculated by subtracting mean low fat scores from the mean for high fat scores, providing a high fat versus low fat foods for each outcome. Fat bias scores range from -48 to 48.

EL and EW category scores (HFSA, LFSA, HFSW, LFSW) are obtained by averaging the ratings for each category for each participant. A higher score indicates a higher EL or EW respectively.

A forced choice procedure in which food images are paired in such a way that every image used is compared to every other image over ninety-six trials, to provide an assessment of implicit wanting (IW). Participants answer the question “Which food do you most want to eat now?”. Please see Figure 3.2 for a visual representation of the IW trials. Participants are instructed to respond as quickly and as accurately as possible and focus on only the type of food shown (i.e. not the quantity). IW scores are relative to the other food choices with a frequency weighted algorithm (see Figure 3.3) used that is influenced by both the frequency of food choices and the reaction times of answers (Dalton & Finlayson, 2014). Scores for IW

usually range from -100 to 100 as there is no fixed minimum/maximum value due to the inclusion of reaction time.

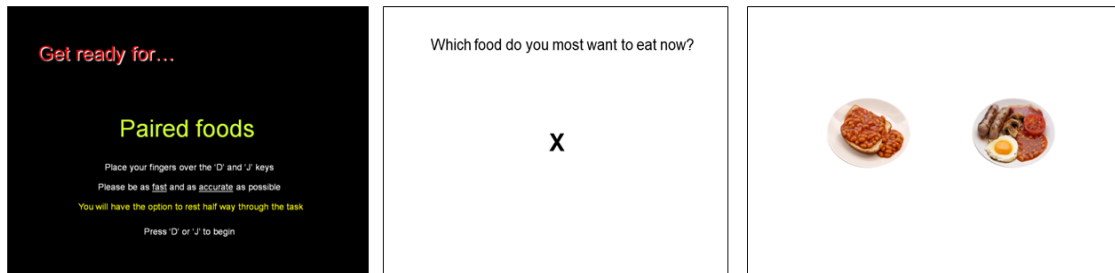


Figure 3.2. Representation of the Implicit Wanting trials in the Leeds Food Preference Questionnaire.

$$\text{'Frequency-weighted algorithm': } I_A = \sum_{i=1}^{N_{\text{win}}} \frac{\bar{t}}{t_i} - \sum_{j=1}^{N_{\text{lose}}} \frac{\bar{t}}{t_j}$$

Figure 3.3. Frequency weighted algorithm used to score IW in the LFPQ.

Note. I_A = Implicit wanting for category. A; N_{win} = number of times category A was selected. N_{lose} = number of times category A was not selected. \bar{t} = mean of all reaction times. \bar{t}_{win} = mean reaction time for category A selections; \bar{t}_{lose} = mean reaction time for non category A selections.

The acceptability of the food images was confirmed at screening in both study 1 and study 2 to improve the internal validity – it is believed that an alternative food from the same category will yield better than a fixed food that is avoided. Participants also completed practise trials of the LFPQ at screening. The format of questions was delivered randomised in order to prevent order effects, with some participants completing the single image trials first and the paired image trials second and vice versa.

Participants sat at a desktop terminal in an isolated room. Before the trial began participants were instructed that the questionnaire would measure their food preferences and involved images of real foods. Participants were instructed to answer single images of foods by clicking the mouse at the point on the line that best represented how they felt at that moment in time. Once the mouse had been clicked the next question was automatically loaded on screen. Images of two paired foods required participants to place their left hand on the 'D' key and right hand on the 'J' key on the keyboard which corresponded to the images on screen. Participants were

instructed to choose which food item they wanted most at that moment in time and focus only on the type of food shown rather than the quantity shown.

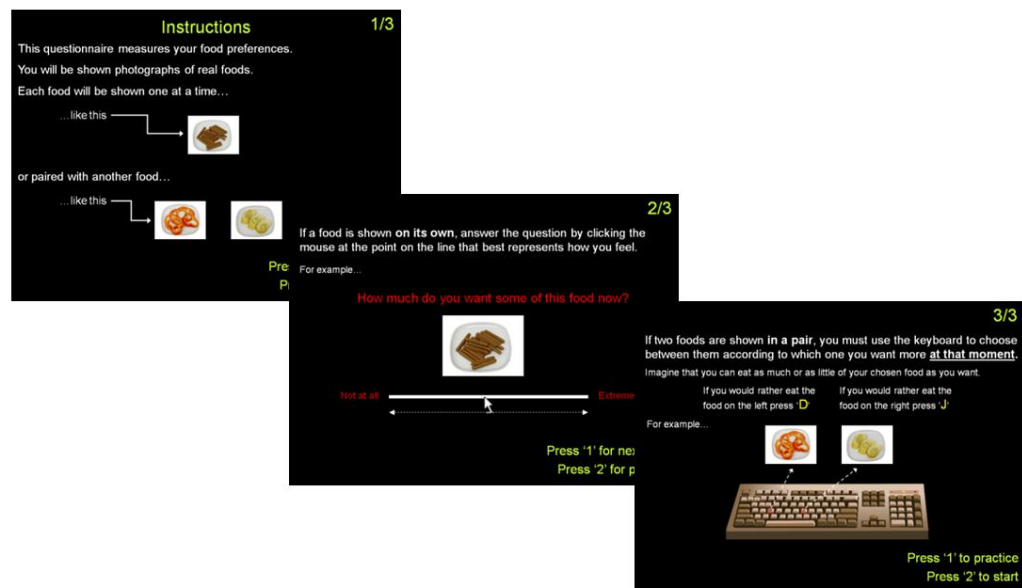


Figure 3.4. Instructions provided to participants prior Leeds Food Preference Questionnaire completion.

3.1.3 Height, Weight, BMI and Body Composition

Height, weight and BMI were confirmed at screening to ensure participant eligibility prior to any measures being completed. Height was measured using a wall-mounted stadiometer to the nearest 0.1cm (Seca Ltd). Weight was measured using electronic weighing scales to the nearest 0.1kg. BMI was calculated by dividing the weight in kilograms by height in metres squared ($BMI = kg/m^2$). Participants wore light clothing and were not required to be in a fasted state whilst measures were conducted. In analysis, the weight provided by the BodPod was utilised.

3.1.3.1 Air-displacement plethysmography

Air-displacement plethysmography is considered to be an accurate method of assessing body composition (Collins & McCarthy, 2003) and is highly correlated with other methods employed including hydrostatic weighing, bioelectrical impedance and dual-x-ray-absorptiometry (Levenhagen et al., 1999). Testing requires approximately 5 minutes and is capable of providing estimates of both fat-mass (FM) and fat-free mass (FFM) within the body,

which are reliable (Vescovi et al., 2001), in addition to total body weight and body fat percentage.

Assessment requires participants to wear tight fitting clothing and remove all jewellery as well as wear a swim cap. Participants are required to sit in the chamber whilst two measurements are taken, during which time they are instructed to sit still and breath normally.

The Bodpod uses body density to determine body composition:

$$\text{Body density} = \text{body weight} / \text{body volume}$$

Boyle's Law – “for a fixed mass of ideal gas at fixed temperature, the product of pressure and volume is a constant” – is used to measure total volume. Participants are required to sit in a chamber which creates a change in air pressure and volume. A diaphragm then measures these changes and Boyle's Law is applied to measure whole body volume. Once overall body density is established, equations relating to body density can be applied to calculate the proportions of FM and FFM within the body. The Siri equation is used to translate whole body density into body fat percentage (%BF):

$$\text{Percent fat} = (495/\text{density}) - 450$$

The percentage of fat-free mass is then calculated using the percentage of fat mass:

$$\text{Percent fat-free mass} = 100 - \text{percent fat}$$

3.1.4 Sensory Visual Analogue Scales (VAS)

VAS scores are consistently used within appetite research to provide continuous monitoring of a range of subjective assessments (Andriessen et al., 2018; Gilbert et al., 2009) and have been established as reliable measures within the field (Rahemtulla et al., 2005). In study 1 VAS responses were completed using a handheld personal digital assistant and with pen and paper (see Appendix 2 for questions asked). In study 2 an online quantitative data platform (QDP) was used, with participants providing responses on a 100mm anchored scale (see Appendix 8 for questions asked).

3.1.5 Ethical Considerations

Both studies required consideration of risk to the participant and approval by the local ethics committee. There was a small risk of food allergies as the two studies involved eating. This risk was minimised by excluding participants with known food allergies, as were any participants with a history of eating disorders excluded, to minimise the risk of distress caused by eating within the laboratory. The food products used in both studies were safe for human consumption.

Measurements taken using the BodPod may trigger claustrophobia, due to the small confined space. Any participants with concerns had the option to exclude this measure. All researchers involved in assessment days were fully trained and experienced at working with participants with overweight and obesity and were able to put any individuals at ease in the case of embarrassment arising due to wearing tight fitting clothing. The option for a male/female researcher for this measure was offered to each participant to prevent embarrassment during assessment.

Both studies required a 12-hr overnight fast to be completed. The risk of a sudden fall in blood glucose (hypoglycaemia) was minimal, however research staff qualified in first aid were available on assessment days.

3.2 General Methodology: Study One, Diet Induced Variability in Appetite (DIVA)

The data presented in Chapters 4, 5, and 6 were collected within a wider research project (Diet-Induced Variability in Appetite – DIVA study, ClinicalTrials.gov reference: NCT03447600).

Only measures and procedures that are relevant to the pertinent research questions outlined are reported.

3.2.1 Design

A repeated-measures, between-subjects design was employed with two separate groups established on the basis of BMI - participants were classified as either ‘overweight’ with a BMI of 25-34.9kg/m² or ‘lean’ with a BMI within a range of 18.5-24.9kg/m². Participant eligibility

was established via an initial screening questionnaire, with those eligible invited to attend the Human Appetite Research Unit (HARU), at the University of Leeds for a pre-screening session (visit 1). Following visit 1, participants completed 7 days of 24-hr food diaries before returning to the lab for an assessment day (visit 2). At visit 2, anthropometrics, body composition and resting metabolic rate were measured, and participants completed the LFPQ at three time points (using a breakfast and lunch version of the LFPQ) and serial appetite VAS. Participants in the overweight and obese group were then allocated one of two diets to follow until $\geq 5\%$ weight-loss was achieved, or 12 weeks had passed and completed a second assessment day after 2 weeks of the diet and attended weekly meetings with a trained dietitian. Elsewhere it is presented that the mean rate of weight loss was similar between groups at week 2, but greater in the CER group (0.8%/week) than the IER group (0.6%/week) through the entire intervention (Casanova et al., 2023). However, due to the small differences between groups it is not anticipated that this will impact outcomes of interest. Once weight-loss had been achieved or the time limit reached, participants completed a third assessment day. Participants in the overweight/obese category were invited to a 1-year follow-up to complete a fourth assessment day. Participants in the lean condition completed a single assessment day only to act as a age-matched lean comparison group.

3.2.2 Participants

Participants from the University of Leeds and surrounding areas volunteered their time for the study. Recruitment methods included posters advertising the study around the University of Leeds campus and surrounding areas, an undergraduate participant pool scheme (in which students obtain credits via study participant in order to progress in their studies) as well as departmental email lists. Power calculations (G*Power v3.1) were calculated prior to the current thesis proposal, and estimated that a sample size of $n=34$ would be necessary to detect interactions in self-selected meal size (*ad libitum* energy intake $\eta^2 = 0.06$) between 2 groups and 2 repeated measurements ($r = 0.5$, based on data from a prior 12-wk intervention (Caudwell et al., 2013)) with $\alpha = 0.05$ and $1-\beta = 0.8$. Significant differences were noted between dietary intervention groups at baseline in analysis of the *ad libitum* energy intake,

demonstrating sufficient power within the sample to detect differences in self-selected meal size (Beaulieu et al., 2020).

Recruitment involved two stages. First, participants characterised as overweight or obese (by BMI) were recruited for a randomized trial examining a dietary weight-loss intervention between February and August 2018. Participants characterised as lean were recruited between March and September 2019 and age-matched to the overweight group based on four age group blocks (18-25, 26-34, 35-43 and 44-54 years of age) – matching the mean age of each block, in order to minimise any age related effects exerting an influence of food preferences and behaviours (Boesveldt et al., 2018).

Participants volunteered their time for the study in return for information regarding their physical activity, metabolism and body composition. In the case of participants defined as overweight, there was the additional incentive of the dietary weight-loss intervention.

Recruitment was restricted to individuals who exercised no more than three times a week, all participants were non-smokers, did not have a history of eating disorders or food intolerances and were not taking any medications that may impact appetite or mood. A full list of the inclusion and exclusion criteria for the DIVA study can be seen in Table 3.1.

Table 3.1. Full inclusion and exclusion criteria for study 1 (DIVA).

Inclusion Criteria	Exclusion Criteria
Female participants aged 18 to 55 years at the time of signing informed consent	Significant health problems which in the opinion of the researcher, may jeopardise participant's safety or compliance with the protocol
BMI of 18.5–24.9 kg/m ² (L)	History of eating disorders including binge eating
BMI of ≥25-34.9 kg/m ² (OW)	Taking medication or supplements known to alter appetite or weight within the past month and/or during the study
	Pregnant, planning to become pregnant or currently breastfeeding
	History of anaphylaxis to food
	Known food allergies of food intolerances
	Smokers and those who have recently ceased smoking (<6 months)
	BMI < 18.5 kg/m ²
	BMI > 35 kg/m ²
	Volunteers having lost significant amount of weight in the previous 6 months (±4kg)
	Volunteers who exercise >3 days per week or have significantly changed their physical activity patterns in the past 6 months or who intend to change them during the study
	Participants receiving systemic or local treatment likely to interfere with evaluation of the study parameters
	Participants who work in appetite or feed related areas.
	Participants who do shift work

Abbreviations: BMI – body mass index. L – lean. OW – overweight.

3.2.3 Procedure

Participants with overweight and obesity were allocated to one of two dietary interventions and thus attended three separate measures days at the lab. Potential participants attended an initial screening session to confirm eligibility, before attending a baseline measures day and being randomly allocated to a diet condition. All participants then returned to the lab at week 2 to complete a second measures day. Weight loss and compliance to the dietary protocols was monitored on a weekly basis during the intervention via meetings with a trained dietitian, once 5% weight loss had been achieved, the participants attended the lab for a third and final, post-

intervention measures day. If 5% weight loss was not achieved during the intervention, a maximum time of 12 weeks was allowed before the post-intervention measures day was completed. A timeline of the diet interventions can be seen in Figure 3.5.

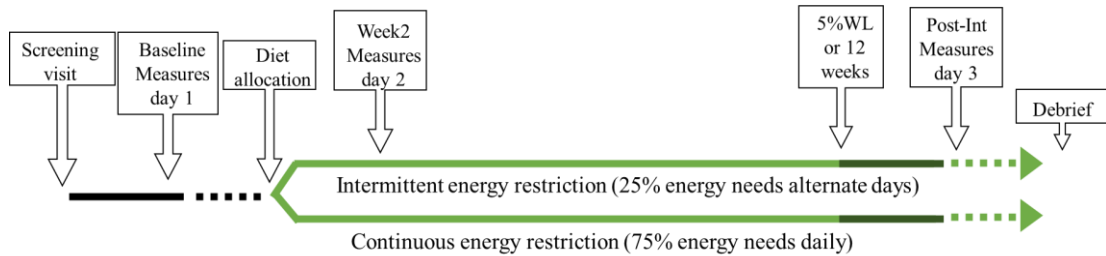


Figure 3.5. Study 1 timeline of diet intervention and measures days for participants with overweight and obesity.

Assessment days were split into two sections: a morning session and an afternoon session, a timeline of each measures day protocol can be seen in. For the morning session, participants arrived at the HARU following completion of an overnight fast, avoiding strenuous physical activity or exercise and alcohol for 24-hrs, and caffeine for 12-hrs prior to the assessment day commencing. The LFPQ breakfast version was completed before body composition was measured using air displacement plethysmography (Bodpod, Concord, USA), followed by resting metabolic rate (RMR) using indirect calorimetry (GEM Nutrition, Cheshire, UK). A standardised breakfast – calculated at 25% of RMR – was provided to participants (see Figure 3.7) and once consumed participants were instructed to return to the HARU exactly 2-hrs 45-mins later to ensure that the initiation of the breakfast and lunch meals were as close as possible to being 3-hrs apart. Participants were instructed to refrain from eating or drinking during this period – with the exception of a bottle of water provided by research staff. Whilst away from the lab, participants were required to answer a series of VAS questionnaires at seven time points. Additional VAS responses were obtained at arrival in the morning, before consumption of the standardised breakfast, and after consumption of the *ad libitum* test meal.

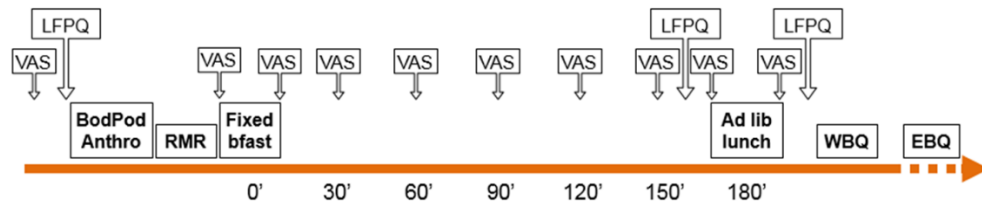


Figure 3.6. Study 1 measures day protocol.

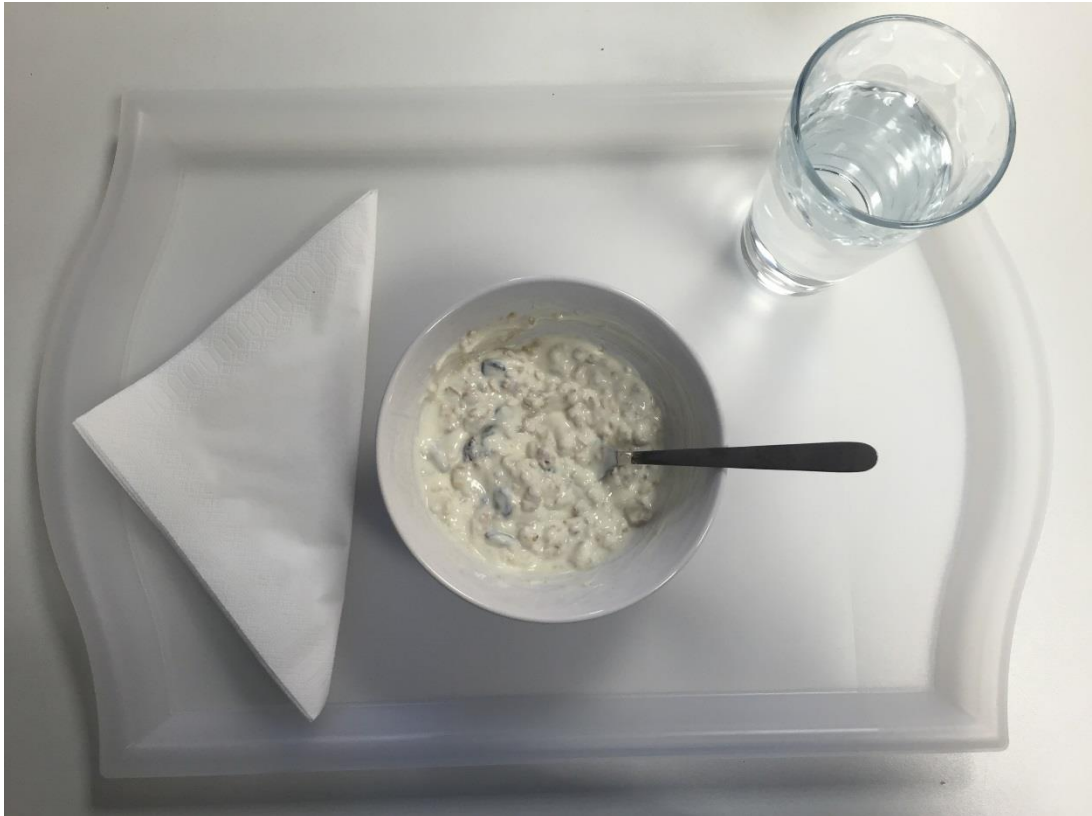


Figure 3.7. Image of standardised breakfast presentation.

The afternoon session began with participants completing the LFPQ for lunch food items and a VAS questionnaire. The *ad libitum* test meal was provided following completion of the questionnaires with participants instructed to “eat as much or as little as you would like until comfortably full”. See Figure 3.8 for the *ad libitum* test meal presentation. Following consumption of the lunch meal, participants completed VAS palatability ratings and the LFPQ for lunch food items a second time. Following completing of the LFPQ participants were free to leave the lab and the assessment day was completed. A full timeline a visit to the lab can be seen in Figure 3.6.



Figure 3.8. *Ad libitum* test meal presentation.

3.2.4 Dietary Interventions

Participants in the overweight/obese category were allocated one of two meal dietary intervention strategies to follow – either continual energy restriction (CER) or intermittent energy restriction (IER). Weight-loss was monitored each week through a weigh-in and discussion with a trained dietitian and energy intake was adjusted if weight-loss was not achieved or plateaued with full adherence. Upon reaching ~5% weight-loss at a weekly weigh in, participants continued a final measures week while continuing the dietary intervention and providing a daily fasted body weight measurement. For those participants that did not achieve a 5% weight-loss, a maximum time of 12-weeks of dietary intervention was allowed.

During CER, participants were required to consume 75% of their daily energy requirements each day from commercially available food products provided by the researchers. Three main meals and snacks were provided and no time restrictions were applied, nor was there specified a specific number of eating episodes. In line with national nutrition guidelines (British Nutrition Foundation), the macronutrient composition of the diet was 50-55% carbohydrates, 15-20% protein and 30-35% fat.

During IER, participants alternated between ‘fasting’ days and *ad libitum* consumption days. During a fasting day, participants consumed 25% of their daily energy requirements from total diet replacement products (LighterLife Ltd) provided by researchers. On the alternate *ad libitum* consumption days, volunteers were free to consume their own foods. The calorie content of each product was similar (~150kcal, ~36% carbohydrates, ~37% protein and ~27% fat) and ensured a daily protein intake of 49.1 ± 8.2 g. Products ranged from 3-5 full packs and an additional bar portion (if required) and were typically evenly distributed throughout the day. If requested, participants were also provided milk portion for hot beverages.

3.2.5 Measures

3.2.5.1 Gas Exchange Monitor

Guidelines outlined by the American Dietetic Association (Compher et al., 2006) were followed to assess RMR using an indirect calorimeter fitted with a ventilated hood. The GEM indirect calorimeter provides a measure of RMR, respiratory quotient and energy expenditure.

Assessment required participants to remain awake and motionless in a supine position for ~40-mins (~10-mins calibration and 30-mins data collection). RMR was taken as the average of the 30-min period. VO_2 and VCO_2 were calculated from O_2 and CO_2 concentrations in inspired and expired air diluted in a constant airflow of ~40 L/min, individually calibrated for each participant and averaged over 30-second intervals. Standard stoichiometric equations used by the software calculated respiratory exchange rate (RER).

3.2.5.2 Fixed energy test meal

A fixed energy test meal allows the energy and nutrient intake to be controlled by the experimenter, enabling standardisation across participants. Participants consumed a standardised breakfast (see Figure 3.7), to navigate the differences in energy requirements the meal was based on 25% of their resting metabolic rate (RMR) – calculated previously using indirect calorimetry (GEM Nutrition, Cheshire, UK). The meal consisted of 66% carbohydrates, 14.1% protein and 19.9% dietary fat. Commercially available products were used (Neal’s Yard Muesli, Neal’s Yard Raisins, Neal’s Yard Sultanas, Yeo Valley Natural

Yoghurt, Sainsbury's Runny Honey, Sainsbury's Semi Skimmed Milk). Participants were provided with a drink of tea, coffee or water. In those participants who did not drink milk in their tea/coffee, or only opted for water, milk was added to the bowl of food to ensure consistency.

Coffee was 5g of Nescafe instant coffee grounds, with 500ml of water added to it and participants were provided with 300g. Tea was made by brewing two Yorkshire tea bags in 500ml of boiling water. Similarly, participants were provided with 300g of tea. If participants opted for neither tea nor coffee, they were provided with 300g of chilled water.

3.2.5.3 *Ad libitum* test meal

Ad libitum test meals require an experimenter to weigh the foods before and after participant consumption to assess the amount of self-determined energy and nutrient intake. *Ad libitum* test meals are highly reproducible (Gregersen et al., 2008) and are more naturalistic than fixed energy meals, allowing participants to determine the amount eaten, similar to in everyday life.

Participants were provided with an *ad libitum* lunch meal consisting of water (500g) and two different commercially available foods (Uncle Ben's tomato and herb risotto and Yeo Valley strawberry flavoured yoghurt), representing a sweet and savoury option, matched for energy density (see Table 3.2). The yoghurt was lower in energy density than the risotto and so flavourless maltodextrin (MyProtein, UK) was included. Portions were provided in excess of consumption although participants were instructed that if desired, more was available. Upon serving the meal, participants were instructed "to eat and drink as much or as little as you like, until you are comfortably full. If you finish, there is more available". The presentation of the *ad libitum* lunch meal can be seen in Figure 3.8.

Table 3.2. Calorie and macronutrient composition of the *ad libitum* test meal.

	Weight (g)	Kcal	CHO (%)	Protein (%)	Fat (%)
Uncle Bens' tomato and herb risotto (+ hot water)	900 (+100)* ¹	1511.2	70.1	8.9	21.0
Yeo Valley strawberry flavoured yoghurt (+Maltodextrin)	425 (+100)* ²	403.2 (+375)* ³	70.5	8.9	19.7

Abbreviations: CHO = Carbohydrates.

*¹ = additional weight of hot water.

*² = additional weight of maltodextrin.

*³ = additional energy supplied by maltodextrin.

3.2.5.4 MyFood24

MyFood24 - an online food diary program was used to measure daily energy intake. This is a validated tool to measure of energy and macronutrient intake and has been shown to be more time efficient measure than interview led recall (Wark et al., 2018). As the software is unfamiliar to participants, participants were screened and shown how to properly complete a day's diary entry. MyFood24 utilised a food and drink database developed using approximately 50,000 commercially available 'back of pack' nutritional labels mapped onto generic data available from 'The Composition of Foods' (McCance & Widdowson, 2014) which was specifically made for MyFood24 (Carter et al., 2015) (Figure 3.10).

Participants provided 7 continuous days of 24 hr food diaries between visit 1 and visit 2. A daily email at 7pm was sent to each participant with the link for that day's food diary – if this was not completed then the following morning a reminder email was issued. Participants selected the meal that an entry was to be added to (i.e. breakfast, lunch, dinner or snack) (Figure 3.9) and searched the online database for the food or drink consumed (Figure 3.10). Participants then either selected a weight or a portion size image selected the portion that best represented their meal (Figure 3.11). This process was repeated for every food and drink consumed on that day.

Figure 3.9. MyFood24 meal selection and database search bar.

Item	Brand	
Porridge made with water and milk	Non brand (generic)	+ Add item
Porridge made with whole milk	Non brand (generic)	+ Add item
Porridge, made with water	Non brand (generic)	+ Add item

Figure 3.10. MyFood24 example of a food search.

Select box or enter weight directly in "Total portion size"

I consumed around
Please select one

60g (1 tablespoon (as made)	110g (small portion (as made up))	160g (average portion (as made up))	210g (large portion (as made up))

Quantity:

Total portion size: g [Need a converter?](#)

Select meal:

What time did you eat?:

Figure 3.11. MyFood24 portion size selection.

3.2.6 Ethical Approval

The present study was granted ethical approval by the University of Leeds, School of Psychology Research Ethics Committee (ethical approval number: PSC-238 and PSC-551). All procedures were explained in full to each participant prior to obtaining informed consent. The specific objectives of the study were not disclosed to the participant to avoid the potential effects of demand characteristics. A full debrief was provided following the final measure on the final assessment day. Participants were informed of their right to withdraw and have any data already collected up until that point destroyed.

3.3 General Methodology: Study Two, Sweeteners & Sweetener Enhancers (SWEET)

The study protocol described herein was conducted within the remit of a wider research project (Clinical trials registration: NCT04633681) and consequently participants completed a number of measures otherwise unreported in the results. Only measures and procedures included in analysis are outlined.

3.3.1 Design

A double blind, randomised crossover trial, utilising a within-subjects design was employed. An initial pre-screening questionnaire established potential participant eligibility, with those deemed eligible invited to an information meeting and subsequent screening session, at which eligibility was confirmed. Participants were offered the opportunity to complete these two sessions virtually, in order to minimise Covid-19 risk. In the event of a virtual screening session, a screening pack was sent via postal service enabling participants to self-report weight, height, hip and waist measurements absent an in-person visit – which were verified at their first visit to the lab. Following confirmation of eligibility, participants were invited to a clinical investigation day (CID) at the Human Appetite Research Unit (HARU) at the University of Leeds (CID1). Anthropometric measurements and protocol compliance were confirmed, a cannula was inserted and participants completed the LFPQ and provided VAS questionnaire responses in fasted states as well as providing a fasted blood sample. Participants consumed the study food and completed a post-ingestion LFPQ response, and provided serial appetite VAS responses alongside serial blood draws.

3.3.2 Participants

Participants were recruited from the University of Leeds and surrounding areas on a voluntary basis. Methods of recruitment included, posters advertising the study around the University of Leeds campus, distribution of flyers to local businesses and residences, departmental mailing lists and social media campaigns.

Strict eligibility criteria were defined and adhered to throughout the recruitment process.

Participants were aged between 18-60 years, with a body mass index (BMI) between 25-35 kg/m² and regularly consumed sugar containing foods and were willing to consume foods containing high-intensity sweeteners. The full details of the eligibility criteria can be seen in Table 3.3.

Table 3.3. Table of inclusion and exclusion criteria for the SWEET study.

Inclusion criteria	Exclusion criteria
<ol style="list-style-type: none"> 1. Age: 18-60 years. 2. BMI: 25 to 35 kg/m². 3. For women: Use of contraceptive methods or not planning to become pregnant for the duration of the study. 4. Regular consumption of sugar-containing foods and willing to consume sugar and artificially sweetened food products. 5. Liking of the study foods. 6. Able to participate on the visit days during normal working hours. 7. Healthy as determined from self-reported medical history or when no relevant medical condition exists. 8. Consuming breakfast (before 10.30am) regularly (at least 5 days per week). 9. Able to understand and be willing to sign the informed consent form, and to follow all the study procedures and requirements. 10. Capacity to store at-home quantity of intervention product 	<p><u>General</u></p> <ol style="list-style-type: none"> 1. Blood donation < 3 months prior to study or for full duration of the study. 2. Food allergy, intolerance, restriction or avoidance of any of the study foods (e.g., veganism). 3. Likelihood for disordered eating. 4. Currently dieting to lose weight. 5. Having lost or gained >4.5 kg in the last 3 months. 6. Smoking or having quit <3 months prior to study. 7. Habitually consuming >14 units/week of alcohol in women or >21 units/week in men in the last 3 months. 8. Performing >10 h of intense physical activity per week in the last 3 months. 9. Previous university or college training related to eating behaviour research. 10. Night or late (after 11 PM) shift work. 11. Self-reported use of drugs of abuse within the previous 12 months. 12. For women: Pregnancy, lactation. 13. Persons who do not have access to either (mobile) phone or internet (this is necessary when being contacted by the study personnel during the study). 14. Insufficient communication in the national language. 15. Proven or suspected inability, physically or mentally, to comply with the procedures required by the study protocol as evaluated by the daily study manager or research staff. 16. Subject's general condition contraindicates continuing in the study as evaluated by the daily study manager or research staff. 17. Simultaneous participation in other relevant clinical intervention studies. <p><u>Medical conditions</u></p> <ol style="list-style-type: none"> 18. Self-reported eating disorders 19. Diagnosed anaemia 20. Diagnosed diabetes (any type) 21. Abnormal gastro-intestinal (gut) function or structure such as malformation, angiodysplasia or active peptic ulcer 22. Active inflammatory bowel disease, celiac disease, chronic pancreatitis or other disorder potentially causing malabsorption.

-
23. History of gastro-intestinal surgery with permanent effect (i.e. surgical treatment of obesity)
 24. Medical history of cardio-vascular disease
 25. Significant liver disease.
 26. Malignancy (cancer) which is currently active or in remission for less than 5 years after last treatment.
 27. Thyroid diseases, except if you are on specific medication on a stable dose for at least 3 months.
 28. Mental health illness (e.g., major depression, bipolar disorders)

Medication

29. Current use or within the previous 3 months of prescription or over the counter medication that has the potential of affecting body weight including food supplements (specific medications will be assessed on a one-to-one basis).
 30. Cholesterol lowering medication if dose has changed during the last 3 months (i.e. the medication is allowed if you have been on a stable dose for at least 3 months)
-

3.3.3 Procedure

Total time in the study for each participant was 70-84 days (as shown in Figure 3.12). Eligible participants were required to attend the HARU at the University of Leeds on 6 occasions for CIDs, with 2 virtual sessions also taking place. Intervention blocks began with probe day 1 (day 1), ended with probe day 2 (day 14) and were spaced apart by, 12 ± 2 day periods of at home testing involving daily consumption of the study food. Intervention blocks were separated by a two week wash-out period.



Figure 3.12. Number of laboratory visits and time commitment per volunteer in the FAST study. Each visit is scheduled in the morning and lasts up to 4 hours.

3.3.4 Screening

Interested participants were invited to complete an initial pre-screening questionnaire with those deemed potentially eligible invited to attend a virtual information meeting. During the information meeting study protocols were described to participants and the opportunity to ask

any questions answered was provided. Participants provided informed consent and signed the GDPR form using online software. Following the information session a pre-screening pack was sent to participants, containing materials to measure height, weight and hip and waist circumference, in addition to a demo study food to complete the food taste test.

During the screening session participants completed the eating attitudes test (EAT-26) and short-food frequency questionnaire (sFFQ) before a food taste test – which were further used to determine eligibility. Once eligibility was confirmed participants completed the socio-demographic questionnaire, SWITCH-food frequency questionnaire (SFFQ), international physical activity questionnaire (IPAQ), and site optional questionnaires (Binge Eating Scale), Three Factor Eating Questionnaire and Perceived Stress Scale. All questionnaires were completed using the Qualtrics data collection platform and exported directly to the consortium data hub. Following successful completion of the screening session participant's CIDs were arranged and a pre-CID pack was sent via postal service containing: a urine collection pack and instructions on how to collect samples and participant instruction booklet.

3.3.5 Clinical Investigation Days

Participants arrived at the HARU after completing an overnight fast (minimum of 12-hrs but not exceeding 15-hrs) and maintaining habitual levels of physical activity and eating patterns. Upon arrival at CID1, participants' height and weight were confirmed before completing the protocol compliance questionnaire and Control of Eating Questionnaire. Body composition was measured using air displacement plethysmography (BodPod, Concord, USA) and 200ml of water was provided to participants to standardise thirst levels and facilitate blood taking procedures (not included in analysis). A trained researcher inserted a cannula into the antecubital vein with the participant in a seated position 15minutes prior to the first sample draw. Participants completed a fasting sensation VAS response followed by LFPQ (-12mins) (see Appendix 9 for images used at the two different sites), with a second appetite VAS (-2mins). Participants were served three of the intervention biscuits (0mins) and instructed to "take one bite and complete the '1 bite' sensory-specific satiety and expected satiety questions" on the QDP on a desktop computer. Once these were completed participants received 9minutes

to consume the portion in full with 200ml of water – a timer was placed in the cubicle with participants with a researcher prompting consumption if necessary. Participants were instructed to spread consumption of the portion across the entire 9minutes. Post-consumption, serial sensation VAS responses and blood draws were obtained (times shown in

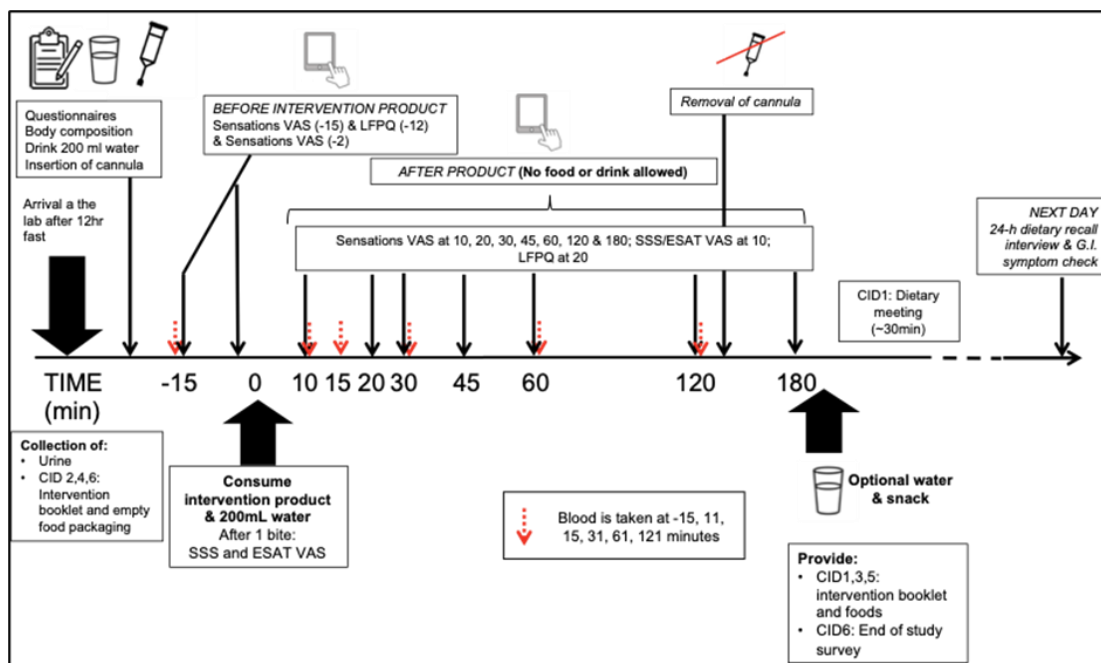


Figure 3.13) with a post-consumption LFPQ (20mins). At 120mins post-consumption the cannula was removed and at 180mins the final sensation VAS was recorded. During CID1, 3 and 5, a dietary discussion took place with a member of the research team using responses from the SWITCH food frequency questionnaire to develop a substitution strategy (see section 3.3.6) and a urine collection kit was provided for the next CID. During CID2 and 4 instructions were given for the washout period and for CID6 only, the end of study survey was completed and £200 compensation provided.

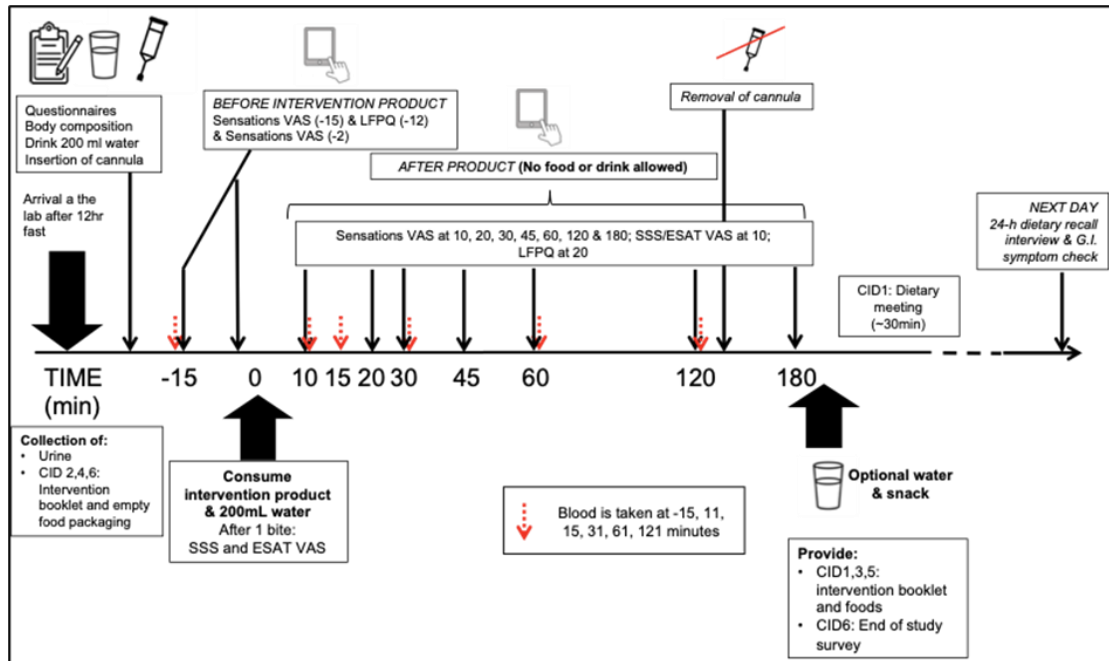


Figure 3.13. Timeline of Clinical Investigation Days during the SWEET protocol.

3.3.6 Substitution Strategy

At the end of CID1 a trained member of the research team explained the substitution strategy to participants and the same strategy was employed at each intervention period unless requested to be changed by participants. The approximate time of eating was agreed participants were asked to return any empty packaging to ensure compliance. Participants were instructed that in the event of a missed dose, the biscuits could be consumed later on in the day, but could not be consumed the next day.

Responses obtained via the SWFFQ informed the substitution strategy, with a substitution based on 245-405kcal based on the control products being $325\text{kcal} \pm 25\%$. Ideal foods to substitute were those most closely resembling the intervention product. However, if multiple foods from the SWFFQ are consumed at a low frequency, these foods were able to be combined within the overall strategy. Ultimately, the strategy was discussed with and agreed upon by the participant so that a sweet food was substituted with the intervention product each day.

3.3.7 Wash-out Period

Following CIDs 2 and 4, participants completed a 2-week wash-out period. During this time participants were instructed to keep their diet and physical activity levels as habitual as possible.

3.3.8 Measures

All participants completed the following measures unless otherwise stated.

3.3.8.1 Eating Attitudes Test 26 (EAT-26)

The Eating Attitudes Test 26 (EAT-26) is a 26-item questionnaire used to detect the likelihood of eating disorders. It represents the most widely utilised self-report measure of symptoms and characteristics of eating disorders and is a refined version of the original EAT-40 and has been validated against the diagnostic and statistical manual version IV (DSM-IV) eating disorder criteria (Mintz & O'Halloran, 2000). The EAT-26 is not designed to provide a diagnosis of an eating disorder, although does provide a cut-off score and is to be used as an objective index of symptoms frequently observed in anorexia nervosa (Garner & Garfinkel, 1979).

In the present study the EAT-26 was used as a screening tool, with participants providing a score >20 considered ineligible for inclusion in the study (Appendix 3).

3.3.8.2 Short Sweet Food Frequency Questionnaire (sFFQ)

The short sweet food frequency questionnaire is a 2 part self-report assessment of habitual sweet food consumption. It has not been validated but has been developed based on the previously published SWITCH questionnaire (Masic et al., 2017).

The sFFQ was used as a screening tool, with only participants scoring a score of ≥ 3 in part Part 1 and answer Yes to all questions in Part 2 deemed eligible to volunteer (Appendix 4).

3.3.8.3 International Physical Activity Questionnaire (IPAQ)

The international physical activity questionnaire (IPAQ) is a 27-item validated questionnaire assessing participant's habitual physical activity patterns (Booth, 2000). The IPAQ consists of four domains: i) transportation, ii) work, iii) household and gardening tasks and iv) leisure time

and has been shown to have acceptable patterns of validity when assessing physical activity in healthy adults (Hagströmer et al., 2006).

3.3.8.4 SWITCH Sweet Food Frequency Questionnaire (SWFFQ)

The SWITCH Sweet Food Frequency Questionnaire is a 39-item questionnaire assessing the frequency of eating particular types of foods. The questionnaire deciphers frequency of consumption of sugar and sugar free alternatives for various categories of foods. This questionnaire has not been validated but has been used in previous research studies (Masic et al., 2017).

The SWFFQ was used to develop a substitution strategy for the at-home intervention periods.

3.3.8.5 Socio-Demographic Questionnaire

The socio-demographic questionnaire (SDG) is a 7-item questionnaire regarding ethnic origin, education, marital status, household composition and employment status. This questionnaire has not been validated.

3.3.8.6 Protocol Compliance Questionnaire

The protocol compliance questionnaire consists of 4-items, with a yes/no or optional open-ended response related to protocol aspects (fasting, alcohol intake, exercise and general diet).

3.3.8.7 Control of Eating Questionnaire (CoEQ)

The CoEQ is a 21-item questionnaire with twenty VAS questions and one open ended question. Responses culminate in four outcome measures: craving control, craving for sweet, craving for savoury and positive mood. The CoEQ has been demonstrated to have acceptable internal consistency and has been validated (Dalton et al., 2015) (Appendix 5).

3.3.8.8 24-h Dietary Recall

A trained member of the research team conducted a telephone interview 24-hrs after leaving the lab after each CID (CID+1). In this, participants were asked to recall everything that had been eaten and drunk since leaving the lab the previous day. To aid in the participant's recollection, the Australian Food Portion Guide was provided to all participants and space to write down

everything consumed was provided in the participant intervention booklet. Data were then manually entered in WinDiets, an online analysis software (Wise, 2008).

WinDiets requires a researcher to manually search for a food or drink item (e.g., jam filled biscuit) and select the most suitable option available. The time of the day that the food or drink item is consumed, as well as the portion size is entered. WinDiets software collates this information and provides a downloadable output, providing energy and macronutrient total values.

3.3.8.9 Binge Eating Scale (BES)

The BES is a 16-item questionnaire relating to the presence of binge eating behaviours. 8-items reflect behavioural characteristics in the participants relating to the amount of food eaten. An additional 8-items reflect the emotional aspects (e.g., guilt or shame) associated with food consumption. The BES provides an assessment of binge eating as a behavioural trait and is not suitable for a diagnosis of Binge Eating Disorder (Appendix 6).

3.3.8.10 Three Factor Eating Questionnaire (TFEQ)

The TFEQ is a 51-item questionnaire consisting of 21-items assessing cognitive restraint, 16-items assessing disinhibition and 14-items assessing hunger. The TFEQ has been demonstrated to be valid (Stunkard & Messick, 1985) (Appendix 7).

3.3.9 Ethical Approval

The present study was granted ethical approval by the University of Leeds, School of Psychology Research Ethics Committee (ethical approval number: PSYC-127; 19-Nov-2020) and was in compliance with the Declaration of Helsinki. Prior to the commencement of the study, all procedures were explained in full to the participants before obtaining informed consent. To avoid the confounding effects of demand characteristics, the project was described as ‘this study will look at the immediate and medium-term effects of consuming foods sweetened with sugar or sweeteners on blood markers, body sensations and consumer acceptance’. Following the completion of all measures during CID6, the aim of the study was

provided to participants. All participants were informed of their right to withdraw, anonymity and signed a general data protection regulation document (GDPR).

3.3.10 Statistical Analysis

All data were analysed using Statistical Package for the Social Sciences v.25 (SPSS; IBM Corporation, Somers, New York). All data in study were exported into MS Excel which was used to calculate variables for export into SPSS. LFPQ data were collected using E-Prime v.2.0 software and exported into MS Excel.

Specific statistical procedures can be seen in greater detail in each experimental chapter.

4 The stability of sweet food preference within one day and after diet-induced weight loss

Aims: Sweet food preferences are subject to individual variability but may alter within the day or after weight loss. The stability of individual differences in sweet food preference (e.g., strong sweet likers) is often assumed but rarely tested in different acute or chronic states. The aim of the following chapter is to assess 1) the stability of sweet food preference in different states following an overnight fast, in a non-fasted state and following consumption of an *ad libitum* lunch meal, and 2) the stability of sweet food preferences taken at the same time-points at baseline, after 2 weeks and after completion of a dietary weight loss intervention. The outcome will provide evidence for the extent to which sweet food preference is a stable trait and will address the second outcome listed in section 1.8.1.

4.1 Introduction

Some people exhibit a strong liking for sweet foods, popularly referred to as possessing a ‘sweet tooth’. Currently, there is no standardised method of assessing sweet liker status, thereby generating difficulties with cross study comparisons (Yang et al., 2019) and raising the uncertainty as to whether a sweet food preference is an enduring characteristic. Without a reliable method to define sweet food preferences, the ability to identify factors affecting food preferences and between-group differences is limited. However, limited evidence is available regarding the reproducibility of sweet food preferences which has produced conflicting reports (Asao et al., 2012; Coulon et al., 2012). Sweet preferences have been suggested to be reproducible over the short-term (e.g., 9 days and 15 days) in individuals with substance dependence and psychiatric disorders (Kampov-Polevoy, 2001; Kampov-Polevoy et al., 2003) or through responses to sucrose solutions (Asao et al., 2012) and the use of VAS measurements (Coulon et al., 2012). This is problematic as the scales and stimuli utilised may not be capable of accurately capturing various forms of sweetness in foods or varying presentations of

sweet foods. Furthermore, the reliability of other aspects of food preferences such as liking vs wanting have not been examined (Asao et al., 2012).

Evidence has demonstrated that when deprived of energy, the reward value of a food increases in healthy weight participants (Epstein et al., 2003; Raynor & Epstein, 2003). It is possible that this increase occurs as a means of obtaining nutrients and energy, suggested so by the appeal of energy-dense foods being greater than that of lower energy foods when in a fasted state (Goldstone et al., 2009). Similarly, it was reported by Zverev (2004) that short-term energetic deprivation is associated with significant increases in taste sensitivity in response to sucrose solutions in lean individuals – suggesting that when in a fasted state the hedonic value of a sweet food may be elevated relative to when in a non-fasted state. However, when comparing individuals with obesity to lean participants, fasting has been shown to increase gaze duration to food images in both groups (Castellanos et al., 2009). This evidence may suggest that the influence of being in a fasted state on the stability of sweet food preferences may not be impacted by body weight.

It has been stated that food liking and wanting can be diminished by satiety, but cannot be completely eliminated (Berridge et al., 2010; Cabanac, 1971). However, the extent of this diminishment may vary between individuals. In one study, pleasantness ratings declined from the start to the end of a test meal in all participants, whereas palatability demonstrated variability. Although the majority of participants (72%) rated palatability as declining in a similar manner to that of pleasantness, a separate set of participants (28%) reported no change in palatability across the meal (Yeomans & Symes, 1999). Similarly in another study, the hedonic value of sweet tastes did not change in sweet likers across nutritionally deprived and satiated states, whereas sweet dislikers displayed an attenuated dislike for concentrated sucrose when in a deprived state (Looy & Weingarten, 1991). Consequently, the magnitude of decline in the hedonic value of sweet foods from satiety related influences may vary between individuals.

Moreover, taste preferences are altered following successful weight loss (Burgess et al., 2016), although these alterations are likely caused by secondary effects of the obese state (Berthoud & Zheng, 2012) such as differences in hedonic hunger (Ribeiro et al., 2018). For example, in participants with obesity following a 12 week weight loss program was shown to produce a reduction in hedonic hunger (O'Neil et al., 2012). Similarly, dietary induced weight loss has been shown to produce a decrease in liking in participants with overweight or obesity - particularly for high-fat, high-carbohydrate and low-energy products (Andriessen et al., 2018). Previous research using the current data set, reported a reduction in liking for images of food from sweet, savoury, high and low fat food categories from baseline to post $\geq 5\%$ weight loss (Oustric et al., 2021). Alternatively, contrary evidence suggested that sweet taste preference is not lowered following weight loss (Rodin et al., 1976). In participants having undergone Roux-Y gastric bypass surgery, hedonic taste ratings of sucrose remained unchanged post-surgery (Bueter et al., 2011), a finding repeated elsewhere following an intensive medical weight loss program (Asao et al., 2016). However, it is important to note that O'Neil (2012) specified an upper BMI limit of 35kg/m^2 , whereas in both Bueter (2011) and Asao (2016) the mean post-weight loss BMI was greater than this value (38.4kg/m^2 and 36.7kg/m^2 respectively). Subsequently, the extent to which the stability of sweet food preferences are dependent on body weight or alterations to body composition remains uncertain.

The evidence outlined above suggests that the nutritional status of an individual may result in alterations to sweet taste preferences in a multitude of ways. However, there is limited evidence directly investigating the stability of sweet taste preferences in different acute or chronic nutritional states. One study demonstrated high short-term (3-7 days) reproducibility of a forced-choice staircase procedure using varying concentrations of sucrose in participants with a range of BMIs (Asao et al., 2012), whilst another demonstrated moderate strength correlations between measures recorded 7 ± 2 days apart (Coulon et al., 2012). Given that taste preferences are often shown to determine eating habits (e.g., Asano et al., 2016; Dotson et al., 2012; Drewnowski, 1997, 1999), it is imperative to understand the extent to which sweet food preferences are acutely and chronically stable traits. The following chapter aims to investigate

the stability and reproducibility of sweet food preferences across the morning in a fasted (pre-breakfast), non-fasted (pre-lunch) and fed (post-lunch) state, in a sample of lean women and women with overweight or obesity, and at the same times of day at baseline, and in response to diet-induced weight loss in those with overweight/obesity.

Hypotheses:

- Sweet food preferences will be elevated in a fasted and fed state than a non-fasted state.
- There will be a reduction in sweet food preference in response to weight loss in participants with overweight/obesity.
- Sweet food preferences will display a degree of stability acutely across a single day in different nutritional states.
- Sweet food preferences will display a degree of stability pre, during (2-weeks) and after ($\geq 5\%$ weight loss achieved) a dietary weight loss intervention.

4.2 Method

Ninety-four participants (with a mean age of 35.1 years, SD 10.2 years) were recruited on a voluntary basis at the University of Leeds and surrounding locations. Eligibility was determined using an online screening questionnaire. Participants were recruited separately as women with overweight or obesity, and lean women – determined by BMI, although for the purposes of the present chapter were combined into a single homogenous group. Eligible participants were invited to a screening session to confirm eligibility and screen the images used during the LFPQ task (for further details see section 3.1.2). This study was approved by the University of Leeds School of Psychology Ethics Committee (ref: PSC-238, date: 10-01-2018).

All measures were conducted within the HARU at the University of Leeds – except the screening questionnaire which was completed online. Participants arrived at the HARU after completing a 12-hr overnight fast and avoiding alcohol intake and physical activity for 24 hrs. Participants completed the LFPQ breakfast version before body composition was measured using air displacement plethysmography (Bodpod, Concord, USA). Following consumption of the sweet breakfast participants were free to leave the lab and return 2-hrs 45-mins later – ensuring breakfast and lunch meals were as close to 3-hrs apart as possible (further details see section 3.2.5.2 and section 3.2.5.3). Upon returning to the lab, participants completed the LFPQ lunch version before being provided an *ad libitum* lunch meal, representing savoury (risotto) and sweet (yoghurt) options, matched for energy density. Following consumption of the *ad libitum* lunch meal, participants completed the LFPQ lunch version a second time.

Participants with overweight or obesity completed a baseline measures day, before beginning a weight loss intervention (for further details, see section 3.2.4). Participants completed a second measures day at 2-weeks, and a third measures day once weight loss $\geq 5\%$ was achieved or at 12-weeks – depending on which occurred first. Participants were randomly allocated to one of two dietary weight loss interventions (CER or IER), for analyses of the effects of the separate intervention protocols on sweet food preferences see Chapter 6.

4.2.1 Data Analysis

Data were analysed using Statistical Programme for Social Sciences (SPSS) Version 25. For all analyses, an α -level of .05 was used to determine significance. Sweet food preference was operationalised through EL, EW, IW and choice (frequency of selection) sweet bias. High fat sweet (HFSW) and low fat sweet (LFSW) outcomes are also included in analyses. For further details regarding the LFPQ please see section 3.1.2. The differences between LFPQ outcomes across the day were assessed using a series of repeated measures ANOVAs (time x LFPQ outcome). Differences across baseline, week2 and post-intervention were examined using a series of repeated measures ANOVAs (time x LFPQ) outcome. For significant effects, post-hoc analyses with a Bonferroni correction for multiple comparisons were conducted. LFPQ outcomes included overall sweet bias, HFSW and LFSW. Where appropriate Greenhouse-Geisser probability levels were used to adjust for non-sphericity. Bivariate Pearson's correlations were used to investigate the strength of associations between mean LFPQ outcomes across the day.

Finally, the intraclass correlation coefficient (ICC) was used to investigate the strength of associations within individuals between LFPQ outcomes on the same day in both lean and individuals with overweight or obesity, and the same time point assessed at baseline, after 2 weeks and post-intervention. Previous studies have utilised the intraclass correlation coefficient (ICC) to assess the reproducibility of appetite scores in boys (Bellissimo et al., 2008) as well as the agreement between daily average and weekly appetite scores in adults (Womble et al., 2003). As such, the present chapter utilises the ICC analysis via a two-way mixed models approach and strength of scores as set out by (Koo & Li, 2016) with an r value rated as poor 0.5 and below, moderate 0.5-0.75, good 0.75-0.9 and excellent 0.9 and above. Adjustments for multiple associations were not made.

4.3 Results

Participant descriptive statistics (means and standard deviations) are presented in Table 4.1 with the means for the full study sample outcomes produced by the LFPQ ($n=94$) displayed in Table 4.2. Mean sweet bias scores were elevated at breakfast, declined at pre-lunch to provide negative values before increasing post-lunch, whilst remaining below breakfast values.

Table 4.1. Descriptive statistics for full study sample ($n=94$).

	Mean (SD)	Minimum	Maximum
Age (years)	35.1 (10.2)	20.0	55.0
Height (cm)	164.8 (7.3)	152.5	186.0
Weight (kg)	69.3 (14.0)	44.6	112.4
BMI (kg/m ²)	25.4 (4.2)	18.2	34.6
Body Fat %	34.2 (8.9)	34.2	55.9
Fat Mass (kg)	24.7 (10.8)	4.5	59.4
Fat-Free Mass (kg)	44.7 (5.4)	35.5	61.5
Waist (cm)	84.0 (11.7)	59.9	112.4
Hip (cm)	102.3 (9.4)	85.9	124.8
W:H Ratio	0.8 (0.1)	0.7	1.0
Risotto (g)	409.7 (125.3)	68.7	723.3
Yoghurt (g)	162.9 (96.7)	0.0*	428.6
Total Intake (g)	572.6 (162.6)	167.5	973.4
Risotto % Total Intake	70.8 (17.6)	0.0	100.0

Abbreviations: SD = standard deviation. BMI = body mass index. W:H = waist:hip.

*One individual did not consume any yoghurt and was excluded from analysis where necessary.

Table 4.2. Means and Standard Deviations from Leeds Food Preference Questionnaire for full study sample ($n=94$).

	Breakfast M(SD)	Pre-Lunch M(SD)	Post-Lunch M(SD)
EL Sweet Bias (mm)	11.56 (20.25)	-4.21 (20.86)	17.88 (20.25)
EW Sweet Bias (mm)	9.18 (19.15)	-7.66 (22.11)	11.37 (15.70)
IW Sweet Bias (ms)	13.2 (37.6)	-20.9 (40.1)	35.5 (33.7)
Choice Sweet Bias (count)	4.77 (13.51)	-7.74 (15.13)	13.02 (12.92)
EL HFSW (mm)	51.25 (22.59)	54.47 (24.20)	36.54 (26.00)
EL LFSW (mm)	67.08 (19.34)	53.04 (20.12)	41.26 (25.43)
EW HFSW (mm)	45.37 (23.94)	46.84 (25.11)	19.68 (18.51)
EW LFSW (mm)	63.78 (20.69)	46.27 (21.02)	26.18 (22.90)
IW HFSW (ms)	-10.8 (28.4)	-7.5 (27.0)	5.0 (27.0)
IW LFSW (ms)	23.5 (34.1)	-13.4 (27.0)	30.5 (22.7)
Choice HFSW (count)	20.72 (10.64)	21.23 (11.56)	26.63 (10.24)
Choice LFSW (count)	32.04 (11.88)	18.60 (10.10)	34.39 (8.18)

Abbreviations: M = mean. SD = standard deviation. IW = implicit wanting. EW = explicit wanting. EL = explicit liking. HFSW = high fat sweet. LFSW = low fat sweet.

4.3.1 Differences in Sweet Food Preferences in Fasted, Non-fasted and Fed states

4.3.1.1 Explicit Liking

Repeated measures ANOVAs was conducted to compare the difference in means for EL sweet bias, HFSW and LFSW at three time points (breakfast vs. pre-lunch vs. post-lunch). For EL sweet bias, sphericity was assumed ($p=.776$) and results showed a main effect of time, $F(2,184) = 38.333, p<.001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p<.001$), breakfast and post-lunch ($p=.037$), and pre-lunch and post-lunch ($p<.001$) and shown in Figure 4.1.

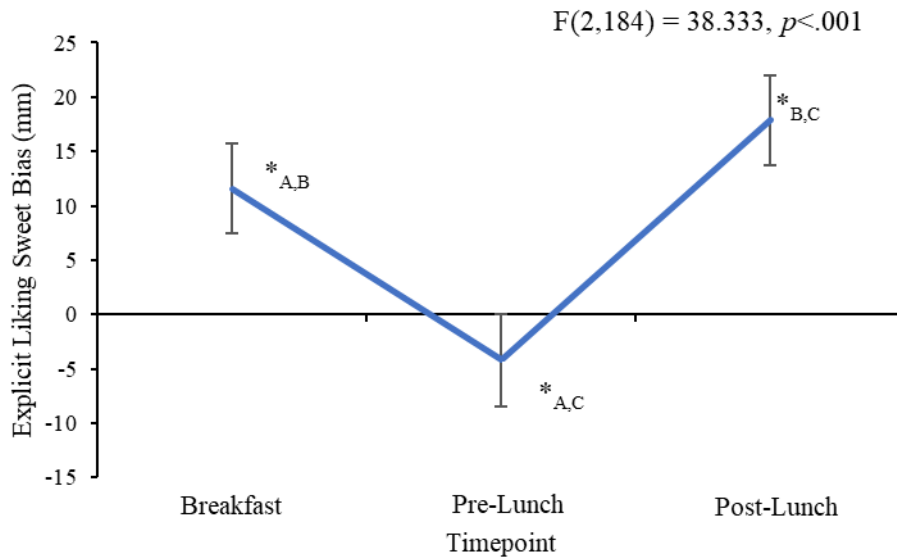


Figure 4.1. Changes in Explicit Liking Sweet Bias across baseline.

Error bars represent standard deviation.

* = significant difference between scores.

For EL HFSW, sphericity was violated ($p < .001$) and a significant main effect of time was shown, $F(1.727, 158.847) = 24.212, p < .001$. Pairwise comparisons showed significant differences between breakfast and post-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$), but not between breakfast and pre-lunch ($p = .405$). For EL LFSW, sphericity was violated ($p = .040$) and results showed a significant main effect of time, $F(1.872, 172.241) = 48.623, p < .001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p < .001$), breakfast and post-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$). Changes in mean EL HFSW and LFSW can be seen in Figure 4.2.

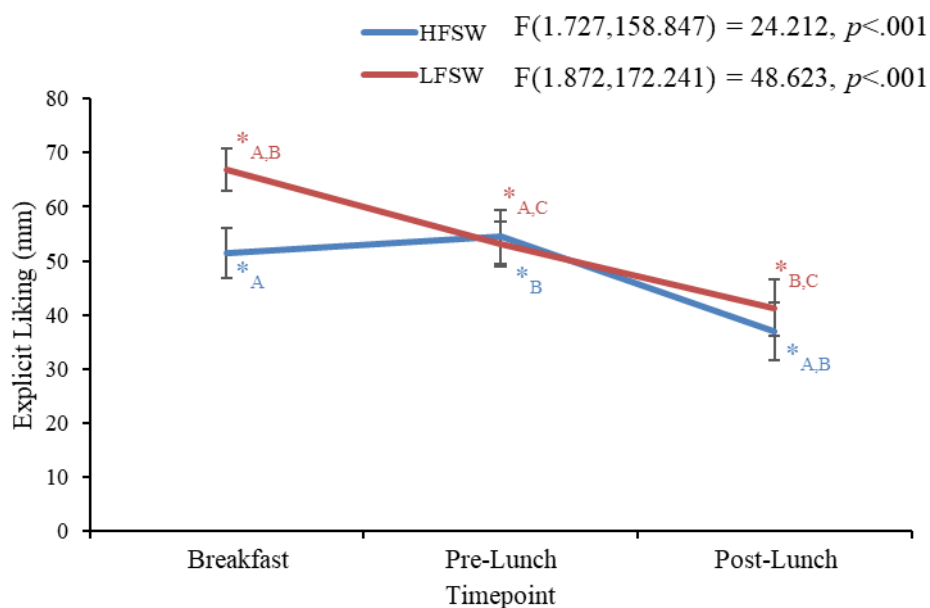


Figure 4.2. Changes in Explicit Liking High Fat Sweet and Low Fat Sweet across Baseline. Error bars represent standard deviation.

* = significant difference between scores.

4.3.1.2 Explicit Wanting

Repeated measures ANOVAs were conducted with time (3 levels) comparing the differences in explicit wanting for EW sweet bias, HFSW and LFSW. For EW sweet bias, sphericity was assumed ($p = .147$) and a significant main effect of time was displayed, $F(2, 184) = 32.422, p < .001$. Pairwise comparisons showed significant differences between breakfast and pre-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$). However, the difference between breakfast and post-lunch was non-significant ($p > .99$) (see Figure 4.3).

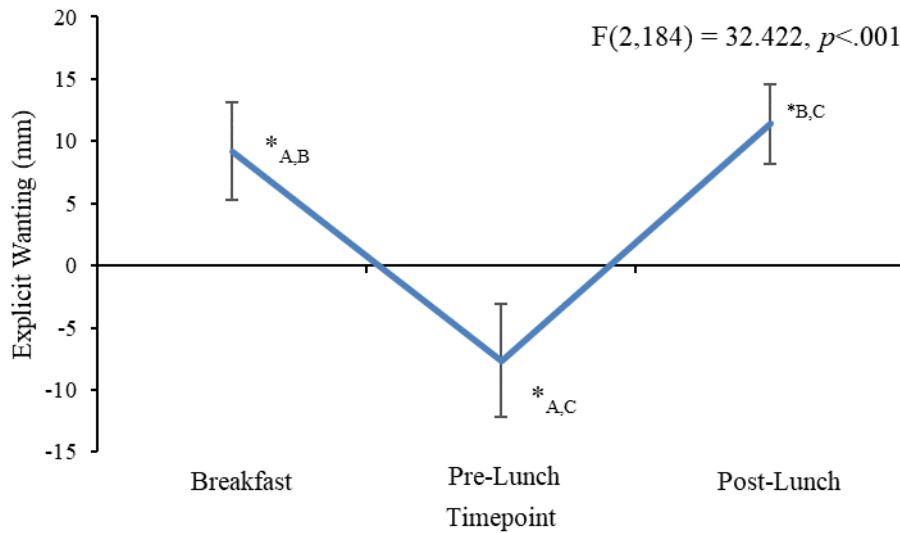


Figure 4.3. Change in Explicit Wanting Sweet Bias within a single day. Error bars represent standard deviation. * = significant difference between scores.

For EW HFSW, sphericity was violated ($p=.002$) and a significant main effect of time was shown, $F(1.765,162.421) = 66.039, p < .001$. Pairwise comparisons showed a non-significant difference between breakfast and pre-lunch ($p > .999$) but significant differences between breakfast and post-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$). For EW LFSW, sphericity was assumed ($p=.095$) and a significant main effect of time was shown, $F(2,184) = 91.261, p < .001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p < .001$) and post-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$). Changes in EW HFSW and LFSW can be seen in Figure 4.4.

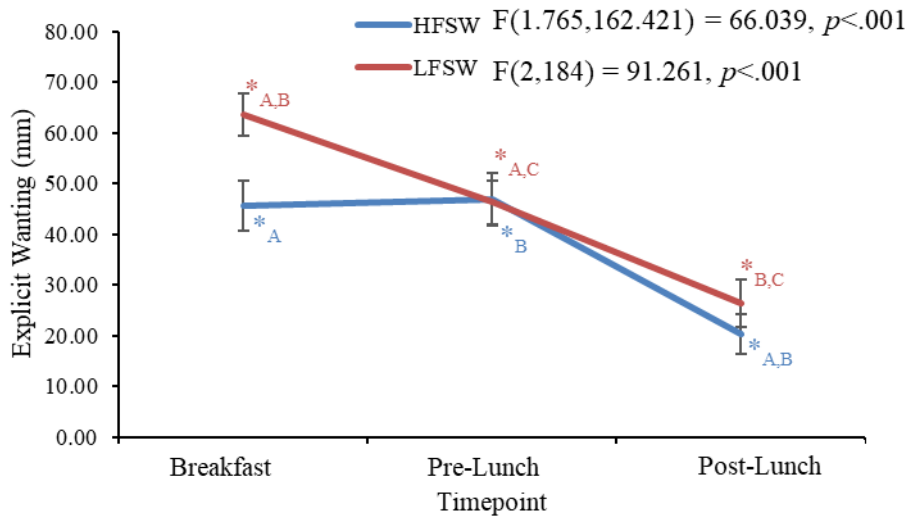


Figure 4.4. Changes in Explicit Wanting High Fat Sweet and Low Fat Sweet within a single day.

Error bars represent standard deviation.

* = significant difference between scores.

4.3.1.3 Implicit Wanting

Repeated measures ANOVAs were conducted with time (3 levels) comparing the differences in implicit wanting (IW) sweet bias, HFSW and LFSW. For IW sweet bias, sphericity was assumed ($p = .382$) and results displayed a significant main effect of time, $F(2, 184) = 74.566, p < .001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p < .001$), pre-lunch and post-lunch ($p < .001$) and breakfast and post-lunch ($p < .001$) (see Figure 4.5).

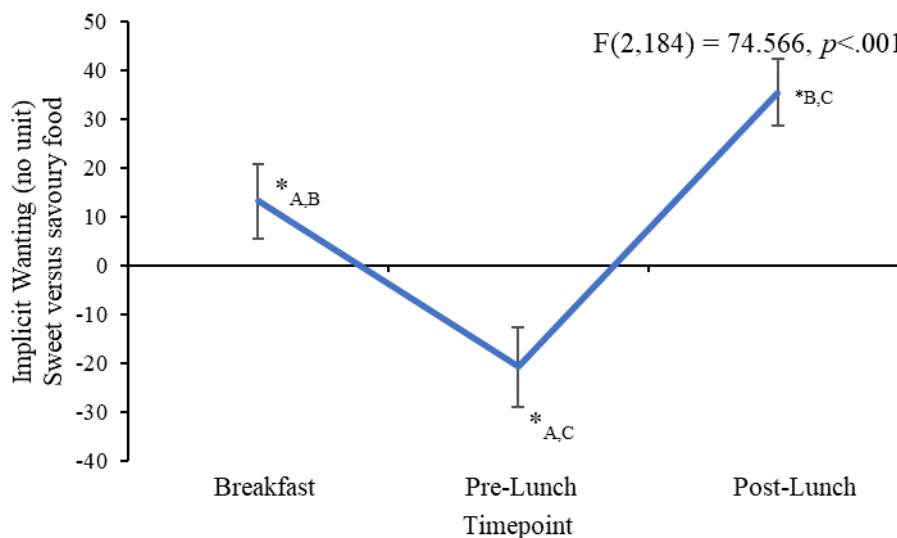


Figure 4.5. Change in Implicit Wanting Sweet Bias within a single day.

Error bars represent standard deviation.

* = significant difference between scores.

For IW HFSW, sphericity was violated ($p=.004$), and a significant main effect of time was shown, $F(1.792,164.858) = 13.475, p<.001$. Pairwise comparisons showed a non-significant difference between breakfast and pre-lunch ($p=.635$), but significant differences between breakfast and post-lunch ($p<.001$) and pre-lunch and post-lunch ($p<.001$). For IW LFSW, sphericity was assumed ($p=.062$) and a significant main effect of time was shown, $F(2,184) = 97.726, p<.001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p<.001$) and pre-lunch and post-lunch ($p<.001$), but a non-significant difference between breakfast and post-lunch ($p=.197$). Changes in IW HFSW and LFSW are shown in Figure 4.6.

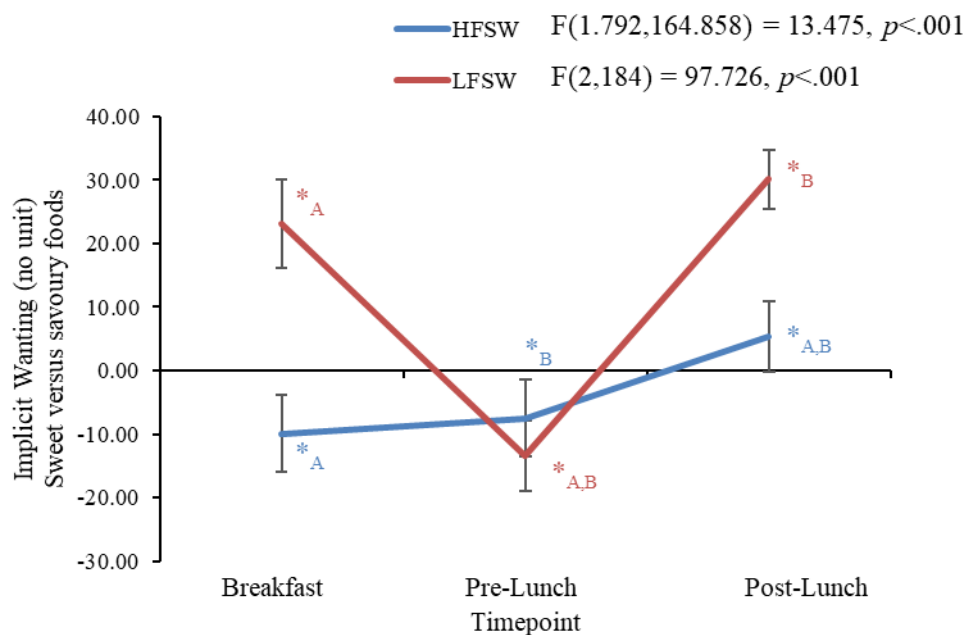


Figure 4.6. Changes in Implicit Wanting High Fat Sweet and Low Fat Sweet within a single day.

Error bars represent standard deviation.

* = significant difference between scores.

4.3.1.4 Food Choice

Repeated measures ANOVAs were conducted to compare the differences in means for choice sweet bias, HFSW and LFSW at three time points in different nutritional states (fasted - breakfast vs. non-fasted - pre-lunch vs. fed - post-lunch). Results showed sphericity was assumed ($p=.322$) and a main effect of time on Choice sweet bias, $F(2,184) 69.778, p<.001$.

Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p < .001$), breakfast and post-lunch ($p < .001$), and pre-lunch and post-lunch sweet bias scores ($p < .001$). Figure 4.7 displays the changes in mean sweet bias outcomes within one day.

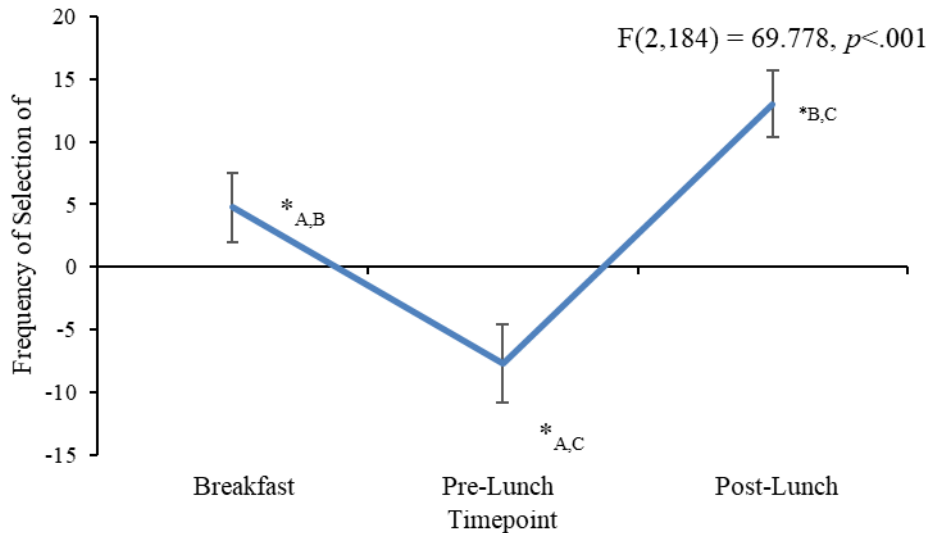


Figure 4.7. Change in Choice Sweet Bias within a single day.

Error bars represent standard deviation.

* = significant difference between scores.

For Choice HFSW, sphericity was assumed ($p = .169$). Results showed a significant main effect of time, $F(2,186) = 13.947, p < .001$. Pairwise comparisons showed a significant difference between breakfast and post-lunch ($p < .001$), and pre-lunch and post-lunch ($p < .001$). Differences between breakfast and pre-lunch were non-significant ($p > .99$). For Choice LFSW, sphericity was assumed ($p = .067$) and results showed a significant main effect of time, $F(2,186) = 95.744, p < .001$. Pairwise comparisons showed a significant difference between breakfast and pre-lunch ($p < .001$) and pre-lunch and post-lunch ($p < .001$). Differences between breakfast and post-lunch were non-significant ($p = .251$). Changes in Choice HFSW and LFSW can be seen in Figure 4.8.

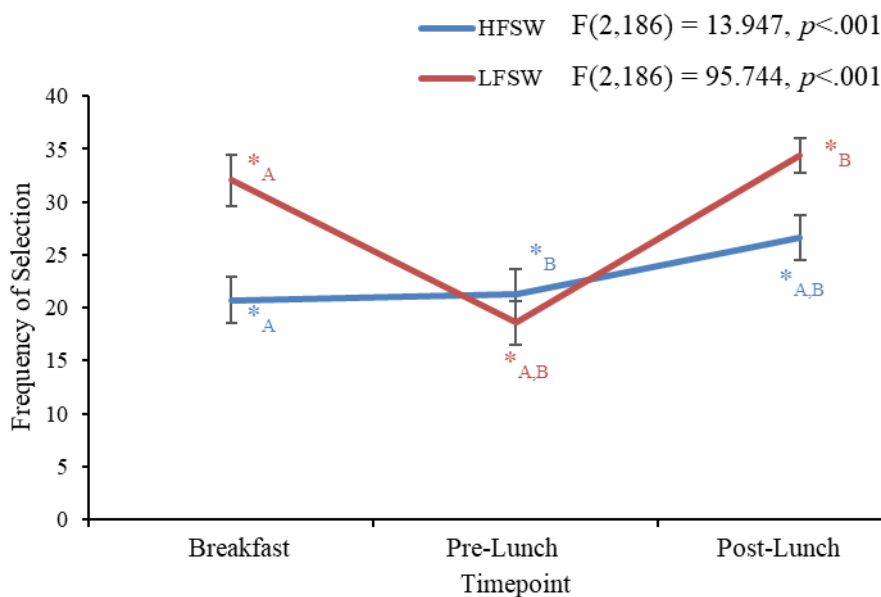


Figure 4.8. Changes in mean Choice High Fat Sweet and Low Fat Sweet within a single day. Error bars represent standard deviation. * = significant difference between scores.

4.3.2 Association between Sweet Food Preferences in different states within one day

Bivariate Pearson's correlations were conducted to assess the strength of the associations between baseline sweet bias at three time points (breakfast, pre-lunch and post-lunch).

Correlation coefficients and p -values can be seen in Table 4.3.

EL sweet bias was significantly positively correlated at breakfast with pre-lunch, breakfast with post-lunch, and pre-lunch with post-lunch. EW sweet bias was significantly positively correlated at breakfast with pre-lunch only. IW sweet bias was significantly positively correlated at breakfast with pre-lunch and breakfast with post-lunch, but not pre-lunch with post-lunch. Choice sweet bias was significantly positively correlated at breakfast with pre-lunch, but breakfast with pre-lunch and pre-lunch with post-lunch displayed non-significant associations.

Table 4.3. Correlation coefficients for Bivariate Pearson's correlations of Leeds Food Preference Scores scores at three time points across baseline in the full sample ($n=94$).

		Bkfst	PreLu	PoLu
EL Sweet Bias	Breakfast	-	.248 ($p=.016$)	.305 ($p=.003$)
	Pre-Lunch	.248 ($p=.016$)	-	.233 ($p=.024$)
	Post-Lunch	.305 ($p=.003$)	.233 ($p=.024$)	-
EW Sweet Bias	Breakfast	-	.306 ($p=.003$)	.167 ($p=.107$)
	Pre-Lunch	.306 ($p=.003$)	-	.008 ($p=.941$)
	Post-Lunch	.167 ($p=.107$)	.008 ($p=.941$)	-
IW Sweet Bias	Breakfast	-	.422 ($p<.001$)	.208 ($p=.044$)
	Pre-Lunch	.422 ($p<.001$)	-	.187 ($p=.073$)
	Post-Lunch	.208 ($p=.044$)	.187 ($p=.073$)	-
Choice Sweet Bias	Breakfast	-	.369 ($p<.001$)	.155 ($p=.135$)
	Pre-Lunch	.369 ($p<.001$)	-	.178 ($p=.088$)
	Post-Lunch	.155 ($p=.135$)	.178 ($p=.088$)	-

Abbreviations: *IW* = implicit wanting. *EW* = explicit wanting. *EL* = explicit liking.

4.3.3 The stability of sweet food preferences within one day

The stability of sweet food preferences across a single day (i.e. the acute stability) was assessed via the ICC, applying a two-way mixed model for EL, EW, IW and choice, i) sweet bias ii) HFSW and iii) LFSW outcomes at breakfast, pre-lunch and post-lunch, results of which can be seen in Table 4.4. EL sweet bias, HFSW and LFSW displayed weak significant correlations ($r = .374-.496, p<.001$). All other variables displayed moderate significant correlations ($r = .515-.610, p<.001$). Choice HFSW displayed a moderate significant correlation ($r = .653, p<.001$), whilst choice sweet bias and choice LFSW, displayed weak significant correlations ($r = .496-.574, p<.001$) at time points across the day.

Table 4.4. Intraclass correlation coefficients for Leeds Food Preference Questionnaire choice bias at breakfast, pre-lunch and post-lunch across baseline.

Variable	ICC	Significance (<i>p</i>)	Strength
EL Sweet Bias	.515	<.001	Moderate
EL HFSW	.672	<.001	Moderate
EL LFSW	.593	<.001	Moderate
EW Sweet Bias	.374	.004	Weak
EW HFSW	.630	<.001	Moderate
EW LFSW	.464	<.001	Weak
IW Sweet Bias	.535	<.001	Moderate
IW HFSW	.668	<.001	Moderate
IW LFSW	.610	<.001	Moderate
Choice Sweet Bias	.496	<.001	Weak
Choice HFSW	.653	<.001	Moderate
Choice LFSW	.574	<.001	Moderate

Abbreviations: ICC – intraclass correlation coefficient. HFSW – high fat sweet. LFSW – low fat sweet. HFSA – high fat savoury. LFSA – low fat savoury

4.3.4 Differences in sweet food preference across a dietary weight loss intervention

4.3.4.1 Explicit Liking

Repeated measures ANOVAs were conducted to compare differences in EL sweet bias, at three time points (baseline, week2, post-intervention). For EL sweet bias, sphericity was violated ($p=.002$) and results showed no effect of time, $F(1.533,55.18) = .160$, $p=.795$ (see Figure 4.9).

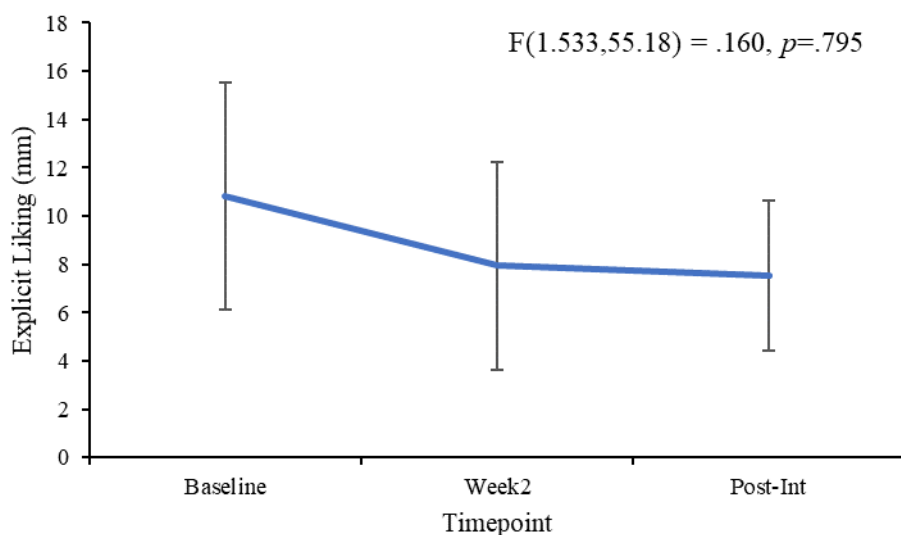


Figure 4.9. Changes in Explicit Liking Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention. Error bars represent standard deviations.

4.3.4.2 Explicit Wanting

Repeated measures ANOVAs were conducted to compare differences in means for EW sweet bias, at three time points (baseline, week2, post-intervention). For EW sweet bias, sphericity was assumed ($p=.203$), although results showed no effect of time, $F(2,72).130, p=.878$ (Figure 4.10).

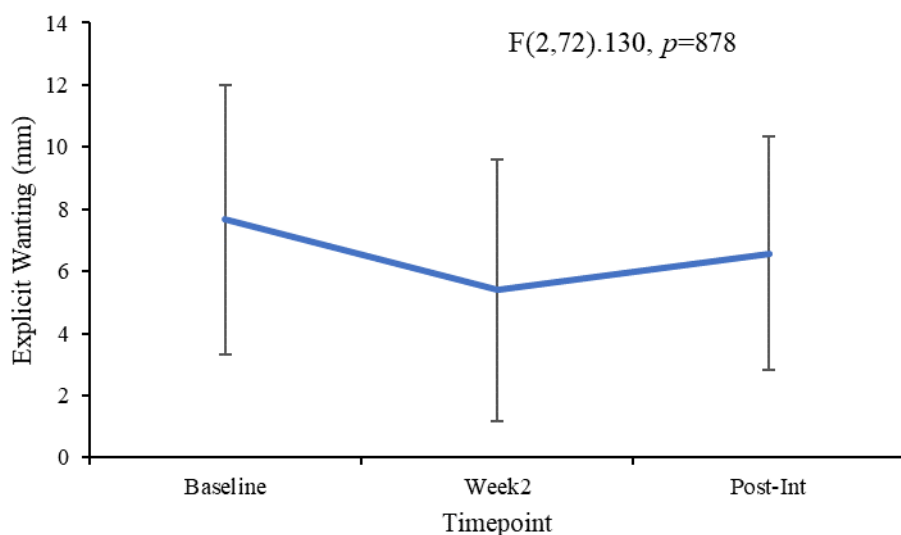


Figure 4.10. Changes in EW Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention. Error bars represent standard deviation.

4.3.4.3 Implicit Wanting

Repeated measures ANOVAs were conducted to compare differences in means for IW sweet bias, at three time points (baseline, week2, post-intervention). For IW sweet bias, sphericity was violated ($p=.006$), although results showed no effect of time, $F(1.597,57.491) = 2.059$, $p=.146$ (see Figure 4.11).

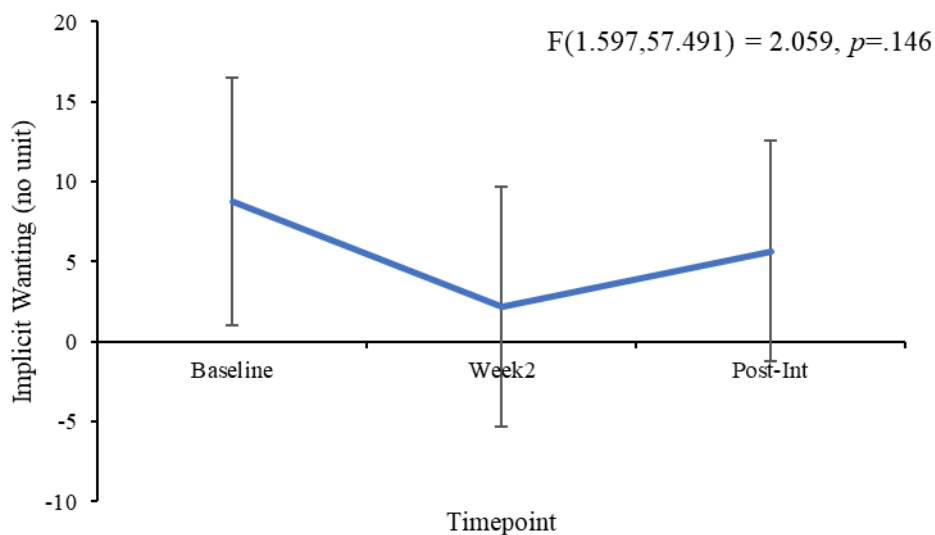


Figure 4.11. Changes in IW Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention. Error bars represent standard deviation.

4.3.4.4 Choice

Repeated measures ANOVAs were conducted to compare differences in means for Choice sweet bias, at three time points (baseline, week2, post-intervention). For Choice sweet bias, sphericity was assumed ($p = .071$), although results showed no effect of time, $F(2,72) = .674$, $p = .513$ (Figure 4.12).

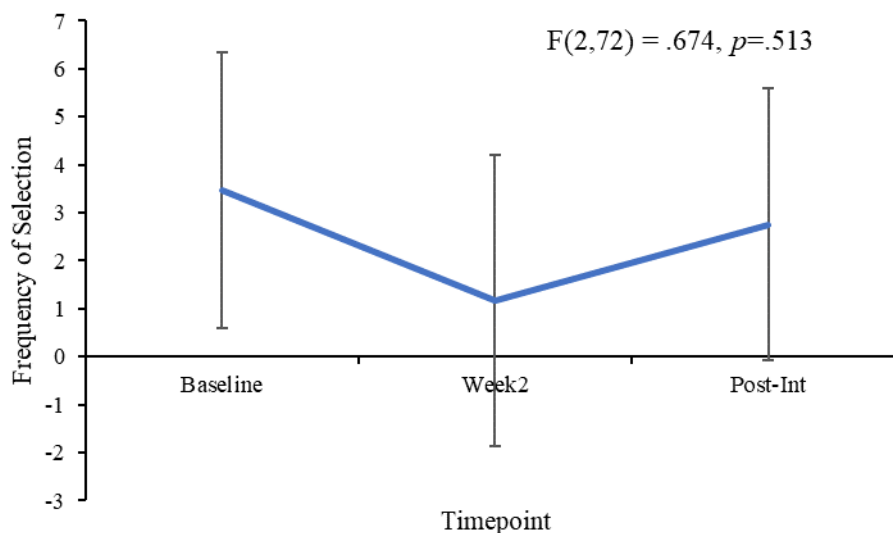


Figure 4.12. Changes in Choice Sweet Bias (fasted/breakfast) from baseline, week2 and post-intervention. Error bars represent standard deviation.

4.3.5 Associations in sweet food preference across a dietary weight loss intervention

Associations between LFPQ outcomes at a single time point during the weight loss intervention (baseline, week2 and week12) are displayed in Table 4.5. All correlations across baseline, week2 and post-intervention were significant. The strength of correlation coefficients between baseline and post-intervention ranged from $r=.399$ (EW sweet bias at post-lunch) to $r=.719$ (EW sweet bias at breakfast).

Table 4.5. Correlation coefficients for Leeds Food Preference Questionnaire outcomes across a weight loss intervention.

Variable		Baseline (n=46)	Week2 (n=42)	Post-Int (n=37)
EL Sweet Bias Bkfst	Baseline	-	.753 ($p<.001$)	.612 ($p<.001$)
	Week2	.753 ($p<.001$)	-	.816 ($p<.001$)
	Post-Int	.612 ($p<.001$)	.816 ($p<.001$)	-
EL Sweet Bias PrLu	Baseline	-	.462 ($p=.002$)	.583 ($p<.001$)
	Week2	.462 ($p=.002$)	-	.869 ($p<.001$)
	Post-Int	.583 ($p<.001$)	.869 ($p<.001$)	-
EL Sweet Bias PoLu	Baseline	-	.684 ($p<.001$)	.515 ($p=.001$)
	Week2	.684 ($p<.001$)	-	.694 ($p<.001$)
	Post-Int	.515 ($p=.001$)	.694 ($p<.001$)	-
EW Sweet Bias Bkfst	Baseline	-	.711 ($p<.001$)	.719 ($p<.001$)
	Week2	.711 ($p<.001$)	-	.807 ($p<.001$)
	Post-Int	.719 ($p<.001$)	.807 ($p<.001$)	-
EW Sweet Bias PrLu	Baseline	-	.599 ($p<.001$)	.571 ($p<.001$)
	Week2	.599 ($p<.001$)	-	.872 ($p<.001$)
	Post-Int	.571 ($p<.001$)	.872 ($p<.001$)	-
EW Sweet Bias PoLu	Baseline	-	.577 ($p<.001$)	.399 ($p=.014$)
	Week2	.577 ($p<.001$)	-	.722 ($p<.001$)
	Post-Int	.399 ($p=.014$)	.722 ($p<.001$)	-
IW Sweet Bias Bkfst	Baseline	-	.715 ($p<.001$)	.498 ($p=.002$)
	Week2	.715 ($p<.001$)	-	.732 ($p<.001$)
	Post-Int	.498 ($p=.002$)	.732 ($p<.001$)	-
IW Sweet Bias PrLu	Baseline	-	.747 ($p<.001$)	.583 ($p<.001$)
	Week2	.747 ($p<.001$)	-	.681 ($p<.001$)
	Post-Int	.583 ($p<.001$)	.681 ($p<.001$)	-
IW Sweet Bias PoLu	Baseline	-	.750 ($p<.001$)	.583 ($p<.001$)
	Week2	.750 ($p<.001$)	-	.714 ($p<.001$)
	Post-Int	.583 ($p<.001$)	.715 ($p<.001$)	-
Choice Sweet Bias Bkfst	Baseline	-	.717 ($p<.001$)	.540 ($p=.001$)
	Week2	.717 ($p<.001$)	-	.770 ($p<.001$)
	Post-Int	.540 ($p=.001$)	.770 ($p<.001$)	-
Choice Sweet Bias PrLu	Baseline	-	.715 ($p<.001$)	.498 ($p=.002$)
	Week2	.715 ($p<.001$)	-	.732 ($p<.001$)
	Post-Int	.498 ($p=.002$)	.732 ($p<.001$)	-
Choice Sweet Bias PoLu	Baseline	-	.771 ($p<.001$)	.599 ($p<.001$)
	Week2	.771 ($p<.001$)	-	.776 ($p<.001$)
	Post-Int	.599 ($p<.001$)	.776 ($p<.001$)	-

Abbreviations: IW – Implicit wanting. EW – explicit wanting. EL – explicit liking. Bkfst – breakfast. PrLu – Pre-lunch. PoLu – Post-Lunch.

4.3.6 The stability of sweet food preference across a dietary weight loss intervention

The stability of sweet food preference in participants with overweight and obesity at baseline, 2 weeks and post-intervention for LFPQ sweet bias variables at each time point across measures days was examined, to assess whether sweet food preferences measured at the same time of day

are consistent during and after weight loss. As shown in Table 4.6, the strength of coefficients across a dietary weight loss intervention was classified as good ($r = .792 - .898$).

Table 4.6. Intraclass correlation coefficients for Leeds Food Preference Questionnaire Sweet Bias at breakfast, pre-lunch and post-lunch across baseline, week2 and post-intervention in participants with overweight or obesity ($n=37$).

Variable	ICC	Significance (p)
EL Sweet Bias Bkfst	.873	<.001
EL Sweet Bias PrLu	.870	<.001
EL Sweet Bias PoLu	.840	<.001
EW Sweet Bias Bkfst	.898	<.001
EW Sweet Bias PrLu	.844	<.001
EW Sweet Bias PoLu	.792	<.001
IW Sweet Bias Bkfst	.834	<.001
IW Sweet Bias PrLu	.864	<.001
IW Sweet Bias PoLu	.870	<.001
Choice Sweet Bias Bkfst	.853	<.001
Choice Sweet Bias PrLu	.890	<.001
Choice Sweet Bias PoLu	.885	<.001

Abbreviations: Bkfst – breakfast. PrLu – Pre-Lunch. PoLu – Post-Lunch.

4.4 Discussion

The aim of the present chapter was to assess the stability of sweet food preferences within one day in different nutritional states (fasted, non-fasted and fed) in both lean women and those with overweight and obesity, as well as during diet-induced weight loss in only a sample of women with overweight or obesity. Findings showed that sweet food preference was higher when in a fasted state in the morning and was lower immediately before lunch (3 hours post-breakfast consumption). Immediately following a standardised lunch meal, sweet preference increased significantly, surpassing values obtained before breakfast consumption. There was poor to moderate stability of choice LFPQ variables across a single day. Inspection of sweet food preferences during dietary weight loss demonstrated no significant differences across time points, indicating stability in sweet food preferences following weight loss. The strength of stability for LFPQ variables taken at the same time of day on different days (baseline, week 2 and week 12) was good, according to criteria outlined by (Koo & Li, 2016). These findings demonstrate that sweet food preferences may be less consistent across the course of a single day, but appear to be stable traits over time despite weight loss.

4.4.1 Differences in Food Preferences

The first hypothesis that sweet food preferences would be elevated in a fasted and fed state than a non-fasted state was supported. Choice sweet bias demonstrated a significant reduction from breakfast to pre-lunch, and increased post-lunch to exceed breakfast levels. Previous evidence has demonstrated sweet food intake to be lower than that of savoury during main meals (Louie, 2017; van Langeveld et al., 2018), which the present findings appear to support, with a reduced sweet food selection frequency in the LFPQ prior to a lunch meal.

The second hypothesis, that sweet food preferences would display a reduction in response to weight loss is not supported – with no significant effect of time on EL, EW, IW or Choice sweet bias. However, previously demonstrated by Oustric (2021) was a significant decline in EL for all food groups in the LFPQ, which only included those participants who had achieved a $\geq 5\%$ weight loss, whereas the current chapter included all participants who completed the trial, regardless of weight loss achieved and so represented a larger sample. The lack of a reduction

in the current analysis is suggestive that an alteration to food liking may occur as a consequence of successful weight loss – with 5% possibly a minimum point at which this is observed. The inclusion of participants whom had not achieved this 5% minimum, may obscure this effect. Alternatively, Oustric examined food groups (i.e., HFSW, LFSW, HFSA, LFSA) within the LFPQ, whereas the current analysis examined solely sweet preferences. It is possible that an overall sweet preference measure is not sensitive enough to capture the reduction in liking following weight loss, which is better captured by examination of the differing food groups.

The similar patterns of variation shown in EL and Choice sweet bias suggests that the two are closely related, with frequency of selection likely influenced by the magnitude of liking. It is highly likely that the decrease in sweet preferences from breakfast to pre-lunch may have been influenced by the consumption of a sweet breakfast meal (for details see section 3.2.5.2) in the lab as well as the anticipation of a lunch meal. Moreover, participants completed both breakfast and lunch versions of the LFPQ to account for differences in foods commonly consumed during these meals, although this may in part contribute to differences between breakfast and pre-lunch scores. It is therefore possible that sweet food preferences were elevated in the morning, in part due to participants presenting in a fasted state, before decreasing at pre-lunch due to the consumption of the low fat, high carbohydrate sweet breakfast meal.

The changes from pre-lunch to post-lunch in sweet preference may highlight the effect of sensory specific satiety. On average, the *ad libitum* lunch meal intake constituted of 71% risotto and so participants consumed more of the savoury food than the sweet food. This would also explain the elevated Choice sweet bias at post-lunch, as well as the increase in IW LFSW at post-lunch – with the greater intake of savoury than sweet food at the lunch meal displaying the transfer effect of sensory specific satiety. Interestingly, despite a decline in EL and EW LFSW at post-lunch, these variables remained elevated relative to other LFPQ outcomes at these time points. EL and EW LFSW were highest at breakfast, with a significant decline at pre-lunch. It is not possible to determine whether this was caused by the consumption of the sweet breakfast, or due to the time of day – reflecting anticipation of a savoury lunch meal (Louie, 2017; van

Langeveld et al., 2018). However, this finding demonstrates the potential for acute variation in sweet food preferences dependent on nutritional state. This requires further investigation in future to determine the influence of time of day as well as nutritional state, and any possible interaction, exerted over food preferences.

4.4.2 The Stability of Sweet Food Preferences

The third hypothesis that sweet food preference would demonstrate stability acutely across a single day in different nutritional states was not supported as although sweet preferences demonstrated significant correlations, the size of these associations was weak and the intraclass correlation coefficients were poor to moderate in strength. Previous evidence has demonstrated that nutritional state may be capable of altering sweet preferences (Epstein et al., 2003; Raynor & Epstein, 2003), for example, food reward brain signalling in response to visual food stimuli was elevated when in a fasted state compared to when satiated in both normal weight and participants with overweight (Martens et al., 2013). Similarly, females in a fasted state have been shown to increase pleasantness ratings of food images (Stoeckel et al., 2007). Moreover, Stoeckel and colleagues (2007) identified that alliesthesia (i.e., the manner in which the hedonic value of an item is modulated by the motivational state (Cabanac, 1971)) was stronger in female participants, and more recent evidence has demonstrated that females are more sensitive to food hedonics when in a fasted state compared to males (Legget et al., 2018). This finding is particularly important for the present study, which was conducted in females only which may have contributed towards the increased variability in preferences. Additionally the extent to which the present variations in sweet food preferences may be replicated in male participants remains unknown.

However, the above findings demonstrate that sweet food preference remains highly stable across a period of diet-induced weight loss, demonstrated by the good intraclass correlation coefficient strengths and supporting the fourth hypothesis. This finding contradicts early findings which demonstrated reductions in hedonic ratings to highly concentrated sucrose solutions following weight loss (Esses & Herman, 1984) as well as similar findings drawn more recently (Nishihara et al., 2019; Umabiki et al., 2010). Furthermore, this builds upon

previous work examining the reproducibility of sweet preference assessment which has been limited to periods of 3-7 days (Asao et al., 2012), 7 ± 2 days (Coulon et al., 2012) as well as studies in patients with alcoholism (Kampov-Polevoy, 2001) and substance dependence (Kampov-Polevoy et al., 2003).

This is the first study to date which has examined the stability of sweet food preferences in individuals with a range of BMI scores, on an acute basis and over a prolonged period of dietary induced weight loss. Although it is not possible to disentangle the effects of diurnal variation from nutritional state, these findings remain important for the assessment of sweet preferences and the categorisation of sweet liker status as a phenotypic trait.

4.4.3 Conclusion

The findings of the present chapter demonstrate that sweet food preference may be influenced by the nutritional state of an individual varying across the day. The acute variations in preferences as a result of nutritional state differing throughout the day require consideration in future study designs in order to accurately capture reliable measures of sweet food preferences during assessment. However, the finding that sweet food preference may be viewed as a stable trait across time during weight loss is also demonstrated. This is supported by the finding that sweet food preferences did not differ across a weight loss intervention, suggesting that sweet food preference may be a stable trait, independent of weight loss. The findings, when taken together, suggest that sweet food preferences are not strongly consistent in the context of acute changes in nutritional state, but remain a stable trait across more prolonged periods of time.

5 The influence of sweet food preferences on eating behaviours in women across a range of BMIs

Aims: It was demonstrated within the previous chapter that sweet food preferences remain stable after $\geq 5\%$ weight loss during a dietary intervention. Evidence has shown that sweet food preferences are associated with eating behaviour traits that may contribute to diet failure. Therefore, a sweet food preference may make weight management more difficult via these associations. The aim of the present chapter is to investigate the influence of sweet food preferences on eating behaviours in a sample of women across a range of BMIs in a cross-sectional analysis, and in those with overweight and obesity before and after weight loss. The implications of which may assist in identifying potential barriers to successful weight loss or a susceptibility to overconsume in those with a sweet food preference and will address the third objective listed in section 1.8.1.

5.1 Introduction

There are a number of possible routes towards obesity and weight gaining individuals may possess a number of susceptibility factors (Blundell et al., 2005). Behavioural traits are capable of only indirectly influencing physical conditions such as body weight via a relationship with covariates such as eating behaviour (Davis et al., 2007). For example, individuals with a strong sweet preference report a greater number of binge episodes per month than lower sweet preferers (Goodman et al., 2018). Similarly, in female participants, cravings tend to be positively associated with BMI, and are specifically directed towards sweet foods (Christensen, 2007). With intake of sweet, energy-dense foods postulated as a cause of obesity rates increasing globally (Mullee et al., 2019; Swinburn et al., 2011), it is important to understand the role of sweet food preferences on eating behaviours as this may highlight potential factors contributing to overconsumption or barriers to weight loss.

A sweet food preference is associated with a number of eating behaviour traits which may predispose an individual to overconsumption and subsequent weight gain. For example, women scoring highly on trait binge eating also display an elevated wanting for sweet (Dalton &

Finlayson, 2014) and subsequently a high intake of HFSW food (de Zwaan, 2001; de Zwaan et al., 1992; Finlayson et al., 2011). Additionally, both lean-binge and obese-binge type participants exhibit a greater preference for sweet foods and greater energy intake than non-binge individuals (Dalton et al., 2013a). Notably Dalton (2013a) also reported that in obese-binge type individuals wanting for sweet foods was greater when fed than in a fasted state, suggesting a susceptibility to overeating as a possible mechanism by which weight gain occurs.

Additionally, consumption of sweet foods is related to higher disinhibition (Chambers & Yeomans, 2011; Lähteenmäki & Tuorila, 1995), evidenced by a positive association between disinhibition (assessed via the TFEQ) and the consumption of sweet foods in women (Bryant, 2001; Haynes et al., 2003). As such, a higher degree of disinhibition is detrimental to diet quality (Aguirre et al., 2017; Bernstein et al., 2015) and is associated with obesity (Lindroos et al., 1997). Similarly, women with low restraint scores display a higher energy intake and sweet intake than women scoring highly on restraint (French et al., 1994), and subsequently, restraint is positively associated with BMI (Blumfield et al., 2018; Cornelis et al., 2014).

Moreover, a predilection for sweet foods has also been suggested to be a risk factor for overeating as a result of greater cravings (Dalton et al., 2013a). Frequency of food cravings are positively associated with BMI (Burton et al., 2007; Chao et al., 2014), with cravings for sweet associated with sweet liking ratings and the frequency of consumption of sweet foods (Keskitalo, 2007). Furthermore, during a weight loss intervention there also exists an association between trait craving and weight change, with greater reductions in trait craving associated with greater weight loss (Batra et al., 2013). Therefore, it may be anticipated that individuals with an elevated sweet food preference may also present elevated trait cravings, which change during weight loss, with changes occurring due to alterations in dietary intake, rather than reductions in body weight (Martin et al., 2006).

Similarly, diet induced weight loss may alter other eating behaviour traits, with decreases in disinhibition and increases in restraint associated with greater reductions in waist circumference (Bryant et al., 2012). However, the potential differences between individuals

categorised as high or low sweet likers remain unclear. It has been suggested recently that high sweet likers possess higher interoceptive abilities, particularly in relation to traits such as trait hunger, than low sweet likers (Iatridi et al., 2020). As an examination of changes in eating behaviour traits between phenotype groups is yet to be conducted, a preliminary exploratory investigation is therefore warranted.

The aim of the current chapter is to investigate the influence of sweet food preferences on eating behaviours in women with a range of BMIs using cross-sectional analysis, as well as in those with overweight and obesity before (baseline), during (week 2) and after weight loss (post-intervention). Differences between baseline and week 2 of the weight loss intervention are examined, due to interest in any effects produced by the reduction in dietary intake by participants before significant changes to body weight have occurred that may also impact eating behaviour traits. Similarly, differences between baseline and post-intervention are considered (after \sim >5% weight loss had been achieved), with changes to eating behaviour traits between these time points hypothesised to be a result of a combination of changes in body weight and composition, and changes to dietary intake whilst following the weight loss intervention. Differences between week 2 and post-intervention were not considered in this chapter.

Hypotheses:

- Participants with overweight and obesity will present at baseline with a significantly higher sweet food preference than lean weight controls.
- Sweet food preferences will be significantly positively associated with BES score, TFEQ disinhibition and craving for sweet across a range of BMIs and significantly inversely associated with craving control and TFEQ restraint scores across a range of BMIs.
- High sweet likers will display a lower rate of weight change per week than low sweet likers.

- There will be a significant difference between sweet liker phenotype groups in eating behaviour traits at baseline, as well as the changes at week 2 and post-intervention.
- There will be a significant increase in craving control, trait hunger and restraint and reduction in sweet/savoury cravings at week 2, but not at post-intervention, with no difference between phenotype groups.

5.2 Methods

Ninety-four women, forty-six with overweight or obesity and forty-eight with normal weight took part in the current study. Participants with overweight/obesity and those with normal weight were recruited separately. Individuals with overweight and obesity were recruited to take part in a dietary intervention designed to reduce body weight by $\geq 5\%$, whilst participants with normal weight were recruited later to provide a control comparison. A total of fifty-four participants with overweight/obesity were enrolled into the trial, with forty-six participants providing baseline measures. Thirty-seven participants completed the diet intervention whilst twenty-nine achieved $\geq 5\%$ weight loss and eight participants failed to achieve 5% weight loss within the 12-week timeframe. All available data were utilised during analysis with instances of missing data indicated where relevant.

Participants with overweight and obesity undertook one of two dietary induced weight loss interventions, although are treated within the current chapter as a single group. The current examination is of the effects of weight loss only, an investigation into the effects of intervention type is provided in the following chapter (Chapter 6). Differences at baseline between participants with overweight and obesity relative to a lean control group will be examined, as well as the associations between sweet food preferences and eating behaviour traits, prior to an exploratory examination of the differences in the rate of weight change and eating behaviour traits between high and low sweet liker phenotype groups before, during and after weight loss.

Each measures day followed the same format for all participants. All measures were conducted within the HARU at the University of Leeds. Participants arrived at the HARU after completing a 12-hr overnight fast and avoiding alcohol intake and physical activity for 24 hrs. During a measures day participants completed the LFPQ breakfast version before body composition was measured using air displacement plethysmography (Bodpod, Concord, USA). Following consumption of a standardised sweet breakfast (calculated at 25% of resting metabolic rate using GEM indirect calorimetry) participants were free to leave the lab and

return 2-hrs 45-mins later – ensuring breakfast and lunch meals were as close to 3-hrs apart as possible. Upon returning to the lab, participants completed the LFPQ lunch version before being provided an *ad libitum* lunch meal, representing savoury (risotto) and sweet (yoghurt) options, matched for energy density (consumption amounts not reported in the current analysis). Following consumption of the *ad libitum* lunch meal, participants completed the LFPQ lunch version a second time. Participants were then free to leave the lab and were provided a booklet containing eating behaviour questionnaires (Three-Factor Eating Questionnaire, Binge Eating Scale and Control of Eating Questionnaire reported in the current chapter), to complete at home that evening and to return to the lab.

Participants with overweight or obesity completed a baseline measures day, before beginning a weight loss intervention. Participants then completed a second measures day at 2 weeks, and a third measures day once weight loss $\geq 5\%$ was achieved or at 12 weeks – dependent on which occurred first. Participants were randomly allocated to one of two dietary weight loss interventions, for analyses of the effects of the separate intervention protocols on sweet food preferences as well as details as to how the diets differed, see Chapter 6. For further details on the methodology please refer to Chapter 3.

5.2.1 Sweet Liker Phenotype Categorisation

Participants with overweight and obesity were grouped on the basis of sweet liker phenotype. Fasted explicit liking sweet bias score at baseline was selected as a baseline measure of sweet liking, although as shown previously in the current thesis (see Chapter 4), sweet preferences were highly stable across the dietary intervention and so baseline, week2 and post-intervention are unlikely to impact sweet liker phenotype classification. Due to the manner in which sweet bias is calculated within the LFPQ, it represents a sweet relative to savoury preference, therefore any negative sweet bias values represent a savoury preference. As such, sweet liker phenotype is presented in the current analysis as a dichotomised variable, with participants coded as a high sweet liker if their explicit liking sweet bias was >0 , and a low sweet liker if it was <0 . Defining further phenotype groups was not possible within the present thesis due to the operationalisation of sweet liking. Moreover, although the desired sweet intensity and relative

sweet preference may differ between groups, participants are described as high sweet likers and low sweet likers – not sweet likers/dislikers – due to sweetness possessing a universal hedonic reward (Blundell, 2018; Yang et al., 2019).

5.2.2 Data Analysis

The statistical analysis consisted of two stages, an examination of baseline LFPQ sweet bias scores and eating behaviour traits values in both lean participants and those with overweight and obesity, before an examination of the same values during the weight loss intervention in participants with overweight and obesity compared by sweet liker phenotype group. Data were visually explored for extreme outliers using histograms and boxplots, with no extreme outliers being identified according to the Tukey method (Tukey, 1977).

Firstly, independent groups t-tests were conducted between lean participants and those with overweight and obesity, on all outcomes. Next, bivariate Pearson's correlations were conducted to identify any associations between the sweet bias scores and eating behaviour trait variables (binge eating score, craving control and three-factor eating questionnaire scores) at baseline in both participants with overweight and obesity and normal weight participants as a single group. For any outcomes which differed significantly by BMI group within the tests of difference, a partial correlation was conducted controlling for BMI to factor in these groups differences and reduce the potential for BMI to serve as a confound.

Bivariate Pearson's correlations were also conducted between all LFPQ sweet bias outcomes and rate of weight change in participants completing the dietary intervention. Rate of weight change was included in the analyses as the amount of absolute weight-loss may be impacted by participants exhibiting different starting body masses (Hatoum, 2013) as well as to identify the influence of sweet food preferences as a potential barrier to successful weight-loss. The mean rate of weight change was calculated at post-intervention and was calculated using the following formula:

$$\text{Rate of Weight Change (\% per week)} = \frac{\text{Total Weight Loss (\%)}}{\text{Time (Weeks)}}$$

T-tests were conducted to compare differences in body composition values at baseline and rate of weight change between sweet liker phenotype groups. A series of 2x3 repeated measures ANOVAs were then conducted to compare differences in eating behaviour traits across the

dietary induced weight loss intervention. Post-hoc tests were conducted to examine any significant effects with no multiple comparison adjustments made, the significance threshold for all the following tests was set at $p \leq 0.05$.

5.3 Results

5.3.1 Descriptive Statistics and Baseline Differences Between BMI Groups

Baseline descriptive statistics are displayed in Table 5.1. As would be expected, body fat percentage, fat mass, fat-free mass and body weight were significantly higher in the group with overweight/obesity (Table 5.1). Furthermore, at baseline participants with overweight and obesity displayed significantly greater TFEQ disinhibition, TFEQ hunger and BES scores, with lean participants presenting a greater craving control. There were no differences between groups regarding LFPQ sweet bias outcomes, sweet or savoury craving, positive mood or TFEQ restraint.

Table 5.1. Participant baseline descriptive statistics in both participants with overweight and obesity and normal weight by group.

Variable	Mean (SD)	Mean (SD)	Significance (<i>p</i>)
	Overweight/Obesity (n = 45)	Lean (n = 42)	
Age (years)	34.94 (10.28)	34.94 (10.28)	.913
Height (cm)	165.22 (8.11)	164.40 (6.38)	.585
BMI (kg/m ²)	29.17 (2.40)	21.84 (1.75)	<.001
Body Fat Percentage (%)	41.42 (5.16)	27.18 (5.39)	<.001
Fat Mass (kg)	33.44 (8.17)	16.22 (4.05)	<.001
Fat-Free Mass (kg)	46.53 (5.65)	42.87 (4.43)	.001
Body Weight (kg)	79.92 (11.62)	59.09 (6.47)	<.001
EL Sweet Bias	10.83 (22.90)	12.25 (17.57)	.738
EW Sweet Bias	10.83 (21.16)	10.63 (17.10)	.457
IW Sweet Bias	8.76 (37.63)	17.39 (37.51)	.269
Choice Sweet Bias	3.48 (14.03)	6.00 (13.02)	.368
TFEQ Restraint	7.96 (3.89)	7.57 (5.18)	.695
TFEQ Disinhibition	9.76 (3.00)	6.45 (3.66)	<.001
TFEQ Hunger	6.96 (3.18)	4.72 (2.90)	.001
Craving Control	40.94 (21.60)	64.91 (20.04)	<.001
Sweet Craving	50.13 (28.14)	44.86 (24.60)	.357
Savoury Craving	42.37 (24.59)	46.32 (19.57)	.412
Positive Mood	65.12 (13.96)	67.70 (14.32)	.454
Binge Eating Score	16.47 (8.70)	7.10 (5.73)	<.001

Abbreviations: BMI – body mass index. EL – explicit liking. EW – explicit wanting. IW – implicit wanting. TFEQ – three factor eating questionnaire. BES – binge eating score. Kg – kilogram. SD – standard deviation. Association Between Baseline Sweet Food Preferences and Eating Behaviour Traits

As displayed in Table 5.2, when controlling for BMI via a partial correlation, there were also significant negative associations between IW sweet bias and TFEQ disinhibition, IW sweet bias and BES score, and Choice sweet bias and BES score, which were weak in strength.

Individuals displaying an elevated IW sweet bias also demonstrate lower disinhibition and BES scores. Similarly, those that displayed greater frequency of sweet items selected in the LFPQ also displayed a lower BES score.

Table 5.2. Correlation matrix displaying baseline correlations between Leeds Food Preference Questionnaire sweet bias outcomes and eating behaviour trait variables ($n=87$).

Variable	TFEQ Restraint		TFEQ Disinhibition*		TFEQ Hunger*		Craving Control*		Sweet Craving		Savoury Craving		BES*	
	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>	<i>r</i>	<i>p</i>
EL Sweet Bias	.237	.020	-.046	.679	.047	.674	-.030	.783	.186	.085	-.130	.231	-.120	.275
EW Sweet Bias	.232	.031	-.073	.508	.030	.789	.010	.928	.165	.127	-.085	.434	-.179	.102
IW Sweet Bias	.185	.086	-.223	.042	-.187	.088	.142	.198	.105	.332	-.120	.270	-.246	.024
Choice Sweet Bias	.180	.095	-.201	.067	-.168	.127	.141	.201	.122	.261	-.117	.280	-.239	.029

Abbreviations: EL – explicit liking. EW – explicit wanting. IW – implicit wanting. TFEQ – three factor eating questionnaire. BES – binge eating score.
 *Indicates partial correlation controlling for BMI.

5.3.2 Effects of Sweet Food Preferences on Rate of Weight Change

A further Bivariate Pearson's correlation was performed to examine the associations between baseline sweet bias outcomes and rate of weight change (per week) in the participants with overweight/obesity in response to the dietary intervention. As can be seen in Table 5.3 there were no significant associations.

Table 5.3. Table of correlations between Leeds Food Preference Questionnaire sweet bias outcomes and Rate of Weight Change ($n=37$).

Variable	Rate of Weight Change (p/w)	
	<i>r</i>	<i>p</i>
EL Sweet Bias	.136	.421
EW Sweet Bias	.124	.465
IW Sweet Bias	.064	.706
Choice Sweet Bias	.071	.676

Abbreviations: p/w – per week. EL – explicit liking. EW – explicit wanting. IW – implicit wanting.

Participants were then categorised as a high or low sweet liker phenotype, using LFPQ EL sweet bias responses at baseline, with differences in outcome variables compared between the phenotype groups. The sample consisted of 25 high sweet likers and 11 low sweet likers who completed the trial and mean EL sweet bias by phenotype group can be seen in Figure 5.1. Independent groups t-tests were conducted to compare differences in rate of weight change, results demonstrated no significant differences between high (mean (SD) = -0.79 (0.34)) and low (mean (SD) = -0.66 (0.25)) sweet liker phenotype groups in rate of weight change (per week) ($t(35) = -1.375, p = .178$).

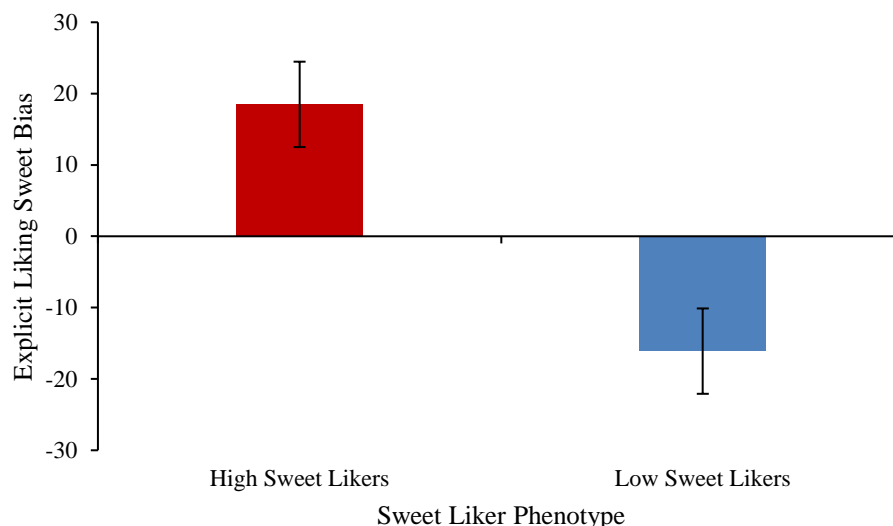


Figure 5.1. Mean explicit liking sweet bias for high ($n=25$) and low ($n=11$) sweet likers at baseline ($t(34)=-6.796$, $p<.001$). Error bars represent standard deviation.

5.3.3 Differences in Eating Behaviour traits between Sweet Liker Phenotypes in response to Weight Loss

Independent groups t-tests were conducted initially to compare differences between high and low sweet liker phenotype groups in participant characteristics at baseline (see Table 5.4). Participants did not differ by age or height at baseline, but high sweet likers presented with a significantly greater body fat percentage than low sweet likers.

Displayed in Table 5.4. Participant baseline descriptive statistics and differences in High ($n=25$) and Low ($n=11$) Sweet Liker Phenotype Groups.

Variable	Mean (SD)	Mean (SD)	t – Value	p – value
	High Sweet Likers	Low Sweet Likers		
Age (years)	35.66 (11.08)	33.29 (8.27)	-.716	.478
Height (cm)	165.13 (8.02)	165.44 (8.61)	.117	.907
BMI (kg/m ²)	29.59 (2.52)	28.20 (1.86)	-1.864	.069
Body Fat Percentage (%)	42.55 (5.16)	38.83 (4.28)	-2.363	.023
Fat Mass (kg)	34.74 (8.16)	30.48 (7.64)	-1.657	.105
Fat-Free Mass (kg)	46.22 (5.51)	47.24 (6.09)	.556	.581
Body Weight (kg)	80.96 (11.27)	77.72 (12.50)	-.868	.390

Abbreviations: BMI – body mass index. TFEQ – three factor eating questionnaire. BES – binge eating score. Kg – kilogram. SD – standard deviation.

Table 5.5 are the means and standard deviations of the eating behaviour trait scores for both high and low sweet liker groups at each time point during the weight loss intervention. A series

of 2x3 repeated measures ANOVAs with sweet phenotype (2 levels) and time (3 levels) were conducted to examine the differences in eating behaviour traits by sweet liker phenotype group during the dietary intervention. Mauchley's test of sphericity was examined and where appropriate Greenhouse-Geisser corrections were applied.

Table 5.4. Participant baseline descriptive statistics and differences in High ($n=25$) and Low ($n=11$) Sweet Liker Phenotype Groups.

Variable	Mean (SD)	Mean (SD)	t – Value	p – value
	High Sweet Likers	Low Sweet Likers		
Age (years)	35.66 (11.08)	33.29 (8.27)	-.716	.478
Height (cm)	165.13 (8.02)	165.44 (8.61)	.117	.907
BMI (kg/m ²)	29.59 (2.52)	28.20 (1.86)	-1.864	.069
Body Fat Percentage (%)	42.55 (5.16)	38.83 (4.28)	-2.363	.023
Fat Mass (kg)	34.74 (8.16)	30.48 (7.64)	-1.657	.105
Fat-Free Mass (kg)	46.22 (5.51)	47.24 (6.09)	.556	.581
Body Weight (kg)	80.96 (11.27)	77.72 (12.50)	-.868	.390

Abbreviations: BMI – body mass index. TFEQ – three factor eating questionnaire. BES – binge eating score. Kg – kilogram. SD – standard deviation.

Table 5.5. Baseline, Week 2 and Post-Intervention Eating Behaviour Trait Scores for High and Low Sweet Liker Phenotype Groups.

Variable	Mean (SD)	Mean (SD)		Mean (SD)	Mean (SD)	
	Baseline	Week 2	Post-Int	Baseline	Week 2	Post-Int
	High Sweet Likers (n = 25)			Low Sweet Likers (n = 11)		
TFEQ Restraint	8.60 (3.83)	9.72 (4.43)	11.92 (4.45)	8.27 (4.45)	10.36 (5.61)	13.27 (4.69)
TFEQ Disinhibition	9.448 (3.23)	9.12 (3.75)	8.28 (3.97)	10.18 (3.06)	9.27 (3.00)	7.36 (2.69)
TFEQ Hunger	6.68 (3.30)	7.00 (3.19)	5.20 (2.99)	6.55 (2.46)	5.82 (3.55)	2.46 (1.86)
CoEQ Craving	44.08 (44.16)	58.99 (22.81)	63.74 (22.71)	44.33 (26.36)	54.86 (25.22)	64.78 (22.45)
CoEQ Sweet	50.68 (24.35)	34.40 (24.70)	36.68 (25.14)	40.33 (30.16)	28.67 (33.09)	24.24 (26.25)
CoEQ Savoury	44.52 (27.26)	37.52 (23.35)	34.86 (22.67)	45.64 (17.84)	38.61 (22.86)	23.41 (16.94)
CoEQ Pos. Mood	66.00 (12.80)	65.94 (14.62)	67.86 (16.73)	63.50 (15.59)	61.59 (16.46)	72.41 (12.66)
BES*	16.04 (9.74)	14.38 (9.80)	12.54 (8.80)	17.18 (7.52)	13.73 (6.86)	10.27 (4.92)

Abbreviations: SD – standard deviation. Post-Int – post-intervention. TFEQ – three factor eating questionnaire. CoEQ – control of eating questionnaire. Pos – positive. BES – binge eating scale.

*Binge eating scale high sweet likers $n = 24$.

5.3.3.1 Three Factor Eating Questionnaire

For TFEQ restraint sphericity was assumed ($p=.307$) and between-subjects effects displayed a non-significant effect of sweet liker phenotype, $F(1,34) = 0.151$, $p=.700$. Within-subjects effects displayed a significant main effect of time $F(2,68) = 21.106$, $p < .001$, and no effect of time*sweet liker phenotype, $F(2,68) = 0.853$, $p=.431$. Pairwise comparisons displayed a significant difference between baseline and week 2 ($p=.020$), and baseline and post-intervention ($p<.001$) as displayed in Figure 5.2A.

Sphericity was assumed ($p=.200$) for TFEQ disinhibition. Between-subjects effects displayed no effect of sweet liker phenotype, $F(1,34) = 0.000$, $p=.986$. Within-subjects effects displayed a significant main effect of time, $F(2,68) = 10.232$, $p<.001$, but no time*sweet liker phenotype interaction, $F(2,68) = 1.642$, $p=.201$. Pairwise comparisons highlighted a significant difference between baseline and post-intervention ($p < .001$) as displayed in Figure 5.2B.

For TFEQ hunger sphericity was assumed ($p= .118$). Between-subjects effects displayed no effect of sweet liker phenotype, $F(1,34) = 2.149$, $p= .152$. Within-subjects effects displayed a significant main effect of time, $F(2,68) = 18.165$, $p < .001$, and a significant time*sweet liker phenotype interaction, $F(2,68) = 3.252$, $p=.045$. Pairwise comparisons demonstrated no difference between baseline and week2 ($p > .999$), but a significant difference between baseline and post-intervention ($p < .001$). As displayed in Figure 5.2C there was a significant difference between high and low sweet liker phenotypes at post-intervention ($p=.008$). Whilst both groups displayed a reduction in trait hunger at post-intervention, low sweet likers displayed a significantly larger reduction than high sweet likers. This difference was not present at baseline or at week 2.

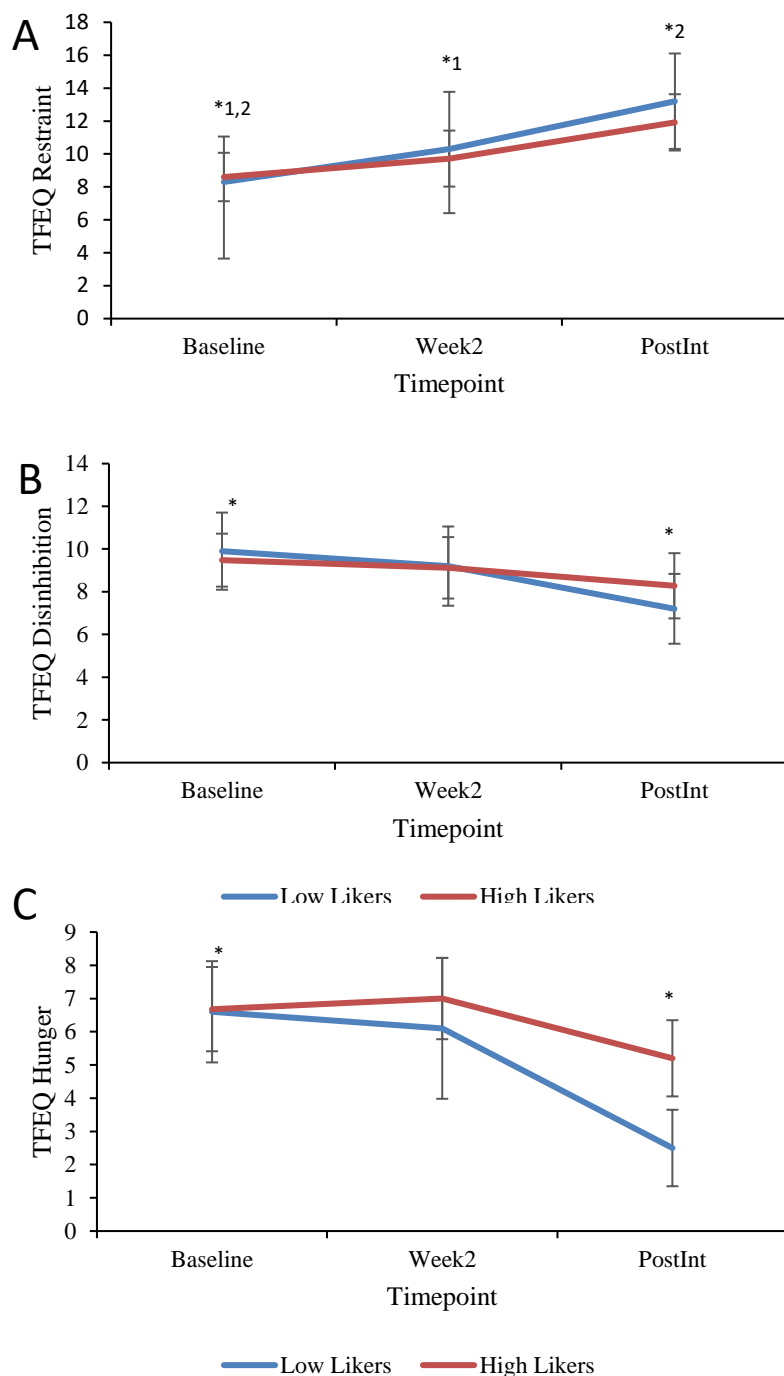


Figure 5.2. Differences in Three Factor Eating Questionnaire outcomes across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure A. Differences in Trait Restraint across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure B. Differences in Trait Disinhibition across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure C. Differences in Trait Hunger across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Error bars represent standard deviation.

Significant differences: A, *¹ difference between baseline and week2, *² difference between baseline and post-intervention. B, * difference between baseline and post-intervention. C, * baseline and post-intervention.

5.3.3.2 Binge Eating Scale

For BES scores sphericity was assumed ($p=.855$). Between-subjects effects displayed no effect of sweet liker phenotype, $F(1,33) = 0.041$, $p=.840$. Within-subjects effects displayed a significant main effect of time, $F(2,66) = 12.219$, $p<.001$, but no time*sweet liker phenotype interaction, $F(2,66) = 1.312$, $p=.276$. Pairwise comparisons displayed a trend towards a significant difference between baseline and week 2 ($p=.051$), and a significant difference between baseline and post-intervention ($p<.001$) as displayed in Figure 5.3.

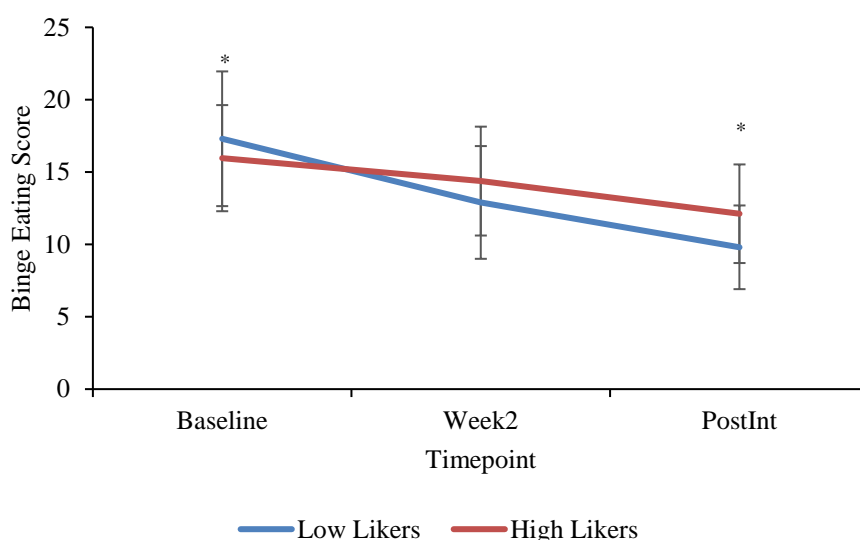


Figure 5.3. Differences in Binge Eating Scale scores across a dietary induced weight loss intervention in high ($n=24$) and low ($n=11$) sweet liker phenotype groups. Error bars represent standard deviation. Significant differences between baseline and post-intervention denoted by *.

5.3.3.3 Control of Eating Questionnaire

Sphericity was assumed ($p=.963$) for craving control. Between-subjects effects demonstrated no effect of sweet liker phenotype, $F(1,34) = 0.017$, $p=.899$. Within-subjects effects displayed a significant main effect of time, $F(2,68) = 18.988$, $p<.001$, but no time*sweet liker phenotype interaction, $F(2,68) = 0.359$, $p=.700$. Pairwise comparisons displayed a significant difference between baseline and week 2 ($p=.001$) and baseline and post-intervention ($p<.001$) which can be seen in Figure 5.4A. For sweet craving, sphericity was assumed ($p=.404$). Between-subjects effects displayed no effect of sweet liker phenotype, $F(1,34) = 1.247$, $p=.272$. Within-subjects effects displayed a significant main effect of time, $F(2,68) = 10.096$, $p<.001$, but no

time*sweet liker phenotype interaction, $F(2,68) = 0.422, p = .657$. Pairwise comparisons displayed a significant difference between baseline and week 2 ($p = .001$), and baseline and post-intervention ($p = .003$) which can be seen in Figure 5.4B. Finally, for savoury craving, sphericity was assumed ($p = .181$). Between-subjects effects displayed no effect of sweet liker phenotype, $F(1,34) = 0.185, p = .670$. Within-subjects effects displayed a significant main effect of time, $F(2,68) = 9.158, p < .001$, but no time*sweet liker phenotype interaction, $F(2,68) = 1.883, p = .160$. Pairwise comparisons displayed a significant difference between baseline and post-intervention ($p < .001$) which can be seen in Figure 5.4C.

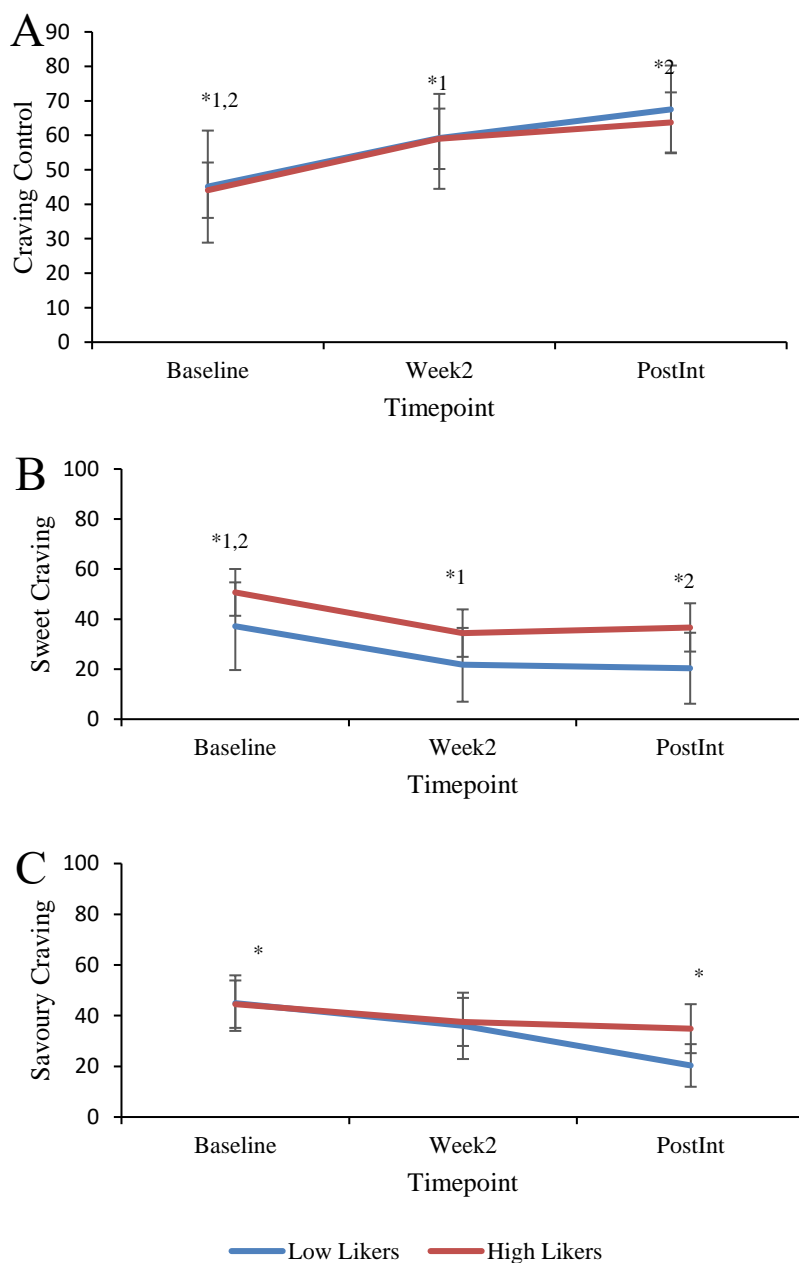


Figure 5.4. Differences in Control of Eating Questionnaire outcomes across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure A. Differences in Craving Control across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure B. Differences in Sweet Craving across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Figure C. Differences in Savoury Craving across a dietary induced weight loss intervention in high ($n=25$) and low ($n=11$) sweet liker phenotype groups.

Error bars represent standard deviation.

Significant differences: A, *¹ difference between baseline and week2, *² difference between baseline and post-intervention. B, *¹ difference between baseline and week2, *² difference between baseline and post-intervention. C, * difference between baseline and post-intervention.

5.4 Discussion

The aim of the current chapter was to examine the influence of sweet food preferences on eating behaviours in female participants across a range of BMIs as well as in those with overweight and obesity before, during and after a dietary weight loss intervention. The first hypothesis was rejected, as at baseline there were no differences between BMI groups in sweet food preference outcomes. The second hypothesis was supported, as when controlling for BMI there existed positive associations between IW sweet bias with both TFEQ disinhibition and BES score, as well as choice sweet bias and BES score, indicating that individuals with an increased IW for sweet presenting with an increased likelihood of losing control around food. However, the strength of the associations remained weak, and both EL and EW sweet bias demonstrated positive associations with TFEQ restraint scores, and so the second hypothesis is only partially supported. The third hypothesis is rejected as there was not a significant difference in the rate of weight change between sweet liker phenotype groups. Significant differences were demonstrated in a number of eating behaviour traits over the course of the dietary induced weight loss intervention, however, these differences did not differ between sweet liker phenotype groups with the exception of trait hunger. There was a significant time*sweet liker phenotype interaction, with both groups displaying a decrease in trait hunger over the dietary weight loss intervention, although a greater decrease at post-intervention was observed in low sweet likers, thereby supporting the fourth hypothesis and highlighting trait hunger as a key difference between phenotype groups. There were also reductions in dietary restraint, sweet cravings and an increase in craving control in both sweet phenotype groups at week 2 as hypothesised, however these changes were also present at post-intervention, as well as a differences at post-intervention in disinhibition and BES score.

5.4.1 Differences and Associations between Baseline Sweet Food Preferences and Eating Behaviour Traits

There were no differences between BMI groups in any sweet food preference outcomes at baseline. This may be due to differences in the expression of sweet preference between BMI groups. Via food preference checklists it has been demonstrated that individuals with obesity

prefer foods high in both carbohydrates and fats (Drewnowski et al., 1992), with more recent evidence demonstrating females with obesity report a preference for HFSW, whereas women who are lean have a preference for LFSW foods (Jayasinghe et al., 2017). Therefore, the fat content of a sweet stimulus may be of greater importance when examining BMI group differences, than has previously been considered. Indeed, liking for a combination of sweet and fat is associated with an increased risk of obesity in females specifically, whereas a preference for natural sweetness is protective against obesity risk (Lampuré et al., 2016). However, it should be noted that the current study was sufficiently powered to detect interactions in self-selected meal size between two groups, and as such the study may lack sufficient power to detect significant differences.

Conversely, at baseline, participants with overweight and obesity presented with elevated scores on TFEQ disinhibition and hunger, as well as BES scores relative to their lean counterparts. Within the wider literature, not only is disinhibition score positively associated with BMI (Ernst et al., 2015; Thomas et al., 2013) and adiposity specifically (Lawson et al., 1995), but it is also associated with poorer quality diet choices which in turn contribute towards the development of obesity and poor health (Bryant, 2001). This indication that individuals with overweight and obesity are at a greater risk of a loss of control around food may partly explain why these individuals developed overweight and obesity in the first instance. However the current analysis cannot establish cause and effect and so this argument must be made with caution.

Previous evidence has also demonstrated that individuals with a lower restraint score report a greater energy intake from sweet foods than individuals with a higher restraint score (French et al., 1994). If energy intake from sweet foods is assumed to be a proxy indication of sweet preferences, the current findings displaying a positive association between EL sweet bias and TFEQ restraint – albeit weak in strength - would disagree with this. The current findings also disagree with previous evidence demonstrating that disinhibition and hunger scores are related to liking for foods in dieters (Lähteenmäki & Tuorila, 1995), leading authors to conclude that disinhibition and hunger are associated with hedonic responses to food. However, the current

findings do not replicate this, and instead demonstrate a positive association between restraint and hedonic responses of liking and wanting. These incongruent findings may be explained via the samples utilised and the data collection methods employed. Lahteenmaki and Tuorila (1995) utilised questionnaire responses from 253 female respondents taking part in a commercial weight loss program, with such techniques requiring interpretation with caution due to factors such as social desirability bias (Hebert et al., 1995). Furthermore, the mean rate of attendance at group meetings was 11 weeks, meaning these participants were not assessed at the beginning of a weight loss attempt and so any weight loss associated changes may have already occurred at the time of data collection, whereas the current study represents a clinical trial in a lab environment with baseline data recorded at the beginning of, during and end of an intervention. Additionally, the age range was from 15 to 79 years compared to 20 to 54 years in the current sample, considering that it is known that age creates variation in sweet preferences (Bartoshuk, 2000; Desor & Beauchamp, 1987) this may explain differences in findings between the current study and that of Lahteenmaki and Tuorila.

In the present analyses, sweet food preferences were not associated with binge eating score at baseline as was hypothesised based on evidence that sweet preferers report a greater number of binge eating episodes than lower sweet preferers (Goodman et al., 2018). However, Goodman examined binge eating frequency as a proxy for eating disorder symptomatology in the previous 28 days, rather than binge eating score, which may explain the difference in findings. Moreover, the Eating Disorder Examination-Questionnaire used in the Goodman study has demonstrated low inter-rater reliability on individual items that rate binge eating in a non-clinical sample (Rosen et al., 1990). This may suggest that binge eating frequency is not necessarily indicative of trait binge eating, as it has been previously stated that other variables are also important in triggering a binge episode, such as negative affect (Schulz & Laessle, 2010), which may further explain the unexpected findings present. Additionally, previously it was demonstrated that specifically HFSW intake was positively associated with binge eating score (de Zwaan, 2001; de Zwaan et al., 1992; Finlayson et al., 2011), which may suggest that an overall sweet food preference is not sensitive enough to display an association if one is

indeed present. Indeed, foods selected during a binge episode are typically those high in both sugar and fat (Yanovski et al., 1992).

The absence of a significant association between sweet food preferences and either craving control or sweet craving suggests that preferences and cravings, although often associated, are distinct aspects of food reward. This extends the claim by Robinson and Berridge (1993) - that craving (described as pathological wanting in the context of substance abuse) and liking are distinct - beyond pathological wanting and can be applied to cravings for foods or nutrients. Additionally, the most commonly craved food item is chocolate, an example of a HFSW food, not only sweet, (Zellner et al., 1999; Zellner et al., 2004) and tend cravings to be directed towards specific foods, rather than a general taste (i.e., sweet).

When controlling for BMI there exists in the current data weak but significant negative associations between IW sweet bias with disinhibition and BES scores, as well as choice sweet bias and BES score. This indicates that individuals scoring highly in disinhibition and BES scores have less of a bias towards sweet foods, and a greater bias towards savoury foods, which is independent of BMI. This is an unexpected finding, as elsewhere an elevated IW for HFSW foods was shown to interact with disinhibition (Finlayson et al., 2012) and is related to increased consumption of sweet foods (Bryant, 2001; Haynes et al., 2003). Specifically, individuals presenting high disinhibition scores report increased intake of sweet foods such as ice-cream (Lähtenmäki & Tuorila, 1995), therefore it may be that an overall sweet bias is not sufficiently sensitive enough to capture this relationship, if it is only expressed for HFSW foods. Nevertheless, future research may wish to further explore this negative association.

Furthermore, recent evidence has claimed that due to the overlapping nature of a number of eating behaviour trait constructs, a reconsideration is due, grouping these traits under the term 'uncontrolled eating', specifically grouping together disinhibition and hunger (Vainik et al., 2015). As disinhibition and hunger strongly correlate (Price et al., 2015) this may indicate that participants do not adequately differentiate between the different reasons for over consumption. Therefore when examining possible phenotypes of obesity, disinhibition and hunger may be

better phrased as an uncontrolled eating phenotype. This notion is strengthened when considering the current finding that BES score was also significantly higher in participants with overweight and obesity, a measure which assesses the behavioural and affective symptoms preceding a binge, or loss of control surrounding food intake. It has previously been suggested that binge eating should be characterised by the loss of control as opposed to the quantity of food ingested (Niego et al., 1997). Loss of control surrounding eating has been suggested to be expressed as a continuum, with the lower end of the spectrum regarded as passive overeating and binge episodes occurring at the other (Vainik et al., 2015). Therefore, these three eating behaviour traits may reflect a single obesity phenotype, however further research is required specifically examining this in order to draw firm conclusions as this was beyond the scope of the current analysis. Nonetheless, the inverse association shared between implicit sweet preferences and TFEQ disinhibition and BES may suggest that the presence of an implicit sweet bias in an individual is not sufficient to illicit a loss of control around food. The fact that this relationship was shown whilst controlling for BMI indicates that it may be a relationship that is uninfluenced by body mass.

5.4.2 Differences in Eating Behaviour Traits between Sweet Liker Phenotypes in Individuals with Overweight and Obesity in Response to Weight Loss

The evidence outlined a significant difference between high and low sweet liker phenotype groups on body fat percentage at baseline, but not differences in body weight, fat-free mass or fat mass. This is informative as during the assessment of body composition, body fat percentage provides a more informative measure than absolute values relying on weight (e.g., body weight, fat-free mass or fat mass), as it accounts for an individual's adipose tissue as a proportion of their overall body composition. However, this should not be interpreted as meaning that a low sweet preference is protective of obesity, as the analysis involving sweet phenotype groups consisted of only participants with overweight and obesity. Moreover, the study protocol employed was not designed to detect differences between phenotype groups, and so recruitment did not factor equal group size, resulting in unequal groups. Therefore

differences between groups in body composition will foremost be small, and must be made with caution. Similarly, phenotype groups were also not associated with rate of weight loss, findings which are supportive of a recent review which concluded that there is a lack of clear support for sweet liking as a major risk factor for obesity (Armitage et al., 2021). However, the current findings fail to demonstrate support for other evidence which has suggested differences in BMI, fat-free mass or fat mass levels between sweet liker phenotype groups (Armitage et al., 2021). However, these differences between phenotype groups were noted when age was considered, which was not observed within the current analysis. This consideration is not possible in the current data set as the protocol employed was not designed for a comparison of sweet phenotype groups and conclusions must be drawn with an awareness of the limitations of the protocol. Subsequently, these conclusions must be made with caution. Moreover, equal groups were not observed between high and low sweet liker phenotypes, as it was not a consideration during the recruitment and randomisation processes. Indeed, the unequal group sizes may be interpreted to indicate that individuals with overweight and obesity are more likely to be a high sweet liker than a low sweet liker. However, the current analysis represents an exploration into differences between sweet liker phenotype groups, and as such it is recommended that future research is conducted before drawing firm conclusions. Nonetheless, it is cautiously interpreted from these findings that a sweet food preference does not present a barrier to successful weight loss.

Over the course of the weight loss intervention, there were favourable alterations to a number of eating behaviour traits, with increases displayed in restraint and craving control, and declines in disinhibition, hunger, BES, sweet craving and savoury craving. Food craving is associated with difficulties in weight loss maintenance following successful weight loss (Fabbricatore et al., 2013) and may be a cause for concern in those having lost weight, however, the current findings demonstrate that cravings for both sweet and savoury foods can be diminished, as well as total craving control improved, following successful weight loss. Moreover, the significant differences reported between baseline and week 2 in craving control and sweet craving indicate that these cravings may not be directly linked to body weight. These alterations at week 2 occur

too rapidly to be created by meaningful changes in body fat. Food cravings are associated with lower diet quality and poor eating patterns (Taetzsch et al., 2020), therefore the reductions observed are likely due to the implementation of specific dietary protocols (i.e., the act of trying to restrict intake), which involved participants receiving food parcels from members of the research team on a weekly basis. Similarly, the increase in restraint score at week 2 must also therefore have been as a consequence of dietary alterations, it is anticipated that TFEQ restraint scores will increase whilst under conditions of energy restriction.

The interaction between time and sweet liker phenotype with changes in trait hunger is of interest, with high sweet likers displaying less of a reduction in trait hunger over a period of weight loss. This builds on previous evidence which has demonstrated extreme sweet likers score higher than other phenotype groups regarding TFEQ hunger (Iatridi et al., 2020). Early claims suggested that differences in sweet liking reflect an underlying physiologically sensed nutrient deficit (Cabanac, 1971) with support provided via studies which highlight stronger sweet liking ratings when in a hungry state relative to a satiated state (Rolls et al., 1983). More recent claims suggest that differences in sweet liking arise as high sweet likers are more sensitive to appetite signalling and therefore reflect state rather than trait differences (Armitage et al., 2021) in light of evidence demonstrating enhanced interoceptive eating in extreme sweet likers (Iatridi et al., 2020). However, research on sweet liker phenotypes is currently lacking, and so it is recommended that further work investigates the topic before firm conclusions are drawn.

5.4.3 Conclusions

Within the present chapter it was demonstrated that participants with overweight and obesity relative to lean controls at baseline did not differ in sweet food preferences, although they did present with greater scores on eating behaviour traits that involve a greater loss of control around food. Explicit components of food reward displayed weak, positive associations with TFEQ restraint scores, demonstrating an association independent of BMI. In individuals with overweight/obesity, there was no association between sweet food preferences and rate of weight loss, and there were no differences between sweet phenotype groups on eating

behaviour traits (except trait hunger) over the course of a dietary induced weight loss intervention, despite there being favourable improvements in a number of eating behaviour traits. Although it is acknowledged that further research is required in this area, it is cautiously concluded that a sweet food preference does not present a barrier to weight loss. However, high relative to low sweet likers experience less of a reduction in trait hunger during a period of dietary induced weight loss, which may contribute to poorer self-control around food.

6 A comparison of IER and CER pre- and post- 5% weight loss on sweet food preferences

Aims: Short-term energy restriction tends to increase food hedonics whereas sustained energy restriction produces a reduction. This generates the question of whether there are different responses produced by continual energy-restriction compared to intermittent energy-restriction. In the previous chapter, it was demonstrated that sweet food preferences vary within individuals acutely throughout a single day but remain highly stable across a period of weight-loss. The present chapter will examine the extent to which different dietary strategies lead to changes to sweet food preferences and eating behaviours during a dietary weight-loss intervention addressing the fourth objective listed in section 1.8.1.

6.1 Introduction

Any obesity associated alterations not reversed through weight-loss are likely pre-existing traits that may contribute to excess body weight and adipose tissue, for example, sensitivity to external signals related to the control of food intake (Berthoud & Zheng, 2012; Rodin et al., 1977), whereas those that are reversed are likely to be secondary effects of the obese state (Berthoud & Zheng, 2012), such as taste sensitivity and reward functions (Thirlby et al., 2006; Thomas & Marcus, 2008). Whilst evidence has demonstrated a significant association between fat-loss and changes to appetite sensations (Gilbert et al., 2009) as well as alterations to food preferences (Andriessen et al., 2018), it remains to be demonstrated as to whether the method of dietary restriction may interfere with these processes by altering the effect that weight loss has on specific traits such as sweet food preferences.

Via an increase in food reward (Berthoud et al., 2011) weight loss is able to create a compensatory drive to eat (Melby et al., 2017). The available literature highlights the duration of energy restriction as a key variable that may alter the impact on food reward – with short-term energy-restriction (<1day) demonstrated to increase the hedonic response to food

(Cameron et al., 2012; Stoeckel et al., 2007; Thivel et al., 2018) whereas, long-term restriction (>4weeks) reduces reward (Kahathuduwa et al., 2016; Meule, 2020b).

Continuous energy restriction (CER) is recommended to achieve clinically important weight-loss (NICE, 2014). Whereas, intermittent energy-restriction (IER) involves alternating periods of *ad libitum* energy intake and fasting days whereby participants consume 25% of energy requirements (Varady et al., 2009). The two methods have been demonstrated to produce comparable weight loss in adults with overweight or obesity (Davis et al., 2016; Harris et al., 2018). However, severe energy-restriction may increase energy intake on *ad libitum* days. In one study, energy intake was restricted to two thirds of estimated energy needs for two days, and on days three and four participants ate *ad libitum*. Findings showed positive associations between the degree of energy-restriction and *ad libitum* energy intake (Mars et al., 2005). Similarly, *ad libitum* energy intake increased by 74% following consumption following a completion of a fast (Cameron et al., 2014). Furthermore, in the current data set, it was highlighted that an increased feeling of hunger was reported in IER (means of fast and feed days) relative to CER, in addition to no change in sweet cravings in the IER condition versus a reduction at each time point in the CER condition (Beaulieu et al., 2021). This evidence demonstrates that the means of dietary induced weight loss may produce different outcomes in certain aspects of appetite and eating behaviour traits, and that specifically, following a fast *ad libitum* energy intake may increase. However, an examination on the influence exerted over sweet food preferences or intake remains to be conducted.

The above evidence generates the question of whether diet protocols varying in the pattern of energy restriction (namely CER or IER) impose differing effects on food preferences. In the current data set, Oustric (2021) demonstrated reductions to liking in LFPQ food groups (HFSA, LFSA, HFSW and LFSW) following weight-loss independent of intervention type in those participants whom achieved $\geq 5\%$ weight-loss. It remains to be demonstrated what the precise effects are of the differing diet interventions specifically on measures of sweet food preferences, intake and subsequent palatability ratings. The evidence outlined suggests that bouts of acute energy-restriction may increase food preferences as assessed by liking and

wanting ratings, comparatively, it may be hypothesised that CER may decrease food preferences at post-intervention. The aim of the current chapter is to compare the effects of IER and CER on female participant's measures of sweet food preference and intake during a dietary weight-loss intervention. This will provide a comparison of short-term extreme energy restriction, with continual moderate energy restriction.

Hypotheses:

- There will be a significant increase in sweet food preferences in both groups from baseline to week 2.
- There will be significant differences in sweet food preferences between groups across the diet intervention.
- Sweet food preferences will be significantly higher at week 2 in the IER group than in the CER group.
- Participants in the IER group will consume significantly more calories during the *ad libitum* meal at post-intervention than those in the CER group.

6.2 Methods

Forty-six participants were recruited on a voluntary basis at the University of Leeds and surrounding locations with eligibility determined using an online screening questionnaire. Participants were recruited separately as women with overweight or obesity, as determined by BMI. This study was approved by the University of Leeds School of Psychology Ethics Committee (ref: PSC-238, date: 10-01-2018). Participants consisted of individuals with overweight or obesity who then completed a baseline measures day, before beginning a weight loss intervention. Participants allocated the CER diet were required to consume 75% of energy needs daily, whereas those in the IER diet were required to consume 25% of energy needs one day, alternated with *ad libitum* feed days as shown in Figure 6.1.

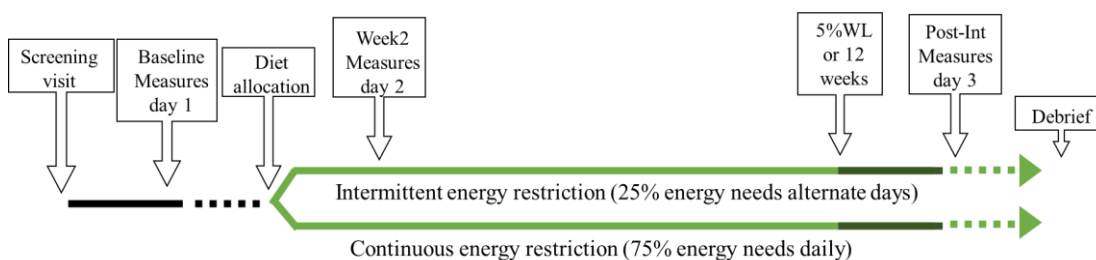


Figure 6.1. Study 1 timeline of diet intervention and measures days for participants with overweight and obesity.

All measures were conducted within the HARU at the University of Leeds. Participants arrived at the HARU after completing a 12-hr overnight fast and avoiding alcohol intake and physical activity for 24-hrs. Participants completed the LFPQ before body composition was measured using air displacement plethysmography (Bodpod, Concord, USA). Following consumption of the sweet breakfast participants were free to leave the lab and return 2-hrs 45-mins later – ensuring breakfast and lunch meals were as close to 3-hrs apart as possible. Upon returning to the lab, participants were provided an *ad libitum* lunch meal, representing savoury (risotto) and sweet (yoghurt) options, matched for energy density. Immediately following consumption of the *ad libitum* lunch meal, participants completed post-meal palatability VAS responses.

Participants were issued LighterLife food packets (see

Table 6.1), those in the CER group were provided with two packets per day (of sweet foods) as well as pre-portioned food parcels, whereas those in the IER group were provided four packets on the fast days (three sweet and one savoury food) and ate foods of their own selection *ad libitum* on feed days. Food packets were provided in powder form, requiring participants to add boiling water and heat in the microwave to prepare. Participants engaged in a weekly meeting with a trained dietitian to discuss compliance and if required (in the event of no weight-loss) reduce the allotted calorie intake. Participants completed a second measures day after 2 weeks, and a third measures day once weight loss $\geq 5\%$ was achieved or at 12 weeks – depending which occurred first. Weekly meetings took place with a trained dietitian where compliance was checked and if deemed necessary, adjusted to the allotted calorie intake were made.

A member of the research team calculated energy requirements based on measures RMR x PAL obtained from the SenseWear Armband. Weekly feedback provided by participants were used to adapt meal plans based on individual requirements and preferences. Foods were all pre-portioned (excluding milk, for which a measuring cup was also provided) resulting in minimal preparation time and were accompanied by daily food checklists. Consumption of beverages such as tea/coffee with the milk provided by the researchers was permitted (otherwise only black tea/coffee or herbal teas were permitted) as well as other energy-free beverages and participants were encouraged to drink plenty of water. Participants were required to report whether all food items provided were consumed, or specify how much remained in addition to any foods not included in the meal plan that were consumed.

Table 6.1. Table of LighterLife food products and energy and macronutrient compositions.

Food Item	Kcal	Fat (%)	CHO(%)	Pro (%)
Porridge	144.2	28.1	32.5	39.4
Vegetable soup	142.6	31.6	33.4	35.1
Scotch Broth	139.3	23.3	33.7	43.1
Three cheese pasta	142.2	27.8	35.6	36.6
Shepherd's pie	140.1	27.6	36.7	35.7
Spaghetti	139.9	27.0	37.3	35.7
Mug cake	137.8	29.4	34.0	36.6
Banana shake	142.6	29.0	33.4	37.6
Chocolate shake	132.2	25.2	35.8	39.0
Strawberry shake	140.7	29.4	33.3	37.2
Vanilla shake	141.1	29.3	33.0	37.7
Toffee bar	153.5	24.0	41.5	34.4
Peanut bar	148.8	24.8	41.3	33.9
Nut fudge bar	154.7	26.2	41.0	32.8

Abbreviations: CHO – carbohydrates. Pro – protein.

6.2.1 Data Analysis

The interaction effects of two key factors (diet type and time) were examined in order to compare differences between diet types at each time point. Anticipated differences between baseline and week 2 are likely to be as a consequence of changes to eating patterns and the act of dietary restriction. The degree of weight-loss that may have occurred at week 2 is anticipated

to be insufficient to elicit changes in the outcome variables of interest, with any effects observed at this time point attributable to the changes in eating behaviours (i.e., the dietary interventions). It is anticipated that differences between baseline and post-intervention are a consequence of changes in body composition and weight following the dietary weight loss intervention (Beaulieu et al., 2020). 3-way mixed ANOVAs were used to compare the differences between diet type (CER vs. IER) at each time point (baseline, week2 and post-intervention) as well as interaction effects.

Data were visually explored for extreme outliers using histograms and boxplots, with no extreme outliers being identified according to the Tukey method (Tukey, 1977). Mauchley's test of Sphericity was conducted and where necessary Greenhouse-Geisser corrections were applied. All data were analysed using SPSS v25.

6.3 Results

A total of 46 participants were included at baseline, with 22 being allocated to the CER diet and 24 the IER diet. Two participants in the CER group withdrew before week 2 and one withdrew before post-intervention. In the IER group, six withdrew before week 2 and six withdrew before post-intervention. There were no baseline differences between diet type, those that completed the intervention and those that achieved $\geq 5\%$ weight-loss (Beaulieu et al., 2020). Baseline study characteristics for each group can be seen in Table 6.2.

Table 6.2. Baseline study characteristics of participants by group.

	CER	IER
<i>n</i>	22	24
Age (years)	34 (9)	35 (11)
Body mass (kg)	78.6 (10.0)	81.2 (13.0)
Height (m)	1.65 (0.7)	1.66 (0.9)
BMI (kg/m ²)	28.9 (2.3)	29.4 (2.5)
Fat Mass (kg)	32.3 (7.3)	34.5 (8.7)
Fat Free Mass (kg)	46.3 (5.5)	46.7 (5.9)
Body Fat %	40.8 (5.7)	42.0 (4.6)
Total energy intake (Kcal/day)	1951 (374)	1783 (438)

Abbreviations: CER = continual energy restriction. IER = intermittent energy restriction. BMI = body mass index.

There were no significant differences between groups at baseline in anthropometrics, nor were there differences in self-reported energy intake. All participants reduced BMI, body mass, fat mass, fat-free mass and body fat percentage a significant amount across the intervention, although there were no differences between groups (Beaulieu et al., 2020).

6.3.1 Sweet Food Preferences (LFPQ)

A mixed ANOVA with time (3 levels) was conducted to compare differences in diet groups on choice, EL, EW and IW sweet bias scores, as well as the interaction effects between group and time. As shown in Chapter 4 there was not a main effect of time on sweet bias outcomes.

Further results displayed here show that there was not a significant time x diet type interaction effect for Choice sweet bias, $F(2,70) = .098, p=.906$, EL sweet bias, $F(1.527,53.452) = 1.095$,

$p=.340$, EW sweet bias, $F(2,70) = .293$, $p=.747$, nor IW sweet bias, $F(2,70) = .027$, $p=.974$.

Results can be seen in Figure 6.2.

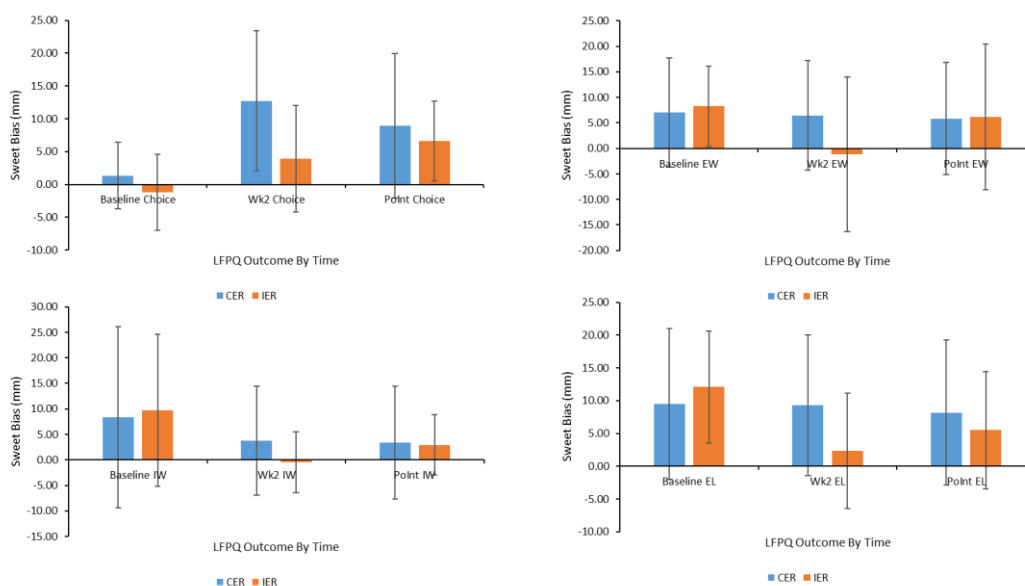


Figure 6.2. Leeds Food Preference Questionnaire Sweet Bias scores by group across the weight-loss intervention.

Error bars represent standard deviation.

Differences between groups were not significant.

6.3.2 *Ab Libitum* Intake

A mixed ANOVA was conducted with time (3 levels) on yoghurt intake (kcal) during the *ad libitum* test meal with diet type as the between subjects factor. Mauchley's test of Sphericity was non-significant ($p=.733$). Results did not show a significant main effect of time, $F(2,70) = 1.140$, $p=.326$, nor a significant time x diet type interaction, $F(2, 70) = 2.956$, $p=.059$.

A mixed ANOVA was conducted with time (3 levels) on risotto intake (kcal) during the *ad libitum* test meal with diet type as the between subjects factor. Mauchley's test of Sphericity was non-significant ($p=.134$). There was not a significant main effect of time, $F(2,70) = .492$, $p=.614$, nor was there a significant time x diet type interaction, $F(2,70) = 1.439$, $p=.244$.

6.3.3 Post-Meal Palatability Responses

A mixed ANOVA was conducted with time (3 levels) on sweet liking ratings and diet type as the between subjects factor, to examine differences between groups at time points. Mauchley's test of Sphericity was significant ($p=.009$). There was not a significant main effect of time

found, $F(1.594, 52.592) = .011, p=.975$, nor a significant time x diet type interaction, $F(1.594, 52.592) = .613, p=.510$.

A mixed ANOVA was conducted with time (3 levels) on savoury liking ratings and diet type as the between subjects factor, to examine differences at time points. One participant was excluded as an extreme outlier. Mauchley's test of Sphericity was non-significant ($p=.124$). There was not a significant main effect of time, $F(2,70) = 2.327, p=.105$, as well as a non-significant time x diet type interaction, $F(2,70) = .951, p=.391$.

A mixed ANOVA was conducted with time (3 levels) on subjective ratings of 'want more sweet' and diet type as the between subjects factor, to examine differences at time points. One participant whom did not consume any yoghurt at post-intervention was not included in the analysis. Mauchley's test of Sphericity was significant ($p=.043$). There was a significant main effect of time, $F(1.697, 56.015) = 4.368, p=.017$. Pairwise comparisons showed that subjective ratings of 'want more sweet' at baseline significantly differed to those at week 2 ($p=.024$) with a higher score at baseline than week 2, the difference between baseline and post-intervention was non-significant ($p=.088$) as was the difference between week2 and post-intervention ($p>.999$). There was also not a significant time x diet type interaction, $F(1.697, 56.015) = 2.928, p=.070$. A further one-way ANOVA was conducted to explore subjective ratings of 'want more sweet' between groups at time points. Results showed a significant difference between groups at baseline, $F(1,44) = 4.628, p=.037$, and at week2, $F(1,40) = 5.408, p=.025$, although there was not a significant difference between groups at post-intervention, $F(1,34) = .243, p=.625$. Findings can be viewed in Figure 6.3.

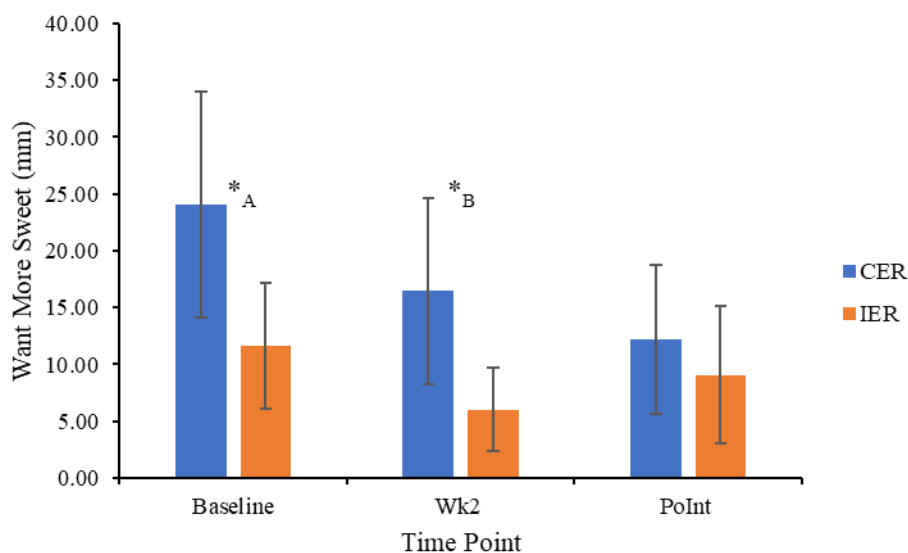


Figure 6.3. Bar chart displaying post-*ad libitum* wanting more sweet VAS ratings across the intervention by group.

Error bars represent standard deviation.

*A = significant difference between groups at baseline.

*B = significant difference between groups at week 2.

Abbreviations: CER = continuous energy restriction. IER = intermittent energy restriction.

Wk2 = week 2. PoInt = post-intervention.

A mixed ANOVA was conducted with time (3 levels) on savoury wanting more ratings and diet type as the between subjects factor, to examine differences at time points. One participant was removed from baseline, one was removed from week2 and one from post-intervention as extreme outliers. Mauchley's test of Sphericity was non-significant ($p=.152$). There was not a significant main effect of time, $F(2,64) = 1.510$, $p=.229$. There was also not a significant time x diet type interaction, $F(2,64) = .568$, $p=.570$.

A mixed ANOVA was conducted with time (3 levels) on sweetness ratings with diet type as the between subjects factor, to examine differences between groups at time points. Mauchley's test of Sphericity was significant ($p=.046$). Results did not show a significant main effect of time, $F(1.702, 52.592) = 2.067$, $p=.143$, nor a significant time x group interaction, $F(1.702, 52.592) = .015$, $p=.975$.

A mixed ANOVA was conducted with time (3 levels) on savoury ratings with diet type as the between subjects factor, to examine differences at time points. Mauchley's test of Sphericity

was non-significant ($p=.083$). There was not a significant main effect of time, $F(2,70) = .278$, $p=.758$. There was also not a significant time x diet type interaction, $F(2,70) = .105$, $p=.900$.

6.4 Discussion

The aim of the present chapter was to compare the effects of IER and CER dietary weight loss interventions on sweet food preference and intake in women with overweight and obesity. No difference was found between diet groups at any time point on sweet food preferences, nor was there a difference in *ad libitum* intake or post-meal palatability responses between groups. Both groups displayed a reduction in post-meal subjective ratings of ‘want more sweet’ from baseline to week 2, although differences between groups were only significant at baseline and week 2 and not at post-intervention.

The evidence outlined suggests that there was no difference in sweet food preferences or intake produced by either diet protocol. This refutes the hypothesis that acute yet extreme bouts of energy restriction will produce increases in sweet food preferences, due to the persistent acute energy restriction. It is possible that any alterations that may have occurred due to the energy restriction, were negated by intake on feed days – however future work may wish to investigate this further. The findings also refute the notion that CER will produce an increase in sweet food preferences over the short-term, proceeded by a reduction over a sustained period.

Similarly, the increased exposure of sweet LighterLife products provided to the IER group did not impact preferences. However, it was not possible to control for food selection by participants in the IER group nor was it possible to quantify sweet exposure on feed days in the IER group, with it possible that food preferences were impacted by this as those in the IER were able to consume foods of their choice during feed days.

6.4.1 Differences Between Groups in Sweet Food Preferences During and Following Weight Loss

The available data demonstrated that there was no impact of either of the implemented diet types at any time point during the intervention and that there were no differences between the groups at any time point on overall sweet food preferences as assessed via the LFPQ, thereby refuting all hypotheses. This is in contrast to previous work demonstrating alterations to food preferences following weight-loss in overweight and obese adults (Andriessen et al., 2018),

although participants in the Andriessen study experienced a greater degree of weight loss than participants within the current study (8% versus 5% in the current data set). It may be possible that a minimum of 5% weight loss was not sufficient to produce comparable alterations.

However, Beaulieu et al., (2020) previously demonstrated no differences at post-intervention

Although, within the present data set, it was shown that food liking for specific food categories varying in fat and taste reduced in both diet groups (Oustric et al., 2021). Therefore, it may be that an overall sweet preference such as the method employed in the current analysis, is unable to detect changes to preferences. It may be that you need to look at different forms of sweet foods (i.e., those varying in fat content as well) to identify any differences.

It is of interest that there were no differences between the diet groups despite evidence suggesting that short-term energy restriction increases food hedonics (Berthoud et al., 2011; Cameron et al., 2012; Stoeckel et al., 2007; Thivel et al., 2018). It was hypothesised that acute, yet extreme bouts of energy-restriction may increase sweet food preferences, an effect which would be observed following the completion of the dietary intervention, however from the present findings it is suggested that the alternated *ad libitum* feed days potentially negated this effect. Taken together, these findings may suggest that overall sweet or fat food preferences remain unaltered through weight-loss, with changes actually occurring within specific food groups.

Differences at week 2 were included in the analysis as any differences between groups were hypothesised to be due to the implementation of the differing diet protocols, with changes to body weight being insufficient at this stage to illicit a response. However, there were no differences between groups at week2 on any outcome, refuting the third hypothesis although this finding strengthens the conclusion that neither diet strategy sufficiently impacted sweet food preferences. However, this finding is in line with previous evidence demonstrating that IER failed to generate compensatory mechanisms in appetite regulatory systems (Alhamdan et al., 2016; Coutinho et al., 2018).

6.4.2 Differences in *Ad Libitum* Intake

It was hypothesised that for those in the IER group, a measures day would represent a non-restricted feed day and so would lead to a greater energy intake. However, this was not found, with there being no differences between groups at any time point in either sweet or savoury food intake, refuting the fourth hypothesis. This may be due to the *ad libitum* test meal representing only a single eating occasion, if energy intake was assessed across the entire 24hr period, findings may have been similar to those by Mars et al., (2005). Furthermore, the *ad libitum* test meal consisted of risotto (savory) and yoghurt (sweet) which differed in their relative sweet/savory taste, but not in their fat content. However, given that there was no alteration to sweet food preferences within the present analysis, it may be concluded that alterations to energy intake (Mars et al., 2005) is not due to alterations to preferences, and is likely explained via alternative compensatory drivers. Given the findings by Oustric et al., (2021) that preferences for foods varying in fat and sweet/savory composition, this may represent a methodological flaw. Future studies may wish to provide a selection of foods varying along these parameters to provide a more in depth consideration of food preferences and intake.

6.4.3 Differences in Post-Meal Palatability Responses

There were no differences between groups in post-meal palatability responses, except wanting for more sweet ratings which were elevated in the CER group at baseline. Both groups displayed a reduction in wanting for more sweet between baseline and week 2. Previous evidence has highlighted that calorie deprivation may increase food reward independent of weight-loss (Cameron et al., 2008) and so it is of interest that ratings declined in those in the CER over the course of the intervention. The elevation at baseline is likely due to random error within the data and is not a meaningful difference as participants were randomised to conditions and recruitment followed a stratified process and had not begun the dietary intervention at the time of baseline measurements being taken. However, this finding may represent an alteration in sweet food preferences in the CER group. The large change noted at week 2 – prior to meaningful alterations to body weight or composition – support the findings

of Cameron (2008) that alterations to food reward occur independently of weight-loss, however, occurring in the opposite direction in the present data. From week2 to post-intervention, ratings declined in the CER, providing a non-significant difference between the two diet groups. The reduction in ratings at week2 also contradicts previous findings demonstrating short-term restriction increases food hedonics (Cameron et al., 2012; Stoeckel et al., 2007; Thivel et al., 2018). However, the non-significant difference at post-intervention is supportive of findings demonstrating that long-term energy restriction produces a reduction in hedonics (Kahathuduwa et al., 2016; Meule, 2020b).

6.4.4 Conclusions

The findings of this chapter demonstrate that CER or IER do not differently impact sweet food preferences and intake during weight-loss. Taken together these findings suggest that diet modality exerts limited influence over food preferences and eating behaviours with it likely that any effects produced are as a result of simply undergoing a diet-induced weight-loss intervention.

7 The Effects of Acute and Repeated Ingestion of Sucrose vs. High-Intensity Sweeteners formulated in biscuits on Sweet Food Preferences and Related Eating Behaviours

Aims: The use of HIS as a means to retain sweetness while replacing or reducing sugar in solid foods has increased in recent years. Currently, there is only limited evidence available regarding their effects when consumed as part of reformulated biscuits or on subsequent food preferences and eating behaviours. The aim of the current chapter is to use the Leeds Food Preference Questionnaire as a measure of sweet food preferences, to assess the acute and repeated effects of sucrose or two types of HIS (Neotame or Stevia Rebaudioside M) on sweet food preferences and eating behaviours in men and women with overweight and obesity, addressing the fifth objective listed in section 1.8.1.

7.1 Introduction

There remains much speculation regarding the effects of HIS on subsequent food preferences and food intake, particularly in light of evidence suggesting that consumption of HIS leads to an increased risk of overweight and obesity (Azad et al., 2017; Bruyère et al., 2015). However the abundance of observational studies and lack of randomised controlled trials leaves the issue of reverse causation largely unaddressed (Borges et al., 2017). Moreover, the preponderance of evidence available utilises beverages as the vehicle of administration (O'Connor et al., 2021), due in part to the difficulties of replacing caloric sweeteners for HIS in solid food matrices (Luo, 2019), which requires a greater degree of reformulation than beverages. Therefore it is imperative to investigate the effects of HIS reformulated foods, in comparison to sucrose controls, on subsequent sweet food preferences and eating behaviours.

There is only limited evidence available regarding the effects of HIS on subsequent food preferences and food intake, with a greater weight of evidence examining preferences for HISs compared to caloric sweeteners. For example, despite a poor ability to discriminate between sweetener types in products, foods utilising caloric sweeteners may be more well-liked relative to their HIS counterparts (Delogu et al., 2016; Li et al., 2015). Similarly, following acute

consumption HIS beverages received lower liking ratings (Thai et al., 2011) and lower pleasantness ratings (Delogu et al., 2016) than SSB counterparts. Differences tend to be driven by sweetener type and bitterness perception (Kamerud & Delwiche, 2007), as some sweeteners have been reported to leave a metallic aftertaste (Portmann & Kilcast, 1996). From this, it may be cautiously inferred that HIS sweetened products are less well accepted than caloric sweeteners and may as a consequence reduce sweet food preferences.

Concerns have been expressed that acute consumption of HIS will increase sweet food preferences and subsequent intake (Mooradian et al., 2017). Early evidence demonstrated that acute HIS consumption increased motivational ratings and food preference checklist responses, although there was no differentiation made between sweet and savoury foods (Rogers et al., 1988). Although limited, contemporary evidence is contradictory, demonstrating acute consumption of HIS increases motivation towards sweet foods (Casperson et al., 2017), or exerts no effect (Fantino et al., 2018b). Review of the evidence highlights that HIS does not increase acute energy intake (O'Connor et al., 2021; Rogers et al., 2016) which may be interpreted as not impacting subsequent food preferences as food intake is considered to be an indication of preferences (Drewnowski & Hann, 1999). However, when examining energy intake in previous studies, the primary consideration is subsequent energy intake in its entirety and studies fail to differ between energy provided from different sources (e.g., energy from sweet or energy from savoury). Through the use of the LFPQ, this will be addressed by providing an examination of differences in preferences for sweet and savoury.

Additionally, the wider literature tends to treat HIS as a single homogenous group, despite some being plant-based whilst others are artificially manufactured. Emerging evidence also demonstrates a potential for different effects in body weight or energy intake across HIS type, with saccharin producing an increase in body weight over 12 weeks of consumption, whilst rebA and sucralose did not produce any alteration over the 12 weeks (Higgins & Mattes, 2019). It is therefore overly simplistic to treat all HIS types as synonymous with one another and potential differences across sweetener types warrants investigation.

Repeated exposure to a specific flavour promotes an increased preference (Liem & De Graaf, 2004) and habitually consumed items become liked and preferred over initially equivalent or initially preferred alternatives (Mela, 1999). This generates concerns that habitual HIS consumption may encourage sugar cravings and sugar intake precisely because of their sweet taste (Bello et al., 2018; Yang, 2010). However, following sweet taste stimulation low habitual HIS consumers demonstrate an increased appetite and energy intake in a test meal compared to high habitual HIS consumers (Appleton & Blundell, 2007). Therefore, although repeated exposure may increase a subsequent preference, it may also produce a decoupling of the association between sweet taste and energy (Delogu et al., 2016; Dhillon et al., 2017) resulting in different appetite responses.

Moreover, in participants categorised as high or low sweetened beverage consumers high consumers reported greater liking for increasing sweetness concentrations, with no effect of sweetener type on liking ratings (Mahar & Duizer, 2007) - thus sweetener type may be of less importance than general consumption of sweet food and drinks, when influencing preferences. Supporting this notion, is evidence provided via a 12-month intervention, in which consumption of HIS beverages or SSB did not result in any change in preferred sweetness concentration, whereas consumption of an unsweetened beverage resulted in a significant decline (Ebbeling et al., 2020). Similarly, in a small sample, following exclusion of all added sugars and HIS for 2 weeks, 95% of responders reported that sweet food and drink tasted either sweeter or too sweet (Bartolotto, 2015). This evidence highlights the impact of regular exposure to sweetened products on sweet food preferences, with a greater exposure possibly increasing preferences. However, a review of available literature has indicated that repeated exposure may reduce sweet preferences on an acute basis (Appleton et al., 2018). Therefore, it is necessary to understand the impact of repeated exposure of HIS foods on subsequent sweet preferences.

In addition to potentially impacting preferences, it has been suggested that HIS may encourage sweet cravings (Mooradian et al., 2017; Roberts, 2015). Alternatively, more moderate suggestions are that specifically repeated exposure to HIS may establish and maintain

preferences for sweet items in the diet (Mattes & Popkin, 2009), as repeated exposure trains food preferences (Liem & De Graaf, 2004). However, within the CHOICE trial, HIS beverages when compared to water produced a reduction in energy intake from sugars and desserts (Piernas et al., 2013), thereby indicating that HIS may actually satisfy sweet cravings (Rogers, 2018). Elsewhere, it has been stated that individuals with a habitually elevated sweet craving may benefit from access to HIS, precisely for this reason (O'Connor et al., 2021). When examining differences in experimentally induced cravings in habitual and non-consumers of HIS, it was found that frequent consumers of HIS did not alter their eating behaviours, whereas non-users ate significantly more calories – from both sweet and savoury sources – relative to a control condition (Maloney et al., 2018). However, the literature surrounding the impact of HIS ingestion on food reward is not well understood, and additional studies are required (O'Connor et al., 2021).

The aim of the following chapter is to provide an examination of the impact of acute and repeated consumption of HIS and sucrose sweetened products on subsequent food preferences and eating behaviours. The current study represents a randomised split-site cross-over trial, utilising three distinct sweetener conditions. The three conditions will be compared both acutely (i.e., after consumption in the morning) and repeated (i.e., after 14 consecutive days of consumption). The primary outcomes will be LFPQ sweet preference outcomes (i.e., EL, EW, IW and Choice sweet bias). Secondary outcomes are the CoEQ and subjective appetite ratings, whilst exploratory outcomes are body weight and composition, and 24hr dietary recall outcomes. At the time of writing sweetener conditions remain blinded and so attributing differences in effects on outcomes to a specific sweetener type is not possible. Additionally, as demonstrated in Chapter 2, there is a scarcity of evidence available examining the effect of consumption of different sweetener types on subsequent sweet food preferences, particularly when sweeteners are provided in a solid food product rather than a beverage, and there is a focus on a select number of HIS within the literature currently. As such it is not possible to provide directional hypotheses within the current analysis. Therefore, analysis will consist of exploratory comparisons across conditions and hypotheses will not specify differences between

conditions. In the event of significant main effects of condition on the outcomes are observed, it can be concluded that either HIS are different to sucrose, or that HIS are different to each other. However, in light of evidence which demonstrates differences in the reward value of sucrose and HIS (e.g., Delogu et al., 2016 or Smeets et al., 2011) it may be surmised that the absence of sucrose in the HIS conditions may lead to a compensatory increase in the reward value (i.e., EL, EW, IW and choice sweet bias) for sweet sucrose-containing foods in the LFPQ.

Hypotheses:

- There will be a significant reduction in co-primary outcomes (EL, EW, IW and choice sweet bias) following acute exposure to the intervention products.
- There will be a significant difference in co-primary outcomes (EL, EW, IW and choice sweet bias) and secondary outcomes (CoEQ and subjective appetite ratings) following repeated exposure (14 days) to the intervention products.
- There will be a significant difference between conditions in co-primary outcomes (EL, EW, IW and Choice sweet bias) and secondary outcomes (CoEQ and subjective appetite ratings) following acute consumption.
- There will be a significant difference between conditions in co-primary outcomes (EL, EW, IW and Choice sweet bias) and secondary outcomes (CoEQ and subjective appetite ratings) following repeated exposure (14 days) to the intervention products.

7.2 Methods

The details outlined herein pertain to the specific analyses conducted within the present thesis chapter. A detailed outline of the SWEET trial can be seen in Chapter 3, section 3.3, and has published elsewhere (see Gibbons et al., 2022). The current findings have been collected as part of a wider EU project entitled SWEET. The wider SWEET project was a randomised split-site crossover trial, conducted across five intervention sites in four countries across three regions of Europe, with each site testing a different intervention product (matrix) whilst following the same protocol. The primary objective of the wider SWEET trial was to evaluate the effect of HIS reformulated products compared to a sucrose sweetened control product on a number of outcomes, including behavioural, metabolic and health related endpoints. Intervention products covered bakery (cake and biscuits), dairy (yoghurt), confectionary (chocolate) and breakfast cereal, the findings within the present chapter pertain to only those participants consuming biscuit products (data were collected at University of Leeds, England and Centre de Recherche en Nutrition Humain Rhône Alpes, France).

The primary endpoint of the wider SWEET trial was 180min iAUC (incremental area under the curve) composite score for all appetite sensations in response to each product, calculated using the trapezoid method (Blundell et al., 2010), and subsequent power calculations identified a sample size of 48 participants was required. Power calculations were not performed for secondary outcomes within the wider SWEET trial. However, published literature utilising comparable designs were consulted (e.g., Yeomans et al., (2016)) which demonstrated effects of small nutritional manipulations on various gut peptides. Sample sizes ranged in these studies from 12 to 23 participants, providing confidence that a sample size of 48 participants per matrix should be sufficient to detect differences with clinical significance (Gibbons et al., 2022).

Within the present analysis co-primary outcomes were LFPQ sweet bias outcomes (EL, EW, IW and Choice sweet bias), with secondary outcomes consisting of the CoEQ and subjective appetite ratings (VAS) and exploratory outcomes of body weight and composition and 24hr dietary recall outcomes.

7.2.1 Design

The procedures described here pertain only to variables relevant to the current analysis, other processes were included that are not mentioned here. A within-subjects design was utilised, with participants required to complete three distinct conditions utilising one of three different sweeteners, either sucrose (caloric sugar), stevia reb M (natural high-intensity sweetener) or neotame (artificial high-intensity sweetener) (conditions 550, 199 and 647). A balanced block design to randomly allocate product sequence, with each sequence stratified by sex and age group. Both participants and investigators remained blind to biscuit conditions throughout the data collection process and analyses. At the time of writing conditions have not been unblinded.

The duration of the protocol (displayed in Figure 7.1) for each participant was a minimum of 70 days and a maximum of 84 days. Each intervention period began with a probe day (PD 1), followed by 12-days of at-home intervention (PD 2-13) and finished with a probe day (PD 14). All probe days followed the same format, began at the same time for each participant and required participants to arrive at the lab in a fasted state between 07:30-10:00am.



Figure 7.1. Number of laboratory visits and time commitment per volunteer in the FAST study. Each visit is scheduled in the morning and lasts up to 4 hours.

7.2.2 Materials

The materials described herein pertain only to the current analysis. A number of additional materials and measures that are omitted within the current thesis were included within the SWEET protocol, for further details see Gibbons et al., (2022).

Sweet food preferences were assessed via the LFPQ, a computer based assessment technique which provides measurement of liking and wanting in explicit and implicit components, via visual image presentation. As outlined, the LFPQ sweet bias outcomes (EL, EW, IW and Choice) were the co-primary outcomes within the current chapter. Secondary outcomes were

assessed via the use of the CoEQ, providing a measurement of craving control as well as cravings for specifically sweet and savoury, in addition to positive mood. VAS iAUC subjective appetite ratings were also included as a secondary outcome, with the trapezoid method used for the calculation of iAUC (Blundell et al., 2010). Body weight and composition were assessed via the BodPod, with 24hr dietary recall assessed via phone calls with a trained member of the research team as close to 24 hours after leaving the lab as possible. Dietary recall provided values of total energy intake (kcal) and macronutrient intake (grams).

Intervention products represented jam filled biscuits and were selected to be representative of commonly consumed snack foods within the target population. Conditions comprised of one control product sweetened using sucrose, and two no added/reduced sugar reformulated products using one of two HIS (Neotame and Reb M). Intervention products were matched for sweetness intensity, flavour and physical appearance, and there was no difference in the perceived pleasantness between the biscuits. Stevia Reb M (95% Steviol Glycosides, 80% Rebaudioside M) as a stevia leaf extract was provided by Cargill (Vilvoorde, BE). Neotame was provided by ManusBio (Augusta, GA).

The reformulation of food products is extremely complex due to sugars contributing to a number of sensory aspects of food products and as such very limited amounts of sucrose can be reduced from cakes and biscuits without being replaced by suitable alternatives (Luo, 2019). Polyols are particularly important as sugar substitutes, which is explained in greater detail in (Roze et al., 2021). The difficulties involved in reformulating sugar sweetened products is reflected in the energy and macronutrient composition of the intervention products. Full nutritional information is available in Table 7.1

For further details of the materials utilised within the SWEET trial see sections 3.1 and 3.3 of Chapter 3.

Table 7.1. Energy and macronutrient composition of the intervention products.

	Control product		Reformulated product	
	Per 100g	Per portion (3 biscuits)	Per 100g	Per portion (3 biscuits)
Energy (kcal)	423	360	384	326
CHO (g)	75.9	64.5	76.2	64.8
Sugar (g)	24.7	21.0	1.8	1.5
Polyols (g)	3.7	3.1	22.7	19.3
Fat (g)	11.2	9.5	11.5	9.8
Sat. fat (g)	7.11	6.0	7.33	6.2
Protein (g)	6.5	5.5	6.6	5.6
Fibre (g)	0.7	0.6	2.4	2.0
Salt (mg)	0.7	0.6	0.7	0.6

Abbreviations: Sat – saturated. CHO – carbohydrate.

7.2.3 Procedure

Participants were screened prior to enrolment in the study. During screening visits, participants at the University of Leeds completed site optional questionnaires – the TFEQ and BES. These questionnaires were only completed at the University of Leeds and as such, any analyses utilising these measures are conducted on a sub-set of the total study population. At screening, the sFFQ was completed by participants at all locations.

Participants were instructed to consume a similar meal the evening before each CID, before completing a fast for a minimum of 12hrs prior to attending the laboratory. Participants arrived at the lab for a CID between 07:30-10:00am in a fasted state. Upon arrival in the lab the protocol compliance questionnaire was completed, in the event of non-compliance the session was cancelled and re-arranged. Participants then completed a computer-based version of the CoEQ before body weight and composition was assessed using the BodPod.

A timeline of measures taken during a probe day can be seen in Figure 7.2, displaying measures included in the current analysis as well as those collected for use within the wider SWEET analysis. Participants provided fasting VAS (time -12mins, -2mins) and LFPQ responses (time -12mins) prior to consumption of the intervention product. At time point 0mins the intervention product was served alongside 200ml of still water (Figure 7.3). Participants were instructed that

they were allowed 9 minutes to consume the full portion of the intervention product and the water and were required to spread their consumption across the full 9 minutes. Further VAS responses were completed (+10, 20, 30, 45, 60, 120 and 180mins) in addition to a post-consumption LFPQ (+20mins). Following completion of the final VAS response (180mins), participants were free to leave the lab.

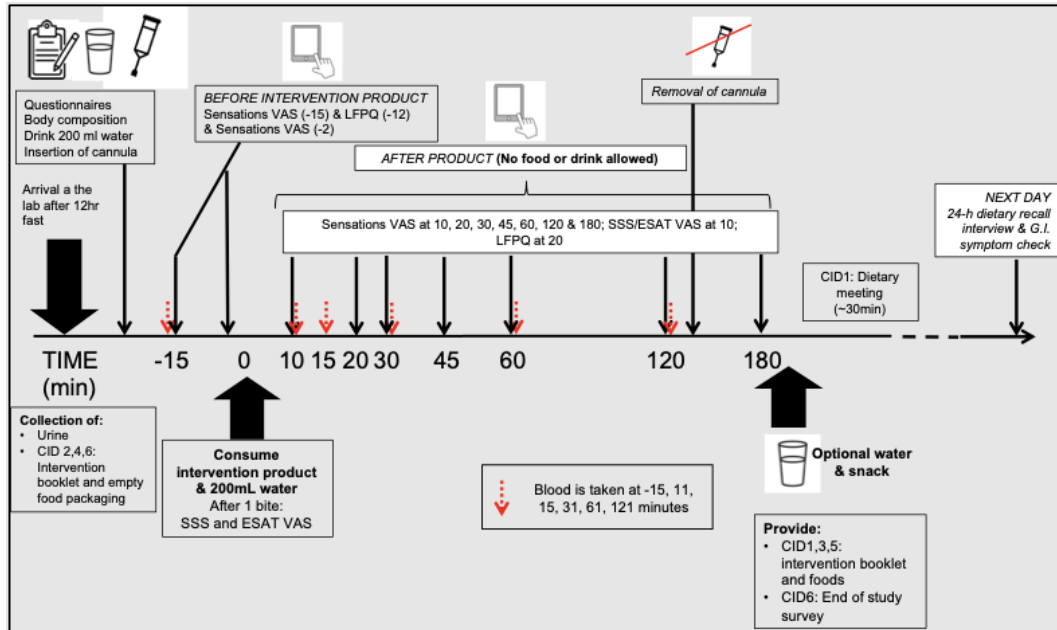


Figure 7.2. Timeline of a probe day during the SWEET trial.



Figure 7.3. Presentation of intervention products to participants at each probe day.

At PD1 participants were provided with a portion of study food for each of their at-home intervention days and a substitution strategy was agreed upon (for full details see Chapter 3). On PD14 participants began a 14-day wash-out period in which they were instructed to maintain their habitual dietary and physical activity patterns. 24-hrs after leaving the lab participants received a phone call from a trained member of the research team and asked to recall all food and drink that had been consumed since leaving the lab, until the phone call. This information was then manually entered into relevant dietary recall software (WinDiets) to provide total energy and macronutrient intake values.

The product sequence was randomly allocated into blocks of six via a Latin square design (six treatment orders). The individual responsible for generating the sequences for all sites did not have any study related tasks (e.g., inclusion or exclusion of participants, data collection). Each sequence was stratified by sex (female/male) and age (18-45 years/46-60 years). The blinding of the intervention codes and allocation of product codes (i.e., 199, 550 and 647) was completed by the manufacturers of the intervention products, thereby enabling the blinding of the research staff.

7.2.3.1 Data Analysis

The data in the current chapter was collected at two sites – the University of Leeds, England (UoL) and Centre de Recherche en Nutrition Humaine Rhône-Alpes, France (CRNH-RA). Analyses including the TFEQ and BES consisted of only UoL participants with all available data used ($n=28$ due to missing data for one participant). Where possible, analyses included participants from both intervention sites, with collection site controlled for as a covariate. Other covariates considered were age, gender and habitual sweet consumption (sFFQ), due to their known impact on sweet preferences (Desor & Beauchamp, 1987; Drewnowski, 1997; Wansink et al., 2003).

The primary outcomes of interest for this thesis were sweet bias outcomes as assessed by the LFPQ. A series of 3x2x2 repeated measures ANOVAs compared differences across intervention conditions between PD1 and PD14, and pre- and post-ingestion. Secondary outcomes of interest were the CoEQ and VAS appetite ratings (specifically appetite for sweet

and appetite for savoury). CoEQ outcomes were assessed via 3x2 repeated measures ANOVAs, examining differences between conditions from PD1 to PD14. Appetite ratings were assessed via iAUC composite scores, with a 3x2 repeated measures ANOVA comparing differences across conditions and between PD1 and PD14. As outlined, due to the CoEQ being completed prior to ingestion of the intervention product, it was not possible to provide an examination of the effects of acute exposure on cravings utilising the CoEQ. Therefore acute effects on cravings were assessed via the use of VAS questions on probe days (i.e., appetite for sweet and appetite for savoury).

Finally, exploratory outcomes were assessed. Body composition outcomes over the 2-week intervention periods were assessed via 3x2 repeated measures ANOVAs to compare changes in body composition across conditions from PD1 to PD14. 24hr dietary recall was assessed via 3x2 repeated measures ANOVAs to compare differences in total energy (kcal) and macronutrient (g) intake across conditions and between PD1 and PD14.

Sphericity was examined via Mauchley's test of Sphericity and where appropriate Greenhouse-Geisser corrections were applied. Significant differences in ANOVA main effects or interactions were investigated further via post-hoc analysis utilising the Bonferroni correction. Significance levels were set at $p \leq .05$.

Potential covariates to be included were variables which have been demonstrated within the wider literature as having a capacity to influence sweet food preferences (habitual sweet intake, BMI, age and gender), as well as data collection site. Unadjusted and adjusted models were reported, with only covariates of note being included in the final adjusted models (i.e. those that were significant in the model). In the event that there were no significant covariates, only unadjusted models were reported.

7.3 Results

7.3.1 Participant Descriptive Statistics

A total of 1,106 participants were screened to take part in the study, as displayed in Figure 7.4.

A total of 34 participants were recruited into the trial at UoL, of which 29 completed all six probe days. CRNH-RA recruited 28 participants, of which 21 completed all six probe days, giving a combined final total of 50 participants in the present analysis. 19 participants were male and 31 were female. Baseline eating behaviour traits and body composition are displayed in Table 7.2.

Table 7.2. Participant descriptive statistics at PD1 $n=50$.

Variable	Total sample	UoL ($n=29$)	CRNH-RA ($n=21$)
Variable	Mean (SD)	Mean (SD)	Mean (SD)
Age	41.78 (11.04)	41.21 (11.66)	42.57 (10.37)
EAT-26	3.82 (3.78)	3.48 (2.76)	4.29 (4.89)
Short sFFQ	8.48 (1.68)	8.69 (1.23)	8.19 (2.16)
CoEQ Craving Control	48.84 (20.28)	48.07 (21.01)	49.91 (19.68)
CoEQ Sweet Craving	50.90 (19.93)	51.27 (19.79)	50.38 (20.59)
CoEQ Savoury Craving	47.65 (15.73)	49.33 (16.46)	45.32 (14.74)
CoEQ Positive Mood	64.14 (15.37)	63.53 (17.74)	64.99 (11.69)
BMI (kg/m ²)	28.59 (2.75)	28.16 (2.95)	29.20 (2.39)
Bodyweight (kg)	82.04 (10.45)	80.55 (11.08)	84.11 (9.39)
Fat mass (kg)	29.08 (7.68)	27.93 (7.51)	30.67 (7.81)
Fat-free mass (kg)	52.97 (9.94)	52.62 (10.00)	53.44 (10.07)
Body fat percentage	35.49 (8.27)	34.71 (8.01)	36.57 (8.71)
TFEQ Restraint*	-	7.18 (4.63)	-
TFEQ Disinhibition*	-	6.46 (2.99)	-
TFEQ Hunger*	-	5.86 (3.40)	-
BES*	-	9.50 (6.41)	-

Abbreviations: EAT – eating attitudes test. sFFQ – sweet food frequency questionnaire. CoEQ – control of eating questionnaire. BMI – body mass index. SD – standard deviation.

*Represent site optional questionnaires $n=28$.

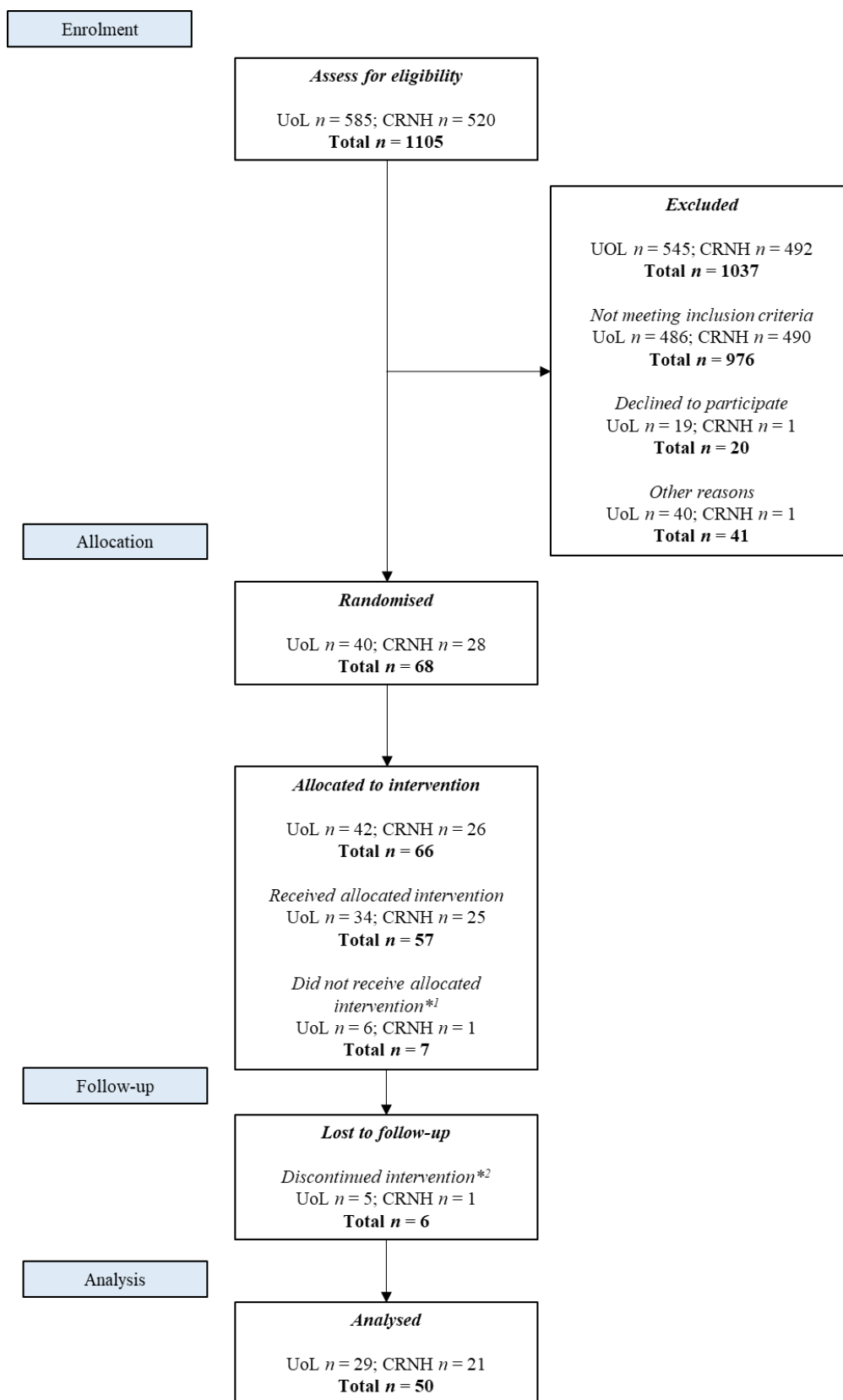


Figure 7.4. Participant recruitment flow diagram.

Abbreviations: UoL = University of Leeds. CRNH-RA = Centre de Recherche en Nutrition Humaine Rhône-Alpes.

7.3.2 Co-Primary Outcome

A series of 3x2x2 repeated measures ANOVAs with condition (3 levels), probe day (2 levels) and time (2 levels) included as fixed-factors, were conducted to compare differences between conditions between PD1 and PD14 from pre- and post-ingestion on LFPQ sweet bias outcomes. Sphericity was assumed for both time (pre/post-ingestion) and probe day with both having two levels. Means and standard deviations for LFPQ sweet bias outcomes can be seen in Table 7.3 with F and *p* values for both unadjusted and adjusted models reported in Table 7.4, with covariates of note reported.

Table 7.3. Means and Standard Deviations for Leeds Food Preference Questionnaire Sweet Bias Outcomes Pre- and Post-Ingestion across Probe Days and Conditions.

		199		199		550		550		647		647	
		PD1		PD14		PD1		PD14		PD1		PD14	
		Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
EL Sweet Bias	Mean	13.49	2.50	10.57	1.42	13.45	1.36	10.45	-0.91	10.63	3.13	11.20	1.45
	SD	24.99	19.96	25.47	24.92	23.53	22.26	23.68	28.22	27.61	26.01	23.98	22.33
EW Sweet Bias	Mean	11.44	1.22	8.42	-0.41	11.10	-1.51	8.26	-1.19	9.69	3.73	9.64	1.17
	SD	25.86	21.42	24.40	21.90	23.65	20.29	24.51	24.55	24.91	24.95	23.59	19.18
IW Sweet Bias	Mean	23.98	5.14	14.09	-1.71	19.26	2.67	17.25	4.93	18.60	3.32	20.73	2.57
	SD	42.01	43.11	43.29	54.00	41.26	45.66	42.48	41.08	43.92	50.22	44.72	44.38
C Sweet Bias	Mean	8.69	2.26	5.72	1.30	7.58	1.18	6.76	2.20	7.30	1.08	8.22	0.96
	SD	15.05	16.45	16.95	17.32	15.62	17.22	16.71	16.51	16.36	18.56	16.25	16.68

Abbreviations: EL – explicit liking. EW – explicit wanting. IW – implicit wanting. C – Choice. SD – standard deviation. PD – probe day.

Table 7.4. Models of adjusted and unadjusted repeated measures ANOVA results for Leeds Food Preference Questionnaire sweet bias outcomes.

		Unadjusted		Adjusted	
		F	<i>p</i>	F	<i>p</i>
EL Sweet Bias	Cond	0.145	.831	2.509	.097
	PD	3.530	.066	.280	.599
	Time	17.667	<.001	.655	.422
	Cond*PD	0.387	.680	.305	.738
	Cond*Time	1.125	.329	2.228	.113
	PD*Time	0.004	.947	.908	.345
	Cond*PD*Time	.424	.655	3.166	.054^a
EW Sweet Bias	Cond	0.677	.510	2.695	.081
	PD	2.555	.116	2.171	.147
	Time	14.545	<.001	8.116	.006^b
	Cond*PD	.143	.867	.369	.692
	Cond*Time	1.699	.188	4.011	.021^b
	PD*Time	0.141	.709	.002	.964
	Cond*PD*Time	0.911	.406	.711	.494
IW Sweet Bias	Cond	0.072	.896	-	-
	PD	1.859	.179	-	-
	Time	20.184	<.001	-	-
	Cond*PD	2.246	.111	-	-
	Cond*Time	0.312	.733	-	-
	PD*Time	0.207	.652	-	-
	Cond*PD*Time	0.860	.426	-	-
C Sweet Bias	Cond	0.007	.986	-	-
	PD	0.706	.405	-	-
	Time	18.625	<.001	-	-
	Cond*PD	1.134	.326	-	-
	Cond*Time	0.700	.499	-	-
	PD*Time	0.925	.341	-	-
	Cond*PD*Time	1.649	.198	-	-

^aExplicit liking sweet bias: sFFQ was included as the covariate in the adjusted model.

^bExplicit wanting sweet bias: gender was included as the covariate in the adjusted model.

Implicit wanting sweet bias and Choice sweet bias did not include any significant covariates

7.3.2.1 Explicit Liking Sweet Bias

Sphericity was violated for condition ($p=.008$) but was assumed for condition*probe day ($p=.459$), condition*time ($p=.432$) and condition*probe day*time interactions ($p=.074$). As displayed in Table 7.4 in unadjusted models there was a significant main effect of time only and a trend towards a significant effect of probe day (Figure 7.5). Pairwise comparisons demonstrated a significant difference between pre- and post-ingestion EL sweet bias (mean difference = 10.14, $p<.001$), with pre-ingestion values higher than post-ingestion and a trend towards lower values at PD14 than PD1, with no difference across conditions reported. In the adjusted model sFFQ was included as a covariate, and a significant interaction of condition, probe day and time was demonstrated, displaying a reduction in EL sweet bias from pre- to post-ingestion, and from PD1 to PD14 across all conditions. However, in condition 550 at post-consumption on PD14, EL sweet bias represented a negative value and thereby a savoury preference, whilst conditions 199 and 647 remained as a sweet preference.

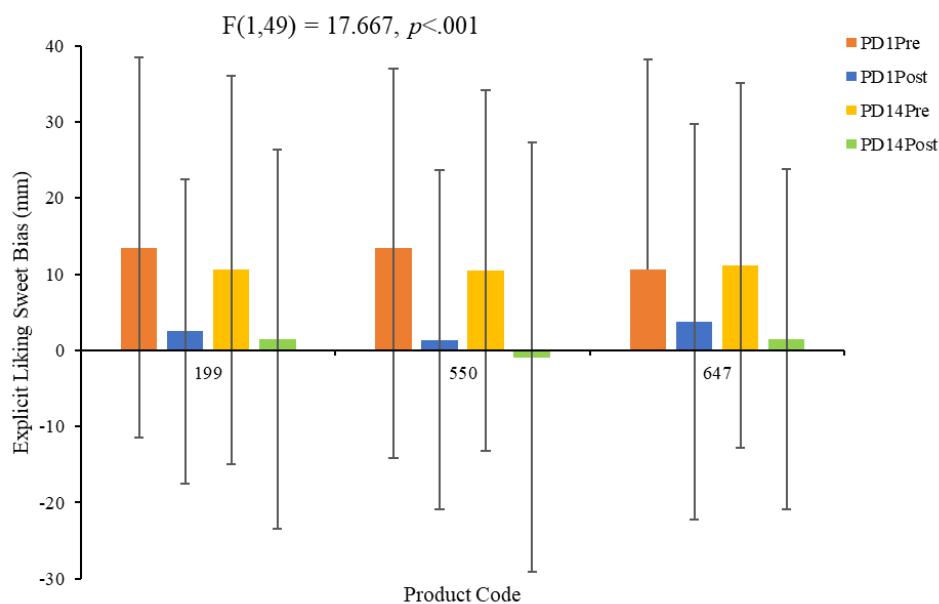


Figure 7.5. Changes in Leeds Food Preference Explicit Liking Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models. Error bars represent standard deviation.

7.3.2.2 Explicit Wanting Sweet Bias

Sphericity was violated for condition ($p=.012$) but was assumed for condition*probe day ($p=.203$), condition*time ($p=.482$) and condition*probe day*time interactions ($p=.236$). As displayed in Table 7.4 there was a significant main effect of time only (Figure 7.6). Pairwise comparisons demonstrated a significant difference between pre- and post-ingestion EW sweet bias (mean difference = 9.26, $p < .001$), with pre-ingestion values higher than post-ingestion. When including covariates into the model, gender displayed a trend towards a significant interaction with condition, and was a significant covariate of the effect of time, and condition*time interaction. In condition 550 at post-consumption EW sweet bias displayed a negative value, representing a savoury preference, whereas in conditions 199 and 647 it remained a positive value and therefore a sweet preference.

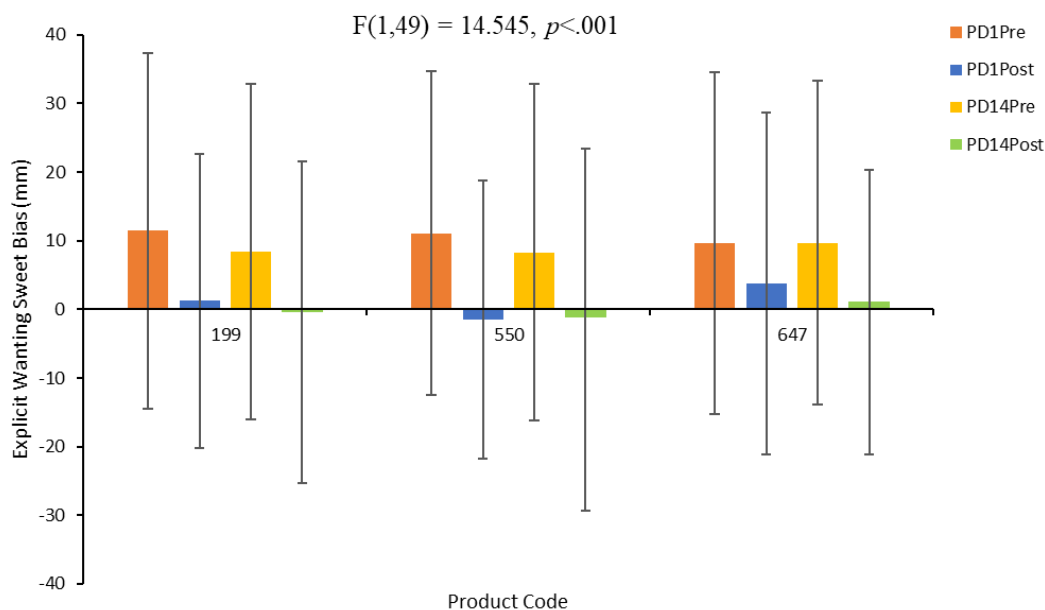


Figure 7.6. Changes in Leeds Food Preference Explicit Wanting Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models. Error bars represent standard deviation.

7.3.2.3 Implicit Wanting Sweet Bias

For IW sweet bias sphericity was violated for condition ($p = .002$) but was assumed for condition*probe day ($p = .269$), condition*time ($p = .921$) and condition*probe day*time interactions ($p = .849$). As shown in Table 7.4 there was a significant main effect of time only

(Figure 7.7). Pairwise comparisons demonstrated a significant difference between pre- and post-ingestion IW sweet bias (mean difference = 16.16, $p < .001$), with pre-ingestion values higher than post-ingestion. Adjusted models demonstrated no covariates of note.

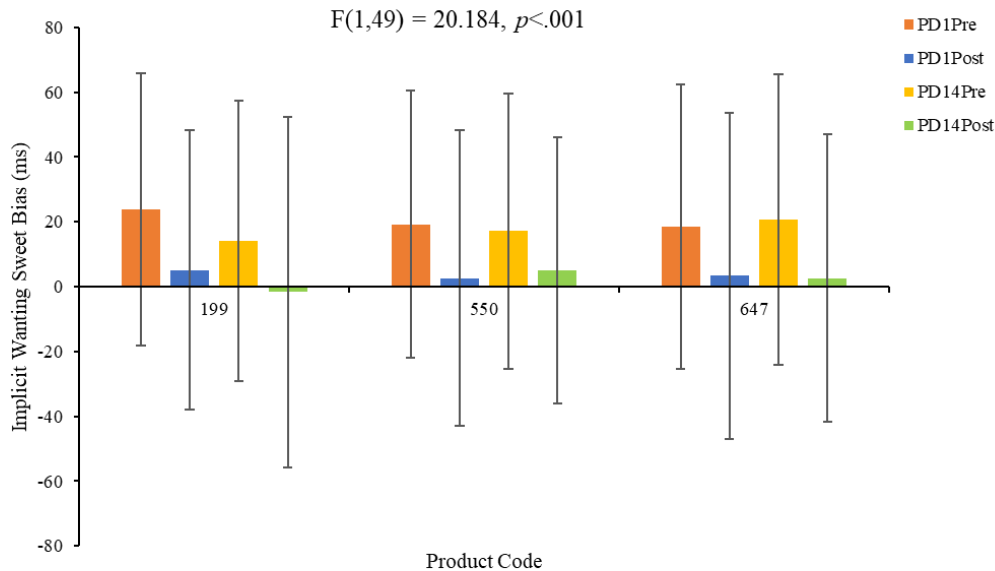


Figure 7.7. Changes in Leeds Food Preference Implicit Wanting Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models. Error bars represent standard deviation.

7.3.2.4 Choice Sweet Bias

Sphericity was violated for condition ($p = .005$) but was assumed for condition*probe day ($p = .579$), condition*time ($p = .128$) and condition*probe day*time interactions ($p = .115$). As shown in Table 7.4 there was a significant main effect of time only (Figure 7.8). Pairwise comparisons demonstrated a significant difference between pre- and post-ingestion choice sweet bias (mean difference = 5.88, $p < .001$), with pre-ingestion values higher than post-ingestion.

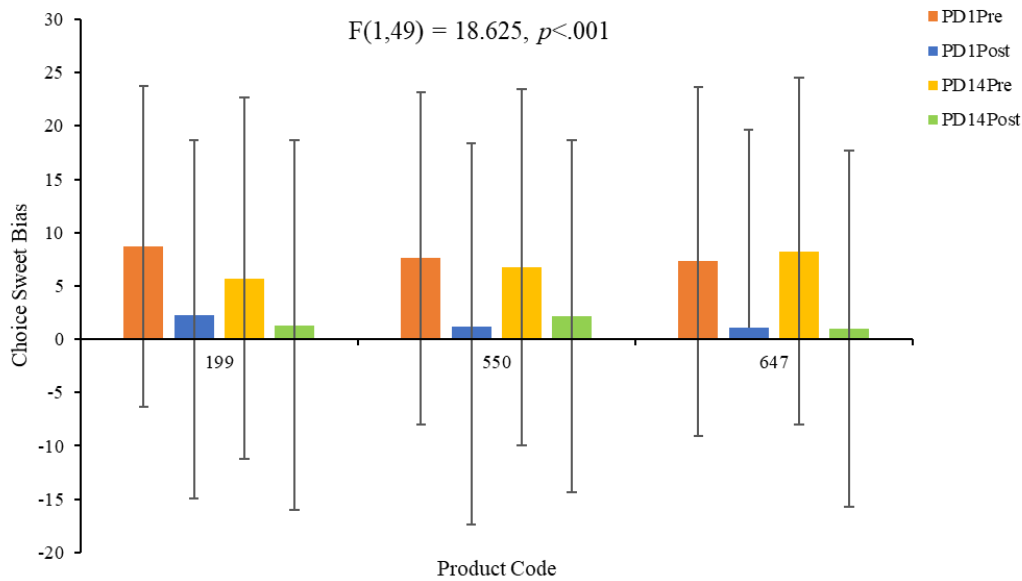


Figure 7.8. Changes in Leeds Food Preference Choice Sweet Bias outcomes from pre- to post-ingestion across probe days (PD) and condition in unadjusted models. Error bars represent standard deviation.

7.3.3 Secondary Outcomes

7.3.3.1 Subjective Appetite Sensations

A series of 3x2 repeated measures ANOVAs were conducted with condition (3 levels) and probe day (2 levels) as fixed-factors, to compare differences between conditions across PD1 and PD14 on iAUC subjective appetite sensations. Due to having only two levels, sphericity was assumed for probe day. Means and standard deviations can be seen in Table 7.5, whilst results for adjusted and unadjusted models can be seen in Table 7.6.

Table 7.5. Means and Standard Deviations of iAUC appetite ratings.

		647		199		550	
		PD1	PD14	PD1	PD14	PD1	PD14
iAUC Appetite for Sweet (mm)* ¹	Mean	-4648.13	-4391.81	-5090.28	-3615.36	-4185.44	-3332.71
	SD	4306.53	4364.97	4246.07	4841.74	4529.59	3681.99
iAUC Appetite for Savoury (mm)* ²	Mean	-1674.43	-1714.08	-1390.14	-879.36	-1686.64	-1425.14
	SD	3891.61	4065.26	3859.94	3594.83	4390.40	4297.69
iAUC Desire to Eat (mm)* ²	Mean	-4535.13	-4089.54	-4060.98	-3464.39	-4600.18	-3867.16
	SD	4164.33	4262.67	4585.16	4424.56	5104.90	3694.47
iAUC Hunger (mm)* ²	Mean	-4620.10	-4189.72	-4352.68	-4010.35	-4307.34	-3971.00
	SD	4721.06	4347.17	4254.43	5073.83	4671.98	3834.78

Abbreviations: PD – probe day. iAUC – incremental area under the curve. SD – standard deviation.

*¹n=49. *²n=50.

Table 7.6. Models of adjusted and unadjusted repeated measures ANOVA for iAUC subjective appetite ratings.

		Unadjusted		Adjusted	
		F	<i>p</i>	F	<i>p</i>
iAUC Appetite for Sweet (mm) ^{*1}	Cond	2.397	.104	4.576	.018^a
	PD	6.232	.016	6.360	.015^a
	Cond*PD	1.086	.342	.297	.743
iAUC Appetite for Savoury (mm) ^{*2}	Cond	1.324	.271	1.736	.182
	PD	.710	.403	5.285	.026^c
	Cond*PD	.335	.716	.132	.877
iAUC Desire to Eat (mm) ^{*2}	Cond	1.291	.280	5.497	.006^a
	PD	5.433	.024	2.758	.103
	Cond*PD	.066	.937	.007	.993
iAUC Hunger (mm) ^{*2}	Cond	.209	.786	.520	.575
	PD	1.519	.224	4.553	.038^d
	Cond*PD	.008	.992	.159	.853

^a iAUC appetite for sweet included age at screening as a covariate in the adjusted model.

^b iAUC appetite for savoury included site, gender and sFFQ as covariates in the adjusted model.

^c iAUC desire to eat included age at screening and sFFQ as covariates in the adjusted model.

^d iAUC hunger included gender as a covariate in the adjusted model.

Abbreviations: iAUC – incremental area under the curve. Cond – condition. PD – probe day.

^{*1}*n*=49. ^{*2}*n*=50.

For iAUC appetite for sweet, sphericity was violated for condition ($p=.028$) but was assumed for condition*probe day ($p=.533$). Tests of within-subjects effects demonstrated an effect of probe day only (Table 7.6 and Figure 7.9). Pairwise comparisons demonstrated a significant reduction in iAUC appetite for sweet from PD1 to PD14 (mean difference = -782.39, $p = .016$), in addition to a significant difference between conditions 647 and 550 (mean difference = -809.02, $p=.043$). The adjusted model demonstrated in addition to a main effect of probe day, a significant main effect of condition when including age at screening as a covariate (Table 7.6), in which condition 550 was lower to condition 647 (mean difference = -809.12, $p = .018$),

thereby highlighting the potential for age to influence the effect of sweetener condition (Figure 7.10).

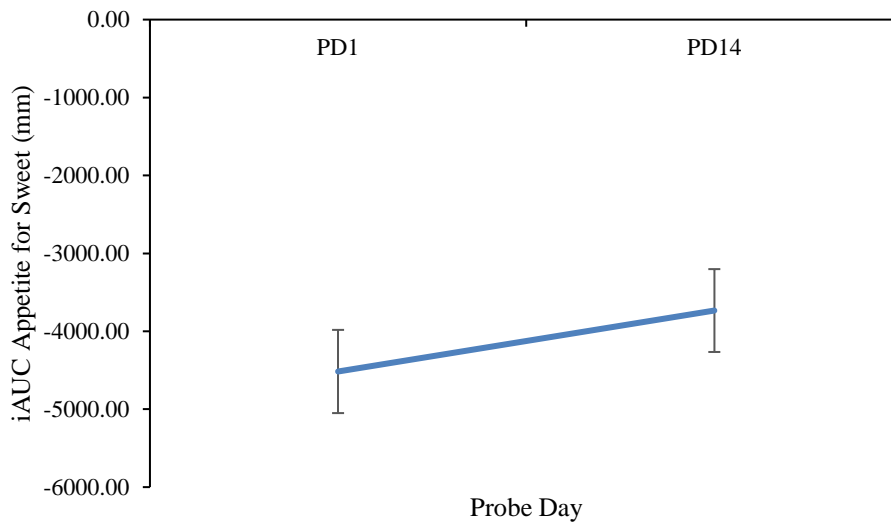


Figure 7.9. Unadjusted models iAUC Appetite for Sweet subjective VAS rating change from PD1 to PD14.

Error bars represent standard error of the mean.

Abbreviations: PD – probe day. iAUC – incremental area under the curve.

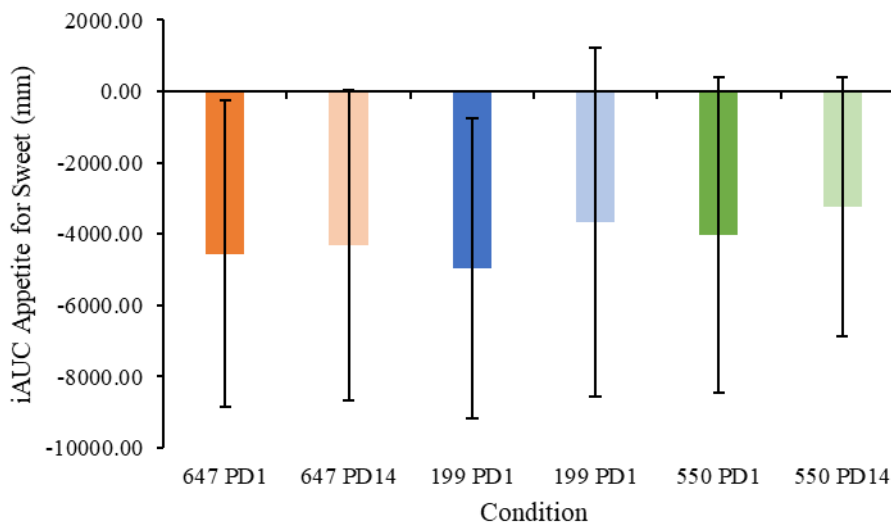


Figure 7.10. Adjusted model iAUC Appetite for Sweet subjective VAS rating change from PD1 to PD14 across conditions.

Error bars represent standard deviation.

Abbreviations: PD – probe day. iAUC – incremental area under the curve.

For iAUC appetite for savoury sphericity was assumed for condition ($p=.179$) and condition*probe day ($p=.084$). There was no effect of condition, probe day or condition*probe day interaction on iAUC appetite for savoury. Adjusted models included site, gender and sFFQ as significant covariates, with a significant effect of probe day emerging (Table 7.6). Pairwise

comparisons did not demonstrate a significant difference between PD1 and PD14 (mean difference = -244.21, $p=.339$).

For iAUC hunger sphericity was violated for condition ($p=.038$), but was assumed for condition*probe day ($p=.293$). There was no effect of condition, probe day or condition*probe day interaction on iAUC hunger. Adjusted models included gender as a significant covariate, with a significant effect of probe day emerging (Table 7.6). Pairwise comparisons did not demonstrate a difference between PD1 and PD14 (mean difference = -369.68, $p=.213$).

For iAUC desire to eat Mauchley's test of sphericity was assumed for condition ($p=.547$) and condition*probe day ($p=.875$). There was a significant effect of probe day only (Table 7.6 and Figure 7.11). Pairwise comparisons demonstrated a significant reduction in iAUC appetite for sweet from PD1 to PD14 (mean difference = -591.73, $p = .024$). Adjusted models included age at screening and habitual sweet food intake (sFFQ) as covariates, and demonstrated an effect of condition, in which condition 647 (mean = -4,312.34) and condition 550 (mean = -4,233.67) were greater than condition 199 (mean = -3,762.69), although pairwise comparisons did not demonstrate significant differences (Table 7.6 and Figure 7.12).

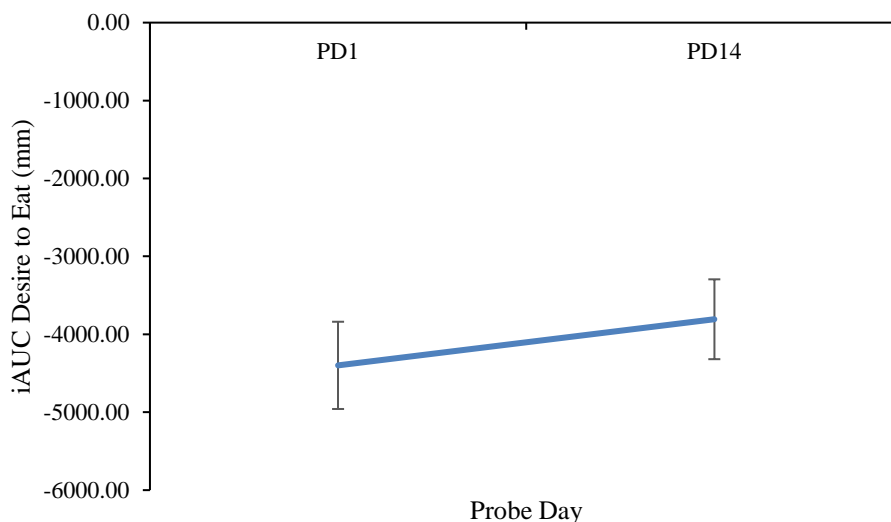


Figure 7.11. Unadjusted models iAUC Desire to Eat VAS rating change from PD1 to PD14. Error bars represent standard error of the mean. Abbreviations: PD – probe day. iAUC – incremental area under the curve.

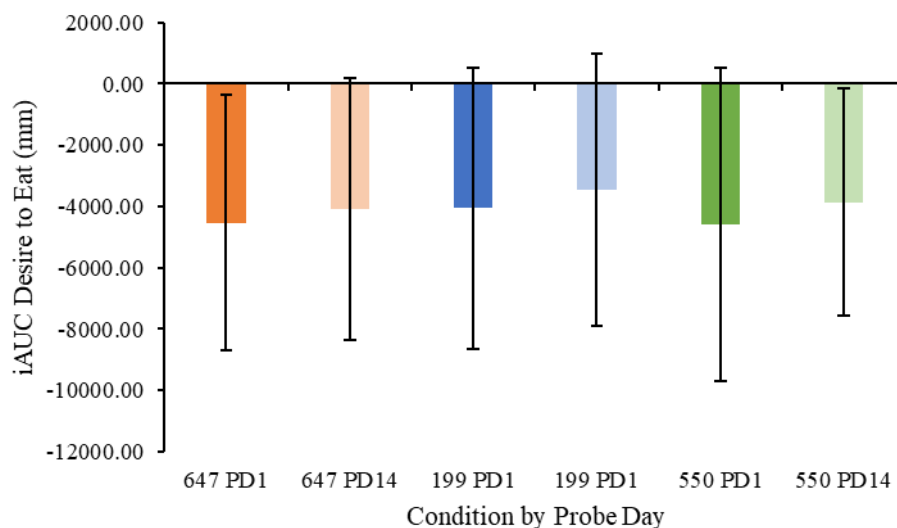


Figure 7.12. Adjusted model iAUC Desire to Eat subjective VAS rating change from PD1 to PD14 across conditions.

Error bars represent standard deviation.

Abbreviations: PD – probe day. iAUC – incremental area under the curve.

7.3.3.2 Control of Eating Questionnaire

A series of 3x2 repeated measures ANOVAs with condition (3 levels) and probe day (2 levels) as fixed-factors, were conducted to examine the effect of repeated exposure to the intervention foods on craving related outcomes. Means and standard deviations are displayed in

Table 7.7, whilst adjusted and unadjusted models can be seen in Table 7.8, with covariates on note reported. Sphericity was automatically assumed for probe day due to only having two levels.

Table 7.7. Means and standard deviations for Control of Eating Questionnaire outcomes ($n=50$).

		647		199		550	
		PD1	PD14	PD1	PD14	PD1	PD14
Craving Control	Mean	50.60	57.59	49.69	60.94	51.09	55.41
	SD	18.19	20.18	21.11	22.38	23.13	22.98
Sweet Craving	Mean	49.65	42.81	47.52	41.59	48.03	41.53
	SD	19.13	17.38	20.55	21.49	20.81	22.03
Savoury Craving	Mean	49.75	45.79	50.99	42.84	47.29	45.72
	SD	16.80	18.26	18.51	21.56	18.88	21.21
Positive Mood	Mean	65.69	66.00	63.50	66.30	64.27	66.75
	SD	17.12	16.22	18.68	19.11	17.79	19.30

Abbreviations: PD – probe day. iAUC – incremental area under the curve. SD – standard deviation

Table 7.8. Models of adjusted and unadjusted repeated measures ANOVA results for Control of Eating Questionnaire outcomes ($n=50$).

		Unadjusted		Adjusted	
		F	<i>p</i>	F	<i>p</i>
Craving Control	Cond	.384	.625	3.761	.038^a
	PD	20.163	< .001	.231	.633
	Cond*PD	2.685	.073	4.286	.016^a
Sweet Craving	Cond	.455	.636	1.730	.183
	PD	15.566	< .001	.899	.348
	Cond*PD	.042	.959	3.033	.053 ^a
Savoury Craving	Cond	.226	.798	2.274	.109
	PD	8.507	.005	5.991	.018^b
	Cond*PD	2.234	.112	3.227	.044
Positive Mood	Cond	.148	.863	-	-
	PD	1.747	.192	-	-
	Cond*PD	.521	.595	-	-

^a Craving control and sweet craving included sFFQ as a covariate in the adjusted model.

^b Savoury craving included sFFQ and gender as covariates in the adjusted model.

Positive mood did not include any covariates in the adjusted model.

Abbreviations: Cond – condition. PD – probe day.

For craving control, sphericity was not assumed for condition ($p<.001$) but was assumed for condition*probe day interaction ($p=.766$). Tests of within-subjects effects demonstrated a main effect of probe day only (

Table 7.7). Pairwise comparisons demonstrated a significant difference between PD1 and PD14 (mean difference = -7.52, $p<.001$), in which craving control improved at PD14 (Figure 7.13).

When controlling for covariates, a significant effect of condition emerged, although pairwise comparisons did not demonstrate a significant difference between conditions 647 and 199 (mean difference = -1.22, $p>.999$), conditions 647 and 550 (mean difference = .844, $p>.999$), or conditions 199 and 550 (mean difference = 2.066, $p=.601$). The adjusted model also demonstrated a significant interaction of condition*probe day, displayed in Figure 7.14.

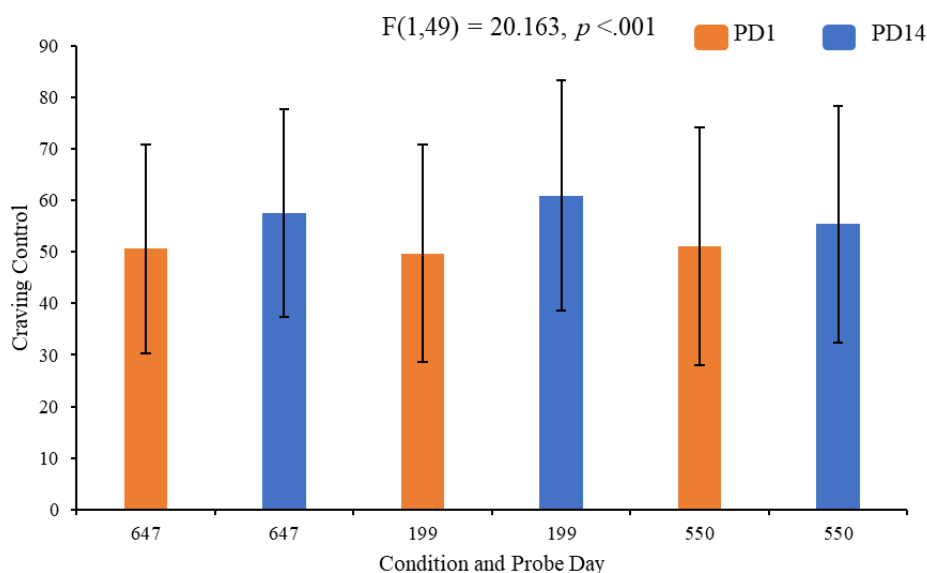


Figure 7.13. Control of Eating Questionnaire Craving Control as a function of condition and probe day.
Error bars represent standard deviation.
Abbreviations: PD – probe day.

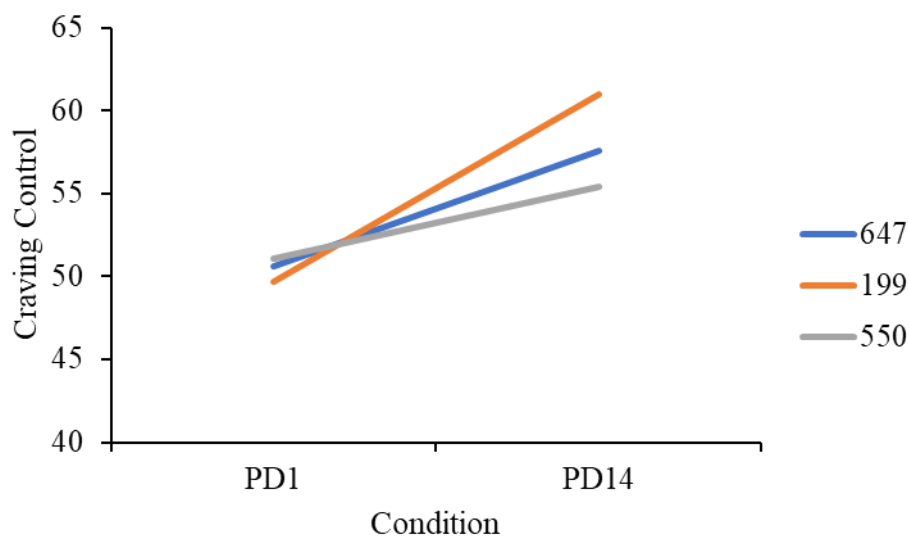


Figure 7.14. Control of Eating Questionnaire Craving Control adjusted model condition*probe day interaction effect.
Abbreviations: PD – probe day.

For sweet craving, sphericity was assumed for condition ($p=.137$) and condition*probe day interaction ($p=.326$). Tests of within-subjects effects demonstrated a significant main effect of probe day only (

Table 7.7). Pairwise comparisons demonstrated significant difference between PD1 and PD14 (mean difference = 6.43, $p<.001$), in which sweet craving decreased at PD14 (Figure 7.15).

Adjusted models included sFFQ as a covariate, with a trend emerging for an interaction effect of condition*probe day, and no effect of probe day remaining (

Table 7.7).

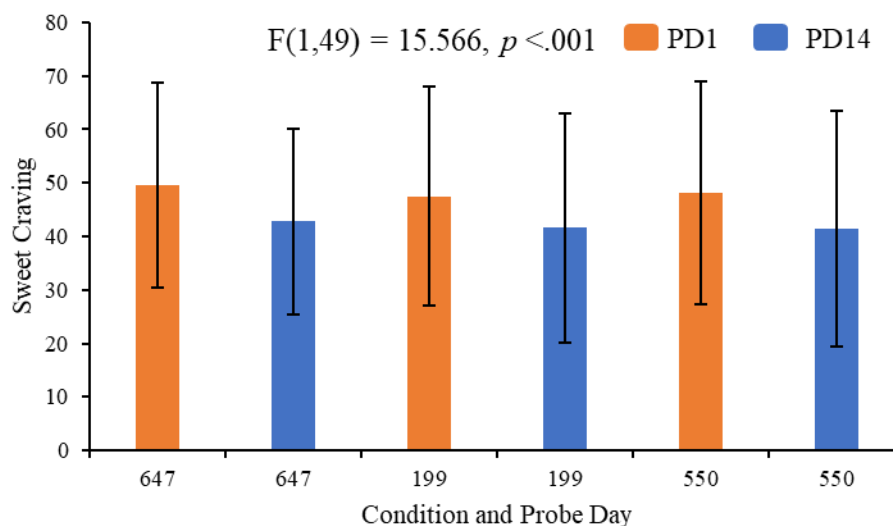


Figure 7.15. Control of Eating Questionnaire Sweet Craving as a function of condition and probe day.

Error bars represent standard deviation.

Abbreviations: PD – probe day.

For savoury craving, sphericity was assumed for condition ($p=.790$) and condition*probe day interaction ($p=.114$). Within-subjects effects demonstrated a significant main effect of probe day only (

Table 7.7). Pairwise comparisons demonstrated a significant difference between PD1 and PD14 (mean difference = 4.56, $p=.005$), in which craving control decreased at PD14 (Figure 7.16).

Adjusted models included sFFQ and gender as covariates, with the effect of probe day remaining significant (

Table 7.7).

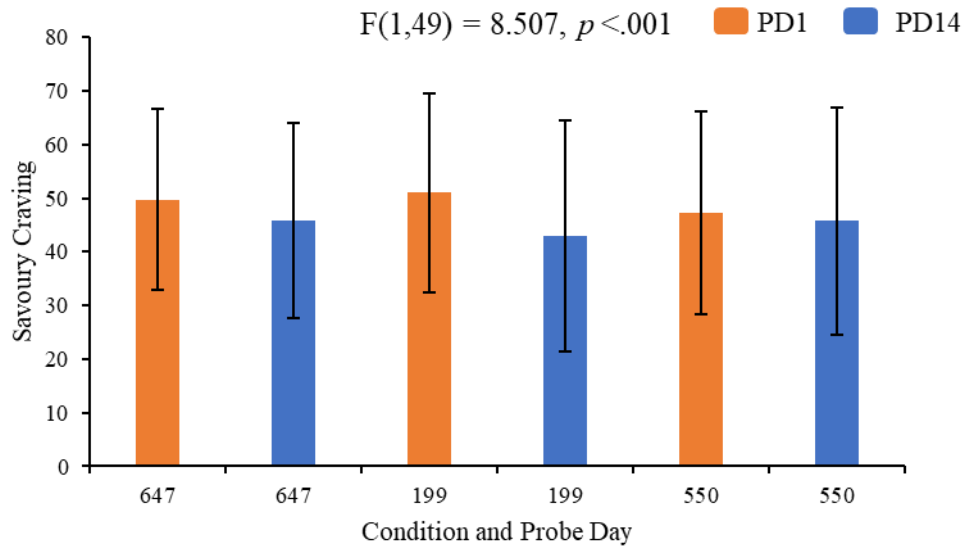


Figure 7.16. Control of Eating Questionnaire Savoury Craving as a function of condition and probe day.
Error bars represent standard deviation.
Abbreviations: PD – probe day.

For positive mood, sphericity was assumed for condition ($p=.876$) and condition*probe day interaction ($p=.475$). Tests of within-subjects effects did not report any significant effects on positive mood, nor did adjusted models highlight any significant covariates for inclusion in the model (

Table 7.7).

7.3.4 Exploratory Outcomes

7.3.4.1 Body weight and composition

A series of 3x2 repeated measures ANOVAs with condition (3 levels) and probe day (2 levels) as fixed-factors were conducted to compare body weight and composition outcomes across conditions and from PD1 to PD14. For probe day sphericity was automatically assumed due to only presenting with two levels. Means and standard deviations can be seen in Table 7.9, whilst results for unadjusted and adjusted models can be seen in Table 7.10 with covariates of note outlined.

Table 7.9. Means and standard deviations for body weight and composition outcomes ($n=50$).

647		199		550	
PD1	PD14	PD1	PD14	PD1	PD14

Body weight (kg)	Mean	82.19	82.14	82.64	82.78	82.46	82.47
	SD	10.64	10.71	10.36	10.75	10.82	10.93
Fat mass (kg)	Mean	29.29	29.52	29.46	29.71	29.43	28.85
	SD	7.83	7.85	7.67	7.72	7.66	8.09
Fat-free mass (kg)	Mean	52.89	52.62	53.00	52.67	52.94	53.52
	SD	10.23	10.38	9.95	10.51	9.97	10.59
Body fat percentage (%)	Mean	35.70	36.01	35.78	35.99	35.78	35.16
	SD	8.52	8.55	8.31	8.46	8.18	8.78

Abbreviations: PD – probe day. SD – standard deviation. Kg – kilogram.

Table 7.10. Models of adjusted and unadjusted repeated measures ANOVA results for body weight and composition outcomes (n = 48).

		Unadjusted		Adjusted ^a	
		F	<i>p</i>	F	<i>p</i>
Body weight (kg)	Cond	1.076	.337	.206	.776
	PD	2.513	.120	9.198	.004^a
	Cond*PD	1.455	.239	1.035	.359
Fat mass (kg)	Cond	.692	.506	-	-
	PD	.289	.594	-	-
	Cond*PD	1.002	.363	-	-
Fat-free mass (kg)	Cond	1.308	.273	5.436	.006^a
	PD	.280	.599	4.863	.032^a
	Cond*PD	.685	.482	13.373	< .001^a
Body fat (%)	Cond	1.088	.341	1.505	.227
	PD	.729	.398	.013	.909
	Cond*PD	.942	.384	4.225	.021^a

Abbreviations: Cond – condition. PD – probe day. SD – standard deviations. Kg – kilograms.

^a Body weight, fat-free mass and body fat percentage included sFFQ as a covariate in the adjusted model.

For body weight, sphericity was violated for condition ($p=.011$) but was assumed for condition*probe day interaction ($p=.543$). Unadjusted models demonstrated no effect of condition, probe day, an interaction of condition*probe day. Adjusted models included sFFQ as a covariate and a significant effect of probe day emerged (Table 7.9), although pairwise comparisons demonstrated no difference between PD1 and PD14 (mean difference=-0.134) suggesting a lack of sufficient power to detect significant differences in post-hoc tests.

For fat mass, sphericity was assumed for condition ($p=.420$) but was violated for condition*probe day ($p=.035$). Tests of within-subjects effects demonstrated no effect of condition or of probe day and no interaction of condition*probe day. When testing for covariates there was no effect of any covariate and as such only unadjusted models are reported (Table 7.10).

For fat-free mass, sphericity was violated for condition ($p=.010$) and condition*probe day ($p=.006$). Unadjusted models demonstrated no effect of condition, probe day or interaction of condition*probe day. Adjusted models included sFFQ as a covariate, with a significant effect of condition, probe day and condition*probe day emerging (Table 7.10). Adjusted means did not display a difference between condition 199 and 550 (mean difference =-.003, $p>.999$), 199 and 647 (mean difference =-.311, $p=.380$), nor 550 and 647 (mean difference =-.308, $p=.678$). Pairwise comparisons did not display a difference between PD1 and PD14 (mean difference =-0.110, $p=.582$).

For body fat percentage, sphericity was assumed for condition ($p=.210$) but was violated for condition*probe day ($p=.045$). Unadjusted models demonstrated no effect of condition or probe day, and no interaction of condition*probe day. Adjusted models included sFFQ as a covariate, with an interaction of condition*probe day emerging (Table 7.10).

7.3.4.2 24hr Dietary Recall

A series of 3x2 repeated measures ANOVAs were conducted to compare differences in energy and macronutrient intake with conditions (3 levels) and of probe day (2 levels) included as fixed-factors to compare differences across conditions from PD1 to PD14. Sphericity was

assumed for probe day due to being two levels. Means and standard deviation of dietary intake outcomes can be seen in Table 7.11, whilst unadjusted and adjusted models can be seen in Table 7.12 with covariates of note defined.

Table 7.11. Means and standard deviations for 24hr dietary recall outcomes.

		647		199		550	
		PD1	PD14	PD1	PD14	PD1	PD14
Total energy intake (kcal)	Mean	2164.86	2109.98	1990.60	2073.12	2160.80	2139.22
	SD	712.20	624.10	678.74	606.74	802.59	670.15
CHO (g)	Mean	256.06	241.84	239.40	253.96	252.80	251.76
	SD	99.36	77.70	95.62	91.37	110.05	97.87
Sugar (g)	Mean	96.54	89.57	95.68	103.44	100.66	97.00
	SD	51.46	42.87	53.15	61.44	72.94	58.95
Fat (g)	Mean	83.74	82.65	76.56	76.42	85.36	84.22
	SD	34.64	31.42	35.23	29.39	38.83	31.72
PRO (g)	Mean	88.10	92.12	81.80	85.36	85.50	89.22
	SD	32.55	44.87	33.89	30.32	32.73	36.97
Fibre (g)	Mean	19.68	18365	17.82	18.50	19.24	19.16
	SD	7.94	8.46	9.11	8.29	8.90	6.38

Abbreviations: PD – probe day. SD – standard deviation. CHO – carbohydrates. PRO – protein. g – grams.

Table 7.12. Models of adjusted and unadjusted repeated measures ANOVA results for 24hr dietary recall outcomes ($n=48$).

		Unadjusted		Adjusted	
		F	p	F	p

Total energy intake (kcal)	Cond	1.578	.212	-	-
	PD	.032	.859	-	-
	Cond*PD	.870	.422	-	-
CHO (g)	Cond	.226	.798	-	-
	PD	.042	.839	-	-
	Cond*PD	1.714	.186	-	-
Sugar (g)	Cond	.873	.412	.639	.530
	PD	.192	.664	6.931	.012^a
	Cond*PD	1.200	.306	.090	.914
Fat (g)	Cond	2.694	.073	-	-
	PD	.089	.767	-	-
	Cond*PD	.110	.896	-	-
PRO (g)	Cond	1.477	.234	-	-
	PD	.789	.379	-	-
	Cond*PD	.003	.997	-	-
Fibre (g)	Cond	.525	.593	-	-
	PD	.259	.613	-	-
	Cond*PD	.608	.547	-	-

Abbreviations: Cond – condition. PD – probe day. CHO – carbohydrates. PRO – protein. Kcal – kilocalories. g – grams.

^a Sugar (g) included BMI as a covariate in the adjusted model. Total energy intake (kcal), CHO (g), fat (g), PRO (g) and Fibre (g) did not include any significant covariates.

For total energy intake (kcal), sphericity was assumed for condition ($p=.868$) and condition*probe day interaction ($p=.126$). Tests of within-subjects effects demonstrated no effect of condition or of probe day and no interaction of condition*probe day. When including covariates there were no effects of any covariates, as such only unadjusted models are reported

(

Table 7.11).

For carbohydrate intake, sphericity was assumed for condition ($p=.808$) and condition*probe day ($p=.379$). Within-subjects effects demonstrated no effect of condition or of probe day and interaction of condition*probe day. When including covariates there were no effects of any covariates, as such only unadjusted models are reported (

Table 7.11).

For sugar intake, sphericity was assumed for condition ($p=.319$) and condition*probe day interaction ($p=.187$). Tests of within-subjects effects demonstrated no effect of condition or of probe day and no interaction of condition*probe day. When controlling for covariates there was a significant effect of BMI on the effect of probe day (

Table 7.11), in which PD1 was greater (mean = 98.98) than PD14 (mean = 97.05), although pairwise comparisons did not demonstrate a significant difference (mean difference = 1.93, $p=.644$).

For fat intake, sphericity was assumed for condition ($p=.320$) and condition*probe day ($p=.259$). Tests of within-subjects effects demonstrated no effect of condition or probe day as well as no interaction of condition*probe day. When including covariates there were no effects of any covariates, as such only unadjusted models are reported (

Table 7.11).

For protein intake, sphericity was assumed for condition ($p=.130$) and condition*probe day interaction ($p=.210$). Tests of within-subjects effects demonstrated no effect of condition nor of probe day, neither was there an interaction effect of condition*probe day. When including covariates there were no effects of any covariates, as such only unadjusted models are reported (

Table 7.11).

For fibre intake, sphericity was assumed for condition ($p=.106$) and condition*probe day interaction ($p=.554$). Tests of within-subjects effects demonstrated no effect of condition or of probe day and interaction of condition*probe day. When including covariates there were no effects of any covariates, as such only unadjusted models are reported (

Table 7.11).

7.4 Discussion

The aim of the present chapter was to provide an examination of the acute and repeated exposure effects of three different sweeteners in a solid food matrix on subsequent sweet food preferences and eating behaviours. All sweet bias outcomes demonstrated a reduction from pre- to post-ingestion, thereby supporting the first hypothesis regarding the acute ingestion. There was also a reduction in iAUC appetite for sweet VAS ratings, cravings for sweet and cravings for savoury, and improvement in overall craving control following repeated ingestion, supporting the second hypothesis. Findings showed no effect of condition on overall sweet food preferences or subjective appetite ratings and cravings in adjusted models, leading to the conclusion that the three different sweetener types do not impact subsequent sweet food preferences differently. There was also no impact of probe day, indicating that repeated consumption of a sweet food daily for 14 days did not impact subsequent sweet food preferences, thereby rejecting the third and fourth hypotheses. There was no effect of condition or probe day on body weight or composition, or 24hr dietary intake in unadjusted models, although adjusted models highlighted the influence of habitual sweet food intake (sFFQ) in influencing this.

7.4.1 Sweet Food Preferences – High-Intensity Sweeteners versus Sugar

Sweet food preferences declined immediately following acute exposure in line with sensory specific satiety (Griffioen-Roose et al., 2010; Hetherington, 1996), however this effect did not differ across sweetener types. Neuroimaging techniques highlight the increased reward value of sucrose sweetened products relative to HIS sweetened products (Smeets et al., 2011), however, the current analysis highlights that this does not translate into an implicit effect on subsequent sweet food preferences. This is in line with previous evidence which has illustrated sweetener type is of less importance than general consumption of sweet food and drinks when influencing subsequent preferences (Mahar & Duizer, 2007). Additionally, it should be noted that a so-called ‘transfer effect’ of preference from savoury to sweet tasting foods has been demonstrated in the context of sensory specific satiety (Griffioen-Roose et al., 2010). This transfer effect is less pronounced from sweet to savoury – which the current findings support as on the whole,

post-ingestion preferences represented an overall sweet bias rather than savoury bias. In this manner, sweet food preference was diminished but not entirely eliminated.

Previous evidence has suggested that motivational aspects of food preferences (i.e., wanting) are increased following acute consumption of HIS. In one such study, when using a computer-based task HIS beverages increased motivation to gain access to sweet snacks relative to savoury snack foods later in the day in (Casperson et al., 2017). The discrepancy with the current findings in the present chapter which demonstrate a reduction in wanting following acute consumption may be due to the timeline of assessment employed, with the current study assessing sweet food preferences 20 minutes post-ingestion, whereas Casperson's findings demonstrate increased motivation 4hrs post-ingestion. Therefore, the present protocol may not accurately capture any increases in motivation towards sweet foods due to the time of assessment. Moreover, Casperson utilised healthy weight adults, whereas the current study employed individuals with overweight and obesity, with previous evidence highlighting differences in motivational aspects of food reward in the obese state (Stoeckel et al., 2008). Taken together these findings may indicate a delayed increase towards motivation for sweet foods, as well as potential differences in participants varying by BMI, however future studies are required to elucidate this further.

The lack of a significant effect of probe day on subsequent sweet food preferences in the current study is in line with the limited available literature. For example, repeated consumption of HIS sweetened beverages or SSB for 12-months did not result in any change in preferred sweetness concentration (Ebbeling et al., 2020). It has elsewhere been shown that in individuals regularly consuming sweetened beverages there was no effect of sweetener type (Mahar & Duizer, 2007). Current findings support this and demonstrate that over 14 days of repeated daily exposure there is also no impact on subsequent sweet preferences.

Furthermore, habitual sweet food and drink consumption was highlighted as a covariate in the model in regards to condition. Notably, in adjusted models condition 550 demonstrated a greater reduction in liking from pre- to post-consumption, as well as from PD1 to PD14. This

finding is in contrast to previous evidence demonstrating that individuals regularly consuming sweetened beverages report a greater liking for increasing sweetness concentrations, which is not affected by sweetener type (Mahar & Duizer, 2007). The discrepancy here may be attributable to differences in experimental designs, with the current study finding weak effects of condition in an intervention which has required participants to be exposed daily for 14 days to the intervention foods before assessing food preferences, whereas the Mahar study consisted of a single sensory testing session. Moreover, differences between conditions were marginal, therefore firm conclusions cannot be drawn regarding the interaction between habitual sweet food consumption and effects of different sweetener reformulations. Nonetheless, habitual sweet consumption has been highlighted as a potentially importance variable, possibly capable of influencing the effect of different sweetener types on subsequent sweet preferences and it is recommended that future studies examine this in greater detail.

7.4.2 Food Cravings, Appetite for Sweet/Savoury and Subsequent Intake

One concern surrounding the use of HIS consumption in consumers is whether their ingestion results in an increase in cravings. It has been stated that it is precisely because HIS are sweet that sugar cravings are encouraged following their consumption (Mooradian et al., 2017; Yang, 2010). However, the current findings demonstrating a lack of effect of condition on subjective appetite ratings at PD1 demonstrates that specific appetite for sweet and savoury foods does not differ by sweetener type, nor does hunger or desire to eat, following a single exposure event.

Moreover, repeated exposure is suggested to increase appetite specifically for sweet foods (Malek et al., 2018). One such study demonstrated that consumption of sucralose relative to sucrose resulted in a higher motivation to gain access to sweet snacks relative to savoury foods (Casperson et al., 2017). However, the current findings demonstrate a reduction in cravings for sweet and savoury and increase in craving control from repeat exposure to both sucrose and HIS sweetened products. Moreover, with a lack of a significant effect between conditions, this also indicates that this reduction is not the result of the sweetener itself. It is instead most likely as a result of sweet cravings being satisfied via daily consumption of a sweet food. Cravings are satisfied by through consumption (Van Kleef et al., 2013) and specific cravings can be

satisfied by a substitute that serves the same basic goal (Huh et al., 2016), therefore it is concluded that repeated exposure to HIS sweetened foods does not promote an increase in sweet cravings, but may instead assist in reducing them (in line with findings outlined in Chapter 2).

Interestingly, dietary variety within food groups is an important predictor of body fatness (McCrary et al., 2000). There also exists a specific association between dietary variety in sweets, snacks and carbohydrates with body fatness suggesting that dietary variety in a food group assists in contributing to excess energy intake (McCrary et al., 1999). Conversely, limiting diet variety in a food group is associated with a reduction in the consumption of that food group (Raynor, 2012). It has been hypothesised elsewhere that this is a consequence of dietary monotony producing a decrease in hedonic ratings of foods (Raynor & Epstein, 2001). Within the current study which required participants to consume a portion of the same food each day for 14 days there was a reduction in iAUC appetite for sweet and desire to eat subjective ratings following repeated exposure. Although there was not a significant effect of probe day on sweet food preferences, the differences in subjective ratings suggests that a different element of sweet food preferences, may be impacted from repeated consumption of the same food.

The current findings are in line with the wider literature which tend to demonstrate that HIS sweetened foods do not increase sweet food preferences or energy intake (Wilk et al., 2022), and expand upon this by demonstrating that the effects do not differ when compared to a sucrose sweetened product. In the present study there was no effect of repeated consumption on 24-hr dietary energy or macronutrient intake, although these findings should be interpreted with caution, due to limitations in under-reporting of free-living measures of habitual intake (Poppitt et al., 1998). Assessment of dietary recall suffers from a reliance on the accuracy of a participant's memory and a willingness to accurately report all intake (Johnson et al., 2005; Krall et al., 1988). Similarly, the act of reporting food intake has been previously demonstrated to inadvertently reduce food intake because of increased self-monitoring (Goris et al., 2000). However it has been stated elsewhere that allowing participants to perform their habitual

behaviours in a familiar and comfortable environment ensures that food diaries and dietary records as a measurement of habitual intake can be regarded as highly reliable (Albar et al., 2016). Moreover, the 24hr period in which participants reported their dietary intake represented an atypical day for participants and so may not be a true representation of free-living eating behaviours. Nonetheless, these findings are in line with those concluded in Chapter 2 of the current thesis as well as a narrative review (O'Connor et al., 2021), in which it was concluded that the inclusion of HIS in the diet does not impact subsequent eating behaviours. In Chapter 2 it was highlighted that substitution of HIS for sugar in the diet may facilitate a reduction in sugars and energy intake in individuals with an elevated BMI. However this conclusion was drawn from a study which employed a beverage as the mode of administration. The current study utilised a solid food matrix with a minor difference in total energy between conditions (~30kcal), therefore the inclusion of HIS in the diet appears to not be due to toxicology of the sweetener, but the propensity to provide an energy reduced alternative. In instances where a HIS sweetened product is unable to provide a significant reduction of the product's energy density, it is unlikely that there will be any alteration to dietary intake in participants with overweight and obesity.

7.4.3 Body Weight and Composition and 24hr Dietary Recall

Findings demonstrated no effect of any sweetener condition on body weight or body composition outcomes, or 24hr dietary recall outcomes. This finding is not unanticipated, the intervention product was included in the diet through use of a substitution strategy in which a similar food at a similar energy density was replaced with the intervention products. Also, a 12 day intervention period was used which is unlikely to be long enough to elicit difference in body weight or composition. This finding is supported by the finding that demonstrated that there was no impact of the intervention on subsequent energy intake. In studies which demonstrate a reduction in body weight through HIS inclusion in the diet, it is also highlighted that in individuals already engaged in an energy deficit-inducing diet there is no further benefit to weight loss from the inclusion of HIS (Laviada-Molina et al., 2020). Studies which compare HIS consumption with water tend to report no effect of body weight outcomes in contrast with

those comparing to a sucrose control conditions, leading authors to conclude that the effects of HIS in the diet are most pronounced when they are used as a sugar substitute that actively produces a reduction in energy intake (Toews et al., 2019; Wilk et al., 2022). Similar findings through use of rodent models have concluded that the inclusion of neotame within the diet results in reductions to body weight as a consequence of a reduced food consumption, not toxicity of neotame (Mayhew et al., 2003). As the current intervention products were comparable in their energy density, their ingestion is therefore unlikely to produce a reduction in energy intake and subsequent body weight.

Moreover, in adjusted models habitual sweet consumption (sFFQ) was identified as an influential variable, producing an effect of probe day on body weight, as well as effects of condition, probe day and a condition*probe day interaction on fat-free mass. However, this is likely a type-I error due to multiple comparisons, as pairwise comparisons did not demonstrate significant effects after using Bonferroni corrections. Post-hoc comparisons demonstrated marginal differences that were not significant and therefore of limited practical importance in the current analysis.

7.4.4 Conclusions

It can be concluded that there was no difference between two forms of HIS and a sucrose control when included in a solid food matrix (biscuit) on subsequent sweet food preferences following acute or repeated exposure. Repeated exposure demonstrated an improvement in overall craving control and a reduction in both subjective ratings of appetite for sweet, desire to eat and cravings for both sweet and savoury, with no difference between sweetener type. Additionally, the inclusion of HIS in a solid food matrix is not sufficient to produce an alteration to energy or macronutrient intake, nor body weight or composition, instead reformulated products may provide a means of maintaining consumer palatability whilst implementing alternative energy reduction strategies. HIS therefore may not provide passive energy reduction when included in a solid food matrix, but appear unlikely to impact subsequent sweet food preferences or encourage sweet food intake. However, the influence of habitual sweet food intake was identified as a potentially important variable in determining the

effects of condition, which warrants examination in future to a greater degree and will be the subject of the proceeding chapter. Nonetheless, the evidence outlined leads to the conclusion that HIS sweetened products may be included in the diet to provide consumer desired palatability in conjunction with other calorie control methods to aid in achieving successful weight loss in individuals with overweight and obesity.

8 An Exploration into Sweet Phenotype Differences in the Effects of Sucrose and High-Intensity Sweeteners on Subsequent Sweet Food Preferences

Aims: Sweet liking varies amongst individuals, these individual differences may in turn influence sweet food preferences and subsequent intake. Within the previous chapter it was demonstrated that habitual sweet food intake was a covariate influencing the effect of HIS or sucrose condition (i.e., sweetener type) on subsequent sweet preferences. Within the wider literature, sweet liker phenotype is also demonstrated to be associated with habitual intake of sweet foods, therefore by extension, sweet liker phenotype may impact the effect of HIS or sucrose condition on sweet food preferences via this mechanism. Moreover, current evidence surrounding the effects of acute and repeated exposure to HIS relative to sucrose sweetened products investigating effects of sweet liker phenotype groups is lacking. Therefore, the aim of the current chapter will address the sixth objective in section 1.8.1, which is to explore whether sweet liker phenotype group (as measured by the LFPQ explicit liking sweet bias) is associated with differing effects of HIS or sucrose consumption on subsequent sweet food preferences.

Introduction

Sweet liker phenotype describes the differences between sweet likers and sweet dislikers (referred to as high and low sweet likers in the current thesis and operationalised through the explicit liking sweet bias score from the LFPQ) outlining the different hedonic responses to sweet stimuli (Armitage et al., 2021; Iatridi et al., 2020). Currently it remains unknown what specifically causes differences in sweet-liker phenotype (Hwang et al., 2015), however, individuals with increased sensitivity to the bitter tastant 6-n-propylthiouracil (PROP) are reported to perceive greater sweetness intensity from solutions (Looy & Weingarten, 1992). This has been exhibited for both HISs such as saccharin (Bartoshuk, 1979) as well as sucrose (Gent & Bartoshuk, 1983).

Despite a number of HIS producing a bitter or metallic aftertaste (Muenprasitivej et al., 2022), both stevia Reb M and neotame (used within the current study) do not produce this effect.

Neotame produces an intense sweet aftertaste (Cardoso et al., 2008; Rocha & Bolini, 2015) whereas stevia Reb M possesses a taste profile that is defined as creamy and closer to that of sucrose than other HIS (Prakash et al., 2014). However, wide variability in the patterns of intensity ratings between participants for a variety of sweetener types (Schiffman et al., 1979) indicates that various sweeteners may be experienced differently by individuals, which has been reiterated by more recent evidence also (Kamerud & Delwiche, 2007). As taste perception and liking are two connected elements of food preferences (Jayasinghe et al., 2017) it remains possible, but as yet unclear whether there will be differences between high and low sweet likers in the effects of different sweetener types.

There is only limited evidence investigating differences between sweet phenotype groups in the context of sweetener types. One study demonstrated no difference in liking ratings for sucrose and HIS (aspartame and acesulfame-K) sweetened ice tea in a pooled sample, before demonstrating significant differences in desired sweetness concentrations between sweet liker phenotype groups (Yang et al., 2019). However, the study failed to compare differences in liking ratings between phenotype groups for the two forms of sweetener used. Therefore although phenotype impacts desired sweetness preference, it has not been examined whether there is an interaction between sweet liker phenotype and the effects of different sweetener types, which will be addressed within the present chapter.

Moreover, within the previous chapter it was demonstrated that there was no effect of condition (sweetener type) on subsequent sweet food preferences. However, adjusted models demonstrated that habitual consumption of sweet foods may be influential in determining the impact of different sweetener types on sweet food preferences (see Chapter 7 – sFFQ adjusted models increased the effect of condition). Sweet liker phenotype has within the wider literature been demonstrated to be positively associated with dietary intake (Garneau et al., 2018; Holt et al., 2000; Iatridi et al., 2020), with liking for sweet positively associated with an increased intake of sweet foods (Jayasinghe et al., 2017). Therefore by extension, it may be anticipated that sweet liker phenotype is able to impact the effect of condition (sweetener type) on sweet food preferences via this mechanism and the potential for this to occur warrants further

investigation using sweet phenotype as a covariate in ANOVA models. The following chapter represents an extension of the previous SWEET chapter (Chapter 7) by provision of an examination of the potential effects sweet liker phenotype on reported models. The current analysis, due to the limited available previous evidence, remains exploratory and as such no formal hypotheses are defined.

8.2 Methods

The overarching methodology for the current chapter was identical to that of Chapter 7, for specific details surrounding participant recruitment and protocol procedures please see Chapter 3, section 3.3 and Chapter 7, section 7.2.

8.2.1 Sweet liker phenotype

Participants were grouped on the basis of sweet liker phenotype using the same method as employed previously in the thesis (see Chapter 5). Pre-ingestion explicit liking sweet bias score at CID1 (which was balanced across conditions) was selected as a baseline measure. This was recorded prior to exposure to the intervention product, thereby eliminating any effects of condition on sweet liker phenotypes. Although as shown previously in the current thesis, sweet preferences are a highly stable trait and so no differences between CID1, 3 and 5 pre-ingestion values were anticipated. Due to the manner in which sweet bias is calculated within the LFPQ, sweet bias outcomes represent a sweet relative to savoury preference, therefore any negative sweet bias values represent a savoury preference. As such, participants were coded as a high sweet-liker if their EL sweet bias was >0 , and a low sweet liker if it was <0 at CID1.

Participants were described as high sweet likers and low sweet likers in line with the relative nature of the metric and evidence outlining that the sweet taste is universally rewarding (Drewnowski, 1997; Reed et al., 2006; Yang et al., 2019). Indeed, despite there being previous evidence highlighting variability in the expression of preferred sweet foods (Lampuré et al., 2016, 2019), it remains that sweet foods possess a powerful hedonic potential in all people (Blundell, 2018; Blundell, 1991).

8.2.2 Data Analysis

As with the previous chapter, at the time of writing, the intervention conditions have not been unblinded within the wider SWEET trial. As such, conditions will be referred to as their codes – 199, 550 and 647, which represent randomly generated 3-digit sequences. Primary outcomes consist of sweet food preferences as assessed via the LFPQ, secondary outcomes were eating

behaviour traits (TFEQ, BES and CoEQ), whilst body weight and composition and 24hr dietary recall (energy, protein, fat, carbohydrate and sugar intake) remained exploratory outcomes.

Participant descriptive statistics were explored as a function of sweet liker phenotype group and independent groups t-tests were conducted to compare differences at baseline (i.e., CID1).

Differences in sweet food preferences across the intervention were examined through 3x2x2 mixed ANOVA (condition, 3 levels, pre/post-consumption, 2 levels and PD, 2 levels) using sweet liker phenotype as a between-subjects factor. Secondly, a series of 3x2 repeated measures ANOVAs were conducted (conditions, 3 levels, PD, 2 levels) including sweet liker phenotype as a between-subjects factor to examine differences in the intervention effects on iAUC appetite sensations, CoEQ outcomes, body weight and composition as well as dietary recall outcomes. Mauchley's test of sphericity was examined for ANOVAs, with Greenhouse-Geisser corrections being applied where necessary. Significant differences were investigated via Bonferroni post-hoc analysis, with the significant values set at $p \leq 0.05$.

Potential covariates to be included were variables which have been demonstrated within the wider literature as having a capacity to influence sweet food preferences (BMI, age and gender), as well as data collection site. Unadjusted and adjusted models were reported, with only covariates of note being included in the final adjusted models (i.e. those that were significant in the model). In the event that there were no significant covariates, only unadjusted models were reported. For this reason, both unadjusted and adjusted (with significant covariates) models were reported.

8.3 Results

Using the same method of defining sweet liker phenotype groups as that outlined in Chapter 5, in the present data set high sweet likers accounted for 36 participants and low sweet likers accounted for 14 participants. Descriptive statistics for both groups can be seen in Table 8.1 as well as independent groups t-test results, which demonstrate no significant differences between groups regarding age, EAT-26 or sFFQ results at baseline. Similarly, participants did not differ in their CoEQ scores or BMI and body composition values at their first visit to the lab, prior to exposure to the intervention product (i.e., baseline).

Table 8.1. Descriptive statistics of high and low sweet liker phenotype groups, with independent groups t-test values ($n=50$).

Variable	High sweet likers	Low sweet likers		
	Mean (SD)	Mean (SD)	t	p
Age (years)	41.92 (11.04)	41.43 (11.47)	.139	.890
EAT-26	3.83 (4.21)	3.79 (2.49)	.040	.969
sFFQ	8.50 (1.80)	8.43 (1.40)	.134	.894
EAT-26	3.83 (4.21)	3.79 (2.49)	.040	.969
BES* ¹	10.06 (6.43)	8.18 (6.95)	.731	.471
Craving Control* ²	51.33 (18.44)	42.43 (23.92)	1.408	.165
Sweet Craving* ²	52.33 (18.73)	47.21 (23.06)	.812	.421
Savoury Craving* ²	48.32 (15.14)	45.91 (17.63)	.482	.632
BMI (Kg/m ²)* ²	28.41 (2.60)	29.06 (3.17)	-.739	.464
Body weight (kg)* ²	80.98 (8.94)	84.80 (13.62)	-.971	.345
Fat mass (kg)* ²	29.13 (8.13)	28.95 (6.66)	.072	.943
Fat-free mass (kg)* ²	51.85 (8.81)	55.84 (12.28)	-1.285	.205
Body fat percentage * ²	35.91 (8.63)	34.41 (7.47)	.570	.571

Abbreviations: SD – standard deviation. BMI – body mass index. EAT – eating attitudes test. sFFQ – sweet food frequency questionnaire. *¹ n = 28 representing site optional questionnaires. *² values recorded during a participant's first visit to the lab, prior to exposure of the intervention product.

8.3.1 Co-Primary Outcomes

A series of 3x2x2 mixed ANOVAs with sweet liker phenotype as a between-subjects variable and condition (3 levels), probe day (2 levels) and time (2 levels) as fixed-factors were

conducted to examine differences in the effect of the intervention of sweet food preferences between high ($n=36$) and low ($n=14$) sweet likers. Means and standard deviations can be seen by sweet liker phenotype in Table 8.2, for each co-primary outcome, by condition, probe day and time of measurement. Sphericity was automatically assumed for probe day and time due to both only possessing two levels. Results for unadjusted and adjusted models can be seen in Table 8.3.

Table 8.2. Means and standard deviations of Leeds Food Preference Questionnaire outcomes in high ($n=36$) and low ($n=14$) sweet likers both condition, PD and time.

		Condition	199				550				647			
			PD1 Pre	PD1 Post	PD14 Pre	PD14 Post	PD1 Pre	PD1 Post	PD14 Pre	PD14 Post	PD1 Pre	PD1 Post	PD14 Pre	PD14 Post
Explicit Liking Sweet Bias	Mean (SD)	High Sweet Likers	23.62 (20.94)	8.94 (17.77)	20.12 (23.13)	7.02 (24.76)	21.48 (21.89)	7.56 (21.35)	19.15 (20.43)	4.21 (30.12)	20.45 (24.84)	10.48 (24.71)	19.86 (20.67))	7.25 (22.52)
		Low Sweet Likers	-12.55 (12.58)	-14.08 (15.52)	-13.97 (10.21)	-12.99 (19.50)	-7.18 (12.69)	-14.57 (16.15)	-11.93 (15.53)	-14.07 (17.32)	-14.63 (16.10)	-15.77 (19.30)	-11.08 (16.57)	-13.46 (13.47)
Explicit Wanting Sweet Bias	Mean (SD)	High Sweet Likers	21.1 (22.11)	8.37 (17.51)	17.51 (21.57)	4.73 (22.12)	19.31 (21.53)	4.13 (18.09)	16.85 (21.65)	2.51 (26.42)	19.08 (21.28)	10.51 (24.55))	17.97 (21.00)	4.99 (19.18)
		Low Sweet Likers	-13.4 (16.78)	-17.15 (19.99)	-14.96 (13.19)	-13.63 (15.14)	-10.04 (13.86)	-16.03 (18.84)	-13.84 (16.50)	-10.72 (16.02)	-14.48 (15.68)	-13.7 (16.40)	-11.8 (14.92)	-8.66 (15.87)
Implicit Wanting Sweet Bias	Mean (SD)	High Sweet Liker	42.25 (29.24)	18.36 (39.94)	30.49 (35.74)	14.46 (40.44)	37.21 (29.01)	15.62 (44.84)	35.61 (30.81)	19.02 (36.47)	34.79 (35.17)	20.68 (44.55)	36.91 (35.87)	14.37 (44.61)
		Low Sweet Likers	-23.00 (32.42)	-28.85 (31.35)	-28.08 (63.28)	-43.27 (63.28)	-26.89 (31.07)	-30.64 (28.04)	-29.97 (30.15)	-31.31 (28.56)	-23.04 (36.69)	-41.33 (34.39)	-20.89 (38.52)	-27.77 (26.21)
Choice Sweet Bias	Mean (SD)	High Sweet Liker	15.19 (10.50)	7.19 (15.43)	12.17 (14.09)	6.03 (16.70)	14.25 (11.92)	6.22 (16.62)	13.86 (12.29)	7.71 (14.88)	13.22 (13.09)	7.44 (16.54)	14.06 (12.70)	5.92 (16.00)
		Low Sweet Liker	-8.04 (11.73)	-10.43 (11.75)	-10.86 (11.72)	-10.86 (12.72)	-9.57 (10.46)	-11.79 (11.10)	-11.50 (12.11)	-11.96 (11.46)	-7.93 (14.16)	-15.29 (12.67)	-6.79 (14.99)	-11.79 (10.76)

Abbreviations: PD – probe day. SD – standard deviation.

Table 8.3. Models of adjusted and unadjusted mixed ANOVA results for Leeds Food Preference Questionnaire sweet bias outcomes.

		Unadjusted		Adjusted	
Effect*Sweet liker phenotype		F	<i>p</i>	F	<i>p</i>
EW Sweet Bias	Cond	.719	.471	-	-
	PD	4.929	.031	-	-
	Time	6.361	.019	-	-
	Cond*PD	.379	.686	-	-
	Cond*Time	.075	.928	-	-
	PD*Time	2.910	.094	-	-
	Cond*PD*Time	.779	.462	-	-
IW Sweet Bias	Cond	.084	.883	-	-
	PD	.179	.675	-	-
	Time	.1.770	.190	-	-
	Cond*PD	.889	.414	-	-
	Cond*Time	.830	.439	-	-
	PD*Time	.000	.998	-	-
	Cond*PD*Time	4.420	.015	3.400	.038^a
Choice Sweet Bias	Cond	.315	.691	-	-
	PD	.331	.568	-	-
	Time	1.889	.176	-	-
	Cond*PD	.624	.538	-	-
	Cond*Time	2.207	.116	-	-
	PD*Time	.615	.437	-	-
	Cond*PD*Time	.778	.432	-	-

Abbreviations: Cond – condition. PD – probe day. EL – explicit liking. EW – explicit wanting. IW – implicit wanting.

^agender and BMI were included as covariates in the adjusted model for IW sweet bias.

8.3.1.1 Explicit Wanting Sweet Bias

Tests of between-subjects effects demonstrated a significant main effect of sweet liker phenotype ($F(1,48) = 30.129, p < .001$), in which high sweet likers presented a greater EW sweet bias (mean = 12.26) than low sweet likers (mean = -13.20).

Mauchly's test of Sphericity was violated for condition ($p = .016$) but was assumed for condition*PD ($p = .226$), condition*time ($p = .493$) and condition*PD*time ($p = .242$). Tests of within-subjects effects demonstrated a significant effect of PD*sweet liker phenotype and time*sweet liker phenotype only (Table 8.3). High sweet likers displayed a reduction in EW sweet bias following consumption of the intervention product (pre-consumption mean = 18.64, post-consumption mean = 5.87), whereas low sweet likers did not demonstrate a significant change (pre-consumption mean = -13.08, post-consumption mean = -13.32) (Figure 8.1).

Similarly, high sweet likers displayed a reduction from PD1 (mean = 13.75) to PD14 (mean = 10.76) whereas low sweet likers displayed an increase from PD1 (mean = -14.13) to PD14 (mean = -12.27) (Figure 8.2) in EW sweet bias. Adjusted models did not identify any covariates for inclusion and so only unadjusted models are reported.

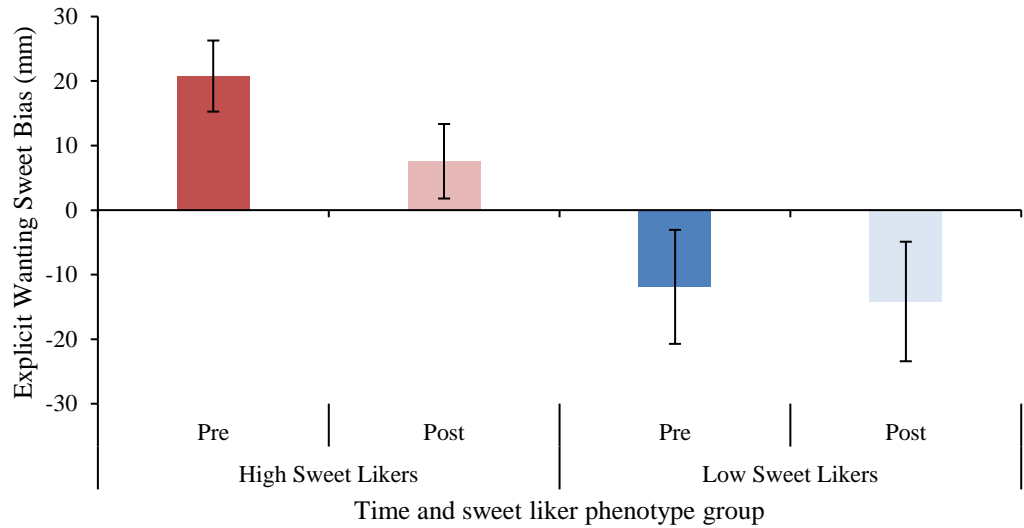


Figure 8.1. Differences in the change from pre- to post-ingestion explicit wanting sweet bias in high ($n=36$) and low ($n=14$) sweet likers. Error bars represent standard deviation.

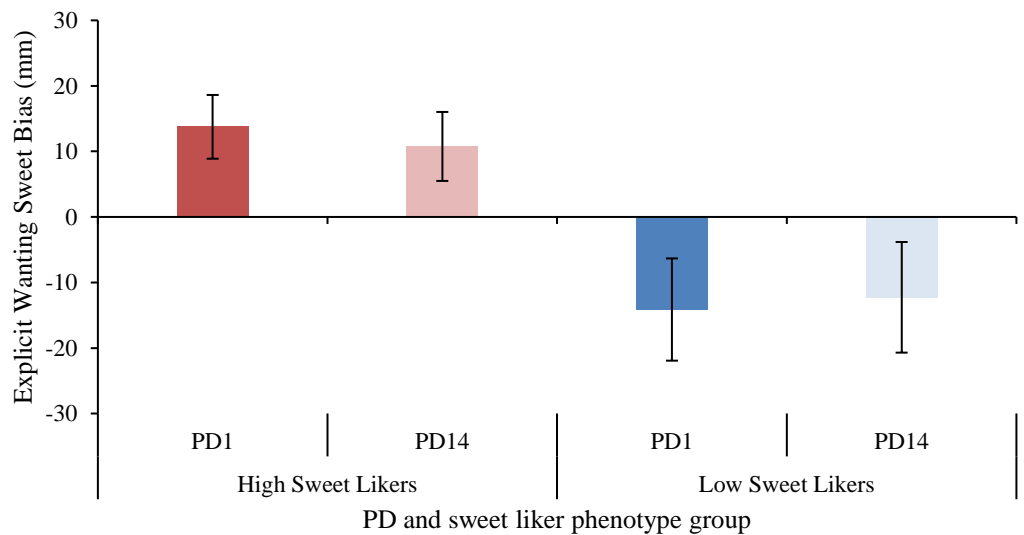


Figure 8.2. Differences in the change from PD1 to PD14 explicit wanting sweet bias in high ($n=36$) and low ($n=14$) sweet likers. Error bars represent standard deviation.

8.3.1.2 Implicit Wanting Sweet bias

Tests of between-subjects effects demonstrated a significant main effect of sweet liker phenotype ($F(1,48) = 37.855, p < .001$), in which high sweet likers presented a greater IW sweet bias (mean = 26.65) than did low sweet likers (mean = -29.59).

Mauchly's test of Sphericity was violated for condition ($p = .002$) but was assumed for condition*PD ($p = .324$), condition*time ($p = .905$) and condition*PD*time ($p = .802$). Tests of within-subjects effects demonstrated a significant interaction of condition*PD*time*sweet liker phenotype only (Table 8.3). The final adjusted model included gender and BMI as covariates, although this did not impact the results.

8.3.1.3 Choice Sweet Bias

Tests of between-subjects effects demonstrated a significant main effect of sweet liker phenotype ($F(1,48) = 35.072, p < .001$), in which high sweet likers presented a greater choice sweet bias (mean = 10.27) than did low sweet likers (mean = -10.57).

Mauchly's test of Sphericity was violated for condition ($p = .005$) but was assumed for condition*PD ($p = .620$), condition*time ($p = .140$) and condition*PD*time ($p = .137$). Tests of within-subjects demonstrated no significant effects, with adjusted models not altering this result, as such only unadjusted models are reported (Table 8.3).

8.3.2 Secondary Outcomes

8.3.2.1 Subjective Appetite Sensations

A series of 3x2 mixed ANOVAs with sweet liker phenotype as a between-subjects factor and condition (3 levels) and probe day (2 levels) as fixed-factors were conducted to examine differences in high ($n=36$) and low ($n=14$) sweet likers in the effect of condition and probe day on iAUC subjective appetite sensations. Sphericity was automatically assumed for probe day due to having two levels. Unadjusted models of tests of within-subjects effects can be seen in Table 8.4.

Table 8.4. Models of unadjusted mixed ANOVA results for iAUC subjective appetite rating outcomes.

		Effect*Sweet liker phenotype	F	<i>p</i>
iAUC	Appetite for Sweet (mm)	Cond	1.616	.207
		PD	.883	.352
		Cond*PD	.824	.442
iAUC	Appetite for Savoury (mm)	Cond	1.763	.177
		PD	.158	.693
		Cond*PD	.302	.740
iAUC	Hunger (mm)	Cond	1.574	.215
		PD	3.273	.077
		Cond*PD	1.218	.300
iAUC	Desire to Eat (mm)	Cond	.129	.879
		PD	.005	.945
		Cond*PD	.313	.732

Abbreviations: iAUC – incremental area under the curve. Cond – condition. PD – probe day.

Tests of between-subjects effects demonstrated no effect of sweet liker phenotype on appetite for sweet, ($F(1,47) = .040, p=.843$). Mauchley's test of Sphericity was violated for condition ($p=.039$) but was assumed for condition*PD ($p=.489$) and tests of within-subjects effects demonstrated no significant effects. Adjusted models did not alter these results with the inclusion of covariates and so only unadjusted models are reported (Table 8.4).

Tests of between-subjects effects demonstrated a significant effect of sweet liker phenotype on appetite for savoury, ($F(1,47) = 5.524, p = .023$). Pairwise comparisons demonstrated a

significant difference between high and low sweet likers in iAUC appetite for savoury (mean difference = 2363.19), with low sweet likers displaying a significantly greater iAUC appetite for savoury. Mauchley's test of Sphericity was assumed for condition ($p=.260$) and condition*PD ($p=.091$). Tests of within-subjects effects demonstrated no significant effects. The final adjusted models (Table 8.4) included BMI, site, gender and age as covariates, although this did not alter the tests of within-subjects effects and so are not reported, however, when including covariates, tests of between-subjects effects demonstrated no difference between sweet liker phenotype groups ($F(1,45) = 3.652, p=.062$).

Tests of between-subjects effects demonstrated no effect of sweet liker phenotype on subjective hunger ($F(1,47) = .188, p=.666$). Mauchley's test of sphericity was violated for condition ($p=.028$) but was assumed for condition*PD ($p=.352$). Tests of within-subjects effects demonstrated no significant effects (Table 8.4), and inclusion of covariates did not alter this finding, as such, only unadjusted models are reported.

Tests of between-subjects effects demonstrated no effect of sweet liker phenotype on desire to eat, ($F(1,47) = .380, p=.541$). Mauchley's test of sphericity was assumed for condition ($p=.544$) and condition*PD ($p=.856$). Tests of within-subjects effects demonstrated no significant effects and adjusted models did not alter this finding (Table 8.4).

8.3.2.2 Control of Eating Questionnaire

A series of 3x2 mixed ANOVAs were conducted with sweet liker phenotype as a between-groups factor and with condition (3 levels) and probe day (2 levels) as fixed-factors to examine differences between high ($n=36$) and low ($n=14$) sweet likers on the effects of condition and repeated exposure on CoEQ outcomes. Sphericity was automatically assumed for probe day due to having two levels. Unadjusted values can be seen in Table 8.5 with no adjusted models included as no covariates of note were identified.

Table 8.5. Model of unadjusted mixed ANOVA results for Control of Eating Questionnaire outcomes.

		Unadjusted	
		F	<i>p</i>
Effect*Sweet liker phenotype			
Craving Control	Cond	.144	.808
	PD	.201	.656
	Cond*PD	1.623	.203
Sweet Craving	Cond	1.102	.336
	PD	.000	.992
	Cond*PD	.994	.374
Savoury Craving	Cond	2.127	.125
	PD	.110	.742
	Cond*PD	1.174	.314

Abbreviations: Cond – condition. PD – probe day. EL – explicit liker.

^aGender was included as a covariate within the adjusted model for savoury craving.

Tests of between-subjects effects demonstrated no effect of sweet liker phenotype on craving control, ($F(1,48) = .290, p=.593$). Sphericity was violated for condition ($p<.001$) but was assumed for condition*PD ($p=.623$). Tests of within-subjects effects demonstrated no significant effects (Table 8.5).

Tests of between-subjects effects demonstrated no effect of sweet liker phenotype on sweet craving, ($F(1,48) = 3.581, p=.064$). Sphericity was assumed for condition ($p=.096$) and condition*PD ($p=.326$). Tests of within-subjects effects demonstrated no significant effects (Table 8.5).

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on savoury craving, ($F(1,48) = .503, p=.482$). Sphericity was assumed for condition ($p=.772$) and condition*PD ($p=.149$). Tests of within-subjects effects demonstrated no significant effects (Table 8.5).

8.3.3 Body Weight and Composition

A series of 3x2 mixed ANOVAs were conducted with condition (3 levels) and PD (2 levels) as fixed-factors and sweet liker phenotype as a between-groups factor to examine differences between high and low sweet likers on the effects of condition and repeated exposure on body weight and composition outcomes. Sphericity was assumed for PD automatically due to having

two levels. Unadjusted models can be seen in Table 8.6, with no final adjusted models included as no covariates of note were identified.

Table 8.6. Model of unadjusted values for mixed ANOVA results for body weight and composition outcomes.

	Effect*Sweet liker phenotype	Unadjusted	
		F	p
Body weight (kg)	Cond	.238	.752
	PD	4.512	.039
	Cond*PD	.286	.752
Fat mass (kg)	Cond	1.481	.233
	PD	1.392	.244
	Cond*PD	.001	.998
Fat free mass (kg)	Cond	1.059	.341
	PD	4.377	.042
	Cond*PD	.275	.720
Body fat %	Cond	2.080	.131
	PD	2.087	.155
	Cond*PD	.007	.989

Abbreviations: Cond – condition. PD – probe day. Kg – kilogram.

Tests of between-subjects effects demonstrated there was no effect of sweet liker phenotype on body weight (kg) ($F(1,46) = .956, p=.333$). Sphericity was violated for condition ($p=.011$) but was assumed for condition*probe day ($p=.526$). Tests of within-subjects effects demonstrated a significant probe day*sweet liker phenotype interaction only (Table 8.6). As demonstrated in Figure 8.3 body weight did not change in high sweet likers between PD1 (mean = 81.32) and PD14 (mean = 81.35) whereas the low sweet likers increased from PD1 (mean = 84.52) to PD14 (mean = 84.93).

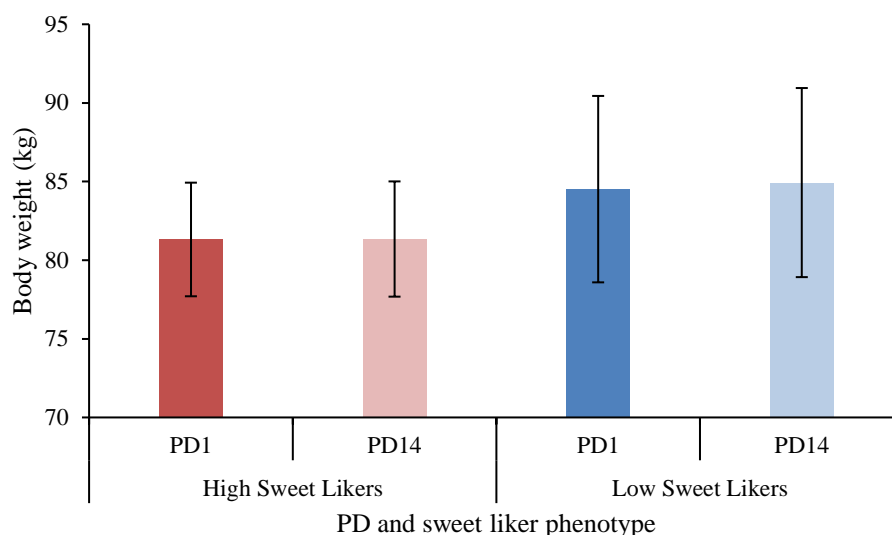


Figure 8.3. Differences in change in body weight (kg) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers. Error bars represent standard error of the mean.

Tests of between-subjects effects demonstrated no difference between high and low sweet liker phenotype groups on fat mass (kg) ($F(1,46)=.025, p=.875$). Sphericity was assumed for condition ($p=.515$) but was violated for condition*PD ($p=.037$). Tests of within-subjects effects demonstrated no significant effects (Table 8.6).

Tests of between-subjects effects demonstrated no difference between high and low sweet liker phenotype groups on fat-free mass (kg), ($F(1,46)=1.438, p=.237$). Sphericity was violated for condition ($p=.005$) and condition*probe day ($p=.007$). Tests of within-subjects effects demonstrated a significant effect of probe day*sweet liker phenotype only (Table 8.6). As displayed in Figure 8.4 high sweet likers displayed a small reduction from PD1 (mean = 51.60kg) to PD14 (mean = 51.46kg), whereas low sweet likers displayed an increase from PD1 (mean = 55.05kg) to PD14 (mean = 55.84kg).

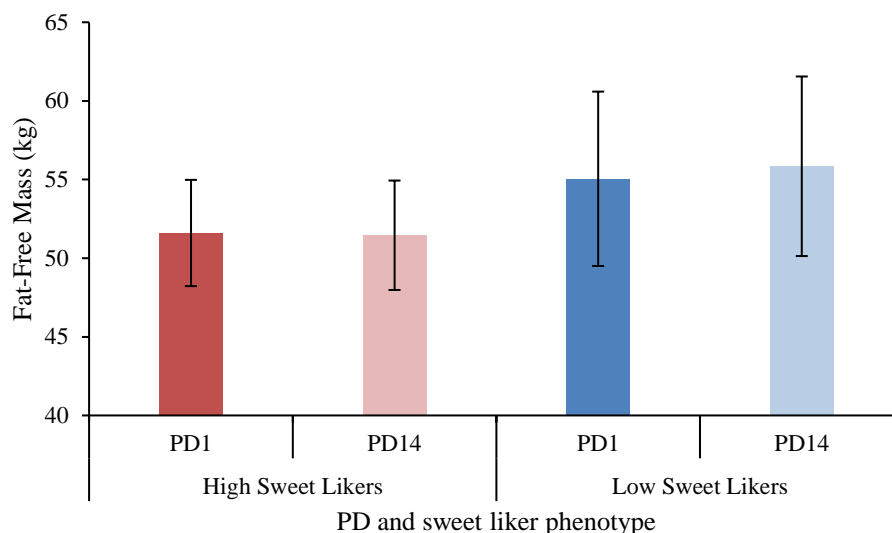


Figure 8.4. Differences in changes in fat-free mass (kg) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers. Error bars represent standard error of the mean.

Tests of between-subjects effects demonstrated no difference between high and low sweet liker phenotype groups on body fat percentage, ($F(1,46) = .350, p=.557$). Sphericity was assumed for condition ($p=.259$) but was violated for condition*PD ($p=.048$). Within-subjects effects demonstrated no significant effects (Table 8.6).

8.3.4 Diet Recall

A series of 3x2 mixed ANOVAs with sweet liker phenotype as a between subjects factor and condition (3 levels) and probe day (2 levels) as fixed-factors were conducted to examine differences in dietary recall values between high ($n=35$) and low ($n=13$) sweet likers Table 8.7 displays the unadjusted and adjusted values.

Table 8.7. Model of unadjusted mixed ANOVA results for dietary recall outcomes.

		Unadjusted	
Effect*Sweet liker phenotype		F	<i>p</i>
Total Energy Intake (kcal)	Cond	.224	.800
	PD	11.997	.001
	Cond*PD	1.655	.197
CHO (g)	Cond	.703	.498
	PD	11.641	.001
	Cond*PD	1.646	.198
Fibre (g)	Cond	.473	.625
	PD	5.594	.022
	Cond*PD	.235	.791
Sugar (g)	Cond	.549	.579
	PD	3.079	.086
	Cond*PD	.045	.956
Protein (g)	Cond	.541	.584
	PD	.686	.412
	Cond*PD	.092	.913
Fat (g)	Cond	.289	.749
	PD	6.363	.015
	Cond*PD	1.398	.252

Abbreviations: Cond – condition. PD – probe day. EL – explicit liker.

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on total energy intake (kcal), ($F(1,46) = .024, p = .878$). Mauchley's test of sphericity was assumed for condition ($p = .854$) and condition*probe day ($p = .129$). Tests of within-subjects effects showed a significant interaction of probe day*sweet liker phenotype only (Table 8.7). High sweet likers displayed an increase from PD1 (mean = 2,075.21) to PD14 (mean = 2,188.44) whereas low sweet likers displayed a decrease from PD1 (mean = 2,280.59) to PD14 (mean = 1,932.23) as shown in Figure 8.5. No covariates of note were reported and so only unadjusted models are reported for total energy intake.

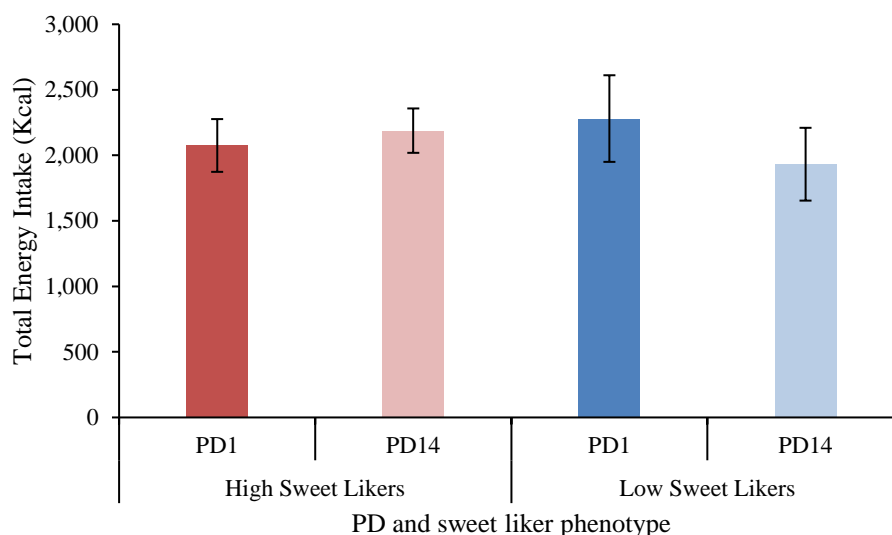


Figure 8.5. Differences in changes in total energy intake (kcal) between high ($n=35$) and low ($n=13$) sweet likers between PDs. Error bars represent standard error of the mean.

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on carbohydrate intake (g), ($F(1,46)=.057, p=.813$). Mauchley's test of sphericity was assumed for condition ($p=.853$) and condition*probe day ($p=.251$). Tests of within-subjects effects demonstrated a significant interaction of probe day*sweet liker phenotype (Table 8.7). As displayed in Figure 8.6 high sweet likers displayed an increase in carbohydrate intake (g) from PD1 (mean = 245.14g) to PD14 (mean = 259.03g) whereas low sweet likers displayed a decrease from PD1 (mean = 268.23) to PD14 (mean = 224.59). Adjusted models did not note any covariates for inclusion and so only the unadjusted model is reported.

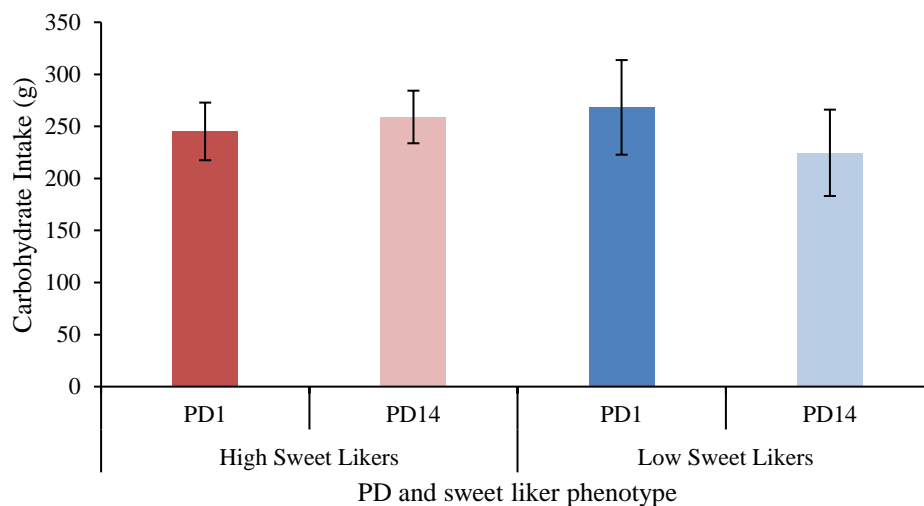


Figure 8.6. Difference in change in carbohydrate intake (g) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers. Error bars represent standard error of the mean.

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on fibre intake (g), ($F(1,46)=.131, p=.719$). Mauchly's test of sphericity was assumed for condition ($p=.091$) and condition*probe day ($p=.572$). Tests of within-subjects effects demonstrated a significant interaction of probe day*sweet liker phenotype (Table 8.7). As displayed in Figure 8.7 high sweet likers displayed an increase in fibre intake (g) from PD1 (mean = 18.82g) to PD14 (mean = 19.46g) whereas low sweet likers displayed a reduction from PD1 (mean = 20.21g) to PD14 (mean = 16.92g). Adjusted models did not note any covariates for inclusion and so only the unadjusted model is reported.

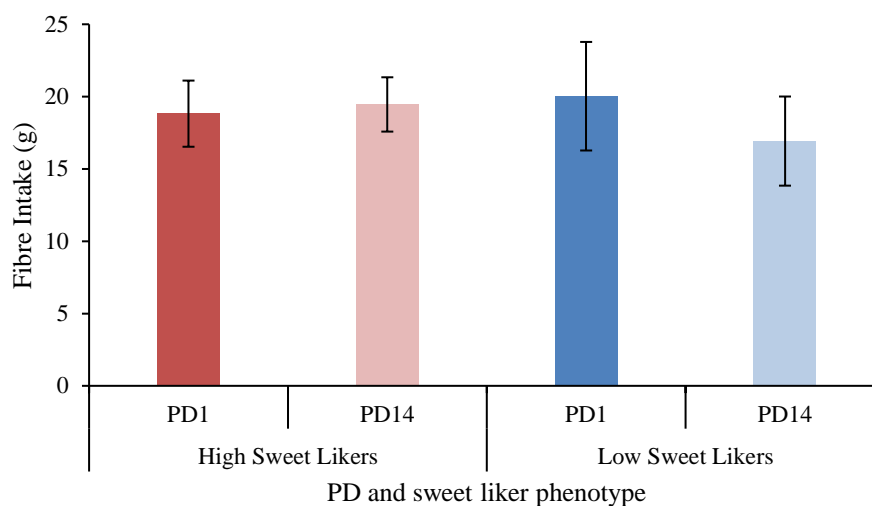


Figure 8.7. Differences in change in fibre intake (g) from PD1 to PD14 between high ($n=35$) and low ($n=13$) sweet likers. Error bars represent standard error of the mean.

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on sugar intake (g), ($F(1,46)=1.690$, $p=.200$). Mauchly's test of sphericity was assumed for condition ($p=.360$) and condition*probe day ($p=.196$). Unadjusted models did not demonstrate a significant effect and adjusted models did not alter this finding, as such only the unadjusted model is reported (Table 8.7).

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on protein intake (g), ($F(1,46)=.155$, $p=.696$). Mauchly's test of sphericity was assumed for condition ($p=.109$) and condition*probe day ($p=.221$). Tests of within-subjects effects demonstrated no significant effects (Table 8.7) and adjusted models did not note any covariates for inclusion, therefore only the unadjusted model is reported.

Tests of between-subjects effects demonstrated no main effect of sweet liker phenotype on fat intake (g), ($F(1,46)=.046$, $p=.832$). Mauchly's test of sphericity was assumed for condition ($p=.316$) and condition*probe day ($p=.215$). Tests of within-subjects effects demonstrated a significant interaction of probe day*sweet liker phenotype only (Table 8.7). As displayed in Figure 8.8 high sweet likers displayed an increase in fat intake (g) from PD1 (mean = 85.12g) to PD14 (mean = 89.64g) whereas low sweet likers displayed a decrease from PD1 (mean = 91.28g) to PD14 (mean = 89.74g).

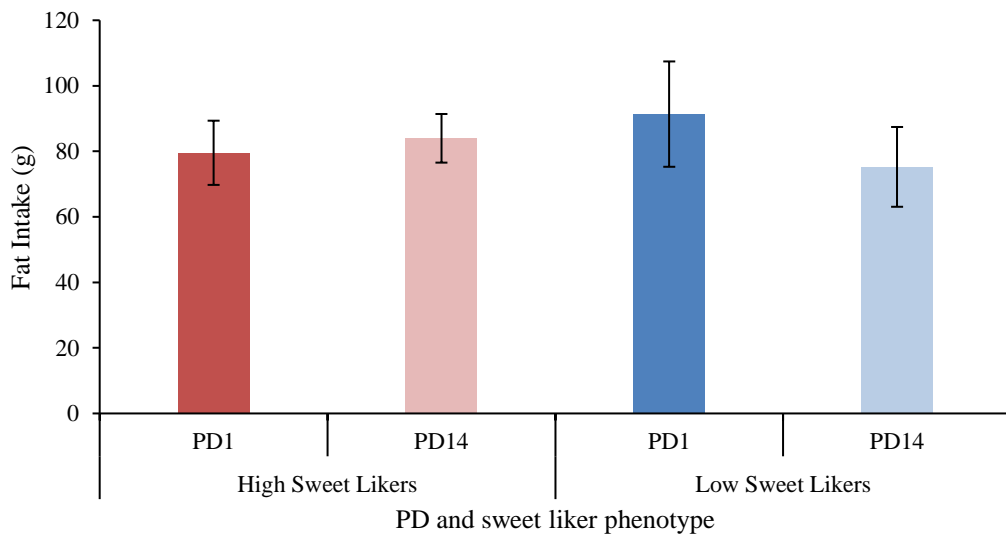


Figure 8.8. Differences in change in fat intake (g) from PD1 to PD14 in high ($n=35$) and low ($n=13$) sweet likers.

Error bars represent standard error of the mean.

8.4 Discussion

The aim of the present chapter was to explore the potential differences between high and low sweet liker phenotypes in the response to acute and repeated exposure of two different sweetener types or sucrose in a solid food matrix on subsequent sweet food preferences and associated outcomes. Results demonstrated that there were significant differences in sweet food liking and wanting between sweet phenotype groups as would be anticipated. Further to this, there were differences between groups in the changes to sweet food preferences as a result of acute exposure to the intervention products, with high sweet likers displaying a reduction in EL and EW sweet bias following acute consumption of the intervention products. Furthermore, high sweet likers also displayed a reduction in EW sweet bias following repeated exposure, whereas low sweet likers displayed a savoury preference which remained relatively unaltered. However, there was no interaction between sweetener condition or phenotype for either acute or repeated exposure. Additionally, there was no effect of phenotype on CoEQ related outcomes or subjective appetite sensations, with the exception of iAUC appetite for savoury, which was significantly greater in low sweet likers. Low sweet likers also displayed a reduction in total energy intake from PD1 to PD14, which appeared to be driven by a reduction in carbohydrate, fat and fibre.

8.4.1 Sweet Food Preferences

Following the findings of Chapter 7 which identified the potential of habitual sweet food consumption (which did not differ between sweet liker phenotype groups) to impact the effect of condition on subsequent sweet food preferences, the current findings demonstrated a number of interesting differences in the role of sweet liker phenotype. As would be anticipated sweet phenotype groups differed significantly in their sweet liking and wanting. Groups differed in their EL sweet bias as a consequence of the means of categorising sweet liker status within the current study and so a main effect of phenotype on EL sweet bias is uninformative. However, it is rather more illuminating to examine the effects on wanting as well as interaction effect of sweet liker phenotype and time, an interaction which was present for EW sweet bias, as this identified potential differences between phenotype groups in the strength of sensory specific

satiety. Previously, normal weight participants and those with obesity were shown to not differ in their sensitivity to sensory specific satiety (Snoek et al., 2004), however, this is the first study to date that has shown potential differences in the effect of sensory-specific satiety in individuals categorised on the basis of their taste preferences. A so-called transfer effect of sensory-specific satiety occurs, whereby consumption of a taste serves to diminish preferences for that taste, whilst increasing alternate tastes, an effect which is stronger for savoury to sweet, than sweet to savoury (Griffioen-Roose et al., 2010). The findings of the previous chapter supported this claim by demonstrating a lack of a transfer for sweet to savoury, the findings of the current chapter on the other hand elaborate this via demonstration of the impact of sweet liker phenotype. The diminishing effects of sensory specific satiety following consumption of a sweet food is stronger in high sweet likers as low sweet likers present with a savoury preference which remains comparatively unaltered. Alternatively, this may be caused by a floor effect within the data, indicating that low sweet likers have already reached a point at which a further decrease in sweet food preferences is no longer possible.

Moreover, there was an effect of repeated exposure on EW sweet bias which differed between phenotype groups. Low sweet likers demonstrated an increase in their sweet preference, which due to the operationalisation of the LFPQ presenting a sweet relative to savoury preference, is displayed as a reduction in savoury preference and should be framed as such. Previously it has been shown that repeated consumption of a flavour trains preferences (Liem & De Graaf, 2004), which appears to be true in the current data set for low sweet likers, displaying a reduction in savoury relative to sweet preference. However, high sweet likers demonstrated a reduction in EW sweet bias following repeated exposure, thereby highlighting differences between sweet liker phenotype groups. Elsewhere evidence has examined the effect of repeated consumption of novel foods, highlighting dietary monotony (Zandstra et al., 2000) or boredom as key influences in reducing preferences (Hoek et al., 2013). In a study employing daily exposure for 3 weeks, it was demonstrated that boredom occurred even in participants who did not think they would become bored with a food (Zandstra et al., 2004) and another study highlighted that women (across a range of BMIs) consuming 300kcal portions of a snack

displayed a reduction in liking following 2 weeks of daily exposure (Temple et al., 2009).

These findings highlight that boredom is a key influence in reducing subsequent food preferences following repeated consumption. The findings of the current chapter further elaborate this by indicating that a reduction in preference may only occur in individuals presenting with a preference for the corresponding taste (i.e., consumption of a sweet food will reduce sweet preference in those with a sweet bias). In those individuals with alternative preferences (i.e., savoury bias) the repeated consumption of sweet foods will have a markedly reduced impact on preferences. Thereby whether repeated exposure will train or reduce preferences may be dependent on the pre-existing preferences to begin with.

Moreover, in the previous chapter it was outlined that the type of sweetener may be of minimal importance when impacting subsequent food preferences (Bartolotto, 2015; Ebbeling et al., 2020; Mahar & Duizer, 2007) and there was not an effect of condition demonstrated. The current findings develop this further by outlining that there remains no effect of condition on subsequent sweet preferences when dichotomising participants on the basis of sweet liker phenotype status. From the findings of the current and previous chapter, it is concluded that sweetener type does not impact subsequent sweet food preferences, rather it is the consumption of a sweet food in and of itself that impacts preferences. The current findings in particular highlight differences in the effect of sweet food consumption between sweet liker phenotype groups, following both acute and repeated consumption.

8.4.2 Appetite Sensations and Food Cravings

Firstly, the current findings demonstrate at a participants' first visit to the lab, prior to exposure of the intervention products (i.e., baseline) high and low sweet likers did not differ in their craving control or cravings for sweet or savoury, thereby reiterating findings presented in Chapter 5 - that dichotomised sweet phenotype groups do not differ in their cravings. To date the relationship between sweet liker phenotype and sweet craving has not been studied (Yang et al., 2020) and so this represents novel findings which partly addresses this gap within the literature, although further research is recommended employing a protocol specifically designed and powered to detect differences between phenotype groups. Secondly, there was not

an effect of condition following acute exposure on iAUC subjective appetite ratings, suggesting that there is no difference between sweet phenotype groups in the acute exposure effects of differing sweeteners. However, despite no difference in baseline cravings, following ingestion of the study food there was however a significant main effect of sweet liker phenotype on iAUC appetite for savoury. Low sweet likers reported greater subjective ratings of appetite for savoury than did high sweet likers. This is an important finding as it highlights that low sweet likers not only present with a stronger savoury relative to sweet preference (demonstrated in the LFPQ), but they present with a generalised preference for savoury, that is independent of other tastes. This finding demonstrates therefore that it is not merely that low sweet likers in the current data set prefer savoury to sweet, but that their preference is for savoury foods overall.

Furthermore, there was a lack of difference present between high and low sweet likers on changes to cravings following repeated exposure to the intervention products. When examining subjective appetite sensations there was no impact of sweet liker phenotype on iAUC appetite for sweet or other subjective appetite related sensations. Similarly participants did not differ in craving control, sweet craving or savoury craving. It was previously concluded in Chapter 7 that HIS do not promote sweet cravings, as is expressed as a concern elsewhere (Liem & De Graaf, 2004; Yang, 2010), the present findings provide further credence to this conclusion and develop it further via demonstrating that sweet liker phenotype is not an influential factor. In both high and low sweet likers, craving control improves and sweet cravings reduce following repeated consumption of a sweet product, independent of sweetener type employed. Therefore recommendations for consumption of reformulated products specifically to satisfy cravings (O'Connor et al., 2021) may be made regardless of sweet liker phenotype, as craving control has been shown to improve in those individuals with both an elevated and depressed sweet food preference.

8.4.3 Body Weight and Composition

There were no differences at baseline in body composition outcomes between phenotype groups. However, recruitment criteria specified a BMI between 25-35 kg/m², and so all participants within both groups represented individuals with overweight and obesity. Moreover,

the study protocol employed was not designed to detect differences between phenotype groups, and so the lack of difference may be due to methodological differences.

Interestingly there was a significant interaction of sweet liker phenotype and probe day for both body weight and fat-free mass. It is likely that the effect of body weight was driven by the effect of fat-free mass, as any change in fat-free mass will consequently result in a change to body weight, assuming fat mass remains unaltered, as in this scenario. There is limited evidence regarding H₂O consumption and consequential water retention, however examination of a rodent model highlighted that when comparing diets sweetened with sucrose, saccharin or aspartame when energy intake was held constant, there was greater weight gain in the saccharin and aspartame conditions, speculated to be produced by an increase in water retention (Feijó et al., 2013). However, as there was no effect of condition it cannot be stated that these effects are produced by the consumption of different sweetener types. The precise reason for the increase in body weight and fat-free mass in low sweet likers remains unclear, although it is noted that the increases were marginal and therefore likely represent random variation within the present data set. Therefore, no firm conclusions are drawn surrounding differences in phenotype groups on the effects of the intervention on body weight or composition.

8.4.4 Diet Recall

Findings did not reveal any effects of sweet liker phenotypes and due to the study protocol employed it was not possible to examine the differences in dietary intake prior to exposure of the study foods (i.e., habitual intake). However there were noted some important interaction effects. First to note is the lack of interaction with condition, within the previous chapter it was concluded that condition did not subsequently impact dietary intake, the current analysis further develops this conclusion by highlighting that sweet liker phenotype does not impact this.

Previous evidence has demonstrated that reformulated products do not impact total energy intake because a reduction in carbohydrate and sugar intake leads to an increase in protein and fat intake (Markey et al., 2016). However, this was demonstrated in participants within a normal range BMI. Within Chapter 2 it was demonstrated that in participants with overweight

and obesity, reformulated products may facilitate a reduction in energy intake, specifically from carbohydrates and sugars. However, the current intervention products provided minimal difference in energy (see Chapter 3). Therefore although reformulated products may not guarantee a reduction in the energy density of the diet, there is no consequential increase in intake which is often stated as a concern (Yang, 2010).

On the other hand, low sweet likers reported interactions with probe day, displaying greater reductions in energy, carbohydrate, fibre and fat intake than the changes observed in high sweet likers. These findings when considered in line with the reductions in liking and wanting observed earlier in the chapter, suggest that any effects of dietary boredom following repeated consumption, result in a reduction in intake of corresponding foods in low sweet likers. However, important to note is that in the current protocol participants were required to consume a daily portion of the intervention product, it remains to be seen whether when freely choosing to consume the products or not, whether the alterations to dietary intake would be observed.

Liking for sweet is commonly thought to have developed as a mechanism to identify carbohydrate sources (Armitage et al., 2021). Therefore, the daily consumption of a sweet food product may be responsible for a reduction in other sweet carbohydrates in low sweet likers. However, this conclusion must be made with caution due to the caveats within the method of diet recall employed in the current study such as only representing a 24 hr window of eating, that was an atypical day for participants. It is recommended that future studies investigate this potential effect further. Furthermore, it has been demonstrated elsewhere that high sweet likers present with a higher intake of sugars, (Holt et al., 2000) and less fibre intake (Turner-McGrievy et al., 2013). However, the current findings disagree with this, and instead support findings demonstrating no difference in intake between sweet liker phenotype groups (Methven et al., 2016). To date no study has examined the effect of different sweetener types on subsequent food intake in high and low sweet likers, and so although the findings of the current chapter are made with caution with an acknowledgement of the caveats of the data, they represent a novel examination that future studies may wish to re-examine in future.

8.4.5 Conclusion

The findings of the present chapter represent a novel exploratory examination of differences in the responses to an intervention involving the acute and repeated consumption of biscuits containing three different sweetener types. The findings confirm that high and low sweet likers also differ in their wanting for sweet – with low sweet likers presenting savoury wanting - as well as highlighting differences in the acute effects of ingestion of a sweet stimuli. It is concluded that high sweet likers display a greater reduction in sweet liking and wanting following both acute and repeated exposure to a sweet food, although this is not impacted by condition and the effects appear to be generalisable to sweet foods overall rather than sweetener type. The relationship between sweet liker phenotype and cravings has not been previously examined, and so the current conclusion that phenotype groups do not differ in their baseline cravings nor the effect of the intervention on their cravings is novel. Finally, high and low sweet likers differed in their diet recall, with low sweet likers displaying a reduction in total energy intake, likely driven by a reduction of carbohydrate and fibre intake. Overall there was no interaction between condition and sweet liker phenotype, thereby highlighting that sweet phenotype groups do not respond differently to different types of sweeteners when included in a solid food matrix. Any differences observed between sweet phenotype groups were due to the general consumption of a sweet food and not the type of sweetener used.

9 General Discussion

9.1 Overview of thesis

Following a systematic review of the literature, the research presented in the current thesis has provided an examination of sweet food preferences in the context of weight loss in females with overweight and obesity as well as the impact of a sweet food containing sucrose or reformulated with no added sugar and sweeteners on subsequent sweet food preferences, by means of two separate clinical trials. Through making use of a validated behavioural measure of food reward in which liking and wanting for sweet relative to savoury foods are assessed with both explicit and implicit components (Finlayson et al., 2007b), the current thesis sought to expand upon current knowledge of sweet food preferences and factors contributing to appetite regulation. The use of the behavioural measure of food reward also enabled the examination of a novel method of categorising sweet liker phenotype status in participants and whether high and low sweet likers displayed differences in the relationships between sweet food preferences and changes in eating behaviour traits, as well as changes in sweet food preferences following consumption of a sweet food in response to both interventions.

Through the use of a systematic review the effects of sweet consumption on appetite, food reward and body weight were identified, highlighting that HIS consumption does not impact subjective appetite, food intake or appetite-related biomarkers, but may facilitate a reduction in sugar and energy intake. Utilising a RCT trial in both lean women and those with overweight and obesity, sweet food preferences were demonstrated to display acute variation over a single day, whilst remaining highly stable in women with overweight and obesity during a period of dietary induced weight loss. It was also demonstrated that women with overweight and obesity present at baseline with a number of eating behaviour traits that may be characterised by a loss of control around food, relative to their lean counterparts. This RCT also demonstrated that two different methods of dietary induced weight loss did not differentially impact sweet food preferences or weight loss. A second RCT examined the subsequent effects on sweet food preferences of reformulated foods, demonstrating sweetener type exerts limited effect on

subsequent sweet food preferences, with any effect exerted primarily caused by the consumption of a sweet stimulus. Together, these findings have enhanced our understanding of sweet food preferences and the associated appetite regulatory mechanisms.

9.2 Systematic review of RCTs comparing HIS vs. sucrose and/or water controls on appetite, food reward and body weight

A systematic review was conducted in Chapter 2 to examine RCTs in which HIS were compared to sucrose and/or water controls in their effects on appetite, food reward and body weight related outcomes. The review included 58 studies, 42 studies included appetite related outcomes, 18 studies included food reward related outcomes and 16 studies included body weight related outcomes. Key findings from the review highlight HIS do not appear to impact appetite related outcomes, with no observable changes to subjective appetite ratings, appetite related hormones or ingestive behaviours. Notably, when HIS beverages substitute sugar sweetened beverages alongside a meal, meal energy intake remains unaltered and when including the energy content of the beverages in the model, HIS conditions have a lower overall total energy intake than sugar sweetened beverage conditions. This finding is interpreted to mean that the energy content of sucrose sweetened products when consumed alongside a meal are not fully compensated for by participants during the meal. HIS therefore can facilitate a reduction in energy intake by substituting energy containing beverages with low or no energy alternatives. However, this finding can only be applied to HIS sweetened beverages with extremely limited evidence available regarding the use of HIS in solid foods. A meta-analysis of subjective appetite ratings was anticipated, however due to inconsistencies in methodology, data proved to be insufficient, a finding corroborated by other recent evidence (Mehat et al., 2022). Similarly, it was not possible to draw meaningful conclusions regarding the effects of HIS on food reward due to inconsistent methodologies and definitions surrounding food reward assessment. Finally, effects of HIS on body weight related outcomes differ between population sub-groups, with no impact on lean individuals whereas in individuals with overweight and obesity there is a reduction in body fat. Overall the substitution of sugars for HIS in the diet

may facilitate a reduction in energy intake via a specific reduction in sugar and carbohydrate intake in individuals with an elevated BMI and aid in weight loss attempts.

The review also identified a number of issues surrounding the study of HIS, primarily despite there being a diverse array of HIS available for public consumption which demonstrate the potential for differing effects, research treats HIS as a homogenous group and conclusions are not drawn for distinct HIS separately. Additionally, the preponderance of evidence available is provided via use of a minority of HIS types and the vehicle of ingestion tends to be beverages, and so further research is required to fully understand the impact of HIS inclusion in the diet. Overall the findings of Chapter 2 demonstrate a potential for HIS to facilitate a reduction in energy intake via a substitutive strategy, although this does not operate by altering appetite or food intake, but rather the lower energy content of sweetened products.

9.3 Are sweet food preferences stable?

The experimental protocol employed in Chapters 4, 5 and 6 provided an examination of sweet food preferences in women with overweight and obesity in the context of a weight loss intervention comparing two dietary strategies. The first experimental chapter (Chapter 4) highlighted that individuals experience a high degree of variability in their sweet food preferences across a single day when fasted and in response to consumption of a meal. This is likely due to the sensory aspects and energy content of foods consumed throughout the day. The so-called transfer effect of sensory specific satiety is well documented (e.g., Griffioen-Roose et al., 2010), in which there is a definite and immediate increase in sweet food preferences following consumption of a savoury food or meal, providing an explanation for the increased sweet preference immediately post *ad libitum* meal consumption. It has been highlighted elsewhere that preferences for savoury foods increase around meal times (Forde et al., 2013), which may be in-part responsible for the drop in sweet relative to savoury preference prior to the lunch meal. Regarding the elevated sweet preference observed in the morning, this may be in part influenced by cultural experiences around meal times. In Western societies certain foods are considered appropriate or inappropriate for particular meals (Birch et al.,

1984; Kramer et al., 1992). For example, breakfast foods tend to be higher in carbohydrates whilst dinner tends to be higher in fats (Westerterp-Plantenga, 1999). This may also be reflective of nutritional requirements, as in the morning the body is required to load up on energy-dense, carbohydrate rich foods (Adolphus et al., 2013; Jakubowicz et al., 2012).

Alternatively, the acute variation in sweet preferences may also be created by food intake throughout the day. Food images are rated as more pleasant when fasted than when satiated (Uher et al., 2006), which is in line with the findings of Chapter 4. Elsewhere it has been demonstrated that sweet foods tend to be consumed between meals during snacking occasions, with main meals constituting savoury foods (van Langeveld et al., 2018), therefore assessment of food preferences prior to a main meal may reflect a participant's anticipation of this. The large increase in sweet preference immediately after a meal is similarly observed in the second study of the thesis, whereby sweet food preferences declined immediately following consumption of sweetened biscuits, which is also likely attributable to sensory specific satiety (Chapter 7). These findings taken together suggest that food consumption throughout the day may be capable of impacting sweet food preferences on an acute basis, and should be considered in future research protocols in which food preferences are an objective outcome.

In Chapter 4 sweet food preferences were also demonstrated to be highly stable across a period of dietary induced weight loss and Chapter 5 highlighted no significant change to sweet preferences following weight loss. This is counter to currently available literature which tends to demonstrate alteration to sweet preferences following weight loss (e.g., Rodin et al., 1976; Umabiki et al., 2010). For example, in a sample of female participants following 14.6% weight loss over 30 weeks, authors described the change in preferences as the normalisation of a sucrose preference – in that following the reductions in body weight, sweet preferences was comparable to that of normal weight controls (Nishihara et al., 2019). This statement in particular would suggest that an individual's food preferences are a consequence of body composition, specifically body fat levels, rather than body weight being a consequence of an elevated sweet preference. Moreover, the findings of Chapter 4, due to being illuminated via an intra-individual analysis, should be interpreted as indicating that potential changes to food

preferences occur equivocally across participants. Therefore, although it remains possible that a sweet preference may decline during weight loss, this occurs to a similar degree across all women with overweight/obesity. Despite literature demonstrating the sweet preferences may become altered during weight loss, this was not shown within the findings of Chapter 4 within the present thesis, therefore it may be concluded from the current that sweet food preferences are stable across a period of weight loss. However it should be noted that the amount of weight loss is not equivocal across studies, with 14.6% weight loss occurring over 30 weeks in Nishihara (2019), or 8% occurring over 8 weeks in Andriessen (2018) which also demonstrated alterations to taste preferences. The current study involved a protocol to elicit 5% weight loss, and so it remains a possibility that a greater degree of weight loss is necessary to elicit alterations to taste preferences.

Moreover, Chapter 6 demonstrated that the dietary protocol employed did not impact sweet food preferences either. This is a key finding for practitioners when determining dietary protocols for individuals seeking to reduce body weight, as this indicates that the means of achieving weight loss may be of limited importance. Within the same participants it was shown elsewhere that the two dietary protocols did not impact appetite ratings differently, nor were there group differences in body composition outcomes (Beaulieu et al., 2020). Therefore, this finding supports a person-centred approach to nutritional interventions, seeking to adopt a protocol which aligns with an individual's lifestyle and goals, which may aid in adherence rates (Fastenau et al., 2019). Although the study did not attempt to assess or measure participant satisfaction with the dietary protocols beyond the reason for participant discontinuation, it should also be noted that 5 participants withdrew from the IER condition due to issues with the foods or meal plan provided, compared to only 1 participant from the CER group for the same reason.

From these findings it can be concluded that sweet food preferences represent a highly stable trait in individuals. Despite displaying variation on an acute basis due to physiological factors, as well as responding to dietary intake, preferences do not change across prolonged periods of time. Therefore, the findings of Chapter 4, specifically the strength of the intraclass correlation

coefficients and Chapter 6, highlighting that the method of weight loss is of limited relevance when impacting sweet food preferences, indicate that phenotyping individuals on the basis of their sweet liking can be conducted with confidence, as from the current analysis it appears unlikely that an individual's sweet preferences change over a period of dietary induced weight loss.

9.4 Eating behaviour traits and sweet food preferences

It is well established within the wider literature that eating behaviour traits differ by BMI groups (Burton et al., 2007; Cappelleri et al., 2009; Chao et al., 2014; Christensen, 2007; Gallant et al., 2010) which is reinforced by the findings of the current thesis (see Chapter 5), as participants with overweight and obesity presented higher scores on TFEQ disinhibition, TFEQ hunger and BES, with simultaneously lower scores on CoEQ craving control than lean participant counterparts. However, little evidence exists examining differences in eating behaviour traits between sweet liker phenotype groups. Improvements were observed in both phenotype groups between baseline and week 2 on CoEQ craving control and sweet cravings specifically. In the context of a dietary weight loss intervention, these significant changes occurred too rapidly to be a consequence of weight loss. Review of the wider literature demonstrated that avoiding specific foods in the short-term (1-14 days) tends to result in an increase in cravings (Meule, 2020b). However, it is important to note that none of the studies reviewed imposed instructions on total energy intake and only restricted specific foods. Therefore, the food deprivation instructions in these studies did not result in a nutrient deficiency, highlighting that perceived deprivation – a feeling of not eating as much of a food as one would like to – is an important factor in determining cravings. It is therefore possible that when participants retain a level of autonomy with their food selection choices, that specific cravings may be satisfied more readily. Moreover, multiple reviews of studies that employ energy-restriction diets demonstrate a reduction to cravings (i.e., improvement) (Kahathuduwa et al., 2017; Meule, 2020b; Oustric et al., 2018). However, these reviews involve intervention periods ranging from 4 weeks to 2 years, therefore the findings of Chapter 5 represent novel findings, demonstrating improvements to cravings over a 2 week period.

The present thesis provides evidence that sweet food preferences are associated with a number of eating behaviour traits which are thought to be illuminative of a loss of control around food. ‘Uncontrolled eating’ has been created as an umbrella term representing the intersection of different but correlated eating behaviour traits (Vainik et al., 2019). However, the separate questionnaires may still be informative, with some measuring aspects of a loss of control around food (e.g., binge eating scale) whereas others attempt to illuminate the reasons behind the loss of control (e.g., three-factor eating questionnaire or the control of eating questionnaire). Despite this, the strong correlations between questionnaires (e.g., Price et al., 2015 or Vainik et al., 2015) suggests that participants do not adequately differentiate between the reasons for overconsumption. Moreover, when a new questionnaire is developed and proposed to be measuring a new construct, it is validated against currently existing questionnaires (e.g., Gearhardt et al., 2009). This may represent the ‘jangle fallacy’ in which an erroneous assumption is made that two almost identical things are different because they are labelled as such. It has been proposed that the jangle fallacy is common within obesity research (Vainik et al., 2015; Vainik & Meule, 2018). Furthermore, examination of the overlapping eating behaviour traits highlights patterns with phenotypic, genetic and environmental associations (Vainik et al., 2019). Therefore given the issues present in eating behaviour trait assessment, it is concluded from the current findings that a sweet food preference demonstrates associations with a number of eating behaviour traits which may be characterised by a loss of control around food intake.

9.5 Sweet liker phenotype

The present method of defining sweet liker phenotype, which categorised individuals as high or low-sweet likers through the use of a computer-based questionnaire (LFPQ) may be reasoned to be a more ecologically valid method than those methods previously employed within the literature. Participants provided EL ratings of common high fat and low fat sweet foods - foods which were screened prior to the commencement of the study for their acceptability, and is therefore more in line with preferences and eating behaviours in real life away from the lab environment. It is clear that current classification systems within the literature of sweet liker

phenotypes are lacking (Iatridi et al., 2019b, 2019a), which may be contributing to a level of incongruence within the literature and is therefore detrimental to our understanding of how sweet liker status impacts appetite and eating behaviours. The method employed in the current study represents a novel approach, an advantage of which is the lack of limitation in individual variability in taste sensitivity due to the omission of stimuli presentation (which common methods of sweet liker phenotyping typically consists of). However, it is limited by the manner in which it is only able to provide a dichotomised variable of sweet phenotypes, as the method employed utilised sweet relative to savoury preference within the LFPQ, employing a pre-defined cut-off point to distinguish low and high sweet liker groups. Although the extent to which there are multiple phenotypes remains inconclusive, this data could also be used to view sweet liker phenotype as a continuous variable. Given the findings of Chapter 4 which highlighted the stability of sweet preferences across prolonged periods, the day-to-day variability should be minimal, but may be possible to be captured through the use of a continuous variable.

9.5.1 LFPQ use in the assessment of sweet liker phenotype

When assessing food preferences visual presentation is an important factor as visualisation provides information on the edibility, palatability and satiating properties of a food (de Bruijn et al., 2017). Viewing a food enables the awareness of availability and potential palatability (Lieberman, 2006). As such, tasks utilising images of food, as opposed to presenting the food in person, are demonstrated to be a reliable and valid method of assessing food preferences (de Bruijn et al., 2017). However, the accuracy of the test is dependent on the quality and familiarity of food images included (Oustric et al., 2020) – an issue which is addressed within the present thesis during screening sessions. Therefore, the method of sweet food preference assessment employed in the current thesis is a reliable and valid method and can be employed in the phenotyping of sweet liker status.

9.5.2 Dichotomisation of sweet liker phenotype groups

Within the present thesis the variable of sweet liker phenotype has been dichotomised, using high and low sweet liker types. Similarly, early work regarding sweet liker phenotypes

employed a method of phenotyping participants as a dichotomous variable (Drewnowski et al., 1997; Drewnowski & Schwartz, 1990; Yeomans et al., 2007), although more contemporary work utilising hierarchical cluster analysis (HCA) has identified multiple phenotype groups (Iatridi et al., 2019a - outlined in Chapter 1). Within the contemporary work are two groups of interest - a sweet liker group who display an increased liking for solutions increasing in intensity, as well as an inverted U-shaped group, displaying a similar increase in liking with increasing intensity, which plateaus and declines after reaching a maximal point (Iatridi et al., 2019b). However, during the final stage of the HCA process the inverted-U shape phenotype merged with the sweet-liker group, demonstrating a degree of commonality between the groups. This may be due to the methods typically employed to investigate sweet phenotypes, which involves the presentation of multiple stimuli varying in concentration. It may therefore be the reality that all sweet likers will reach a maximal sweetness intensity point whereby liking will begin to decline, an inflection point which varies amongst individuals and is currently higher than the most intense sweet solutions provided in current studies. Indeed further evidence has concluded the existence of an extreme-sweet liker phenotype group suggesting that there is a wide degree of variation in the maximum sweet concentration levels amongst individuals. A review of 71 studies identified four main phenotyping techniques, each of which involved presentation of stimuli varying in sweetness intensity (Iatridi et al., 2019a). However, it may be argued that this is not a true representation of sweet liking as in real life individuals rarely are provided the opportunity to manipulate the sweetness intensity of food products, particularly if these products are purchased away from the home. Therefore, it may be reasoned that maximum tolerance levels and overall liking are not equivalent concepts in the assessment of food preferences. For example, the LFPQ is a method of assessment which provides images of commonly consumed foods, rather than presenting sweet solutions that vary in intensity which is unrepresentative of sweet consumption in everyday life.

Moreover, previous work has defined a sweet disliker (i.e., low liker in the current thesis) either as a monotonically decreasing liking with increasing concentrations of sweetness (Drewnowski et al., 1997), or a liking for moderate levels of sweetness (i.e. an inverted-U shape) (Methven et

al., 2016). Elsewhere these two groups are categorised into a single group (Yeomans et al., 2007) or more recently defined as separate sub-groups (Iatridi et al., 2019b). Within the present thesis a low sweet liker was defined as an individual presenting with a higher savoury relative to sweet preference. Due to claims that sweet foods are universally rewarding (Blundell, 1991), it is reasoned that a sweet disliker phenotype may be a misrepresentation of the reality of sweet preferences. Due to the manner of assessment (i.e., visual food image presentation) and the manner in which sweet liking is operationalised, the LFPQ can be reasoned to provide a greater representation of real-world sweet liking in this manner.

9.6 Expression of sweet food preferences

Within Chapter 5 it was demonstrated that a sweet preference may not present as a barrier to weight loss which is supportive of findings provided by a recent review in which sweet liking was shown to not be a major risk factor for obesity (Armitage et al., 2021). However, it has been previously suggested that individuals with a larger body size also present with an increased liking for sweetness (Ettinger et al., 2012). Elsewhere it has been suggested that the differences observed between individuals differing by body weight and composition in their sweetness preference is dependent on the fat content of the stimulus (van Langeveld et al., 2018), with HFSW foods preferred by individuals with increasing levels of body fatness (Drewnowski, 1997, 2007; Drewnowski & Greenwood, 1983). Furthermore, a preference for natural sweetness has been demonstrated to be protective of obesity (Lampuré et al., 2016) as well as type-II diabetes mellitus (Lampuré et al., 2019). The combination of findings therein serves to demonstrate the numerous manifestations of a sweet food preference, as well as the variation in the effects of these preferences.

The LFPQ may be limited in that it only contains two macronutrient-based categories, divided into sweet and savoury taste preferences (with the inclusion of high and low fat). The current version of the LFPQ represents a development on the original version which only utilised high and low fat foods (Finlayson et al., 2007a), with later studies adapting this to include high and low protein (Griffioen-Roose et al., 2011) and high and low energy (Zoon et al., 2014). The

Macronutrient and Taste Preference Ranking Task (MTPRT) on the other hand, was developed as a means of assessing a full range of macronutrients (de Bruijn et al., 2017) which can consequently be used in a wider range of studies. The MTPRT is similar to the LFPQ in that it is a computer-based assessment, can be utilised for both hypothesis-driven and exploratory studies to examine the influence of a range of factors on changes in food preferences (de Bruijn et al., 2017).

Therefore, it is suggested that an examination of an overall sweet food preference in the context of obesity, or as a phenotype in the study of obesity, may be insufficient. Indeed, sweet foods consist of a wide variety of foods, naturally occurring fruits that are high in fructose are sweet, although these same fruits contain high levels of micronutrients and fibres, resulting in different effects to so-called ultra-processed sweet foods, such as candy/sweets, sugar sweetened beverages, which in turn should not be categorised as the same as sweetened fats. The expression of this preference should not be overlooked as it may be highly influential in determining the extent to which this preference will be protective or destructive in health related outcomes. Findings provided within Chapter 6 of the current thesis highlights the associations between sweet food preferences and numerous eating behaviour traits which may increase an individual's susceptibility to over consumption, whereas findings provided in Chapter 5 which demonstrate successful weight loss via two interventions, highlight the manner in which it is challenging to state whether a sweet food preference is undesirable in the context of obesity. The reality of food preferences is that they are nuanced and require careful consideration.

9.7 Effect of sweetener type and subsequent sweet food preferences

Highlighted within Chapter 2, the preponderance of literature available regarding HIS types favours a select few (also highlighted within (Higgins & Mattes, 2019)) and the available evidence for reformulated solid matrices is sparse. The second study within the present thesis provided an opportunity to address this limitation of the literature, employing a protocol

utilising both a natural and an artificial form of HIS, as well as sucrose, in a reformulated intervention product (i.e., a jam filled biscuit).

It has been claimed that consumption of HIS serves to promote sweet cravings and encourage subsequent intake (Yang, 2010), However findings in Chapters 2, 7 and 8 serve to dispel this concern. Review of the available literature of RCTs, although limited in that the current literature primarily employs beverages as the mode of ingestion, demonstrated that consumption of HIS does not promote either *ad libitum* or free-living energy intake. Within the second study of this thesis, there was no effect of sweetener condition on cravings, and instead there were notable reductions in sweet food preferences, which did not differ as a function of sweetener condition. Taken together these findings suggest that HIS do not promote sweet cravings or future intake, instead findings of Chapter 7 and 8 suggest that repeated consumption for 14-days instead decreases sweet food preferences. The lack of difference across sweetener conditions suggests that the influential factor is the consumption of a sweet food in and of itself, with the sweetener type of less importance.

Moreover, sweeteners are perceived differently by individuals in their sweetness intensity and taste profile (Kamerud & Delwiche, 2007; Muenprasitvej et al., 2022; Schiffman et al., 1979). The existence of sweet liker phenotypes demonstrates the range in differences in preference for sweet foods (Bartoshuk, 1979; Gent & Bartoshuk, 1983; Looy & Weingarten, 1992). Previous evidence which has attempted to examine potential phenotypic variance in sweetener types has failed to also examine differences in the effect on subsequent preferences. One study through multivariate modelling identified similar heritability in the sweetness perception of two sugars and two HIS between phenotype groups. Greater than 75% of genetic variance was accounted for by a common genetic factor, thereby indicating that individual differences in the perception of sweetness may be attributable to a single set of genes (Hwang et al., 2015). Another study demonstrated similar liking ratings between phenotype groups for sucrose and HIS sweetened beverages (Yang et al., 2019). However, this is the first study to date to examine potential differences between phenotype groups in the effect on subsequent food preferences. Within Chapter 8 it was demonstrated that the lack of effect of sweetener condition on subsequent

sweet food preferences did not differ between phenotype groups. However, high sweet likers displayed a greater reduction in EL and EW sweet bias following acute consumption of the intervention product. This represents a preliminary finding, as it is the first of its kind to demonstrate that high and low sweet liker phenotype groups display different changes to subsequent sweet food preferences following acute and repeated consumption of a sweet stimuli, which is not impacted by the sweetener type. Due to being preliminary findings this effect should be investigated in more depth in future.

9.8 Issues Regarding Sweetener use in Processed Foods

Food processing involves any method or process that alters a food from its raw state, this includes cooking, seasoning, preserving or combining with other foods and has been part of the human diet for thousands of years (Carretero et al., 2020). With the introduction of affordable and efficient machinery, food processing became industrialised. However, there is a growing concern that there may be a cost of this to public health (Petrus et al., 2021), despite many consumers understanding the potential benefits of food processing (Lazarides, 2012; Sadler et al., 2021), such as fortified foods for individuals with nutrient deficiencies (Martínez-Navarrete et al., 2002). Nonetheless, the consumption of processed foods has been systematically criticized over the previous decade, with the underlying assumption that processed foods are dilute in nutrient content compared to homemade dishes (Petrus et al., 2021). Current evidence however highlights that future research is required to establish whether associations present between obesity with ultra-processed food intake are due to the processing itself, or the nutrient content of the foods (Poti et al., 2017).

Currently, the NOVA classification system divides foods into four groups on the basis of their degree of processing (Monteiro et al., 2019). Consequently, according to the NOVA classification of so-called ultra-processed foods (Monteiro et al., 2010) reformulated products utilising HIS fall under this category. However, this presents an issue as the term ultra-processed has been adopted by conventional press and social media with negative connotations (Knorr & Watzke, 2019). Ultra-processed foods in the NOVA classification refers to reformulations containing five or more ingredients (Monteiro et al., 2019), which in the current

food environment is common both in home recipes and industrial products (Sacchi et al., 2019) and so this may be an overly broad definition. Moreover, a recent systematic review commissioned by the WHO led to a draft recommendation that HIS should not be used as a means of achieving weight control or reducing the risk of non-communicable diseases (World Health Organization, 2022a). The systematic review and meta-analysis demonstrated the facilitative capacity of HIS products in reducing energy intake and assisting in modest weight loss, highlighted in RCTs (Rios-Leyvraz & Montez, 2022) and thereby supporting findings of previous reviews (Laviada-Molina et al., 2020; Lee et al., 2021; Miller & Perez, 2014; Rogers et al., 2016) as well as the findings demonstrated in Chapter 2. However, the review also highlighted that cohort studies do not demonstrate significant long term reductions to body weight with the consumption of HIS. The WHO recommendations state that for this reason, there is not sufficient evidence to support the use of HIS consumption in the diet as a means of controlling or reducing energy intake to aid in weight loss attempts.

However, the current recommendations to avoid the use of HIS and reformulated foods is made in light of associations between obesity related comorbidities such as type-II diabetes mellitus or cardiovascular disease with HIS consumption in cohort studies, which may be subject to reverse causality. Indeed, a number of studies demonstrate a positive association between HIS consumption and obesity (Fowler et al., 2008, 2015; Stellman & Garfinkel, 1986). For example, a cohort study of 1,454 participants consuming HIS displayed a significantly increased BMI and increased waist circumference when compared to non-users (Chia et al., 2016). Nonetheless, the mechanism by which HIS ingestion would promote obesity and related co-morbidities cannot be illuminated in controlled studies. This evidence cannot state a causal effect of HIS consumption, and so the possibility that this association is an example of reverse causation cannot be ignored. The recommendations of the WHO to avoid consumption of HIS products appears to be overly influenced by evidence provided by cohort studies – which fail to demonstrate long term reductions in energy intake and significant weight loss. However, cohort studies are observational in nature and the variables of interest (in this instance HIS intake and obesity rates) have not undergone manipulation by a researcher, whilst this provides an

understanding of the correlates of HIS consumption under strong ecologically valid conditions, there remains a degree of uncertainty around data obtained, creating an inability to draw definitive conclusions. Therefore research providing a focus on potential mechanisms of action explaining associations are needed.

Randomised controlled trials provide a greater degree of control around variables as well as providing manipulation of variables of interest, as such controlled trials are able to indicate causation, when present. For example, within Chapter 2 - a review of controlled trials - it was identified that consumption of HIS did not impact energy intake, in addition to Chapter 7 and Chapter 8 demonstrating no effect of sweetener condition on dietary recall, or sweet cravings. These findings taken together lead to the conclusion that HIS does not seem to promote energy intake and consequently it remains necessary to illuminate precisely how HIS consumption may contribute to obesity levels. The findings of controlled trials provide a clearer understanding of the precise effects of HIS consumption, due to the stricter control over variables. The findings of the present thesis do not support the recommendations to avoid HIS consumption, as the findings indicate that sweetener type is of limited importance in influencing sweet food preferences and eating behaviours.

9.8.1 NOVA classification system

The NOVA classification system uses ‘industrial processes’ as the crucial determinant of food and diet quality and as such ignores previously used conventional groups, such as cereals and cereal products, or meats and meat products (Monteiro & Cannon, 2012). It is therefore necessary to examine the NOVA classification system, questioning whether foods which have undergone an element of industrial processing are inherently detrimental to health or whether the system is limited in its application.

This approach relies upon the premise that ultra-processed foods are by definition detrimental to health, and reformulation cannot improve them (Gibney, 2019). By extension, this definition includes reformulated food products that have the capacity to facilitate a reduction in energy intake and aid in weight loss attempts – as demonstrated within Chapter 2. In addition to

reformulated, or energy reduced products, this classification system also includes nutrient fortified foods which have been demonstrated as having a significant beneficial effect on health related outcomes (Cormick et al., 2021) as well as cognitive function (Cohen et al., 2020).

Despite evidence demonstrating that so called ultra-processed foods are associated with negative health outcomes (Elizabeth et al., 2020), as outlined in section 1.7.2 of Chapter 1, a single nutrient or food focused approach is overly narrow in its approach. The Australia paradox demonstrates that a narrow approach to understanding the effects of foods and nutrients on obesity levels is inaccurate, and as such it is necessary to adopt a whole diet based approach. Moreover, the findings of Chapter 7 and Chapter 8 indicate that sweetener type is of less importance when considering the effects of sweet food intake. Therefore, it is suggested that the NOVA classification system is limited, as defining foods that have undergone an element of industrial processing as inherently unhealthy is misleading.

9.9 Strengths and limitations of this thesis

The two studies utilised within the current thesis provide an examination of sweet food preferences in response to dietary induced weight loss, as well as the effect of acute and repeated ingestion of different types of sweetener in a solid food matrix. However, the value of the results presented and the conclusions drawn must be made with an awareness of the relative strengths and limitations in the study protocols employed.

9.9.1 Protocol Limitations in both studies

As briefly mentioned, the LFPQ is unable to categorise sweet food preferences beyond an overall preference and high or low fat sweet preferences. Given that not all sweet foods within these LFPQ categories are equivalent - e.g., images of fruit and images of candy are both included as LFSW foods, but previous evidence has demonstrated that fruits may be categorised as a distinct form of sweet preference (Lampuré et al., 2016, 2019) - it is reasonably possible to make a distinction between types of sweet preferences that are not accurately reflected in the current measure. Future work in this area may wish to consider types of sweet foods beyond high fat and low fat sweet.

Within the wider literature, the existence of associations between sweet food intake and eating behaviour traits has been demonstrated. However, food intake remains only a proxy indication of sweet food preferences, with just 15.3% of women stating pleasure as the reason for food selection (McGill & Appleton, 2009), it is evident that there are other variables influencing food selection and intake than merely preferences. Thus when reviewing the available literature surrounding the associations between preferences and behavioural traits, caution must be employed when considering studies examining food intake as an outcome rather than an overt measure of preference. Additionally, study 1 utilised a female only sample, due to food preference differences observed between males and females (van Langeveld et al., 2018) and so these findings may not be applicable to males. Current literature has provided examination of eating behaviour traits in women (Bryant, 2001; Dalton et al., 2013a; French et al., 1994) with findings highlighting differences between male and female participants. Furthermore, the possibility of a bi-directional association between sweet food preferences and eating behaviour traits cannot be disregarded. However, currently there is insufficient evidence examining the direction of this association or considering the extent to which eating behaviour traits influence food preferences. And so it cannot be ruled out that trait binge eating, craving control, or trait restraint, disinhibition or hunger are capable of influencing sweet food preferences.

As outlined in section 9.5, the current method of sweet liker phenotyping represents a novel technique. A limitation of the current sweet liker phenotype classification is suggested in that it may present an over simplification of phenotypes. Recent evidence has demonstrated an additional two phenotype groups, namely an inverted-U shape and non-sensitivity response group (Iatridi et al., 2019b). Therefore categorising individuals as high and low sweet likers may represent a limitation in the current methodology, however establishing further phenotype groups was beyond the scope of the current analysis and was not possible due to the measurement of sweet liking in the LFPQ. Nonetheless, previous evidence has considered sweet phenotypes as a dichotomous variable (Looy & Weingarten, 1991) with more recent evidence demonstrating the possibility to dichotomise the additional groups identified in hierarchical cluster analysis (Kim et al., 2017). Moreover, not all non-dichotomised phenotype

study's findings are replicated (Iatridi et al., 2019b), with some studies treating the inverse-U shape response as an outlier (Drewnowski et al., 1997; Drewnowski & Schwartz, 1990; Yeomans et al., 2007). Additionally in Iatridi's (2019) hierarchical cluster analysis, sweet likers merge with the inverse-U shape response pattern at the final stage of the process, thereby demonstrating a strong degree of resemblance between the sweet likers and the inverse-U response pattern.

Within both studies exploratory analyses were conducted, which increases the probability of a type-I error occurring (Othus et al., 2022), and as such should be interpreted as non-definitive. Additionally, the exploratory results presented within the present thesis should be used to aid future work in hypothesis generation.

9.9.2 Protocol strengths in both studies

Both studies in the current thesis were carefully designed to measure a range of variables associated with sweet food preferences with careful consideration applied to potential limitations in the quality of the data obtained. In the first study (DIVA) participants were age matched during recruitment and in study 2 (SWEET) a stratified recruitment method was employed, consequently potential age (Desor & Beauchamp, 1987; Yoshinaka et al., 2016) and gender (van Langeveld et al., 2018) related effects on food preferences were mitigated. Additionally, all participants in both studies attended screening sessions prior to their first clinical investigation days. During these sessions food images in the LFPQ were screened to ensure foods used were not disliked and would be freely chosen. In the event that participants disliked and would not typically consume a food, an appropriate substitute was used. However, the data in study 2 (SWEET) were collected from two sites with different cultures. To navigate the cultural differences in food types, culturally appropriate images were used in both sites (see appendix 9).

Furthermore, when examining sweet liker phenotype groups, there remains the question of whether a high sweet liker is always a sweet liker (and the same for low sweet likers), however within the current thesis (Chapter 4) sweet preferences were demonstrated to be a stable trait,

serving to alleviate this concern. Although a sweet preference does display some variation throughout the day, intraclass correlation coefficients demonstrated strong stability across a period of weight loss. Therefore the extent to which an individual's sweet liker phenotype may change from pre- to post-weight loss within the current data analysis remains unlikely.

9.9.3 Limitations to Study 1: DIVA

One limitation of the present study is that whilst those in the CER group were provided pre-portioned foods for all days, those in the IER received food packs for only the fast days. However, to somewhat mitigate this, both groups received food packs (LighterLife). The macronutrient content of each diet differed but was similar to those employed during clinical or commercial weight-loss interventions. Although it cannot be ruled out that this may have impacted results as the food packs represented unfamiliar foods (Nacef et al., 2019; Tuorila et al., 2001). Moreover, it was not possible to account for any effects produced by the menstrual cycle as the post-intervention measures day was scheduled after 5% weight-loss occurred or at 12 weeks. Although food cravings may differ during stages of the menstrual cycle, this does not necessarily impact food intake or anthropometric measures (Souza et al., 2018). Future studies may also wish to increase the sample size, as the current dataset represents a small sample and thus any conclusions drawn must be made with caution. An additional limitation of the protocol employed was that a female only sample was utilised in part to avoid gender presenting a confounding variable due to physiological differences, but menopausal state and menstrual cycle were variables that were not controlled for during recruitment. Taste preferences have been demonstrated to vary across the menstrual cycle (Bowen & Grunberg, 1990; Sevim & Yağar, 2022) and so represents a confounding variable in the investigation of the stability of food preferences.

9.9.4 Limitations to Study 2: SWEET

In study 2 (SWEET) 24 hour dietary recall was used with an awareness of the caveats of this method for measuring energy and nutrient intake. The accuracy of any energy and nutrient intake information provided by participants is contingent on the reliability of the information

provided. Retrospective methods of dietary intake assessment are therefore flawed due to a reliance on the participant's memory of the diet, and as such may not accurately measure the diet itself (Krall et al., 1988). Self-reported dietary recall is also impacted by social desirability bias, and subsequently the validity of the measure is dependent on the accurate and honest recall by participants (Hebert et al., 1995).

Moreover, attendance at the lab represented an atypical day for participants and the potential that this impacted eating behaviours cannot be dismissed. Moreover, at the time of writing the intervention products were not unblinded and so the energy and macronutrient compositions of the biscuits was not included in the data analysis.

It was not possible to examine the acute effects of different sweetener types on subsequent cravings. The CoEQ was completed by participants prior to consumption of the intervention products when in the lab, consequently only the effect of repeated consumption was possible to examine. This therefore represents a limitation in the study protocol employed, which may be a point to be considered in future work.

9.10 Concluding Remarks

The present thesis has demonstrated that sweet food preferences are highly stable, however the time of assessment should be carefully considered in future work as the time of day and composition of meals influences these preferences. Therefore when characterising individuals as sweet likers, it can confidently be stated that a high sweet liker (and vice versa a low sweet liker) remains as such over prolonged periods of time. However, preferences were shown to not change during dietary weight loss, which disagrees with the preponderance of literature.

Considering the current findings with the wider literature it may be concluded that any changes to sweet food preferences may occur as a consequence of weight loss, with the method of weight loss being of less importance. Moreover, reformulated products may be a viable means of facilitating a reduction in energy density of the diet whilst maintaining the palatability of foods which is desirable for the consumer. This was demonstrated in Chapters 2, 7 and 8 and it appears that the type of sweetener does not impact subsequent sweet preferences, rather it is the

consumption of a sweet stimulus in and of itself that exerts an influence. However, it should be stated that reformulated products do not guarantee a reduction in energy density of the diet, particularly in solid foods, and if being used as a means of weight loss should be employed alongside alternative weight reduction strategies.

References

- Abete, I., Astrup, A., Martínez, J. A., Thorsdottir, I., & Zulet, M. A. (2010). Obesity and the metabolic syndrome: Role of different dietary macronutrient distribution patterns and specific nutritional components on weight loss and maintenance. *Nutrition Reviews*, 68(4), 214–231. <https://doi.org/10.1111/j.1753-4887.2010.00280.x>
- Adolphus, K., Lawton, C. L., & Dye, L. (2013). The effects of breakfast on behavior and academic performance in children and adolescents. *Frontiers in Human Neuroscience*, 7. <https://doi.org/10.3389/fnhum.2013.00425>
- Aguirre, T. M., Kuster, J. T., & Koehler, A. E. (2017). Relationship Between Eating Behavior and Dietary Intake in Rural Mexican-American Mothers. *Journal of Immigrant and Minority Health*, 19(1), 225–227. <https://doi.org/10.1007/s10903-015-0324-8>
- Akella, G. D., Henderson, S. A., & Drewnowski, A. (1997). Sensory acceptance of Japanese green tea and soy products is linked to genetic sensitivity to 6-*n*-propylthiouracil. *Nutrition and Cancer*, 29(2), 146–151. <https://doi.org/10.1080/01635589709514616>
- Albar, S. A., Alwan, N. A., Evans, C. E. L., Greenwood, D. C., & Cade, J. E. (2016). Agreement between an online dietary assessment tool (myfood24) and an interviewer-administered 24-h dietary recall in British adolescents aged 11–18 years. *British Journal of Nutrition*, 115(9), 1678–1686. <https://doi.org/10.1017/S0007114516000593>
- Alhamdan, B. A., Garcia-Alvarez, A., Alzahrnai, A. H., Karanxha, J., Stretchberry, D. R., Contrera, K. J., Utria, A. F., & Cheskin, L. J. (2016). Alternate-day versus daily energy restriction diets: Which is more effective for weight loss? A systematic review and meta-analysis: Alternate-day fasting versus daily energy restriction. *Obesity Science & Practice*, 2(3), 293–302. <https://doi.org/10.1002/osp4.52>
- Almiron-Roig, E., Chen, Y., & Drewnowski, A. (2003). Liquid calories and the failure of satiety: How good is the evidence? *Obesity Reviews*, 4(4), 201–212. <https://doi.org/10.1046/j.1467-789X.2003.00112.x>
- Altun, H., Hanci, D., Altun, H., Batman, B., Serin, R. K., Karip, A. B., & Akyuz, U. (2016). Improved Gustatory Sensitivity in Morbidly Obese Patients After Laparoscopic Sleeve Gastrectomy. *Annals of Otolaryngology, Rhinology & Laryngology*, 125(7), 536–540. <https://doi.org/10.1177/0003489416629162>
- Anderson, Cohen, P., Naumova, E. N., & Must, A. (2006). Association of Depression and Anxiety Disorders With Weight Change in a Prospective Community-Based Study of Children Followed Up Into Adulthood. *Archives of Pediatrics & Adolescent Medicine*, 160(3), 285. <https://doi.org/10.1001/archpedi.160.3.285>
- Anderson, G. H., Foreyt, J., Sigman-Grant, M., & Allison, D. B. (2012). The Use of Low-Calorie Sweeteners by Adults: Impact on Weight Management. *The Journal of Nutrition*, 142(6), 1163s–1169s. <https://doi.org/10.3945/jn.111.149617>
- Anderson, P. M., & Butcher, K. F. (Kristin F. (2006). Childhood Obesity: Trends and Potential Causes. *The Future of Children*, 16(1), 19–45. <https://doi.org/10.1353/foc.2006.0001>
- Andriessen, C., Christensen, P., Vestergaard Nielsen, L., Ritz, C., Astrup, A., Meinert Larsen, T., Martinez, J. A., Saris, W. H. M., van Baak, M. A., Papadaki, A., Kunesova, M., Jebb, S., Blundell, J., Lawton, C., & Raben, A. (2018). Weight loss decreases self-reported appetite and alters food preferences in overweight and obese adults: Observational data from the DiOGenes study. *Appetite*, 125, 314–322. <https://doi.org/10.1016/j.appet.2018.02.016>
- Angotzi, A. R., Puchol, S., Cerdá-Reverter, J. M., & Morais, S. (2020). Insights into the Function and Evolution of Taste 1 Receptor Gene Family in the Carnivore Fish

- Gilthead Seabream (*Sparus aurata*). *International Journal of Molecular Sciences*, 21(20), 7732. <https://doi.org/10.3390/ijms21207732>
- Appleton, K. M., & Blundell, J. E. (2007). Habitual high and low consumers of artificially-sweetened beverages: Effects of sweet taste and energy on short-term appetite. *Physiology & Behavior*, 92(3), 479–486. <https://doi.org/10.1016/j.physbeh.2007.04.027>
- Appleton, K. M., & Conner, M. T. (2001). Body weight, body-weight concerns and eating styles in habitual heavy users and non-users of artificially sweetened beverages. *Appetite*, 37(3), 225–230. <https://doi.org/10.1006/appe.2001.0435>
- Appleton, K. M., Rogers, P. J., & Blundell, J. E. (2004). Effects of a sweet and a nonsweet lunch on short-term appetite: Differences in female high and low consumers of sweet/low-energy beverages. *Journal of Human Nutrition and Dietetics*, 17(5), 425–434. <https://doi.org/10.1111/j.1365-277X.2004.00548.x>
- Appleton, K. M., Tuorila, H., Bertenshaw, E. J., de Graaf, C., & Mela, D. J. (2018). Sweet taste exposure and the subsequent acceptance and preference for sweet taste in the diet: Systematic review of the published literature. *The American Journal of Clinical Nutrition*, 107(3), 405–419. <https://doi.org/10.1093/ajcn/nqx031>
- Archer, E., Hand, G. A., & Blair, S. N. (2013). Validity of U.S. Nutritional Surveillance: National Health and Nutrition Examination Survey Caloric Energy Intake Data, 1971–2010. *PLoS ONE*, 8(10), e76632. <https://doi.org/10.1371/journal.pone.0076632>
- Armitage, R. M., Iatridi, V., & Yeomans, M. R. (2021). Understanding sweet-liking phenotypes and their implications for obesity: Narrative review and future directions. *Physiology & Behavior*, 235, 113398. <https://doi.org/10.1016/j.physbeh.2021.113398>
- Armitage, R., Vi, C., Iatridi, V., & Yeomans, M. R. (2021). Understanding sweet-liking phenotypes: Exploring habitual intake of Western diet, impulsivity and childhood eating experience. *Appetite*, 157, 104939. <https://doi.org/10.1016/j.appet.2020.104939>
- Asano, M., Hong, G., Matsuyama, Y., Wang, W., Izumi, S., Izumi, M., Toda, T., & Kudo, T. (2016). Association of Oral Fat Sensitivity with Body Mass Index, Taste Preference, and Eating Habits in Healthy Japanese Young Adults. *The Tohoku Journal of Experimental Medicine*, 238(2), 93–103. <https://doi.org/10.1620/tjem.238.93>
- Asao, K., Luo, W., & Herman, W. H. (2012). Reproducibility of the measurement of sweet taste preferences. *Appetite*, 59(3), 927–932. <https://doi.org/10.1016/j.appet.2012.09.002>
- Asao, K., Rothberg, A. E., Arcori, L., Kaur, M., Fowler, C. E., & Herman, W. H. (2016). Sweet taste preferences before and after an intensive medical weight loss intervention. *Obesity Science & Practice*, 2(2), 189–195. <https://doi.org/10.1002/osp4.33>
- Azad, M. B., Abou-Setta, A. M., Chauhan, B. F., Rabbani, R., Lys, J., Copstein, L., Mann, A., Jeyaraman, M. M., Reid, A. E., Fiander, M., MacKay, D. S., McGavock, J., Wicklow, B., & Zarychanski, R. (2017). Nonnutritive sweeteners and cardiometabolic health: A systematic review and meta-analysis of randomized controlled trials and prospective cohort studies. *Canadian Medical Association Journal*, 189(28), E929–E939. <https://doi.org/10.1503/cmaj.161390>
- Azaïs-Braesco, V., Sluik, D., Maillot, M., Kok, F., & Moreno, L. A. (2017). A review of total & added sugar intakes and dietary sources in Europe. *Nutrition Journal*, 16(1), 6. <https://doi.org/10.1186/s12937-016-0225-2>
- Baghurst, K. I., Baghurst, P. A., & Record, S. J. (1994). Demographic and dietary profiles of high and low fat consumers in Australia. *Journal of Epidemiology & Community Health*, 48(1), 26–32. <https://doi.org/10.1136/jech.48.1.26>
- Baker-Smith, C. M., de Ferranti, S. D., & Cochran, W. J. (2019). *The Use of Nonnutritive Sweeteners in Children*. 144(5), 18.

- Barclay, A. W., & Brand-Miller, J. (2011). The Australian Paradox: A Substantial Decline in Sugars Intake over the Same Timeframe that Overweight and Obesity Have Increased. *Nutrients*, 3(4), 491–504. <https://doi.org/10.3390/nu3040491>
- Barkeling, B., Andersson, I., Lindroos, A., Birkhed, D., & Rössner, S. (2001). Intake of sweet foods and counts of cariogenic microorganisms in obese and normal-weight women. *European Journal of Clinical Nutrition*, 55(10), 850–855. <https://doi.org/10.1038/sj.ejcn.1601237>
- Barragán, R., Coltell, O., Portolés, O., Asensio, E., Sorlí, J., Ortega-Azorín, C., González, J., Sáiz, C., Fernández-Carrión, R., Ordovas, J., & Corella, D. (2018). Bitter, Sweet, Salty, Sour and Umami Taste Perception Decreases with Age: Sex-Specific Analysis, Modulation by Genetic Variants and Taste-Preference Associations in 18 to 80 Year-Old Subjects. *Nutrients*, 10(10), 1539. <https://doi.org/10.3390/nu10101539>
- Bartolotto, C. (2015). Does Consuming Sugar and Artificial Sweeteners Change Taste Preferences? *The Permanente Journal*. <https://doi.org/10.7812/TPP/14-229>
- Bartoshuk, L. M. (1979). Bitter Taste of Saccharin Related to the Genetic Ability to Taste the Bitter Substance 6-*n*-Propylthiouracil. *Science*, 205(4409), 934–935. <https://doi.org/10.1126/science.472717>
- Bartoshuk, L. M. (2000). Comparing Sensory Experiences Across Individuals: Recent Psychophysical Advances Illuminate Genetic Variation in Taste Perception. *Chemical Senses*, 25(4), 447–460. <https://doi.org/10.1093/chemse/25.4.447>
- Bartoshuk, L. M., Duffy, V. B., Hayes, J. E., Moskowitz, H. R., & Snyder, D. J. (2006). Psychophysics of sweet and fat perception in obesity: Problems, solutions and new perspectives. *Philosophical Transactions of the Royal Society B: Biological Sciences*, 361(1471), 1137–1148. <https://doi.org/10.1098/rstb.2006.1853>
- Batra, P., Das, S. K., Salinardi, T., Robinson, L., Saltzman, E., Scott, T., Pittas, A. G., & Roberts, S. B. (2013). Relationship of cravings with weight loss and hunger. Results from a 6month worksite weight loss intervention. *Appetite*, 69, 1–7. <https://doi.org/10.1016/j.appet.2013.05.002>
- Beaulieu, K., Casanova, N., Oustric, P., Hopkins, M., Varady, K., Finlayson, G., & Gibbons, C. (2021). An exploratory investigation of the impact of ‘fast’ and ‘feed’ days during intermittent energy restriction on free-living energy balance behaviours and subjective states in women with overweight/obesity. *European Journal of Clinical Nutrition*, 75(3), 430–437. <https://doi.org/10.1038/s41430-020-00740-1>
- Beaulieu, K., Casanova, N., Oustric, P., Turicchi, J., Gibbons, C., Hopkins, M., Varady, K., Blundell, J., & Finlayson, G. (2020). Matched Weight Loss Through Intermittent or Continuous Energy Restriction Does Not Lead To Compensatory Increases in Appetite and Eating Behavior in a Randomized Controlled Trial in Women with Overweight and Obesity. *The Journal of Nutrition*, 150(3), 623–633. <https://doi.org/10.1093/jn/nxz296>
- Beaulieu, K., Hopkins, M., Blundell, J., & Finlayson, G. (2016). Does Habitual Physical Activity Increase the Sensitivity of the Appetite Control System? A Systematic Review. *Sports Medicine*, 46(12), 1897–1919. <https://doi.org/10.1007/s40279-016-0518-9>
- Beaver, J. D. (2006). Individual Differences in Reward Drive Predict Neural Responses to Images of Food. *Journal of Neuroscience*, 26(19), 5160–5166. <https://doi.org/10.1523/JNEUROSCI.0350-06.2006>
- Bello, J. K., Stulberg, D. B., Zhou, Y., & Wang, C. (2018). Physical Activity and Consumption Patterns of Reproductive-Aged Women by BMI Category. *Maternal and Child Health Journal*, 22(5), 713–724. <https://doi.org/10.1007/s10995-018-2440-2>

- Benedict, C., Brooks, S. J., O'Daly, O. G., Almèn, M. S., Morell, A., Åberg, K., Gingnell, M., Schultes, B., Hallschmid, M., Broman, J.-E., Larsson, E.-M., & Schiöth, H. B. (2012). Acute Sleep Deprivation Enhances the Brain's Response to Hedonic Food Stimuli: An fMRI Study. *The Journal of Clinical Endocrinology & Metabolism*, *97*(3), E443–E447. <https://doi.org/10.1210/jc.2011-2759>
- Bennett, E., Peters, S. A. E., & Woodward, M. (2018). Sex differences in macronutrient intake and adherence to dietary recommendations: Findings from the UK Biobank. *BMJ Open*, *8*(4), e020017. <https://doi.org/10.1136/bmjopen-2017-020017>
- Bernstein, E. E., Nierenberg, A. A., Deckersbach, T., & Sylvia, L. G. (2015). Eating behavior and obesity in bipolar disorder. *Australian & New Zealand Journal of Psychiatry*, *49*(6), 566–572. <https://doi.org/10.1177/0004867414565479>
- Berridge, K. C. (1996). Food reward: Brain substrates of wanting and liking. *Neuroscience & Biobehavioral Reviews*, *20*(1), 1–25. [https://doi.org/10.1016/0149-7634\(95\)00033-B](https://doi.org/10.1016/0149-7634(95)00033-B)
- 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. <https://doi.org/10.1016/j.brainres.2010.04.003>
- Berridge, K. C., & Kringelbach, M. L. (2015). Pleasure Systems in the Brain. *Neuron*, *86*(3), 646–664. <https://doi.org/10.1016/j.neuron.2015.02.018>
- Berridge, K. C., & Robinson, T. E. (2003). Parsing reward. *Trends in Neurosciences*, *26*(9), 507–513. [https://doi.org/10.1016/S0166-2236\(03\)00233-9](https://doi.org/10.1016/S0166-2236(03)00233-9)
- Bertéus Forslund, H., Torgerson, J. S., Sjöström, L., & Lindroos, A. K. (2005). Snacking frequency in relation to energy intake and food choices in obese men and women compared to a reference population. *International Journal of Obesity*, *29*(6), 711–719. <https://doi.org/10.1038/sj.ijo.0802950>
- Berthoud, H.-R. (2002). Multiple neural systems controlling food intake and body weight. *Neuroscience & Biobehavioral Reviews*, *26*(4), 393–428. [https://doi.org/10.1016/S0149-7634\(02\)00014-3](https://doi.org/10.1016/S0149-7634(02)00014-3)
- 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. <https://doi.org/10.1016/j.conb.2011.09.004>
- Berthoud, H.-R., Lenard, N. R., & Shin, A. C. (2011). Food reward, hyperphagia, and obesity. *American Journal of Physiology-Regulatory, Integrative and Comparative Physiology*, *300*(6), R1266–R1277. <https://doi.org/10.1152/ajpregu.00028.2011>
- Berthoud, H.-R., Münzberg, H., & Morrison, C. D. (2017). Blaming the Brain for Obesity: Integration of Hedonic and Homeostatic Mechanisms. *Gastroenterology*, *152*(7), 1728–1738. <https://doi.org/10.1053/j.gastro.2016.12.050>
- Berthoud, H.-R., & Zheng, H. (2012). Modulation of taste responsiveness and food preference by obesity and weight loss. *Physiology & Behavior*, *107*(4), 527–532. <https://doi.org/10.1016/j.physbeh.2012.04.004>
- Black, R. M., Leiter, L. A., & Anderson, G. H. (1993). Consuming aspartame with and without taste: Differential effects on appetite and food intake of young adult males. *Physiology & Behavior*, *53*(3), 459–466. [https://doi.org/10.1016/0031-9384\(93\)90139-7](https://doi.org/10.1016/0031-9384(93)90139-7)
- Black, R. M., Tanaka, P., Leiter, L. A., & Anderson, G. H. (1991). Soft drinks with aspartame: Effect on subjective hunger, food selection, and food intake of young adult males. *Physiology & Behavior*, *49*(4), 803–810. [https://doi.org/10.1016/0031-9384\(91\)90321-E](https://doi.org/10.1016/0031-9384(91)90321-E)
- Blumfield, M. L., Bei, B., Zimberg, I. Z., & Cain, S. W. (2018). Dietary disinhibition mediates the relationship between poor sleep quality and body weight. *Appetite*, *120*, 602–608. <https://doi.org/10.1016/j.appet.2017.10.022>

- Blundell. (1991). The biology of appetite. *Clin Appl Nutr*, 1, 21–31.
- Blundell. (2019). Low-calorie sweeteners: More complicated than sweetness without calories. *The American Journal of Clinical Nutrition*. <https://doi.org/10.1093/ajcn/nqz015>
- Blundell, J. (2018). Biology of the drive to eat. Implications for understanding human appetite and obesity. *Ernahrungs Umschau*, 126–133. <https://doi.org/10.4455/eu.2018.029>
- Blundell, J., de Graaf, C., Hulshof, T., Jebb, S., Livingstone, B., Lluch, A., Mela, D., Salah, S., Schuring, E., van der Knaap, H., & Westerterp, M. (2010). Appetite control: Methodological aspects of the evaluation of foods. *Obesity Reviews*, 11(3), 251–270. <https://doi.org/10.1111/j.1467-789X.2010.00714.x>
- 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. <https://doi.org/10.1016/j.physbeh.2004.04.021>
- Blundell, J. E., & Macdiarmid, J. I. (1997). Passive Overconsumption Fat Intake and Short-Term Energy Balance. *Annals of the New York Academy of Sciences*, 827(1 Lipids and Sy), 392–407. <https://doi.org/10.1111/j.1749-6632.1997.tb51850.x>
- Blundell, J., Stubbs, R., Golding, C., Croden, F., Alam, R., Whybrow, S., Lenoury, 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. <https://doi.org/10.1016/j.physbeh.2005.08.052>
- Boesveldt, S., Bobowski, N., McCrickerd, K., Maître, I., Sulmont-Rossé, C., & Forde, C. G. (2018). The changing role of the senses in food choice and food intake across the lifespan. *Food Quality and Preference*, 68, 80–89. <https://doi.org/10.1016/j.foodqual.2018.02.004>
- Bolhuis, D. P., Costanzo, A., & Keast, R. S. J. (2018). Preference and perception of fat in salty and sweet foods. *Food Quality and Preference*, 64, 131–137. <https://doi.org/10.1016/j.foodqual.2017.09.016>
- Bonnet, F., Tavenard, A., Esvan, M., Laviolle, B., Viltard, M., Lopicard, E. M., & Lainé, F. (2018). Consumption of a Carbonated Beverage with High-Intensity Sweeteners Has No Effect on Insulin Sensitivity and Secretion in Nondiabetic Adults. *The Journal of Nutrition*, 148(8), 1293–1299. <https://doi.org/10.1093/jn/nxy100>
- Booth, M. (2000). Assessment of Physical Activity: An International Perspective. *Research Quarterly for Exercise and Sport*, 71(sup2), 114–120. <https://doi.org/10.1080/02701367.2000.11082794>
- Borges, M. C., Louzada, M. L., de Sá, T. H., Laverty, A. A., Parra, D. C., Garzillo, J. M. F., Monteiro, C. A., & Millett, C. (2017). Artificially Sweetened Beverages and the Response to the Global Obesity Crisis. *PLOS Medicine*, 14(1), e1002195. <https://doi.org/10.1371/journal.pmed.1002195>
- Bowen, D. J., & Grunberg, N. E. (1990). Variations in food preference and consumption across the menstrual cycle. *Physiology & Behavior*, 47(2), 287–291. [https://doi.org/10.1016/0031-9384\(90\)90144-S](https://doi.org/10.1016/0031-9384(90)90144-S)
- Braude, L., & Stevenson, R. J. (2014). Watching television while eating increases energy intake. Examining the mechanisms in female participants. *Appetite*, 76, 9–16. <https://doi.org/10.1016/j.appet.2014.01.005>
- Bray, G. A., Paeratakul, S., & Popkin, B. M. (2004). Dietary fat and obesity: A review of animal, clinical and epidemiological studies. *Physiology & Behavior*, 83(4), 549–555. <https://doi.org/10.1016/j.physbeh.2004.08.039>
- Bray, G. A., & Popkin, B. M. (2014). Dietary Sugar and Body Weight: Have We Reached a Crisis in the Epidemic of Obesity and Diabetes?: Health Be Damned! Pour on the Sugar. *Diabetes Care*, 37(4), 950–956. <https://doi.org/10.2337/dc13-2085>

- British Nutrition Foundation. (n.d.). *Nutrition Requirements*.
https://www.nutrition.org.uk/attachments/article/907/nutrition%20requirements_revised%20oct%202017.pdf/
- Brown, R. J., & Rother, K. I. (2012). Non-Nutritive Sweeteners and their Role in the Gastrointestinal Tract. *The Journal of Clinical Endocrinology & Metabolism*, 97(8), 2597–2605. <https://doi.org/10.1210/jc.2012-1475>
- Brunstrom, J. M. (2011). The control of meal size in human subjects: A role for expected satiety, expected satiation and premeal planning. *Proceedings of the Nutrition Society*, 70(2), 155–161. <https://doi.org/10.1017/S002966511000491X>
- Bruyère, O., Ahmed, S. H., Atlan, C., Belegaud, J., Bortolotti, M., Canivenc-Lavier, M.-C., Charrière, S., Girardet, J.-P., Houdart, S., Kalonji, E., Nadaud, P., Rajas, F., Slama, G., & Margaritis, I. (2015). Review of the nutritional benefits and risks related to intense sweeteners. *Archives of Public Health*, 73(1). <https://doi.org/10.1186/s13690-015-0092-x>
- Bryant, E. J., Caudwell, P., Hopkins, M. E., King, N. A., & Blundell, J. E. (2012). Psychomarkers of weight loss. The roles of TFEQ Disinhibition and Restraint in exercise-induced weight management. *Appetite*, 58(1), 234–241. <https://doi.org/10.1016/j.appet.2011.09.006>
- Bryant, E. J., King, N. A., & Blundell, J. E. (2001). Disinhibition: Its effects on appetite and weight regulation. *Obesity Reviews*, 9(5), 409–419.
- Buchanan, K., & Sheffield, J. (2017). Why do diets fail? An exploration of dieters' experiences using thematic analysis. *Journal of Health Psychology*, 22(7), 906–915. <https://doi.org/10.1177/1359105315618000>
- Buckland, N. J., Swinnerton, L. F., Ng, K., Price, M., Wilkinson, L. L., Myers, A., & Dalton, M. (2021). Susceptibility to increased high energy dense sweet and savoury food intake in response to the COVID-19 lockdown: The role of craving control and acceptance coping strategies. *Appetite*, 158, 105017. <https://doi.org/10.1016/j.appet.2020.105017>
- Buemann, B., Toubro, S., & Astrup, A. (1998). D-Tagatose, a Stereoisomer of D-Fructose, Increases Hydrogen Production in Humans without Affecting 24-Hour Energy Expenditure or Respiratory Exchange Ratio. *The Journal of Nutrition*, 128(9), 1481–1486. <https://doi.org/10.1093/jn/128.9.1481>
- Bueter, M., Miras, A. D., Chichger, H., Fenske, W., Ghatge, M. A., Bloom, S. R., Unwin, R. J., Lutz, T. A., Spector, A. C., & le Roux, C. W. (2011). Alterations of sucrose preference after Roux-en-Y gastric bypass. *Physiology & Behavior*, 104(5), 709–721. <https://doi.org/10.1016/j.physbeh.2011.07.025>
- Burgess, B., Rao, S. P., & Tepper, B. J. (2016). Changes in liking for sweet and fatty foods following weight loss in women are related to prep phenotype but not to diet: Weight Loss and Taste. *Obesity*, 24(9), 1867–1873. <https://doi.org/10.1002/oby.21570>
- Burton, P., J. Smit, H., & J. Lightowler, H. (2007). The influence of restrained and external eating patterns on overeating. *Appetite*, 49(1), 191–197. <https://doi.org/10.1016/j.appet.2007.01.007>
- Cabanac, M. (1971). Physiological Role of Pleasure. *Science*, 173(4002), 1103–1107. <https://doi.org/10.1126/science.173.4002.1103>
- Cabanac, M. (1989). Palatability of Food and the Ponderostat. *Annals of the New York Academy of Sciences*, 575(1 The Psychobio), 340–352. <https://doi.org/10.1111/j.1749-6632.1989.tb53255.x>
- Cameron, J. D., Goldfield, G. S., Cyr, M.-J., & Doucet, É. (2008). The effects of prolonged caloric restriction leading to weight-loss on food hedonics and reinforcement. *Physiology & Behavior*, 94(3), 474–480. <https://doi.org/10.1016/j.physbeh.2008.02.014>

- Cameron, J. D., Goldfield, G. S., & Doucet, É. (2012). Fasting for 24h improves nasal chemosensory performance and food palatability in a related manner. *Appetite*, *58*(3), 978–981. <https://doi.org/10.1016/j.appet.2012.02.050>
- Cameron, J. D., Goldfield, G. S., Finlayson, G., Blundell, J. E., & Doucet, É. (2014). Fasting for 24 Hours Heightens Reward from Food and Food-Related Cues. *PLoS ONE*, *9*(1), e85970. <https://doi.org/10.1371/journal.pone.0085970>
- Canty, D. J., & Chan, M. M. (1991). Effects of consumption of caloric vs noncaloric sweet drinks on indices of hunger and food consumption in normal adults. *The American Journal of Clinical Nutrition*, *53*(5), 1159–1164. <https://doi.org/10.1093/ajcn/53.5.1159>
- Cappelleri, J. C., Bushmakin, A. G., Gerber, R. A., Leidy, N. K., Sexton, C. C., Lowe, M. R., & Karlsson, J. (2009). Psychometric analysis of the Three-Factor Eating Questionnaire-R21: Results from a large diverse sample of obese and non-obese participants. *International Journal of Obesity*, *33*(6), 611–620. <https://doi.org/10.1038/ijo.2009.74>
- Cardoso, P., Bolini, A., Maria, J., & Maria, H. (2008). Descriptive profile of peach nectar sweetened with sucrose and different sweeteners. *Journal of Sensory Studies*, *23*(6), 804–816. <https://doi.org/10.1111/j.1745-459X.2008.00187.x>
- Carnell, S., Gibson, C., Benson, L., Ochner, C. N., & Geliebter, A. (2012). Neuroimaging and obesity: Current knowledge and future directions: Neuroimaging and obesity. *Obesity Reviews*, *13*(1), 43–56. <https://doi.org/10.1111/j.1467-789X.2011.00927.x>
- Carretero, C., Clotet, R., & Colomer, Y., García de Fernando, G., Frías, J., Vaque, L. GRomero Rodriguez, M. (2020). Food Classification Report: The Concept Ultra-Processed. *Eur. Food & Feed L. Rev.*, *15*, 357.
- Carter, M., Albar, S., Morris, M., Mulla, U., Hancock, N., Evans, C., Alwan, N., Greenwood, D., Hardie, L., Frost, G., Wark, P., & Cade, J. (2015). Development of a UK Online 24-h Dietary Assessment Tool: Myfood24. *Nutrients*, *7*(6), 4016–4032. <https://doi.org/10.3390/nu7064016>
- Casanova, N., Beaulieu, K., Oustric, P., O'Connor, D., Gibbons, C., Blundell, J. E., Finlayson, G. S., & Hopkins, M. E. (2023). Increases in physical activity are associated with a faster rate of weight loss during dietary energy restriction in women with overweight and obesity. *British Journal of Nutrition*, *129*(8), 1451–1461. <https://doi.org/10.1017/S000711452200023X>
- Casperson, S. L., Johnson, L., & Roemmich, J. N. (2017). The relative reinforcing value of sweet versus savory snack foods after consumption of sugar- or non-nutritive sweetened beverages. *Appetite*, *112*, 143–149. <https://doi.org/10.1016/j.appet.2017.01.028>
- Castellanos, E. H., Charboneau, E., Dietrich, M. S., Park, S., Bradley, B. P., Mogg, K., & Cowan, R. L. (2009). Obese adults have visual attention bias for food cue images: Evidence for altered reward system function. *International Journal of Obesity*, *33*(9), 1063–1073. <https://doi.org/10.1038/ijo.2009.138>
- Catenacci, V. A., Pan, Z., Thomas, J. G., Ogden, L. G., Roberts, S. A., Wyatt, H. R., Wing, R. R., & Hill, J. O. (2014). Low/No calorie sweetened beverage consumption in the National Weight Control Registry: LNCSB Consumption in the NWCR. *Obesity*, *22*(10), 2244–2251. <https://doi.org/10.1002/oby.20834>
- Caudwell, P., Finlayson, G., Gibbons, C., Hopkins, M., King, N., Näslund, E., & Blundell, J. E. (2013). Resting metabolic rate is associated with hunger, self-determined meal size, and daily energy intake and may represent a marker for appetite,.. *The American Journal of Clinical Nutrition*, *97*(1), 7–14. <https://doi.org/10.3945/ajcn.111.029975>
- Chambers, E. S., Bridge, M. W., & Jones, D. A. (2009). Carbohydrate sensing in the human mouth: Effects on exercise performance and brain activity: Oral carbohydrate and

- exercise performance. *The Journal of Physiology*, 587(8), 1779–1794.
<https://doi.org/10.1113/jphysiol.2008.164285>
- Chambers, L., & Yeomans, M. R. (2011). Individual differences in satiety response to carbohydrate and fat. Predictions from the Three Factor Eating Questionnaire (TFEQ). *Appetite*, 56(2), 316–323. <https://doi.org/10.1016/j.appet.2011.01.003>
- Chao, Grilo, C. M., White, M. A., & Sinha, R. (2014). Food cravings, food intake, and weight status in a community-based sample. *Eating Behaviors*, 15(3), 478–482.
<https://doi.org/10.1016/j.eatbeh.2014.06.003>
- Chao, Jastreboff, A. M., White, M. A., Grilo, C. M., & Sinha, R. (2017). Stress, cortisol, and other appetite-related hormones: Prospective prediction of 6-month changes in food cravings and weight. *Obesity*, 25(4), 713–720.
- Chaput, J.-P., Klingenberg, L., Astrup, A., & Sjödén, A. M. (2011). Modern sedentary activities promote overconsumption of food in our current obesogenic environment: Modern activities and energy balance. *Obesity Reviews*, 12(5), e12–e20.
<https://doi.org/10.1111/j.1467-789X.2010.00772.x>
- Chen, L., Appel, L. J., Loria, C., Lin, P.-H., Champagne, C. M., Elmer, P. J., Ard, J. D., Mitchell, D., Batch, B. C., Svetkey, L. P., & Caballero, B. (2009). Reduction in consumption of sugar-sweetened beverages is associated with weight loss: The PREMIER trial. *The American Journal of Clinical Nutrition*, 89(5), 1299–1306.
<https://doi.org/10.3945/ajcn.2008.27240>
- Chern, C., & Tan, S.-Y. (2019). Energy Expenditure, Carbohydrate Oxidation and Appetitive Responses to Sucrose or Sucralose in Humans: A Pilot Study. *Nutrients*, 11(8), 1782.
<https://doi.org/10.3390/nu11081782>
- Chia, C. W., Shardell, M., Tanaka, T., Liu, D. D., Gravenstein, K. S., Simonsick, E. M., Egan, J. M., & Ferrucci, L. (2016). Chronic Low-Calorie Sweetener Use and Risk of Abdominal Obesity among Older Adults: A Cohort Study. *PLOS ONE*, 11(11), e0167241. <https://doi.org/10.1371/journal.pone.0167241>
- Christensen, L. (2007). Craving for sweet carbohydrate and fat-rich foods? Possible triggers and impact on nutritional intake. *Nutrition Bulletin*, 32(s1), 43–51.
<https://doi.org/10.1111/j.1467-3010.2007.00615.x>
- Cleobury, L., & Tapper, K. (2014). Reasons for eating ‘unhealthy’ snacks in overweight and obese males and females. *Journal of Human Nutrition and Dietetics*, 27(4), 333–341.
<https://doi.org/10.1111/jhn.12169>
- Cohen, J. F. W., Sagar, K., Dahlgren, M. K., Kurdziel, L. B. F., & Gruber, S. A. (2020). The Impact of Micronutrient Fortified Foods on Cognitive Functioning among Low-Income Children: A Pilot and Feasibility Study. *Nutrients*, 12(11), 3351.
<https://doi.org/10.3390/nu12113351>
- Collins, A. L., & McCarthy, H. D. (2003). Evaluation of factors determining the precision of body composition measurements by air displacement plethysmography. *European Journal of Clinical Nutrition*, 57(6), 770–776. <https://doi.org/10.1038/sj.ejcn.1601609>
- Compher, C., Frankenfield, D., Keim, N., & Roth-Yousey, L. (2006). Best Practice Methods to Apply to Measurement of Resting Metabolic Rate in Adults: A Systematic Review. *Journal of the American Dietetic Association*, 106(6), 881–903.
<https://doi.org/10.1016/j.jada.2006.02.009>
- Conroy, R., Febres, G., McMahon, D. J., Thorner, M. O., Gaylinn, B. D., Conwell, I., Aronne, L., & Korner, J. (2014). Recombinant Human Leptin Does Not Alter Gut Hormone Levels after Gastric Bypass but May Attenuate Sweet Cravings. *International Journal of Endocrinology*, 2014, 1–8. <https://doi.org/10.1155/2014/120286>

- Cordain, L., Eaton, S. B., Sebastian, A., Mann, N., Lindeberg, S., Watkins, B. A., O'Keefe, J. H., & Brand-Miller, J. (2005). Origins and evolution of the Western diet: Health implications for the 21st century. *The American Journal of Clinical Nutrition*, *81*(2), 341–354. <https://doi.org/10.1093/ajcn.81.2.341>
- Cordain, L., Watkins, B., Florant, G., Kelher, M., Rogers, L., & Li, Y. (2002). Fatty acid analysis of wild ruminant tissues: Evolutionary implications for reducing diet-related chronic disease. *European Journal of Clinical Nutrition*, *56*(3), 181–191. <https://doi.org/10.1038/sj.ejcn.1601307>
- Cormick, G., Betran, A., Romero, I., Cormick, M., Belizán, J., Bardach, A., & Ciapponi, A. (2021). Effect of Calcium Fortified Foods on Health Outcomes: A Systematic Review and Meta-Analysis. *Nutrients*, *13*(2), 316. <https://doi.org/10.3390/nu13020316>
- Cornelis, M. C., Rimm, E. B., Curhan, G. C., Kraft, P., Hunter, D. J., Hu, F. B., & Van Dam, R. M. (2014). Obesity susceptibility loci and uncontrolled eating, emotional eating and cognitive restraint behaviors in men and women: Obesity Loci and Eating Behavior. *Obesity*, *22*(5), E135–E141. <https://doi.org/10.1002/oby.20592>
- Coulon, S. M., Miller, A. C., Reed, J. M., & Martin, C. K. (2012). Reliability of a common solution-based taste perception test: Implications for validity and a briefer test. *Eating Behaviors*, *13*(1), 42–45. <https://doi.org/10.1016/j.eatbeh.2011.09.003>
- Coutinho, S. R., Halset, E. H., Gåsbakk, S., Rehfeld, J. F., Kulseng, B., Truby, H., & Martins, C. (2018). Compensatory mechanisms activated with intermittent energy restriction: A randomized control trial. *Clinical Nutrition*, *37*(3), 815–823. <https://doi.org/10.1016/j.clnu.2017.04.002>
- Cox, Hendrie, G. A., & Carty, D. (2016). Sensitivity, hedonics and preferences for basic tastes and fat amongst adults and children of differing weight status: A comprehensive review. *Food Quality and Preference*, *48*, 359–367. <https://doi.org/10.1016/j.foodqual.2015.01.006>
- Cox, Perry, L., Moore, P., Vallis, L., & Mela, D. (1999). Sensory and hedonic associations with macronutrient and energy intakes of lean and obese consumers. *International Journal of Obesity*, *23*(4), 403–410. <https://doi.org/10.1038/sj.ijo.0800836>
- Crézé, C., Candal, L., Cros, J., Knebel, J.-F., Seyssel, K., Stefanoni, N., Schneiter, P., Murray, M., Tappy, L., & Toepel, U. (2018). The Impact of Caloric and Non-Caloric Sweeteners on Food Intake and Brain Responses to Food: A Randomized Crossover Controlled Trial in Healthy Humans. *Nutrients*, *10*(5), 615. <https://doi.org/10.3390/nu10050615>
- Curran, F., Davis, M. E., Murphy, K., Tersigni, N., King, A., Ngo, N., & O'Donoghue, G. (2023). Correlates of physical activity and sedentary behavior in adults living with overweight and obesity: A systematic review. *Obesity Reviews*, *24*(11), e13615. <https://doi.org/10.1111/obr.13615>
- Dalton, M., Blundell, J., & Finlayson, G. (2013a). Effect of BMI and Binge Eating on Food Reward and Energy Intake: Further Evidence for a Binge Eating Subtype of Obesity. *Obesity Facts*, *6*(4), 348–359. <https://doi.org/10.1159/000354599>
- Dalton, M., Blundell, J., & Finlayson, G. S. (2013b). Examination of food reward and energy intake under laboratory and free-living conditions in a trait binge eating subtype of obesity. *Frontiers in Psychology*, *4*. <https://doi.org/10.3389/fpsyg.2013.00757>
- Dalton, M., & Finlayson, G. (2014). Psychobiological examination of liking and wanting for fat and sweet taste in trait binge eating females. *Physiology & Behavior*, *136*, 128–134. <https://doi.org/10.1016/j.physbeh.2014.03.019>
- Dalton, M., Finlayson, G., Hill, A., & Blundell, J. (2015). Preliminary validation and principal components analysis of the Control of Eating Questionnaire (CoEQ) for the experience

- of food craving. *European Journal of Clinical Nutrition*, 69(12), 1313–1317.
<https://doi.org/10.1038/ejcn.2015.57>
- Davis, C., & Fox, J. (2008). Sensitivity to reward and body mass index (BMI): Evidence for a non-linear relationship. *Appetite*, 50(1), 43–49.
<https://doi.org/10.1016/j.appet.2007.05.007>
- Davis, C., Patte, K., Levitan, R., Reid, C., Tweed, S., & Curtis, C. (2007). From motivation to behaviour: A model of reward sensitivity, overeating, and food preferences in the risk profile for obesity. *Appetite*, 48(1), 12–19. <https://doi.org/10.1016/j.appet.2006.05.016>
- Davis, C. S., Clarke, R. E., Coulter, S. N., Rounsefell, K. N., Walker, R. E., Rauch, C. E., Huggins, C. E., & Ryan, L. (2016). Intermittent energy restriction and weight loss: A systematic review. *European Journal of Clinical Nutrition*, 70(3), 292–299.
<https://doi.org/10.1038/ejcn.2015.195>
- de Bruijn, S. E. M., de Vries, Y. C., de Graaf, C., Boesveldt, S., & Jager, G. (2017). The reliability and validity of the Macronutrient and Taste Preference Ranking Task: A new method to measure food preferences. *Food Quality and Preference*, 57, 32–40.
<https://doi.org/10.1016/j.foodqual.2016.11.003>
- de Castro, J. (2000). Palatability and intake relationships in free-living humans characterization and independence of influence in North Americans. *Physiology & Behavior*, 70(3–4), 343–350. [https://doi.org/10.1016/S0031-9384\(00\)00264-X](https://doi.org/10.1016/S0031-9384(00)00264-X)
- De Koning, L., Merchant, A. T., Pogue, J., & Anand, S. S. (2007). Waist circumference and waist-to-hip ratio as predictors of cardiovascular events: Meta-regression analysis of prospective studies. *European Heart Journal*, 28(7), 850–856.
<https://doi.org/10.1093/eurheartj/ehm026>
- de Ruyter, J. C., Katan, M. B., Kuijper, L. D. J., Liem, D. G., & Olthof, M. R. (2013). The Effect of Sugar-Free Versus Sugar-Sweetened Beverages on Satiety, Liking and Wanting: An 18 Month Randomized Double-Blind Trial in Children. *PLoS ONE*, 8(10), e78039. <https://doi.org/10.1371/journal.pone.0078039>
- De Vogli, R., Kouvonen, A., & Gimeno, D. (2011). ‘Globesization’: Ecological evidence on the relationship between fast food outlets and obesity among 26 advanced economies. *Critical Public Health*, 21(4), 395–402. <https://doi.org/10.1080/09581596.2011.619964>
- de Zwaan, (2001). Binge eating disorder and obesity. *International Journal of Obesity*, 5.
- de Zwaan, Nutzinger, D. O., & Schoenbeck, G. (1992). Binge eating in overweight women. *Comprehensive Psychiatry*, 33(4), 256–261. [https://doi.org/10.1016/0010-440X\(92\)90050-Z](https://doi.org/10.1016/0010-440X(92)90050-Z)
- Deglaire, A., Méjean, C., Castetbon, K., Kesse-Guyot, E., Hercberg, S., & Schlich, P. (2015). Associations between weight status and liking scores for sweet, salt and fat according to the gender in adults (The Nutrinet-Santé study). *European Journal of Clinical Nutrition*, 69(1), 40–46. <https://doi.org/10.1038/ejcn.2014.139>
- DellaValle, D. M., Roe, L. S., & Rolls, B. J. (2005). Does the consumption of caloric and non-caloric beverages with a meal affect energy intake? *Appetite*, 44(2), 187–193.
<https://doi.org/10.1016/j.appet.2004.11.003>
- Delogu, F., Huddas, C., Steven, K., Hachem, S., Lodhia, L., Fernandez, R., & Logerstedt, M. (2016). A Dissociation Between Recognition and Hedonic Value in Caloric and Non-caloric Carbonated Soft Drinks. *Frontiers in Psychology*, 7.
<https://doi.org/10.3389/fpsyg.2016.00036>
- Desor, J. A., & Beauchamp, G. K. (1987). Longitudinal changes in sweet preferences in humans. *Physiology & Behavior*, 39(5), 639–641. [https://doi.org/10.1016/0031-9384\(87\)90166-1](https://doi.org/10.1016/0031-9384(87)90166-1)

- Desor, J. A., Maller, O., & Turner, R. E. (1973). Taste in acceptance of sugars by human infants. *Journal of Comparative and Physiological Psychology*, *84*(3), 496–501. <https://doi.org/10.1037/h0034906>
- Dhillon, J., Lee, J. Y., & Mattes, R. D. (2017). The cephalic phase insulin response to nutritive and low-calorie sweeteners in solid and beverage form. *Physiology & Behavior*, *181*, 100–109. <https://doi.org/10.1016/j.physbeh.2017.09.009>
- Donaldson, L. F., Bennett, L., Baic, S., & Melichar, J. K. (2009). Taste and weight: Is there a link? *The American Journal of Clinical Nutrition*, *90*(3), 800S–803S. <https://doi.org/10.3945/ajcn.2009.27462Q>
- Dotson, C. D., Babich, J., & Steinle, N. I. (2012). Genetic Predisposition and Taste Preference: Impact on Food Intake and Risk of Chronic Disease. *Current Nutrition Reports*, *1*(3), 175–183. <https://doi.org/10.1007/s13668-012-0021-3>
- Doucet, É., McInis, K., & Mahmoodianfard, S. (2018). Compensation in response to energy deficits induced by exercise or diet: Weight loss and compensation. *Obesity Reviews*, *19*, 36–46. <https://doi.org/10.1111/obr.12783>
- Drewnowski, A. (1997). Taste preferences and food intake. *Annual Review of Nutrition*, *17*(1), 237–253. <https://doi.org/10.1146/annurev.nutr.17.1.237>
- Drewnowski, A. (1999). Intense sweeteners and energy density of foods: Implications for weight control. *European Journal of Clinical Nutrition*, *53*(10), 757–763. <https://doi.org/10.1038/sj.ejcn.1600879>
- Drewnowski, A. (2007). The Real Contribution of Added Sugars and Fats to Obesity. *Epidemiologic Reviews*, *29*(1), 160–171. <https://doi.org/10.1093/epirev/mxm011>
- Drewnowski, A., Brunzell, J., Sande, K., Iverius, P., & Greenwood, M. (1985). Sweet tooth reconsidered: Taste responsiveness in human obesity. *Physiology & Behavior*, *35*(4), 617–622. [https://doi.org/10.1016/0031-9384\(85\)90150-7](https://doi.org/10.1016/0031-9384(85)90150-7)
- Drewnowski, A., & Hann, C. (1999). Food preferences and reported frequencies of food consumption as predictors of current diet in young women. *The American Journal of Clinical Nutrition*, *70*(1), 28–36. <https://doi.org/10.1093/ajcn/70.1.28>
- Drewnowski, A., Henderson, S. A., & Shore, A. B. (1997). Genetic Sensitivity to 6-n-Propylthiouracil (PROP) and Hedonic Responses to Bitter and Sweet Tastes. *Chemical Senses*, *22*(1), 27–37. <https://doi.org/10.1093/chemse/22.1.27>
- Drewnowski, A., Krahn, D. D., Demitrack, M. A., Nairn, K., & Gosnell, B. A. (1995). *Taste Responses and Preferences for Sweet High-Fat Foods: Evidence for Opioid Involvement I*. 9.
- Drewnowski, A., & Schwartz, M. (1990). Invisible fats: Sensory assessment of sugar/fat mixtures. *Appetite*, *14*(3), 203–217. [https://doi.org/10.1016/0195-6663\(90\)90088-P](https://doi.org/10.1016/0195-6663(90)90088-P)
- Drewnowski, & Greenwood, M. R. C. (1983). Cream and sugar: Human preferences for high-fat foods. *Physiology & Behavior*, *30*(4), 629–633. [https://doi.org/10.1016/0031-9384\(83\)90232-9](https://doi.org/10.1016/0031-9384(83)90232-9)
- Drewnowski, Kurth, C., Holden-Wiltse, J., & Saari, J. (1992). Food preferences in human obesity: Carbohydrates versus fats. *Appetite*, *18*(3), 207–221. [https://doi.org/10.1016/0195-6663\(92\)90198-F](https://doi.org/10.1016/0195-6663(92)90198-F)
- Drewnowski, Mennella, J. A., Johnson, S. L., & Bellisle, F. (2012). Sweetness and Food Preference. *The Journal of Nutrition*, *142*(6), 1142S–1148S. <https://doi.org/10.3945/jn.111.149575>
- Dye, L., Warner, P., & Bancroft, J. (1995). Food craving during the menstrual cycle and its relationship to stress, happiness of relationship and depression; a preliminary enquiry. *Journal of Affective Disorders*, *34*(3), 157–164. [https://doi.org/10.1016/0165-0327\(95\)00013-D](https://doi.org/10.1016/0165-0327(95)00013-D)

- Ebbeling, C. B., Feldman, H. A., Steltz, S. K., Quinn, N. L., Robinson, L. M., & Ludwig, D. S. (2020). *Effects of Sugar-Sweetened, Artificially Sweetened, and Unsweetened Beverages on Cardiometabolic Risk Factors, Body Composition, and Sweet Taste Preference: A Randomized Controlled Trial*. *9*(20).
- Elizabeth, L., Machado, P., Zinöcker, M., Baker, P., & Lawrence, M. (2020). Ultra-Processed Foods and Health Outcomes: A Narrative Review. *Nutrients*, *12*(7), 1955. <https://doi.org/10.3390/nu12071955>
- Elliott, S. S., Keim, N. L., Stern, J. S., Teff, K., & Havel, P. J. (2002). Fructose, weight gain, and the insulin resistance syndrome. *The American Journal of Clinical Nutrition*, *76*(5), 911–922. <https://doi.org/10.1093/ajcn/76.5.911>
- Emmett, P. M., & Heaton, K. W. (1995). Is extrinsic sugar a vehicle for dietary fat? *The Lancet*, *345*(8964), 1537–1540. [https://doi.org/10.1016/S0140-6736\(95\)91087-5](https://doi.org/10.1016/S0140-6736(95)91087-5)
- Epstein, L. H., Truesdale, R., Wojcik, A., Paluch, R. A., & Raynor, H. A. (2003). Effects of deprivation on hedonics and reinforcing value of food. *Physiology & Behavior*, *78*(2), 221–227. [https://doi.org/10.1016/S0031-9384\(02\)00978-2](https://doi.org/10.1016/S0031-9384(02)00978-2)
- Esses, V., & Herman, C. (1984). Palatability of sucrose before and after glucose ingestion in dieters and nondieters. *Physiology & Behavior*, *32*(5), 711–715. [https://doi.org/10.1016/0031-9384\(84\)90183-5](https://doi.org/10.1016/0031-9384(84)90183-5)
- Ettinger, L., Duizer, L., & Caldwell, T. (2012). Body Fat, Sweetness Sensitivity, and Preference: Determining the Relationship. *Canadian Journal of Dietetic Practice and Research*, *73*(1), 45–48. <https://doi.org/10.3148/73.1.2012.45>
- Fabbricatore, M., Imperatori, C., Contardi, A., Tamburello, A., & Innamorati, M. (2013). Food craving is associated with multiple weight loss attempts. *Mediterranean Journal of Nutrition and Metabolism*, *6*(1), 79–83. <https://doi.org/10.1007/s12349-012-0115-x>
- Fantino, M., Fantino, A., Matray, M., & Mistretta, F. (2018a). Beverages containing low energy sweeteners do not differ from water in their effects on appetite, energy intake and food choices in healthy, non-obese French adults. *Appetite*, *125*, 557–565. <https://doi.org/10.1016/j.appet.2018.03.007>
- Fantino, M., Fantino, A., Matray, M., & Mistretta, F. (2018b). Reprint of ‘Beverages containing low energy sweeteners do not differ from water in their effects on appetite, energy intake and food choices in healthy, non-obese French adults’. *Appetite*, *129*, 103–112. <https://doi.org/10.1016/j.appet.2018.06.036>
- Farhat, G., Berset, V., & Moore, L. (2019). Effects of Stevia Extract on Postprandial Glucose Response, Satiety and Energy Intake: A Three-Arm Crossover Trial. *Nutrients*, *11*(12), 3036. <https://doi.org/10.3390/nu11123036>
- Fastenau, J., Kolotkin, R., L., Fujioka, K., Alba, M., Canovatchel, W., & Traina, S. (2019). A call to action to inform patient-centred approaches to obesity management: Development of a disease-illness model. *Clinical Obesity*, *9*(3).
- Fedoroff, I., Polivy, J., & Peter Herman, C. (2003). The specificity of restrained versus unrestrained eaters’ responses to food cues: General desire to eat, or craving for the cued food? *Appetite*, *41*(1), 7–13. [https://doi.org/10.1016/S0195-6663\(03\)00026-6](https://doi.org/10.1016/S0195-6663(03)00026-6)
- Feig, E. H., Piers, A. D., Kral, T. V. E., & Lowe, M. R. (2018). Eating in the absence of hunger is related to loss-of-control eating, hedonic hunger, and short-term weight gain in normal-weight women. *Appetite*, *123*, 317–324. <https://doi.org/10.1016/j.appet.2018.01.013>
- Feijó, F. de M., Ballard, C. R., Foletto, K. C., Batista, B. A. M., Neves, A. M., Ribeiro, M. F. M., & Bertoluci, M. C. (2013). Saccharin and aspartame, compared with sucrose, induce greater weight gain in adult Wistar rats, at similar total caloric intake levels. *Appetite*, *60*, 203–207. <https://doi.org/10.1016/j.appet.2012.10.009>

- Fernandez-Garcia, J. C., Alcaide, J., Santiago-Fernandez, C., Roca-Rodriguez, M., Aguera, Z., Baños, R., Botella, C., de la Torre, R., Fernandez-Real, J. M., Fruhbeck, G., Gomez-Ambrosi, J., Jimenez-Murcia, S., Menchon, J. M., Casanueva, F. F., Fernandez-Aranda, F., Tinahones, F. J., & Garrido-Sanchez, L. (2017). An increase in visceral fat is associated with a decrease in the taste and olfactory capacity. *PLOS ONE*, *12*(2), e0171204. <https://doi.org/10.1371/journal.pone.0171204>
- Field, A. E., Willett, W. C., Lissner, L., & Colditz, G. A. (2007). Dietary Fat and Weight Gain Among Women in the Nurses' Health Study*. *Obesity*, *15*(4), 967–976. <https://doi.org/10.1038/oby.2007.616>
- Finlayson, G. (2017). Food addiction and obesity: Unnecessary medicalization of hedonic overeating. *Nature Reviews Endocrinology*, *13*(8), 493–498. <https://doi.org/10.1038/nrendo.2017.61>
- Finlayson, G., Arlotti, A., Dalton, M., King, N., & Blundell, J. E. (2011). Implicit wanting and explicit liking are markers for trait binge eating. A susceptible phenotype for overeating. *Appetite*, *57*(3), 722–728. <https://doi.org/10.1016/j.appet.2011.08.012>
- Finlayson, G., Bordes, I., Griffioen-Roose, S., de Graaf, C., & Blundell, J. E. (2012). Susceptibility to Overeating Affects the Impact of Savory or Sweet Drinks on Satiation, Reward, and Food Intake in Nonobese Women. *The Journal of Nutrition*, *142*(1), 125–130. <https://doi.org/10.3945/jn.111.148106>
- 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*(1), 42–49. <https://doi.org/10.1007/s13679-011-0007-2>
- Finlayson, G., King, N., & Blundell, J. (2008). The role of implicit wanting in relation to explicit liking and wanting for food: Implications for appetite control. *Appetite*, *50*(1), 120–127. <https://doi.org/10.1016/j.appet.2007.06.007>
- Finlayson, G., King, N., & Blundell, J. E. (2007a). Is it possible to dissociate 'liking' and 'wanting' for foods in humans? A novel experimental procedure. *Physiology & Behavior*, *90*(1), 36–42. <https://doi.org/10.1016/j.physbeh.2006.08.020>
- Finlayson, G., King, N., & Blundell, J. E. (2007b). Liking vs. wanting food: Importance for human appetite control and weight regulation. *Neuroscience & Biobehavioral Reviews*, *31*(7), 987–1002. <https://doi.org/10.1016/j.neubiorev.2007.03.004>
- Fletcher, B. (C), Pine, K. J., Woodbridge, Z., & Nash, A. (2007). How visual images of chocolate affect the craving and guilt of female dieters. *Appetite*, *48*(2), 211–217. <https://doi.org/10.1016/j.appet.2006.09.002>
- Flint, A., Raben, A., Blundell, J., & Astrup, A. (2000). Reproducibility, power and validity of visual analogue scales in assessment of appetite sensations in single test meal studies. *International Journal of Obesity*, *24*(1), 38–48. <https://doi.org/10.1038/sj.ijo.0801083>
- Forde, C. G., van Kuijk, N., Thaler, T., de Graaf, C., & Martin, N. (2013). Texture and savoury taste influences on food intake in a realistic hot lunch time meal. *Appetite*, *60*, 180–186. <https://doi.org/10.1016/j.appet.2012.10.002>
- Fowler. (2016). Low-calorie sweetener use and energy balance: Results from experimental studies in animals, and large-scale prospective studies in humans. *Physiology & Behavior*, *164*, 517–523. <https://doi.org/10.1016/j.physbeh.2016.04.047>
- Fowler, Williams, K., & Hazuda, H. P. (2015). Diet Soda Intake Is Associated with Long-Term Increases in Waist Circumference in a Biethnic Cohort of Older Adults: The San Antonio Longitudinal Study of Aging. *Journal of the American Geriatrics Society*, *63*(4), 708–715. <https://doi.org/10.1111/jgs.13376>

- Fowler, Williams, K., Resendez, R. G., Hunt, K. J., Hazuda, H. P., & Stern, M. P. (2008). Fueling the Obesity Epidemic? Artificially Sweetened Beverage Use and Long-term Weight Gain. *Obesity*, *16*(8), 1894–1900. <https://doi.org/10.1038/oby.2008.284>
- Frank, G. K. W., Oberndorfer, T. A., Simmons, A. N., Paulus, M. P., Fudge, J. L., Yang, T. T., & Kaye, W. H. (2008). Sucrose activates human taste pathways differently from artificial sweetener. *NeuroImage*, *39*(4), 1559–1569. <https://doi.org/10.1016/j.neuroimage.2007.10.061>
- Franken, I. H. A., & Muris, P. (2005). Individual differences in reward sensitivity are related to food craving and relative body weight in healthy women. *Appetite*, *45*(2), 198–201. <https://doi.org/10.1016/j.appet.2005.04.004>
- Fraser, L. K., & Edwards, K. L. (2010). The association between the geography of fast food outlets and childhood obesity rates in Leeds, UK. *Health & Place*, *16*(6), 1124–1128. <https://doi.org/10.1016/j.healthplace.2010.07.003>
- French, S. A., Jeffery, R. W., & Wing, R. R. (1994). Food intake and physical activity: A comparison of three measures of dieting. *Addictive Behaviors*, *19*(4), 401–409. [https://doi.org/10.1016/0306-4603\(94\)90063-9](https://doi.org/10.1016/0306-4603(94)90063-9)
- Gadah, N. S., Kyle, L. A., Smith, J. E., Brunstrom, J. M., & Rogers, P. J. (2016). No difference in compensation for sugar in a drink versus sugar in semi-solid and solid foods. *Physiology & Behavior*, *156*, 35–42. <https://doi.org/10.1016/j.physbeh.2015.12.025>
- Gallant, A. R., Tremblay, A., Pérusse, L., Bouchard, C., Després, J.-P., & Drapeau, V. (2010). The Three-Factor Eating Questionnaire and BMI in adolescents: Results from the Québec Family Study. *British Journal of Nutrition*, *104*(7), 1074–1079. <https://doi.org/10.1017/S0007114510001662>
- Garneau, N. L., Nuessle, T. M., Mendelsberg, B. J., Shepard, S., & Tucker, R. M. (2018). Sweet liker status in children and adults: Consequences for beverage intake in adults. *Food Quality and Preference*, *65*, 175–180. <https://doi.org/10.1016/j.foodqual.2017.10.005>
- Garner, D. M., & Garfinkel, P. E. (1979). The Eating Attitudes Test: An index of the symptoms of anorexia nervosa. *Psychological Medicine*, *9*(2), 273–279. <https://doi.org/10.1017/S0033291700030762>
- Gaudette, N. J., & Pickering, G. J. (2012). Optimizing the Orosensory Properties of Model Functional Beverages: The Influence of Novel Sweeteners, Odorants, Bitter Blockers, and Their Mixtures on (+)-Catechin. *Journal of Food Science*, *77*(6), S226–S232. <https://doi.org/10.1111/j.1750-3841.2012.02707.x>
- Gearhardt, A. N., Corbin, W. R., & Brownell, K. D. (2009). Preliminary validation of the Yale Food Addiction Scale. *Appetite*, *52*(2), 430–436. <https://doi.org/10.1016/j.appet.2008.12.003>
- Gendall, K. A., Joyce, P. R., & Abbott, R. M. (1999). The effects of meal composition on subsequent craving and binge eating. *Addictive Behaviors*, *24*(3), 305–315. [https://doi.org/10.1016/S0306-4603\(98\)00046-X](https://doi.org/10.1016/S0306-4603(98)00046-X)
- Gendall, K. A., Joyce, P. R., Sullivan, P. F., & Bulik, C. M. (1998). Food cravers: Characteristics of those who binge. *International Journal of Eating Disorders*, *23*(4), 353–360. [https://doi.org/10.1002/\(SICI\)1098-108X\(199805\)23:4<353::AID-EAT2>3.0.CO;2-H](https://doi.org/10.1002/(SICI)1098-108X(199805)23:4<353::AID-EAT2>3.0.CO;2-H)
- Gent, J. F., & Bartoshuk, L. M. (1983). Sweetness of sucrose, neohesperidin dihydrochalcone, and saccharin is related to genetic ability to taste the bitter substance 6-*n*-propylthiouracil. *Chemical Senses*, *7*(3–4), 265–272. <https://doi.org/10.1093/chemse/7.3-4.265>

- Gibbons, C., O'Hara, B., O'Connor, D., Hardman, C., Wilton, M., Harrold, J. A., Almiron-Roig, E., Navas-Carretero, S., Hodgkins, C. E., Nazare, J. A., Alligier, M., Martínez, J. A., Scott, C., Kjølbaek, L., Normand, M., Rannou, C., Blaak, E. E., Feskens, E., Moshoyiannis, H., ... Finlayson, G. (2022). Acute and repeated impact of sweeteners and sweetness enhancers in solid and semi-solid foods on appetite: Protocol for a multicentre, cross-over, RCT in people with overweight/obesity – the SWEET Project. *BMJ Open*, *12*(12), e063903. <https://doi.org/10.1136/bmjopen-2022-063903>
- Gibney, M. J. (1990). Dietary guidelines: A critical appraisal. *Journal of Human Nutrition and Dietetics*, *3*(4), 245–254. <https://doi.org/10.1111/j.1365-277X.1990.tb00064.x>
- Gibney, M. J. (2019). Ultra-Processed Foods: Definitions and Policy Issues. *Current Developments in Nutrition*, *3*(2). <https://doi.org/10.1093/cdn/nzy077>
- Gibson, S. A. (1996). Are High-Fat, High-Sugar Foods and Diets Conducive to Obesity? *International Journal of Food Sciences and Nutrition*, *47*(5), 405–415. <https://doi.org/10.3109/09637489609006954>
- Gilbert, J.-A., Drapeau, V., Astrup, A., & Tremblay, A. (2009). Relationship between diet-induced changes in body fat and appetite sensations in women. *Appetite*, *52*(3), 809–812. <https://doi.org/10.1016/j.appet.2009.04.003>
- Goldstone, A. P., Precht de Hernandez, C. G., Beaver, J. D., Muhammed, K., Croese, C., Bell, G., Durighel, G., Hughes, E., Waldman, A. D., Frost, G., & Bell, J. D. (2009). Fasting biases brain reward systems towards high-calorie foods. *European Journal of Neuroscience*, *30*(8), 1625–1635. <https://doi.org/10.1111/j.1460-9568.2009.06949.x>
- Goodman, E. L., Breithaupt, L., Watson, H. J., Peat, C. M., Baker, J. H., Bulik, C. M., & Brownley, K. A. (2018). Sweet taste preference in binge-eating disorder: A preliminary investigation. *Eating Behaviors*, *28*, 8–15. <https://doi.org/10.1016/j.eatbeh.2017.11.005>
- Goris, A. H., Westterp-Plantenga, M. S., & Westterp, K. R. (2000). Undereating and underrecording of habitual food intake in obese men: Selective underreporting of fat intake. *The American Journal of Clinical Nutrition*, *71*(1), 130–134. <https://doi.org/10.1093/ajcn/71.1.130>
- Great Britain: Scientific Advisory Committee on Nutrition. (2015). *Carbohydrates and Health Report*. TSO.
- Green, E., & Murphy, C. (2012). Altered processing of sweet taste in the brain of diet soda drinkers. *Physiology & Behavior*, *107*(4), 560–567. <https://doi.org/10.1016/j.physbeh.2012.05.006>
- Green, Rogers, P. J., & Elliman, N. A. (2000). Dietary restraint and addictive behaviors: The generalizability of Tiffany's Cue Reactivity Model. *International Journal of Eating Disorders*, *27*(4), 419–427. [https://doi.org/10.1002/\(SICI\)1098-108X\(200005\)27:4<419::AID-EAT6>3.0.CO;2-Z](https://doi.org/10.1002/(SICI)1098-108X(200005)27:4<419::AID-EAT6>3.0.CO;2-Z)
- Gregersen, N. T., Flint, A., Bitz, C., Blundell, J. E., Raben, A., & Astrup, A. (2008). Reproducibility and power of ad libitum energy intake assessed by repeated single meals. *The American Journal of Clinical Nutrition*, *87*(5), 1277–1281. <https://doi.org/10.1093/ajcn/87.5.1277>
- Gregory, N. G. (2000). *Consumer Concerns about Food*. *29*(4), 7.
- Griffioen-Roose, S., Finlayson, G., Mars, M., Blundell, J. E., & de Graaf, C. (2010). Measuring food reward and the transfer effect of sensory specific satiety. *Appetite*, *55*(3), 648–655. <https://doi.org/10.1016/j.appet.2010.09.018>
- Griffioen-Roose, S., Hogenkamp, P. S., Mars, M., Finlayson, G., & de Graaf, C. (2012). Taste of a 24-h diet and its effect on subsequent food preferences and satiety. *Appetite*, *59*(1), 1–8. <https://doi.org/10.1016/j.appet.2012.03.013>

- Griffioen-Roose, S., Mars, M., Finlayson, G., Blundell, J. E., & de Graaf, C. (2011). The effect of within-meal protein content and taste on subsequent food choice and satiety. *British Journal of Nutrition*, *106*(5), 779–788. <https://doi.org/10.1017/S0007114511001012>
- Griffioen-Roose, S., Smeets, P. A. M., Weijzen, P. L. G., van Rijn, I., van den Bosch, I., & de Graaf, C. (2013). Effect of Replacing Sugar with Non-Caloric Sweeteners in Beverages on the Reward Value after Repeated Exposure. *PLoS ONE*, *8*(11), e81924. <https://doi.org/10.1371/journal.pone.0081924>
- Guyatt, O., A. D. ., Vist, G. E. ., Kunz, R. ., Falck-Ytter, Y. ., Alonso-Coello, P. ., & Schünemann, H. J. Oxman, A. D. ., Vist, G. E. ., Kunz, R. ., Falck-Ytter, Y. ., Alonso-Coello, P. . & Schünemann, H. (2008). GRADE: an emerging consensus on rating quality of evidence and strength of recommendations. *Bmj*, *336*(7650), 924–926.
- Haase, L., Cerf-Ducastel, B., & Murphy, C. (2009). Cortical activation in response to pure taste stimuli during the physiological states of hunger and satiety. *NeuroImage*, *44*(3), 1008–1021. <https://doi.org/10.1016/j.neuroimage.2008.09.044>
- Hagströmer, M., Oja, P., & Sjöström, M. (2006). The International Physical Activity Questionnaire (IPAQ): A study of concurrent and construct validity. *Public Health Nutrition*, *9*(6), 755–762. <https://doi.org/10.1079/PHN2005898>
- Han, P., Bagenna, B., & Fu, M. (2018). The sweet taste signalling pathways in the oral cavity and the gastrointestinal tract affect human appetite and food intake: A review. *International Journal of Food Sciences and Nutrition*, *0*(0), 1–11. <https://doi.org/10.1080/09637486.2018.1492522>
- Han, P., Keast, R. S. J., & Roura, E. (2017). Salivary leptin and *TAS1R2/TAS1R3* polymorphisms are related to sweet taste sensitivity and carbohydrate intake from a buffet meal in healthy young adults. *British Journal of Nutrition*, *118*(10), 763–770. <https://doi.org/10.1017/S0007114517002872>
- Hare, T. A., Camerer, C. F., Knopfle, D. T., O’Doherty, J. P., & Rangel, A. (2010). Value Computations in Ventral Medial Prefrontal Cortex during Charitable Decision Making Incorporate Input from Regions Involved in Social Cognition. *Journal of Neuroscience*, *30*(2), 583–590. <https://doi.org/10.1523/JNEUROSCI.4089-09.2010>
- Harris, L., McGarty, A., Hutchison, L., Ells, L., & Hankey, C. (2018). Short-term intermittent energy restriction interventions for weight management: A systematic review and meta-analysis: Daily Intermittent Energy Restriction. *Obesity Reviews*, *19*(1), 1–13. <https://doi.org/10.1111/obr.12593>
- Hatoum, I. J., & Kaplan, L. M. (2013). Advantages of percent weight loss as a method of reporting weight loss after Roux-en-Y gastric bypass. *Obesity*, *21*(8), 1519–1525.
- Haynes, C., Lee, M. D., & Yeomans, M. R. (2003). Interactive effects of stress, dietary restraint, and disinhibition on appetite. *Eating Behaviors*, *4*(4), 369–383. <https://doi.org/10.1016/j.eatbeh.2003.07.005>
- Hebert, J. R., Clemow, L., Pbert, L., Ockene, I. S., & Ockene, J. K. (1995). Social Desirability Bias in Dietary Self-Report May Compromise the Validity of Dietary Intake Measures. *International Journal of Epidemiology*, *24*(2), 389–398. <https://doi.org/10.1093/ije/24.2.389>
- Heini, A. F., & Weinsier, R. L. (1997). Divergent trends in obesity and fat intake patterns: The american paradox. *The American Journal of Medicine*, *102*(3), 259–264. [https://doi.org/10.1016/S0002-9343\(96\)00456-1](https://doi.org/10.1016/S0002-9343(96)00456-1)
- Herman, C. P., & Mack, D. (1975). Restrained and unrestrained eating. *Journal of Personality*, *43*(4), 647–660. <https://doi.org/10.1111/j.1467-6494.1975.tb00727.x>

- 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. <https://doi.org/10.1016/j.jcps.2014.03.002>
- Hetherington, M. M. (1996). Sensory-Specific Satiety and its Importance in Meal Termination. *Neuroscience & Biobehavioral Reviews, 20*(1), 5.
- Higgins, K. A., Considine, R. V., & Mattes, R. D. (2018). Aspartame Consumption for 12 Weeks Does Not Affect Glycemia, Appetite, or Body Weight of Healthy, Lean Adults in a Randomized Controlled Trial. *The Journal of Nutrition, 148*(4), 650–657. <https://doi.org/10.1093/jn/nxy021>
- Higgins, K. A., & Mattes, R. D. (2019). A randomized controlled trial contrasting the effects of 4 low-calorie sweeteners and sucrose on body weight in adults with overweight or obesity. *The American Journal of Clinical Nutrition.* <https://doi.org/10.1093/ajcn/nqy381>
- 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. <https://doi.org/10.1177/0269881117736917>
- Hill, A. J., Weaver, C. F. L., & Blundell, J. E. (1991). Food craving, dietary restraint and mood. *Appetite, 17*(3), 187–197. [https://doi.org/10.1016/0195-6663\(91\)90021-J](https://doi.org/10.1016/0195-6663(91)90021-J)
- Hill, Prokosch, M. L., Morin, A., & Rodeheffer, C. D. (2014). The effect of non-caloric sweeteners on cognition, choice, and post-consumption satisfaction. *Appetite, 83*, 82–88. <https://doi.org/10.1016/j.appet.2014.08.003>
- Hill, S. A. B. (1965). *The Environment and Disease: Association or Causation?* 6.
- Hoch, T., Pischetsrieder, M., & Hess, A. (2014). Snack food intake in ad libitum fed rats is triggered by the combination of fat and carbohydrates. *Frontiers in Psychology, 5*. <https://doi.org/10.3389/fpsyg.2014.00250>
- Hoek, A. C., Elzerman, J. E., Hageman, R., Kok, F. J., Luning, P. A., & Graaf, C. de. (2013). Are meat substitutes liked better over time? A repeated in-home use test with meat substitutes or meat in meals. *Food Quality and Preference, 28*(1), 253–263. <https://doi.org/10.1016/j.foodqual.2012.07.002>
- Holt, S. H. A., Cobiac, L., Beaumont-Smith, N. E., Easton, K., & Best, D. J. (2000). Dietary habits and the perception and liking of sweetness among Australian and Malaysian students: A cross-cultural study. *Food Quality and Preference, 11*(4), 299–312. [https://doi.org/10.1016/S0950-3293\(99\)00076-2](https://doi.org/10.1016/S0950-3293(99)00076-2)
- Hormes, J. M. (2014). Perimenstrual chocolate craving: From pharmacology and physiology to cognition and culture. *Handbook of Diet and Nutrition in the Menstrual Cycle, Periconception and Fertility, 7*(137).
- Howard, B. V., & Wylie-Rosett, J. (2002). Sugar and Cardiovascular Disease: A Statement for Healthcare Professionals From the Committee on Nutrition of the Council on Nutrition, Physical Activity, and Metabolism of the American Heart Association. *Circulation, 106*(4), 523–527. <https://doi.org/10.1161/01.CIR.0000019552.77778.04>
- Hu, F. B. (2013). Resolved: There is sufficient scientific evidence that decreasing sugarsweetened beverage consumption will reduce the prevalence of obesity and obesity-related diseases. *Obesity Reviews, 14*.
- Huh, Y. E., Vosgerau, J., & Morewedge, C. K. (2016). More Similar but Less Satisfying: Comparing Preferences for and the Efficacy of Within- and Cross-Category Substitutes for Food. *Psychological Science, 27*(6), 894–903. <https://doi.org/10.1177/0956797616640705>

- Hutchinson, S. A., Ho, G. S., & Ho, C. (1999). Stability and degradation of the high-intensity sweeteners: Aspartame, Alitame, and Sucralose. *Food Reviews International*, *15*(2), 249–261. <https://doi.org/10.1080/87559129909541189>
- Hwang, L.-D., Zhu, G., Breslin, P. A. S., Reed, D. R., Martin, N. G., & Wright, M. J. (2015). A Common Genetic Influence on Human Intensity Ratings of Sugars and High-Potency Sweeteners. *Twin Research and Human Genetics*, *18*(4), 361–367. <https://doi.org/10.1017/thg.2015.42>
- Iatridi, V., Armitage, R. M., Yeomans, M. R., & Hayes, J. E. (2020). Effects of Sweet-Liking on Body Composition Depend on Age and Lifestyle: A Challenge to the Simple Sweet-Liking—Obesity Hypothesis. *Nutrients*, *12*(9), 2702. <https://doi.org/10.3390/nu12092702>
- Iatridi, V., Hayes, J. E., & Yeomans, M. R. (2019a). Reconsidering the classification of sweet taste liker phenotypes: A methodological review. *Food Quality and Preference*, *72*, 56–76. <https://doi.org/10.1016/j.foodqual.2018.09.001>
- Iatridi, V., Hayes, J., & Yeomans, M. (2019b). Quantifying Sweet Taste Liker Phenotypes: Time for Some Consistency in the Classification Criteria. *Nutrients*, *11*(1), 129. <https://doi.org/10.3390/nu11010129>
- Iatridi, V., Quadt, L., Hayes, J. E., Garfinkel, S. N., & Yeomans, M. R. (2021). Female sweetlikers have enhanced cross-modal interoceptive abilities. *Appetite*, *165*, 105290. <https://doi.org/10.1016/j.appet.2021.105290>
- Izquierdo, A. G., Crujeiras, A. B., Casanueva, F. F., & Carreira, M. C. (2019). Leptin, Obesity, and Leptin Resistance: Where Are We 25 Years Later? *Nutrients*, *11*(11), 2704. <https://doi.org/10.3390/nu11112704>
- Jakubowicz, D., Froy, O., Wainstein, J., & Boaz, M. (2012). Meal timing and composition influence ghrelin levels, appetite scores and weight loss maintenance in overweight and obese adults. *Steroids*, *77*(4), 323–331. <https://doi.org/10.1016/j.steroids.2011.12.006>
- Jamel, H. A., Sheiham, A., Cowell, C. R., & Watt, R. G. (1996). Taste Preference for Sweetness in Urban and Rural Populations in Iraq. *Journal of Dental Research*, *75*(11), 1879–1884. <https://doi.org/10.1177/00220345960750111001>
- James. (2018). Obesity: A Global Public Health Challenge. *Clinical Chemistry*, *64*(1), 24–29. <https://doi.org/10.1373/clinchem.2017.273052>
- James, Seward, M. W., James O'Malley, A., Subramanian, S., & Block, J. P. (2017). Changes in the food environment over time: Examining 40 years of data in the Framingham Heart Study. *International Journal of Behavioral Nutrition and Physical Activity*, *14*(1), 84. <https://doi.org/10.1186/s12966-017-0537-4>
- Janssen, F., Bardoutsos, A., & Vidra, N. (2020). Obesity Prevalence in the Long-Term Future in 18 European Countries and in the USA. *Obesity Facts*, *13*(5), 514–527. <https://doi.org/10.1159/000511023>
- Jayasinghe, S., Kruger, R., Walsh, D., Cao, G., Rivers, S., Richter, M., & Breier, B. (2017). Is Sweet Taste Perception Associated with Sweet Food Liking and Intake? *Nutrients*, *9*(7), 750. <https://doi.org/10.3390/nu9070750>
- Johnson, R. K., Friedman, A. B., Harvey-Berino, J., Gold, B. C., & McKenzie, D. (2005). Participation in a Behavioral Weight-Loss Program Worsens the Prevalence and Severity of Underreporting among Obese and Overweight Women. *Journal of the American Dietetic Association*, *105*(12), 1948–1951. <https://doi.org/10.1016/j.jada.2005.09.006>
- Julia, C., Martinez, L., Allès, B., Touvier, M., Hercberg, S., Méjean, C., & Kesse-Guyot, E. (2018). Contribution of ultra-processed foods in the diet of adults from the French

- NutriNet-Santé study. *Public Health Nutrition*, 21(1), 27–37.
<https://doi.org/10.1017/S1368980017001367>
- Kahathuduwa, C. N., Binks, M., Martin, C. K., & Dawson, J. A. (2017). Extended calorie restriction suppresses overall and specific food cravings: A systematic review and a meta-analysis: Calorie restriction suppresses food cravings. *Obesity Reviews*, 18(10), 1122–1135. <https://doi.org/10.1111/obr.12566>
- Kahathuduwa, C. N., Boyd, L. A., Davis, T., O’Boyle, M., & Binks, M. (2016). Brain regions involved in ingestive behavior and related psychological constructs in people undergoing calorie restriction. *Appetite*, 107, 348–361.
<https://doi.org/10.1016/j.appet.2016.08.112>
- Kahn, R., & Sievenpiper, J. L. (2014). Dietary Sugar and Body Weight: Have We Reached a Crisis in the Epidemic of Obesity and Diabetes?: We Have, but the Pox on Sugar Is Overwrought and Overworked. *Diabetes Care*, 37(4), 957–962.
<https://doi.org/10.2337/dc13-2506>
- Kamerud, J. K., & Delwiche, J. F. (2007). Individual Differences in Perceived Bitterness Predict Liking of Sweeteners. *Chemical Senses*, 32(9), 803–810.
<https://doi.org/10.1093/chemse/bjm050>
- Kampov-Polevoy. (2001). Sweet liking and family history of alcoholism in hospitalized alcoholic and non-alcoholic patients. *Alcohol and Alcoholism*, 36(2), 165–170.
<https://doi.org/10.1093/alcalc/36.2.165>
- Kampov-Polevoy, Ziedonis, D., Steinberg, M. L., Pinsky, I., Krejci, J., Eick, C., Boland, G., Khalitov, E., & Crews, F. T. (2003). Association Between Sweet Preference and Paternal History of Alcoholism in Psychiatric and Substance Abuse Patients: *Alcoholism: Clinical & Experimental Research*, 27(12), 1929–1936.
<https://doi.org/10.1097/01.ALC.0000099265.60216.23>
- Kartheuser, A. H., Leonard, D. F., Penninckx, F., Paterson, H. M., Brandt, D., Remue, C., Bugli, C., Dozois, E., Mortensen, N., Ris, F., & Tiret, E. (2013). Waist Circumference and Waist/Hip Ratio Are Better Predictive Risk Factors for Mortality and Morbidity after Colorectal Surgery Than Body Mass Index and Body Surface Area. *Annals of Surgery*, 258(5), 722–730. <https://doi.org/10.1097/SLA.0b013e3182a6605a>
- Kawai, K., Sugimoto, K., Nakashima, K., Miura, H., & Ninomiya, Y. (2000). Leptin as a modulator of sweet taste sensitivities in mice. *Proceedings of the National Academy of Sciences*, 97(20), 11044–11049. <https://doi.org/10.1073/pnas.190066697>
- Keaver, L., Xu, B., Jaccard, A., & Webber, L. (2020). Morbid obesity in the UK: A modelling projection study to 2035. *Scandinavian Journal of Public Health*, 48(4), 422–427.
<https://doi.org/10.1177/1403494818794814>
- Kemps, E., Tiggemann, M., & Grigg, M. (2008). Food cravings consume limited cognitive resources. *Journal of Experimental Psychology: Applied*, 14(3), 247–254.
<https://doi.org/10.1037/a0012736>
- Keskitalo, K., Tuorila, H., Spector, T. D., Cherkas, L. F., Knaapila, A., Silventoinen, K., & Perola, M. (2007). Same genetic components underlie different measures of sweet taste preference. *The American Journal of Clinical Nutrition*, 86(6), 1663–1669.
- Kim, J.-Y., Prescott, J., & Kim, K.-O. (2017). Emotional responses to sweet foods according to sweet liker status. *Food Quality and Preference*, 59, 1–7.
<https://doi.org/10.1016/j.foodqual.2017.01.013>
- King, N. A., Appleton, K., Rogers, P. J., & Blundell, J. E. (1999). Effects of Sweetness and Energy in Drinks on Food Intake Following Exercise. *Physiology & Behavior*, 66(2), 375–379. [https://doi.org/10.1016/S0031-9384\(98\)00280-7](https://doi.org/10.1016/S0031-9384(98)00280-7)

- Knorr, D., & Watzke, H. (2019). Food Processing at a Crossroad. *Frontiers in Nutrition*, 6, 85. <https://doi.org/10.3389/fnut.2019.00085>
- Kohno, D. (2017). Sweet taste receptor in the hypothalamus: A potential new player in glucose sensing in the hypothalamus. *The Journal of Physiological Sciences*, 67(4), 459–465. <https://doi.org/10.1007/s12576-017-0535-y>
- Koo, T. K., & Li, M. Y. (2016). A Guideline of Selecting and Reporting Intraclass Correlation Coefficients for Reliability Research. *Journal of Chiropractic Medicine*, 15(2), 155–163. <https://doi.org/10.1016/j.jcm.2016.02.012>
- Krall, E. A., Dwyer, J. T., & Ann Coleman, K. (1988). Factors influencing accuracy of dietary recall. *Nutrition Research*, 8(7), 829–841. [https://doi.org/10.1016/S0271-5317\(88\)80162-3](https://doi.org/10.1016/S0271-5317(88)80162-3)
- Kuzma, J. N., Cromer, G., Hagman, D. K., Breymeyer, K. L., Roth, C. L., Foster-Schubert, K. E., Holte, S. E., Callahan, H. S., Weigle, D. S., & Kratz, M. (2015). No difference in ad libitum energy intake in healthy men and women consuming beverages sweetened with fructose, glucose, or high-fructose corn syrup: A randomized trial. *The American Journal of Clinical Nutrition*, 102(6), 1373–1380. <https://doi.org/10.3945/ajcn.115.116368>
- Lafay, L., Thomas, F., Mennen, L., Charles, M. A., Eschwege, E., Borys, J.-M., & Basdevant, A. (2001). *Gender differences in the relation between food cravings and mood in an adult community: Results from the Fleurbaix Laventie Ville Santé Study*. 10.
- Lähteenmäki, L., & Tuorila, H. (1995). Three-factor eating questionnaire and the use and liking of sweet and fat among dieters. *Physiology & Behavior*, 57(1), 81–88. [https://doi.org/10.1016/0031-9384\(94\)00210-V](https://doi.org/10.1016/0031-9384(94)00210-V)
- Lampuré, A., Adriouch, S., Castetbon, K., Deglaire, A., Schlich, P., Péneau, S., Fezeu, L., Hercberg, S., & Méjean, C. (2019). Relationship between sensory liking for fat, sweet or salt and cardiometabolic diseases: Mediating effects of diet and weight status. *European Journal of Nutrition*. <https://doi.org/10.1007/s00394-019-01904-x>
- Lampuré, A., Castetbon, K., Deglaire, A., Schlich, P., Péneau, S., Hercberg, S., & Méjean, C. (2016). Associations between liking for fat, sweet or salt and obesity risk in French adults: A prospective cohort study. *International Journal of Behavioral Nutrition and Physical Activity*, 13(1). <https://doi.org/10.1186/s12966-016-0406-6>
- Laviada-Molina, H., Molina-Segui, F., Pérez-Gaxiola, G., Cuello-García, C., Arjona-Villicaña, R., Espinosa-Marrón, A., & Martínez-Portilla, R. J. (2020). Effects of nonnutritive sweeteners on body weight and BMI in diverse clinical contexts: Systematic review and meta-analysis. *Obesity Reviews*, 21(7). <https://doi.org/10.1111/obr.13020>
- Lawlor, D. A., & Chaturvedi, N. (2006). Treatment and prevention of obesity—Are there critical periods for intervention? *International Journal of Epidemiology*, 35(1), 3–9. <https://doi.org/10.1093/ije/dyi309>
- Lazar, M. A. (2005). How Obesity Causes Diabetes: Not a Tall Tale. *Science*, 307(5708), 373–375. <https://doi.org/10.1126/science.1104342>
- Lazarides, H. N. (2012). Challenges and Opportunities for the community of Food Sciences to contribute Towards a Society of Healthier Consumers and a Better World. *International Journal of Food Studies*, 1(1). <https://doi.org/10.7455/ijfs/1.1.2012.a10>
- Lee, H. Y., Jack, M., Poon, T., Noori, D., Venditti, C., Hamamji, S., & Musa-Veloso, K. (2021). Effects of Unsweetened Preloads and Preloads Sweetened with Caloric or Low-/No-Calorie Sweeteners on Subsequent Energy Intakes: A Systematic Review and Meta-Analysis of Controlled Human Intervention Studies. *Advances in Nutrition*, nmaa157. <https://doi.org/10.1093/advances/nmaa157>

- Legget, K. T., Cornier, M.-A., Bessesen, D. H., Mohl, B., Thomas, E. A., & Tregellas, J. R. (2018). Greater Reward-Related Neuronal Response to Hedonic Foods in Women Compared with Men: Sex Differences in Neuronal Response to Food. *Obesity*, *26*(2), 362–367. <https://doi.org/10.1002/oby.22082>
- Lemmens, S. G. T., Schoffelen, P. F. M., Wouters, L., Born, J. M., Martens, M. J. I., Rutters, F., & Westerterp-Plantenga, M. S. (2009). Eating what you like induces a stronger decrease of ‘wanting’ to eat. *Physiology & Behavior*, *98*(3), 318–325. <https://doi.org/10.1016/j.physbeh.2009.06.008>
- Levenhagen, D. K., Borel, M. J., Welch, D. C., Piasecki, J. H., Piasecki, D. P., Chen, K. Y., & Flakoll, P. J. (1999). A Comparison of Air Displacement Plethysmography with Three Other Techniques to Determine Body Fat in Healthy Adults. *Journal of Parenteral and Enteral Nutrition*, *23*(5), 293–299. <https://doi.org/10.1177/0148607199023005293>
- Levine, A. S., Kotz, C. M., & Gosnell, B. A. (2003). Sugars and Fats: The Neurobiology of Preference. *The Journal of Nutrition*, *133*(3), 831S-834S. <https://doi.org/10.1093/jn/133.3.831S>
- Li, Lopetcharat, K., & Drake, M. A. (2015). Parents’ and Children’s Acceptance of Skim Chocolate Milks Sweetened by Monk Fruit and Stevia Leaf Extracts: Parents’ and children’s acceptance.... *Journal of Food Science*, *80*(5), S1083–S1092. <https://doi.org/10.1111/1750-3841.12835>
- Li, X., Staszewski, L., Xu, H., Durick, K., Zoller, M., & Adler, E. (2002). Human receptors for sweet and umami taste. *Proceedings of the National Academy of Sciences*, *99*(7), 4692–4696. <https://doi.org/10.1073/pnas.072090199>
- Lieberman, L. S. (2006). Evolutionary and anthropological perspectives on optimal foraging in obesogenic environments. *Appetite*, *47*(1), 3–9. <https://doi.org/10.1016/j.appet.2006.02.011>
- Liem, D., & De Graaf, C. (2004). Sweet and sour preferences in young children and adults: Role of repeated exposure. *Physiology & Behavior*, *83*(3), 421–429. <https://doi.org/10.1016/j.physbeh.2004.08.028>
- Lim, J., & Pullicin, A. J. (2019). Oral carbohydrate sensing: Beyond sweet taste. *Physiology & Behavior*, *202*, 14–25. <https://doi.org/10.1016/j.physbeh.2019.01.021>
- Lindroos, A.-K., Lissner, L., Mathiassen, M. E., Karlsson, J., Sullivan, M., Bengtsson, C., & Sjöström, L. (1997). Dietary Intake in Relation to Restrained Eating, Disinhibition, and Hunger in Obese and Nonobese Swedish Women. *Obesity Research*, *5*(3), 175–182. <https://doi.org/10.1002/j.1550-8528.1997.tb00290.x>
- Liu, D., Archer, N., Duesing, K., Hannan, G., & Keast, R. (2016). Mechanism of fat taste perception: Association with diet and obesity. *Progress in Lipid Research*, *63*, 41–49. <https://doi.org/10.1016/j.plipres.2016.03.002>
- Looy, H., & Weingarten, H. P. (1991). Effects of metabolic state on sweet taste reactivity in humans depend on underlying hedonic response profile. *Chemical Senses*, *16*(2), 123–130. <https://doi.org/10.1093/chemse/16.2.123>
- Looy, H., & Weingarten, H. P. (1992). Facial expressions and genetic sensitivity to 6-n-propylthiouracil predict hedonic response to sweet. *Physiology & Behavior*, *52*(1), 75–82. [https://doi.org/10.1016/0031-9384\(92\)90435-5](https://doi.org/10.1016/0031-9384(92)90435-5)
- Louie. (2017). Patterns of added sugars intake by eating occasion among a nationally representative sample of Australians. *European Journal of Nutrition*, *57*(1), 137–154.
- Louzada, M. L. da C., Ricardo, C. Z., Steele, E. M., Levy, R. B., Cannon, G., & Monteiro, C. A. (2018). The share of ultra-processed foods determines the overall nutritional quality of diets in Brazil. *Public Health Nutrition*, *21*(1), 94–102. <https://doi.org/10.1017/S1368980017001434>

- Lucas, F. (1985). *Hyperphagia in Rats Produced by a Mixture of Fat and Sugar I*. 5.
- Luo, X. (2019). A review of food reformulation of baked products to reduce added sugar intake. *Trends in Food Science & Technology*, 86, 412–425.
<https://doi.org/10.1016/j.tifs.2019.02.051>
- Ma, Y., He, F. J., Yin, Y., Hashem, K. M., & MacGregor, G. A. (2016). Gradual reduction of sugar in soft drinks without substitution as a strategy to reduce overweight, obesity, and type 2 diabetes: A modelling study. *The Lancet Diabetes & Endocrinology*, 4(2), 105–114. [https://doi.org/10.1016/S2213-8587\(15\)00477-5](https://doi.org/10.1016/S2213-8587(15)00477-5)
- Maersk, Belza, A., Holst, J. J., Fenger-Grøn, M., Pedersen, S. B., Astrup, A., & Richelsen, B. (2012). Satiety scores and satiety hormone response after sucrose-sweetened soft drink compared with isocaloric semi-skimmed milk and with non-caloric soft drink: A controlled trial. *European Journal of Clinical Nutrition*, 66(4), 523–529.
<https://doi.org/10.1038/ejcn.2011.223>
- Maersk, Belza, A., Stødkilde-Jørgensen, H., Ringgaard, S., Chabanova, E., Thomsen, H., Pedersen, S. B., Astrup, A., & Richelsen, B. (2012). Sucrose-sweetened beverages increase fat storage in the liver, muscle, and visceral fat depot: A 6-mo randomized intervention study. *The American Journal of Clinical Nutrition*, 95(2), 283–289.
<https://doi.org/10.3945/ajcn.111.022533>
- Mahar, A., & Duizer, L. M. (2007). The Effect of Frequency of Consumption of Artificial Sweeteners on Sweetness Liking by Women. *Journal of Food Science*, 72(9), S714–S718. <https://doi.org/10.1111/j.1750-3841.2007.00573.x>
- Malek, A. M., Hunt, K. J., DellaValle, D. M., Greenberg, D., St. Peter, J. V., & Marriott, B. P. (2018). Reported Consumption of Low-Calorie Sweetener in Foods, Beverages, and Food and Beverage Additions by US Adults: NHANES 2007–2012. *Current Developments in Nutrition*, 2(9). <https://doi.org/10.1093/cdn/nzy054>
- Malik, V. S., & Hu, F. B. (2012). Sweeteners and Risk of Obesity and Type 2 Diabetes: The Role of Sugar-Sweetened Beverages. *Current Diabetes Reports*, 12(2), 195–203.
<https://doi.org/10.1007/s11892-012-0259-6>
- Maloney, N. G., Christiansen, P., Harrold, J. A., Halford, J. C. G., & Hardman, C. A. (2018). The effect of experimentally induced food cravings on energy intake in frequent and non-frequent consumers of Low-calorie Sweetened (LCS) beverages. *Appetite*, 123, 458. <https://doi.org/10.1016/j.appet.2017.11.042>
- Margolskee, R. F., Dyer, J., Kokrashvili, Z., Salmon, K. S. H., Ilegems, E., Daly, K., Maillet, E. L., Ninomiya, Y., Mosinger, B., & Shirazi-Beechey, S. P. (2007). T1R3 and gustducin in gut sense sugars to regulate expression of Na⁺-glucose cotransporter 1. *Proceedings of the National Academy of Sciences*, 104(38), 15075–15080.
<https://doi.org/10.1073/pnas.0706678104>
- Markey, O., Le Jeune, J., & Lovegrove, J. A. (2016). Energy compensation following consumption of sugar-reduced products: A randomized controlled trial. *European Journal of Nutrition*, 55(6), 2137–2149. <https://doi.org/10.1007/s00394-015-1028-5>
- Mars, M., de Graaf, C., de Groot, L. C., & Kok, F. J. (2005). Decreases in fasting leptin and insulin concentrations after acute energy restriction and subsequent compensation in food intake. *The American Journal of Clinical Nutrition*, 81(3), 570–577.
<https://doi.org/10.1093/ajcn/81.3.570>
- Martens, M. J., Born, J. M., Lemmens, S. G., Karhunen, L., Heinecke, A., Goebel, R., Adam, T. C., & Westerterp-Plantenga, M. S. (2013). Increased sensitivity to food cues in the fasted state and decreased inhibitory control in the satiated state in the overweight. *The American Journal of Clinical Nutrition*, 97(3), 471–479.
<https://doi.org/10.3945/ajcn.112.044024>

- Martin, C. K., O'Neil, P. M., & Pawlow, L. (2006). Changes in Food Cravings during Low-Calorie and Very-Low-Calorie Diets*. *Obesity, 14*(1), 115–121. <https://doi.org/10.1038/oby.2006.14>
- Martínez Steele, E., Baraldi, L. G., Louzada, M. L. da C., Moubarac, J.-C., Mozaffarian, D., & Monteiro, C. A. (2016). Ultra-processed foods and added sugars in the US diet: Evidence from a nationally representative cross-sectional study. *BMJ Open, 6*(3), e009892. <https://doi.org/10.1136/bmjopen-2015-009892>
- Martínez Steele, E., Raubenheimer, D., Simpson, S. J., Baraldi, L. G., & Monteiro, C. A. (2018). Ultra-processed foods, protein leverage and energy intake in the USA. *Public Health Nutrition, 21*(1), 114–124. <https://doi.org/10.1017/S1368980017001574>
- Martínez-Navarrete, N., Camacho, M. M., Martínez-Lahuerta, J., Martínez-Monzó, J., & Fito, P. (2002). Iron deficiency and iron fortified foods—A review. *Food Research International, 35*(2–3), 225–231. [https://doi.org/10.1016/S0963-9969\(01\)00189-2](https://doi.org/10.1016/S0963-9969(01)00189-2)
- Martyn, D., Darch, M., Roberts, A., Lee, H., Yaqiong Tian, T., Kaburagi, N., & Belmar, P. (2018). Low-/No-Calorie Sweeteners: A Review of Global Intakes. *Nutrients, 10*(3), 357. <https://doi.org/10.3390/nu10030357>
- Masic, U., Harrold, J. A., Christiansen, P., Cuthbertson, D. J., Hardman, C. A., Robinson, E., & Halford, J. C. G. (2017). Effect S of non-nutritive s W eetened beverages on appet IT e during a C tive weig H t loss (SWITCH): Protocol for a randomized, controlled trial assessing the effects of non-nutritive sweetened beverages compared to water during a 12-week weight loss period and a follow up weight maintenance period. *Contemporary Clinical Trials, 53*, 80–88. <https://doi.org/10.1016/j.cct.2016.12.012>
- Matsuda, M., Liu, Y., Mahankali, S., Pu, Y., Mahankali, A., Wang, J., DeFronzo, R. A., Fox, P. T., & Gao, J. H. (1999). Altered hypothalamic function in response to glucose ingestion in obese humans. *Diabetes, 48*(9), 1801–1806. <https://doi.org/10.2337/diabetes.48.9.1801>
- Matsushita, Y., Mizoue, T., Takahashi, Y., Isogawa, A., Kato, M., Inoue, M., Noda, M., & Tsugane, S. (2009). Taste preferences and body weight change in Japanese adults: The JPHC Study. *International Journal of Obesity, 33*(10), 1191–1197. <https://doi.org/10.1038/ijo.2009.153>
- Mattes, R. D. (1996). Dietary Compensation by Humans for Supplemental Energy Provided as Ethanol or Carbohydrate in Fluids. *Physiology & Behavior, 59*(1), 179–187. [https://doi.org/10.1016/0031-9384\(95\)02007-1](https://doi.org/10.1016/0031-9384(95)02007-1)
- Mattes, R. D., & Popkin, B. M. (2009). Nonnutritive sweetener consumption in humans: Effects on appetite and food intake and their putative mechanisms. *The American Journal of Clinical Nutrition, 89*(1), 1–14. <https://doi.org/10.3945/ajcn.2008.26792>
- May, C. E., Vaziri, A., Lin, Y. Q., Grushko, O., Khabiri, M., Wang, Q.-P., Holme, K. J., Pletcher, S. D., Freddolino, P. L., Neely, G. G., & Dus, M. (2019). High Dietary Sugar Reshapes Sweet Taste to Promote Feeding Behavior in *Drosophila melanogaster*. *Cell Reports, 27*(6), 1675-1685.e7. <https://doi.org/10.1016/j.celrep.2019.04.027>
- Mayhew, D. A., Phil Comer, C., & Wayne Stargel, W. (2003). Food consumption and body weight changes with neotame, a new sweetener with intense taste: Differentiating effects of palatability from toxicity in dietary safety studies. *Regulatory Toxicology and Pharmacology, 38*(2), 124–143. [https://doi.org/10.1016/S0273-2300\(03\)00074-6](https://doi.org/10.1016/S0273-2300(03)00074-6)
- McCull, K. A. (1988). The sugar-fat seesaw. *Nutrition Bulletin, 13*(2), 114–118. <https://doi.org/10.1111/j.1467-3010.1988.tb00281.x>
- McCrorry, M. A., Fuss, P. J., McCallum, J. E., Yao, M., Vinken, A. G., Hays, N. P., & Roberts, S. B. (1999). Dietary variety within food groups: Association with energy intake and

- body fatness in men and women. *The American Journal of Clinical Nutrition*, 69(3), 440–447. <https://doi.org/10.1093/ajcn/69.3.440>
- McCrary, M. A., Fuss, P. J., Saltzman, E., & Roberts, S. B. (2000). Dietary Determinants of Energy Intake and Weight Regulation in Healthy Adults. *The Journal of Nutrition*, 130(2), 276S–279S. <https://doi.org/10.1093/jn/130.2.276S>
- McGill, R., & Appleton, K. M. (2009). Reasons for snack food choice and the prevalence of fruit snacking in Northern Ireland. *Proceedings of the Nutrition Society*, 68(OCE3), E144. <https://doi.org/10.1017/S0029665109991005>
- Mehat, K., Chen, Y., & Corpe, C. P. (2022). The Combined Effects of Aspartame and Acesulfame-K Blends on Appetite: A Systematic Review and Meta-Analysis of Randomized Clinical Trials. *Advances in Nutrition*, nmac072. <https://doi.org/10.1093/advances/nmac072>
- Mela, D. J. (1999). Food choice and intake: The human factor. *Proceedings of the Nutrition Society*, 58(3), 513–521. <https://doi.org/10.1017/S0029665199000683>
- 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. <https://doi.org/10.1016/j.appet.2006.02.006>
- Melby, C., Paris, H., Foright, R., & Peth, J. (2017). Attenuating the Biologic Drive for Weight Regain Following Weight Loss: Must What Goes Down Always Go Back Up? *Nutrients*, 9(5), 468. <https://doi.org/10.3390/nu9050468>
- Methven, L., Xiao, C., Cai, M., & Prescott, J. (2016). Rejection thresholds (RjT) of sweet likers and dislikers. *Food Quality and Preference*, 52, 74–80. <https://doi.org/10.1016/j.foodqual.2016.03.012>
- Meule, A. (2020a). The Psychology of Food Cravings: The Role of Food Deprivation. *Current Nutrition Reports*, 9(3), 251–257. <https://doi.org/10.1007/s13668-020-00326-0>
- Meule, A. (2020b). The Psychology of Food Cravings: The Role of Food Deprivation. *Current Nutrition Reports*, 9(3), 251–257. <https://doi.org/10.1007/s13668-020-00326-0>
- Meule, A. (2020c). Twenty Years of the Food Cravings Questionnaires: A Comprehensive Review. *Current Addiction Reports*, 7(1), 30–43. <https://doi.org/10.1007/s40429-020-00294-z>
- Meyer-Gerspach, A. C., Biesiekierski, J. R., Deloosse, E., Clevers, E., Rotondo, A., Rehfeld, J. F., Depoortere, I., Van Oudenhove, L., & Tack, J. (2018). Effects of caloric and noncaloric sweeteners on antroduodenal motility, gastrointestinal hormone secretion and appetite-related sensations in healthy subjects. *The American Journal of Clinical Nutrition*, 107(5), 707–716. <https://doi.org/10.1093/ajcn/nqy004>
- Miller, & Perez, V. (2014). Low-calorie sweeteners and body weight and composition: A meta-analysis of randomized controlled trials and prospective cohort studies. *The American Journal of Clinical Nutrition*, 100(3), 765–777. <https://doi.org/10.3945/ajcn.113.082826>
- Miller, W. C., Lindeman, A. K., Wallace, J., & Niederpruem, M. (1990). Diet composition, energy intake, and exercise in relation to body fat in men and women. *The American Journal of Clinical Nutrition*, 52(3), 426–430. <https://doi.org/10.1093/ajcn/52.3.426>
- Mintz, L. B., & O'Halloran, M. S. (2000). The Eating Attitudes Test: Validation With DSM-IV Eating Disorder Criteria. *Journal of Personality Assessment*, 74(3), 489–503. https://doi.org/10.1207/S15327752JPA7403_11
- Monsivais, P., Perrigue, M. M., & Drewnowski, A. (2007). Sugars and satiety: Does the type of sweetener make a difference? *The American Journal of Clinical Nutrition*, 86(1), 116–123. <https://doi.org/10.1093/ajcn/86.1.116>

- Monteiro, C. A., Cannon, G., Levy, R. B., Moubarac, J.-C., Louzada, M. L., Rauber, F., Khandpur, N., Cediel, G., Neri, D., Martinez-Steele, E., Baraldi, L. G., & Jaime, P. C. (2019). Ultra-processed foods: What they are and how to identify them. *Public Health Nutrition*, 22(5), 936–941. <https://doi.org/10.1017/S1368980018003762>
- Monteiro, C. A., Levy, R. B., Claro, R. M., Castro, I. R. R. de, & Cannon, G. (2010). A new classification of foods based on the extent and purpose of their processing. *Cadernos de Saúde Pública*, 26(11), 2039–2049. <https://doi.org/10.1590/S0102-311X2010001100005>
- Monteiro, C., & Cannon, G. (2012). *Product reformulation will not improve public health*. 3(9), 29.
- Moodie, R., Stuckler, D., Monteiro, C., Sheron, N., Neal, B., Thamarangsi, T., Lincoln, P., & Casswell, S. (2013). Profits and pandemics: Prevention of harmful effects of tobacco, alcohol, and ultra-processed food and drink industries. *The Lancet*, 381(9867), 670–679. [https://doi.org/10.1016/S0140-6736\(12\)62089-3](https://doi.org/10.1016/S0140-6736(12)62089-3)
- Mooradian, A. D., Smith, M., & Tokuda, M. (2017). The role of artificial and natural sweeteners in reducing the consumption of table sugar: A narrative review. *Clinical Nutrition ESPEN*, 18, 1–8. <https://doi.org/10.1016/j.clnesp.2017.01.004>
- Mora, M. R., & Dando, R. (2021). The sensory properties and metabolic impact of natural and synthetic sweeteners. *Comprehensive Reviews in Food Science and Food Safety*, 20(2), 1554–1583. <https://doi.org/10.1111/1541-4337.12703>
- Morales, I., & Berridge, K. C. (2020). ‘Liking’ and ‘wanting’ in eating and food reward: Brain mechanisms and clinical implications. *Physiology & Behavior*, 227, 113152. <https://doi.org/10.1016/j.physbeh.2020.113152>
- Mortensen, A. (2006). Sweeteners permitted in the European Union: Safety aspects. *Scandinavian Journal of Food and Nutrition*, 50(3), 104–116. <https://doi.org/10.1080/17482970600982719>
- Morton, G. J., Cummings, D. E., Baskin, D. G., Barsh, G. S., & Schwartz, M. W. (2006). Central nervous system control of food intake and body weight. *Nature*, 443(7109), 289–295. <https://doi.org/10.1038/nature05026>
- Moskowitz, H. R., Kluter, R. A., Westerling, J., & Jacobs, H. L. (1974). Sugar Sweetness and Pleasantness: Evidence for Different Psychological Laws. *Science*, 184(4136), 583–585. <https://doi.org/10.1126/science.184.4136.583>
- Mossavar-Rahmani, Y., Kamensky, V., Manson, J. E., Silver, B., Rapp, S. R., Haring, B., Beresford, S. A. A., Snetselaar, L., & Wassertheil-Smoller, S. (2018). *Artificially Sweetened Beverages and Stroke, Coronary Heart Disease, and All-Cause Mortality in the Women’s Health Initiative*. 8.
- Moubarac, J.-C., Batal, M., Louzada, M. L., Martinez Steele, E., & Monteiro, C. A. (2017). Consumption of ultra-processed foods predicts diet quality in Canada. *Appetite*, 108, 512–520. <https://doi.org/10.1016/j.appet.2016.11.006>
- Muenprasitvej, N., Tao, R., Nardone, S. J., & Cho, S. (2022). The Effect of Steviol Glycosides on Sensory Properties and Acceptability of Ice Cream. *Foods*, 11(12), 1745. <https://doi.org/10.3390/foods11121745>
- Mullee, A., Romaguera, D., Pearson-Stuttard, J., Viallon, V., Stepien, M., Freisling, H., Fagherazzi, G., Mancini, F. R., Boutron-Ruault, M.-C., Kühn, T., Kaaks, R., Boeing, H., Aleksandrova, K., Tjønneland, A., Halkjær, J., Overvad, K., Weiderpass, E., Skeie, G., Parr, C. L., ... Murphy, N. (2019). Association Between Soft Drink Consumption and Mortality in 10 European Countries. *JAMA Internal Medicine*, 179(11), 1479. <https://doi.org/10.1001/jamainternmed.2019.2478>

- Murtaugh, M. A., Herrick, J. S., Sweeney, C., Baumgartner, K. B., Guiliano, A. R., Byers, T., & Slattery, M. L. (2007). Diet Composition and Risk of Overweight and Obesity in Women Living in the Southwestern United States. *Journal of the American Dietetic Association, 107*(8), 1311–1321. <https://doi.org/10.1016/j.jada.2007.05.008>
- Nacef, M., Lelièvre-Desmas, M., Symoneaux, R., Jombart, L., Flahaut, C., & Chollet, S. (2019). Consumers' expectation and liking for cheese: Can familiarity effects resulting from regional differences be highlighted within a country? *Food Quality and Preference, 72*, 188–197. <https://doi.org/10.1016/j.foodqual.2018.10.004>
- Nakamura, Y., Sanematsu, K., Ohta, R., Shirosaki, S., Koyano, K., Nonaka, K., Shigemura, N., & Ninomiya, Y. (2008). Diurnal Variation of Human Sweet Taste Recognition Thresholds Is Correlated With Plasma Leptin Levels. *Diabetes, 57*(10), 2661–2665. <https://doi.org/10.2337/db07-1103>
- Nelson, G., Hoon, M. A., Chandrashekar, J., Zhang, Y., Ryba, N. J. P., & Zuker, C. S. (2001). Mammalian Sweet Taste Receptors. *Cell, 106*(3), 381–390. [https://doi.org/10.1016/S0092-8674\(01\)00451-2](https://doi.org/10.1016/S0092-8674(01)00451-2)
- NHS Digital. (2019). *Health survey for England*. <https://digital.nhs.uk/data-and-information/publications/statistical/health-survey-for-england/2019#>
- NICE. (2014). *National Institute for Health and Clinical Excellence (NICE). Obesity: Identification, assessment and management of overweight and obesity in children, young people and adults* (p. CG189).
- Niego, S. H., Pratt, E. M., & Agras, W. S. (1997). Subjective or objective binge: Is the distinction valid? *International Journal of Eating Disorders, 22*(3), 291–298. [https://doi.org/10.1002/\(SICI\)1098-108X\(199711\)22:3<291::AID-EAT8>3.0.CO;2-I](https://doi.org/10.1002/(SICI)1098-108X(199711)22:3<291::AID-EAT8>3.0.CO;2-I)
- Nielsen, S. J., Siega-Riz, A. M., & Popkin, B. M. (2002). Trends in Energy Intake in U.S. between 1977 and 1996: Similar Shifts Seen across Age Groups. *Obesity Research, 10*(5), 370–378. <https://doi.org/10.1038/oby.2002.51>
- Nielsen, Schmidt, J. B., le Roux, C. W., & Sjödin, A. (2019). Effects of Roux-en-Y Gastric Bypass and Sleeve Gastrectomy on Food Preferences and Potential Mechanisms Involved. *Current Obesity Reports*. <https://doi.org/10.1007/s13679-019-00354-0>
- Nishihara, T., Nozaki, T., Sawamoto, R., Komaki, G., Miyata, N., Hosoi, M., & Sudo, N. (2019). Effects of Weight Loss on Sweet Taste Preference and Palatability following Cognitive Behavioral Therapy for Women with Obesity. *Obesity Facts, 12*(5), 529–542. <https://doi.org/10.1159/000502236>
- Normand, M., Ritz, C., Mela, D., & Raben, A. (2021). Low-energy sweeteners and body weight: A citation network analysis. *BMJ Nutrition, Prevention & Health, 4*(1), 319–332. <https://doi.org/10.1136/bmjnph-2020-000210>
- O'Connor, D., Pang, M., Castelnuovo, G., Finlayson, G., Blaak, E., Gibbons, C., Navas-Carretero, S., Almiron-Roig, E., Harrold, J., Raben, A., & Martinez, J. A. (2021). A rational review on the effects of sweeteners and sweetness enhancers on appetite, food reward and metabolic/adiposity outcomes in adults. *Food & Function, 12*(2), 442–465. <https://doi.org/10.1039/D0FO02424D>
- Odegaard, A. O., Koh, W. P., Yuan, J.-M., Gross, M. D., & Pereira, M. A. (2012). Western-Style Fast Food Intake and Cardiometabolic Risk in an Eastern Country. *Circulation, 126*(2), 182–188. <https://doi.org/10.1161/CIRCULATIONAHA.111.084004>
- Odermatt, A. (2011). The Western-style diet: A major risk factor for impaired kidney function and chronic kidney disease. *American Journal of Physiology-Renal Physiology, 301*(5), F919–F931. <https://doi.org/10.1152/ajprenal.00068.2011>

- Olszewski, P. K., Wood, E. L., Klockars, A., & Levine, A. S. (2019). Excessive Consumption of Sugar: An Insatiable Drive for Reward. *Current Nutrition Reports*.
<https://doi.org/10.1007/s13668-019-0270-5>
- O'Neil, P. M., Theim, K. R., Boeka, A., Johnson, G., & Miller-Kovach, K. (2012). Changes in weight control behaviors and hedonic hunger during a 12-week commercial weight loss program. *Eating Behaviors*, *13*(4), 354–360.
<https://doi.org/10.1016/j.eatbeh.2012.06.002>
- Othus, M., Zhang, M.-J., & Gale, R. P. (2022). Clinical trials: Design, endpoints and interpretation of outcomes. *Bone Marrow Transplantation*, *57*(3), 338–342.
<https://doi.org/10.1038/s41409-021-01542-0>
- Oustric, P., Beaulieu, K., Casanova, N., O'Connor, D., Gibbons, C., Hopkins, M., Blundell, J., & Finlayson, G. (2021). Food Liking but Not Wanting Decreases after Controlled Intermittent or Continuous Energy Restriction to $\geq 5\%$ Weight Loss in Women with Overweight/Obesity. *Nutrients*, *13*(1), 182. <https://doi.org/10.3390/nu13010182>
- Oustric, P., Gibbons, C., Beaulieu, K., Blundell, J., & Finlayson, G. (2018). Changes in food reward during weight management interventions - a systematic review: Role of food reward during weight management. *Obesity Reviews*. <https://doi.org/10.1111/obr.12754>
- Oustric, P., Thivel, D., Dalton, M., Beaulieu, K., Gibbons, C., Hopkins, M., Blundell, J., & Finlayson, G. (2020). Measuring food preference and reward: Application and cross-cultural adaptation of the Leeds Food Preference Questionnaire in human experimental research. *Food Quality and Preference*, *80*, 103824.
<https://doi.org/10.1016/j.foodqual.2019.103824>
- Pangborn, R. M. (1970). Individual variation in affective responses to taste stimuli. *Psychonomic Science*, *21*(2), 125–126. <https://doi.org/10.3758/BF03335798>
- Patriarca, L., Magerowski, G., & Alonso-Alonso, M. (2017). Functional neuroimaging in obesity: *Current Opinion in Endocrinology & Diabetes and Obesity*, *24*(3), 260–265.
<https://doi.org/10.1097/MED.0000000000000340>
- Peeters, A., Barendregt, J. J., Willekens, F., Mackenbach, J. P., Mamun, A. A., Bonneux, L., & and for NEDCOM, the Netherlands Epidemiology and Demography Compression of Morbidity Research Group*. (2003). Obesity in Adulthood and Its Consequences for Life Expectancy: A Life-Table Analysis. *Annals of Internal Medicine*, *138*(1), 24.
<https://doi.org/10.7326/0003-4819-138-1-200301070-00008>
- Pelchat, M. L. (2002). *Of human bondage: Food craving, obsession, compulsion, and addiction*. 6.
- Pelchat, M. L., & Schaefer, S. (2000). Dietary monotony and food cravings in young and elderly adults. *Physiology & Behavior*, *68*(3), 353–359. [https://doi.org/10.1016/S0031-9384\(99\)00190-0](https://doi.org/10.1016/S0031-9384(99)00190-0)
- Pepino, M. Y., & Mennella, J. A. (2012). Habituation to the pleasure elicited by sweetness in lean and obese women. *Appetite*, *58*(3), 800–805.
<https://doi.org/10.1016/j.appet.2012.01.026>
- Peterson, J. M., Bartoshuk, L. M., & Duffy, V. B. (1999). Intensity and preference for sweetness is influenced by genetic taste variation. *Journal of American Dietetic Association*, *99*(9), A28.
- Petrus, R. R., do Amaral Sobral, P. J., Tadini, C. C., & Gonçalves, C. B. (2021). The NOVA classification system: A critical perspective in food science. *Trends in Food Science & Technology*, *116*, 603–608. <https://doi.org/10.1016/j.tifs.2021.08.010>
- Pielak, M., Czarniecka-Skubina, E., Trafiałek, J., & Głuchowski, A. (2019). Contemporary Trends and Habits in the Consumption of Sugar and Sweeteners—A Questionnaire

- Survey among Poles. *International Journal of Environmental Research and Public Health*, 16(7), 1164. <https://doi.org/10.3390/ijerph16071164>
- Piernas, C., Tate, D. F., Wang, X., & Popkin, B. M. (2013). Does diet-beverage intake affect dietary consumption patterns? Results from the Choose Healthy Options Consciously Everyday (CHOICE) randomized clinical trial. *The American Journal of Clinical Nutrition*, 97(3), 604–611. <https://doi.org/10.3945/ajcn.112.048405>
- Poppitt, S., Swann, D., Black, A., & Prentice, A. (1998). Assessment of selective under-reporting of food intake by both obese and non-obese women in a metabolic facility. *International Journal of Obesity*, 22(4), 303–311. <https://doi.org/10.1038/sj.ijo.0800584>
- Porikos, P., Hesser, F., & Itallie, B. V. (1982). *Caloric Regulation in Normal-Weight Men Maintained on a Palatable Diet of Conventional Foods I*. 8.
- Portmann, M.-O., & Kilcast, D. (1996). Psychophysical characterization of new sweeteners of commercial importance for the EC food industry. *Food Chemistry*, 56(3), 291–302. [https://doi.org/10.1016/0308-8146\(96\)00026-X](https://doi.org/10.1016/0308-8146(96)00026-X)
- Poti, J. M., Braga, B., & Qin, B. (2017). Ultra-processed Food Intake and Obesity: What Really Matters for Health—Processing or Nutrient Content? *Current Obesity Reports*, 6(4), 420–431. <https://doi.org/10.1007/s13679-017-0285-4>
- Prakash, I., Markosyan, A., & Bunders, C. (2014). Development of Next Generation Stevia Sweetener: Rebaudioside M. *Foods*, 3(1), 162–175. <https://doi.org/10.3390/foods3010162>
- Price, M., Higgs, S., & Lee, M. (2015). Self-reported eating traits: Underlying components of food responsiveness and dietary restriction are positively related to BMI. *Appetite*, 95, 203–210. <https://doi.org/10.1016/j.appet.2015.07.006>
- Public Health England. (2017). *Health matters_ obesity and the food environment—GOV.UK.pdf*. <https://www.gov.uk/government/publications/health-matters-obesity-and-the-food-environment/health-matters-obesity-and-the-food-environment--2>
- Raben, A., Møller, BenteK., Flint, A., Vasilaras, TatjanaH., Christina Møller, A., Juul Holst, J., & Astrup, A. (2011). Increased postprandial glycaemia, insulinemia, and lipidemia after 10 weeks' sucrose-rich diet compared to an artificially sweetened diet: A randomised controlled trial. *Food & Nutrition Research*, 55(1), 5961. <https://doi.org/10.3402/fnr.v55i0.5961>
- Raben, A., Vasilaras, T. H., Møller, A. C., & Astrup, A. (2002). Sucrose compared with artificial sweeteners: Different effects on ad libitum food intake and body weight after 10 wk of supplementation in overweight subjects. *The American Journal of Clinical Nutrition*, 76(4), 721–729. <https://doi.org/10.1093/ajcn/76.4.721>
- Rahemtulla, Z., Baldwin, C., Spiro, A., Mcgough, C., Norman, A., Frost, G., Cunningham, D., & Andreyev, H. (2005). The palatability of milk-based and non-milk-based nutritional supplements in gastrointestinal cancer and the effect of chemotherapy. *Clinical Nutrition*, 24(6), 1029–1037. <https://doi.org/10.1016/j.clnu.2005.08.003>
- Rangel, A. (2013). Regulation of dietary choice by the decision-making circuitry. *Nature Neuroscience*, 16(12), 1717–1724. <https://doi.org/10.1038/nn.3561>
- Rangel, A., & Hare, T. (2010). Neural computations associated with goal-directed choice. *Current Opinion in Neurobiology*, 20(2), 262–270. <https://doi.org/10.1016/j.conb.2010.03.001>
- Rauber, F., Louzada, M. L. da C., Martinez Steele, E., Rezende, L. F. M. de, Millett, C., Monteiro, C. A., & Levy, R. B. (2019). Ultra-processed foods and excessive free sugar intake in the UK: A nationally representative cross-sectional study. *BMJ Open*, 9(10), e027546. <https://doi.org/10.1136/bmjopen-2018-027546>

- Raynor, H. A. (2012). Can limiting dietary variety assist with reducing energy intake and weight loss? *Physiology & Behavior*, *106*(3), 356–361. <https://doi.org/10.1016/j.physbeh.2012.03.012>
- Raynor, H. A., & Epstein, L. H. (2001). Dietary variety, energy regulation, and obesity. *Psychological Bulletin*, *127*(3), 325–341. <https://doi.org/10.1037/0033-2909.127.3.325>
- Raynor, H. A., & Epstein, L. H. (2003). *The relative-reinforcing value of food under differing levels of food deprivation and restriction*. 10.
- Reardon, T., Timmer, C. P., Barrett, C. B., & Berdegue, J. (2003). The Rise of Supermarkets in Africa, Asia, and Latin America. *American Journal of Agricultural Economics*, *85*(5), 1140–1146. <https://doi.org/10.1111/j.0092-5853.2003.00520.x>
- Reed, D. R., & McDaniel, A. H. (2006). The Human Sweet Tooth. *BMC Oral Health*, *6*(S1), S17. <https://doi.org/10.1186/1472-6831-6-S1-S17>
- Reed, D. R., Tanaka, T., & McDaniel, A. H. (2006). Diverse tastes: Genetics of sweet and bitter perception. *Physiology & Behavior*, *88*(3), 215–226. <https://doi.org/10.1016/j.physbeh.2006.05.033>
- Reid, M., Hammersley, R., & Duffy, M. (2010). Effects of sucrose drinks on macronutrient intake, body weight, and mood state in overweight women over 4 weeks. *Appetite*, *55*(1), 130–136. <https://doi.org/10.1016/j.appet.2010.05.001>
- Reid, M., Hammersley, R., Duffy, M., & Ballantyne, C. (2014). Effects on obese women of the sugar sucrose added to the diet over 28 d: A quasi-randomised, single-blind, controlled trial. *British Journal of Nutrition*, *111*(3), 563–570. <https://doi.org/10.1017/S0007114513002687>
- Reid, M., Hammersley, R., Hill, A. J., & Skidmore, P. (2007). Long-term dietary compensation for added sugar: Effects of supplementary sucrose drinks over a 4-week period. *British Journal of Nutrition*, *97*(01), 193. <https://doi.org/10.1017/S0007114507252705>
- Ribeiro, G., Camacho, M., Santos, O., Pontes, C., Torres, S., & Oliveira-Maia, A. J. (2018). Association between hedonic hunger and body-mass index versus obesity status. *Scientific Reports*, *8*(1), 5857. <https://doi.org/10.1038/s41598-018-23988-x>
- Rimal, A., Fletcher, S. M., McWatters, K. H., Misra, S. K., & Deodhar, S. (2001). *Perception of food safety and changes in food consumption habits: A consumer analysis*. 10.
- Rios-Leyvraz, M., & Montez, J. (2022). *Health effects of the use of non-sugar sweeteners*.
- Rippin, H. L., Hutchinson, J., Jewell, J., Breda, J. J., & Cade, J. E. (2019). Comparison of consumed portion sizes and on-pack serving sizes of UK energy dense foods. *Appetite*, *134*, 193–203. <https://doi.org/10.1016/j.appet.2018.12.018>
- Roberts, J. R. (2015). The Paradox of Artificial Sweeteners in Managing Obesity. *Current Gastroenterology Reports*, *17*(1), 1. <https://doi.org/10.1007/s11894-014-0423-z>
- Robinson, E., Boyland, E., Chisholm, A., Harrold, J., Maloney, N. G., Marty, L., Mead, B. R., Noonan, R., & Hardman, C. A. (2021). Obesity, eating behavior and physical activity during COVID-19 lockdown: A study of UK adults. *Appetite*, *156*, 104853. <https://doi.org/10.1016/j.appet.2020.104853>
- Robinson, T. E., & Berridge. (1993). The neural basis of drug craving: An incentive sensitization theory of addiction. *Brain Research Reviews*, *18*, 247–291.
- Rocha, I. F. de O., & Bolini, H. M. A. (2015). Passion fruit juice with different sweeteners: Sensory profile by descriptive analysis and acceptance. *Food Science & Nutrition*, *3*(2), 129–139. <https://doi.org/10.1002/fsn3.195>
- Rodin, J. (1990). Comparative effects of fructose, aspartame, glucose, and water preloads on calorie and macronutrient intake. *The American Journal of Clinical Nutrition*, *51*(3), 428–435. <https://doi.org/10.1093/ajcn/51.3.428>

- Rodin, J., Moskowitz, H. R., & Bray, G. A. (1976). Relationship between obesity, weight loss, and taste responsiveness. *Physiology & Behavior*, *17*(4), 591–597. [https://doi.org/10.1016/0031-9384\(76\)90157-8](https://doi.org/10.1016/0031-9384(76)90157-8)
- Rodin, J., Slochow, J., & Fleming, B. (1977). Effects of degree of obesity, age of onset, and weight loss on responsiveness to sensory and external stimuli. *Journal of Comparative and Physiological Psychology*, *91*(3), 586–597. <https://doi.org/10.1037/h0077354>
- Rogers, Carlyle, J.-A., Hill, A. J., & Blundell, J. E. (1988). Uncoupling sweet taste and calories: Comparison of the effects of glucose and three intense sweeteners on hunger and food intake. *Physiology & Behavior*, *43*(5), 547–552. [https://doi.org/10.1016/0031-9384\(88\)90207-7](https://doi.org/10.1016/0031-9384(88)90207-7)
- Rogers, Hogenkamp, P. S., de Graaf, C., Higgs, S., Lluch, A., Ness, A. R., Penfold, C., Perry, R., Putz, P., Yeomans, M. R., & Mela, D. J. (2016). Does low-energy sweetener consumption affect energy intake and body weight? A systematic review, including meta-analyses, of the evidence from human and animal studies. *International Journal of Obesity*, *40*(3), 381–394. <https://doi.org/10.1038/ijo.2015.177>
- Rogers, P. J. (2018). The role of low-calorie sweeteners in the prevention and management of overweight and obesity: Evidence v . conjecture. *Proceedings of the Nutrition Society*, *77*(3), 230–238. <https://doi.org/10.1017/S0029665117004049>
- Rogers, P. J., Fleming, H. C., & Blundell, J. E. (1990). Aspartame ingested without tasting inhibits hunger and food intake. *Physiology & Behavior*, *47*(6), 1239–1243. [https://doi.org/10.1016/0031-9384\(90\)90377-G](https://doi.org/10.1016/0031-9384(90)90377-G)
- Rolls, B. J., Hetherington, M., & Burley, V. J. (1988). The specificity of satiety: The influence of foods of different macronutrient content on the development of satiety. *Physiology & Behavior*, *43*(2), 145–153. [https://doi.org/10.1016/0031-9384\(88\)90230-2](https://doi.org/10.1016/0031-9384(88)90230-2)
- Rolls, B. J., Hetherington, M., & Laster, L. J. (1988). *Comparison of the Effects of Aspartame and Sucrose on Appetite and Food Intake*. 6.
- Rolls, B. J., Laster, L. J., & Summerfelt, A. (1989). Hunger and food intake following consumption of low-calorie foods. *Appetite*, *13*(2), 115–127. [https://doi.org/10.1016/0195-6663\(89\)90109-8](https://doi.org/10.1016/0195-6663(89)90109-8)
- Rolls, E., Rolls, B., & Rowe, E. (1983). Sensory-specific and motivation-specific satiety for the sight and taste of food and water in man. *Physiology & Behavior*, *30*(2), 185–192. [https://doi.org/10.1016/0031-9384\(83\)90003-3](https://doi.org/10.1016/0031-9384(83)90003-3)
- Rolls, Kim, S., & Fedoroff, I. C. (1990). Effects of drinks sweetened with sucrose or aspartame on hunger, thirst and food intake in men. *Physiology & Behavior*, *48*(1), 19–26. [https://doi.org/10.1016/0031-9384\(90\)90254-2](https://doi.org/10.1016/0031-9384(90)90254-2)
- Rosen, J. C., Vara, L., Wendt, S., & Leitenberg, H. (1990). Validity studies of the eating disorder examination. *International Journal of Eating Disorders*, *9*(5), 519–528. [https://doi.org/10.1002/1098-108X\(199009\)9:5<519::AID-EAT2260090507>3.0.CO;2-K](https://doi.org/10.1002/1098-108X(199009)9:5<519::AID-EAT2260090507>3.0.CO;2-K)
- Ross, S. E., Flynn, J. I., & Pate, R. R. (2016). What is really causing the obesity epidemic? A review of reviews in children and adults. *Journal of Sports Sciences*, *34*(12), 1148–1153. <https://doi.org/10.1080/02640414.2015.1093650>
- Rothmund, Y., Preuschhof, C., Bohner, G., Bauknecht, H.-C., Klingebiel, R., Flor, H., & Klapp, B. F. (2007). Differential activation of the dorsal striatum by high-calorie visual food stimuli in obese individuals. *NeuroImage*, *37*(2), 410–421. <https://doi.org/10.1016/j.neuroimage.2007.05.008>
- Roze, M., Crucean, D., Diler, G., Rannou, C., Catanéo, C., Jonchère, C., Le-Bail, A., & Le-Bail, P. (2021). Impact of Maltitol and Sorbitol on Technological and Sensory Attributes of Biscuits. *Foods*, *10*(11), 2545. <https://doi.org/10.3390/foods10112545>

- Sacchi, R., Caporaso, N., Squadrilli, G. A., Paduano, A., Ambrosino, M. L., Cavella, S., & Genovese, A. (2019). Sensory profile, biophenolic and volatile compounds of an artisanal ice cream ('gelato') functionalised using extra virgin olive oil. *International Journal of Gastronomy and Food Science*, *18*, 100173. <https://doi.org/10.1016/j.ijgfs.2019.100173>
- Sadler, C. R., Grassby, T., Hart, K., Raats, M., Sokolović, M., & Timotijevic, L. (2021). Processed food classification: Conceptualisation and challenges. *Trends in Food Science & Technology*, *112*, 149–162. <https://doi.org/10.1016/j.tifs.2021.02.059>
- Sadler, M. J., McNulty, H., & Gibson, S. (2015). Sugar-Fat Seesaw: A Systematic Review of the Evidence. *Critical Reviews in Food Science and Nutrition*, *55*(3), 338–356. <https://doi.org/10.1080/10408398.2011.654013>
- Sakurai, M., Nakamura, K., Miura, K., Takamura, T., Yoshita, K., Nagasawa, S. Y., Morikawa, Y., Ishizaki, M., Kido, T., Naruse, Y., Suwazono, Y., Sasaki, S., & Nakagawa, H. (2014). Sugar-sweetened beverage and diet soda consumption and the 7-year risk for type 2 diabetes mellitus in middle-aged Japanese men. *Eur J Nutr*, *8*.
- Salbe, A. D., DelParigi, A., Pratley, R. E., Drewnowski, A., & Tataranni, P. A. (2004). Taste preferences and body weight changes in an obesity-prone population. *The American Journal of Clinical Nutrition*, *79*(3), 372–378. <https://doi.org/10.1093/ajcn/79.3.372>
- Sartor, F., Donaldson, L. F., Markland, D. A., Loveday, H., Jackson, M. J., & Kubis, H.-P. (2011). Taste perception and implicit attitude toward sweet related to body mass index and soft drink supplementation. *Appetite*, *57*(1), 237–246. <https://doi.org/10.1016/j.appet.2011.05.107>
- Scarborough, P., Burg, M. R., Foster, C., Swinburn, B., Sacks, G., Rayner, M., Webster, P., & Allender, S. (2011). Increased energy intake entirely accounts for increase in body weight in women but not in men in the UK between 1986 and 2000. *British Journal of Nutrition*, *105*(9), 1399–1404. <https://doi.org/10.1017/S0007114510005076>
- Schiffman, S. S., Reilly, D. A., & Clark, T. B. (1979). Qualitative differences among sweeteners. *Physiology & Behavior*, *23*(1), 1–9. [https://doi.org/10.1016/0031-9384\(79\)90113-6](https://doi.org/10.1016/0031-9384(79)90113-6)
- Schoeller, D. A. (2009). The energy balance equation: Looking back and looking forward are two very different views. *Nutrition Reviews*, *67*(5), 249–254. <https://doi.org/10.1111/j.1753-4887.2009.00197.x>
- Schoeller, D. A., Cella, L. K., Sinha, M. K., & Caro, J. F. (1997). Entrainment of the diurnal rhythm of plasma leptin to meal timing. *Journal of Clinical Investigation*, *100*(7), 1882–1887. <https://doi.org/10.1172/JCI119717>
- Schröder, H., Fito, M., & Covas, M. I. (2007). Association of fast food consumption with energy intake, diet quality, body mass index and the risk of obesity in a representative Mediterranean population. *British Journal of Nutrition*, *98*(6), 1274–1280. <https://doi.org/10.1017/S0007114507781436>
- Schulz, S., & Laessle, R. G. (2010). Associations of negative affect and eating behaviour in obese women with and without binge eating disorder. *Eating and Weight Disorders - Studies on Anorexia, Bulimia and Obesity*, *15*(4), e287–e293. <https://doi.org/10.1007/BF03325311>
- Schwartz, M. W. (2006). Central Nervous System Regulation of Food Intake. *Obesity*, *14*(2S), 1S-8S. <https://doi.org/10.1038/oby.2006.275>
- Schwartz, M. W., Woods, S. C., Seeley, R. J., Barsh, G. S., Baskin, D. G., & Leibel, R. L. (2003). Is the Energy Homeostasis System Inherently Biased Toward Weight Gain? *Diabetes*, *52*(2), 232–238. <https://doi.org/10.2337/diabetes.52.2.232>
- Scientific Advisory Committee on Nutrition. (2015). *Carbohydrates and health*.

- Sclafani, A. (2007). Sweet taste signaling in the gut. *Proceedings of the National Academy of Sciences*, *104*(38), 14887–14888. <https://doi.org/10.1073/pnas.0707410104>
- Scott, K. (2004). The sweet and the bitter of mammalian taste. *Current Opinion in Neurobiology*, *14*(4), 423–427. <https://doi.org/10.1016/j.conb.2004.06.003>
- Sevim, Y., & Yağar, H. (2022). Changes in healthy women's food preferences, taste, body, and mood before and during menstruation. *Izmir Democracy University Health Sciences Journal*, *5*(2), 402–418. <https://doi.org/10.52538/iduhes.1142164>
- Shigemura, N., Ohta, R., Kusakabe, Y., Miura, H., Hino, A., Koyano, K., Nakashima, K., & Ninomiya, Y. (2004). Leptin Modulates Behavioral Responses to Sweet Substances by Influencing Peripheral Taste Structures. *Endocrinology*, *145*(2), 839–847. <https://doi.org/10.1210/en.2003-0602>
- Shoar, S., Naderan, M., Shoar, N., Modukuru, V. R., & Mahmoodzadeh, H. (2019). Alteration Pattern of Taste Perception After Bariatric Surgery: A Systematic Review of Four Taste Domains. *Obesity Surgery*. <https://doi.org/10.1007/s11695-019-03730-w>
- Small, D. M. (2001). Changes in brain activity related to eating chocolate: From pleasure to aversion. *Brain*, *124*(9), 1720–1733. <https://doi.org/10.1093/brain/124.9.1720>
- Small, D. M., Jones-Gotman, M., & Dagher, A. (2003). Feeding-induced dopamine release in dorsal striatum correlates with meal pleasantness ratings in healthy human volunteers. *NeuroImage*, *19*(4), 1709–1715. [https://doi.org/10.1016/S1053-8119\(03\)00253-2](https://doi.org/10.1016/S1053-8119(03)00253-2)
- Smeets, de Graaf, C., Stafleu, A., van Osch, M. J., & van der Grond, J. (2005). Functional magnetic resonance imaging of human hypothalamic responses to sweet taste and calories. *The American Journal of Clinical Nutrition*, *82*(5), 1011–1016. <https://doi.org/10.1093/ajcn/82.5.1011>
- Smeets, P. A. M., Weijzen, P., de Graaf, C., & Viergever, M. A. (2011). Consumption of caloric and non-caloric versions of a soft drink differentially affects brain activation during tasting. *NeuroImage*, *54*(2), 1367–1374. <https://doi.org/10.1016/j.neuroimage.2010.08.054>
- Snoek, H. M., Huntjens, L., van Gemert, L. J., de Graaf, C., & Weenen, H. (2004). Sensory-specific satiety in obese and normal-weight women. *The American Journal of Clinical Nutrition*, *80*(4), 823–831. <https://doi.org/10.1093/ajcn/80.4.823>
- Søberg, S., Sandholt, C. H., Jespersen, N. Z., Toft, U., Madsen, A. L., von Holstein-Rathlou, S., Grevengoed, T. J., Christensen, K. B., Bredie, W. L. P., Potthoff, M. J., Solomon, T. P. J., Scheele, C., Linneberg, A., Jørgensen, T., Pedersen, O., Hansen, T., Gillum, M. P., & Grarup, N. (2017). FGF21 Is a Sugar-Induced Hormone Associated with Sweet Intake and Preference in Humans. *Cell Metabolism*, *25*(5), 1045-1053.e6. <https://doi.org/10.1016/j.cmet.2017.04.009>
- Solomi, L. (2020). *Diet cola and glycaemia: The acute effects of a preload containing the non-nutritive sweeteners aspartame and acesulfame-K on the glycaemic response to a glucose load*. 16.
- Sørensen, L. B., Møller, P., Flint, A., Martens, M., & Raben, A. (2003). Effect of sensory perception of foods on appetite and food intake: A review of studies on humans. *International Journal of Obesity*, *27*(10), 1152–1166. <https://doi.org/10.1038/sj.ijo.0802391>
- Sørensen, Raben, A., Stender, S., & Astrup, A. (2005). Effect of sucrose on inflammatory markers in overweight humans. *The American Journal of Clinical Nutrition*, *82*(2), 421–427.
- Souza, L., Martins, K., Cordeiro, M., Rodrigues, Y., Rafacho, B., & Bomfim, R. (2018). Do Food Intake and Food Cravings Change during the Menstrual Cycle of Young Women?

Revista Brasileira de Ginecologia e Obstetrícia / RBGO Gynecology and Obstetrics, 40(11), 686–692. <https://doi.org/10.1055/s-0038-1675831>

- Spiegel, T. A., Shrager, E. E., & Stellar, E. (1989). Responses of lean and obese subjects to preloads, deprivation, and palatability. *Appetite*, 13(1), 45–69. [https://doi.org/10.1016/0195-6663\(89\)90026-3](https://doi.org/10.1016/0195-6663(89)90026-3)
- Steel, D., Kemps, E., & Tiggemann, M. (2006). Effects of hunger and visuo-spatial interference on imagery-induced food cravings. *Appetite*, 46(1), 36–40. <https://doi.org/10.1016/j.appet.2005.11.001>
- Steinert, R. E., Frey, F., Töpfer, A., Drewe, J., & Beglinger, C. (2011). Effects of carbohydrate sugars and artificial sweeteners on appetite and the secretion of gastrointestinal satiety peptides. *British Journal of Nutrition*, 105(9), 1320–1328. <https://doi.org/10.1017/S000711451000512X>
- Stellman, S. D., & Garfinkel, L. (1986). Artificial sweetener use and one-year weight change among women. *Preventive Medicine*, 15(2), 195–202. [https://doi.org/10.1016/0091-7435\(86\)90089-7](https://doi.org/10.1016/0091-7435(86)90089-7)
- Stender, S., Dyerberg, J., & Astrup, A. (2007). Fast food: Unfriendly and unhealthy. *International Journal of Obesity*, 31(6), 887–890. <https://doi.org/10.1038/sj.ijo.0803616>
- Stice, E., Burger, K., & Yokum, S. (2013). Caloric deprivation increases responsivity of attention and reward brain regions to intake, anticipated intake, and images of palatable foods. *NeuroImage*, 67, 322–330. <https://doi.org/10.1016/j.neuroimage.2012.11.028>
- Stice, E., Spoor, S., Bohon, C., Veldhuizen, M. G., & Small, D. M. (2008). Relation of reward from food intake and anticipated food intake to obesity: A functional magnetic resonance imaging study. *Journal of Abnormal Psychology*, 117(4), 924–935. <https://doi.org/10.1037/a0013600>
- Stoeckel, L. E., Cox, J. E., Cook, E. W., & Weller, R. E. (2007). Motivational state modulates the hedonic value of food images differently in men and women. *Appetite*, 48(2), 139–144. <https://doi.org/10.1016/j.appet.2006.07.079>
- Stoeckel, L. E., Weller, R. E., Cook, E. W., Twieg, D. B., Knowlton, R. C., & Cox, J. E. (2008). Widespread reward-system activation in obese women in response to pictures of high-calorie foods. *NeuroImage*, 41(2), 636–647. <https://doi.org/10.1016/j.neuroimage.2008.02.031>
- 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. [https://doi.org/10.1016/0022-3999\(85\)90010-8](https://doi.org/10.1016/0022-3999(85)90010-8)
- Swinburn, Sacks, G., Hall, K. D., McPherson, K., Finegood, D. T., Moodie, M. L., & Gortmaker, S. L. (2011). The global obesity pandemic: Shaped by global drivers and local environments. *The Lancet*, 378(9793), 804–814. [https://doi.org/10.1016/S0140-6736\(11\)60813-1](https://doi.org/10.1016/S0140-6736(11)60813-1)
- Swithers. (2013). Artificial sweeteners produce the counterintuitive effect of inducing metabolic derangements. *Trends in Endocrinology & Metabolism*, 24(9), 431–441. <https://doi.org/10.1016/j.tem.2013.05.005>
- Sylvetsky, A. C., Jin, Y., Mathieu, K., DiPietro, L., Rother, K. I., & Talegawkar, S. A. (2017). Low-Calorie Sweeteners: Disturbing the Energy Balance Equation in Adolescents?: Low-Calorie Sweeteners and Obesity in Adolescents. *Obesity*, 25(12), 2049–2054. <https://doi.org/10.1002/oby.22005>
- Sylvetsky, Brown, R. J., Blau, J. E., Walter, M., & Rother, K. I. (2016). Hormonal responses to non-nutritive sweeteners in water and diet soda. *Nutrition & Metabolism*, 13(1), 71. <https://doi.org/10.1186/s12986-016-0129-3>

- Sylvetsky, & Rother, K. I. (2016). Trends in the consumption of low-calorie sweeteners. *Physiology & Behavior, 164*, 446–450. <https://doi.org/10.1016/j.physbeh.2016.03.030>
- Sylvetsky, Welsh, J. A., Brown, R. J., & Vos, M. B. (2012). Low-calorie sweetener consumption is increasing in the United States. *American Journal of Clinical Nutrition, 96*(3), 640–646. <https://doi.org/10.3945/ajcn.112.034751>
- Taetzsch, A., Roberts, S. B., Gilhooly, C. H., Lichtenstein, A. H., Krauss, A. J., Bukhari, A., Martin, E., Hatch-McChesney, A., & Das, S. K. (2020). Food cravings: Associations with dietary intake and metabolic health. *Appetite, 152*, 104711. <https://doi.org/10.1016/j.appet.2020.104711>
- Tahmassebi, J. F., & BaniHani, A. (2019). Impact of soft drinks to health and economy: A critical review. *European Archives of Paediatric Dentistry*. <https://doi.org/10.1007/s40368-019-00458-0>
- Talukdar, S., Owen, B. M., Song, P., Hernandez, G., Zhang, Y., Zhou, Y., Scott, W. T., Paratala, B., Turner, T., Smith, A., Bernardo, B., Müller, C. P., Tang, H., Mangelsdorf, D. J., Goodwin, B., & Kliewer, S. A. (2016). FGF21 Regulates Sweet and Alcohol Preference. *Cell Metabolism, 23*(2), 344–349. <https://doi.org/10.1016/j.cmet.2015.12.008>
- Tan, & Tucker, R. (2019). Sweet Taste as a Predictor of Dietary Intake: A Systematic Review. *Nutrients, 11*(1), 94. <https://doi.org/10.3390/nu11010094>
- Tang, D. W., Fellows, L. K., & Dagher, A. (2014). Behavioral and Neural Valuation of Foods Is Driven by Implicit Knowledge of Caloric Content. *Psychological Science, 25*(12), 2168–2176. <https://doi.org/10.1177/0956797614552081>
- Tate, D. F., Turner-McGrievy, G., Lyons, E., Stevens, J., Erickson, K., Polzien, K., Diamond, M., Wang, X., & Popkin, B. (2012). Replacing caloric beverages with water or diet beverages for weight loss in adults: Main results of the Choose Healthy Options Consciously Everyday (CHOICE) randomized clinical trial. *The American Journal of Clinical Nutrition, 95*(3), 555–563. <https://doi.org/10.3945/ajcn.111.026278>
- Te Morenga, L., Mallard, S., & Mann, J. (2012). Dietary sugars and body weight: Systematic review and meta-analyses of randomised controlled trials and cohort studies. *BMJ, 346*(jan15 3), e7492–e7492. <https://doi.org/10.1136/bmj.e7492>
- Temizkan, S., Deyneli, O., Yasar, M., Arpa, M., Gunes, M., Yazici, D., Sirikci, O., Haklar, G., Imeryuz, N., & Yavuz, D. G. (2015). Sucralose enhances GLP-1 release and lowers blood glucose in the presence of carbohydrate in healthy subjects but not in patients with type 2 diabetes. *European Journal of Clinical Nutrition, 69*(2), 162–166. <https://doi.org/10.1038/ejcn.2014.208>
- Temple, J. L., Bulkley, A. M., Badawy, R. L., Krause, N., McCann, S., & Epstein, L. H. (2009). Differential effects of daily snack food intake on the reinforcing value of food in obese and nonobese women. *The American Journal of Clinical Nutrition, 90*(2), 304–313. <https://doi.org/10.3945/ajcn.2008.27283>
- Tey, S. L., Salleh, N. B., Henry, C. J., & Forde, C. G. (2017a). Effects of non-nutritive (artificial vs natural) sweeteners on 24-h glucose profiles. *European Journal of Clinical Nutrition, 71*(9), 1129–1132. <https://doi.org/10.1038/ejcn.2017.37>
- Tey, S. L., Salleh, N. B., Henry, J., & Forde, C. G. (2017b). Effects of aspartame-, monk fruit-, stevia- and sucrose-sweetened beverages on postprandial glucose, insulin and energy intake. *International Journal of Obesity, 41*(3), 450–457. <https://doi.org/10.1038/ijo.2016.225>
- Thai, P.-K., Tan, E.-C., Tan, W.-L., Tey, T.-H., Kaur, H., & Say, Y.-H. (2011). Sweetness intensity perception and pleasantness ratings of sucrose, aspartame solutions and cola

- among multi-ethnic Malaysian subjects. *Food Quality and Preference*, 22(3), 281–289. <https://doi.org/10.1016/j.foodqual.2010.11.004>
- The Canadian Heart Health Surveys Research Group, Dobbelsteyn, C., Joffres, M., MacLean, D., & Flowerdew, G. (2001). A comparative evaluation of waist circumference, waist-to-hip ratio and body mass index as indicators of cardiovascular risk factors. The Canadian Heart Health Surveys. *International Journal of Obesity*, 25(5), 652–661. <https://doi.org/10.1038/sj.ijo.0801582>
- Thirlby, R., Bahiraei, F., Randall, J., & Drewnoski, A. (2006). Effect of Roux-en-Y Gastric Bypass on Satiety and Food Likes: The Role of Genetics. *Journal of Gastrointestinal Surgery*, 10(2), 270–277. <https://doi.org/10.1016/j.gassur.2005.06.012>
- Thivel, D., Finlayson, G., Miguet, M., Pereira, B., Duclos, M., Boirie, Y., Doucet, E., Blundell, J. E., & Metz, L. (2018). Energy depletion by 24-h fast leads to compensatory appetite responses compared with matched energy depletion by exercise in healthy young males. *British Journal of Nutrition*, 120(5), 583–592. <https://doi.org/10.1017/S0007114518001873>
- Thomas, G. N., Cockram, C. S., & Chan, J. C. N. (2001). Relationships between the TaqI polymorphism of the dopamine D2 receptor and blood pressure in hyperglycaemic and normoglycaemic Chinese subjects. *Clinical Endocrinology*, 7.
- Thomas, J. R., & Marcus, E. (2008). High and Low Fat Food Selection with Reported Frequency Intolerance Following Roux-en-Y Gastric Bypass. *Obesity Surgery*, 18(3), 282–287. <https://doi.org/10.1007/s11695-007-9336-3>
- Tiggemann, M., & Kemps, E. (2005). The phenomenology of food cravings: The role of mental imagery. *Appetite*, 45(3), 305–313. <https://doi.org/10.1016/j.appet.2005.06.004>
- Toews, I., Lohner, S., de Gaudry, D. K., Sommer, H., & Meerpohl, J. J. (2019). *Association between intake of non-sugar sweeteners and health outcomes: Systematic review and meta-analyses of randomised and non-randomised controlled trials and observational studies*. 13.
- Tordoff, & Alleva, A. M. (1989). Oral stimulation with aspartame increases hunger. *Physiology & Behavior*, 47(3), 555–559. [https://doi.org/10.1016/0031-9384\(90\)90126-O](https://doi.org/10.1016/0031-9384(90)90126-O)
- Tordoff, & Alleva, A. M. (1990). Effect of drinking soda sweetened with aspartame or high-fructose corn syrup on food intake and body weight. *The American Journal of Clinical Nutrition*, 51(6), 963–969. <https://doi.org/10.1093/ajcn/51.6.963>
- Tsouristakis, A. I., Febres, G., McMahon, D. J., Tchang, B., Conwell, I. M., Tsang, A. J., Ahmed, L., Bessler, M., & Korner, J. (2019). Long-Term Modulation of Appetitive Hormones and Sweet Cravings After Adjustable Gastric Banding and Roux-en-Y Gastric Bypass. *Obesity Surgery*, 29(11), 3698–3705. <https://doi.org/10.1007/s11695-019-04111-z>
- Tukey, J. (1977). *Exploratory Data Analysis* (pp. 43–44). Addison-Wesley.
- Tuorila, H., Lähteenmäki, L., Pohjalainen, L., & Lotti, L. (2001). Food neophobia among the Finns and related responses to familiar and unfamiliar foods. *Food Quality and Preference*, 12(1), 29–37. [https://doi.org/10.1016/S0950-3293\(00\)00025-2](https://doi.org/10.1016/S0950-3293(00)00025-2)
- Turner-McGrievy, G., Tate, D. F., Moore, D., & Popkin, B. (2013). Taking the Bitter with the Sweet: Relationship of Supertasting and Sweet Preference with Metabolic Syndrome and Dietary Intake: Sweet preference and dietary intake *Journal of Food Science*, 78(2), S336–S342. <https://doi.org/10.1111/1750-3841.12008>
- Uher, R., Treasure, J., Heining, M., Brammer, M. J., & Campbell, I. C. (2006). Cerebral processing of food-related stimuli: Effects of fasting and gender. *Behavioural Brain Research*, 169(1), 111–119. <https://doi.org/10.1016/j.bbr.2005.12.008>

- Umabiki, M., Tsuzaki, K., Kotani, K., Nagai, N., Sano, Y., Matsuoka, Y., Kitaoka, K., Okami, Y., Sakane, N., & Higashi, A. (2010). The Improvement of Sweet Taste Sensitivity with Decrease in Serum Leptin Levels During Weight Loss in Obese Females. *The Tohoku Journal of Experimental Medicine*, *220*(4), 267–271.
<https://doi.org/10.1620/tjem.220.267>
- Vainik, U., García-García, I., & Dagher, A. (2019). Uncontrolled eating: A unifying heritable trait linked with obesity, overeating, personality and the brain. *European Journal of Neuroscience*, *50*(3), 2430–2445. <https://doi.org/10.1111/ejn.14352>
- Vainik, U., & Meule, A. (2018). Jangle fallacy epidemic in obesity research: A comment on Ruddock et al. (2017). *International Journal of Obesity*, *42*(3), 585–586.
<https://doi.org/10.1038/ijo.2017.264>
- 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. <https://doi.org/10.1016/j.appet.2015.03.004>
- Van de Ven, W.-P., M. S. ., Wouters, L. ., & Saris, W. H. M. (1994). Effects of liquid preloads with different fructose/fibre concentrations of subsequent food intake and ratings of hunger in women. *Appetite*, *23*(2), 139–146.
- Van Kleef, E., Shimizu, M., & Wansink, B. (2013). Just a bite: Considerably smaller snack portions satisfy delayed hunger and craving. *Food Quality and Preference*, *27*(1), 96–100. <https://doi.org/10.1016/j.foodqual.2012.06.008>
- van Langeveld, A. W. B., Teo, P. S., de Vries, J. H. M., Feskens, E. J. M., de Graaf, C., & Mars, M. (2018). Dietary taste patterns by sex and weight status in the Netherlands. *British Journal of Nutrition*, *119*(10), 1195–1206.
<https://doi.org/10.1017/S0007114518000715>
- Van Opstal, A. M., Hafkemeijer, A., van den Berg-Huysmans, A. A., Hoeksma, M., Mulder, Theo. P. J., Pijl, H., Rombouts, S. A. R. B., & van der Grond, J. (2019). Brain activity and connectivity changes in response to nutritive natural sugars, non-nutritive natural sugar replacements and artificial sweeteners. *Nutritional Neuroscience*, 1–11.
<https://doi.org/10.1080/1028415X.2019.1639306>
- van Opstal, A. M., Kaal, I., van den Berg-Huysmans, A. A., Hoeksma, M., Blonk, C., Pijl, H., Rombouts, S. A. R. B., & van der Grond, J. (2019). Dietary sugars and non-caloric sweeteners elicit different homeostatic and hedonic responses in the brain. *Nutrition*, *60*, 80–86. <https://doi.org/10.1016/j.nut.2018.09.004>
- Varady, K. A., Bhutani, S., Church, E. C., & Klempel, M. C. (2009). Short-term modified alternate-day fasting: A novel dietary strategy for weight loss and cardioprotection in obese adults. *The American Journal of Clinical Nutrition*, *90*(5), 1138–1143.
<https://doi.org/10.3945/ajcn.2009.28380>
- Veldhuizen, M. G., Babbs, R. K., Patel, B., Fobbs, W., Kroemer, N. B., Garcia, E., Yeomans, M. R., & Small, D. M. (2017). Integration of Sweet Taste and Metabolism Determines Carbohydrate Reward. *Current Biology*, *27*(16), 2476–2485.e6.
<https://doi.org/10.1016/j.cub.2017.07.018>
- Venditti, C., Musa-Veloso, K., Lee, H. Y., Poon, T., Mak, A., Darch, M., Juana, J., Fronda, D., Noori, D., Pateman, E., & Jack, M. (2020). Determinants of Sweetness Preference: A Scoping Review of Human Studies. *Nutrients*, *12*(3), 718.
<https://doi.org/10.3390/nu12030718>
- Vescovi, J. D., Zimmerman, S. L., Miller, W. C., Hildebrandt, L., Hammer, R. L., & Fernhall, B. (2001). Evaluation of the BOD POD for estimating percentage body fat in a heterogeneous group of adult humans. *European Journal of Applied Physiology*, *85*(3–4), 326–332. <https://doi.org/10.1007/s004210100459>

- Vignini, A., Borroni, F., Sabbatinelli, J., Pugnali, S., Alia, S., Taus, M., Ferrante, L., Mazzanti, L., & Fabri, M. (2019). General Decrease of Taste Sensitivity Is Related to Increase of BMI: A Simple Method to Monitor Eating Behavior. *Disease Markers*, 2019, 1–8. <https://doi.org/10.1155/2019/2978026>
- 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. <https://doi.org/10.1016/j.tics.2010.11.001>
- Volkow, N. D., Wang, G.-J., Telang, F., Fowler, J. S., Thanos, P. K., Logan, J., Alexoff, D., Ding, Y.-S., Wong, C., Ma, Y., & Pradhan, K. (2008). Low dopamine striatal D2 receptors are associated with prefrontal metabolism in obese subjects: Possible contributing factors. *NeuroImage*, 42(4), 1537–1543. <https://doi.org/10.1016/j.neuroimage.2008.06.002>
- Volkow, N. D., Wise, R. A., & Baler, R. (2017). The dopamine motive system: Implications for drug and food addiction. *Nature Reviews Neuroscience*, 18(12), 741–752. <https://doi.org/10.1038/nrn.2017.130>
- Vucenik, I., & Stains, J. P. (2012). Obesity and cancer risk: Evidence, mechanisms, and recommendations. *Annals of the New York Academy of Sciences*, 1271(1), 37–43. <https://doi.org/10.1111/j.1749-6632.2012.06750.x>
- Wandel, M., & Fagerli, R. A. (2001). Consumer concern about food related health risks and their trust in experts. *Ecology of Food and Nutrition*, 40(3), 253–283. <https://doi.org/10.1080/03670244.2001.9991652>
- Wanders, A. J., van den Borne, J. J. G. C., de Graaf, C., Hulshof, T., Jonathan, M. C., Kristensen, M., Mars, M., Schols, H. A., & Feskens, E. J. M. (2011). Effects of dietary fibre on subjective appetite, energy intake and body weight: A systematic review of randomized controlled trials: Dietary fibres, appetite and body weight. *Obesity Reviews*, no-no. <https://doi.org/10.1111/j.1467-789X.2011.00895.x>
- Wang, G.-J., Volkow, N. D., Felder, C., Fowler, J. S., Levy, A. V., Pappas, N. R., Wong, C. T., Zhu, W., & Netusil, N. (2002). Enhanced resting activity of the oral somatosensory cortex in obese subjects: *Neuroreport*, 13(9), 1151–1155. <https://doi.org/10.1097/00001756-200207020-00016>
- Wang, G.-J., Volkow, N. D., Thanos, P. K., & Fowler, J. S. (2004). Similarity Between Obesity and Drug Addiction as Assessed by Neurofunctional Imaging: A Concept Review. *Journal of Addictive Diseases*, 23(3), 39–53. https://doi.org/10.1300/J069v23n03_04
- Wansink, B., Cheney, M., & Chan, N. (2003). Exploring comfort food preferences across age and gender. *Physiology & Behavior*, 79(4–5), 739–747. [https://doi.org/10.1016/S0031-9384\(03\)00203-8](https://doi.org/10.1016/S0031-9384(03)00203-8)
- Wardle, J., Carnell, S., Haworth, C. M., & Plomin, R. (2008). Evidence for a strong genetic influence on childhood adiposity despite the force of the obesogenic environment. *The American Journal of Clinical Nutrition*, 87(2), 398–404. <https://doi.org/10.1093/ajcn/87.2.398>
- Wark, P. A., Hardie, L. J., Frost, G. S., Alwan, N. A., Carter, M., Elliott, P., Ford, H. E., Hancock, N., Morris, M. A., Mulla, U. Z., Noorwali, E. A., Petropoulou, K., Murphy, D., Potter, G. D. M., Riboli, E., Greenwood, D. C., & Cade, J. E. (2018). Validity of an online 24-h recall tool (myfood24) for dietary assessment in population studies: Comparison with biomarkers and standard interviews. *BMC Medicine*, 16(1). <https://doi.org/10.1186/s12916-018-1113-8>
- Warrilow, A., Mellor, D., McKune, A., & Pumpa, K. (2019). Dietary fat, fibre, satiation, and satiety—A systematic review of acute studies. *European Journal of Clinical Nutrition*, 73(3), 333–344. <https://doi.org/10.1038/s41430-018-0295-7>

- Wee, M., Tan, V., & Forde, C. (2018). A Comparison of Psychophysical Dose-Response Behaviour across 16 Sweeteners. *Nutrients*, *10*(11), 1632. <https://doi.org/10.3390/nu10111632>
- Weingarten, H. P., & Elston, D. (1990). The phenomenology of food cravings. *Appetite*, *15*(3), 231–246. [https://doi.org/10.1016/0195-6663\(90\)90023-2](https://doi.org/10.1016/0195-6663(90)90023-2)
- Westerterp-Plantenga, M. S. (1999). Effects of extreme environments on food intake in human subjects. *Proceedings of the Nutrition Society*, *58*(4), 791–798. <https://doi.org/10.1017/S002966519900107X>
- Westwater, M. L., Fletcher, P. C., & Ziauddeen, H. (2016). Sugar addiction: The state of the science. *European Journal of Nutrition*, *55*(S2), 55–69. <https://doi.org/10.1007/s00394-016-1229-6>
- Whitelock, V., Nouwen, A., van den Akker, O., & Higgs, S. (2018). The role of working memory sub-components in food choice and dieting success. *Appetite*, *124*, 24–32. <https://doi.org/10.1016/j.appet.2017.05.043>
- Wilk, K., Korytek, W., Pelczyńska, M., Moszak, M., & Bogdański, P. (2022). The Effect of Artificial Sweeteners Use on Sweet Taste Perception and Weight Loss Efficacy: A Review. *Nutrients*, *14*(6), 1261. <https://doi.org/10.3390/nu14061261>
- Wise, A. (2008). Developments in nutritional programming illustrated by WinDiets. *Nutrition Bulletin*, *33*(1), 55–57. <https://doi.org/10.1111/j.1467-3010.2007.00660.x>
- Wise, P. M., Nattress, L., Flammer, L. J., & Beauchamp, G. K. (2016). Reduced dietary intake of simple sugars alters perceived sweet taste intensity but not perceived pleasantness. *The American Journal of Clinical Nutrition*, *103*(1), 50–60. <https://doi.org/10.3945/ajcn.115.112300>
- Woods, S. C., Lutz, T. A., Geary, N., & Langhans, W. (2006). Pancreatic signals controlling food intake; insulin, glucagon and amylin. *Philosophical Transactions of the Royal Society B: Biological Sciences*, *361*(1471), 1219–1235. <https://doi.org/10.1098/rstb.2006.1858>
- Woods, S. C., Seeley, R. J., Porte, D., & Schwartz, M. W. (1998). Signals That Regulate Food Intake and Energy Homeostasis. *Science*, *280*(5368), 1378–1383. <https://doi.org/10.1126/science.280.5368.1378>
- Wooley, O. W., Wooley, S. C., & Dunham, R. B. (1972). Calories and sweet taste: Effects on sucrose preference in the obese and nonobese. *Physiology & Behavior*, *9*(5), 765–768. [https://doi.org/10.1016/0031-9384\(72\)90048-0](https://doi.org/10.1016/0031-9384(72)90048-0)
- World Health Organization. (2015). *Guideline*. World Health Organization. <https://public.ebookcentral.proquest.com/choice/publicfullrecord.aspx?p=2033879>
- World Health Organization. (2022a). *Draft WHO guideline: Use of non-sugar sweeteners* (Online Public Consultation: Draft Guideline on Use of Non-Sugar Sweeteners). <https://www.who.int/news-room/articles-detail/online-public-consultation-draft-guideline-on-use-of-non-sugar-sweeteners>
- World Health Organization. (2022b). *WHO NUGAG Use of Non-Sugar Sweeteners Report Release Webinar*.
- World Health Organization, Rios-Leyvraz, M., & Montez, J. (2022). *Health effects of the use of non-sugar sweeteners: A systematic review and meta-analysis*. World Health Organization. <https://apps.who.int/iris/handle/10665/353064>
- Wu, T., Zhao, B. R., Bound, M. J., Checklin, H. L., Bellon, M., Little, T. J., Young, R. L., Jones, K. L., Horowitz, M., & Rayner, C. K. (2012). Effects of different sweet preloads on incretin hormone secretion, gastric emptying, and postprandial glycemia in healthy humans. *The American Journal of Clinical Nutrition*, *95*(1), 78–83. <https://doi.org/10.3945/ajcn.111.021543>

- Yang, Q. (2010). *Gain weight by “going diet?” Artificial sweeteners and the neurobiology of sugar cravings*. 8.
- Yang, Q., Kraft, M., Shen, Y., MacFie, H., & Ford, R. (2019). Sweet Liking Status and PROP Taster Status impact emotional response to sweetened beverage. *Food Quality and Preference*, *75*, 133–144. <https://doi.org/10.1016/j.foodqual.2019.02.016>
- Yang, Q., Williamson, A.-M., Hasted, A., & Hort, J. (2020). Exploring the relationships between taste phenotypes, genotypes, ethnicity, gender and taste perception using Chi-square and regression tree analysis. *Food Quality and Preference*, *83*, 103928. <https://doi.org/10.1016/j.foodqual.2020.103928>
- Yanovski, Leet, M., Yanovski, J. A., Flood, M., Gold, P. W., Kissileff, H. R., & Walsh, B. T. (1992). Food selection and intake of obese women with binge-eating disorder. *The American Journal of Clinical Nutrition*, *56*(6), 975–980. <https://doi.org/10.1093/ajcn/56.6.975>
- Yeomans, M. R., Re, R., Wickham, M., Lundholm, H., & Chambers, L. (2016). Beyond expectations: The physiological basis of sensory enhancement of satiety. *International Journal of Obesity*, *40*(11), 1693–1698. <https://doi.org/10.1038/ijo.2016.112>
- Yeomans, M. R., & Symes, T. (1999). Individual Differences in the Use of Pleasantness and Palatability Ratings. *Appetite*, *32*(3), 383–394. <https://doi.org/10.1006/appe.1999.0224>
- Yeomans, M. R., Tepper, B. J., Rietzschel, J., & Prescott, J. (2007). Human hedonic responses to sweetness: Role of taste genetics and anatomy. *Physiology & Behavior*, *91*(2–3), 264–273. <https://doi.org/10.1016/j.physbeh.2007.03.011>
- Yoshinaka, M., Ikebe, K., Uota, M., Ogawa, T., Okada, T., Inomata, C., Takeshita, H., Mihara, Y., Gondo, Y., Masui, Y., Kamide, K., Arai, Y., Takahashi, R., & Maeda, Y. (2016). Age and sex differences in the taste sensitivity of young adult, young-old and old-old Japanese. *Geriatrics & Gerontology International*, *16*(12), 1281–1288. <https://doi.org/10.1111/ggi.12638>
- Young, L. R., & Nestle, M. (2002). The Contribution of Expanding Portion Sizes to the US Obesity Epidemic. *American Journal of Public Health*, *92*(2), 246–249. <https://doi.org/10.2105/AJPH.92.2.246>
- Zandstra, E. H., de Graaf, C., & van Trijp, H. C. M. (2000). Effects of variety and repeated in-home consumption on product acceptance. *Appetite*, *35*(2), 113–119. <https://doi.org/10.1006/appe.2000.0342>
- Zandstra, E. H., Weegels, M. F., Van Spronsen, A. A., & Klerk, M. (2004). Scoring or boring? Predicting boredom through repeated in-home consumption. *Food Quality and Preference*, *15*(6), 549–557. <https://doi.org/10.1016/j.foodqual.2003.12.001>
- Zellner, D. A., Garriga-Trillo, A., Centeno, S., & Wadsworth, E. (2004). Chocolate craving and the menstrual cycle. *Appetite*, *42*(1), 119–121. <https://doi.org/10.1016/j.appet.2003.11.004>
- Zellner, D., Garriga-Trillo, A., Rohm, E., Centeno, S., & Parker, S. (1999). Food Liking and Craving: A Cross-cultural Approach. *Appetite*, *33*(1), 61–70. <https://doi.org/10.1006/appe.1999.0234>
- Zhang, Y., Proenca, R., Maffei, M., Barone, M., & Leopold, L. (1994). *Positional cloning of the mouse obese gene and its human homologue*. 8.
- Zhao, G. Q., Zhang, Y., Hoon, M. A., Chandrashekar, J., Erlenbach, I., Ryba, N. J. P., & Zuker, C. S. (2003). The Receptors for Mammalian Sweet and Umami Taste. *Cell*, *115*(3), 255–266. [https://doi.org/10.1016/S0092-8674\(03\)00844-4](https://doi.org/10.1016/S0092-8674(03)00844-4)
- Zheng, H., Lenard, N. R., Shin, A. C., & Berthoud, H.-R. (2009). Appetite control and energy balance regulation in the modern world: Reward-driven brain overrides repletion

- signals. *International Journal of Obesity*, 33(S2), S8–S13.
<https://doi.org/10.1038/ijo.2009.65>
- Zobel, E. H., Hansen, T. W., Rossing, P., & Von Scholten, B. J. (2016). Global Changes in Food Supply and the Obesity Epidemic. *Current Obesity Reports*, 5(4), 449–455.
<https://doi.org/10.1007/s13679-016-0233-8>
- Zoon, H. F. A., He, W., de Wijk, R. A., de Graaf, C., & Boesveldt, S. (2014). Food preference and intake in response to ambient odours in overweight and normal-weight females. *Physiology & Behavior*, 133, 190–196. <https://doi.org/10.1016/j.physbeh.2014.05.026>
- Zverev, Y. P. (2004). Effects of caloric deprivation and satiety on sensitivity of the gustatory system. *BMC Neuroscience*, 5.

Appendix 1 Leeds Food Preference Breakfast and Lunch Versions
(Study1)

	High-fat	Low-fat
<i>Breakfast</i>		
Sweet	<ul style="list-style-type: none"> • Cream-filled crepe • Glazed donut • Blueberry muffin • Cinnamon twist pastry 	<ul style="list-style-type: none"> • Grapes • Muesli • Yogurt with berries • Banana
Savoury	<ul style="list-style-type: none"> • Ham and cheese croissant sandwich • Sausage sandwich • English breakfast • Sausage roll 	<ul style="list-style-type: none"> • Smoked salmon and cream cheese crackers • Ham • Beans on toast • White bread roll
<i>Lunch</i>		
Sweet	<ul style="list-style-type: none"> • Milk chocolate with nuts • Jam doughnut • Blueberry muffin • Cream doughnut 	<ul style="list-style-type: none"> • Fruit salad • Marshmallows • Sweet popcorn • Jelly babies
Savoury	<ul style="list-style-type: none"> • Crisps (chips) • Salted peanuts • Swiss cheese • Chips (fries) 	<ul style="list-style-type: none"> • Boiled potatoes • Pilau rice • White bread roll • Spaghetti in sauce

Appendix 2 Visual Analogue Scale Questions (Study1)

Part A

Please complete this section, after consuming the foods provided, by placing a vertical mark through the line.

1. How sweet did you find the **RISOTTO**?

Not at all _____ Extremely
Sweet Sweet

2. How savoury did you find the **RISOTTO**?

Not at all _____ Extremely
Savoury Savoury

3. How fatty did you find the **RISOTTO**?

Not at all _____ Extremely
Fatty Fatty

4. How tasty did you find the **RISOTTO**?

Not at all _____ Extremely
Tasty Tasty

5. How pleasant did you find the **RISOTTO**?

Not at all _____ Extremely
Pleasant Pleasant

6. How filling did you find the **RISOTTO**?

Not at all _____ Extremely

Filling _____ Filling

7. How satisfying did you find the **RISOTTO**?

Not at all _____ Extremely

Satisfying _____ Satisfying

8. How much did you like the **RISOTTO**?

Not at all _____ Extremely

9. How much more of the **RISOTTO** do you think you could eat?

A Small _____ A Large

Amount _____ Amount

Part B

Please complete this section, after consuming the foods provided, by placing a vertical mark through the line.

1. How sweet did you find the **YOGURT**?

Not at all _____ Extremely

Sweet _____ Sweet

2. How savoury did you find the **YOGURT**?

Not at all _____ Extremely

Savoury
Savoury

3. How fatty did you find the **YOGURT**?

Not at all _____ Extremely
Fatty _____ Fatty

4. How tasty did you find the **YOGURT**?

Not at all _____ Extremely
Tasty _____ Tasty

5. How pleasant did you find the **YOGURT**?

Not at all _____ Extremely
Pleasant _____ Pleasant

6. How filling did you find the **YOGURT**?

Not at all _____ Extremely
Filling _____ Filling

7. How satisfying did you find the **YOGURT**?

Not at all _____ Extremely
Satisfying _____ Satisfying

8. How much did you like the **YOGURT**?

Not at all _____ Extremely

9. How much more of the **YOGURT** do you think you could eat?

A Small
Amount



A Large
Amount

Appendix 3 Eating Attitudes Test 26

Please fill out the form below as accurately, honestly and completely as possible. There are no right or wrong answers. All of your responses are confidential.

	Always	Usually	Often	Sometimes	Rarely	Never
1. I am terrified about being overweight.						
2. I avoid eating when I am hungry.						
3. I find myself preoccupied with food.						
4. I have gone on eating binges where I feel that I may not be able to stop.						
5. I cut my food into small pieces.						
6. I am aware of the calorie content of foods that I eat.						
7. I particularly avoid food with a high carbohydrate content (i.e. bread, rice, potatoes, etc.)						
8. I feel that others would prefer if I ate more.						
9. I vomit after I have eaten.						
10. I feel extremely guilty after eating.						
11. I am occupied with a desire to be thinner.						
12. I think about burning up calories when I exercise.						
13. Other people think that I am too thin.						
14. I am preoccupied with the thought of having fat on my body.						
15. I take longer than others to eat my meals.						
16. I avoid foods with sugar in them.						
17. I eat diet foods.						
18. I feel that food controls my life.						
19. I display self-control around food.						
20. I feel that others pressure me to eat.						
21. I give too much time and thought to food.						
22. I feel uncomfortable after eating sweets.						
23. I engage in dieting behavior.						
24. I like my stomach to be empty.						
25. I enjoy trying new rich foods.						
26. I have the impulse to vomit after meals.						

Appendix 4 Short sweet Food Frequency Questionnaire

Part 1: Have you consumed these products during the last month?

[Each site to provide a list with examples of products to support the participant if necessary]

Product	Yes	No
Sugar soft drinks, energy drinks, juice, nectars, mixture of fruit syrup		
Candy		
Jam, honey, compote		
Breakfast cereals and bars		
Flavoured yoghurt or fermented milk		
Bakery and pastry items, breakfast cake		
Added table sugar to breakfast, coffee, tea, etc.		
Chocolate and chocolate paste/spread		
Cake and biscuits		
Low-alcohol-content beverages		
Ice cream		
Other sugar-containing foods/drinks (specify): _____		

For researcher Part 1:

Scoring will be applied automatically by the QDP software. 1 point is assigned for each Yes and 0 points for No. Subjects need to score at least 3 to be eligible.

Part 2: Food Product Use and Acceptability

1. Do you like the product shown in the picture? YES / NO
2. Do you like low-calorie versions of the product shown in the picture? YES / NO
3. Are you willing to consume the product shown in the picture during the probe days and each day during the 2-week at-home intervention of the trial? YES / NO

Appendix 5 Control of Eating Questionnaire

Please read each question carefully and put a mark through the line at the point that best represents your experience. Answer all questions according to your experience over the **last 7 days**.

1. How hungry have you felt?

Not at all hungry	Extremely hungry
----------------------	---------------------
 2. How full have you felt?

Not at all full	Extremely full
--------------------	-------------------
 3. How strong was your desire to eat sweet foods?

Not at all strong	Extremely strong
----------------------	---------------------
 4. How strong was your desire to eat savoury foods?

Not at all strong	Extremely strong
----------------------	---------------------
 5. How happy have you felt?

Not at all happy	Extremely happy
---------------------	--------------------
 6. How anxious have you felt?

Not at all anxious	Extremely anxious
-----------------------	----------------------
 7. How alert have you felt?

Not at all alert	Extremely alert
---------------------	--------------------
 8. How contented have you felt?

Not at all contented	Extremely contented
-------------------------	------------------------
- A food craving is a strong urge to eat a particular food or drink
9. During the last 7 days how often have you had food cravings?

Not at all	Very often
------------	------------
 10. How strong have any food cravings been?

Not at all strong	Extremely strong
----------------------	---------------------
 11. How difficult has it been to resist any food cravings?

Not at all difficult	Extremely difficult
-------------------------	------------------------
 12. How often have you eaten in response to food cravings?

Not at all

After every one

How often have you had food cravings for the following types of food/drink?

13. Chocolate or chocolate flavoured foods

Not at all

Extremely
often

14. Other sweet foods (cakes, pastries, biscuits, etc)

Not at all

Extremely
often

15. Fruit or fruit juice

Not at all

Extremely
often

16. Dairy foods (cheese, yoghurts, milk, etc)

Not at all

Extremely
often

17. Starchy foods (bread, rice, pasta, etc)

Not at all

Extremely
often

18. Savoury foods (french fries, crisps, burgers, pizza, etc)

Not at all

Extremely
often

19. Generally, how difficult has it been to control your eating?

Not at all

Extremely

difficult

difficult

20. Which one food makes it most difficult for you to control eating?

.....

21. How difficult has it been to resist eating this food during the last 7 days?

Not at all

Extremely

difficult

difficult

Appendix 6 Binge Eating Scale

Eating habits checklist

Instructions

Below are groups of numbered statements. Read all of the statements in each group and mark on this sheet the one that describes you the best by circling the good number.

A.

1. I don't feel self-conscious about my weight or body size when I'm with others.
2. I feel concerned about how I look to others, but it normally does not make me feel disappointed with myself.
3. I do get self-conscious about my appearance and weight which makes me feel disappointed in myself.
4. I feel very self-conscious about my weight and frequently, I feel intense shame and disgust for myself. I try to avoid social contacts because of my self-consciousness.

B.

1. I don't have any difficulty eating slowly in the proper manner.
2. Although I seem to "gobble down" foods, I don't end up feeling stuffed because of eating too much.
3. At times, I tend to eat quickly and then, I feel uncomfortably full afterwards.
4. I have the habit of bolting down my food, without really chewing it. When this happens I usually feel uncomfortably stuffed because I've eaten too much.

C.

1. I feel capable to control my eating urges when I want to.
2. I feel like I have failed to control my eating more than the average person.
3. I feel utterly helpless when it comes to feeling in control of my eating urges.
4. Because I feel so helpless about controlling my eating I have become very desperate about trying to get in control.

D.

1. I don't have the habit of eating when I'm bored.
2. I sometimes eat when I'm bored, but often I'm able to "get busy" and get my mind off food.
3. I have a regular habit of eating when I'm bored, but occasionally, I can use some other activity to get my mind off eating.
4. I have a strong habit of eating when I'm bored. Nothing seems to help me break the habit.

E.

1. I'm usually physically hungry when I eat something.
2. Occasionally, I eat something on impulse even though I really am not hungry.
3. I have the regular habit of eating foods, that I might not really enjoy, to satisfy a hungry feeling even though physically, I don't need the food.
4. Even though I'm not physically hungry, I get a hungry feeling in my mouth that only seems to be satisfied when I eat a food, like a sandwich, that fills my

mouth. Sometimes, when I eat the food to satisfy my mouth hunger, I then spit the food out so I won't gain weight.

F.

1. I don't feel any guilt or self-hate after I overeat.
2. After I overeat, occasionally I feel guilt or self-hate.
3. Almost all the time I experience strong guilt or self-hate after I overeat.

G.

1. I don't lose total control of my eating when dieting even after periods when I overeat.
2. Sometimes when I eat a "forbidden food" on a diet, I feel like I "blew it" and eat even more.
3. Frequently, I have the habit of saying to myself, "I've blown it now, why not go all the way" when I overeat on a diet. When that happens I eat even more.
4. I have a regular habit of starting strict diets for myself, but I break the diets by going on an eating binge. My life seems to be either a "feast" or "famine."

H.

1. I rarely eat so much food that I feel uncomfortably stuffed afterwards.
2. Usually about once a month, I eat such a quantity of food, I end up feeling very stuffed.
3. I have regular periods during the month when I eat large amounts of food, either at mealtime or at snacks.
4. I eat so much food that I regularly feel quite uncomfortable after eating and sometimes a bit nauseous.

I.

1. My level of calorie intake does not go up very high or go down very low on a regular basis.
2. Sometimes after I overeat, I will try to reduce my caloric intake to almost nothing to compensate for the excess calories I've eaten.
3. I have a regular habit of overeating during the night. It seems that my routine is not to be hungry in the morning but overeat in the evening.
4. In my adult years, I have had week-long periods where I practically starve myself. This follows periods when I overeat. It seems I live a life of either "feast or famine."

J.

1. I usually am able to stop eating when I want to. I know when "enough is enough."
2. Every so often, I experience a compulsion to eat which I can't seem to control.
3. Frequently, I experience strong urges to eat which I seem unable to control, but at other times I can control my eating urges.
4. I feel incapable of controlling urges to eat. I have a fear of not being able to stop eating voluntarily.

K.

1. I don't have any problem stopping eating when I feel full.
2. I usually can stop eating when I feel full but occasionally overeat leaving me feeling uncomfortably stuffed.
3. I have a problem stopping eating once I start and usually I feel uncomfortably stuffed after I eat a meal.
4. Because I have a problem not being able to stop eating when I want, I sometimes have to induce vomiting to relieve my stuffed feeling.

L.

1. I seem to eat just as much when I'm with others (family, social gatherings) as when I'm by myself.
2. Sometimes, when I'm with other persons, I don't eat as much as I want to eat because I'm self-conscious about my eating.
3. Frequently, I eat only a small amount of food when others are present, because I'm very embarrassed about my eating.
4. I feel so ashamed about overeating that I pick times to overeat when I know no one will see me. I feel like a "closet eater."

M.

1. I eat three meals a day with only an occasional between meal snack.
2. I eat 3 meals a day, but I also normally snack between meals.
3. When I am snacking heavily, I get in the habit of skipping regular meals.
4. There are regular periods when I seem to be continually eating, with no planned meals.

N.

1. I don't think much about trying to control unwanted eating urges.
2. At least some of the time, I feel my thoughts are pre-occupied with trying to control my eating urges.
3. I feel that frequently I spend much time thinking about how much I ate or about trying not to eat anymore.
4. It seems to me that most of my waking hours are pre-occupied by thoughts about eating or not eating. I feel like I'm constantly struggling not to eat.

O.

1. I don't think about food a great deal.
2. I have strong cravings for food but they last only for brief periods of time.
3. I have days when I can't seem to think about anything else but food.
4. Most of my days seem to be pre-occupied with thoughts about food. I feel like I live to eat.

P.

1. I usually know whether or not I'm physically hungry. I take the right portion

of food to satisfy me.

2. Occasionally, I feel uncertain about knowing whether or not I'm physically hungry. At these times it's hard to know how much food I should take to satisfy me.

3. Even though I might know how many calories I should eat, I don't have any idea what is a "normal" amount of food for me.

Appendix 7 Three Factor Eating Questionnaire

This booklet contains a number of statements. Each statement should be answered either TRUE or FALSE. Read each statement and decide how you feel about it in PART 1.

If you agree with the statement , or if you feel that it is true about you then circle T next to the statement.

If you disagree with a statement, or if you feel that it is false as applied to you, circle the F next to the statement.

- | | | |
|--|---|---|
| 1) 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 | T | F |
| 2) I usually eat too much at social occasions, like parties and picnics. | T | F |
| 3) I am usually so hungry that I eat more than 3 times a day. | T | F |
| 4) When I have eaten my quota of calories I am usually very good about not eating any more. | T | F |
| 5) Dieting is so hard for me because I just get too hungry. | T | F |
| 6) I deliberately take small helpings as a means of controlling my weight. | T | F |
| 7) Sometimes things just taste so good that I keep on eating, even when I am no longer hungry. | T | F |
| 8) 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. | T | F |
| 9) When I feel anxious I find myself eating. | T | F |
| 10) Life is too short to worry about dieting. | T | F |
| 11) Since my weight goes up and down, I have gone on reducing diets more than once. | T | F |
| 12) I often feel so hungry I just have to eat something. | T | F |
| 13) When I am with someone who is overeating I usually overeat too. | T | F |

- | | | |
|--|---|---|
| 14) I have a pretty good idea of the number of calories in common foods | T | F |
| 15) Sometimes when I start eating, I just can't seem to stop. | T | F |
| 16) It is not difficult for me to leave something on my plate. | T | F |
| 17) At certain times of the day I get hungry because I have gotten used to eating then. | T | F |
| 18) 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. | T | F |
| 19) Being with someone who is overeating often makes me hungry enough to eat also. | T | F |
| 20) When I feel blue I often overeat. | T | F |
| 21) I enjoy eating too much to spoil it by counting calories or watching my weight. | T | F |
| 22) When I see a real delicacy I often get so hungry that I have to eat it right away. | T | F |
| 23) I often stop eating when I am not really full as a conscious means of limiting the amount I eat. | T | F |
| 24) I get so hungry my stomach feels like a bottomless pit. | T | F |
| 25) My weight has hardly changed at all in the last ten years. | T | F |
| 26) I am always hungry so it is hard for me to stop eating before I finish the food on my plate. | T | F |
| 27) When I feel lonely, I console myself by eating. | T | F |
| 28) I consciously hold back at meals in order not to gain weight. | T | F |
| 29) I sometimes get very hungry late in the evening or at night. | T | F |
| 30) I eat anything I want, anytime. | T | F |
| 31) Without even thinking about it I take a long time to eat. | T | F |
| 32) I count calories as a conscious means of controlling my weight. | T | F |
| 33) I do not eat some foods because they make me fat. | T | F |

- 34) I am always hungry enough to eat at anytime. **T** **F**
- 35) I pay a great deal of attention to changes in my figure. **T** **F**
- 36) While on a diet, if I eat food that is not allowed, I often then splurge and eat other high calorie foods. **T** **F**

Appendix 9 Leeds Food Preference Questionnaire core images**UoL:**



CRNH:



