The Perception of Sweet, Bitter and Fatty Acid Taste and Sensitivity to Fat by Mouthfeel and Olfaction: Associations with Dietary Intake, Eating Behaviour and Obesity in Premenopausal Women

A thesis presented in partial fulfilment of the requirements for the degree of Doctor of Philosophy in Nutritional Science

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Abstract

Background

Individual variability in taste perception may influence diet, possibly modifying eating behaviour and long-term food choice. Research into taste perception and weight status, dietary intake, eating behaviour and endocrine regulators of metabolic health could provide new important insights. Taste perception may be modifiable, and as such may be a target for future intervention strategies which may have the potential to prevent or treat obesity.

Objectives

The aims of this study were to determine associations between (1) fatty acid taste, olfaction, mouthfeel of fat, dietary intake, eating behaviour and body mass index (BMI), and (2) associations between taste perception of glucose (sweet taste), quinine (bitter taste) and milk (fat perception) with body composition and hormonal adiposity signals (fasting insulin and leptin), dietary intake and eating behaviour.

Design

For the first cross-sectional study, 50 premenopausal women assessed oleic acid taste and olfaction thresholds. BMI was calculated from weight (kg) and height (m). Dietary intake and eating behaviour were evaluated using a food frequency and three-factor eating questionnaire (TFEQ), respectively. Binomial regression analysis was used to model fatty acid taste and olfaction data and fatty acid taster status was determined (hypersensitive, n= 22; hyposensitive, n= 28).

For the second cross-sectional study (the PROMISE study), 351 premenopausal Pacific and New Zealand (NZ) European women were recruited and stratified by non-obese and obese groups. Suprathreshold intensity, hedonic liking, and discrimination of taste by a ranking task were measured using a range of concentrations of sweet, bitter, and fat

solutions. Participants were classified as likers or dislikers for each tastant using a hierarchical cluster analysis. Body fat (BF) was quantified by dual x-ray absorptiometry. Total energy and macronutrient intake were assessed using a 5-day estimated food record and eating behaviour was assessed by TFEQ. Socioeconomic status was measured by deprivation index (NZDep2013). Logistic and linear regression analyses were used to analyse study outcomes and to adjust for potential confounders (socioeconomic position, age, etc). Both studies were undertaken in Auckland, NZ.

Outcomes

The results of the first study showed taste and olfactory detection for oleic acid were positively correlated (r= 0.325; P< 0.02). The eating behaviour disinhibition and BMI were higher in women who were hyposensitive to oleic acid taste (P< 0.05).

The PROMISE study showed women who incorrectly discriminated sweet taste by ranking task were nearly three times more likely to have >35 BF% (adjusted, OR 2.9, P< 0.01). Cluster analysis revealed distinct patterns of liking for each tastant. NZ European sweet likers were twice as likely to have >35 BF% compared to sweet dislikers (adjusted, OR 2.1, P< 0.05), however, this comparison was not significant in Pacific women. Conversely, bitter likers had a decreased likelihood of having >35 BF% in comparison to bitter dislikers (adjusted, OR 0.4, P< 0.01). Having higher fasting plasma leptin concentration significantly increased the likelihood of being a sweet liker in Pacific and in NZ European combined (adjusted; OR 1.7, P< 0.05), but in NZ European women, the likelihood of this was further increased (adjusted; OR 3.6, P< 0.001). Higher fasting plasma insulin concentration also increased the likelihood of being a sweet liker (adjusted, OR 1.7, P< 0.05).

New Zealand European sweet likers had a significantly higher intake of carbohydrates, all sugars and starch (199.4 ± 51.1, 87.9 ± 27.4 and 111.1 ± 34.6 g/day) when compared to sweet taste dislikers (165.9 ± 48.7, 71.4 ± 25.2 and 94.1 ± 34.7 g/day, P< 0.001, P< 0.01, and P< 0.01, respectively), however, this comparison was not significant in Pacific

women. NZ European women in the sweet likers group had an increased disinhibited eating behaviour score ($P < 0.01$).

Conclusions

Fatty acid taste perception was found to be associated with olfaction, eating behaviour and body composition. The findings from the PROMISE study have shown differences in sweet taste perception in relation to adiposity which is further associated with circulating plasma leptin and insulin concentrations. Sweet taste perception was associated with dietary intake and eating behaviour in NZ European women. The taste-diet associations observed in NZ European women were not observed in Pacific women. Therefore, population groups with lower metabolic disease risk may have dissimilar taste-diet associations compared to those with a higher metabolic disease risk. Taste perception is a promising target for future weight-loss and intervention strategies due to demonstrating links with dietary intake, eating behaviour and body composition.

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List of Abbreviations

3-AFC	Three-alternative forced choice
Ach	Acetylcholine
AMDR	Acceptable macronutrient distribution range
ASTM	American Society for Testing and Materials
BF%	Body fat percentage
BIA	Bioelectrical impedance analysis
BMI	Body mass index
BP	Blood pressure
Ca^{2+}	Calcium ions
CCK	Cholecystokinin
CD36	Cluster of differentiation-36
CHO	Carbohydrate
CI	Confidence interval
CN	Cranial nerve
CNS	Central nervous system
CV	Coefficient of variation
DA	Dietary assessment
DFE	Daily frequency equivalent
DXA	Dual-energy x-ray absorptiometry
EB	Eating behaviour
EDTA	Ethylenediaminetetraacetic acid
EE	Enteroendocrine

EI	Energy intake
EXPLORE	Examining the Predictors Linking Obesity Related Elements (study)
FFQ	Food frequency questionnaire
fMRI	Functional magnetic resonance imaging
FR	Food record
g	Grams
GABA	Gamma-aminobutyric acid
GI	Gastrointestinal
GIP	Glucose-dependent insulinotropic polypeptide
GLM	General Linear Model
gLMS	general labelled magnitude scale
GLP-1	Glucagon-like peptide-1
GPCR	G-protein-coupled receptor
GPR 40	G-protein receptor 40
GPR41	G-protein receptor 41
GPR43	G-protein receptor 43
GPR120	G-protein receptor 120
H^+	Hydrogen ion
HbA1c	Glycated haemoglobin
HC	Hip circumference
HDL	High-density lipoprotein cholesterol
HNRU	Human Nutrition Research Unit (Massey University)
ICC	Intra-class correlation
ISAK	International Society of the Advancement of Kinanthropometry

JAR	Just about right
K^+	Potassium ion
kcal	Kilocalorie
kJ	Kilojoule
LAM	Labelled affective magnitude
LCFA	Long chain fatty acid
LDL	Low-density lipoprotein cholesterol
LMS	Labelled magnitude scale
MJ	Megajoule
mM	Millimolar
MoH	Ministry of Health
ng	Nanograms
Ob	Obese
Ow	Overweight
PBF	Percent body fat
PCR	Polymerase chain reaction
PKD2L1	Polycystic kidney disease like ion channel (Polycystin 2 Like 1)
PROMISE	PRedictors linking Obesity and gut MIcrobiomE (study)
PROP	6-n-propylthiouracil
PTC	Phenylthiocarbamide
PYY	Peptide YY
mGLUR1	Metabotropic glutamate receptor 1
mGLUR4	Metabotropic glutamate receptor 4
MSG	Monosodium glutamate

NA	Noradrenaline
NaCl	Sodium chloride
NHANES	National Health and Nutrition Examination Survey
NT	Neurotensin
NZDep2013	New Zealand Deprivation index 2013
NZE	New Zealand European
NZW-FFQ	New Zealand Women's Food Frequency Questionnaire
OA	Oleic acid
Ob-Rb	Leptin receptor, long-form
OEA	Oleoylethanolamine
OECD	The Organisation for Economic Cooperation and Development
OFC	Orbitofrontal cortex
OR	Odds ratio
SD	Standard deviation
sem	Standard error of the mean
SFA	Saturated fatty acid
SNP	Single-nucleotide polymorphism
T2D	Type 2 diabetes
T1R2	Taste receptor type 1 member 2
T1R3	Taste receptor type 1 member 3
TC	Total cholesterol
TDI	Threshold discrimination identification
TRC	Taste receptor cell
TFEQ	Three-factor eating questionnaire

UHT	Ultra Heat Treatment
VAS	Visual analogue scale
WC	Waist circumference
WHO	World Health Organisation
WS	Western style

Researcher's contribution to the thesis

Contributions to the Dessert Taste study

Researchers	Contributions to the thesis
Sophie Kindleysides	• Main researcher/coordinator for the Dessert Taste Study • Application for ethics • Study design and sensory methodology development • Coordinated recruitment and screening • Data collection • Data entry and analysis • Statistical analysis • Interpretation and discussion of the results • Results and discussion • Author of the thesis
Prof Bernhard H Breier	Academic primary supervisor; research conception, strategy and direction; development of study design; research funding support; supervision of all aspects and interpretation of results; thesis revision and approval.
Dr Kathryn Beck	Academic co-supervisor; research strategy; development of the dietary component of study design; interpretation of results; thesis revision.
Prof Matt Golding	Academic co-supervisor; supervision of study design.
Dr Daniel C I Walsh	Supervision of statistical analysis and interpretation of results.
Lisa Henderson	MSc student; participant recruitment and testing; data entry; interpretation and discussion of results.

Contributions to the PROMISE study (HRC funded trial)

Researchers	Contributions to the thesis
Sophie Kindleysides	• Coordinator of the taste testing procedures of the PROMISE Study • Taste testing methodology development • Recruitment and screening (shared) • Data collection (shared) • Data entry and analysis (shared) • Laboratory work (biological samples) (shared) • Statistical analysis • Interpretation and discussion of the results • Results and discussion • Author of the thesis
Prof Bernhard H Breier	Primary investigator of the PROMISE study; Academic primary supervisor; research conception, strategy and direction; development of study design; research funding support; supervision of all aspects and interpretation of results; thesis revision and approval.
Dr Kathryn Beck	Academic co-supervisor; interpretation of results (Chapter 5); thesis revision and approval.
A/Prof Rozanne Kruger	Academic co-supervisor; research conception; development of the dietary component of study design; development of body composition component of study design; DXA scanning; interpretation of results; thesis revision and approval.
Prof Jeroen Douwes	Co-investigator of the PROMISE study. Contribution of study design and development; supervision of statistical analysis and interpretation of results.

Dr Ridvan Firestone Prof Gerald Tannock Dr Anne-Thea McGill Dr Lisa Te Morenga A/Prof Leigh Signal Prof Philippa Gander Prof Stephen Stannard Dr Marilize Richter	Co-investigators of the PROMISE study. Contribution of study design and development; development of recruitment strategies; gut microbiome analyses and data collection (not included in this thesis); trial clinician and medical advice; Pacific and Māori community advice and liaison; sleep and physical activity study components (not included in this thesis); results and discussion.
Dr Marine Corbin	Supervision of statistical analysis and interpretation of results.
Niamh Brennan	Trial co-ordinator; Application for ethics and trial registration; management of participant recruitment; phlebotomy; DXA scanning; management of gratuity vouchers and study finances; management of staff and primary contact for study participants and enquiries.
Moana Manukia	The senior nurse from the Fono (Pacific Healthcare). Recruitment and screening of Pacific participants; data collection; phlebotomy; management of Pacific arm of the study; primary contact for Pacific study participants and enquiries.
Nikki Renall Jo Slater	PhD candidates; Registered dietitians. Coordination of dietary component of study design; one-on-one dietary review of food record data; recruitment and screening; data collection; blood processing and laboratory work (biological samples); data entry; results and discussion.
Shivon Singh	MSc student; taste testing data collection and lab work; data entry.
Elizabeth Cullen Bronte Anscombe Beatrice Drury Laura Mickleson	MSc students; dietary data collection; data entry.
Guojiao (Maggie) Cao Sunna Jacobsen Carolin Friedle	Taste testing data collection; lab setup and labwork; data entry.

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Chapter I.

Introduction

1.1. Background

Sensory properties of food influence food selection and the desire to eat. The major sensory properties of food (e.g. taste, texture, aroma, appearance, etc) are perceived by the primary human senses (e.g. gustatory (taste), tactile (touch), olfactory (smell), visual (sight), auditory (hearing), and chemesthesis) (Drewnowski & Almiron-Roig, 2010; Newman, Haryono, & Keast, 2013). Among these sensory properties, taste perception is a key driver of food choice. Taste sensation or perception is triggered by chemicals when they meet taste bud cells (TBCs) of the tongue (Calvo & Egan, 2015). These distinct taste sensations (e.g. sweet, salty, umami, sour) influence the selection and consumption of food or conversely, may trigger the rejection of food due to toxins (e.g. bitter).

Texture is the sensory and functional manifestation of the structural, mechanical and surface properties of foods detected through the senses of vision, hearing, touch, and kinesthetics. In contrast to other sensory food attributes (e.g. taste), there are no single and specific receptors for texture because of its complex multimodal representation (Rolls, 2004). Most textural properties are perceived when the food is deformed on chewing with the teeth, manipulated and moved by the tongue around the oral cavity (Szczesniak, 2002).

Stimulation of smell (olfaction) occurs through odour receptors which bind to specific volatile (mostly organic) aroma compounds. Volatile odour compounds are first detected in the nasal neuroepithelium by olfactory receptor neurons. The interaction of odorous molecules with receptors results in consecutive transduction events that initiate the opening of ion channels in neuronal cell membranes (Swiegers, Chambers, & Pretorius, 2005). Compared to taste, we can detect thousands of different odorants,

which are critical in perceiving differences in flavour.

Some researchers have emphasised the role of food appearance and colour in the selection of foods, for example, phytochemicals account for various colours of fruit and vegetables (Barnes, Prasain, & Kim, 2013). The intensity of fruit colour is intimately associated with ripeness and food acceptance (Delwiche, 2012). Food saliency, visibility and portion size can increase food intake (Dube et al., 2010), and visual presentation can impact the appeal of a food product (i.e. fresh apple compared to an apple puree) (Keller & Duizer, 2014). The multimodal sensory perception of appearance, mouthfeel, aroma, taste (i.e. flavour) and auditory cues impact food choice (Prescott, 2015).

1.1.1. Taste perception

Taste perception is the sensation that results when taste buds convey information about the chemical composition of a soluble stimulus (Bradbury, 2004). Taste and its associated signalling pathways help govern the body's response to incoming food (Trivedi, 2012a). The physiological mechanisms of taste signal the appeal of food items and provide feedback to the digestive system to assist in the regulation of satiety (Trivedi, 2012b). Binding of taste molecules (e.g. glucose, fructose, etc) to specific taste receptors (i.e. G-protein coupled receptors) causes chemical signalling (Lin, 2013). To date there are five recognised primary tastes (bitter, sweet, salt, sour and umami), and the newly recognised fatty acid taste ('oleogustus') (Running, Craig, & Mattes, 2015) which can be described as 'nutrient sensors', facilitating variety seeking and the intake of essential dietary components (van Dongen, van den Berg, Vink, Kok, & de Graaf, 2012). For example, sweet taste facilitates the consumption of energy providing foods which contain sugar (i.e. glucose) (van Dongen et al., 2012) and salt taste facilitates the consumption of sodium which is an essential micronutrient (Liem, Miremadi, & Keast, 2011).

Dependent on life stage, individuals have been shown to vary in their responsiveness to detect and recognise taste (Methven, Allen, Withers, & Gosney, 2012). There is some

evidence that increased density of the fungiform papillae on the tongue is responsible for heightened oral sensitivity for sweet, bitter, and salt tastes (Delwiche, Buletic, & Breslin, 2001; Doty et al., 2016; Haryono, Sprajcer, & Keast, 2014). An individual's responsiveness to a taste stimulus is directly dependent on the concentration of the stimulus. However, there is a wide distribution of detection and recognition thresholds for individual tastants when measured across a population (Keast & Roper, 2007). This heterogeneity in taste behaviour is reflective of the plasticity and the adaptive mechanism of taste receptor expression, as well as a range of other influential factors such as gender, age, and genetics (Dando, 2015).

In order to accurately characterise taste perception, corresponding taste testing methodologies need to be valid, reproducible, and consistent. Taste perception can be measured in a variety of ways, including detection threshold, recognition threshold, discrimination tasks, and ratings of suprathreshold intensity and hedonic liking, which all have important yet distinct characteristics (Haryono et al., 2014; Keast & Roper, 2007). Previous studies on taste perception have mainly used two measures, namely detection threshold, which is defined as 'the weakest stimulus that can be detected', and secondly recognition threshold, which is defined as 'the level at which the stimulus can not only be detected but also recognised' (Bartoshuk, 1978; Cox, Hendrie, & Carty, 2015; Tan & Tucker, 2019). Taste perception can be further defined by the following criteria: (1) there must be a distinct class of affective stimuli; (2) transduction mechanisms include receptors to change the chemical stimulus to an electrical signal; (3) there must be neurotransmission of the electrical signal to the brain; (4) it must have perceptual independence from other taste qualities; (5) there must be physiological effects after activation of taste bud cells, and; (6) it must provide an adaptive (evolutionary) advantage (Mattes, 2011).

1.1.2. Taste perception and dietary intake

Variation in oral sensation and taste hedonics has been proposed to influence dietary intake, as individuals eat what they like and avoid what they dislike (Duffy, 2007). Taste perception or sensitivity to taste may also influence dietary intake through

appetite and satiety mechanisms (Duffy, 2007). Establishing how taste perception is associated with dietary intake may produce significant opportunities for health improvement. However, dietary intake and eating behaviour have been inconsistently linked with taste sensitivity and therefore requires further investigation using robust methodology (Cox et al., 2015).

Recent evidence suggests that taste threshold measurements may have limited use in dietary intake studies, as real-world experiences are not at the low concentrations that are used and measured in threshold studies (Low, Lacy, McBride, & Keast, 2016). The development of the generalised labelled magnitude scale (gLMS) and labelled affective magnitude (LAM) scale has allowed investigating the rating of both intensity and hedonic liking of taste respectively. Measurements using the gLMS can be compared across groups by anchoring each end of the scale (i.e. "strongest imaginable sensation of any kind") negating when taste is perceived differently between individuals (Bartoshuk et al., 2004; Kaufman, Choo, Koh, & Dando, 2018, Schutz and Cardello, 2001). Use of the gLMS at suprathreshold concentration levels has revealed convincing associations between sweet taste perception and dietary intake in more recent studies (Jayasinghe et al., 2017; Low et al., 2016).

There are a range of methods for assessing dietary intake: weighed or estimated food record (in which all foods and beverages are recorded at the time of consumption), and the food frequency questionnaire (FFQ) (in which individuals report how often they have consumed particular foods from a predefined list over a chosen time period, either with or without portion estimation) (Biro, Hulshof, Ovesen, & Amorim Cruz, 2002). Additionally, there is the 24-hour recall (in which individuals recall all foods and beverages consumed over the previous 24-hours), and the diet history (which consists of a series of methods; (i) an interview to determine usual meal patterns, (ii) a food frequency questionnaire, and (iii) a 3 day diet record) (Biro et al., 2002). To date, there is no consensus as to the best dietary assessment method for identifying taste-diet relationships (Tan & Tucker, 2019). It has been recommended that taste-diet studies should measure the whole diet, as opposed to only the comparisons of interest (e.g. associations between bitter taste perception and vegetable intake) to reduce outcome bias (Cox et al., 2015).

The Western-style diet provides a wide range of highly palatable food choices, resulting in dietary intake which is higher in saturated fat and added sugar (Stevenson et al., 2016). Interestingly, intervention studies of both low fat (Newman, Bolhuis, Torres, & Keast, 2016) and low-sugar (Wise, Nattress, Flammer, & Beauchamp, 2016) diets have been shown to increase fat and sweet taste sensitivity, respectively. This suggests that sensitivity for a specific taste can act as a marker of dietary habits in relation to the associated nutrients. Supportive of this, is that taste cells are continually renewed with the average taste cell lifespan of approximately 10-14 days, where normal function of taste buds is dependent on a continuous supply of properly differentiated taste receptor cells (Kaufman et al., 2018). This dynamic taste receptor expression and subsequent taste sensitivity is adaptive, which may be associated with satiety and reward signalling pathways across population groups (Silhan, Robinett, Deshpande, & Liggett, 2012). How the modern food environment interacts with adaptive taste perception mechanisms and subsequent appetite and signalling pathways is yet to be established.

1.2. Taste perception and metabolic health

Despite the emerging evidence of associations between taste perception and dietary intake, individual responsiveness to taste stimuli has not yet been clearly linked to long-term weight status. There is evidence that taste is indicative of appetite regulation and satiety due to its critical role in the signalling of incoming nutrients (Touzani, Bodnar, & Sclafani, 2010). Further signalling occurs as dietary components of the ingested nutrients continues throughout the gastrointestinal (GI) tract (Roper & Chaudhari, 2017). Dysregulation of these signalling and appetitive pathways may be linked to the conditioning of food preferences (Sclafani & Ackroff, 2012).

Causes of obesity are multifaceted and involve complex interactions between genetic, metabolic, cultural, environmental, socio-economic and behavioural factors (Heymsfield & Wadden, 2017; Schwartz et al., 2017). Obesity reflects a state of positive energy balance and arises because of how the body regulates energy intake,

energy expenditure, and energy storage. This imbalance occurs through dietary behaviours that do not trigger strong biological opposition (Hill, 2006). Obesity leads to a state of excessive insulin secretion and a series of metabolic responses that produce systemic insulin resistance (Thompson et al., 2007). Desensitisation to insulin action is accompanied by increased oxidative stress (Hayes & Dinkova-Kostova, 2014), leptin secretion, and inflammation. Subsequent changes in the functioning of endocrine regulators include insulin, leptin, ghrelin and glucagon-like peptide-1 (GLP-1) which disturb appetite regulation (Sumithran et al., 2011). High sugar and fat intake have been proposed as a potential cause of the increasing prevalence of obesity (WHO, 2015, 2018).

1.2.1. Linking taste perception, dietary intake and metabolic health

Sensory perception and the physiological mechanism underlying our taste experience are known to play a role in long-term food choice and eating behaviour (Dando, 2015; Keast, 2016). However, the range of determinants of dietary intake are extensive and include genetic, economic, social, cultural, physiological, environmental, and psychological influences (Story, Kaphingst, Robinson-O'Brien, & Glanz, 2008). By focusing on the biology of taste perception, we gain insight into why people are more vulnerable to a food environment rich in high-fat, sweetened and highly processed foods. Excess energy intake is linked with a multifaceted metabolic condition, it involves interactions between taste, hedonic responses and dietary intake (Calvo & Egan, 2015). Obesity, alongside inflammation, has recently been associated with dysregulation of the renewal of taste buds (Kaufman et al., 2018). Taste perception research may support future intervention studies involving specific dietary approaches to reduce obesity. In turn, weight management and the reduction in metabolic health dysregulation will decrease the risk of type 2 diabetes, some cancers, and heart disease (Dube et al., 2010).

Commonly-cited causes of obesity include major changes in our food environment which have led to over-consumption of inexpensive, highly palatable energy-dense and nutrient-poor foods (Schwartz et al., 2017). Current economic policies expose the

population to strong cues that favour energy availability and a positive energy balance (Story et al., 2008; Swinburn et al., 2013). Previous studies which have observed taste-diet interactions have not adjusted findings for factors such as socioeconomic status. However, this is important as living in a deprived residential setting may lead to decreased dietary choices or variety due to availability and cost (Pechey & Monsivais, 2016). In a recent meta-analysis (Seidelmann et al., 2018), it was found that socioeconomic status was associated with an increased percentage of energy from carbohydrates, primarily associated with higher intakes of refined carbohydrates such as white rice. The prevalence of obesity increases alongside low income, low education and low socioeconomic status (Dube et al., 2010). Associations made between obesity, diet and taste perception should take into consideration the socioeconomic status and health inequities, to determine the key drivers of weight gain (New Zealand Medical Association., 2014).

1.2.2. Sweet and fatty acid taste perception and obesity

Despite knowing that overeating is harmful, many people who are overweight are unable to control their food intake (Calvo & Egan, 2015). The satisfaction gained from eating highly palatable, energy-dense food overcomes satiety feedback mechanisms (Berthoud, 2012). Mechanistic studies suggest that consumption of sugar and fat is driven by taste hedonics, which may exacerbate dietary intake (Keast, 2016). Sweetness and fat have a powerful hedonic appeal, so preferences for sweet and fatty foods are important contributors to increases in body weight and metabolic disease risk (Laffitte, Neiers, & Briand, 2014; Martínez-Ruiz, López-Díaz, Wall-Medrano, Jiménez-Castro, & Angulo, 2014).

Hormones may further modify the intensity of taste perception (Kubasova, Burdakov, & Domingos, 2015; Yoshida et al., 2015). People learn to associate the taste properties from foods with the metabolic consequences of ingestion (e.g. increased feelings of fullness, decreased food cravings, etc), which results in a cycle of learning, expectations, and reward feedback, driving subsequent food selection (Rolls, 2016). Studies in mice have indicated that leptin might function to prevent over-consumption

of sugar by interacting directly with sweet taste receptors at the taste bud (Kawai, Sugimoto, Nakashima, Miura, & Ninomiya, 2000; Yoshida et al., 2015). However, in the leptin-resistant state, such as obesity, this mechanism of regulating sweet-food intake might be blunted (Calvo & Egan, 2015). Additional studies support the existence of functional and homeostatic interactions between glucagon-like peptide 1 (GLP-1) and leptin activity at the level of the taste bud (Martin et al., 2010). If gustatory cues were altered in the obese state, this may lead to increased consumption of food in order to experience the same 'food reward' or satiety signals that a normal weight individual would experience (Maliphol, Garth, & Medler, 2013; Thanos et al., 2015).

In addition, the emergence of unique sweet taster patterns (i.e. 'sweet likers' and 'sweet dislikers') (Kim, Prescott, & Kim, 2014) suggests that a 'one size fits all' dietary recommendation strategy may not be effective. There is recent compelling evidence that in a normal, metabolically healthy state, sweet taste hedonic liking is positively associated with the intake of dietary carbohydrates and sugars (Jayasinghe et al., 2017). Other studies on sweet taste perception have found associations with the hedonic liking of sweet taste and the increased intake of sweet-tasting food and beverages (Garneau, Nuessle, Mendelsberg, Shepard, & Tucker, 2018; Turner-McGrievy, Tate, Moore, & Popkin, 2013). However, taste perception may be inherently different for an obese person in comparison to a normal weight individual, due to the dysregulation of appetite-regulating pathways (Sanematsu, Nakamura, Nomura, Shigemura, & Ninomiya, 2018). Further investigation into taste-diet associations in the non-obese and obese state is warranted.

1.3. The link between taste perception and metabolic health consequences

In our Western society, there is an abundance of energy-dense foods, such as sugar-sweetened beverages and sweet or savoury snacks (Bray et al., 2018). An evolutionary perspective on food and human taste perception suggests that easy access to tasty, energy-dense foods exploits our sensitivities for sugary, and fatty foods that are linked with nutrition-related diseases, such as obesity and type 2 diabetes (Breslin, 2013). In

our current food supply, where many foods are highly processed (van Dongen et al., 2012), it may be that taste perception and the underlying signalling mechanisms associated with taste are to some extent disconnected from the energy content that is being consumed.

Previous studies on taste perception have found associations between psychophysical measurements of taste function and dietary intake. For sweet taste perception studies, sensitivity testing and intensity measures have often lacked any association with dietary intake, whereas hedonic liking ratings have shown clear taste-diet associations (Tan & Tucker, 2019). In contrast, fatty acid taste perception studies have shown taste-diet associations using sensitivity or threshold testing (Cox et al., 2015). Increased sensitivity to fatty acid taste (i.e. hypersensitivity) has been associated with decreased intake of dietary fat (Heinze et al., 2018; Liang et al., 2012; Martínez-Ruiz et al., 2014) and total energy intake (Stewart et al., 2010). The hedonic liking of fatty acid taste in isolation is difficult to measure, as these solutions can evoke 'scratchy' and unlikable taste sensations (Burgess et al., 2018).

Although many studies have investigated the link between sweet and sensitivity to wider associations of fat (i.e. mouthfeel) and/or fatty acid taste perception and obesity, the findings of these studies are inconsistent to date (Cox et al., 2015; Feeney, O'Brien, Scannell, Markey, & Gibney, 2017). These inconsistent and contradicting results highlight the need for further investigation in larger cohorts, with well-defined body fat measurements, using established and validated sensory techniques and assessment of dietary intake.

1.3.1. Significance of research (conducted in my PhD programme)

The current PhD research aims to advance knowledge of the role of taste perception in food choice, food intake, energy balance and weight management. Taste perception phenotypes and taste preferences, with a focus on sweet, bitter and fatty acid taste, will be investigated alongside body composition, dietary intake and eating behaviour measurements. As well as this, the comparison of taste sensitivity, taste preferences and

food choice in different physiological settings (e.g. non-obese and obese) will be conducted. The study population selected for this PhD research are premenopausal women of 18 – 45 years. Women of child-bearing age are an important population to study in this context as increased maternal adiposity has been shown to be related to adverse health outcomes in the next generation (Eriksson, Sandboge, Salonen, Kajantie, and Osmond, 2014). This cross-sectional investigation will expand upon the current literature and will help identify distinct associations between diet and taste perception, and in turn, associations with body composition. Translation of this knowledge will help to identify novel targets for improving dietary intake, eating behaviour and metabolic health.

1.4. Study aims and objectives

1.4.1. Primary aim

The overall aim of this PhD research was to advance our understanding of the relationship between sweet taste, fatty acid taste, bitter taste, additional measurements of sensory perception (i.e. mouthfeel of fat and olfaction) and dietary intake and eating behaviour, and, how this may be associated with biomarkers and endocrine regulators of energy metabolism in premenopausal women in the context of obesity.

1.4.2. Specific objectives

1) To investigate the relationships between (Chapter 3):

1.1. Detection threshold of oleic acid taste, olfactory detection of oleic acid, olfactory detection of *n*-butanol and measurements of fat mouthfeel, and;
1.2. How oleic acid taste, olfactory measurements and measurements of fat mouthfeel may relate to dietary intake and eating behaviour.

The secondary objectives included to:

(i) measure oleic acid taste, olfactory detection and test re-test repeatability;
(ii) explore links between oleic acid taste, olfaction and mouthfeel perception of fat and;
(iii) investigate oleic acid taste detection and associations with eating behaviour, dietary intake, and body composition.

→ **Hypothesis 1:** Hypersensitivity to fatty acid taste detection, olfactory detection of fatty acids and measurements of fat mouthfeel is associated with specific measurements of eating behaviour, adiposity, and dietary intake.

2) To investigate the association between suprathreshold measurements of sweet,

bitter and milk fat mouthfeel perception, intensity, liking and discrimination by ranking task, with (Chapter 4):

2.1. Adiposity in 18-45-year-old women with markedly different metabolic disease risks (Pacific and NZ European women), and;
2.2. The long-term adiposity signals, insulin and leptin, which are known to influence energy balance, body weight and food intake.

The secondary objectives included to:

(i) measure and characterise the perception of suprathreshold concentrations of sweet, bitter and milk fat mouthfeel by intensity rating, liking rating and discrimination by ranking task;
(ii) investigate the liking of suprathreshold concentrations of sweet, bitter and milk fat mouthfeel by hierarchical cluster analysis, alongside comparing different population groups;
(ii) explore associations between measurements of blood glucose regulation and blood cholesterol with sweet taste, bitter taste and milk fat mouthfeel perception and;
(iv) determine the nature of the associations between taste perception and markers of metabolic health by adjusting for additional factors which may influence dietary intake (e.g. age, socioeconomic status).

→ **Hypothesis 2:** Hedonic liking of sweet taste, bitter taste, and milk fat mouthfeel perception varies between people and is associated with specific differences in adiposity and metabolic regulators of adiposity.

3) To increase our understanding of the influence of sweet taste, bitter taste and milk fat mouthfeel perception, intensity, liking and discrimination by ranking task, on (Chapter 5):

3.1. Dietary intake measured by a 5-day non-consecutive estimated food record, and;
3.2. Eating behaviour measured by the three-factor eating questionnaire.

The secondary objectives included to:

(i) determine the strength of the associations between taste perception and dietary intake by adjusting for additional factors which may influence dietary intake (e.g. age, socioeconomic status); and
(ii) determine the strength of the associations between taste perception and eating behaviour by adjusting for additional factors (e.g. age, socioeconomic status).

→ **Hypothesis 3:** Increased hedonic liking of suprathreshold taste solutions (sweet taste, bitter taste and milk fat mouthfeel perception) is associated with specific differences in dietary intake. In addition, hedonic liking of suprathreshold taste solutions is associated with specific differences in cognitive measurements of eating behaviour (i.e. dietary restraint, disinhibition of control and susceptibility to hunger).

1.4.3. Structure of the thesis

This thesis begins with a review of the literature focusing on influences of food choice, the physiology of taste perception, the methodology used in sensory research and links between sensory perception and metabolic health. This is followed by three manuscripts presenting the results of this research (Table 1.1). As each study is presented in the form of a manuscript suitable for publication, there may be some repetition throughout the thesis. For example, there is repetition in the material and methods sections of chapter 4 and 5 as both chapters use data from the same study.

To reach our objectives (or to test these hypotheses) we developed two cross-sectional studies. The first study titled the 'Dessert Taste study' was a cross-sectional study which investigated fatty acid taste perception alongside olfactory and mouthfeel perception, with further associations between eating behaviour and adiposity reported (chapter three). This manuscript was published in the journal '*Nutrients*' (Kindleysides et al., 2017).

Table 1.1. Outline of current publication status of research chapters

Outline of current publication status of research chapters		
Chapter	*Current status*	*Journal*
III	Published	Kindleysides, S.; Beck, K.L.; Walsh, D.C.I.; Henderson, L.; Jayasinghe, S.N.; Golding, M.; Breier, B.H. Fat Sensation: Fatty Acid Taste and Olfaction Sensitivity and the Link with Disinhibited Eating Behaviour. *Nutrients* **2017**, *9*, 879.
IV	Peer reviewed	International Journal of Obesity
V	To be submitted	Nutrients OR American Journal of Clinical Nutrition

The second cross-sectional study titled the 'PROMISE study' was a larger study conducted in healthy, premenopausal Pacific and NZ European women, with different

body fat measurements (chapter four and five; Figure 1). The second manuscript explores the perception of sweet taste, bitter taste, and milk fat and compares differences in taste perception with markers of metabolic health (chapter four). The third research manuscript investigates dietary intake and how sweet taste, bitter taste and milk fat perception influence dietary intake and eating behaviour (chapter five). The study protocol and recruitment strategy manuscript for the PROMISE study was published in the journal '*JMIR Research Protocols*' (Kindleysides et al., 2019). This publication is presented in the appendix (Appendix 1.5).

The overview of participant recruitment for the two studies is shown in Figure 1. The thesis concludes with a discussion on the main results, new insights and the studies' strengths and limitations. The the final discussion also highlights the new knowledge presented in this PhD thesis.

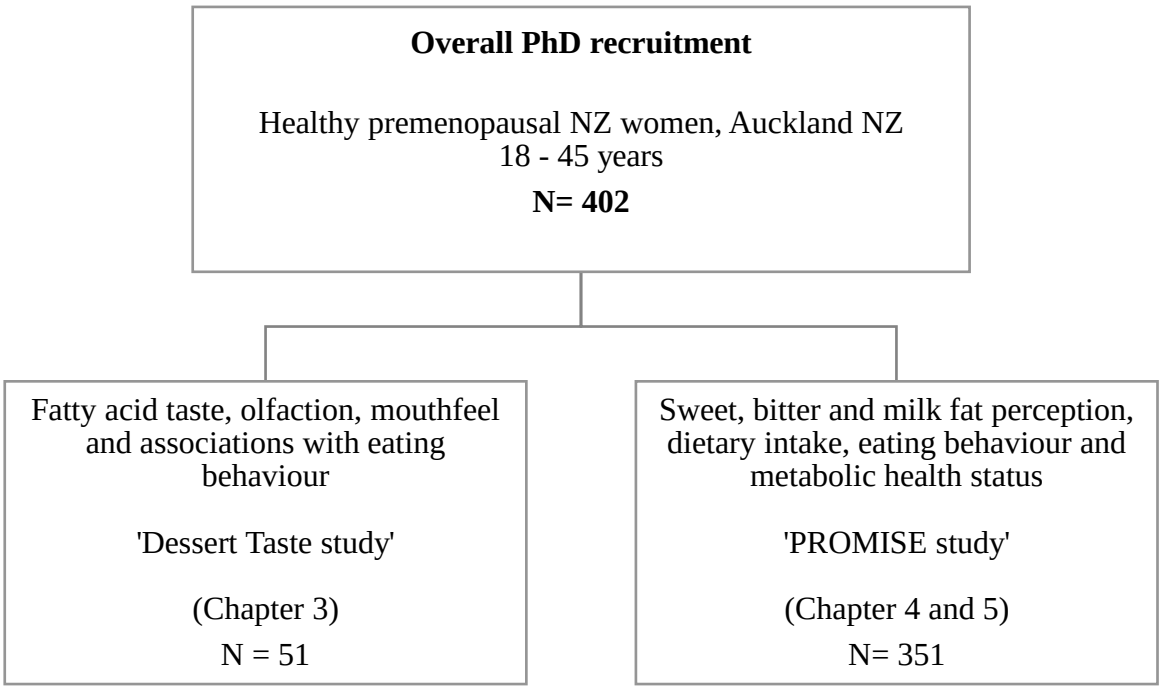

Figure 1.1. Overview of participant recruitment

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Chapter II.

Literature review

2.1. OVERVIEW

In this literature review, determinants of food choice including sensory perception and taste are explored. The role of taste is critically evaluated to better understand its overall physiological function. This review compares taste perception to other primary senses and reviews how recent studies provide evidence for a link between taste sensitivity and health outcomes. The current methodology for the evaluation of taste and aroma is critically evaluated. Individual responsiveness to taste is investigated, as it can vary across different population groups which may be connected to alterations in food selection and long-term dietary behaviour. Dietary intake and eating behaviour methodology are also assessed, with a key focus on developing a link with taste perception. Finally, a critical review of the current literature connecting taste perception, dietary intake and body composition completes this chapter.

2.1.1. DETERMINANTS OF FOOD CHOICE

When choosing food and beverages to eat or drink, taste appears to have a strong influence. Many individuals choose exciting tastes and intense flavours to satisfy their hunger or thirst. However, other influences, both external and internal impact on foods and beverages selected for consumption. These influences may encourage or deter an individual from overeating (Story, Kaphingst, Robinson-O'Brien, & Glanz, 2008; van Dongen, 2012) (see Figure 2.1).

Society	Social environment and community	Individual factors

• Society and cultural norms
• Food industry
  ▪ *Food marketing*
  ▪ *Food production and distribution*
• Government and political systems
	• Family and friends
	• Home
	• Workplace
	• School
	• Neighbours
	• Restaurants and fast food outlets
	• Supermarkets
		• Food preferences
		• Attitude
		• Culture and religion
		• Knowledge and education
		• Skills
		• Demographics
		• Lifestyle
		• Biological factors
		  ▪ *Genetics*
		  ▪ *Age*
		  ▪ *Gender*
		  ▪ *Taste & sensory perception*

Figure 2.1. Factors that influence an individual's eating habits (Story et al., 2008; van Dongen, 2012). Figure used with permission from Annual Reviews, Inc.

2.1.1.1. Society

Societal influences on dietary behaviour and intake include socio-cultural influences, the food industry, government and political systems. Socio-cultural norms influence long-term dietary habits through traditional ideas within a society (Jahoda, 2012). For example, in New Zealand (NZ), differences in eating habits are seen in Pacific people, who have a greater focus on sharing food than NZ European people (Tupai-Firestone et al., 2016). The food industry drives food availability, alongside the industry being profit-driven, which often conflicts with healthy eating recommendations and portion control (Roberto et al., 2015). A range of food products have higher quantities of sugar, fat, and salt, which increases the reward value of foods (Roberto et al., 2015; Wansink & Sobal, 2007). However, there are many products which are marketed as low in sugar, fat, or salt to meet consumer demand (Hutchings, Low, & Keast, 2018). The food industry in NZ is investing in digital media which enables food marketers to directly engage with their target audiences in dynamic ways to sell food products (Vandevijvere, Sagar, Kelly, & Swinburn, 2017). Additionally, there is an association between

geographic access to fast food outlets and neighbourhood deprivation in NZ, which influences food choice and may contribute to obesity (Pearce, Blakely, Witten, & Bartie, 2007).

The political environment can positively influence health and well-being if policies, food regulation and education in schools are carefully instated. For example, the introduction of high salt warning labels in Finland has been successful in reducing the intake of salt and encouraging food manufacturers to develop foods with lower salt content (Pietinen, Valsta, Hirvonen, & Sinkko, 2008). In NZ, there is an ongoing drive to tax sugar which is supported by the World Health Organisation, academics, local health groups and health professionals (Cropp, 2017). A sugar tax is expected to reduce consumption of sugar-sweetened beverages, with a corresponding small decrease in caloric intake, based on the data from high and middle-income countries that have successfully instated a sugar tax (Backholer et al., 2016; Nakhimovsky et al., 2016).

2.1.1.2. Social environment and community

The social environment and the community (e.g. family, friends, workplaces, schools, etc) have an influence on food choice. Families have an influence on food choice due to providing the social environment where eating patterns and food preferences develop during childhood (Story et al., 2008). In children and adolescents, the presence of peers and friends can increase energy intake, except in situations where peers exhibit healthy eating (Salvy, de la Haye, Bowker, & Hermans, 2012). Therefore, friends and social facilitation have a major influence on food and beverage choices, particularly during adolescence (Herman, Roth, & Polivy, 2003). Schools and workplaces can also impact which foods are available to eat and may provide opportunities to facilitate or hinder healthy eating (Gerritsen & Wall, 2017). Accessibility of fast food outlets and takeaway options are increasingly prevalent but may contribute to poorer-quality diets in NZ (Mackay, Vandevijvere, Xie, Lee, & Swinburn, 2017).

Health intervention strategies can be implemented through the social changes of communities. An example of such a health intervention strategy addresses the increase

in levels of childhood obesity in NZ is that of 'Project Energize', which is a region-wide whole-school nutrition and physical activity programme (Rush et al., 2014). Results of this work have shown that the long-term regional commitment to the programme in schools may enhance healthy eating and a reduction in weight gain (Rush et al., 2014).

2.1.1.3. Individual factors

Individual determinants of food choice include food preferences, individual attitudes, culture and religion, knowledge and education, skills, demographics, lifestyle and biological factors. Food preferences can have an important influence on dietary habits that can last over a lifetime. Culture and religion may impact food choice by restricting food intake (i.e. Ramadan) or by eliminating distinct food items from the diet (i.e. restrictions on the consumption of pork in Jews and Muslims) (Ali & Abizari, 2018; Meyer-Rochow, 2009). Knowledge and education may improve dietary intake. It has been previously shown that education level is positively correlated with improved diet quality (Thiele, Mensink, & Beitz, 2004). Conversely, those who are less educated alongside having a lower income, are at an increased risk of weight gain (Johnston & Lordan, 2014). In addition, individual skills (i.e. cooking skills) increase the probability of eating meals at home, which is associated with improved diet quality and therefore a decreased risk of obesity (Zong, Eisenberg, Hu, & Sun, 2016).

Demographics can further impact food choice, for example, males typically consume more food than females due to their higher physiological requirements (Rolls, Fedoroff, & Guthrie, 1991). Lifestyle factors, in addition, can play an important role in dietary intake. For example, it has been shown that individuals who are shift workers skip more meals and have increased intake of foods which are high in sugar and fat (Souza, Sarmento, de Almeida, & Canuto, 2019).

In the current PhD study, biological factors which include genetics, age, gender, taste and sensory perception, are acknowledged as critical influencers of food choice. However, one of the most influential factors governing food intake is taste perception (Breslin, 2013). Consumer behaviour, long-term habits and hedonic liking of food are

affected by taste and flavour perception. For example, sugar-sweetened beverages are a major contributor to weight gain and obesity (Malik, Pan, Willett, & Hu, 2013), and one key reason for high consumption of sugar-sweetened beverages is their enhanced taste and palatability (Garneau, Nuessle, Mendelsberg, Shepard, & Tucker, 2018). The following sections will describe the physiology of taste perception, alongside olfactory and mouthfeel sensory modalities as important factors that impact, influence and motivate food choice.

2.2. SENSORY PERCEPTION

2.2.1. MULTIMODAL SENSORY PERCEPTION - AN OVERVIEW

The ability to ‘sense’ comes from the physiological capacity of an organism to translate external information into a feeling or sensation. This perception requires sensory cell types that are able to respond to a specific physical input and corresponds to a group of regions within the brain where the information is received and decoded (Rolls, 2007). The physical properties of food can affect many of our senses, including taste, touch (e.g. texture), sound (e.g. crunchiness), smell, thermoception, kinaesthetic sense and even pain. Figure 2.2 shows the physiology and the receptors for taste and aroma and how these signals are transmitted to the brain.

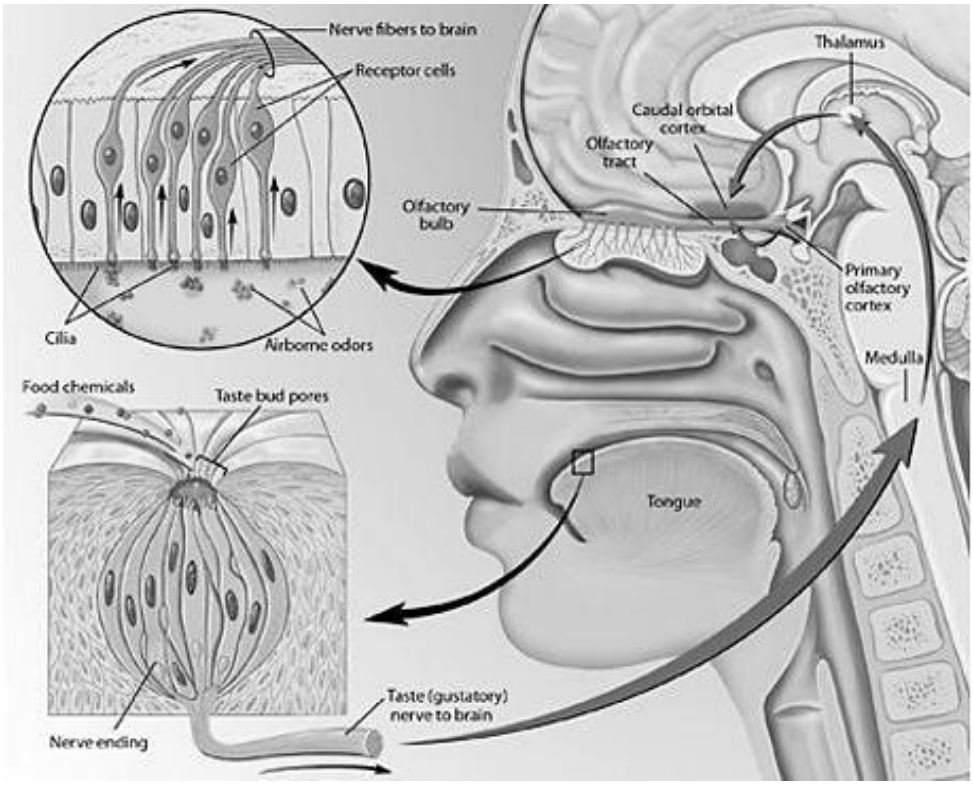

Figure 2.2. Basic physiology of taste and smell receptors signalling to the brain. The combination of sensory signals leads to our interpretation of flavour perception (Society for Neuroscience, 2012). Image used with permission from the Society for Neuroscience (SfN).

2.2.1.1. Sensory perception and the digestive system

Variation in individual responses to sensory stimuli has been reported over a number of decades (Pangborn, 1959). Genetics, age, gender and body composition are all proposed to have differentiated effects on an individual's sensory perception. Further, sensory preferences are thought to be a result of positive energy intake in the gastrointestinal tract creating a feedback loop which induces hunger and craving for familiar foods (Figure 2.3.). For example, the consumption of a highly palatable meal (e.g. a hamburger) is reinforced by appetitive sensory stimulation. As the meal is consumed the person will decide on how much to consume. Satiation occurs during or after the consumption of the meal, which triggers a cascade of hormones that drive short-term (e.g. Glucagon-like peptide 1, Peptide YY) and long-term (e.g. insulin, leptin) appetite regulation (Figure 3). These signals apply the 'brake' on eating when digestive components transit through the small intestine (Shin, Ingram, McGill, & Poppitt, 2013; Steinert et al., 2017).

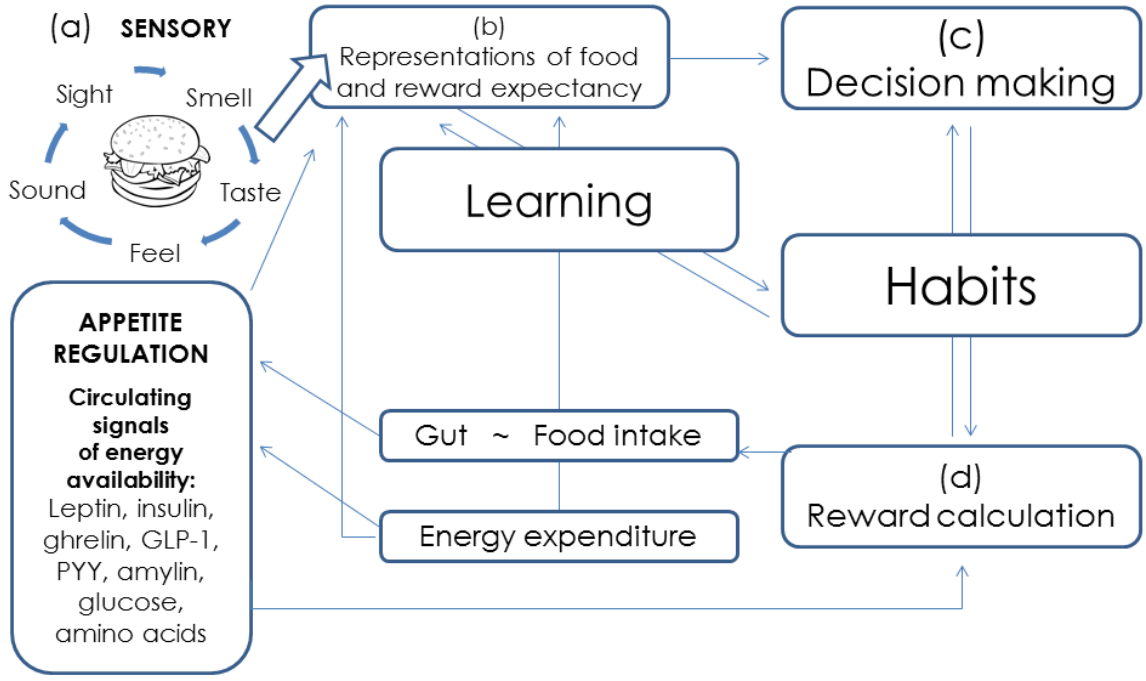

Figure 2.3. The hypothalamus and brainstem play a crucial role in the control of ingestive behaviour (Berthoud, 2011, 2012). The brain aids the modulation of food intake and energy expenditure by (a) external (taste, sight, sound, feel and smell) sensory input to the hypothalamic energy sensor, (b) input from the reward processing system, (c) inputs from the emotional motor system, and (d) voluntary behavioural

control (adapted from Berthoud, 2012). Image (adapted) used with permission from Elsevier.

The brain plays a critical role in anticipating food intake and responds to sensory cues which prepare the body for digestion and absorption of nutrients. The preparation for digestion already occurs during the tasting process and even prior to the experience of taste perception, initiated by the visual and olfaction cues of the food, such as the non-conscious salivating response in anticipation of eating (Krishna, Morrin, & Sayin, 2014). Therefore, taste and oro-sensory cues are quickly and strongly associated with the post-ingestive cues of eating, which includes hormonal and metabolic pre-absorptive reflexes (Swithers & Davidson, 2008).

Appetitive sensory cues (e.g. taste, aroma, mouthfeel) may lead to the programming of food craving and habits, so when signals of hunger are increased, there will be a drive to consume food. This drive will continue until the point at which nutrients are sensed in the gastrointestinal tract, which triggers the release of appetite-regulating hormones indicating fullness (Maljaars, Peters, Mela, & Masclee, 2008; Shin et al., 2013). It has been suggested that this predictive control pathway may be compromised by highly processed food (e.g. flavour or aroma enhancers), due to a discrepancy between sensory signalling and the actual energy content consumed (van Dongen, van den Berg, Vink, Kok, & de Graaf, 2012).

2.2.2. TASTE

Human beings eat in order to meet the energy and nutrient requirement essential for survival. From an evolutionary perspective, taste perception has theoretically evolved primarily as a way to decide if the selected food is safe, suitable and whether or not it will contribute to energy and nutrient demands (Niven & Laughlin, 2008). For example, bitter taste can signal the presence of toxic compounds (Drewnowski, 1997), whereas sour taste can represent the presence of acidic or unripe foods (Huang et al., 2006). The cognitive translation of these signals leads to the development of long-term dietary preferences and habits. The next part of the literature review will describe the

physiology of taste perception and the primary tastes (e.g. sweet, salty, bitter).

2.2.2.1. Basic taste physiology

The gustatory system recognises chemical stimuli that trigger distinct qualities: sweet, salty, bitter, umami and sour. In addition, a proposed sixth taste for fatty acids termed 'oleogustus' has strong supportive evidence of being defined as a basic taste (Running, Craig, & Mattes, 2015). Taste detection occurs primarily through cells present on the tongue, and activation of these taste cells by specific stimuli releases neurotransmitters onto afferent cranial nerve fibres to elicit taste information (Fernstrom et al., 2012). The interaction of taste and flavour (i.e. flavour is the combined sense of taste and odour) occurs during the chewing process (Prescott, 2012). If food is sensed as being unsuitable during this process it will be immediately rejected or it may result in a learned association to avoid that food or taste in future (Mennella, Reiter, & Daniels, 2016).

2.2.2.2. Tongue, fungiform papillae and taste cell types

The tongue is a complex organ containing a variety of papillae types (circumvallate, filiform, fungiform, and foliate papillae). Within each of these papillae, there are taste buds. There are 50 – 100 taste buds per papillae (see Figure 2.4), that are anatomically classified into four types of taste bud (Trivedi, 2012a). It has been proposed that most of the taste cells on the tongue are short-lived, lasting only for a few days (Chandrashekar, Hoon, Ryba, & Zuker, 2006), but that some taste cells are longer-lived cells lasting up to 10 weeks (Hamamichi, Asano-Miyoshi, & Emori, 2006). Whilst specialist taste cells are tuned to one taste quality only (i.e. sweet), there appears to be cell to cell communication (paracrine transmission) between specialist and generalist cell types, which work in unison to send a transmission signal to the brain (Roper & Chaudhari, 2009, 2017). The interaction of multiple tastes and how the signals are transmitted is complex and there are still several interactions that are yet to be understood.

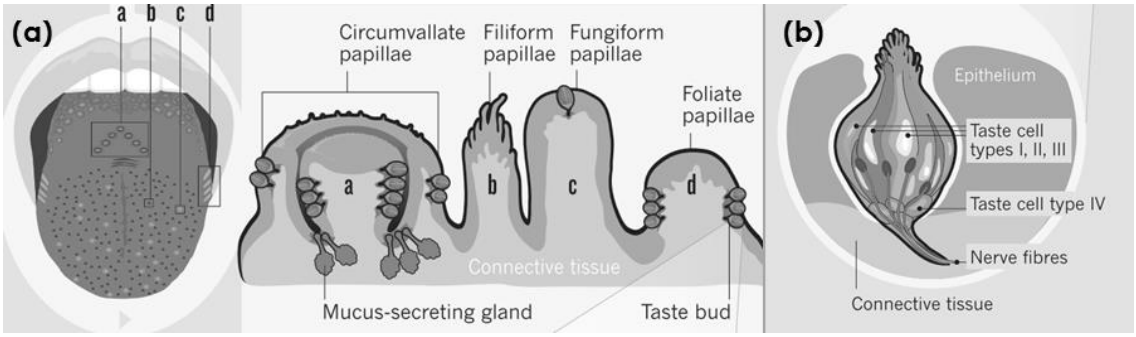

Figure 2.4. Four types of papillae (a) are present on the human tongue (circumvallate, fungiform, filiform and foliate papillae). Circumvallate, fungiform and foliate contain taste buds; (b) whereas the filiform papillae detect the texture of food (adapted from Trivedi, 2012). Image used with permission from Springer Nature.

2.2.2.3. G-protein coupled receptors and taste physiology

The discovery of taste perception via G-protein coupled receptors (GPCRs) was highlighted by the Nobel Prize in chemistry awarded to Lefkowitz and Kobilka in 2012 (Lefkowitz, 2013; Lin, 2013). The receptors for sweet, bitter and umami are GPCRs (Gravitz, 2012), as well as fatty acid taste by GPR120 (Galindo et al., 2012). There is a fast transition from the taste stimulant (ligand) binding to the appropriate GPCR, initiating a signalling cascade that activates a chain of signalling events that lead to taste recognition (Hausch & Holsboer, 2012). Taste sensation is transmitted to the brain by multiple pathways, therefore the total loss of taste is very uncommon (Bromley, 2000).

2.2.2.4. Sweet taste

The innate preference for sweet is present early in life and is arguably one of the key sensory drivers to consume breast milk, which is vital for our survival during infancy (Mennella, 2014). Sweet taste stimuli bind to and activate the taste receptor type 1 member 2 (T1R2) and taste receptor type 1 member 3 (T1R3) receptors present on taste buds on the tongue. Sucrose, aspartame, cyclamate, saccharin and a range of other compounds taste ‘sweet’ by triggering the T1R2 and T1R3 receptors, however, chemically these compounds are relatively diverse (Fernstrom et al., 2012).

2.2.2.5. Salt taste

Salt is an essential micronutrient. Salt or sodium chloride (NaCl) has highly palatable influence on a wide range of foods (McLean & Hoek, 2014). The salt taste mechanism was confirmed in 2010, as an ion channel called the epithelium sodium channel (ENaC) sodium-salt taste receptor (Chandrashekar et al., 2010; Trivedi, 2012a). The brain is said to be finely tuned and highly selective when discerning 'salt taste' and this may be attributed to a strong innate preference for NaCl. This craving or an increased desire for 'salt taste' has been attributed to the importance of salt in our diet which influences many important physiological functions (Liem, Miremadi, & Keast, 2011).

2.2.2.6. Bitter taste

Bitter tastants trigger bitter taste receptors, which also belong to the superfamily of GPCRs. However, unlike the positive response that sweet taste has, bitter taste leads to an adverse reaction and is typically and instinctively 'disliked' (e.g. bitter vegetables, medicine). In some cases, however, bitter taste can be highly enjoyable (e.g. coffee, tea). The initial physiological reaction to bitter taste may be similar to that of poison or something that should not be ingested (Garcia-Burgos & Zamora, 2013; Mennella, 2014). Over time, however, there tends to be a change in the perception of bitter taste components in that individuals accept a wider range of vegetables, or products like coffee and tea, leading to long-term acceptability (Mennella, 2014).

In previous studies, bitter taste was described as a key indicator of taste ability, with the term 'supertaster' applied to those who could perceive the presence of phenylthiocarbamide (PTC) or its chemical relative, 6-n-propylthiouracil (PROP) (Bartoshuk, Duffy, & Miller, 1994). The origins of this classification stem from a laboratory accident in 1931, whereby PTC unintentionally was inhaled and tasted bitter to one individual, but not to the other individual (Anonymous, 1931; Wooding, 2006). Further inquiry by Fox and colleagues determined that some individuals are "taste blind" to PTC/PROP and others are not. Much later it was found that differences in the perception of bitterness recognition can be accounted for by differences in polymorphisms in the bitter receptor gene *hTAS2R38*, coding for distinct receptor types

(Bufe et al., 2005; Kim et al., 2003). A single specific gene detects bitterness in PTC/PROP, but that gene represents one of approximately 25 bitter taste receptors (TAS2R) (Behrens & Meyerhof, 2006; Lossow et al., 2016).

2.2.2.7. Umami taste

The taste 'umami' is a description of the savoury-like taste that results from the amino acid taste receptor. The umami taste is linked with activation of the GPCRs T1R1 and T1R3, which have been termed as broadly tuned to respond to most of the 20 amino acids, but not their D-enantiomers or other compounds (Nelson et al., 2002). It is thought that these savoury tasting percept's binding to the GPCR dimer can also bind to other receptors called metabotropic glutamate receptor 1 and 4 (mGluR1 and mGluR4) (Chaudhari, Pereira, & Roper, 2009). The umami taste is predominately associated with the taste of monosodium glutamate (MSG), which is often used as a flavour enhancer in processed foods and is found naturally in tomatoes, mushrooms and parmesan cheese.

2.2.2.8. Sour taste

Sour taste is triggered by the presence of protons (H^+), a breakdown product from acidic foods. The mechanism of sensing sour is hypothesised to prevent the ingestion of unripe or acidic food choices which may cause acute sickness or harm (Baeyens, Vansteenwegen, De Houwer, & Crombez, 1996). Sour taste can have some appeal and the biological instinct to reject this taste can be altered through habituation, in a similar way to bitter taste. Changes to the sensitivity to sour taste has been further proposed to aid in the detection of key vitamins in the context of deficiency (i.e. Vitamin C) (Teng et al., 2019).

The mechanism for sour taste has been recently described by Teng et al as a proton-selective ion channel Otop1 (Teng et al., 2019). These ion channels are expressed by type III taste receptor cells (TRCs), located in taste buds across the tongue which selectively detect the presence of acidic stimuli (i.e. H+). When studying sour taste

recognition and hedonic liking, the most common substance used is citric acid (Wise & Breslin, 2013). Acceptance of citric acid has been shown to relate to fruit intake and sour taste acceptance in toddlers (Blossfeld et al., 2007).

2.2.2.9. Fatty acid taste

Studies have shown that humans can detect fatty acid taste at low concentrations (Chalé-Rush, Burgess, & Mattes, 2007; Keast & Costanzo, 2015; Mattes, 2005), however, the sensation is extremely complex. Unlike other tastes (e.g. sweet, bitter) the taste of a pure short-chain fatty acid is difficult to describe and can be confused with 'sour' (Running et al., 2015). Fat has gustatory, olfactory, and somatosensory cues, reflecting the broad sensitivity to fatty acid stimuli. There are several proposed receptors for fatty acids which are present on the tongue, as well as on entero-endocrine cells throughout the gastrointestinal (GI) tract (Figure 2.5).

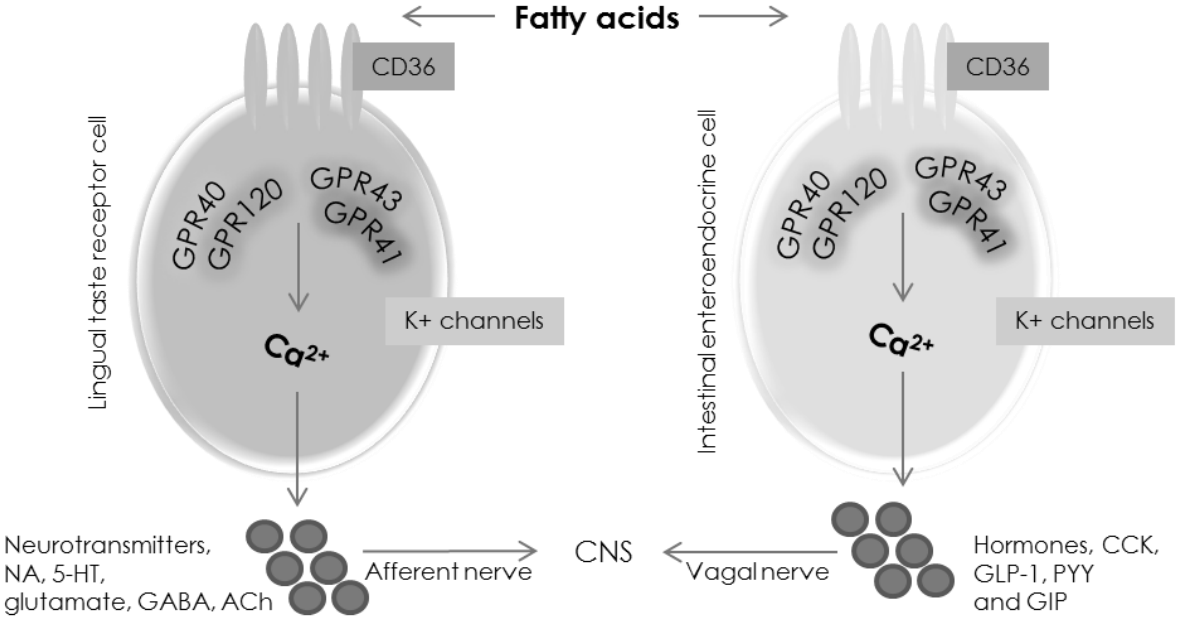

Figure 2.5. Fatty acid receptors and transporters, which have been isolated from gastrointestinal (GI) enteroendocrine cells of both rodents and humans. Receptors include a cluster of differentiation-36 (CD36), as well as G protein-coupled receptors (GPR120, GPR40, GPR41, and GPR43) and delayed rectifying potassium (K^+) channels (Stewart, Feinle-Bisset, & Keast, 2011). Figure (adapted) used with permission from Elsevier.

The gustatory receptor elements that have been attributed to fatty acid taste are CD36, GPR120, GPR40 and two long chain fatty acid (LCFA) specific receptors (Galindo et al., 2012). Evidence suggests that fatty acid stimulation could involve a cascade of more than one of the above receptor candidates (Galindo et al., 2012).

2.2.2.10. Sensory perception of fat terminology

Dietary fats are perceived by multiple sensory pathways. As such, it is important to clearly define each mode of the sensory perception of fat:

(1) Oral perception of fatty acids (**fat taste or fatty acid taste**). Fat taste has been recognised as a primary taste (Running et al., 2015). For fatty acid taste to be perceived, free fatty acids must activate fat taste receptors located on taste cells. Lingual lipases in the oral cavity can hydrolyse triacylglycerides which will increase free fatty acid exposure to taste receptors. However, in large amounts, free fatty acids elicit a rancid taste (Costanzo et al., 2017).

(2) Texture or mouthfeel perception of fat (**fat mouthfeel**). The perception of triacylglycerides differs from the perception of free fatty acids (e.g. fatty acid taste) as triacylglycerides impart odour, textural and irritant dimensions. Textural properties of fat are perceived by multiple routes (i.e. somatosensory). These fat textural cues are transmitted via trigeminal neurons (DiPatrizio, 2014).

(3) Olfaction or odour perception of fat (**fat olfaction**). Aroma of fat (i.e. odour) is perceived by volatiles which are perceived by olfactory receptors (Boesveldt and Lundström, 2014).

(4) Overall perception of dietary fats (**fat perception**). The combined perceived sensation of all the sensory properties of fat (i.e. taste, mouthfeel, olfaction, etc) can be described as fat perception.

2.2.3. OLFACTION

2.2.3.1. Olfaction pathways and physiology

Olfaction is a result of odorants binding to specific sites on olfactory receptors located in the nasal cavity, which form the sense of smell. Humans have around 400 functional olfactory receptors which stem from one of the largest gene families known across all animal species. The olfactory receptor cells themselves are primarily bipolar sensory neurons where the axon extends without synapsing to the central nervous system (Ache & Young, 2005). Due to olfactory receptors working in combination, it has been suggested that humans can detect 1 trillion different olfactory stimuli (Bushdid, Magnasco, Vosshall, & Keller, 2014). In other words, a single receptor is able to recognise multiple odours, and a single odorant is recognised by multiple receptors (Ghinea & Ademoye, 2010). This complex mapping of different combinations of odorant receptors creates a vast array of odours which are interpreted by the brain. Only 10% of olfactory receptors have known agonists and very little is known about specific human olfactory receptors (Gonzalez-Kristeller, do Nascimento, Galante, & Malnic, 2015).

Odorous chemicals enter the nasal passage during inhalation, and they dissolve in the olfactory mucus, where they diffuse or are actively transported to receptors on the cilia of olfactory receptor cells (Bromley, 2000). The anatomic ties between the olfactory system and the hypothalamus, amygdala, and hippocampus help explain the intimate associations between odour perception and cognitive functions such as memory, motivation, activity, and digestion (Doty & Bromley, 2012).

When food is chewed, volatiles are released that are sensed by bipolar neurons which are orthonasal (olfaction arising from odour compounds passing through the external nares or nose) and retronasal (odour compounds passing through the internal nares or through the mouth, more commonly sensed after swallowing) (Small, Gerber, Mak, & Hummel, 2005). Complex aroma signals are interpreted by the brain and the interplay between that input and other sensations (e.g. taste, textural attributes, visual) creates the

final interpretation of the aroma. For example, the perception of strawberries and their flavour is enhanced substantially by breaking down the fruit via homogenisation, which releases more odorant compounds equivalent to chewing (Ingham, 1995).

The reciprocal effects of odour and taste are of interest, due to different types of exposure and learning over our lifetime (Prescott, 2012). When presented simultaneously, ‘strawberry odour’ with sweet taste (i.e. sucrose) and ‘grapefruit odour’ with sour taste (i.e. citric acid) will significantly improve taste recognition speed (White & Prescott, 2007). This phenomenon is thought to be a result of prior co-occurrence and repeated exposure. It is apparent that not only the taste preference but in fact the unique somatosensory interaction, is driving individual dietary behaviour.

2.2.3.2. Olfactory-based fat discrimination

Detection of fatty acids is unique in that humans are able to detect high concentrations of fatty acid by the sense of smell alone. Humans may be able to detect the fat content of food via olfaction due to the evolutionary pressure to detect fat as an important energy source (Boesveldt & Lundström, 2014). In contrast, other tastes (e.g. sweet, bitter) are sole ‘taste’ sensations that do not elicit a sense of smell. Recently, a study has revealed that humans are able to discriminate the presence of varying levels of fat in milk samples using smell and that this ability reflects the ability to detect fat content in a range of real food products (Boesveldt & Lundström, 2014). Another study has demonstrated that odour thresholds for a range of free fatty acids (e.g. linoleic, oleic and stearic acids) can be measured, however, a lack of association was observed between these threshold measures suggesting they function independently (Chalé-Rush et al., 2007). New and innovative future approaches towards a reduction in dietary fat intake may benefit from a better understanding of the physiology of olfactory-based fat discrimination.

2.2.4. MOUTHFEEL / TEXTURE

2.2.4.1. Mouthfeel physiology

Mouthfeel or texture is a product's interaction with the mouth, which can be experienced through the physical interaction of biting, chewing (mastication) and swallowing. During each stage of food fracture and breakdown, the perception of how the product feels in the mouth will change. Mouthfeel can be defined in detail, throughout each stage of mastication. Typical descriptions of the perception of mouthfeel include density, dryness, graininess, gumminess, hardness, heaviness, mouth-coating, roughness, slipperiness and smoothness (Hutchings, Foster, Hedderley, & Morgenstern, 2014). Descriptions such as these often give food their unique quality or characteristic, so the association of mouthfeel with flavour is a crucial relationship for food acceptance. Oral processing is becoming more critical in the understanding of overall sensory perception and hedonic preference. A recent development in this area is the classifications of oral processing in individuals and the way food is orally perceived (see Table 2.1).

Table 2.1. The U.S. population mouth behaviour classification and categorisation. Source: The Understanding and Insight Group, Limited Liability Company (LLC) (Jeltema, Beckley, & Vahalik, 2014). Table used with permission from John Wiley and Sons.

Classification	Qualitative hedonic preference for chocolate	Approx. % of U.S. population
Chewers	Prefer chocolate that has a good chewing texture.	43
Crunchers	Prefer chocolate that contains nuts.	33
Smooshers	Prefer chocolate that melts fast.	16
Suckers	Prefer chocolate that is hard enough to suck on. Prefer to alternate chewing and sucking on chocolates.	8

Mouth behaviour alters the perception of flavour intensity and the availability of aroma volatiles, which in turn, may influence dietary intake and eating behaviour (Jeltema et al., 2014). An example of this is that 'chewers' will breakdown food in the oral cavity more so than 'suckers' would, resulting in an increase in aroma volatiles interacting

with olfactory receptors, which subsequently increases flavour intensity.

2.2.4.2. Mouthfeel-based fat and sugar discrimination

The textural qualities of highly palatable foods will contribute to the overall appeal, which can be altered by ingredients such as fat and sugar (i.e. found in foods such as chocolate). The quantity of fat present in a food product or liquid will significantly alter mouthfeel and textural qualities. Some measurements of the textural properties of dairy products include the tribological measurement, which includes lubricating qualities which can be compared to the oral breakdown rate in the mouth (Nguyen, Bhandari, & Prakash, 2016). It has been reported by trained panels that fattiness perception is highest in emulsions of fat that melts at body temperature, being high in saturated fat (e.g. solid milk fat) in comparison to other oils such as sunflower oil (Vingerhoeds, de Wijk, Zoet, Nixdorf, & van Aken, 2008). The higher degree of saturated fat leans itself toward shear-induced coalescence of these emulsions and therefore increased fat-related textural attributes. Fattiness perception may be determined by the mouthfeel of the fat itself, or instead, by taste receptor interaction of the fatty acid. The combination of taste and textural attributes, as well as olfaction, may enhance fatty acid recognition (Stevenson et al., 2016).

Such an ability to perceive fat content may vary between fatty acid taste hypo- and hypersensitive individuals (Stewart et al., 2010). This question of the liking of textural attributes is of interest, as it is well known that commercial products with ‘fat replacers’ that mimic the textural attributes of fat often fail to attain the same level of hedonic liking or craving response than that of a full fat equivalent (Jervis, Gerard, Drake, Lopetcharat, & Drake, 2014).

Sugar not only contributes sweetness to a product, but it also contributes to the body and mouthfeel of foods and beverages. Significant differences are found between artificially sweetened and caloric sugar equivalents when evaluating mouthfeel (Oliveira et al., 2015). Sucrose mouthfeel detection thresholds have been measured in studies (Hewson, Hollowood, Chandra, & Hort, 2008; Kappes, Schmidt, & Lee, 2006). Therefore, reformulation of products with reduced sugar has the added complexity of

impacting the contribution sugar has on texture, viscosity, and mouthfeel (Hutchings et al., 2018).

2.2.5. REGULATORS OF METABOLIC HEALTH AND DIGESTIVE PHYSIOLOGY

2.2.5.1. Gastrointestinal tract physiology

The physiology of nutrient-sensing involves the multimodal perception of oral taste, olfaction and mouthfeel chemo-sensation, but taste is also further sensed within the GI tract (Janssen & Depoortere, 2013). Food learning in itself has been proposed to involve multiple processes, where oral and post-oral properties of nutrients (e.g. nutrient feedback) allow for the preference of an associated taste (Touzani, Bodnar, & Sclafani, 2010). Gut chemosensors are thought to be linked to digestive effects, providing positive feedback which may condition food preferences over time (Sclafani & Ackroff, 2012). Receptors in the gut have been found to be homologous to the taste receptors found in the oral cavity and consist of GPCR mediated signalling and ion channel pathways (Reimann, Tolhurst, & Gribble, 2012). GPCRs are distributed throughout the stomach, intestine, and pancreas, acting to aid the digestive process by influencing appetite and regulating insulin production.

The GI tract is the largest endocrine organ and hormones (e.g. cholecystokinin (CCK), glucagon-like peptide-1 (GLP-1)) within the GI tract act on the central nervous system to regulate appetite and glycemia post-meal consumption (Côté, Zadeh-Tahmasebi, Rasmussen, Duca, & Lam, 2014). The T1R2 and T1R3 ‘sweet’ receptors are present on enteroendocrine cells, which are homologous to taste receptor cells on the tongue (Spreckley & Murphy, 2015). T1R2 and T1R3 ‘sweet’ receptor cells in the intestine secrete hormones called incretins, which in turn stimulate insulin secretion (Jang et al., 2007). As research continues into this area, it becomes more apparent how the presence of such receptors is vital for normal physiological function (Figure 2.6). As an example, the presence of ‘bitter’ receptors are thought to be protective in the GI tract, as the presence of bitter chemicals (i.e. poison) in the large intestine evokes secretion of anions triggering the entrance of water, which leads to diarrhoea removing the toxins

from the body (Kaji, Karaki, Fukami, Terasaki, & Kuwahara, 2009).

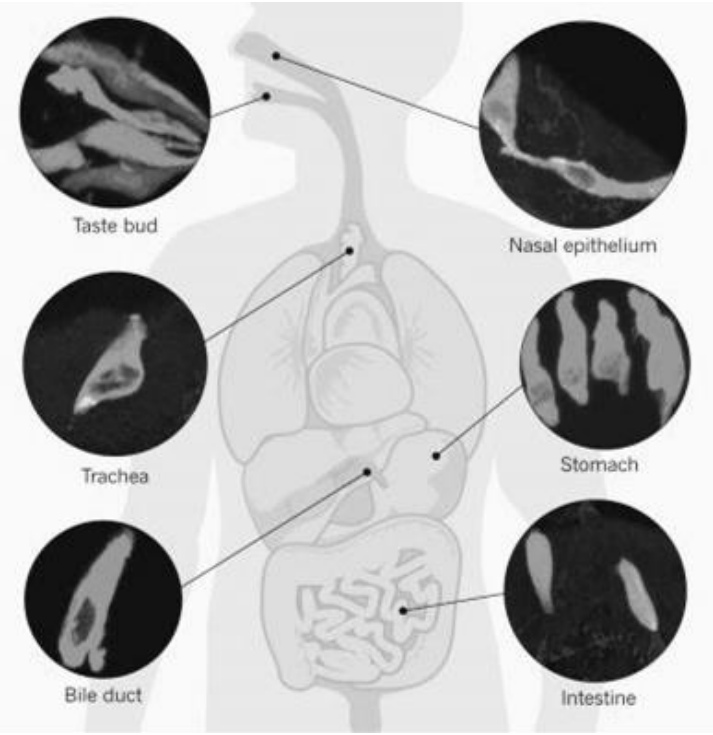

Figure 2.6. Image of the receptors found in the digestive tract that are nutrient sensors homologous to receptors (GPCRs) found in the oral cavity, nasal epithelium and trachea (Trivedi, 2012b). Image used with permission from Springer Nature.

2.2.5.2. Signalling and interactions of the enteroendocrine system

Information regarding incoming nutrients is conveyed by hormones that originate from epithelial cells or bile acids, together with neural signals (Monteiro & Batterham, 2017). Key hormones that are secreted from enteroendocrine cells, which are critical in appetite and food intake control include GLP-1, Peptide YY (PYY), gastric leptin, somatostatin, and CCK, as well as others (Bauer, Hamr, & Duca, 2016). GLP-1 levels increase when nutrients enter the proximal GI tract, leading to a biphasic response of a rapid increase of GLP-1, followed by another peak after 60 - 90 minutes (Monteiro & Batterham, 2017). PYY is co-secreted with GLP-1, and with other peptides such as CCK, secretin, glucose-dependent insulinotropic polypeptide (GIP), and neurotensin (NT). In response to nutrient ingestion, PYY levels increase rapidly and remain elevated

for several hours, with energy content and macronutrient composition impacting upon secretion. In contrast, circulating ghrelin increases before meals and falls rapidly after nutrient ingestion in proportion to the energy intake consumed (Hopkins & Blundell, 2016). Ghrelin, GLP-1, GIP, and CCK are all associated with gastrointestinal taste receptors that may play a functional role in detecting luminal nutrients which affect glucose and energy homeostasis (Janssen et al., 2011).

2.2.5.3. Insulin, leptin and appetite regulation

The early insulin response to meal ingestion is important for the regulation of subsequent glucose tolerance as post-absorptive nutrient status is in part managed by insulin levels (Blundell et al., 2010). It has been suggested that a blunted pre-absorptive insulin response is associated with obesity, which can exacerbate already dysregulated metabolic pathways (Hopkins & Blundell, 2016). Interestingly, the sweet taste receptor is expressed in β-cells, and activation of this receptor further acts to induce insulin secretion (Nakagawa et al., 2009). Chronic, acquired diseases such as type 2 diabetes (T2D) occur when normal physiologic control goes awry, so it is crucial that blood glucose homeostasis and metabolite levels are maintained within physiological parameters (Kotas & Medzhitov, 2015). Therefore, the interaction of the sweet taste receptor with subsequent insulin secretion in β-cells may play a critical role in the long-term regulation of glucose homeostasis and metabolic health.

Insulin increases body fat mass (Benoit, Clegg, Seeley, & Woods, 2004). However, insulin stimulates the production and secretion of leptin, the satiety hormone, that acts centrally to reduce food intake and increase energy expenditure (Amitani, Asakawa, Amitani, & Inui, 2013). Leptin then suppresses insulin secretion by both central and direct actions on the pancreas (Kieffer & Habener, 2000). Leptin is predominantly produced by adipose tissue and enterocytes in the small intestine. As a consequence, plasma levels of leptin are directly proportional to body fat mass (Seufert, 2004). The most significant roles of leptin include regulation of energy homeostasis, neuroendocrine function, energy metabolism and energy expenditure (Covey et al., 2006). Leptin activates a complex neural circuit in the hypothalamus including

anorexigenic (appetite-diminishing) and orexigenic (appetite-stimulating) neuropeptides to control food intake (Amitani et al., 2013). Outside of the hypothalamus, leptin interacts with the mesolimbic dopamine system, which is involved in motivation for and reward of feeding, and the nucleus of the solitary tract of the brainstem to contribute to satiety (Besnard, 2016).

In a healthy physiological state, an increase of adiposity and therefore plasma leptin, results in decreasing insulin production and reducing fat mass (Amitani et al., 2013). However, in the obese state, dysregulation of the insulin-leptin feedback system occurs, characterised by hyperinsulinemia and hyperleptinemia, and endocrine desensitisation (Owei, Umekwe, Provo, Wan, & Dagogo-Jack, 2017; Tchernof & Després, 2013). Leptin also aids to control sugar reward via the central nervous system by acting on glucose-sensing neural circuitry and thus maintains long-term homeostatic control of adipose tissue mass (Kubasova, Burdakov, & Domingos, 2015). Gastric leptin regulates food intake in the short-term and is rapidly secreted in response to both food and peptide hormones, such as CCK and insulin (Monteiro & Batterham, 2017). Further, leptin has been shown to modulate sweet taste sensitivities in mice (Kawai, Sugimoto, Nakashima, Miura, & Ninomiya, 2000). Sweet taste receptors are expressed in taste buds but also in enteroendocrine cells acting as sensors for sugar (Nakagawa et al., 2009).

2.2.5.4. Blood glucose regulation

Blood glucose regulation involves maintaining blood glucose at a constant level. Insulin and glucagon act to maintain an optimal range of blood glucose, where insulin is released when blood glucose levels are elevated, and glucagon is released when there are low levels (Jones, Tan, & Bloom, 2012). However, with obesity, insulin resistance can occur where cells in the muscles, liver and in adipose tissue resist the hormone insulin, resulting in elevated circulating blood glucose. High blood glucose levels over a prolonged period are associated with serious health complications, including T2D (Jones et al., 2012).

Taste receptors are important in the regulation of food intake and nutrient assimilation and therefore may be intimately linked with blood glucose regulation and endocrine responses (Dotson et al., 2008). Differences in sweet and bitter taste receptor efficacy have been hypothesised to influence glucose homeostasis (Dotson et al., 2008). This efficacy or sensitivity to nutrients may be homologous across both oral and gastrointestinal receptors. One study showed a positive correlation between taste responsiveness to sucrose and the number of metabolic disturbances in female adolescents (Pasquet, Laure Frelut, Simmen, Marcel Hladik, & Monneuse, 2007). Additionally, a range of studies has investigated the role of intestinal sweet taste receptors, where low energy sweeteners have been shown to impact on glucose absorption, GLP-1 release and GIP release (Renwick & Molinary, 2010). The role of the sweet taste and bitter taste receptors in regulating metabolic processes indicates that it is a potential target for novel therapeutic treatments of obesity and metabolic dysfunctions (Laffitte, Neiers, & Briand, 2014).

2.2.5.5. Obesity, metabolic health and the current public health setting

Obesity is a global health issue (Ng et al., 2014). The current obesogenic environment suggests that across many people living in westernised countries, energy homeostasis is impaired (Berthoud, 2012). For example, the prevalence in NZ has increased over the last decade, with 1.2 million adults (32% of the population) currently being obese (Ministry of Health, 2016) and NZ ranking as the third most obese country in the Organization for Economic Co-operation and Development (OECD) (Ng et al., 2014). In NZ, Pacific (69%) and Māori (50%) are disproportionately affected by obesity compared with the general population in NZ (32%), and rates are highest in the most deprived areas (Ministry of Health., 2017). Major influences on deprivation and socioeconomic status include employment, income, qualifications, home ownership, family structure and housing (Atkinson, Salmond, & Crampton, 2014). In turn, deprived socioeconomic status is associated with weight gain and obesity risk due to the increased intake of cheap, highly palatable and processed food options (Swinburn et al., 2013), lower adherence to current dietary recommendations (Schwartz et al., 2017) and unhealthy lifestyle choices and habits (Duca & Lam, 2014).

Interventions and government led strategies to halt the obesity epidemic have been unsuccessful to date (Dubé, 2010; Ng et al., 2014). Obesity increases the risk of developing a range of diseases including cardiovascular disease (CVD), T2D and cancer (Heymsfield & Wadden, 2017; Huxley, Mendis, Zheleznyakov, Reddy, & Chan, 2010). The underlying causes of obesity are complex; however, excess energy intake is increased by several factors including cognition, habit-forming, brain reward, nutrient sensing in the gut and predictive control (see Figure 2.3). Obesity is further associated with a mild inflammatory state, which is impacted by elevated circulating leptin (Bauer et al., 2016; Monteiro & Batterham, 2017). Insulin and leptin resistance can both occur as a result of obesity, resulting in the impaired regulation of appetite and metabolism. Other known factors contributing to obesity include imbalances in pathways of glucose and lipid metabolism that occur as a consequence of variations in quantity and quality of the diet, sedentary lifestyle and genetic predisposition (Phillips, 2017; Tchernof & Després, 2013). Obesity arises because of how the body regulates energy intake, energy expenditure and energy storage, and reflects a state of positive energy balance.

Current public health research in obesity aims at generating effective food and nutrition policies (Swinburn et al., 2013), increasing the availability of and access to healthier food choices (Ni Mhurchu et al., 2013) and community-based interventions (Rush et al., 2014). It has become clear that obesity is due to a complex interplay between the central nervous system and metabolic processes that over time contribute to a large range of comorbidities and deterioration in health (Dube et al., 2010). Progress in obesity management will require a greater understanding of the biological, behavioural, and environmental factors associated with lifestyle changes including both diet and taste perception (Hill, 2006; McGill, 2014; N. Thompson et al., 2014). Taste perception, eating behaviour and dietary intake may be important biological factors that require further understanding. Given that both sweet taste and fat have a powerful hedonic appeal, associated preferences for sweet and fatty foods are important contributors to increases in body weight and metabolic disease risk (Laffitte et al., 2014; Martínez-Ruiz, López-Díaz, Wall-Medrano, Jiménez-Castro, & Angulo, 2014). Recent studies have indicated that overweight and obese groups may experience a diminished taste perception compared to normal weight groups (Dando, 2015; Kaufman, Choo, Koh, &

Dando, 2018; Proserpio et al., 2016). This further suggests that overweight and obese groups may have altered taste preferences based on the concentration level of taste (Kure Liu et al., 2019; Proserpio et al., 2016). Taste perception, dietary intake and body composition associations will be reviewed in more detail in section 5.3.

2.3. POTENTIAL INFLUENCES ON TASTE PERCEPTION

Taste perception can be impacted by a range of other biological factors. These important influences on sensory perception are genetics, age, gender, and body weight. Each of these will be discussed as follows.

2.3.1. GENETICS

Humans show substantial differences in taste perception and in part this is due to genetics (Reed & Xia, 2015). For example, differences observed in the CD36 fatty acid taste receptor genotype may lead to weight gain over time (Keast & Costanzo, 2015; Pepino, Kuda, Samovski, & Abumrad, 2014; Sayed et al., 2015). However, this type of association brings up the question '*what came first, the chicken or the egg*' scenario for fat intake; is it an initial impairment in fatty acid taste sensitivity that may lead to obesity, or do individuals with obesity develop this impaired chemoreception overtime? Does this impaired chemoreception lead to overeating and therefore weight gain? In support of this, a recent investigation has found a significantly higher CD36-A allele frequency in young children with obesity when compared to lean children (Sayed et al., 2015). Another study found that acceptance of added fats and oils and perceived creaminess varied between individuals with different CD36 polymorphisms (Keller et al., 2012). Based on molecular evidence, GPCRs and CD36 function in the presence of fatty acids to trigger peptide secretions in the oral cavity and in the gut (see Table 2.2).

Table 2.2. Peptide secretions triggered by fatty acids in both taste cells and enteroendocrine cells and the corresponding fatty acid receptors (Pepino et al., 2014). Table used with permission from Annual Reviews, Inc.

	Peptide	***Primary site***	***Fat ligand***	***FA receptor***
Taste cells	GLP-1	Circumvallate papillae	LCFA	GPR120
	Serotonin	Circumvallate papillae	LCFA	CD36
EE cells				

K cells	GIP	Duodenum/Jejunum	LCFA	GPCR40, 119, 120
S cells	Secretin	Duodenum/Jejunum	LCFA	CD36
I cells	CCK	Duodenum/Jejunum	LCFA	CD36, GPCR40
L cells	PYY	Ileum/Colon	ShFA	GPCR41, 43
L cells	GLP-1	Ileum/Colon	LCFA, OEA	GPCR40, 119, 120

CCK, cholecystokinin; CD36, cluster of differentiation 36; EE, enteroendocrine; FA, fatty acid; GIP, glucose insulinotropic peptide; GLP, glucagon-like peptide; GPCR, G-protein coupled receptor; LCFA, long-chain fatty acid; OEA, oleoylethanolamide; PYY, peptide YY.

2.3.2. AGE (GESTATION, CHILDHOOD, ADULT TO AGING)

The association of early life exposure and taste preferences later in life are still being established. During development, it has been shown that periods of heightened sensitivity to environmental exposure, such as sensory experiences, can strongly influence food preferences (Mennella, 2014). Longitudinal studies suggest that some food habits can be tracked from infancy into adolescence, which is particularly important for fruit and vegetable intake (Lioret, McNaughton, Spence, Crawford, & Campbell, 2013; Mikkilä, Räsänen, Raitakari, Pietinen, & Viikari, 2005; Skinner, Carruth, Bounds, Ziegler, & Reidy, 2002). It is the hedonic liking of specific foods (e.g. broccoli, kale or other bitter-tasting cruciferous vegetables) that may be most influenced by this early exposure. Theoretically, if such 'bitter' taste experiences are acceptable in early life, then the daily intake of healthy vegetables is more likely to occur throughout adult life (Mennella, 2014). Genetic influences interact with this behaviour as it has been found that non-taster PROP children (i.e. less sensitive to bitter taste) consume more vegetables in a free-choice setting (Bell & Tepper, 2006) and those who are sensitive to bitter taste consume fewer vegetables (Keller, Steinmann, Nurse, & Tepper, 2002).

Taste perception has been found to decline with age (Methven, Allen, Withers, & Gosney, 2012) a trend which is seen across all primary tastes. Taste acuity has also been shown to decline with the onset of some diseases such as chronic obstructive pulmonary

disease (Wardwell, Chapman-Novakofski, & Brewer, 2009).

2.3.3. GENDER

There is conflicting evidence as to whether or not there is any difference in sensitivity to primary taste by gender (Chang, Chung, Kim, Chung, & Kho, 2006; Gudziol & Hummel, 2007; James, Laing, & Oram, 1997; Landis et al., 2009; Pingel, Ostwald, Pau, Hummel, & Just, 2010; Wardwell et al., 2009). The studies which have found women to be more sensitive to taste may have observed differences due to hormonal differences between genders (Kuga, Ikeda, & Suzuki, 1999; McNeil, Cameron, Finlayson, Blundell, & Doucet, 2013), behavioural differences (e.g. dieting, restrictive behaviours), differences in neural or endocrine systems and potentially variation in subjective scoring, decision-making and psychology (Doty, 1978; Haase, Green, & Murphy, 2011; Running, Mattes, & Tucker, 2013).

2.3.4. BODY WEIGHT AND OBESITY

A number of studies have raised the question of whether there is a link between increased adiposity and taste perception, or sensitivity to taste (Bartoshuk, Duffy, Hayes, Moskowitz, & Snyder, 2006; Donaldson, Bennett, Baic, & Melichar, 2009; Pepino, Finkbeiner, Beauchamp, & Mennella, 2010; Salbe, DelParigi, Pratley, Drewnowski, & Tataranni, 2004; Sartor et al., 2011). However, testing methods used across these studies are inconsistent (Cox, Hendrie, & Carty, 2015; Low, Lacy, McBride, & Keast, 2017).

Interestingly, the most successful therapeutic treatment for morbid obesity to date has been bariatric surgery (Ashrafian & le Roux, 2009). Bariatric surgery modulates gut and adipose hormones. It has been found that bypass and foregut exclusion operations (i.e. Roux-en-Y gastric bypass) produce stronger gut hormone responses than banding or restrictive procedures (Buchwald et al., 2009). So, what effect does bariatric surgery have on taste perception? Initially, it has been suggested that taste sensation for those who have undergone bariatric surgery increases, with sweet and fatty tastes becoming

less pleasant at higher concentrations. Gastric bypass is therefore thought to reverse taste hedonics (Miras & Le Roux, 2014), for example, the palatability of sweetness as measured by taste testing has been shown to shift from pleasant to unpleasant (Nance, Eagon, Klein, & Pepino, 2017). Roux-en-Y gastric bypass has been found to alter brain activity in areas involved in reward expectation and taste processing upon anticipation of fatty food in rats (Thanos et al., 2015). This information is of great interest as the cerebellar regions related to altered metabolism following gastric bypass may help to highlight novel therapeutic targets for prevention of weight gain.

Establishment of taste perception alongside the measurements of the regulators of appetite and fat storage may elucidate important mechanisms and taste-appetite associations. For example, the adipocyte hormone leptin aids to control sugar reward via the central nervous system by acting on glucose-sensing neural circuitry and may have a distinct influence on sweet taste perception (Kubasova et al., 2015). Compelling work has shown that circulating leptin concentrations correspond with diurnal variation in sweet taste recognition in humans (Nakamura et al., 2008). These associations are complex, as both TAS1R and TAS2R taste receptors detect both sweet and bitter-tasting stimuli (Dotson et al., 2008). The determination of distinct hormonal influences on each primary taste response may reveal important associations between taste perception, body weight and body composition.

2.4. SENSORY PERCEPTION MEASUREMENTS

2.4.1. TASTE METHODOLOGY

Taste perception can be measured by a wide range of techniques. A commonly utilised taste perception measurement is that of 'sensitivity', or the absolute minimum concentration of a stimulus that can be perceived (i.e. detection threshold). An additional measurement that is commonly used is the minimum concentration of a stimulus that can be recognised, or 'named' (i.e. recognition threshold). The perceived intensity and hedonic ratings of suprathreshold concentrations are rated using a scale, general linear magnitude scale (gLMS), or labelled affective magnitude (LAM) scale which have been increasingly used in recent studies (Noel, Sugrue, & Dando, 2017). Other methods include, but are not limited to: ranking tasks, paired comparison taste tests, and discrimination tasks (Ettinger, Duizer, & Caldwell, 2012; Haryono, Sprajcer, & Keast, 2014; Kim, Prescott, & Kim, 2014; Newman, Torres, Bolhuis, & Keast, 2016). These taste testing methodologies are further described below.

2.4.1.1. Taste threshold and taste recognition methodology

Detection thresholds are evaluated using specific testing methods, the two most commonly used methods are 1. staircase method and 2. ascending forced choice procedure (ASTM, 2011; Tucker & Mattes, 2013). Each method has its own advantages; however, the ascending forced choice method has the distinct benefit of minimising desensitisation, as it starts from the lowest concentration and increases until the taste is detected. Furthermore, the ascending forced choice method avoids bias as it doesn't start from a higher concentration or predetermined midpoint (Haryono et al., 2014), and it has less probability of random chance influencing results (3.7%), as opposed to the staircase method (11.1%). Recognition threshold is typically measured from an ascending order, with the detection threshold concentration used as the starting point (Low et al., 2017). Participants continue to trial taste samples until they reach the point at which they can correctly identify the taste quality (i.e. "sour") two or three

times consecutively (Pasquet, Monneuse, Simmen, Marez, & Hladik, 2006).

The ability to recognise a taste (i.e. the ability to describe a taste as ‘sweet’ or ‘salty’) is different from the ability to detect a taste (i.e. noticing the presence of the agonist, without being able to define what it is). For example, the test re-test of salty and sour taste mechanisms were investigated by Wise and Breslin (2013) who suggested that recognition and detection abilities are likely to be controlled by different physiological processes (Wise & Breslin, 2013). More recent studies have found clear differences between detection thresholds and recognition thresholds of taste, as well as suprathreshold intensity ratings of taste (Giguère et al., 2016; Jayasinghe et al., 2017; Low, Lacy, McBride, & Keast, 2016). It is critical that these descriptions of taste perception are clearly distinguished as there appears to be a complex relationship between chemical concentration, detection and suprathreshold intensity (Keast & Roper, 2007).

2.4.1.2. Suprathreshold taste ratings

The suprathreshold intensity assessment of taste uses higher concentration levels than that of the recognition levels of taste. Usually, only a small range of taste concentrations are selected that have been previously described as distinct concentration levels (i.e. ‘weak’, ‘moderate’ and ‘strong’) (Low et al., 2016). Each concentration of stimuli is then rated by participants, often using a validated scale; the most frequently used is the gLMS following a standard protocol (Bartoshuk, 2000). Measurements using these scales (i.e. intensity) are subjective measurements, they can be influenced by opinions or feelings (Hardikar, Höchenberger, Villringer, & Ohla, 2017).

2.4.1.3. Other taste perception measurements

A range of other objective taste perception measurements can be utilised. Previous studies have used ranking tasks, where participants rank four samples from the highest through to the lowest concentration of taste (i.e. 1 = highest concentration of stimulus, 4

= lowest concentration of stimulus). This methodology has been successfully used in previous studies (Haryono et al., 2014; Stewart et al., 2010; Stewart, Newman, & Keast, 2011). Taste sensitivity scores derived from ranking tasks have shown similar results to detection threshold measurements (Stewart et al., 2010). Ranking tasks significantly reduce participant fatigue in comparison to threshold testing.

Impregnated taste strips are commonly used in studies as a measure of taste recognition involving large numbers of participants (Fernandez-Garcia et al., 2017), as well as in studies involving children (Overberg, Hummel, Krude, & Wiegand, 2012; Tucker, Nuessle, Garneau, Smutzer, & Mattes, 2015). These studies can be carried out in off-site locations (i.e. schools). Paired comparison tests, or discrimination tasks, can be used to determine if differences can be perceived between two different samples (i.e. apple juice containing either 4g or 10g of sucrose, presented side by side, where the participant determines which has the highest concentration of sugar) (Alexy et al., 2011). However, both impregnated taste strips and discrimination tasks may only provide broad measurements or single concentration measurements which may be difficult to compare with dietary intake data. However, the distinct advantage of these tasks is the ‘right or wrong’ outcome, which provides non-subjective results with minimal participant fatigue.

2.4.1.4. Fatty acid taste threshold methodology

Unlike the other primary tastes, fat is the most difficult to test because fat has textural, visual and solubility issues when creating test samples. Milk or whey protein-based tasting samples are regarded as a more stable emulsion vehicle, and a number of studies have utilised this milk or protein-based stimuli in lieu of distilled water (Galindo et al., 2012; Keast, Azzopardi, Newman, & Haryono, 2014; Panek-Scarborough, Dewey, & Temple, 2012; Stewart et al., 2010; Stewart & Keast, 2012). Fatty acids can be kept longer and retain a more homogenous mixture in milk than a water-based solution. It has been recommended that non-fat, ultra-high temperature (UHT) processed milk is a suitable base for oral fatty acid threshold assessment (Haryono et al., 2014). In order to evaluate the presence of fatty acid, and the fatty acid taste sensitivity that an individual may have, most studies work on determining the lowest concentration of a fatty acid

which can be distinguished from a background sample (e.g. water or ice-cream) lacking fatty acid (Running et al., 2015).

2.4.2. OLFACTORY METHODOLOGY

When measuring the ability to smell a substance, it is important to note that the interaction of odour volatiles is complex. One of the most common procedures for measuring the ability to sense smell is that of the Sniffin' Sticks test (Burghart Messtechnik GmbH, Germany). Sniffin' Sticks is a method for testing orthonasal stimulation (Hummel, Sekinger, Wolf, Pauli, & Kobal, 1997), and retronasal stimulation (the olfactory stimulation sensed when food is in the mouth) (Heilmann, Strehle, Rosenheim, Damm, & Hummel, 2002). A range of studies using Sniffin' Sticks has utilised a total score (TDI), which combines odour threshold, odour discrimination and odour identification ability (Haehner et al., 2009; Hummel, Kobal, Gudziol, & Mackay-Sim, 2007; Hummel et al., 1997). Odour threshold is measured with a series of sticks containing *n*-butanol or phenyl ethyl alcohol alongside control sticks, and when consecutive correct identifications are made, this is considered to be the detection threshold (lowest concentration at which the odour is detected) (Denzer et al., 2014; Hummel et al., 1997). Discrimination is where three odours are presented, and the individual is asked to identify the stick with the different smell. Identification is assessed with common odours (e.g. peppermint, banana, lemon) and from a multiple-choice question, participants are asked to identify the odour (Freiherr et al., 2012). Individually these measurements can give an indication as to whether a person has "normal" olfactory perception (normosmic), has reduced sensitivity to smell (hyposmic) or no sense of smell (anosmic) (Haxel, Fuchs, Fruth, Mann, & Lippert, 2011; Kobal et al., 2000).

Threshold tests have been designed for measuring olfactory sensitivity with targeted food odours, such as dark chocolate (Stafford & Whittle, 2015). Another useful method for measuring odour recognition is the 40-item University of Pennsylvania Smell Identification Test (UPSIT, Sensonics, Inc. Haddon Heights, NJ, USA) as well as the short version of this - the 8-item test pocket smell test (PST) (Doty, Shaman, & Dann, 1984). These tests have been validated and conducting the test is simple due to the

stimuli being a ‘scratch and sniff’ card (Fornazieri et al., 2013; Haugen et al., 2016; Rawal, Hoffman, Honda, Huedo-Medina, & Duffy, 2015). Another 40-item identification test is one that uses an olfactometer (OLFACT-ID™, Osmic Enterprises, Inc.) which provides better control of aroma stimuli with computerised regulation of aroma output and timing (Rawal et al., 2015).

One of the major challenges for olfactory measurement is ‘adaptation’, which is a phenomenon where intense odours become less intense over time due to physiological desensitisation (Stuck, Fadel, Hummel, & Sommer, 2014). Olfactory tests which test a range of odours at different concentrations (i.e. staircase method) may require repeated testing in order to overcome this limitation at different study visits. Few studies have investigated the test re-test reliability of smell (Rawal et al., 2015).

2.4.3. MOUTHFEEL SENSATION METHODOLOGY

Mouthfeel provides food with desirable qualities that are crucial in product acceptance as well as complimenting taste and flavour attributes (Stokes, Boehm, & Baier, 2013). Textural qualities such as smoothness, fattiness, and creaminess are likely to vary among individuals, however, the measurement of these feelings is challenging.

Measurement of sensation is often subjective and can vary widely between individuals. To overcome this, trained panels are often used who are taught exact mouthfeel or textural sensations. Alternatively, researchers can use visual analogue scales (VAS), or a categorical scale, in an attempt to quantify subjective feelings from untrained panellists (Chaput, Gilbert, Gregersen, Pedersen, & Sjödin, 2010).

2.4.4. SENSORY PERCEPTION SUMMARY

The ‘obesity epidemic’ cannot be solely contributed to hereditary factors, as it has only been of acute concern in recent years (Swinburn et al., 2013). Therefore, multiple influences explain the ‘obesity epidemic’ beyond individual factors (Story et al., 2008; Vandenbroeck, Goossens, & Clemens, 2007). However, individual factors and associated biological mechanisms may be the key to specific targets for therapeutic and

long-term intervention strategies to reduce population obesity. Associations between taste perception, dietary intake, eating behaviour and obesity are reviewed in detail below.

2.5. DIETARY INTAKE AND EATING BEHAVIOUR

2.5.1. DIETARY INTAKE

Individual dietary intake can be influenced by a range of factors (see Section 1.1.), impacting on total energy intake or the intake of individual macronutrients. Whole diets are complex, and the interaction of multiple tastes which would occur during the consumption of a meal may act to enhance or inhibit sensory perception on the oral surface (Newby & Tucker, 2004). Therefore, measurement of the whole diet alongside taste perception is important to advance our understanding of these associations. One example is that of 'sensory-specific satiety', which is the decline in liking for a consumed item relative to other non-consumed items (Rolls, 2015), which is an explanation for increased intake (altered liking) with variety. Studies have shown that when exposed to a wide variety of foods (i.e. buffet-style meal options) the variety will induce increased consumption of up to 44% in comparison with an item restricted buffet (Raynor & Epstein, 2001).

2.5.1.1. Assessing dietary intake

There is a wide range of dietary intake methodologies which are used in sensory perception and taste perception studies. Weighed and estimated food records are the 'gold standard' method for quantifying nutrient intake (Beck & Heath, 2013). However, in large cohort studies, the use of food records may not be feasible. Additional tools for the measurement of dietary data include food frequency questionnaires. Questionnaires of this nature are faster, often computer-based and require the recall of 'usual' foods consumed over a month or over a year (Carroll et al., 2012). The advantage to this is that foods consumed less frequently (i.e. once a month) will be captured by the food frequency questionnaire, whereas this information would be lost in a food record, or possibly over-represented (Beck & Heath, 2013). Other dietary intake methods include

24-hour recalls and diet histories. These interview-style methods involve a higher researcher burden but require less participant literacy and reduce participant-burden. The limitations of 24-hr recalls are the increased likelihood of underreporting, single day reporting may not be representative of usual intake, and poorer estimation of serving sizes and recipes (F. E. Thompson & Subar, 2013).

To date, there are no studies that have drawn an overall conclusion as to the best dietary intake methodology to use together with taste perception data. A review of fatty acid, bitter taste and sweet taste studies show that meaningful diet-taste associations have been found using food records (see Table 2.3, Table 2.4 and Table 2.5). However, in a recent systematic review, there was no recommendation made for the best dietary assessment method, due to the heterogeneity of study outcomes (Tan & Tucker, 2019). Interestingly, the authors concluded that day-to-day variability in dietary intake could obscure immediate or acute diet-taste relationships (Tan & Tucker, 2019). Therefore, dietary information gathered over multiple days (including both weekdays and weekend days) may be an important consideration for taste perception researchers.

2.5.2. EATING BEHAVIOUR

Eating behaviour is a term that encompasses food choice and motives, dieting, eating practices, and eating-related problems (Story et al., 2008). Eating practices and habits such as dietary restraint are suggested to play an important role in the development of eating disorders and obesity (Blundell & Gillett, 2001). Other types of behaviour such as the loss of control over intake and the tendency to overeat (i.e. disinhibition) are also important contributors to weight gain and dietary intake (Stunkard & Messick, 1985). Research has demonstrated that those who have increased restraint have a lower body mass index (BMI), due to being more conscious of their dietary intake (Moor, Scott, & McIntosh, 2012).

Food preferences can be influenced by cognitive responses to food, such as emotionally driven eating. This form of eating can be increased by external factors, however, those who are ‘sensitive to reward’ as a personality trait may learn to continue eating in the

absence of hunger (Davis et al., 2007). This trait has been positively correlated with a preference for sweet and fatty food, which is thought to be a physiological and psychological motivation. In part, this could be due to the endogenous opioid release in the brain when palatable foods are consumed (Drewnowski & Bellisle, 2007). Furthermore, it is thought that high energy diets interfere with the hippocampus, impairing the ability to retrieve the memory of the food-related cue, leading to increased food intake.

There is little information on how sensory perception relates to eating behaviour or related characteristics. Mindful eating has been recognised as a new way in which to consciously heighten our recognition of the amount of food we eat (Moor et al., 2012; Wilson & Dillard, 2015). This process of eating with intention and attention acts to strongly enhance our perception and slow eating speed, as each sensory cue (e.g. taste, aroma, mouthfeel, appearance) becomes a stronger point of focus (Wilson & Dillard, 2015). The reason this may be very effective is that it is believed people with higher disinhibition are generally unaware of all the food decisions they make, which means food intake can occur with little conscious recognition. It is thought that these involuntary decisions can lead to weight gain (Wansink & Sobal, 2007) and that people often underestimate portions consumed. Recent studies have found that when eating speed is slow, total food and energy intake can be significantly reduced in normal-weight subjects when compared to faster eating (Shah et al., 2014). A functional magnetic resonance imaging (fMRI) study measured anticipated responses to the intake of a chocolate milkshake against a tasteless solution, and it was found that the brain response in participants with obesity was different to the response in normal-weight participants, where greater activation of the insular cortex appears to play a role in anticipatory food reward (Stice, Spoor, Bohon, Veldhuizen, & Small, 2008).

2.5.2.1. Assessing eating behaviour

Eating behaviour can be broadly described in terms of three dimensions of human eating behaviour: (1) cognitive dietary restraint (restraint), (2) disinhibition of control

(disinhibition) and (3) susceptibility to hunger (hunger) by calculating scores for these dimensions using the three-factor eating questionnaire (Stunkard & Messick, 1985). Subscales of eating behaviour can be deduced from the same questionnaire; these include flexible and rigid restraint; habitual, emotional and situational susceptibility to disinhibition and internal and external locus of hunger (Bond, McDowell, & Wilkinson, 2001; Westenhoefer, 1991; Westenhoefer, Stunkard, & Pudel, 1999). To score the questionnaire, each item is given either 0 or 1 point with possible scores ranging from '0-0-0' to '21-16-14' (Stunkard & Messick, 1985). High scores denote higher levels of restrained, disinhibited eating or predisposition to hunger.

A range of other validated eating behaviour and associated eating pattern questionnaires can be used to assess specific research questions. Examples of these questionnaires and specific diet-and eating-related assessments are: eating and weight patterns (QEWP), dutch eating behaviour questionnaire (DEBQ), eating attitudes test (EAT), food cravings inventory (FCI) and the binge eating scale (BES) (Bryant et al., 2014; Panek-Scarborough et al., 2012; Pepino & Mennella, 2014).

2.5.3. TASTE, DIETARY INTAKE, EATING BEHAVIOUR AND OBESITY

The prevalence of obesity and excess energy intake among westernised populations is theoretically exacerbated by the increased availability of unhealthy food (Roberto et al., 2015). Therefore, associations between taste sensitivity, dietary intake, eating behaviour and obesity are highly relevant. There is a need to better understand the preferences for specific tastes between people who are obese in order to develop strategies to reduce the prevalence of overweight and obesity (Martinez-Cordero, Malacara-Hernandez, & Martinez-Cordero, 2015).

Generally speaking, increased taste sensitivity to sweet and fatty acids is hypothesised to decrease energy intake (Cox et al., 2015). In contrast, increased taste sensitivity to bitter or sour taste is hypothesised to decrease intake of vegetables and fruit, due to their taste being less palatable to sensitive tasters (Keller & Adise, 2016; Mennella et al., 2016). It is thought that children who reject such tastes and continue to avoid vegetables

will reject these vegetables in later life; as a consequence, bitter taste sensitivity may indirectly influence their future health and wellbeing (Bell & Tepper, 2006). However, genetic factors have been shown to influence taste perception, such as single taste receptor gene variations (i.e. *hTAS2R38*) which leads to taste blindness to PTC and PROP in approximately 20 to 30 percent of individuals (Duffy & Bartoshuk, 2000; Sandell et al., 2014). Genetic differences in *hTAS2R38* have been associated with differences in vegetable and sweet food consumption (Sandell et al., 2014).

There has been little research on savoury taste in relation to dietary intake. However, it is of note that children who are obese eat significantly more savoury snacks than sweet snacks when compared to normal-weight children (Maffeis et al., 2008). This association may be related to the combined effects of salt and savoury taste, which when optimised, is appealing. In addition, monosodium glutamate (MSG) perception may be associated with weight gain, due to MSG enhancing food palatability and increasing overall food intake (Donaldson et al., 2009).

2.5.3.1. Sweet taste perception and dietary intake

The influence of taste and sensory cues on subsequent appetite is of interest, due to the long-term health consequences associated with overeating. Several studies have attempted to measure sensory sensitivity and subsequent food intake, or, measurement of typical food intake and the relationship this may have with taste sensitivity. Sweet taste is a universal trait, however the inter-individual variation in the liking of, and perceived level of sweetness intensity is worthy of further investigation. The level of sweetness in food and the optimal level of sweetness may further influence dietary intake (Sørensen, Møller, Flint, Martens, & Raben, 2003). A key question is whether individuals with obesity or at risk of weight gain have a preferred liking for sweet-tasting foods (Bartoshuk et al., 2006; Ettinger et al., 2012; Jayasinghe et al., 2017). If so, does this lead to increased energy intake and how does this affect the relative perception of sweet taste intensity over time? The ability to detect sweet taste could be improved with an energy-restricted diet (Umabiki et al., 2010), which in turn could strengthen the biological mechanism which triggers satiety.

In Table 3, a range of studies were found that showed positive associations between sweet taste hedonic liking and intake of carbohydrates and sugars (Garneau et al., 2018; Holt, Cobiac, Beaumont-Smith, Easton, & Best, 2000; Jayasinghe et al., 2017; Smith, Ludy, & Tucker, 2016; Turner-McGrievy, Tate, Moore, & Popkin, 2013). In contrast, there was only one study that found a relationship between detection or recognition thresholds of sweet taste, and dietary intake (Martinez-Cordero et al., 2015), and conversely there were a number of studies that found no relationship between these measures (Jayasinghe et al., 2017; Low et al., 2016; Smith et al., 2016). In a recent systematic review, sweet taste as a predictor of dietary intake was investigated (Tan & Tucker, 2019) and it was concluded that hedonic measurements of taste were more likely to be associated with dietary measures. In addition, analysing data from sweet likers separately from sweet dislikers derived from cluster analysis were shown to improve sweet taste and dietary intake relationships (Tan & Tucker, 2019).

Ratings of sweet taste with more than one concentration level of sweet stimulus, have resulted in the subsequent reporting of stronger taste-diet relationships (Garneau et al., 2018; Holt et al., 2000; Jayasinghe et al., 2017) than studies that drew conclusions from perception of taste at one concentration level (Cicerale, Riddell, & Keast, 2012; Leong, Forde, Tey, & Henry, 2018a). The gLMS scale rating method was used in a number of studies measuring sweet taste (i.e. intensity) and a major advantage of this approach is its suitability for comparisons across different population groups (Cicerale et al., 2012; Jayasinghe et al., 2017; Low et al., 2016). This review suggests that suprathreshold ratings of intensity and liking of sweet taste are useful measurements for determining taste-diet relationships (Table 2.3).

2.5.3.2. Sweet taste perception and eating behaviour

There were few sweet taste and dietary intake studies that have measured eating behaviour (i.e. using the TFEQ) (Table 2.3). One study that measured restraint from the three-factor eating questionnaire used this to characterise the normal study population but did not compare the restraint measurement to taste sensitivity (Drewnowski,

Henderson, Levine, & Hann, 1999). Another study measured food behaviour, however, there were no significant associations between food behaviour scores and sweet taste sensitivity (Cicerale et al., 2012).

2.5.3.3. Sweet taste perception and body composition

When investigating sweet taste perception, an interesting inquiry is whether body weight *per se* may influence taste sensitivity. Several studies have investigated this relationship (Table 2.3). There are studies that have found that obese individuals exhibit higher thresholds for sweet tastants than their normal-weight counterparts (Park et al., 2015; Skrandies & Zschieschang, 2015). However, other studies investigating basic tastes (i.e. sweet) have found no association between any taste response and BMI (Martinez-Cordero et al., 2015). Overall, a direct relationship is not yet established and the variation in evidence for this association is at least in part due to a lack of robust or comparable sensory methodology (Cox et al., 2015; Tucker et al., 2017) (Table 3).

One study found evidence for an association between sweet taste and body weight, where diurnal changes in sweet taste recognition were associated with circulating plasma leptin (Nakamura et al., 2008). However, a follow-up study in obese participants did not find the same association (Sanematsu, Nakamura, Nomura, Shigemura, & Ninomiya, 2018). It remains unknown as to whether changes in nutrient sensing, hormonal signalling, or inflammation can impair our sense of sweet taste or taste recognition (Kaufman et al., 2018).

Table 2.3. Studies which have investigated sweet taste sensitivity and dietary intake, eating behaviour and body composition.

Study	Population, participants	Sweet taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Cicerale, Riddell, & Keast, 2012 Cross-sectional observational	Australia. N= 130 adults Age mean±SD 21±4y.	gLMS rated intensity of sucrose at one concentration.	2 x 24hr FR. food & diet questionnaire.	Food behaviours questionnaire.	EI, macronutrients, micronutrients.	BMI	No associations between dietary activities, food beliefs, & sweet taste intensity. Weak association with sodium intake (P<0.05).	None.
Drewnowski, Henderson, Levine, & Hann, 1999 Cross-sectional observational	USA. N= 159 women Age mean(SEM) 27 (0.7) y	Suprathreshold hedonic liking on a 9-point scale. Sucrose in water at 5 concentration levels.	3-day food records (N=87) & food preference checklist.	Restraint scale & eating attitudes test (EAT).	EI, macronutrients, fibre, β-carotene.	BMI	Higher preference for sucrose in water was associated with increased self-reported preference for sweet desserts (P<0.05).	None.
Garneau, et al, 2018 Cross-sectional observational	USA. N=418 adults Age mean±SD 41.8±16.5y	Suprathreshold intensity & liking on VAS. Sucrose in water at 5 concentrations.	Beverage food frequency questionnaire (BEVQ-15).	n/a	EI from beverages. (e.g. coffee, juice).	BMI, BF%	Sweet likers (group) had a greater energy intake from sweetened juice & sweetened tea (P<0.05) compared to the sweet neutral group.	**Yes**, ↑ hedonic liking of sucrose, ↑ sweetened beverage intake, & energy intake.
Han, Keast, & Roura, 2017 Randomised cross-over design experiment	Australia. N=30 adults Age mean±SD 27.1±1.0y	Sensitivity to the 9mM concentration of sucrose in water (triangle test).	Taste & rating of liking of sweet soup. Energy intake from the soup. Food choices.	TFEQ	EI (kJ), macronutrient intake from buffet meal.	BMI	High sensitivity to sweet (group) consumed fewer CHOs (% energy) & more non-sweet foods. No differences in eating behaviour.	**Yes,** ↑ sensitivity to sweet taste, ↓ in CHO intake (% energy) from the soup.
Holt, Cobiac, Beaumont-Smith, Easton, & Best, 2000 Cross-sectional observational	Australia. N=69 Australian & 63 Malaysian adults. Age mean±SD 22.2±2.8y	Suprathreshold intensity & liking rated on VAS. Sucrose in water at 5 concentrations. Ratings of orange juice, custard, biscuit.	FFQ (78 foods).	n/a	Sugar intake (g) as refined & total.	BMI	No association between intensity & sugar intake. Refined sugar intake is higher in sweet likers. Preferred sucrose level was positively associated with sweet food consumption & sugars intake (P<0.05).	**Yes,** ↑ liking to sweet taste, ↑ sugar & sweet food.

Study	Population, participants	Sweet taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Jayasinghe et al., 2017 Cross-sectional observational	New Zealand. N=44 women. Age mean±SD 28±6y	Detection, recognition thresholds & suprathreshold intensity gLMS glucose in water at 4 concentrations.	4-day weighed FR. Sweet food FFQ & sweet beverage liking questionnaire (visual analog scales).	n/a	EI (kJ) & macronutrient intakes. Sugars consumption (e.g. fructose, lactose, maltose).	BMI, BF%	No association between thresholds of glucose & dietary intake. Both intensity & liking gLMS of sweet taste significantly correlated with EI & CHO intake.	**Yes,** ↑ liking to sweet taste (at concentrations above 500 mM), ↑ sugar & sweet food & ↓ intensity to sweet taste (at higher concentrations), ↑ energy & CHO intake).
Leong et al., 2018 Cross-sectional observational	Singapore. 100 adults. Age mean±SD 25.7±4.6y	Suprathreshold intensity & liking on a VAS of sucrose in water at one concentration.	2 x 24hr diet recalls.	n/a	EI (kJ), macronutrients, salt, potassium, dietary fibre, & total sugars.	BMI, BF%, BP	No association between sweet-liking or intensity with dietary intake.	None.
Low, Lacy, McBride, & Keast, 2016 Cross-sectional observational	Australia. 60 adults. Age mean±SD 26±7.8y	Detection, recognition thresholds & suprathreshold intensity (gLMS) in 7 sugars & sweeteners.	FFQ.	n/a	EI (kJ) & macronutrient intake.	BMI, WC	No correlations between thresholds & dietary intake. Intensity rating (high-intensity sweetener, sucralose, Rebaudioside A) significantly correlated with total EI. No association BMI, WC.	**Yes,** ↑ intensity rating to artificial sweetener, ↑ in total energy intake.
Martinez-Cordero et al., 2015 Cross-sectional observational	Mexico. 56 adults. Age mean±SD 33.2±8.7y	The detection threshold of sucrose & aspartame.	7-day FR.	n/a	EI (kcal) & macronutrient intake.	BMI, WC, HC, BF%	No associations with BMI, WC, HC, BF%. Perception threshold of aspartame was negatively associated with energy intake.	**Yes,** ↑ detection threshold to aspartame, ↓ in total energy intake.
Methven et al., 2016 Cross-sectional observational	UK. N=36 adults. Age median 26y.	Hedonic liking of sucrose in water using VAS at 5 concentrations.	FFQ. Sweetness intensity of jelly & juice samples.	n/a	Total carbohydrate & sugar intakes.	BMI	Dietary intake did not differ between sweet likers & dislikers. Difference in ratings of sweetness between sweet likers & dislikers (P=0.019).	None.

Study	Population, participants	Sweet taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Sartor et al., 2011 Cross-sectional observational	UK. Study 1: N=33 (normal & obese); Study 2: N=12 adults Age mean±SD 26±6y	Pleasantness, intensity & preference scores for 5 concentrations of sucrose in water.	14-day FR.	n/a	EI & macronutrient intake.	Body weight, fat & lean mass.	Overweight/obese perceived sweet taste as less intense. No association between taste measurements & dietary intake.	None.
Smith, Ludy, & Tucker, 2016 Cross-sectional/ randomised sleep intervention	USA. N=51 adults Age mean±SD 25±8y	Detection threshold of sucrose (3-AFC). Preferred sweetness concentration using paired tests (sucrose).	2 x 24 hr recalls	n/a	EI & macronutrient intakes.	BMI, BF%	Long-sleepers had a higher sweet taste preference (P=0.04). Sweet preference after short sleep night correlated with EI & CHO intake (P<0.05).	**Yes**, ↑ sweet preference correlated with ↑ energy intake & CHO intake (after short-sleep night, all participants).
Stevenson et al., 2016 Cross-sectional observational	Australia. N=87 adults. Age mean±SD 21±3y	Liking & intensity of sucrose in water (1 strong & 1 weak solution) rated on LMS.	26-item dietary fat & sugar questionnaire (DFS).	n/a	Questionnaire score (approx. higher fat & sugar intake or 'western-style' diet)	BMI	Western-style diet* associated with hedonic differences in taste & flavor perception. No association between diet & BMI.	None.
Turner-McGrievy, et al, 2013 6-month, 3 groups, randomised weight loss trial	USA. N=196 adults. Age mean±SD 42.6±11y	Liking of 5 suprathreshold concentrations of sucrose in water. Rated on LMS.	2 x 24hr recalls.	n/a	EI & macronutrient (focus on beverages).	BMI, WC, BP	Sweet likers are more likely to be African-Americans. Sweet likers & supertaster (PROP bitter taster) interaction associated with fibre, beverage intake & metabolic syndrome.	**Yes**, ↑ sweet liking associated with ↑ energy intake from beverages & decreased fibre intake.

Abbreviations: EB; eating behaviour; DA, dietary assessment; AFC, alternative-forced choice; sem, standard error of the mean; SD, standard deviation; y, years; OA, oleic acid; BMI, body mass index; WC, waist circumference; HC, hip circumference; EI, energy intake; WS, western-style (diet); TFEQ; three-factor eating questionnaire; BP, blood pressure; gLMS, general labelled magnitude scale; LMS, labelled magnitude scale; kcal/d, kilocalories per day; FFQ, food frequency questionnaire; VAS, visual analogue scale; FR, food record; BF%, body fat percentage; CHO, carbohydrate.
*Dietary pattern 'Western-Style' diet determined from food frequency data and compared to chemosensory abilities.

2.5.3.4. Bitter taste perception and dietary intake

The review on bitter taste perception studies conducted for this PhD thesis suggests that increased sensitivity to bitter taste influences dietary intake. Of the 14 studies reviewed, 10 reported a link between bitter taste and dietary intake and/or food preference (Table 4). However, the most convincing association is that bitter taste sensitivity has an impact on the dietary intake or preference of cruciferous or bitter-tasting vegetables (Barajas-Ramírez, Quintana-Castro, Oliart-Ros, & Angulo-Guerrero, 2016; Drewnowski et al., 1999; Duffy et al., 2010; Inoue et al., 2017; Laureati et al., 2018). In addition, increased bitter taste sensitivity was associated with decreased intake or preference for bitter-tasting foods such as green tea (Pasquet, Oberti, El Ati, & Hladik, 2002), healthy food choices (Sharafi, Rawal, Fernandez, Huedo-Medina, & Duffy, 2018) and beer (Perna et al., 2018) (Table 2.4). Differences between tasters and different tastants are variable, as PROP and PTC taster status follow a bimodal distribution (Duffy et al., 2010; U. Kim et al., 2003; Lawless, 1980). Across these bitter taste perception studies there was high variability in chosen bitter taste stimuli (i.e. quinine, caffeine, PTC, PROP), the psychophysical measurements used (i.e. gLMS intensity ratings, detection thresholds, recognition thresholds) and the number of participants which ranged from N=17 through to N=1225 (Table 2.4).

2.5.3.5. Bitter taste perception and eating behaviour

Few studies to date have explored the relationship between eating behaviour with bitter taste perception. Of the 14 studies reviewed, there were 5 that included an eating behaviour questionnaire or measurement (Table 2.4). Sharafi et al (2018) reported that low sensitivity to quinine was associated with a higher restraint score and low intake of high fat/sweet foods; despite an increased preference of high fat/sweet foods (Sharafi et al., 2018). However, other studies which measured eating behaviour did not report any direct bitter taste and eating behaviour association (Barajas-Ramírez et al., 2016; Drewnowski et al., 1999). For example, participants with high levels of cognitive restraint were excluded from analysis in some studies (Barajas-Ramírez et al., 2016;

Duffy et al., 2010), in order to remove the confounding effect of the conscious control of eating, which may mask the associations between taste sensitivity and body weight.

2.5.3.6. Bitter taste perception and body composition

None of the studies reviewed found an association between body composition and bitter taste sensitivity (Table 2.4.).

Table 2.4. Studies which have investigated bitter taste sensitivity and dietary intake, eating behaviour and body composition.

Study	Population, participants	Bitter taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Barajas-Ramírez et al, 2016[†] Cross-sectional observational	Mexico. N=76 adults Age mean (sem) 22.8±0.6y.	Detection threshold PROP (water). Ascending 2-AFC procedure "two down -1 up rule". Intensity of PROP. PROP taster status of non-, med-, & supertasters.	7-day FR (two seasons, 6-months between each)	TFEQ	Energy & macronutrient intake. Vegetables, fruits, grains, protein-rich, dairy, oils, sweets, added sugars & alcohol.	BMI, WC	No association between adiposity and PROP threshold. PROP taste sensitivity associated with linoleic acid taste sensitivity (P<0.05).	**Yes**, ↑ PROP sensitivity consumed ↓ vegetables than non-tasters (P<0.05). Tasters had ↓ dairy & grain (P<0.05).
Cattaneo, Riso, Laureati, Gargari, and Pagliarini, 2019 Cross-sectional observational	Italy. N=59 adults Age mean (sem) 23.3±0.3y.	Recognition threshold caffeine. 7 concentration levels. 3-AFC procedure.	Food and Beverage Questionnaire (FB-FFQ). 7-day food diary.	n/a	Food group intake (DFE of 22-groups). EI, macronutrients.	BMI	Bitter threshold significantly correlated with sweet threshold (r= 0.34, P<0.01) and sour (r = 0.31, P< 0.05).	**Yes**, ↑ caffeine sensitivity ↑ intake of EI and CHO (food record). ↑ caffeine sensitivity ↓ oil intake.
Coltell et al., 2019 Cross-sectional observational	Spain. N=381 adults; including 144 with T2D^. Age mean±SD 65.1±4.7y	Category scale ratings of intensity at 5 concentrations of PTC and PROP solutions. Lowest to highest concentration.	Validated FFQ. 17-item questionnaire for assessing adherence to energy-restricted Mediterranean diet.	n/a	EI, sodium intake, Mediterranean diet adherence.	BMI, WC, BP	Total taste score, and bitter taste intensity ratings at the highest concentration only (PROP and PTC) inversely associated with BMI, WC (P<0.05).	None.
Drewnowski, Henderson, Levine, & Hann, 1999 Cross-sectional observational	USA. N= 159 women Age mean (sem) 27 (0.7) y	Suprathreshold hedonic liking on a 9-point scale. PROP in water at 5 concentration levels.	3-day FR (N=87) & food preference checklist.	Restraint scale & eating attitudes test (EAT).	EI, macronutrients, fibre, β-carotene.	BMI	Subjects were free from eating disorders (EAT test) and had normal restraint scores. PROP thresholds exhibited a bimodal distribution.	**Yes**, ↑ PROP sensitivity ↓ preference for brussel sprouts, cabbage, spinach, coffee (P<0.05)

Study	Population, participants	Bitter taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Duffy et al., 2010 Cross-sectional observational	USA. N=59 adults Age mean (sem) 26.1 (0.55) y	PROP gLMS bitterness and threshold. Quinine gLMS bitterness rating.	5-day non-consecutive FR. Validated FFQ.	TFEQ restraint only (*high restraint score excluded from study*)	EI (kJ), vegetable intake.	BMI, WC	Carriers of the functional PAV allele consumed less vegetables than AVI/AVI homozygotes, and PROP bitterness was negatively correlated with vegetable intake.	**Yes,** ↑ intensity (& genotype) ↓ vegetable intake (all types).
Inoue et al., 2017 Cross-sectional observational	Japan. Study 1: N=50 women; Study 2: N=40 women. Age range: 20-22y	Study 1: Intensity ratings PROP (4 concentrations). Study 2: Intensity ratings PROP (2 concentrations) 0-100 scale.	3-day weighed FR.	n/a	EI, sodium (g), total vegetables (g), Brassica vegetables (g).	BMI	Higher PROP sensitivity associated with higher salt intake (P<0.05).	**Yes,** ↑ PROP intensity ↓ brassica vegetable intake & ↑ salt intake.
Laureati et al., 2018 Cross-sectional observational	Italy. N=1225 adults Age mean±SD 36.9±12.9y	Intensity ratings of dark chocolate pudding at 4 concentrations levels of bitterness. PROP taster status.	Food familiarity & food preference questionnaire.	Food Neophobia Scale (FNS)	Vegetables, beverages & desserts: food familiarity 5-point scale; Preference 9-point scale. Food Neophobia statements.	n/a	High neophobia rated bitterness of dark chocolate more intense than low neophobics.	***Yes***,* ↑ bitter chemosensory intensity (&↑ food neophobia) ↓ liking of bitter vegetables & beverages. No PROP taster association.
Leong, Forde, Tey, and Henry, 2018a Cross-sectional observational	Singapore. N=100 adults. Age mean±SD 25.7±4.6y	Suprathreshold intensity & liking on VAS of PTC or quinine hydrochloride in water.	2 x 24hr diet recalls.	n/a	EI (kJ), macronutrients, salt, potassium, dietary fibre, & total sugars.	BMI, BF%, BP	Bitter-liking and intensity negatively correlated (r= -0.501, P<0.05). Decreased taste preference for PTC correlated with dietary fibre intake (r= -0.030, P<0.05).	None.

Study	Population, participants	Bitter taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Leong, Forde, Tey, and Henry, 2018b Cross-sectional observational	Singapore. N=114 adults. Indian, Chinese. Age mean(SEM) Age 26.6 (0.6)y	7 increasing concentrations of caffeine in water. Sequential monadic order defining. 1) no taste; 2) detection; 3) recognition (defined). Forced choice.	3-day FR.	n/a	EI (kcal), macronutrients, fibre, sodium, potassium.	BMI, BF%, WC	Indians had higher recognition threshold for all tastes, including bitter, compared to Chinese (P<0.05). EI not different between ethnicities.	None.
Lipchock et al., 2017 Cross-sectional observational	USA. N=20 Age mean±SD 31±1y	Rated bitterness on gLMS scale for PROP, caffeine, quinine, benzoate & urea.	FFQ caffeine and quinine (i.e. tonic water) questionnaire.	n/a	7- point scale of habitual caffeine & quinine intake.	n/a	Bitterness of caffeine related to mRNA expression of associated receptors.	**Yes**, ↑ bitterness of caffeine ↑ caffeine daily use (*small sample size to compare groups*).
Martinez-Cordero et al., 2015 Cross-sectional observational	Mexico. N=56 adults. Age mean±SD 33.2±8.7y	Detection threshold of caffeine and quinine hydrocholoride. 2-AFC.	7-day FR.	n/a	EI (kcal) & macronutrient intake.	BMI, WC, HC, BF%	No associations with BMI, WC, HC, BF%. No gender differences in insulin levels or HOMA-IR.	**Yes,** ↑ bitter taste (caffeine) sensitivity ↑ in percent of protein energy intake.
Newman and Keast, 2013 Cross-sectional observational	Australia. N=17 adults. Age mean±SD 28.6±3.2y	Detection threshold of caffeine (8 concentrations).	FR on testing days (30 sessions)	n/a	EI (kJ), macronutrients	BMI	Bitter taste threshold repeatable and reliable measure (test re-test).	None.
Pasquet, Oberti, El Ati, and Hladik, 2002 Cross-sectional observational	Tunisia. N=123 adults. Age mean±SD 36.0±10.7y	Recognition threshold (bitter) quinine hydrocholoride (11 concentrations) & PROP (12 concentrations). Staircase method.	Preference of 43 food items using scale rating.	n/a	Hedonic ratings of 43 food groups.	n/a	PROP sensitivity negatively correlated with hedonic response to NaCl solutions. High sensitivity to PROP associated with greater taste sensitivity to sucrose.	**Yes**, ↑ PROP sensitivity ↑ preference bread, aubergines, oranges, liver & eggs; ↓ preference green tea.

Study	Population, participants	Bitter taste tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Perna et al., 2018 Cross-sectional observational	Italy. N=118 adults. Age mean±SD 45.3±12.8y	Detection threshold test caffeine (5 concentrations) and PROP (6 concentrations);	Preference of 30 items using scale rating.	n/a	Hedonic ratings of 30 food groups.	BMI, BF%, Android fat %, Gynoid fat % (DXA)	Subjects with RS713598 polymorphism had higher PROP and caffeine thresholds ('non-tasters').	***Yes***[‡], bitter genotype nontasters associated with ↑ beer & ↓ butter & meat preference.
Sharafi et al., 2018 Cross-sectional observational	USA. N=110 women. Age mean±SD 45.3±8.7y.	PROP taster status from perceived intensity (5 concentrations)	3 24-hr FR. Block98 FFQ. Food preference 47 food items.	TFEQ, Healthy Eating preference Index (HEPI).	Liking for fats, sweets, salty, protein & fruits/vege. Scores intake-based fruit, vegetables, grains, dairy, proteins & fatty acids.	BMI, WC, BP	Low consumption of sweet/high-fat foods associated with high dietary restraint and low quinine taste function. PROP non-tasters lower diet quality; PROP supertasters consume a healthier diet (but low preference).	**Yes**, ↑ PROP sensitivity ↑ healthy diet consumption.

Abbreviations: EB; eating behaviour; DA, dietary assessment; AFC, alternative-forced choice; sem, standard error of the mean; SD, standard deviation; y, years; BMI, body mass index; WC, waist circumference; HC, hip circumference; EI, energy intake; TFEQ; three-factor eating questionnaire; BP, blood pressure; gLMS, general labelled magnitude scale; LMS, labelled magnitude scale; kcal/d, kilocalories per day; FFQ, food frequency questionnaire; sem, standard error of the mean;VAS, visual analogue scale; FR, food record; BF%, body fat percentage; CHO, carbohydrate; PTC, phenylthiocarbamide; DFE, daily frequency equivalent. [†]Food groups and food consumption frequency of different food groups analysed. ^Subjects with type 2 diabetes (T2D). [‡]Indirect and an implied relationship based on food neophobia scale or genotype/phenotype relationship.

2.5.3.7. Fatty acid taste perception and dietary intake

In metabolic health research, it is of interest whether dietary fat intake correlates to fatty acid taste sensitivity. The review conducted for this PhD thesis suggests that, overall, increased sensitivity to fatty acid taste (or fat) has a significant influence on dietary intake (Table 2.5). This was observed directly and indirectly in 11 out of 22 studies. Several of the reviewed studies found that increased sensitivity to fatty acid taste was significantly associated with decreased energy intake from fat (Costanzo et al., 2018; Costanzo, Orellana, Nowson, Duesing, & Keast, 2017; Heinze et al., 2018; Keller et al., 2012; Liang et al., 2012; Martínez-Ruiz et al., 2014; Stevenson et al., 2016; Stewart et al., 2010; Stewart & Keast, 2012; Tucker, Edlinger, Craig, & Mattes, 2014). However, like the bitter taste perception studies reviewed, there were considerable differences in the (1) taste stimuli used, (2) participants recruited, and (3) dietary intake measurements (Table 2.5). This heterogeneity observed in measurements, stimulus concentrations, and the chosen sample size is likely to obscure consistent relationships. As previously concluded in another recently published systematic review, this means that studies on fatty acid taste perception are difficult to directly compare or to draw meaningful conclusions from (Cox et al., 2015).

2.5.3.8. Fatty acid taste perception and eating behaviour

The current review of the literature revealed a few studies that measured eating behaviour (i.e. TFEQ) (Table 2.5). However, this was commonly done in order to use the cognitive restraint score as a variable to control, or adjust, for population differences (Keller et al., 2012; Liang et al., 2012; Panek-Scarborough et al., 2012) as high cognitive restraint may impact dietary intake and body weight. It was observed that the eating behaviour scores were not directly compared to the taste perception outcomes, or else not reported (Keller et al., 2012; Liang et al., 2012; Panek-Scarborough et al., 2012). In one study, participants with high levels of cognitive restraint were excluded from analysis entirely, as this is an important measurement of the conscious control of eating (Barajas-Ramírez et al., 2016). One study found BMI was significantly associated with disinhibition, however, no direct taste-disinhibition relationship was described (Shen, Kennedy, & Methven, 2017). Therefore, a gap in the current literature

is the direct investigation of associations between fatty acid taste and cognitive eating behaviour (Table 2.5).

2.5.3.9. Fatty acid taste perception and body composition

It has been proposed that sensitivity to fatty acid taste is impaired in obese people, at both levels, the oral and GI tract, when compared to normal-weight subjects (Brennan et al., 2012). This may be due to overeating as a result of the reduced satiating effect of fatty foods. Low-sensitivity to fat has been correlated with higher body fat (Stewart et al., 2010) and increased consumption of fatty foods (Martínez-Ruiz et al., 2014). Out of the 22 studies reviewed (Table 2.5), 8 studies found associations between fatty acid taste perception and body composition (Barajas-Ramírez et al., 2016; Lanfer et al., 2012; Liang et al., 2012; Martínez-Ruiz et al., 2014; Mela & Sacchetti, 1991; Pepino & Mennella, 2014; Ricketts, 1997; Tucker et al., 2014). These studies generally support that increased preference for high-fat foods, or decreased sensitivity to fatty acid taste, are associated with a higher BMI (Table 2.5).

Table 2.5. Studies which have investigated fatty acid taste/aroma sensitivity and dietary intake, eating behaviour and body composition.

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Barajas-Ramírez et al, 2016[†] Cross-sectional observational	Mexico. N=76 adults Age mean (sem) 22.8±0.6y.	Detection threshold linoleic acid (water). Ascending 2-AFC procedure "two down -1 up rule".	7-day FR (two seasons, 6-months between each)	TFEQ	Energy & macronutrient intake. Vegetables, fruits, grains, protein-rich, dairy, oils, sweets, added sugars & alcohol.	BMI, WC	BMI & WC negatively correlated with linoleic acid detection threshold (P<0.05). CHO consumption & linoleic acid detection both negatively correlated with WC (R^2=0.34).	None.
Boesveldt & Lundström, 2014 Cross-sectional observational	1 USA / 2 The Netherlands. 1) N=30 adults & N=30 Normal weight; N=30 ow/ob adults. 2) N=18 adults Age mean (SD) 26.7±4.2.	Odour discrimination, intensity & pleasantness of three milk samples (skim, medium, fat) on 100mm VAS. 3-AFC randomised.	Questionnaire about dairy consumption.	n/a	Total dairy per day, full or reduced milk consumption Total fat from dairy.	BMI	Participants were able to discriminate minute differences in fat content by odour alone (significant across all 3 experiments). No relationships between dairy consumption, BMI & ability to discriminate fat in milk samples.	None.
Costanzo et al., 2018 Co-twin randomised control trial	Australia. Adult twins Age mean (SD) 43.7±15.4y.	Detection threshold oleic acid (FATT) rank in food. Liking ratings for high-fat & reduced-fat foods & intensity ratings of tastants.	Three 24-h dietary recalls at pre & post 8-week dietary intervention.	n/a	Energy (kJ), macronutrient (g) & percentage of energy from macronutrients.	BMI, HC, WC	Significant time x diet interaction for FT rank after the 8-week trial (P< 0.001) where low-fat diet intervention decreased detection threshold & high-fat diet intervention increased detection threshold.	**Yes**, ↑ sensitivity relates to ↓ fat in the diet (intervention)
Costanzo et al, 2017 Baseline from co-twin randomised control trial	Australia. Females, N=69. Age mean (SD) 41.3±15.6y.	Oleic acid detection threshold (milk), fat ranking task (custard samples)	1. 24-h recall 2. FFQ (96 food items)	Food liking based on seven high- & low-fat foods	Total EI (MJ/d), protein (g), fats (g), CHOs (g) & alcohol (g) & % of EI.	BMI, HC, WC	Fat taste threshold associated with % energy from fat & negatively with % energy CHO. Short-term fat intake associated with diet but not body size.	**Yes**, ↑ sensitivity relates to ↓ % energy from fat & ↑ % energy from CHO

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Fisher and Birch, 1995 Cross-sectional observational	USA. N=18 children 3-5 years. Age mean (sem) 4.3±0.2y.	Rank-order preference for foods served at lunch & dinner. Seven items of 5 low-fat & 2 high-fat items alongside samples of food to taste & categorise on a scale.	Weighed food intake data from 6 x30-hour periods of observation (5 meals & 4 snacks during each session). Standard meals used during observational sessions.	TFEQ (parental results only)	Energy intake (kcal/d), macronutrient intake (% intake).	Height/weight triceps & subscapular skinfold measures.	Children who preferred high-fat foods in the ranking task had higher total fat intakes (P<0.05). Fat preferences were significantly related to skinfold measures (P<0.01).	**Yes**, ↑ preference high-fat food relates to ↑ intake of high-fat foods (children).
Heinze et al., 2018 Cross-sectional observational	Australia. N=30 adults. Age mean (SD) 29.2±3.7y.	Oleic acid (milk), paraffin oil, canola oil, & canola oil spiked with oleic acid	1) FFQ 2) 24-h recall (12 in total)	Hunger, satiety, & fullness VAS prior to taste testing (12 sessions)	Total EI, protein (g), fats (g), CHOs (g) & vitamins/minerals. Food groups (e.g. savoury, high-fat, sweet, fruits)	BMI, HC, WC	Detection threshold correlated with high-fat food intake. No association with BMI.	**Yes**, ↑ sensitivity relates to ↓ intake of savoury & high-fat food (R=0.589, P<0.01)
Keast et al., 2014 Blinded cross-over study	Australia. N=24 Age mean (SD) 28±15.6y.	Oleic, ascending 3-AFC method (milk) Classified hypo- or hyper- sensitive	1) Breakfasts with varying macronutrient levels (high fat/protein/CHO/balanced) followed by buffet lunch 2) FFQ (Validated) 3) 4-day FR (weighed/estimated)	Satiety questionnaire	EI at controlled *ad lib* lunch. Satiety measures (fullness, hunger, overeating), liking of food items EI & macronutrient intake	BMI	Those with impaired sensitivity to oleic acid (hyposensitive, n=10) consumed more total energy at a controlled lunch following a high-fat breakfast (P<0.05).	**Yes**, ↑ sensitivity relates to ↓ intake of energy (MJ)[§].
Keller et al., 2012 Cross-sectional observational	USA. N=317 African-American adults. Age mean (SD) 35.5±11.3y.	Ratings of oiliness, fat content, creaminess of 5%, 35% & 55% salad dressings. Rated with 170-mm VAS.	n/a	TFEQ, self-reported liking of 83 fat-containing foods, rated on 170-mm VAS.	Mean liking/acceptance of two food groups: high-fat foods & added fats & oils.	BMI, WC	Variation in rs1761667 was associated with a mean reported liking for added fats & oils (P=0.02).	***Yes***[‡] G/A genotype (↑CD36 expression compared to A/A) associated with ↓ liking of high-fat foods.

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Lanfer et al., 2012 Multi-site Cross-sectional observational	Italy, Estonia, Cyprus, Belgium, Sweden, Germany, Hungary, Spain. N=1,696 children. Age 6-9y.	A sensory testing session where the choice between a high- versus low-fat cracker & a natural versus sugar-sweetened apple juice.	Children's eating habits questionnaire FFQ (43 food items).	Children's eating habits questionnaire.	Weekly consumption frequency of food items & food groups.	BMI, BMI *z*-scores.	Frequent consumption of fatty foods related to fat preference ($P<0.01$). Ow/ob positively associated with fat preference & sweet preference testing (odds ratio 1.8). BMI associated with fat preference for girls (P=0.02).	None.
Liang et al., 2012 Cross-sectional observational	USA. N=317 African-American adults. Age mean (SD) 35.3±11.3y.	Fat discrimination testing (7 samples, ranging from 5%-55% fat content). "Same" or "different" against the second sample.	FFQ (83 food items)	TFEQ, Food preference questionnaire (83 food items)	Food group intake (monthly), food group preferences.	BMI, WC	Fat non-discriminators reported greater consumption of added fats & reduced fat foods ($P<0.05$). Fat non-discriminators had greater abdominal adiposity ($P<0.05$).	**Yes**, ↑ sensitivity (fat discriminators) relates to ↓ intake of added fats & reduced fat foods.
Martínez-Ruiz et al., 2014 Cross-sectional observational	Mexico. N=121. Age mean (SD) 21.1±3.6y.	Linoleic acid FA (water) Rated with intensity scales (not threshold)	24-hour recall for 30-35 days (online self-administered). Food group analysis of most frequently consumed foods.	Preference rating for foods[†].	Total EI (kcal/d) Food liking & preference	BMI, WC, BF% using skinfold measures (bicipital, tricipital, subscapular & suprailiac)	Perceived taste of linoleic acid rated as high intensity correlated with lower consumption of high-fat fast foods (P=0.04). Participants with high-intensity linoleic acid ratings had lower BMI (P=0.04) & WC (P=0.03).	**Yes**, ↑ sensitivity relates to ↓ intake of high-fat foods.
Mela and Sacchetti, 1991 Cross-sectional observational	USA. N=30 adults. Age mean (SD) 27.5±7y.	Preference testing of 10 fat containing stimuli, 2-5 levels of fat (e.g. scrambled eggs with 5, 10, 20, 30 g oil).	10-day FR (estimated)	n/a	EI (kcal/d), Macronutrient intake (kcal/d), dietary fat intake (% of kcal)	BMI, body fat %	No consistent relationships with sensory & dietary measures. A positive correlation between fat preference & BF% ($P<0.01$).	None.

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Panek-Scarborough, Dewey, & Temple, 2012 Cross-sectional observational	USA. 1 N=59. Age mean (sem) 21.9±0.7y. 2 N=34. Age mean (sem) 24.9±1.7y.	Fat from cream mixed with fat-free milk which yields 0.25, 0.5, 1, 2, 4, 6, 10% fat by volume (threshold series). Staircase procedure	24-h dietary intake recall	1) Reinforcing the value of food (computer task) 2) TFEQ 3) Hunger, fullness & food liking	Did not report any 24-h recall results. Reinforcing the value of food Dietary restraint, hunger & disinhibition, & binge eating disorders	BMI	High-fat detection threshold (i.e. insensitivity) predicted the greater reinforcing value of food ratings (P<0.02). No other predictors found.	Relationship not clearly reported.
Pepino et al., 2012 Cross-sectional observational	USA. African American & Caucasian N=21 obese adults. Age mean (SD) 39.1±3.5y.	Oleic acid & triolein FA (water) Tested with & without orlistat added to the emulsion as control (to prevent absorption of fats)	24-h recall with dietitian interview	1) Fat preference questionnaire (FPQ, validated) 2) Food craving inventory	Mean EI (kcal/d), fat consumed, macronutrient distribution Fat preference Food cravings	BMI	CD36 genotype grouping did not influence dietary intake, fat preference or food craving (P>0.20).	None.
Pepino & Mennella, 2014 Cross-sectional observational	USA. N=47 women. Age mean (SD) 28.5±6.7y.	Fat ranking task Intensity perception (gLMS scales) creaminess, oiliness, sweetness, & saltiness	n/a	1) Fat preference 2) Food cravings 3) Restrictive eating	Fat preference Food craving Restrictive eating measures	BMI, HC, WC, BIA	Smokers who were obese rated desserts as less creamy & less sweet than other groups (P<0.02). Smokers identified high-fat food as items that 'taste better', as well as crave & eat more than never-smokers (P<0.03).	Relationship not clearly reported.
Ricketts, 1997 Cross-sectional observational	USA. N=88 children. Age mean 10.8y.	Regular & low-fat versions of baked snacks, cookies. Taste & hedonic evaluation of 9-point scales.	3-day FR (estimated)	n/a	Energy intake (kcal/d), Macronutrient intake (% intake)	BMI, Tricep & subscapular skinfold measures.	Children who preferred high-fat snacks had a high dietary fat intake as a percentage of daily energy intake (P<0.05). Tricep skinfold & BMI measures positively correlated with high-fat food preferences (P<0.05).	**Yes**, ↑ fat preference relates to ↑fat intake (children).

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Shen, et al., 2017 Randomised dietary intervention (low fat or portion control diet)	UK. N=136 Age 18-55y.	Hedonic liking for ice-cream with four concentration levels of fat (6%, 10%, 15%, & 20%), 9-point hedonic scale. Genotyping of CD36 & CA6.	1 FFQ (133 food items) 2 x 3-day FR	TFEQ	EI (kcal), fat intake (g), dairy food group consumption from (FFQ).	BMI, WC, HC, body fat %.	High-fat liker group found to have higher dairy consumption compared to high-fat dislikers (P<0.05). CA6 genotype is significantly related to fat intake as a percentage of energy intake (P<0.02).	Unclear.
Stevenson et al., 2016* Cross-sectional observational	Australia. N=87 Age mean (SD) 59.9±13.7y.	1) Ratings of intensity, fattiness, hedonics, gLMS for test solutions. 2) Sniffin' Sticks test	Dietary fat & sugar questionnaire (DFS) food frequency.	n/a	High saturated fat & added sugar in diet termed WS diet.	BMI	Odour identification poorer in consumers of western-style diet (P<0.05). Fat discrimination was poorer in consumers of WS diet (P<0.01). No association between diet & BMI.	**Yes**, ↑ fat discrimination relates to ↓ intake of dietary fat & sugar.
Stewart et al., 2010 Cross-sectional observational	Australia. 1 N=31 Age mean (sem) 29±1.4y. 2 N=54 Age mean (sem) 20±0.3y.	Oleic, linoleic, lauric fatty acids Ascending 3-AFC method. Fat ranking task with vanilla custard.	2-day diet record (weighed/estimated).	n/a	EI (kJ) & macronutrient distribution	BMI	Hypersensitivity (n=12) associated with lower total energy & lower fat intake in the diet (P<0.05) compared to hyposensitivity (n=42).	**Yes**, ↑ sensitivity relates to ↓ intake of energy & fat intake.
Stewart & Keast, 2012 Randomised cross-over dietary intervention (high fat, low-fat diet)	Australia. N=19. Age mean (SD) 33±13y.	Oleic, ascending 3-AFC method (milk) Ranking task with custard	1) 24-h food recall 2) Dietary intervention (high-fat diet & low-fat diet)- a measure of sensitivity post dietary intervention	Hedonic liking for full-fat & low-fat foods	EI & macronutrient distribution (intervention) & sensitivity to taste measured after. Sensitivity increased post-low-fat diet.	BMI	Consumption of a low-fat diet appeared to increase sensitivity to oleic acid (P<0.05) & increased the ability to rank fat content in custard samples (P=0.05).	**Yes**, ↑ sensitivity relates to ↓ intake of dietary fat (intervention[§]).

Study	Population, participants	Fatty acid / fat related tests	DA tool	EB tool	DA & EB outcomes	Body composition	Outcome	DA & chemo-sensory / EB relationship
Tucker, Edlinger, Craig, & Mattes, 2014 Cross-sectional observational	USA. N=48 Age mean (SD) 28.5±10.4y.	Oleic acid (water) Modified staircase procedure 7 visits for repeated measure	BRFS Block Rapid Fat Screener – a self-administered survey of 100-items to correlate with fat intake in the diet	n/a	Total fat, saturated fat, monounsaturated, cholesterol intake. Sensitivity decreased with higher saturated fat intake in the diet.	BMI	Saturated fat intake & the oleic acid threshold were positively correlated, as fat intake increased sensitivity decreased (P<0.001). The lean & overweight group had total fat intake & oleic acid threshold positively correlated (P<0.001)	**Yes**, baseline ↑ sensitivity relates to ↓ intake of saturated fat intake.
Zhou, Shen, Parker, Kennedy, and Methven, 2016 Cross-sectional observational	UK. 1 N=46 Age 19-53y. 2 N=51 Age 18-55y.	1) Fat intensity ratings under (i.e. mouthfeel odour-masked, etc). OA threshold 3-AFC, staircase procedure. 2) Fat intensity ratings. 2-AFC discrimination test. OA threshold 3-AFC staircase procedure.	FFQ (Validated)	n/a	EI, fat intake	BMI	Odour increases fat intensity perception (P<0.0001). Mouthfeel increases fat intensity perception (P<0.0001). No association between taste detection, nutrient intake or BMI.	None.

Abbreviations: EB; eating behaviour; DA, dietary assessment; AFC, alternative-forced choice; sem, standard error of the mean; SD, standard deviation; y, years; OA, oleic acid; BMI, body mass index; WC, waist circumference; HC, hip circumference; EI, energy intake; BIA, bioelectrical impedance analysis; WS, western-style (diet); TFEQ; three-factor eating questionnaire; BP, blood pressure; gLMS, general labelled magnitude scale; LMS, labelled magnitude scale; kcal/d, kilocalories per day; FFQ, food frequency questionnaire; VAS, visual analogue scale; FR, food record; BF%, body fat percentage; CHO, carbohydrate; ow/ob, overweight/obese; QEWP, Questionnaire on Eating and Weight Patterns; BES, binge eating scale.

*Dietary pattern 'Western-Style' diet determined from food frequency data and compared to chemosensory abilities.

†Food groups and food consumption frequency of different food groups analysed.

§One variable controlled in the relationship (i.e. low-fat diet intervention for 6 weeks = increased fatty acid taste sensitivity post-intervention).

‡Indirect and an implied relationship based on supportive data from the literature (Keller et al., 2012).

2.5.3.10. Olfaction, dietary intake, and obesity

Few studies have been conducted on aroma perception and dietary intake in humans. One study has demonstrated that high aroma intensity was associated with a smaller bite-size of vanilla custard dessert samples (de Wijk, Polet, Boek, Coenraad, & Bult, 2012). In mice, the sense of smell is enhanced, and olfactory circuits are sensitive to changes in energy balance (Palouzier-Paulignan et al., 2012; Soria-Gómez et al., 2014).

Impairment of smell has been associated with eating disorders, such as anorexia (Roessner, Bleich, Banaschewski, & Rothenberger, 2005). The lack of odour recognition in anorexic people has been associated with restrained eating habits (Stafford, Tucker, & Gerstner, 2013). In consideration of weight gain and long-term metabolic health outcomes, how olfactory sensitivity may relate to taste perception and dietary intake is of interest. One study has shown that retro-nasal olfactory perception can be affected by the onset of obesity (Palouzier-Paulignan et al., 2012). Importantly, with increasing BMI the ability to detect and identify odours decreases (Simchen, Koebnick, Hoyer, Issanchou, & Zunft, 2006).

2.5.3.11. Mouthfeel, dietary intake, and obesity

Alongside taste and aroma, the textural influence of food plays another crucial role in overall dietary intake. Previous research on oral processing time and bite-size are linked to the degree of satiety (de Wijk, Zijlstra, Mars, de Graaf, & Prinz, 2008; Zijlstra, de Wijk, Mars, Stafleu, & de Graaf, 2009; Zijlstra, Mars, de Wijk, Westerterp-Plantenga, & de Graaf, 2008). A study on oral processing showed that the intake of hard foods reduces overall energy intake when compared to soft foods (Bolhuis et al., 2014). Studies have found positive associations between body weight status and eating rate speed (Maruyama et al., 2008; Otsuka et al., 2006). This supports the belief that highly processed foods which require minimal oral processing can lead to weight gain and obesity (Stieger & Van de Velde, 2013).

2.6. SUMMARY OF THE LITERATURE REVIEW

From the explored literature it was clear that there are external determinants of food choice such as society, the government and the food industry (Story et al., 2008). For example, the government can introduce policies and taxes which may change long-term dietary habits (i.e. sugar tax) (Cropp, 2017). The second level of influence comes from the social environment and the community. This is where cultural influences, family, friends, schools and workplaces can further shape long-term dietary intake and habits through social facilitation (Gerritsen & Wall, 2017; Herman et al., 2003; Salvy et al., 2012). Finally, individual-level factors which include lifestyle, attitude, and biological factors were reviewed as the third level of influence. Whilst individual and biological factors (e.g. genetics, age, taste and sensory perception) are promising targets for health-related or weight loss intervention studies, it is important that these are addressed in the wider context in which people make decisions.

The biological feedback system is initiated by sensory perception and further leads to hypothalamic signalling, reward processing, and the behavioural control of eating (Rolls, 2015). Adiposity may be associated with taste perception by influencing appetite regulation and satiety (Dando, 2015). The alteration of taste sensitivity and hedonics has been linked to changes in gut-derived appetite signalling hormones (Miras & le Roux, 2014). The most compelling evidence to date of this association is that taste sensitivity has been shown to change after bariatric surgery (Thanos et al., 2015). Taste perception has also been shown to be influenced by genetics, age and life stage, gender and obesity. In addition, olfaction and mouthfeel perception are key drivers of food intake due to their influence on flavour perception and acceptance (Doty & Bromley, 2012; Ingham, 1995; Jeltema et al., 2014).

Detection and recognition threshold measurements have been used to determine individual sensitivity to low concentrations of taste stimuli (Giguère et al., 2016; Tucker et al., 2017). However, a number of recent studies which look to compare dietary intake and taste perception are choosing to measure taste perception with suprathreshold

concentrations of taste (Feeney, O'Brien, Scannell, Markey, & Gibney, 2017; Noel et al., 2017). Suprathreshold taste can be measured with gLMS scales to give an indication of intensity perception (Bartoshuk et al., 2004).

Dietary assessment methods such as food records and FFQs appear to be the most commonly used measures to provide robust associations with taste perception. Measurement of the whole diet was considered to be superior in order to draw associations with taste perception (Stevenson et al., 2016). Additionally, eating behaviour measurements which further impact dietary motives are related to cognitive responses to food, which can be measured with validated questionnaires, the most common of which measures dietary restraint, disinhibition of control and susceptibility to hunger (Stunkard & Messick, 1985).

Studies on sweet taste, bitter taste and fatty acid taste perception were reviewed which also measured dietary intake, body composition and/or eating behaviour. Associations between taste perception and dietary intake were observed in bitter taste and fatty acid taste perception studies (Table 2.4 and Table 2.5). Increased sensitivity to bitter taste was most strongly associated with a decreased intake in vegetables (i.e cruciferous vegetables) and increased fatty acid taste perception was associated with decreased intake of total energy intake (kJ) and dietary fat. In contrast, sweet taste hedonic liking measurements or sweet taste liking clusters were associated with the dietary intake of sugars, but detection threshold measurements were not (Table 2.3). However, there was a significant proportion of studies across sweet taste, bitter taste and fatty acid taste perception studies that showed no diet-taste associations (approx. 40% of the studies reviewed).

The review conducted for this PhD supports that there are a number of studies that have found meaningful associations between taste perception and dietary intake and weight status. However, there is no conclusive evidence confirming the biological basis for these taste perception and dietary intake associations. There are recent studies which have found associations between sweet taste perception and long-term appetite regulators such as plasma leptin (Han, Keast, & Roura, 2017; Overberg et al., 2012) which explains some of the day-to-day variations in taste (Tan & Tucker, 2019).

Endocrine regulation and appetite sensation were shown to be associated with reward signalling pathways which are likely to influence cognitive eating behaviour.

2.6.1. Gaps in the current literature

To date, there is no conclusive evidence of an association between taste perception and dietary intake. However, studies continue to investigate whether the decreased sensitivity of a primary taste is associated with the increased dietary intake of the associated macronutrients (Stewart, Newman, et al., 2011). It has been suggested that measurements of hedonic liking of taste may be more strongly associated with long-term dietary habits (Ettinger et al., 2012), but additional research is required to substantiate this link.

Cognitive domains of eating behaviour may also be associated with taste perception. A number of studies have investigated measurements of dietary restraint in taste perception studies, but have used the restraint score to either exclude participants with high dietary restraint (Barajas-Ramírez et al., 2016) or the measurement was used to adjust for study outcomes as a covariate (Keller et al., 2012; Panek-Scarborough et al., 2012). As taste perception may be intimately linked with cognitive reward signalling pathways and appetite regulation, a better understanding of associations with the cognitive domains of eating behaviour may further support associations with food choice. In addition to eating behaviour, there is emerging evidence of a link between endocrine regulation, appetite regulation and taste perception (Kubasova et al., 2015). There are only a few studies which have measured endocrine regulators of appetite and energy metabolism. Additional research in this area may help to establish the distinct role of these signalling pathways alongside taste perception.

Finally, body composition may be associated with taste perception. It is still unclear as to whether or not specific tastes (i.e. sweet taste) is more likely to be associated with body weight status. It may instead be that inflammation which arises from obesity and metabolic dysregulation causes insensitivity to general taste acuity by inhibiting the renewal of taste buds (Kaufman et al., 2018). Conversely, there are studies which have

observed that obesity is associated with increased taste sensitivity (Hardikar et al., 2017; Pasquet et al., 2007). Further investigation into this area is warranted.

In conclusion, food consumption involves complex taste perception interactions, as well as the added complexity of aroma, visual, auditory and mouthfeel sensations. The information on how these components relate to one another, as well as their influence on long term metabolic health, is scarce and needs to be further explored. A better understanding of sensory perception and associations with dietary intake is an important step in further understanding the biological mechanisms which influence body weight. However, taste testing procedures and dietary intake measurements both must be reliable in order to draw valid conclusions. It has been found that a lack of repeatability is a potentially misleading factor in the design of previous studies which have aimed to determine the taste perception capabilities of an individual. More studies are required to determine the reproducibility of methods for taste perception as well as determining quantitative sensory measures which may have an association with metabolic disease risk or body weight gain. In addition, investigation into whether relationships exist between taste perception, dietary intake, eating behaviour and markers of metabolic health is warranted.

2.7. References

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Chapter III.

Fat Sensation: Oleic Acid Taste and Olfaction Sensitivity and the Link with Disinhibited Eating Behaviour

3.1. Abstract

Perception of fatty acid taste, aroma and texture are proposed to influence food preferences, thus shaping dietary intake and eating behaviour and consequently long-term health. In this study, we investigated associations between fatty acid taste, olfaction, the mouthfeel of fat, dietary intake, eating behaviour and body mass index (BMI). Fifty women attended three sessions to assess oleic acid taste and olfaction thresholds, the olfactory threshold for *n*-butanol and subjective mouthfeel ratings of custard samples. Dietary intake and eating behaviour were evaluated using a food frequency and three-factor eating questionnaire, respectively. Binomial regression analysis was used to model fatty acid taste and olfaction data. Taste and olfactory detection for oleic acid were positively correlated (r=0.325; P< 0.02). Oleic acid taste hypersensitive women had significantly increased *n*-butanol olfactory sensitivity (P< 0.03). The eating behaviour disinhibition and BMI were higher in women who were hyposensitive to oleic acid taste (P< 0.05). Dietary intake of nuts, nut spreads and seeds were significantly correlated with high olfactory sensitivity to oleic acid (r_s= -0.410, < 0.01). These findings demonstrate a clear link between fatty acid taste sensitivity and olfaction and suggest that fatty acid taste perception is associated with specific

characteristics of eating behaviour and body composition.

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3.2. Introduction

Taste is the sensation experienced when a chemical stimulus or tastant in the mouth is recognised by receptors of the taste buds. There are five established taste modalities including sweet, salty, sour, bitter, and umami (savoury). Sweet, umami and bitter molecular sensors have been identified as G-protein coupled receptors (GPCRs), salt is recognised by an ion channel receptor (ENaC) and the sour taste receptor mechanism (yet to be identified) responds to the presence of acid (Trivedi, 2012a, 2012b; Ye et al., 2016).

Fat creates a range of textural qualities which are considered to be the well-known sensory properties of fat, such as creaminess, oiliness and thickness (Chen & Eaton, 2012; Szczesniak, 2002). Until recently, fat was considered only to be perceived through mouthfeel and olfaction, but there is now considerable evidence that fatty acids can be perceived by specific taste receptors of the tongue (Chalé-Rush, Burgess, & Mattes, 2007a; Keast & Costanzo, 2015; Running, Craig, & Mattes, 2015). Several studies have demonstrated varying gustatory sensitivities to fatty acids at low concentrations (Mattes, 2009; Running, Mattes, & Tucker, 2013; Stewart et al., 2010; Stewart, Newman, & Keast, 2011). Furthermore, detection of volatile fatty acids by odour (Boesveldt & Lundström, 2014; Chalé-Rush, Burgess, & Mattes, 2007b) or mouthfeel have been implicated in enhancing enjoyable eating experiences (Liou & Grün, 2007). The oral perception of fatty acids (fat taste or fatty acid taste) is distinct from that of wider textural fat sensations. Fatty acid taste can be confused with or perceived as a ‘sour’ or ‘bitter’ taste sensation. Fatty acid taste is also different to the perception of oxidised or degraded fatty acids, which is instead an irritant ‘rancid’ or ‘acidic’ profile that is perceived via trigeminal signalling pathways (Running, Craig, & Mattes, 2015).

Increasingly, taste is being investigated for its role in the signalling pathways which govern the body’s response to incoming food (Cvijanovic, Feinle-Bisset, Young, & Little, 2015). The physiological mechanisms of taste have multiple functions which include: signalling appeal or safety of items in the oral cavity, providing feedback to the

digestive system about incoming food, and supporting the regulation of satiety (Liu, Archer, Duesing, Hannan, & Keast, 2016). It has recently been suggested that over-consumption of dietary fat might alter the sensitivity or the expression of taste receptors (Stewart, Newman, et al., 2011) and that high-fat diet exposure can decrease sensitivity to fatty acid taste in lean participants (Stewart & Keast, 2012). These data suggest sensitivity to fatty acid taste may have a significant impact on eating behaviour and long-term dietary intake with important health consequences (Cox, Hendrie, & Carty, 2015). Commonly cited causes of obesity include major changes in our food environment (Swinburn et al., 2013) which have led to over-consumption of inexpensive, highly palatable energy-dense and nutrient-poor foods. Given that fat rich foods have a powerful hedonic appeal, preferences for fatty foods are important contributors to increases in body weight and metabolic disease risk (Martínez-Ruiz, López-Díaz, Wall-Medrano, Jiménez-Castro, & Angulo, 2014).

To date, only a few studies have investigated the relationship between olfactory sensitivity to fatty acids and dietary intake, eating behaviour or obesity development (Boesveldt & Lundström, 2014; Stevenson et al., 2016). Human studies have shown that the fat content level of milk can be discriminated by odour alone, however, this ability was shown to have no relationship with BMI or dairy consumption (Boesveldt & Lundström, 2014). Stevenson et al (2016) found that a western-style diet was associated with poor odour identification as well as poor fat discrimination by taste (Stevenson et al., 2016). Other studies have reported that olfaction may be desensitised in individuals who are morbidly obese (Jurowich et al., 2014; Richardson, Vander Woude, Sudan, Thompson, & Leopold, 2004). This may be due to changes in olfactory sensory neurons (OSNs), with a decline of OSNs shown to occur over time during high-fat intake in animal studies (Thiebaud et al., 2014). In contrast, another study found participants with obesity had a stronger hedonic response towards the smell of dark chocolate than non-obese participants, rating the odour as significantly more pleasant (Stafford & Whittle, 2015). Furthermore, there is emerging evidence that variations in human olfactory receptor gene expression can influence eating behaviour, resulting in increased adiposity (Choquette et al., 2012). Similarly, it has been suggested that individuals who are obese may have a better sense of smell for food odours but not to non-food odours

(Stafford & Welbeck, 2011; Stafford & Whittle, 2015). Despite some authors suggesting olfactory cues may be dispensable for the detection of dietary fats (Mattes, 2009), it is not clear whether olfactory sensitivity for fatty acid runs in parallel with an individual's sensitivity to fatty acid taste.

Recent work has found the ability to detect different levels of fat in a food matrix is related to taste sensitivity by comparing results of a fat ranking task with threshold sensitivity to oleic acid (Stewart et al., 2010). Similar fat ranking tasks have classified participants as being fat 'discriminators' or 'non-discriminators', where non-discriminators consumed greater amounts of dietary fat and had higher abdominal adiposity (Liang et al., 2012). Despite mouthfeel perception and the dynamic nature of eating being critical for food acceptance (Appelqvist, Poelman, Cochet-Broch, & Delahunty, 2016), there are still relatively few studies on how mouthfeel perception relates to other sensory attributes such as taste and olfactory modalities (Proserpio, Laureati, Invitti, et al., 2016; Zhou, Shen, Parker, Kennedy, & Methven, 2016).

In the present study, we designed a series of experiments to investigate the relationships between fatty acid taste, olfaction and mouthfeel modalities, and how fatty acid taste perception measurements may relate to dietary intake and eating behaviour. The present study aimed to (i) measure oleic acid taste and olfactory detection, (ii) explore links between oleic acid taste, olfaction and mouthfeel perception of fat, and (iii) investigate oleic acid taste detection and associations with eating behaviour, dietary intake and body composition.

3.3. Materials and Methods

Fatty acid taste and olfactory detection rate of oleic acid were determined in this study by extending the commonly used 3-alternative forced choice (AFC) procedure testing past the assumed taste threshold level and by carrying out three repeated sessions to increase accuracy and precision. The relationship between these sensory modalities was established as well as comparison with eating behaviour, dietary intake, olfactory detection of *n*-butanol, mouthfeel perception of fat and body composition.

3.3.1. Participants

Participants included premenopausal, non-pregnant, non-lactating New Zealand (NZ) European women aged 18-45 years living in Auckland, NZ. All participants self-reported being healthy, had no cold or flu symptoms on test days, had no food allergies or intolerances, nor a dislike towards milk, coconut or dairy based products, were non-smokers and had no medical history or evidence of conditions that could alter gustatory function e.g. undergoing chemotherapy, having diabetes, nor had taken antibiotics over the past three months (Kruger et al., 2015; Steinbach et al., 2009). Participants were recruited using posters, flyers, social media (e.g. Facebook, Twitter) and via email lists (e.g. Massey University staff and student lists). Participants were screened with an online questionnaire to assess the inclusion and exclusion criteria. This study was conducted according to guidelines laid down in the Declaration of Helsinki and all procedures were considered to be low-risk by the Massey University Human Ethics Committee, NZ. Written, informed consent was obtained from all the participants prior to participating in the study.

Participants were required to attend three morning sessions in a fasted state at which they were tested on taste, olfactory and mouthfeel measurements. Participant height and weight were measured at the first visit using a standardised protocol. Body mass index (BMI) was calculated (weight (kg) / height (m^2)). Body fat percentage was measured at

the first visit using bioelectrical impedance (BIA) measurement (InBody230, Biospace Co. Ltd, Seoul, Korea) and standardised techniques (von Hurst et al., 2015). In-between study visits participants were required to complete two online questionnaires to assess dietary intake and eating behaviour.

3.3.2. Stimuli preparation for sensory measurements

3.3.2.1. Stimuli for fatty acid taste measurement

The methodology for taste testing is described in further detail by Haryono et al. (Haryono, Sprajcer, & Keast, 2014). In brief, a milk emulsion vehicle was used and made from non-fat UHT milk (Homebrand, NZ), added to a glass beaker along with food grade gum arabic (Hawkins Watts, NZ). The addition of 0.01% EDTA (FCC, Spectrum Laboratory Products Inc., Gardena CA, USA) was added to prevent oxidation. The milk base had 5% mineral oil added (Purity FCC grade, Canadian Oil Company, Ontario, Canada). This solution was homogenised thoroughly with a Silverson L4RT homogeniser. The milk base solution was divided in half so that a series of the fatty acid vehicle with increasing concentrations of oleic acid could be created. Half of the milk base was used for the blank testing solutions. Each concentration in the series of active stimuli required a separate beaker. In each beaker, in the series, the appropriate amount of oleic acid (Sigma-Aldrich, St Louis, MO, USA) was added to the milk. Homogenisation of each beaker was undertaken in ascending order. The homogeniser was sanitised after contact with oleic acid to prevent any contamination of non-oleic acid solutions. Testing stimuli were made fresh on the day of evaluation. This oleic acid in milk emulsion concentration series (0.02, 0.06, 1, 1.4, 2, 2.8, 3.8, 5, 6.4, 8, 9.8, 12 and 20mM) has previously been used in several studies (Keast, Azzopardi, Newman, & Haryono, 2014; Panek-Scarborough, Dewey, & Temple, 2012; Stewart et al., 2010; Stewart & Keast, 2012).

3.3.2.2. Stimuli for fatty acid olfactory measurement

Olfactory stimuli were created from oleic acid to create a series with an increasing concentration of fatty acid content. The stimuli and procedure were developed in order to align with the taste testing (Haryono et al., 2014), however, our internal testing showed that higher concentrations of oleic acid were required for olfactory detection. The stimuli were prepared by adding the oleic acid to odourless light mineral oil (Sigma-Aldrich, St Louis, MO, USA) in a concentration series. All blank testing solutions bottles contained 5 mL of odourless light mineral oil. The series of active stimuli concentrations ranged from a 6 mM oleic acid solution to a 380 mM oleic acid solution (6, 12, 24, 48, 95, 190 and 380 mM). Olfactory stimuli were kept in small, individual containers with a screw top lid (Figure 3.1). Oleic acid required mixing by drawing 3-4 times with a 10 mL pipette, to ensure an even emulsion of fatty acid and mineral oil. All olfactory stimuli were made fresh on the day of evaluation. All oleic acid used was from the same batch as the tasting procedure and obtained from the same supplier to allow for comparison across testing stimuli. The methodology for olfactory testing used the same procedure as taste testing (3-AFC) but with a decreased number of concentrations (see Table 3.1).

Figure 3.1. Image of sniffing bottles for oleic acid olfactory measurement using the ascending 3-AFC procedure.

3.3.2.3. *n*-butanol threshold test

The overall olfactory performance of each participant was established using a test kit 'Sniffin' Sticks' which has been widely used in research (Hummel, Kobal, Gudziol, & Mackay-Sim, 2007) in order to compare results with the oleic acid olfaction test. The 'Sniffin' Sticks' kit contains 16 pen sets (triplets) with increasing thresholds of the volatile *n*-butanol, alongside blank odour pens (Burghart Instruments, Wedel, Germany). The 16 pen sets require presentation of three pens each time with only one of the three pens containing the target odourant (forced choice procedure). Pen no. 1 is the highest concentration and pen no. 16 is the lowest, with a high score representing increased sensitivity to *n*-butanol, and a score over 6.5 considered to be 'normosmia' (Denzer et al., 2014; Hummel et al., 2007).

Table 3.1. Trial measurements and methods

Measurement	Methods	Reference	Equipment	Outcomes
Body composition profile	Anthropometric measurements (height, weight) and BIA	Ling et al., 2011; von Hurst et al., 2015	Direct segmental measurement (DSM) BIA (InBody230, Biospace Co. Ltd, Seoul, Korea). Stadiometer	Body composition - BMI profiling (height and weight) - fat and lean mass
Taste perception oleic acid (C18:1)	3-AFC procedure ascending method with 6 correct responses (3 at the same concentration and 3 at consecutive higher concentrations)	Developed in this study with reference to Haryono et al., 2014; Keast et al., 2014; Mattes, 2007; Running, 2014; Stewart et al., 2010; Stewart, Feinle-Bisset, et al., 2011; Stewart, Newman, et al., 2011; Tucker & Mattes, 2013	Silverson homogeniser (L4RT)	Sensitivity to oleic acid (C18:1) threshold measurement. Identification of 'hypo' or 'hypersensitivity'
Olfactory perception oleic acid (C18:1)	3-AFC procedure. Maximum of 7 concentration levels	Developed in this study with reference to Boesveldt & Lundström, 2014; Hummel et al., 1997; Kallas & Halpern, 2011	-	Sensitivity to oleic acid (C18:1) olfactory threshold measurement
n-butanol olfactory perception	3-AFC procedure. 16 concentration levels presented in rising order (pens 16, 14, 12, etc)	Denzer et al., 2014; Hummel et al., 2007	Burghart Sniffin' Sticks smell test	Sensitivity to *n*-butanol (Sniffin' Sticks) olfactory threshold
Mouthfeel perception	Subjective hedonic and intensity linear scales, JAR scales	Developed in this study with reference to Ares et al., 2009; Keller et al., 2012; Martínez-Ruiz et al., 2014; Popper, 2014; Worch et al., 2012	-	Subjective rating of mouthfeel (intensity, liking, etc)
Dietary intake	220-item FFQ	Houston, 2014; Kruger et al., 2015	Analysis using Foodworks 7 2012 (Xyris Software, Australia). Questionnaire completed on SurveyMonkey™ online platform	Daily energy, macronutrient and food group intake
Eating behaviour	TFEQ	Stunkard & Messick, 1985	Questionnaire completed on SurveyMonkey™ online platform	Restraint, disinhibition and hunger measurement

Abbreviations: BIA, bioelectrical impedance; BMI, body mass index; AFC, alternative-forced choice; FFQ, food frequency questionnaire; JAR, just about right; TFEQ, three-factor eating questionnaire

3.3.2.4. Subjective mouthfeel measurement test

For testing of mouthfeel and textural influence of fat, vanilla custard tasting stimuli were designed. Test stimuli were created using coconut oil, which has a high saturated fat content providing a high level of fat-related textural attributes (Vingerhoeds, de Wijk, Zoet, Nixdorf, & van Aken, 2008). The base for vanilla custard involved mixing 3 tablespoons of cornstarch (Edmonds, NZ), 3 tablespoons of sugar (Homebrand, NZ), 1 teaspoon vanilla essence (Hansells, NZ), ¼ teaspoon of yellow food colouring (Hansells, NZ) and 500 mL of non-fat milk (Homebrand, NZ). The mixture was heated in a microwave (4 minutes, stirred, 3 minutes, stirred) and homogenised. Coconut oil (Blue Coconut, Canterbury, NZ) was added to each bowl at 0%, 5%, 10%, and 15% quantities and then custard added to give a total weight of 50 g. Each bowl was thoroughly and consistently mixed. Smaller portion cups (35 mL cups) were labelled with individual 3-digit codes and filled with 20 g of custard and then refrigerated. Testing stimuli were made approximately 12 hours prior to taste testing (see Table 1 for details of methodology).

Subjective evaluation of vanilla custard was recorded on paper by placing a vertical line through 150 mm linear scales. The questionnaire and overall evaluation used to assess the vanilla custard (real world food model) was similar to that of consumer sensory evaluation techniques (Haryono et al., 2014; Popper, 2014). At each fat concentration level, a separate questionnaire was administered, with no side-by-side comparisons. Participants were not informed of the fat content levels and were untrained, as we were looking for naïve ratings of attributes and ratings of hedonic liking associated with a real world food model (Ares, Barreiro, & Giménez, 2009). Prior to the assessment, the participant was told that there were no ‘right or wrong’ answers. On each visit, participants evaluated two out of the four custard stimuli. These were selected in a randomised order so that all four custard stimuli were tested at the first two visits. The questions asked related to liking (“How much do you like or dislike the aroma / taste / mouthfeel / sweetness of the vanilla custard?”), ideal preferences (“Compared to your ideal vanilla custard, what do you think of the aroma / flavour strength / mouthfeel / sweetness?”), overall like or dislike (“Overall, how much do you like or dislike this

vanilla custard?"), fat content ("How would you rate the fat content level?"), and fat taste intensity ("How would you rate the intensity of the fat taste?)" (Keller et al., 2012; Martínez-Ruiz et al., 2014). The scales were anchored at either end by statements; "Strongest imaginable dislike / Too weak / Not enough flavour / Too dry / Not sweet enough / Very low fat content (The lowest I have ever tasted in custard)/ Very low fat taste" on the left and "Strongest imaginable like / Too strong / Too much flavour / Too fatty oily / Too sweet / Very high fat content (The highest I have ever tasted in custard / Very high fat taste" on the right. The final question was to circle the % of fat thought to be present in the vanilla custard stimuli from a range of options (0%, 1%, 2%, 5%, 10% or 15%).

3.3.3. Eating behaviour and dietary intake questionnaires

Eating behaviour was assessed using the validated three-factor eating questionnaire (TFEQ) to measure cognitive dietary restraint (21 items), disinhibition of control (16 items) and susceptibility to hunger (14 items) (Stunkard & Messick, 1985). Each item scores either 0 or 1 point with possible scores ranging from 0-0-0 to 21-16-14 (Stunkard & Messick, 1985). TFEQ scores were allocated for each category and associated subscales were calculated under each of the three factors (Bond, McDowell, & Wilkinson, 2001; Westenhoefer, 1991; Westenhoefer, Stunkard, & Pudel, 1999), where higher scores denote higher levels of restrained eating, disinhibited eating and predisposition to hunger (Stunkard & Messick, 1985).

Dietary intake (energy, macronutrients, food groups) was measured by a 220-item food frequency questionnaire (FFQ) developed for the Women's EXPLORE study ("EXamining Predictors Linking Obesity Related Elements") and adapted from the FFQ used in the National Nutrition Survey in NZ (Houston, 2014; Kruger et al., 2015; Quigley & Watts, 1997; Russell, Parnell, & Wilson, 1997). Approximate frequency of food and beverage intake was for items consumed over the previous month. Dietary data from the FFQ were combined into key food groups as recommended by the Eating and Activity Guidelines for NZ Adults of i. fruit, ii. vegetables, iii. grains, iv. milk and milk products, v. nuts, nut spreads and seeds, vi. eggs, poultry and fish, vii, red meat, viii,

takeaways, ix, sugary treats, x. butter, margarine and oil, xi. legumes and xii. alcohol (Ministry of Health. & Health, 2015). Dietary intake data are expressed as daily frequency equivalent's (DFE's) where frequency responses were calculated as a per day value (i.e. '4-6x/week' was calculated as 5 / 7 days = 0.71 DFE's (da Silva, Sichieri, Pereira, da Silva, & Ferreira, 2013). Data from the FFQ and was entered into Foodworks version 7 (Xyris Software 2012, Queensland, Australia). Foodworks uses the NZ Food Composition Database and FOODfiles (New Zealand Institute for Plant and Food Research, 2011) to determine total energy (kJ) and macronutrient (g) intake. In-depth analysis of the dietary intake data collected in this study is reported elsewhere (Henderson, 2016).

3.3.4. Testing procedure

Each participant attended three testing sessions which took place on nonconsecutive days within a period of one month. During all three testing sessions, oleic acid taste and olfaction threshold testing was conducted. Participants were asked not to eat, drink, wear perfume, or taste any other products prior to testing and on arrival had fasted for approximately 12 hours. Testing took place between 0700 and 0930 hours. All stimuli were evaluated at room temperature (20°C) in individual taste testing booths. Tasting stimuli were whole mouth samples which were evaluated using a sip-and-spit procedure with no solution ingested (Mattes, 2009). Participants were asked to wear a nose clip for the taste evaluation only. Prior to sensory testing, a short training procedure was conducted to familiarise each participant with the unique taste of the oleic acid solution (9.8 mM) while wearing a nose-clip and to compare this taste to that of a blank milk emulsion (R. Keast, personal communication, June 2015). If the participant failed to notice any difference between the target (9.8 mM) and the blank, they were asked to try again with higher concentrations, until the participant recognised the taste. During the testing procedure, participants rinsed out their mouth with water between each set of tasting stimuli. Oleic acid taste perception testing was conducted first (wearing nose-clip), followed by oleic acid olfactory testing (nose-clip removed). Red lights were left on throughout the tasting, olfactory and custard stimuli evaluation. To finish, evaluation of vanilla custard tasting stimuli was conducted (visit 1 and 2) or the *n*-butanol

threshold test (visit 3). All testing took approximately an hour to complete. The TFEQ and FFQ were answered in one sitting on SurveyMonkey™ in-between study visits in a location where participants had minimal distraction.

3.3.5. Statistical analysis

Statistical analyses were performed using IBM SPSS software for Windows (version 22.0; SPSS Inc, Armonk, NY: IBM Corp, USA). The sample size calculation was performed based on a pilot study which measured linoleic acid fatty acid taste threshold. It was estimated that 50 participants were required based on alpha risk at 0.05 and beta risk at 0.2 (power 80%) to find a difference in oleic acid taste perception between concentration levels. The sample size is similar to other studies investigating oleic acid taste perception (Stewart et al., 2010; Tucker, Edlinger, Craig, & Mattes, 2014; Zhou et al., 2016). Binomial regression models were performed using R version 3.2.5. Data were checked for normality using the Kolmogorov-Smirnov test. Normally distributed data are presented as mean±SD and non-normally distributed data as median [25, 75 percentiles]. Correlations of fatty acid taste detection, *n*-butanol detection, oleic acid olfaction and dietary intake were tested using Pearson's correlation coefficient and Spearman's correlation coefficient depending on the normality of the data. Intra-class correlation (ICC) using two-way random effects model, single measures was performed to detect associations between fatty acid detection thresholds and proportion of correct trials across testing days. The fitted fatty acid taste models of the probability of correct detection used binomial regression to model the success or failure of taste and olfaction detection. Binomial regression employs a link function to connect the binary outcome variable with the continuous predictor variables. The link functions that gave the most stable fit to each data set were applied, these were the logit link function for the olfaction data, and the complementary-log-log function for the taste data at all three testing sessions (Lawless, 2010). The rationale to interpolate at 0.66 probability was based on the publication by Lawless, 2010 (Lawless, 2010). Lawless (2010) describes an alternative analysis of forced-choice threshold data sets (ASTM, 2011; Lawless, 2010). The interpolation of chance-corrected thresholds does not discount correct responses of early concentrations but does take into account the probability that

guessing correctly may have. This analysis does not exhibit the downward bias that can occur from correct guessing and has proven practical applications (Lawless, 2010).

The probability of guessing would be 0.33 or 1/3 correct trials. Therefore, the probability of 0.66 or 2/3 correct samples ensures that the detection rate is above "chance" level. By using a single 0.66 probability cut-off for all participants we were able to assess their sensitivity at the same point identified by the binomial regression model. A similar approach has been used by Giguère et al, 2016 and Jayasinghe et al, 2017 (Giguère et al., 2016; Jayasinghe et al., 2017). The probability of 0.66 differentiated between participants at a common intercept and is a chance-corrected detection rate.

Sensitivity to oleic acid was treated as a grouping variable and was defined as 'hypersensitive' or 'hyposensitive' to taste similar to previous publications (Stewart et al., 2010; Stewart, Feinle-Bisset, & Keast, 2011). For this purpose, we chose the cut-off of 5.7 mM as this was the median detection threshold on average, across the three days. Group differences between taste detection rate, taste detection threshold, olfactory scores, eating behaviour scores, energy intake, macronutrient intake, food group intake and mouthfeel ratings (continuous variables) were investigated using independent samples t-tests and Mann-Whitney U test for non-normally distributed data. BMI categories and taste sensitivity (hypersensitive versus hyposensitive) were compared using a chi-squared test. A P value of <0.05 was considered as significant.

3.4. Results

3.4.1. Participants

A total of fifty-one female NZ European participants were recruited to take part in the study (Table 3.2). Of these, 50 women completed all three required visitations. The majority of the participants were of normal weight (BMI between 18.5 and 24.9 kg/m^2; 62%), with some overweight (BMI 25-29.9 kg/m^2; 22%) and some obese (BMI≥30 kg/m^2; 16%) participants.

Table 3.2. Anthropometric characteristics of participants

Variable	All (n=50)
Age (y)[1]	26 [22, 32]
Height (cm)[2]	166 ± 6
Weight (kg) [1]	67 [57, 76]
BMI (kg/m²)[1]	24 [21, 28]
PBF (%)[2]	30 ± 8

Abbreviations: y, years; PBF, percentage body fat; SD, standard deviation. [1]Median [25th-75thquartiles]; [2]Values are means±SD.

3.4.2. Taste and olfaction detection curves of oleic acid

Detection curves were modelled from the data obtained from all three sessions in order to interpolate the detection rate of oleic acid taste perception for each individual. Each taste trial, which comprised a set of three samples containing two controls and one oleic acid 'target' sample, was used to create a binomial regression model (incorrect vs. correct identification per 3-AFC set). Figure 3.2a shows the taste detection curves[i] of

[i] Detection curves: concentration of taste stimulus (mM) vs. the probability of correctly identifying taste.

oleic acid using the probability[ii] of correctly identifying a taste stimulus at each trial. Each line represents an individual participant showing that detection ability increases with increasing concentration of oleic acid.

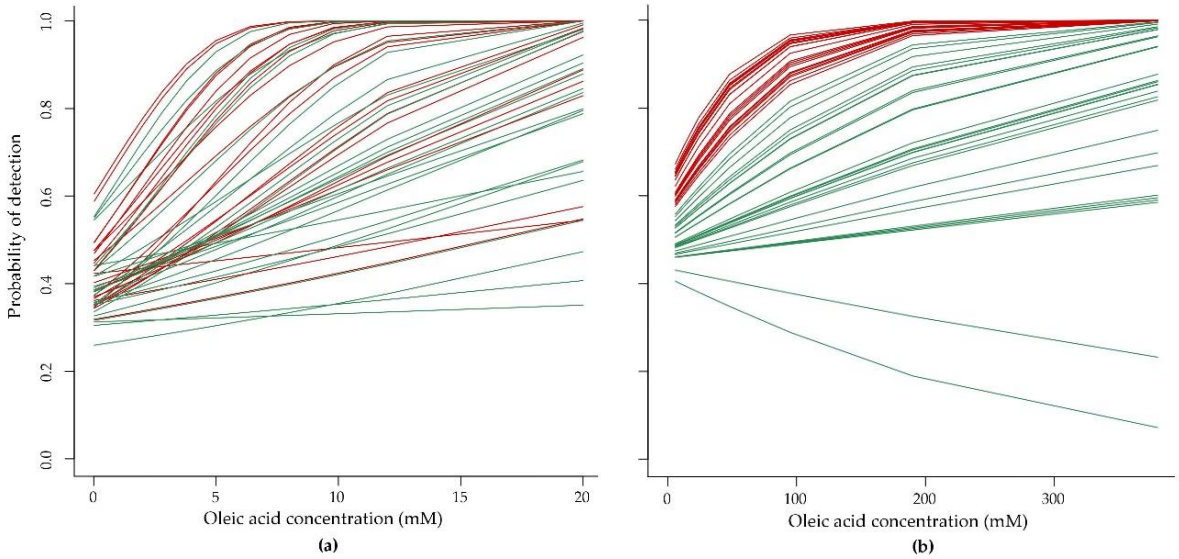

Figure 3.2. Comparison of (a) taste detection curves and (b) olfactory detection curves of oleic acid (n=50). Participants marked in red showed strong olfactory detection rate (b) and those same participants are shown in the taste model also in red (a). The fitted models of binomial regression for taste and olfaction show successful vs. failed individual trials across all 3 testing days (3-AFC) modelled with a link function.

We evaluated between session repeatability of the probability of detection (taste) for each participant, where moderate significant correlations were found across all testing sessions 1, 2 and 3 (ICC=0.52, CI=0.36-0.67, $P< 0.001$, two-way random effects model, single measures). When comparing side-by-side sessions '1 and 2', and '2 and 3', repeatability was stronger, showing significant moderate correlations (ICC=0.67, CI=0.48-0.80, $P< 0.001$ and ICC=0.59, CI=0.38-0.75, $P< 0.001$, respectively). Detection curves for taste were variable across the group of participants but significantly repeatable within an individual's data sets across sessions 1, 2 and 3. In addition, there were no significant differences between taste detection thresholds across the three sessions.

[ii] Probability of detection: the probability of a trial being correctly identified at each concentration (3-AFC method). One in three trials will be correctly identified by chance alone (0.333, or 33.3%).

Figure 3.2b shows the olfactory detection curves using the probability of correctly identifying oleic acid odour at each trial. Olfactory detection curves increased with higher concentrations of oleic acid (data obtained from all three sessions). When evaluating the repeatability of the probability of detection for oleic acid odour, moderate correlations were found across visits for sessions 1, 2 and 3 (ICC=0.41, CI=0.23-0.58, $P< 0.001$, two-way random effects model, single measures). When comparing side-by-side sessions '1 and 2', and '2 and 3', olfactory repeatability was consistent with comparing all three sessions showing significant moderate correlations (ICC=0.44, CI=0.19-0.64, $P< 0.001$ and ICC=0.39, CI=0.13-0.6, $P< 0.002$, respectively). There were no significant differences between olfactory detection thresholds across the three sessions.

3.4.3. Fatty acid taste hypo- and hypersensitivity

Previous studies have defined oleic acid taste hypersensitivity empirically as a detection threshold[iii] at a concentration of less than 3.8 mM (Haryono et al., 2014). In this current study, we used a model to determine taste detection rate. Interpolation of each detection curve was required to characterise participants as hypo- or hypersensitive to oleic acid taste perception. In order to create an equivalent classification to previous studies (Haryono et al., 2014; Keast et al., 2014; Stewart, Newman, et al., 2011), the concentration (mM) at which detection is 0.66 (66%; equivalent to successfully obtaining 2 out of 3 correct trials at that given concentration) was considered to be the detection rate[iv]. Taste hypersensitive participants (n=22) were defined as individuals who obtained a detection rate of less than or equal to 5.7 mM at 0.66 (66%) of the trials, based on their taste detection curve.

[iii] Detection threshold: the lowest concentration at which a stimulus is detected, determined by 3 consecutively correct taste trials at that given concentration (3-AFC method).

[iv] Detection rate: concentration of stimulus at which an individual would correctly identify 2 out of 3 (0.66 or 66%) of trials, using the predictive detection rate curves.

Table 3.3. Comparison of median [25th – 75th quartiles] detection rate and detection threshold of hypersensitive and hyposensitive fatty acid detection (oleic acid) taste groups.

Variable	Hypersensitive (n=22)	Hyposensitive (n=28)	*P*-value
Detection rate[1]	3.36 mM [2.14, 5.53]	12.12 mM [8.91, 19.37]	<0.001
Detection threshold[2] (Haryono et al., 2014; Keast et al., 2014; Stewart, Newman, et al., 2011)	2.58 mM [1.47, 3.35]	11.10 mM [6.07, 12.73]	<0.001

[1]Detection rate: concentration of stimulus at which an individual would correctly identify 2 out of 3 (0.66 or 66%) of trials, using the predictive detection rate curves; [2]Detection threshold: the lowest concentration at which a stimulus is detected, determined by 3 consecutively correct taste trials at that given concentration (3-AFC method).

3.4.4. Relationship between oleic acid taste perception and olfaction

A significant, positive correlation between taste probability of detection and the olfactory probability of detection (r=0.325, n=50, P< 0.02) of oleic acid was found (Figure 3.3).

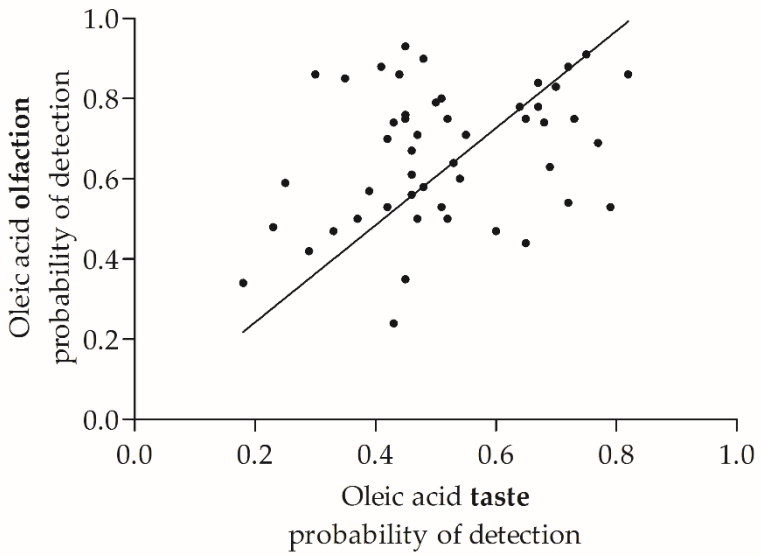

Figure 3.3. Scatterplot of the relationship between oleic acid olfaction and taste (probability of detection), the weighted average across all three sessions.

3.4.5. Relationship between oleic acid taste hypo- and hypersensitivity, olfaction detection rate, n-butanol olfactory threshold and eating behaviour

Taste hypersensitive participants had a lower olfactory detection rate (higher sensitivity) than the hyposensitive taster group (Table 3.4, $P< 0.05$). Scores obtained for *n*-butanol threshold demonstrated a similar relationship to oleic acid olfactory detection rate, taste hypersensitive participants obtained a higher mean score (higher sensitivity) for *n*-butanol threshold 'Sniffin' Sticks' ($P< 0.03$). The mean score for *n*-butanol threshold was 8.7±2.2. Based on normative values, 43 participants were classified as normosmic (test score >6.5), and 7 as hyposmic to odour (test score <6.5, less sensitive). There was a trend for the olfactory oleic acid detection rate (mM) to correlate with *n*-butanol threshold score ($r_s= -0.263$, $P= 0.07$).

The three eating behaviours assessed by the TFEQ were cognitive restraint, disinhibition and hunger as well as associated subscales. Results from the questionnaire were analysed based on scoring criteria established by Stunkard and Messick (1985). For cognitive restraint, the majority of participants (68%) reported low scores (0-10 out of a possible score of 21). Participants also reported mostly low scores for disinhibition (74% scored 0-8 out of 16). For susceptibility to hunger, 80% of participants scored low (0-7 out of 14) (Shen, Kennedy, & Methven, 2017). A significant difference in disinhibition score and emotional disinhibition (subscale) was observed between hypersensitive and hyposensitive groups ($P< 0.05$; $P< 0.03$, respectively). There were no significant correlations between eating behaviour factors when compared to oleic acid olfactory detection or *n*-butanol threshold (all, $P> 0.05$).

Table 3.4. Comparison of TFEQ scores and olfactory detection for hyper- and hyposensitive fatty acid detection (oleic acid) taste groups.

	Hypersensitive (n=22)	**Hyposensitive (n=28)**	**TOTAL (n=50)**	***P*-value**
Oleic acid olfactory detection rate[1] (mM)[2]	24.2 [11, 61]	97.3 [24, 181]	45.4 [16, 158]	**0.041**[4]
n-butanol threshold score[3]	9.5±1.8	8.1±2.3	8.7±2.2	**0.029**[4]
Cognitive dietary restraint[2]	8.0 [4, 11]	10 [7, 12]	9.0 [5, 11]	0.232
Flexible restraint[2]	3.0 [1, 4]	3.5 [2, 5]	3.0 [1.8, 4]	0.159
Rigid restraint[2]	2.0 [1, 3]	3.0 [1.5, 4]	3.0 [1, 4]	0.133
Disinhibition[2]	4.0 [3, 6]	6.5 [3, 10]	5.0 [3, 9]	**0.046**[4]
Habitual susceptibility[2]	0.0 [0, 1]	0.5 [0, 2]	0.0 [0, 1]	0.197
Emotional susceptibility[2]	0.0 [0, 1]	2.0 [0, 3]	1.0 [0, 2]	**0.029**[4]
Situational susceptibility[2]	2.0 [2, 4]	3.0 [1, 4]	3.0 [1, 4]	0.538
Hunger[2]	3.5 [2, 6]	4.0 [2, 7.5]	4.0 [2, 6.3]	0.313
Internal locus[2]	2.0 [0, 3]	2.0 [1, 3]	2.0 [0, 3]	0.638
External locus[2]	1.0 [0, 2]	2.0 [1, 4]	1.5 [0.8, 3]	0.125

Abbreviations: TFEQ, three-factor eating questionnaire; mM, millimolar; SD, standard deviation. [1]Detection rate (mM), defined as the concentration at which correct detection is 0.66 (66% correct trials over 3 days) using the odour detection curves; [2]Median [25th-75th quartiles]; [3]Values are means±SD; [4]Significant difference found between hypersensitive and hyposensitive taste groups ($P< 0.05$).

3.4.6. Relationship between oleic acid taste hypo- and hypersensitivity, mouthfeel rating and olfaction

Distinct from fatty acid taste is the ability to feel the texture of fat in food or drinks in the mouth, which are the tactile sensations that can be described as ‘creamy’ or ‘oily’ (Vingerhoeds et al., 2008). Significant differences were found between hyposensitive and hypersensitive participants when asked to subjectively rate how much they liked the mouthfeel of high-fat custard ($P< 0.05$) and when rating the mouthfeel of the medium fat custard in comparison to what they perceived as an ideal level of fat content ($P< 0.05$) (see Figure 3.4). Additional vanilla custard rating questions (e.g. sweetness intensity, flavour liking, etc.) were not significantly different between hyper- and hyposensitive taste groups (all, $P> 0.05$).

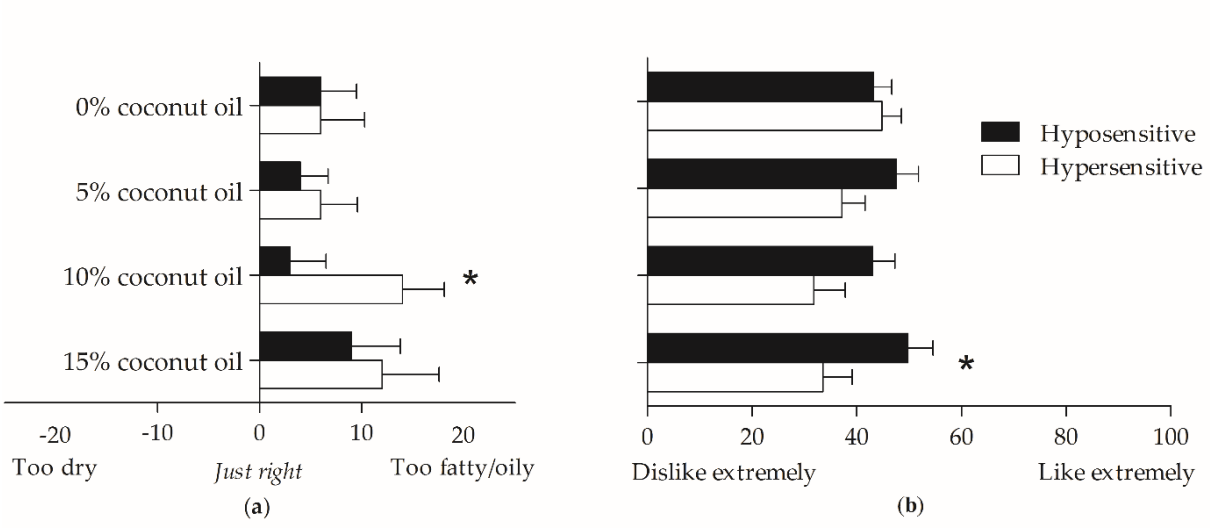

Figure 3.4. Comparison of (a) mouthfeel rating and (b) mouthfeel liking of high (15% coconut oil), medium (10% coconut oil), low (5% coconut oil) and no fat custard (0% coconut oil) split by taste hypersensitive (n=22) and hyposensitive (n=28) participants. Data presented as mean±sem. *P< 0.05.

Mouthfeel perception of high-fat custard (15% coconut oil) was correlated with *n*-butanol sensitivity, where a rating of 'too fatty/oily' being associated with higher olfactory sensitivity (r=0.393, P< 0.01). No other noteworthy significant associations were found between the mouthfeel ratings of custard, *n*-butanol threshold or oleic acid olfactory threshold.

3.4.7. Relationships between oleic acid taste perception, oleic acid olfaction, dietary intake, mouthfeel rating and eating behaviour

In the current study, food group intake was measured, and energy intake evaluated across the study population. An in-depth analysis of dietary patterns and food group data is reported elsewhere (Henderson, 2016). Nuts, nut spreads and seeds food group intake (DFE's) was significantly correlated with oleic acid olfactory detection rate (mM), where high sensitivity (low detection rate) correlated with higher intake of nuts, nut spreads and seeds (r_s= -0.410, P< 0.01). We found no other significant relationships between oleic acid olfactory detection rate, *n*-butanol threshold or oleic acid taste

perception and food group intakes, energy and macronutrient intake. Additionally, there were no significant differences in food groups, energy or macronutrient intake between hypo- and hypersensitive taste groups.

Restraint and hunger eating behaviour factors were related to intake of specific food groups in our sample population. A high hunger score was correlated with higher intake of takeaways (r_s= 0.33, P< 0.02) and butter, margarine and oil (r_s= 0.32, P< 0.03). High sugary treat food intake was inversely associated with restraint (r_s= -0.39, P< 0.01). Vegetable intake was positively correlated with restraint score (r_s= 0.32, P< 0.03) and negatively with hunger score (r_s= -0.31, P< 0.03). No other significant relationships between eating behaviour, food group intake, energy or macronutrient intakes were found.

3.4.8. Oleic acid taste perception and olfaction detection rate and body composition

Oleic acid taste hypersensitive participants were significantly more likely to have a low BMI (be lean) (X^2 (1, n=50) =3.89, P< 0.05) and hyposensitive participants were 3.4 times more likely to be overweight or obese than hypersensitive participants. There was a trend for hypersensitive participants to have a lower PBF than hyposensitive participants (27.8%±7.2 vs. 32.2%±8.8; P= 0.06). There were no relationships found between oleic acid olfactory detection rate and BMI as a continuous variable or between BMI categories or percentage body fat. There were no significant differences in oleic acid olfactory detection rate, oleic acid mouthfeel perception, *n*-butanol threshold, food group intake, energy, macronutrient intakes or eating behaviour between BMI categories.

3.5. Discussion

The present study investigated the relationship between oleic acid taste and olfaction detection rates, and how these measurements may relate to dietary intake, eating behaviour, mouthfeel ratings of fat added to test custard and body composition. The results show that sensitivity to oleic acid taste perception and olfaction varies considerably between participants, with individual detection rates covering three orders of magnitude. The present study shows for the first time that fatty acid olfactory sensitivity is clearly linked with fatty acid taste sensitivity albeit acting through separate pathways. Hyposensitivity to fatty acid taste was associated with disinhibited eating behaviour. Furthermore, participants who were hypersensitive to oleic acid taste perception had lower BMI values than those who were hyposensitive. The findings of this study show remarkable parallels in fatty acid taste and olfaction detection rates with clear and consistent individual differences in detection ability. These individual differences in fat detection appear to be linked with disinhibited eating behaviour that may have implications for long-term metabolic health outcomes (Hays & Roberts, 2008).

3.5.1. Oleic acid taste and olfactory detection rate

In this study, we created tailored models to characterise the detection curves of taste and olfaction with increasing oleic acid concentrations in healthy women. Our data confirm that there is great variability in taste sensitivity between individuals, which is consistent with previous studies in presenting a range of taste detection thresholds across different participants (Chalé-Rush et al., 2007a; Stewart et al., 2010; Zhou et al., 2016). In humans, sensing of ‘fat’ has been attributed to CD36 receptors in taste cells, as well as GPR120, 41, 40 and 43 receptors (Baillie, Coburn, & Abumrad, 1996; Galindo et al., 2012; Pepino, Kuda, Samovski, & Abumrad, 2014). It is thought that CD36 receptors may function in fatty acid recognition at low concentrations, whereas GPR120 may be functioning at higher concentrations, acting to enhance the signalling of fatty acids and

providing sustained taste experiences (Ozdener et al., 2014). Thus, oral detection of fatty acids may be a result of dual, complementary mechanisms (Besnard, Passilly-Degrace, & Khan, 2016). We, therefore, chose to identify the probability of oleic acid taste detection over a wider range of concentrations in the present study, given that fatty acid taste may be detected by multiple receptors and a range of transduction pathways (Abdoul-Azize, Selvakumar, Sadou, Besnard, & Khan, 2014; Besnard et al., 2016; Gilbertson & Khan, 2014; Liu et al., 2016; Ozdener et al., 2014).

In consideration of a postulated multiple receptor mechanism that detects fatty acids, we chose to extend the ascending 3-AFC method to continue testing past three correct evaluations, by adding an additional three higher concentrations past the commonly used 'stopping point' (Mattes, 2007). An extension of the stopping rule was further implemented to collect enough data points across the three repeated sessions to create the binomial regression models. We were then able to interpolate an individual's performance using the model and from this, we were able to classify individuals as hypo- or hypersensitive as a grouping variable. This approach decreases the number of false-positives which can occur through guessing the correct solution by chance alone (Running, 2014). The extension of the stopping rule further enhanced the quality of our data by broadening the range of concentration levels evaluated, which allowed us to model taste behaviour for each participant. One of the limitations of extending the procedure is inducement of fatigue, but the integration of the probability of correct detection at each concentration level obtained from multiple visits decreases the influence of this effect on the detection rate (Mattes, 2007; Running, 2014). The fatty acid detection rates applied in this study account for the possibility of guessing correctly but do not discount the correct responses which may occur at low concentrations. The between-participant variance was further reduced in this study by limiting our participants to the same gender, age range (premenopausal only) and to one ethnic group.

3.5.2. Fatty acid taste hypo- and hypersensitivity

We were able to identify a detection rate for all participants to then further establish our classification into hypo- or hypersensitive fatty acid taster groups, based on their performance across three days of testing, as opposed to a single session measurement. The ratio of participants classified as hypersensitive in our study, based on their detection rate, was comparable to findings in previous studies (Keast et al., 2014; Stewart, Newman, et al., 2011). The repeatability of fatty acid taste threshold assessments has been investigated previously (Newman & Keast, 2013; Tucker & Mattes, 2013), and consistent with these studies, we found significant repeatability across all sessions. Whilst we found that fatty acid taste detection was clearly repeatable, we would recommend a minimum of three testing sessions to measure fatty acid taste or olfaction detection rates, in order to obtain enough data to determine the probability of correctly identifying taste at each concentration level (Newman & Keast, 2013; Tucker et al., 2014; Tucker & Mattes, 2013). In the present study, all sessions were conducted in the morning, prior to consuming breakfast, which we believe enhanced the repeatability of the taste perception data in this study.

3.5.3. Relationship between oleic acid taste perception and olfaction

Our results show that the ability to detect fatty acid by taste was significantly associated with that of olfaction. In a 'real-world' food setting, the recognition of fatty acid taste would be further enhanced by mastication behaviour (chewing), due to the enhanced release of organic odour volatiles (Ployon, Morzel, & Canon, 2017). In support of a fatty acid taste and olfactory relationship, the previous research identified that the expression of the CD36 receptors in the olfactory epithelium may be related to the long-chain fatty acid taste receptor mechanisms (Xavier et al., 2016). There is a possible role in odorant detection by this scavenger receptor (Xavier et al., 2016), suggesting individuals with higher CD36 taste expression potentially have a homologous olfactory detection ability. This has been observed in CD36-deficient mice who displayed altered olfactory behaviour when exposed to long-chain fatty acids (Xavier et al., 2016). In support of our findings, *in vitro* work on human olfactory mucus has found an odorant binding protein which has a strong affinity for long-chain fatty acids, including lauric acid and capric acids (Xavier et al., 2016). Additionally, the variability in a human

odorant-binding protein OBPIIa was associated with individual differences in the bitterness perception of oleic acid (Abdoul-Azize et al., 2014). It would be interesting to investigate in future studies whether individuals who are more sensitive to oleic acid olfaction are carriers of the variation in the olfactory binding protein described by Tomassini Barbarossa et al. (Tomassini Barbarossa et al., 2017). Interestingly, in humans, olfactory-based discrimination of the fat content of milk and specific fatty acids at a range of concentration levels has been demonstrated and supports the notion that humans are able to detect small differences in fat content by odour alone (Boesveldt & Lundström, 2014; Kallas & Halpern, 2011).

In comparison to the oleic acid tasting procedure in this study, the concentration of oleic acid used in the olfactory detection tests went to a considerably higher concentration. This was required due to the stimuli being tested orthonasally, at room temperature (20°C) with fresh oleic acid in partially filled bottles to generate an open headspace for inhalation. Because this is a new procedure we covered a wider range of concentrations but used fewer steps (seven concentrations) as we wanted to avoid adaptation effects which have been reported in some previous olfactory studies (Stuck, Fadel, Hummel, & Sommer, 2014; Thompson & Spencer, 1966).

Our results indicate that there is a difference between hypo- and hypersensitive groups in sensitivity to *n*-butanol odour ('Sniffin' Sticks' score). The *n*-butanol odour sensitivity test is widely used for the evaluation of human olfactory performance and can be used by medical practitioners to assess olfactory dysfunction (Denzer et al., 2014). In the present study, the *n*-butanol threshold test was incorporated to see if a well-established olfactory assessment method may relate to the oleic acid olfactory test introduced in this study. We found that *n*-butanol sensitivity is weakly associated with oleic acid olfactory perception.

3.5.4. Oleic acid taste perception and disinhibited eating behaviour

Significant associations were found between oleic acid taste sensitivity and 'disinhibition' and the eating behaviour sub-category 'emotional disinhibition', where

higher disinhibition scores were obtained by the oleic acid taste hyposensitive group. Disinhibition refers to opportunistic eating behaviour, which could play a role in weight gain (Bryant, King, & Blundell, 2008). Previous studies have found emotional disinhibition significantly predicts body fat percentage in young NZ women (Kruger, De Bray, Beck, Conlon, & Stonehouse, 2016), and another study in a young French cohort found higher disinhibition scores were associated with a higher BMI (Lesdéma et al., 2012). It has been suggested that an inability to detect fat efficiently may result in compensation of cognitive satisfaction with other tastes, such as 'sweet' (Asano et al., 2016) which may account for additional weight gain over time (Newman, Haryono, & Keast, 2013; Rolls, 2007). Eating behaviour and fatty acid taste sensitivity have not been directly compared in previous studies (Liang et al., 2012; Panek-Scarborough et al., 2012; Shen et al., 2017). This study is the first to report a relationship between fatty acid taste sensitivity and disinhibited eating behaviour.

3.5.5. Oleic acid taste perception and mouthfeel

The subjectively rated mouthfeel of fat in the food matrix in the present study varied between hypo- and hypersensitive participants. Given the textural properties of fat, it was important to investigate whether fatty acid taste sensitivity (which is independent of texture or mouthfeel) can be compared to the liking of 'real-world' foods. In this study, we found that oleic acid taste hypersensitivity was significantly related to increased rating of 'oily/fatty' mouthfeel perception, as well as negatively impacting the hedonic liking of a high-fat product. The hedonic liking of fat textural attributes in comparison to fatty acid taste sensitivity was of interest, as it is well known that commercial products with 'fat replacers' often fail to attain the craving response of a full fat equivalent (Jervis, Gerard, Drake, Lopetcharat, & Drake, 2014). This study provides further evidence that fatty acid taste itself (i.e. the presence of fatty acid ligands), in addition to mouthfeel, plays a critical role in the recognition and perception of fat. It is recognised that the presence of fatty acid ligands are critical throughout the digestion process as receptors in the gut are considered to be homologous with oral taste receptors, which may further support an individual's satiety response (Shin, Ingram, McGill, & Poppitt, 2013). In animal studies, it has been shown that consumption of a

high-fat diet related to an increase in CD36 mRNA expression (Primeaux, Braymer, & Bray, 2013). CD36 mRNA expression was found to occur on circumvallate papillae as well as duodenal enterocytes, supporting the possibility that there is complementary sensing of long-chain fatty acids in the two different regions (Primeaux et al., 2013). Receptors isolated from human intestinal enteroendocrine cells include CD36 and G-protein coupled receptors (GPR120, GPR40) (Rasoamanana, Darcel, Fromentin, & Tomé, 2012). The CD36 protein, in particular, is expressed in the duodenum and jejunum and has been proposed to play a role in signaling pathways that mediate fatty acid detection in the gut (Rasoamanana et al., 2012; Shin et al., 2013).

3.5.6. Oleic acid taste perception, olfaction and body composition

The results of the present study showed that participants who were hypersensitive to oleic acid taste had a significantly lower BMI, a finding consistent with previous studies on oleic acid taste perception (Asano et al., 2016; Proserpio, Laureati, Bertoli, Battezzati, & Pagliarini, 2016; Stewart et al., 2010; Stewart, Newman, et al., 2011) and linoleic acid taste perception (Martínez-Ruiz et al., 2014). Positive correlations have also been found between fat preference scores and percent body fat estimates (Mela & Sacchetti, 1991). A comprehensive review by Cox et al. concluded that low sensitivity to fatty acid taste, as well as liking and preference for fat, is related to higher weight status (Cox et al., 2015). However, not all studies have found an association between taste sensitivity and BMI (Chevrot et al., 2014; Tucker, Nuessle, Garneau, Smutzer, & Mattes, 2015; Zhou et al., 2016). A recent meta-analysis of studies on taste sensitivity has concluded that fatty acid taste sensitivity does not precede or result in obesity (Tucker et al., 2017). Of particular interest are studies which have found that a low-fat diet or a high-fat diet can modulate taste sensitivity, where a low-fat diet was shown to significantly increase taste sensitivity to oleic acid over a four-week period while there was no significant difference in sensitivity at baseline (Stewart & Keast, 2012). In support of this, a six-week, low-fat dietary intervention study in overweight and obese participants showed that fatty acid taste sensitivity can be enhanced significantly during this time period (Newman, Bolhuis, Torres, & Keast, 2016). These studies support the notion that fatty acid taste sensitivity can be related to body fat mass in some settings.

Our results did not suggest any direct relationship between oleic acid olfactory sensitivity and body composition. Interestingly, a report by Fernandez-Garcia et al (Fernandez-Garcia et al., 2017) suggests that olfactory function may be desensitised in response to changing levels of endocrine regulation in the obese state (Palouzier-Paulignan et al., 2012). Increased visceral body fat functions as an endocrine gland with increased secretion of adipokines (Fernandez-Garcia et al., 2017). Another recent study has found that a decreased sense of both taste and olfaction correlated with visceral fat rating (Fernandez-Garcia et al., 2017). However, in patients that have had gastric bypass surgery, olfactory function does not change, while in contrast taste sensitivity can improve (Richardson, Vanderwoude, Sudan, Leopold, & Thompson, 2012). Our data further suggests that overall olfactory sensitivity is not directly linked to eating behaviour or dietary intake. In support of this, a previous study found that milk odour discrimination performance was not related to BMI (Boesveldt & Lundström, 2014). Future studies focusing on sensory sensitivities across different BMI categories are required to explore the relationship between taste and odour sensitivity in conjunction with metabolic health status. It is important to note that body fat percentage values are clinically more relevant than BMI categories (Ho-Pham, Lai, Nguyen, & Nguyen, 2015). In the present study we obtained body fat percentage values from BIA measurements, however, it has been shown that typically a BIA will underestimate body fat percentage by 2% (von Hurst et al., 2015). We would recommend that future studies ascertain body fat percentage, ideally from air displacement plethysmography (ADP) or dual-energy X-ray absorptiometry (DXA) and compare these values to chemosensory perception.

3.5.7. Oleic acid taste perception, olfaction and dietary intake

In this study, no major associations were found between taste sensitivity and dietary intake. However, we did find a significant association between sensitivity to olfactory oleic acid and the intake of ‘nuts, nut spreads and seeds’. Olfaction has been identified as an important means for the interpretation of food flavours, and hedonic liking is due to the presence of odour volatiles released during the eating process (Ployon et al.,

2017). It is possible that participants who had a higher intake of nuts and seeds may have an increased ability to recognise the associated odours. To date, there has been no consensus about whether there is a relationship between fatty acid taste sensitivity and dietary intake. It is likely that discrepancies between studies are due to differences in the study participants (e.g. gender, ethnicity, age), assessment methods of fatty acid taste perception (psychophysical measurement, type of fatty acid stimulus) or dietary intake assessment methods which in turn generates inconsistencies about the potential biological or functional relationships (Cox et al., 2015; Zhou et al., 2016). The FFQ used in this study is a retrospective account of dietary intake, which was used to obtain individual energy (kJ), macronutrient (fat, protein, carbohydrate and saturated fat) and food group intakes. A recommendation for future studies would be to measure dietary intake with a four-day food diary directly prior to taste testing. Whilst all self-reported dietary intake assessments are influenced by under- or over-reporting (Gemming, Jiang, Swinburn, Utter, & Mhurchu, 2014), we consider the FFQ would be better used as a population tool for larger studies as opposed to individual comparisons to physiological mechanisms.

3.5.8. Additional strengths and limitations of this study

In this study, the sample size was powered for determining significant differences in oleic acid taste perception. The additional aspects investigated in the study (e.g. mouthfeel ratings, eating behaviour, BMI, etc.) were exploratory variables that were ancillary to the modelling of fatty acid taste perception. In order to extend any of the findings from this study to the wider population, an incorporation of additional participants and representative demographic groupings would be required.

3.5.9. Conclusions

Fatty acid taste detection mechanisms are complex and cannot be explained by a single receptor mechanism (Galindo et al., 2012). Therefore, the methodology chosen for this study optimised taste perception measurements by detection rate of a single fatty acid

across a broad range of concentrations, which modelled each participant's individual taste behaviour. The modelling of taste behaviour was based on the probability of correctly identifying the oleic acid taste at each concentration level, which was unique to this study and was a refined version of previously applied approaches. Furthermore, we were able to apply the same binomial regression model to olfactory detection which allowed us to compare the chemosensitivity of each sensory modality. This study is the first to report a link between fatty acid taste and olfaction sensitivity in humans. Furthermore, we drew conclusions about specific characteristics of disinhibited eating behaviour in hypo- and hypersensitive fatty acid taste groups which were determined from taste detection rate.

Although the ability to perceive fatty acid taste varied markedly between participants, the association between fatty acid taste perception and disinhibited eating behaviour observed in the present study suggests that fatty acid taste perception may influence dietary habits that have long-term metabolic health consequences. Additionally, sensitivity to fatty acid taste was related to body composition and hyposensitivity to oleic acid was clearly associated with a higher BMI. In conclusion, our study presents strong evidence for a link between oleic acid taste perception, olfactory perception and the mouthfeel perception of fat; suggesting there are intimate relationships between multiple modalities of fat sensation in humans. Further research is required to investigate whether there is a causal relationship between fatty acid taste perception and olfaction in the etiology of obesity, especially in an obesogenic environment of highly palatable energy-dense and nutrient-poor foods.

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3.6. References

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Chapter IV.

Taste perception and its association with adiposity and endocrine regulation in Pacific and New Zealand European women

4.1. Abstract

Taste perception may influence food consumption, which can affect long-term weight status and metabolic health. The aims of this study were to determine the association between measures of taste perception, body composition and hormonal adiposity signals (fasting insulin and leptin) in obese and non-obese women from Pacific and NZ European population groups. Participants (n = 351; aged 18 - 45) were recruited in Auckland, New Zealand (NZ). Taste perception was assessed by perceived suprathreshold intensity, hedonic liking, and discrimination of taste by a ranking task using a range of concentrations of sweet (glucose), bitter (quinine), and fat (milk fat) solutions. Participants were classified as likers or dislikers for each tastant using a hierarchical cluster analysis. Body composition was quantified by dual x-ray absorptiometry (DXA).

Women who incorrectly discriminated sweet taste by ranking task were nearly three times more likely to have higher body fat (BF) >35% (adjusted, $P< 0.01$). Cluster analysis revealed distinct patterns of liking for each tastant. NZ European sweet likers were twice as likely to have >35 BF% compared to sweet dislikers (adjusted, $P< 0.05$), however, this comparison was not significant in Pacific women. Conversely, bitter likers had a decreased likelihood of having >35 BF% in comparison to bitter dislikers (adjusted; OR 0.4, $P< 0.01$). Having higher fasting plasma leptin concentration significantly increased the likelihood of being a sweet liker in Pacific and NZ European

combined (adjusted; OR 1.7, $P< 0.05$), but in NZ European women, the likelihood of this was further increased (adjusted; OR 3.6, $P< 0.001$). Higher fasting plasma insulin concentration also increased the likelihood of being a sweet liker (adjusted, $P< 0.05$).

The present study has shown differences in sweet taste perception in relation to adiposity, which is further associated with circulating plasma leptin and plasma insulin concentrations.

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4.2. Introduction

Obesity is a worldwide health issue of epidemic proportion and interventions to halt the epidemic have been largely unsuccessful (WHO, 2018). Commonly-cited causes of obesity include the obesogenic food environment (Lenard & Berthoud, 2008) which has led to over-consumption of inexpensive, highly palatable, energy dense and nutrient poor foods. Foods high in caloric density with added sugar and fat, coupled with a low intake of vegetables and dietary fibre, are associated with weight gain (Swinburn et al., 2011).

The perception of the taste of food is a complex experience based on multiple senses including taste, olfaction and touch (mouthfeel), visual, and other sensory cues (Loper, La Sala, Dotson, & Steinle, 2015). Primary tastes include sweet, sour, salty, bitter, umami and the proposed fatty acid taste (Running, Craig, & Mattes, 2015). Taste detection is carried out by specific chemosensors located on taste buds found in fungiform, foliate and circumvallate papillae (Besnard, 2016). Stimulation of specific chemosensors by a tastant (e.g. glucose) triggers a signalling cascade leading to neurotransmitter release and these signals are subsequently transmitted by nerves to the brain (Besnard, 2016). Individual variation exists in both genotype and taste receptor mRNA expression (Lipchock, Mennella, Spielman, & Reed, 2013) and the cognitive processing of these taste signals (Hayes, Feeney, & Allen, 2013).

Associations between body composition and sweet or fat taste phenotype are of interest because sweet and fat are associated with foods of high energy density, which may contribute to increases in body weight and metabolic disease risk (Burgess, Rao, & Tepper, 2016; Dando, 2015). A range of studies have investigated the perception of sweet or fatty acid taste and have found significant associations between poor taste sensitivity and increased adiposity (Asano et al., 2016; Ettinger, Duizer, & Caldwell, 2012; Pepino, Finkbeiner, Beauchamp, & Mennella, 2010; Proserpio, Laureati, Bertoli, Battezzati, & Pagliarini, 2016). However, the majority of studies to date have not found a conclusive link between taste perception and obesity in human studies (Cox, Hendrie, & Carty, 2015). Genotypic variation in bitter taste receptors (e.g. T2R38) has been

linked to differences in bitter taste perception that influence food preference and intake (Chamoun et al., 2016). For example, increased bitter taste sensitivity has been shown to reduce the intake of healthier food choices, such as cruciferous vegetables (Keller & Adise, 2016). While there are inconsistent data about a proposed link between taste perception and obesity there is a dearth of convincing evidence of physiological mechanisms that may link taste perception and obesity in humans (Laffitte, Neiers, & Briand, 2014; Loper et al., 2015).

Obesity arises as a consequence of how the body regulates energy intake, expenditure and storage, and reflects a state of positive energy balance. The dual hormonal feedback system involving insulin and leptin, produced by the pancreas and adipose tissue respectively, are involved in long-term adiposity signals and the regulation of body weight as well as energy storage as fat (Kieffer & Habener, 2000). Insulin increases body fat mass, and stimulates the production and secretion of leptin, the satiety hormone, that acts centrally to reduce food intake and increase energy expenditure. Leptin in turn suppresses insulin secretion by both central actions and direct actions on the pancreas (D'souza, Neumann, Glavas, & Kieffer, 2017). Plasma levels of leptin are directly proportional to body fat mass. In a healthy physiological state, an increase of adiposity increases plasma leptin, thereby decreasing insulin production and reducing fat mass (Seufert, 2004). However, in the obese state dysregulation of the insulin-leptin feedback system, characterised by hyperinsulinemia and hyperleptinemia, leads to endocrine desensitisation (Owei, Umekwe, Provo, Wan, & Dagogo-Jack, 2017; Tchernof & Després, 2013). With advancing obesity, a decreased ability to metabolise lipid and default energy storage as adipose tissue occurs (D'souza et al., 2017; Kieffer & Habener, 2000). Furthermore, changes in the action of insulin and leptin disturb appetite regulation in the obese state, rendering sustained weight loss difficult to achieve (Amitani, Asakawa, Amitani, & Inui, 2013).

New evidence suggests an important mechanistic link between leptin and sweet taste perception. Yoshida et al. discovered that leptin specifically regulates sweet taste responses in taste buds, and they described a novel peripheral mechanism linking leptin receptors (Ob-Rb) to the suppression of sweet taste in mice. In humans, patterns of sweet taste recognition thresholds are correlated with circulating leptin levels in non-

obese subjects (Nakamura et al., 2008). These findings are further supported by a study which found associations between high salivary leptin levels and decreased sweet taste sensitivity in normal weight children, but not in obese children (Rodrigues et al., 2017). In addition, reduced taste sensitivity to sucrose has been associated with an increased salivary leptin level and higher carbohydrate intake (Han, Keast, & Roura, 2017). Similarly, Sanematsu et al. reported that in obese subjects increased plasma leptin levels may inhibit the modulation of sweet taste intensity (Sanematsu, Nakamura, Nomura, Shigemura, & Ninomiya, 2018). Leptin suppresses sweet taste via specific leptin receptors, but it does not suppress taste cell responses to bitter tasting compounds (Yoshida et al., 2015). Sweet and bitter taste are both mediated by G-protein coupled receptors and their function is associated with shared genes (Hwang et al., 2016). However, sweet and bitter tastes clearly have unique neural and behavioural responses which occur as a result of differentiated molecular pathways (Bufe et al., 2005).

Tasting sweet food stimulates insulin release prior to an increase in plasma glucose levels; this is known as the cephalic phase insulin release and is characterised by the initiation of insulin secretion before the post-absorptive rise in plasma glucose level (Power & Schulkin, 2008). Based on studies in mice, a link between the sweet taste receptor system and insulin has recently been proposed (Maruoka et al., 2015). Although functional insulin receptors have not been identified in taste cells of the tongue, there is increasing evidence that circulating insulin concentrations may influence sweet taste perception indirectly (Behrens & Meyerhof, 2019; Glendinning et al., 2015). These indirect influences of circulating insulin on taste perception may involve central or peripheral pathways. In an obese state, they may be linked with the changing leptin-feedback system and changes in insulin sensitivity and a rise in circulating glucose concentrations discussed above (Schwartz et al., 2017). Therefore, sweet taste perception may be modulated by the long-term adiposity signals, insulin and leptin, that influence energy balance, body weight and food intake (Blundell et al., 2010). The gustatory perception of fat is an interesting contrast to sweet and bitter taste perception, as fat is a key nutrient that is related to energy storage but the mechanistic pathways of fatty acid taste differ markedly from those of sweet and bitter taste (Mattes, 2011).

Underlying the biological mechanisms of taste perception are the diverse taste preferences which exist between different population groups due to genetic, cultural and socio-economic factors (Sable, Warren, DuFlo, Bartoshuk, & Skarulis, 2012; Williams, Bartoshuk, Fillingim, & Dotson, 2016). External influences on different population groups (level of income or socioeconomic setting) have also been shown to further explain metabolic disease risk with the highest obesity rates most prevalent in deprived areas (Ministry of Health., 2017). In New Zealand (NZ), obesity is linked to significant health inequities. Pacific peoples have a higher rate of obesity (69%) compared to the general population (32%). The specific aims of this study were to assess in premenopausal (18-45-year-old) obese and non-obese women from Pacific and NZ European population groups the association between taste perception, body composition and long-term adiposity signals (fasting insulin and leptin). Identification of distinct relationships between taste perception and the endocrine regulation of adiposity will advance our understanding of the aetiology of obesity and may open new avenues for therapeutic targets.

4.3. Materials and methods

4.3.1. Design and participant recruitment

The main research programme in which this study was nested is described in the study protocol (Kindleysides et al., 2019). The screening process was based on self-reported measurements of height and weight, with women invited to participate that had either a normal weight profile (body mass index (BMI): 18.5 – 24.9 kg/m^2) or an obese profile (BMI ≥30 kg/m^2). Subsequently, in this study we grouped women into high and low body fat (BF) %, using a cut point of 35% (Oliveros, Somers, Sochor, Goel, & Lopez-Jimenez, 2014), based on initial screening and inclusion criteria we expected an approximately equal proportion of women in each group. The BF% cut point was used in accordance with the AACE/ACE guidelines (obesity in women was defined as >35 BF%) (Dickey et al., 1998; Jo & Mainous, 2018; Oliveros et al., 2014). Inclusion criteria were: age 18-45 years, being post-menarche and pre-menopausal (as defined by a regular menstrual cycle for the last year), ethnicity (self-identified as NZ European and having lived in NZ for a minimum of 5 years, or self-identified as Pacific and having at least one parent of full Pacific ethnicity), willingness to comply with study requirements and being healthy. Exclusion criteria were: BMI ≤18.5 kg/m^2, pregnant or lactating, presence of any diagnosed chronic illness (e.g. type 2 diabetes, CVD, cancer, etc), bariatric surgery, severe food allergy, use of medication that interferes with appetite or the immune system, smoking, severe dietary restriction or avoidances (e.g. vegan) and antibiotic use in the last three months.

Participants were recruited in Auckland, New Zealand between July 2016 and September 2017 and comprised of 174 Pacific and 177 New Zealand (NZ) European women. Initial participant screening was conducted either online, in-person or over the phone. Further details have been published in the study protocol paper (Kindleysides et al., 2019). The study was approved by the Southern Health and Disability Ethics Committee (16/STH/32) and written informed consent was provided by each participant. The trial was registered at anzctr.org.nz as ACTRN12618000432213. This

study complies with the Declaration of Helsinki for medical research involving human subjects.

4.3.2. Study procedure

All participants attended two clinic visits, 11-14 days apart. At visit one, participants completed a health and demographic face-to-face interview and had their height and weight measured. In addition, they provided a blood sample and underwent taste testing of three tastants (sweet, bitter and milk fat), in a (10-hour) fasted state. Three measurements of taste perception were determined by taste testing; hedonic liking rating, intensity rating and discrimination of taste concentration by ranking task. Participants initially sampled the blank water solution (labelled "sweet"), prior to tasting the four individual 10 mL sweet taste solutions, followed by the sweet taste ranking task. This procedure was repeated for bitter taste solutions and finally for milk fat solutions. All taste stimuli were evaluated at room temperature (20°C) in individual taste testing booths as whole mouth samples which were evaluated using a sip-and-spit procedure (Martinez-Cordero, Malacara-Hernandez, & Martinez-Cordero, 2015; Mattes, 2009). Before taste testing, a short one-on-one training procedure took place to familiarise each participant with the general labelled magnitude scale (gLMS; see below), labelled affective magnitude (LAM) scale and facilities. Taste testing took place between 07:00 and 09:00 at visit one. At visit two, participants underwent a whole-body scan using dual x-ray absorptiometry (DXA).

4.3.3. Taste perception measurements

4.3.3.1. Stimuli for sweet taste, bitter taste and milk fat perception measurement

All taste solutions were prepared on the day of testing. Sweet taste solutions comprised of dissolved glucose (dextrose monohydrate, Sherratt Ingredients, Auckland, New Zealand) in distilled water, similar to previous studies (Jayasinghe et al., 2017). Bitter taste solutions were prepared from a refrigerated stock solution, prepared on a weekly basis, of quinine hydrochloride dehydrate (Sigma-Aldrich, St Louis MO, USA)

dissolved in distilled water. Milk fat samples were made from full-fat milk (Anchor™ Blue Top standardised milk, New Zealand) and cream (Anchor™ Fresh Cream, New Zealand) which, once opened were used on the day of testing. Each individual milk fat sample was thoroughly mixed by drawing the solution 5-6 times with a 10 mL pipette. These were chosen to create a series of milk fat perception tastants using the whole mouth sip and spit technique (Chen & Eaton, 2012).

Glucose was the stimulus chosen for sweet taste perception measurement, as it has been used in similar studies and cited as having clearly defined links with sweet taste perception being a simple sugar (Jayasinghe et al., 2017). Glucose also has clearly defined metabolic links and direct association with intestinal glucose-mediated receptors, subsequent insulin release and energy utilisation (Calvo and Egan, 2015). Quinine has been described as a practical measure for broader bitter taste functioning (Rawal, Hoffman, Honda, Huedo-Medina, & Duffy, 2015). Milk and cream were chosen to create a series of milk fat perception solutions (Zhou, Shen, Parker, Kennedy, & Methven, 2016). Milk and cream is a complex tastant due to comprising of at least three further elements responsible for the sensory properties of milk, including: (1) pleasant mouthfeel due to presence of macromolecules, such as fat globules and colloidal proteins, (2) salt and sweet taste due to milk salts and lactose, respectively, and (3) an aroma profile (Cadwallader, 2010). As such, milk fat solutions were used to measure the perception of fat, but not taste perception.

4.3.3.2. gLMS and LAM measurement of sweet, bitter and milk fat hedonic liking and intensity

The intensity and hedonic liking rating scales were evaluated simultaneously on the same single-sided questionnaire, for each of the individual taste solutions. The blank water solution was evaluated first, followed by individual glucose solutions presented in a randomised order (30 g/L, 60 g/L, 120 g/L and 240 g/L), with no side-by-side comparisons, each identified by a random 3-digit code. The intensity and hedonic liking of quinine samples was evaluated at concentrations of 0.008 g/L, 0.016 g/L, 0.03 g/L and 0.06 g/L (randomised) and milk fat perception samples at concentrations of 3.3%,

11.8%, 20.3% and 37.3% milk fat (randomised). Participants rinsed their palate with distilled water and expelled the water between tasting samples.

Hedonic liking for each solution was rated using a LAM scale with the anchors: 'Strongest imaginable dislike of any kind' (scale score -50) through to 'Strongest imaginable like of any kind' (50). Intermediate labels on the LAM liking scale were: 'very strongly dislike' (-26.5), 'strongly dislike' (-17.5), 'moderately dislike' (-8.5), 'weakly dislike' (-3), 'neutral' (0), 'weakly like' (3), 'moderately like' (8.5), 'strongly like' (17.5), and 'very strongly like' (26.5) (Schutz and Cardello, 2001). Sweet taste intensity for each solution was rated alongside hedonic liking, using a gLMS with the anchors 'No sensation' (scale score =0) and 'Strongest imaginable sensation of any kind' (100), with intermediate labels 'barely detectable' (1.5), 'weak sensation' (6), 'moderate sensation' (17), 'strong sensation' (35), and 'very strong sensation' (53) (Bartoshuk et al., 2004).

4.3.3.3. Taste concentration ranking task

After tasting all the individual solutions of the same type, starting with glucose, four new solutions (30 g/L, 60 g/L, 120 g/L and 240 g/L glucose) were presented as a taste ranking task. All four solutions were presented at once, randomised and identified with unique 3-digit random codes. The four samples were tasted by the participant and placed in order from the highest to the lowest perceived concentration of taste. Similarly, after evaluation of the individual quinine solutions, the ranking task procedure took place for bitter taste (0.008 g/L, 0.016 g/L, 0.03 g/L and 0.06 g/L quinine). To end, the milk fat perception ranking task was completed after evaluating the milk fat solutions. The milk fat ranking task solutions (3.3%, 11.8%, 20.3% and 37.3% fat) were covered in aluminium foil and tasted through a straw to prevent any visuals cues.

4.3.4. Anthropometric measurements

Stretched height (stadiometer) and weight (Sauter platform scale E1200, GmbH, Germany) were measured using the International Society of the Advancement of Kinanthropometry (ISAK) protocols and used to calculate BMI (Stewart, Marfell-Jones, Olds, and de Ridder, 2011). Body composition was measured by DXA to obtain BF% data (Hologic QDR Discovery A, Hologic Inc, Bedford, MA with APEX V. 3.2 software). Prior to DXA scanning, participants changed into appropriate clothing (i.e. tight fitting activewear, swimwear, etc), emptied their bladder, removed jewellery and glasses and re-asked if they were pregnant, had a pacemaker or any metal implants. Research staff conducting these measures were Level 1 ISAK trained.

4.3.5. Blood sample collection

Fasting venous blood samples were collected into pre-chilled tubes containing ethylenediaminetetraacetic acid (EDTA) with dipeptidyl-aminopeptidase IV inhibitor (2 mL Becton Dickinson Vacutainer®, Proprietary Cocktail of Protease, Esterase and DPP-IV Inhibitors; BD P800 hemogard™ closure, NZ) and plain vacutainer tubes (10 mL Becton Dickinson vacutainer® red top, BD hemogard™ closure, NZ) and whole blood EDTA containing vacutainer (10 mL Becton Dickinson vacutainer® lavender top, whole blood tube, BD hemogard™ closure, NZ).

4.3.6. Blood sample processing and analysis

The blood in the EDTA aminopeptidase tube was centrifuged (1500 *g*, 4°C, 15 min) to obtain a plasma sample for the determination of leptin. The blood in the plain tube was allowed to clot and the tube was then centrifuged (1500 *g*, 4°C, 15 min) to obtain a serum sample for determination of insulin, glucose and blood cholesterol. The blood in the whole blood EDTA tube was used for determination of glycated haemoglobin (HbA1c). Appropriate aliquots of plasma and serum were transferred into 1.5 mL microcentrifuge tubes (Eppendorf® safe-lock PCR clean tubes, Hamburg, Germany) and stored immediately at -80°C. Serum insulin, glucose and blood cholesterol markers were analysed with the use of an autoanalyser (Roche Cobas e411). Serum insulin

values are expressed as ng/mL (Rodriguez-Cabaleiro, Van Uytfanghe, Stove, Fiers, & Thienpont, 2007). The serum insulin assay range was 0.10 – 5.06 ng/mL [interassay CV: 0.51%]. Serum glucose range was 4.4 – 15.6 mmol/L [interassay CV: 0.7%]; serum HDL range was 0.86 – 2.94 mmol/L [interassay CV: 6.08%]; serum LDL range was 0.93 – 8.74 mmol/L [interassay CV: 0.92%]; serum triglyceride range was 0.29 – 4.06 mmol/L [interassay CV: 0.84%]; serum cholesterol range was 2.44 – 10.6 mmol/L [interassay CV: 1.97%] and whole blood HbA1c range was 25.3 – 87.1 mmol/mol [interassay CV: 1.56%]. Plasma leptin was analysed using the MILLIPLEX MAP Human Metabolic Hormone Magnetic Bead Panel 96-Well Metabolism Multiplex Assay (Millipore, USA, Cat # HMHEMAG-34K) (Poppitt et al., 2017). Leptin samples were assayed in duplicate, and plates were read with a Bioplex 100 Analyzer System (Bio-Rad). The leptin assay range was 0.37 – 66.0 ng/mL [interassay CV: 4.1%].

4.3.7. Health and demographic face-to-face interview and deprivation index

General information was obtained by a one-on-one interview with participants which included a range of questions about occupation, number of children, alcohol consumption and work patterns (data not reported). The 2013 New Zealand Deprivation (NZDep) index was used as a measurement of socioeconomic status, based on the participant's home address. It is a scale from 1-10, with 1 representing the least deprived and 10 the most deprived (Atkinson, Salmond, & Crampton, 2014).

4.3.8. Data handling

4.3.8.1. Taste perception hedonic liking groups

A hierarchical cluster analysis was conducted to classify participants into sweet/bitter/fat liking/disliking clusters using LAM hedonic ratings (5 concentrations for sweet and 4 concentrations for bitter and milk fat). This analysis grouped participants into either sweet, bitter and milk fat liking status or into disliking status.

This analysis was chosen as it clusters seemingly homogenous data together and does not require any *a priori* decisions regarding the number of clusters, or groups, produced (Garneau, Nuessle, Mendelsberg, Shepard, & Tucker, 2018).

4.3.8.2. Statistical Analysis

Descriptive statistics were reported as mean and standard deviation (SD) or median and 25^{th}, 75^{th} percentiles, and group data presented as the number of participants (n) and %. Data were checked for normality using the Shapiro-Wilks test. Differences between groups were evaluated by t-test for normally distributed data and by Mann-Whitney U test for data that were not normally distributed (Field, 2013). Chi-square tests were used to compare the ranking task outcome (correct vs. incorrect) or taste liking clusters (likers vs. dislikers). We used linear mixed models with a random intercept for subject (taking into account repeated measurements) to assess the association between concentration and taste perception.

A hierarchical cluster analysis was performed using LAM liking ratings for sweet, bitter and milk fat perception. Clusters were assessed using the agglomerative method with Ward's minimum variance algorithm (Asao et al., 2015). Distinct taste liker phenotypes were identified. This method derives meaningful groups (clusters) of participants who shared similar liking patterns within each group but were heterogenous in the between-group contrasts. The squared Euclidean distance between pairs of cases or clusters and the between-groups (averages) linkage method were selected to assist with the merging process (Iatridi et al., 2019). These results were checked with the elbow method, silhouette method and gap statistics to observe the optimal number of clusters. Using the majority rule for each method checked, two liking clusters for each tastant (sweet, bitter and milk fat) were selected (Rousseuw & Kaufman, 1990).

Logistic regression was used to estimate the effects of taste perception (by liking clusters or ranking task) on BF% (high compared to low BF%). Analyses were adjusted for age, deprivation, and ethnicity. Additional analyses were conducted to estimate the effects of fasting plasma leptin and plasma insulin concentration, on taste perception

outcome (by liking clusters or ranking task), adjusted for age, deprivation and ethnicity. Analyses to estimate the effects of taste perception (by liking cluster or ranking task) on fasting blood glucose regulation and cholesterol concentrations were conducted, adjusting for age, deprivation and ethnicity.

In logistic regression models, a median cut-off was used to determine associations between higher compared to lower plasma leptin levels (median = 12 ng/mL) and plasma insulin levels (median = 0.46 ng/mL) as independent variables. Median cut-offs for associations between higher and lower blood glucose and HbA1c concentrations, and blood cholesterol concentrations were used as outcome variables: glucose = 5.30 mmol/L; HbA1c = 32.0 mmol/L; HDL cholesterol = 1.52 mmol/L; LDL cholesterol = 2.92 mmol/L; Total cholesterol = 4.74 mmol/L; Triglycerides = 0.90 mmol/L.

All analyses were conducted separately for Pacific and NZ European women. A *P*-value of less than 0.05 was considered statistically significant. Analyses were conducted using SPSS version 25 (IBM Corporation, New York, USA). Linear mixed model and cluster analyses were conducted using SAS (v9.3, SAS Institute, Cary, NC, USA).

4.4. Results

4.4.1. Participant characteristics

A total of 351 women were recruited. Overall, eleven women were excluded due to exclusion criteria (e.g. pregnancy, medication use). Anthropometric and adiposity characteristics of the study participants are presented in Table 4.1. Pacific women were significantly younger than NZ European women ($P< 0.001$) and measurements of weight, BMI and BF% (with exception of gynoid body fat) were significantly higher among Pacific women with low BF% compared to NZ European women of the same BF% group ($P< 0.001$). Similarly, BMI and weight were significantly higher among Pacific women with high BF% compared to NZ European women of the same BF% group ($P< 0.05$); however, there were no differences in BF% distribution measurements.

Pacific women with <35 BF% had a significantly higher plasma leptin, plasma insulin and glucose concentration than NZ European women of the same BF% group ($P< 0.01$, Table 4.1). Furthermore, Pacific women with >35 BF% had a significantly higher fasting plasma insulin concentration compared to NZ European women with >35 BF% ($P< 0.001$). HbA1c was higher in Pacific women in comparison to NZ European women, irrespective of BF% ($P< 0.05$, Table 4.1). HDL cholesterol was lower in Pacific women compared to NZ European women ($P< 0.05$, Table 4.1) and LDL and total cholesterol levels were higher in NZ European women with >35 BF%, compared to Pacific women with >35 BF% ($P< 0.05$, Table 4.1).

In Pacific women, there were more (64%) sweet dislikers compared to NZ European women (55%). NZ European women with >35 BF% had the highest percentage of sweet likers (51%), significantly higher than in Pacific women of the same BF% group ($P< 0.05$, Table 4.1). A higher percentage of Pacific women with <35 BF% correctly ranked the sweet taste task (89%) compared to those with >35 BF% (73%) (Table 4.1). Pacific women with <35 BF% had a significantly higher number of bitter likers (23%)

compared to Pacific women with >35 BF% (11%). No differences across the groups were observed in terms of milk fat liking.

Table 4.1. Participant characteristics (n=340).

	Pacific		***NZ European***	
	Low BF (<35%)	**High BF (>35%)**	**Low BF (<35%)**	**High BF (>35%)**
	N= 75	N= 94	N= 87	N= 84
	Mean ± SD	Mean ± SD	Mean ± SD	Mean ± SD
BMI (kg/m^2)	25.8 ± 3.78 †, #	35.9 ± 6.56 #	22.6 ± 2.06 †	33.6 ± 4.07
Weight (kg)	73.9 ± 11.5 †, #	101.9 ± 20.5 #	63.1 ± 7.47 †	94.4 ± 13.9
Height (cm)	169.1 ± 6.84	168.3 ± 6.58	167.2 ± 5.57	167.5 ± 6.90
Body fat (%)	30.0 ± 3.23 †, #	40.3 ± 3.84	28.0 ± 4.89 †	41.2 ± 3.76
Android fat (%) §	29.5 ± 5.24 †, #	42.3 ± 5.12	24.9 ± 5.15 †	41.5 ± 4.90
Gynoid fat (%) §	34.8 ± 3.43 †	41.7 ± 3.70	33.5 ± 4.19 †	42.6 ± 4.13
Visceral fat (%) §	27.2 ± 5.67 †, #	40.5 ± 5.29	22.3 ± 5.87 †	39.6 ± 5.14
	Median (25, 75 percentiles)	Median (25, 75 percentiles)	Median (25, 75 percentiles)	Median (25, 75 percentiles)
Age (yrs)	23.0 (20.0, 29.0) #	23.0 (21.0, 27.0) #	29.0 (24.0, 36.0) †	34.0 (28.0, 40.0)
NZDep2013 §	7.5 (5.0, 10.0) #	8.0 (7.0, 9.0) #	3.0 (2.0, 6.0) †	5.0 (3.0, 6.0)
Fasting leptin (ng/mL) §	7.67 (5.02, 12.3) †, #	21.5 (15.7, 29.9)	4.95 (2.79, 8.65) †	20.3 (13.7, 30.6)
Fasting insulin (ng/mL) §	0.47 (0.32, 0.65) †, #	0.86 (0.56, 1.28) #	0.29 (0.19, 0.35) †	0.52 (0.40, 0.76)
Glucose (mmol/L) §	5.25 (5.02, 5.48) †, #	5.43 (5.13, 5.85)	5.09 (4.86, 5.34) †	5.56 (5.12, 5.75)
HbA1c (mmol/mol) §	32.1 (30.8, 33.8) †, #	33.8 (32.3, 36.5) #	30.6 (29.0, 31.9) †	31.1 (28.8, 33.3)
HDL (mmol/L) §	1.50 (1.33, 1.81) †, #	1.32 (1.18, 1.56) #	1.83 (1.63, 2.04) †	1.43 (1.28, 1.66)
LDL (mmol/L) §	2.80 (2.42, 3.26)	2.92 (2.44, 3.32) #	2.76 (2.35, 3.39) †	3.43 (2.72, 4.09)
Total cholesterol (mmol/L) §	4.50 (4.11, 5.07) #	4.52 (4.14, 5.09) #	4.89 (4.31, 5.43) †	5.23 (4.68, 6.09)
Triglycerides (mmol/L) §	0.79 (0.67, 1.07) †	0.99 (0.85, 1.48)	0.70 (0.57, 0.94) †	1.05 (0.79, 1.50)
	n (%)	n (%)	n (%)	n (%)
Sweet likers (group)	28 (37%)	32 (35%) #	34 (39%)	43 (51%)
Bitter likers (group)	17 (23%) †	10 (11%)	18 (21%)	10 (12%)
Milk fat likers (group)	54 (72%)	58 (63%)	63 (72%)	63 (75%)
Sweet ranking task (correct)	67 (89%) †	67 (73%)	76 (87%)	64 (77%)
Bitter ranking task (correct)	22 (29%) #	33 (36%)	43 (49%)	41 (49%)
Milk fat ranking task (correct)	28 (37%)	25 (27%) #	37 (43%)	42 (50%)

Means ± SD; independent samples t-test. Median (25, 75 percentiles); Mann-Whitney U test. Chi-square test for taste liking/ranking task groups, categorical group comparisons. † $P< 0.05$ for test comparing body fat groups within the ethnic group. # $P< 0.05$ for test comparing between ethnic groups within body fat group. BMI, body mass index. §Calculated from complete datasets only (n=304). n (%) refers to the number and the percentage of participants that are likers/taste ranking task correct within the group, compared to dislikers/taste ranking task incorrect.

4.4.2. Sweet, bitter and milk fat perception and liking clusters

Linear mixed model analyses showed that increased glucose concentrations were associated with increased intensity ratings ($P< 0.001$) and a decrease in sweet taste hedonic liking ($P< 0.001$). Bitter taste intensity ratings increased ($P< 0.001$) and liking decreased ($P< 0.001$) with increasing quinine concentration. Ratings of milk fat intensity significantly increased with increasing milk fat concentration ($P< 0.001$), however, milk fat hedonic liking were similar across all concentrations (data not shown).

Hierarchical cluster analysis identified taste 'likers' and 'dislikers' for sweet, bitter and milk fat. Sweet likers rated the intensity of each glucose concentration level as significantly less intense than sweet dislikers at 30 g/L ($P< 0.05$), 60 g/L, 120 g/L and 240 g/L ($P< 0.001$) (Figure 4.1, A). Similarly, bitter dislikers rated all concentrations as significantly more intense than bitter likers (Figure 4.1, B). There was little difference in the intensity rating between milk fat likers and dislikers, with milk fat likers rating intensity of taste as more intense only at the 20.3% concentration level ($P< 0.05$) (Figure 4.1, C).

In NZ European women, the intensity ratings of 30 g/L glucose were significantly higher in sweet likers than dislikers ($P< 0.01$); this was not observed in Pacific women (Figure 4.2, A-B). Similarly, in NZ European women, the intensity rating of 0.008 g/L quinine concentration was significantly higher in bitter likers than dislikers ($P< 0.001$) and this was not observed in Pacific women (Figure 4.2, C-D). In Pacific women, intensity ratings of 20.3% milk fat were higher in sweet likers compared to sweet dislikers ($P< 0.01$); this was not observed in NZ European women (Figure 4.2, E-F).

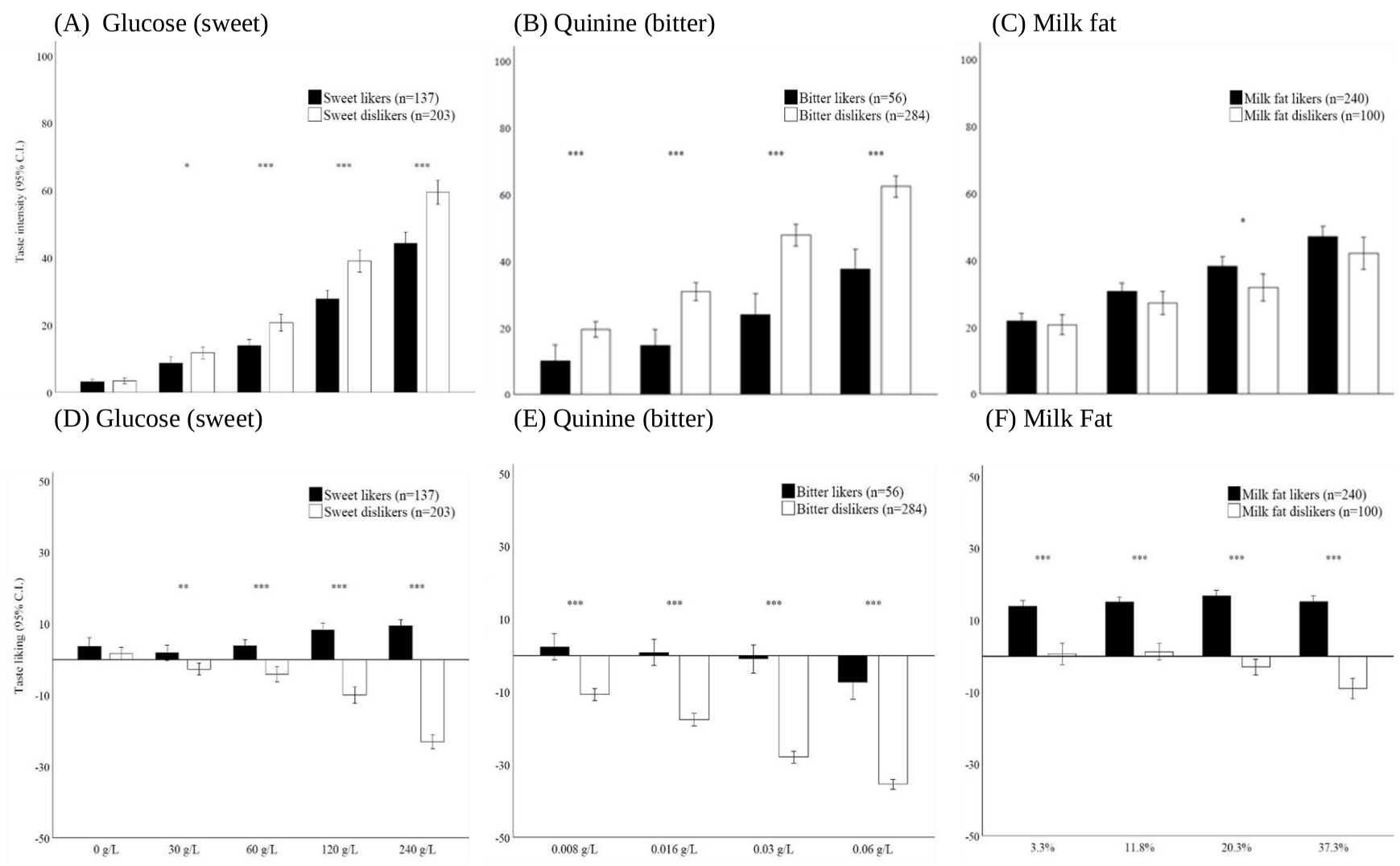

Figure 4.1. The effect of tastant concentration on subjective ratings of suprathreshold taste intensity (A-C) and liking (D-F) for sweet (glucose), bitter (quinine) and fat (milk fat) solutions by liking cluster as determined by hierarchical cluster analysis (N= 340). Independent samples t-test. C.I. confidence interval; g/L, grams per litre. *P< 0.05, **P< 0.01, ***P< 0.001.

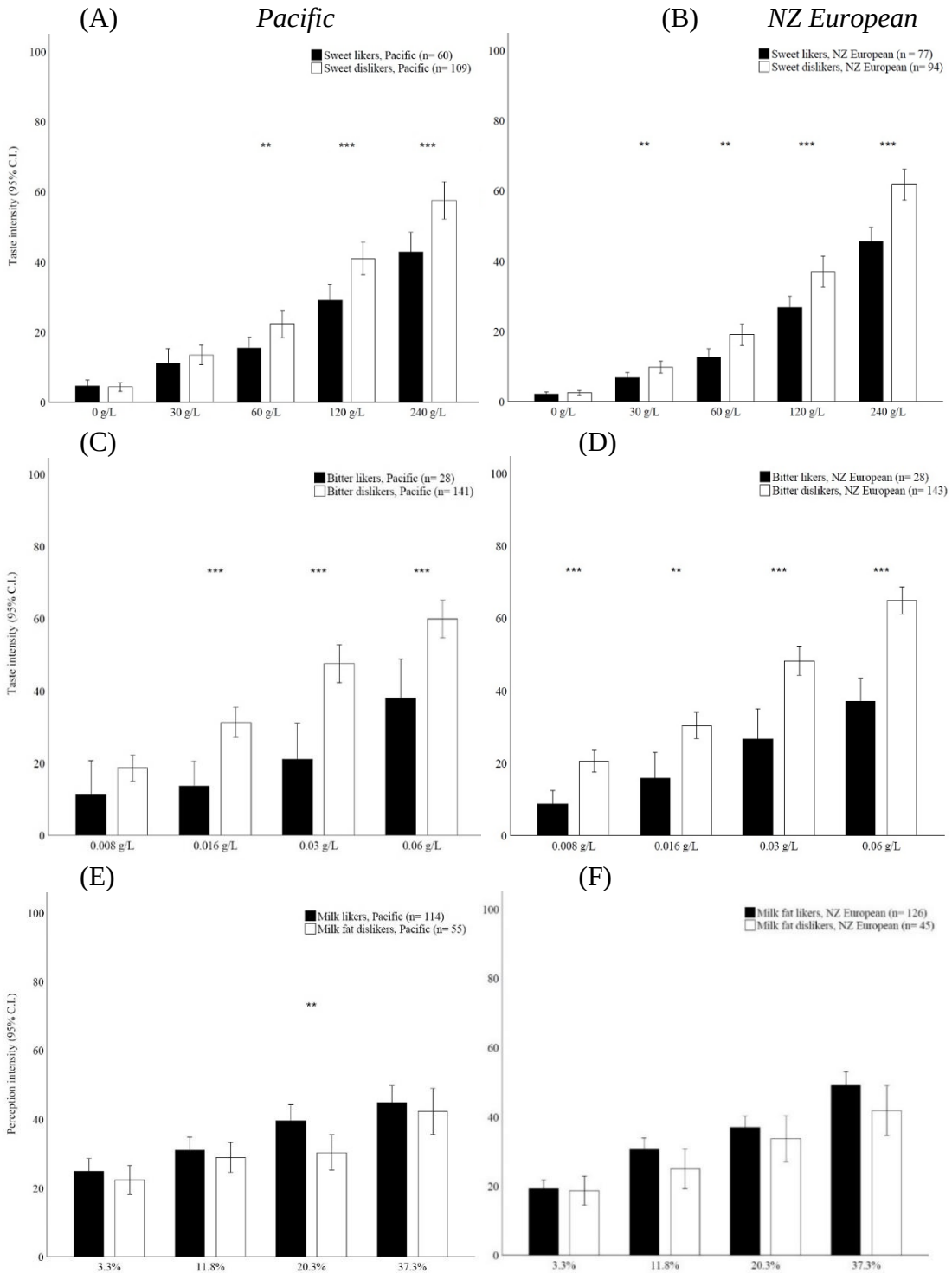

Figure 4.2. The effect of tastant concentration in Pacific and NZ European women, on subjective gLMS ratings of suprathreshold taste intensity for sweet (glucose, A-B), bitter (quinine, C-D) and fat (milk fat, E-F) solutions by liking cluster (N= 340). Independent samples t-test. C.I. confidence interval; g/L, grams per litre. ***P*< 0.01, ****P*< 0.001.

4.4.3. Taste liking and body fat percentage

In NZ European women, sweet likers were twice as likely to have a high >35 BF% compared to sweet dislikers ($P< 0.05$). In contrast, in Pacific women, sweet liking was not associated with BF% (Table 4.2).

Women (Pacific and NZ European combined) who incorrectly discriminated taste by sweet ranking task were nearly three times more likely to have a high >35 BF% compared to those who correctly ranked the task ($P< 0.01$; Table 4.2). This effect was most pronounced in Pacific women (OR 3.8, $P< 0.01$; Table 4.2). In contrast, across both populations, women who were bitter likers had a decreased likelihood (OR = 0.4, $P< 0.01$; Table 4.2) of being in the >35 BF% group, when compared to bitter dislikers. There were no significant associations found when comparing milk fat perception by liking or ranking task outcomes across BF% groups.

Table 4.2. Associations between taste liking and ranking, and body fat % (N=304§).

	Unadjusted			***Adjusted***		
	High vs low BF% (OR, 95% C.I.)			High vs low BF% (OR, 95% C.I.)		
	Pacific (n=142)	NZ European (n=162)	TOTAL ‡ (n=304)	Pacific # (n=142)	NZ European # (n=162)	TOTAL ^ (n=304)
Sweet likers (vs dislikers)	0.99 (0.50, 1.96)	1.53 (0.82, 2.85)	1.25 (0.79, 1.99)	1.02 (0.51, 2.07)	**2.06 (1.04, 4.11)***	1.39 (0.86, 2.24)
Sweet ranking task incorrect (vs correct)	**3.23 (1.27, 8.22)****	1.91 (0.82, 4.42)	**2.43 (1.31, 4.52)****	**3.77 (1.39, 10.2)****	2.41 (0.98, 5.93)	**2.94 (1.53, 5.65)****
Bitter likers (vs dislikers)	0.45 (0.18, 1.10)	0.59 (0.25, 1.37)	0.52 (0.28, 0.96)*	0.35 (0.14, 0.89)*	0.51 (0.21, 1.28)	**0.42 (0.22, 0.80)****
Bitter ranking task incorrect (vs correct)	0.77 (0.38, 1.56)	1.00 (0.54, 1.86)	0.89 (0.56, 1.42)	0.79 (0.38, 1.62)	0.97 (0.50, 1.87)	0.89 (0.55, 1.45)
Milk fat likers (vs dislikers)	0.67 (0.32, 1.37)	0.98 (0.49, 1.95)	0.81 (0.50, 1.34)	0.75 (0.36, 1.58)	1.23 (0.58, 2.61)	0.94 (0.56, 1.58)
Milk fat ranking task incorrect (vs correct)	1.78 (0.88, 3.60)	0.65 (0.35, 1.21)	1.01 (0.63, 1.60)	1.61 (0.75, 3.42)	0.64 (0.33, 1.25)	0.94 (0.58, 1.53)

BF, body fat; OR, odds ratio; C.I. confidence interval. Adjusted for: ‡ ethnicity; # Age and NZDep2013; ^ ethnicity, age and NZDep2013. *P< 0.05, **P< 0.01. §Calculated from complete datasets only (i.e. excludes dropouts and participants with missing blood data).

4.4.4. Fasting plasma leptin and insulin and taste perception

High fasting leptin in NZ European women was strongly associated with the likelihood of being a sweet liker (OR 3.6, $P< 0.001$), whereas no association was observed in Pacific women (Table 4.3). Also, NZ European women with high plasma insulin levels were nearly three times more likely to be a sweet liker ($P< 0.01$; Table 4.3). Again, no association was observed in Pacific women.

Higher plasma insulin increased the likelihood of incorrectly ranking the sweet taste ranking task, but this was only statistically significant for insulin for the combined (Pacific and NZ European women) group, without adjusting for BF% (OR 2.3, $P< 0.05$; Table 4.3). Additional adjustment for BF% to account for study design did not significantly alter the associations between leptin and insulin and taste perception. There were no associations between plasma insulin or leptin levels and bitter and milk fat liking, nor ranking task outcomes (Table 4.3).

Table 4.3. Association between plasma leptin and insulin and taste liking and taste ranking task (N=304§).

	Pacific # (n= 142)	NZ European # (n= 162)	TOTAL ^ (n= 304)
	High versus low plasma leptin (OR, 95% C.I.)		
Sweet likers (vs dislikers)	0.82 (0.40, 1.66)	**3.64 (1.82, 7.27)*** **	**1.72 (1.06, 2.78)***
Sweet ranking task incorrect (vs correct)	1.49 (0.61, 3.65)	1.91 (0.82, 4.49)	1.70 (0.92, 3.16)
Bitter likers (vs dislikers)	0.96 (0.46, 2.04)	0.54 (0.22, 1.33)	0.70 (0.38, 1.30)
Bitter ranking task incorrect (vs correct)	0.65 (0.31, 1.36)	0.94 (0.50, 1.78)	0.81 (0.51, 1.31)
Milk fat likers (vs dislikers)	0.82 (0.40, 1.66)	1.05 (0.51, 2.19)	0.99 (0.59, 1.67)
Milk fat ranking task incorrect (vs correct)	1.36 (0.63, 2.92)	0.81 (0.43, 1.53)	1.00 (0.62, 1.61)
	High versus low plasma insulin (OR, 95% C.I.)		
Sweet likers (vs dislikers)	1.04 (0.50, 2.19)	**2.86 (1.43, 5.72)****	**1.74 (1.06, 2.88)***
Sweet ranking task incorrect (vs correct)	2.37 (0.82, 6.82)	2.27 (0.97, 5.32)	**2.26 (1.17, 4.36)***
Bitter likers (vs dislikers)	0.84 (0.34, 2.09)	0.56 (0.21, 1.49)	0.65 (0.34, 1.23)
Bitter ranking task incorrect (vs correct)	0.90 (0.42, 1.92)	0.90 (0.46, 1.75)	0.91 (0.55, 1.49)
Milk fat likers (vs dislikers)	0.69 (0.31, 1.54)	1.61 (0.74, 3.53)	1.06 (0.62, 1.81)
Milk fat ranking task incorrect (vs correct)	1.08 (0.49, 2.39)	0.94 (0.49, 1.84)	0.92 (0.56, 1.51)

Models adjusted for # age and NZDep2013 and ^ age, NZDep2013, and ethnicity. *P< 0.05, **P< 0.01, ***P< 0.001. OR: odds ratio; C.I. confidence interval. Missing values: insulin n=7, leptin n=2. §Calculated from complete datasets only (i.e. excludes dropouts and participants with missing blood data).

4.4.5. Sweet taste liking and taste ranking task and markers of blood glucose regulation and cholesterol

Due to finding significant associations between sweet taste perception and metabolic regulators of adiposity, the effects that taste perception may have on blood glucose regulation and cholesterol were further investigated. In Pacific women, sweet likers had decreased fasting HbA1c, LDL cholesterol and total cholesterol concentration in comparison to sweet dislikers ($P< 0.05$; Table 4.4).

NZ European sweet likers had fasting circulating glucose concentration which was three times higher than sweet dislikers ($P< 0.01$; Table 4.4). NZ European sweet likers had a decrease in fasting HDL cholesterol compared to sweet dislikers (OR 0.4, $P< 0.01$; Table 4.4). In NZ European and Pacific combined, HDL cholesterol was decreased in sweet likers (OR 0.5, $P< 0.05$). Adjustment for BF% to account for study design did not significantly alter blood glucose regulation or cholesterol associations with taste perception in Pacific and NZ European women. Further, analysis of blood glucose regulation markers and cholesterol as continuous variables (linear regression) gave similar results.

4.4.6. Bitter and milk fat perception liking and ranking task outcome and blood glucose regulation and cholesterol concentrations

Pacific women who incorrectly ranked the bitter taste task were twice as likely to have high HbA1c (OR 2.2, $P< 0.05$) compared to Pacific women who correctly ranked the task. No other clear associations were found between blood glucose regulation markers and cholesterol with bitter taste or milk fat perception outcomes in Pacific or NZ European women (Suppl. 1). Adjusting for BF% did not significantly alter associations between blood glucose or cholesterol and taste perception.

Table 4.4. Associations between sweet taste perception liking and ranking and plasma blood glucose and cholesterol (N= 304§).

	Pacific # (n= 142)	NZ European # (n= 162)	TOTAL ^ (n= 304)
	Sweet likers versus dislikers (OR, 95% C.I.)		
Glucose (mmol/L)	0.71 (0.35, 1.44)	**3.18 (1.62, 6.26)****	1.50 (0.94, 2.40)
HbA1c (mmol/mol)	**0.44 (0.21, 0.95)***	1.80 (0.89, 3.66)	0.92 (0.55, 1.54)
HDL (mmol/L)	1.31 (0.63, 2.73)	**0.40 (0.20, 0.77)****	0.72 (0.45, 1.17)
LDL (mmol/L)	**0.46 (0.21, 0.98)***	1.28 (0.66, 2.48)	0.82 (0.50, 1.32)
Total cholesterol (mmol/L)	**0.44 (0.20, 0.97)***	1.11 (0.57, 2.18)	0.76 (0.46, 1.24)
Triglycerides (mmol/L)	1.00 (0.49, 2.03)	1.10 (0.59, 2.08)	1.05 (0.66, 1.67)
	Sweet ranking task incorrect versus correct (OR, 95% C.I.)		
Glucose (mmol/L)	1.82 (0.74, 4.48)	1.11 (0.48, 2.58)	1.39 (0.76, 2.55)
HbA1c (mmol/mol)	1.43 (0.52, 3.94)	2.07 (0.86, 5.01)	1.77 (0.90, 3.46)
HDL (mmol/L)	0.49 (0.18, 1.33)	0.48 (0.21, 1.12)	**0.50 (0.26, 0.95)***
LDL (mmol/L)	1.37 (0.56, 3.36)	2.25 (0.88, 5.76)	1.68 (0.89, 3.16)
Total cholesterol (mmol/L)	0.74 (0.29, 1.92)	1.76 (0.69, 4.49)	1.11 (0.59, 2.10)
Triglycerides (mmol/L)	1.15 (0.47, 2.77)	1.65 (0.71, 3.82)	1.41 (0.77, 2.58)

Models adjusted for # age and NZDep2013 and ^ age, NZDep2013, and ethnicity. **P*< 0.05, ***P*< 0.01, ****P*< 0.001. OR: odds ratio; CI confidence interval.
§Calculated from complete datasets only (i.e. excludes dropouts and participants with missing blood data).

4.5. Discussion

This cross-sectional study characterises sweet taste, bitter taste and milk fat perception in two population groups with markedly different metabolic disease risk and different body fat measurements. Our study describes that taste perception is significantly influenced by levels of body fat, ethnic-cultural and socio-economic characteristics. We further demonstrate links between taste perception and the long-term adiposity signals, insulin and leptin that influence energy balance, body weight and food intake.

4.5.1. Study population

The present report describes a segment of the PROMISE (PRedictors linking Obesity and gut MIcrobiomE) study (Kindleysides et al., 2019) which investigates obesity in Pacific women, known to have a high metabolic disease risk, and NZ European women, known to have a moderate metabolic disease risk. The study explores whether findings are different in terms of physical, ethnic-cultural and socio-economic characteristics. Pacific women with lower total body fat (<35%) had a higher proportion of android and visceral fat, despite having the same proportion of gynoid fat, when compared to NZ European women. Overall, Pacific women had higher levels of fasting plasma leptin and insulin concentrations. In addition, Pacific women had a significantly higher deprivation index (i.e. more deprived) when compared to NZ European women. In NZ European women, those with higher body fat (>35%) had a significantly higher deprivation index than those with lower body fat. These findings indicate there is marked socio-economic inequity observed between these two ethnic groups, which appears to be linked with obesity profiles. In addition, variability in taste perception and hedonic taste preferences were observed across our study population. Previous studies have shown associations between taste receptors and glucose and insulin homeostasis (Dotson et al., 2008; Park & Song, 2019). Taste perception classification and how this is linked with endocrine and metabolic regulation will be explored in more detail below.

4.5.2. Comparisons of taste perception clusters and population groups

In Pacific women, there were a higher number of sweet dislikers (64%) than in NZ European women (55%). Pacific women were twice as likely to be sweet taste dislikers compared with NZ European women with high BF%. In both Pacific and NZ European, women with lower body fat % had a higher proportion of bitter likers (~22%) compared to women with higher body fat % (~12%). In contrast, more than half of the participants in this study were milk fat likers (~71%) irrespective of ethnicity.

In a recent study by Szajer et al, sweet taste liking was compared between different ethnicities. Comparisons were made between Hispanic and non-Hispanic young adults, where Hispanic overweight individuals had a decreased liking for sweet taste compared to non-Hispanics (Szajer, Jacobson, Green, & Murphy, 2017). Furthermore, the Hispanic group had significantly lower hedonic reward processing during the evaluation of sucrose when compared to the non-Hispanic group (Szajer et al., 2017). Our study may be showing that Pacific women do not perceive the same reward value from higher concentrations of sweet taste. In this context, it is important to acknowledge that dietary intake varies as a function of cultural and socio-economic background, current environment and upbringing, which may have influenced the hedonic evaluation in this study (Shintani & Hughes, 1994). This study suggests that a different sweet taster profile exists for Pacific women in comparison to NZ European women, which may be reflective of differences in genetics (Risso et al., 2017), environmental upbringing or socio-cultural differences (Tupai-Firestone et al., 2016).

4.5.3. Sweet taste ranking task and obesity

In this study of our population of pre-menopausal healthy women, 20% of women incorrectly discriminated sweet taste concentrations. Discrimination of taste at suprathreshold levels was measured using a ranking task, similar to previous studies (Costanzo et al., 2018; Fushan, Simons, Slack, Manichaikul, and Drayna, 2009; Stewart et al., 2010). Both Pacific and NZ European women who incorrectly ranked sweet taste were nearly three times more likely to have more body fat compared to those who

correctly ranked the task. In a study by Fushan and colleagues, a ranking task using sucrose solutions was used to measure sweet taste sensitivity, and they found that the grading of solutions was related to genetic differences in taste receptor type 1 member 3 (T1R3) transcription (Fushan et al., 2009). This sweet taste sensitivity explained 16% of population variability, which was correlated with the T1R3 coding sequence (Fushan et al., 2009), however other studies to date have not replicated these findings (Running & Hayes, 2016). The genetic differences observed by Fushan and colleagues (2009) may be existent in our participants, which would partially explain the differences observed in the grading of sweet ranking task solutions. However, further investigation into genetic data would be required in order to substantiate this.

Complementary to the sweet taste ranking task results, our study further showed a significant difference in body fat between NZ European sweet likers and dislikers. NZ European sweet likers had twice the likelihood of increased body fat in comparison to sweet dislikers. Previous studies have found no differences between normal weight and obese women in sweet taste liking (Alexy et al., 2011; Cox et al., 2015; Garneau et al., 2018). However, there is emerging evidence that there may be some link between decreased taste sensitivity in an obese state with increased markers of inflammation (Kaufman, Choo, Koh, & Dando, 2018). As previously reported, our current data support the notion that obesity and parameters of metabolic health are influenced by income and socioeconomic status (Bray et al., 2018), as well as age (Dubé, 2010). However, after adjusting for these factors, our data strongly suggest that differences in sweet taste perception may be an important contributor to the complex causes of obesity (Donaldson, Bennett, Baic, & Melichar, 2009).

4.5.4. Sweet taste perception and fasting plasma leptin concentration

Women with higher fasting plasma leptin concentrations were more likely to be sweet likers compared to those with lower fasting plasma leptin concentrations. This is in contrast to bitter taste and milk fat perception where no association was found. A possible mechanism for a link between increased sweet taste liking and circulating concentrations of leptin is that leptin specifically inhibits the response to sweet

substances at the level of the sweet receptor of the tongue (Kawai, Sugimoto, Nakashima, Miura, & Ninomiya, 2000). The leptin receptor (Ob-Rb) co-localises with approximately 40% of T1R3 expressing sweet-sensing taste cells (Kubasova, Burdakov, & Domingos, 2015). The important finding of a significant positive association between circulating leptin levels and taste liking was specific to sweet taste perception in our present study which supports the notion of a specific molecular association between sweet taste perception and leptin pathways (Yoshida et al., 2015).

This study suggests that sweet taste suppression by higher levels of circulating fasting leptin may have resulted in decreased gLMS ratings of sweet taste intensity, resulting in the differences observed between sweet likers and dislikers. Additionally, the associations between high fasting insulin and sweet taste liking are of interest, as there is no receptor for insulin on taste cells engaged in sweet taste perception (Behrens & Meyerhof, 2019). Therefore, the observation of an association between sweet taste liking and increased insulin may be due to leptin regulating peripheral insulin sensitivity (D'Elia, Strazzullo, Iacone, Russo, & Galletti, 2019). Leptin levels, in association with the adipoinsular axis (Kieffer & Habener, 2000), have been shown to be predictive of the development of insulin resistance in normal weight individuals (D'Elia et al., 2019). In the pancreas, beta cells located in the islets of Langerhans are involved in the regulation of blood glucose level via insulin secretion (Calvo & Egan, 2015). All the components necessary for the detection of sweet-tasting compounds (e.g. T1R2, T1R3 and α-gustducin) are present on the human beta cell surface (Henquin, 2012) and the presence of sweet compounds results in an increase in insulin (Laffitte et al., 2014). NZ European women in this study who were sweet likers were significantly more likely to have hyperinsulinemia and hyperleptinemia and they also had decreased levels of HDL cholesterol and increased levels of fasting glucose concentrations. The maintenance of body weight and glucose homeostasis is regulated by leptin, which in turn acts to regulate insulin secretion (Covey et al., 2006; D'souza et al., 2017). These results demonstrate that leptin and insulin associations were mirrored by sweet taste perception. Interestingly, these regulators of adiposity were not associated with either bitter taste or milk fat perception.

Interestingly, the link between sweet taste liking and higher fasting leptin levels in NZ

European women was not observed in Pacific women. The reason for this may be due to a state of relative leptin resistance, as Pacific women in our study population had significantly higher fasting leptin concentrations than NZ European women. In a recent study, increased circulating leptin levels were considered to exceed the level at which leptin can be effective in modulating the recognition of sweet taste (Sanematsu et al., 2018). For individuals with higher body fat, correspondingly increased levels of circulating leptin leads to leptin resistance, impacting the reward system of the brain (Lenard & Berthoud, 2008). For example, in the leptin resistant state, the permeability of the blood-brain barrier to leptin is decreased, resulting in insufficient signalling at the hypothalamus, impacting feeding behaviour and appetite (Amitani et al., 2013). In the present study, it is plausible that in the leptin-sensitive state, sweet taste intensity ratings which are influenced by circulating leptin may be significantly influencing sweet taste liking. In contrast, in the leptin resistant state, these associations may be dysregulated. Further investigation into these associations is warranted.

Studies have shown that sweet taste sensitivity increases after bariatric surgery (Shoar, Naderan, Shoar, Modukuru, & Mahmoodzadeh, 2019; Zakeri & Batterham, 2017) alongside altered taste acuity is the decreased liking, enjoyment or desire for sweet and fatty tasting foods (Gero et al., 2017; Nance, Eagon, Klein, & Pepino, 2017; Van Vuuren, Strodl, White, & Lockie, 2017). The exact reason for these changes is unknown, however gut derived signals and appetite signalling mechanisms are considered to be causal factors (Zakeri & Batterham, 2017). In addition to taste changes, rapid improvements in insulin sensitivity and release occur immediately after surgery (Casimiro, Sam, & Brady, 2019). These effects on pancreatic beta cells are mostly due to increased gut hormone secretion due to the improvement of nutrient delivery to the small intestine. Hyperleptinemia may be further alleviated in parallel with reducing fat mass (Sinclair, Docherty, & Roux, 2018). Such changes in gut derived signalling mechanisms are intimately linked with regulating food intake and reward-processing, resulting in favourable changes in eating behaviour and reduced energy intake (Zakeri & Batterham, 2017). Future studies will reveal more detail on associations between the taste perception, food hedonics, brain reward system responses to food, eating behaviours, body fat content, microbiome and insulin sensitivity (Glaysher et al., 2017).

Preventative measures may be introduced to enhance taste perception, potentially improving signalling mechanisms associated with endocrine regulators of adiposity.

4.5.5. Bitter taste and milk fat perception

In this study, being a bitter liker decreased the likelihood of having higher body fat (>35%), compared to those who were bitter dislikers. Differences in bitter liking influencing weight status is feasible. Increased sensitivity and aversion to bitterness may lead to a decrease in the consumption of healthy, bitter tasting foods (i.e. cruciferous vegetables), resulting in weight gain (Ortega et al., 2016; Turner et al., 2018). Minor ethnic-cultural differences in bitter taste perception may have been observed due to differences in long-term adaption, bitter taste recognition or bitter taste genes (Breslin, 2013; Risso et al., 2017). In contrast, no clear associations were found between milk fat perception measurements and BF%, or between milk fat perception and metabolic regulators of adiposity.

4.5.6. Strengths and limitations of this study and public health implications

One of the strengths of this study was the use of the gLMS and LAM scale and the administration of a range of concentration levels for the measurement of taste perception, as food and beverage consumption generally takes place at suprathreshold concentration levels (Hardikar, Höchenberger, Villringer, & Ohla, 2017). Studies investigating taste status have increasingly supported rating suprathreshold taste solutions with the validated gLMS, to align responses from different population groups such as normal weight and obese (Bartoshuk et al., 2004). An additional strength was the measurement of taste perception using the ranking task, as it did not require the interpretation of a scale it is considered a non-subjective comparative tool. In addition, this study used hierarchical cluster analysis, so the likelihood of misclassifying sweet likers was reduced, in comparison to previously used cut-off approaches (Garneau et al., 2018; Methven, Xiao, Cai, & Prescott, 2016). Hierarchical cluster analysis allowed for the determination of taster status measured from a range of different concentration

levels (Asao et al., 2015; Garneau et al., 2018; Iatridi et al., 2019; Kim et al., 2014). Similar results have been observed in other studies, identifying either two groups (sweet likers and dislikers) (Asao et al., 2015; Kim, Prescott, and Kim, 2017), or three groups (sweet likers, neutral and dislikers) (Garneau et al., 2018; Iatridi et al., 2019; Kim et al., 2014; Puputti, Aisala, Hoppu, and Sandell, 2018).

There are some limitations that need to be considered. In this study, all taster groups were described as either 'liker' or 'disliker' groups for ease of interpretation. However, when comparing the average hedonic liking rating at each concentration level, the group 'bitter likers' may be more accurately described as 'bitter neutral'. In addition, the group 'milk fat dislikers' may be more accurately described as 'milk fat neutral'. In sweet taste studies where three groups are derived from hierarchical cluster analysis, the intermediate group or taster 'neutral' group is a distinct and important phenotype (Garneau et al., 2018; Iatridi et al., 2019; Kim et al., 2014). It is acknowledged that in this study the lack of an intermediate phenotype for each taste type may have obfuscated potential relationships between each phenotype and health outcomes of interest.

In this study the sweet taste stimulus chosen was glucose because glucose is a simple sugar/monosaccharide that has clearly defined metabolic links and glucose sensors are ubiquitous throughout the body, including the GI tract and hypothalamus (Jayasinghe et al., 2017). However, the majority of previous studies have used sucrose as a sweet taste stimulus (Cox et al, 2015, Tan and Tucker, 2019). The choice of sweet stimulus and the range of concentrations tested (0 g/L – 240 g/L) may partially explain the lack of an intermediate or neutral phenotype derived from the hierarchical cluster analysis. A broader range of sweetness or the use of sucrose, which is perceived differently to glucose (Peng, Hautus, Oey, and Silcock, 2016), may have also resulted in a decreased number of sweet taste dislikers, which would be more consistent with the proportion of sweet taste likers and dislikers found in data from the US (Garneau et al., 2018), Korea (Kim et al., 2017) and the UK (Iatridi et al., 2019) where typically 20-25% of participants are sweet dislikers. Future studies will continue to compare different types of sweeteners and how these influence sweet taste phenotype by hierarchical cluster analysis for clarification of these associations.

Measurements of milk fat perception (liking, intensity, and ranking task) were not a direct measurement of 'taste', primarily due to the presence of non-taste sensory inputs such as mouthfeel and aroma. Due to the study aims, it was inappropriate to measure the hedonic liking of a single fatty acid, as it is generally an unrecognisable taste which evokes 'irritation' or 'scratchy' sensations at suprathreshold levels (Burgess et al., 2018). In this study, the milk fat perception samples were administered without the use of a nose-clip. For measurements of hedonic liking, it seemed inappropriate to block the nares as the true experience and sensation of fat in the mouth includes aroma (Boesveldt & Lundström, 2014). Further, in previous studies conducted in our lab (Kindleysides et al., 2017), we found that nose-clips can cause moderate discomfort and sinus irritation which in some cases causes a runny nose. Therefore, we chose to create an environment which enhanced engagement and minimised fatigue, particularly as the study included rating the hedonic liking of solutions. However, in order to determine associations with adiposity and markers of metabolic health, we recommend that future studies incorporate a measurement of fatty acid taste perception (Kindleysides et al., 2017; J. Stewart et al., 2010).

A further limitation of this study is the cross-sectional design, which was chosen to characterise women with different body fat measurements (normal and obese) in two population groups (Pacific and NZ European). The allocation of participants into distinct categories (i.e. lower BF% and higher BF%) allows for stratification and effect modification. A cross-sectional study does not infer causality (Hanage, 2014), however, it is a highly efficient approach that may help to inform future longitudinal and intervention studies.

In New Zealand, health inequities exist between population groups which are impacting on health and wellbeing, resulting in weight gain, obesity and the onset of poor metabolic health outcomes (Roberto et al., 2015). Of major concern is that these health inequities are impacting younger adults, adolescents, and children (Schwartz et al., 2017; Tupai-Firestone et al., 2016; Verbiest et al., 2018). Poor metabolic health outcomes are further influenced by ethnic-cultural and socio-economic characteristics (Dubé, 2010; Wen, Rush, & Plank, 2010).

Government and political actions, such as the introduction of a ‘sugar tax’, could be a successful approach to reduce population weight gain (Backholer et al., 2016; Nakhimovsky et al., 2016). Weight loss interventions, which may further improve sweet taste acuity, may have the long-term effect of reducing hyperinsulinemia and hyperleptinemia which would improve cephalic responses and regulatory actions determined by the hypothalamus (Amitani et al., 2013). Improved appetite response mechanisms and signalling by the hypothalamus may diminish food reward and enhance the response to satiety signals that are generated during food consumption (Farooqi et al., 2007). Future studies may look at determining the most efficient intervention strategies to improve insulin sensitivity to reverse the cascade of metabolic dysregulation occurring prior to the onset of metabolic disease. For example, one previous intervention study demonstrated the successful enhancement of the perception of sweet-taste intensity ratings with dietary intervention (Wise, Nattress, Flammer, & Beauchamp, 2016). Taste modification could be administered in the form of a pill or functional food ingredient, such as taste-modifying proteins similar to that of ‘Miraculin’ (Misaka, 2013; Swamy, Hadi, Sekaran, & Pichika, 2014), alongside dietary intervention and weight loss.

4.5.7. Conclusions

In this study, we found that decreased sweet taste perception is associated with higher body fat mass. In contrast, increased bitter taste liking was associated with lower body fat mass. Our data show a relationship between obesity and sweet taste perception. Given the cross-sectional nature of this study, our results do not allow us to explore the direction of this relationship, whether increasing obesity may influence sweet taste perception through dysregulation of the insulin-leptin feedback system (Amitani et al., 2013; Behrens & Meyerhof, 2019), or *vice-versa*, whether an enhanced sweet liking may cause obesity. Importantly, the data presented in this paper suggest that the hyperinsulinemia and hyperleptinemia in the obese state is linked with an endocrine desensitisation of the sweet taste receptor. It is tempting to speculate that alterations in signal transduction pathways at the sweet taste receptors in the obese state may lead to long-term changes of taste preferences, thus contributing to a cycle of unhealthy food

choices and disturbed appetite regulation and further increasing adiposity. The sweet taste receptor may be a powerful candidate and a potential target for future weight control interventions. The therapeutic potential of the taste receptor system is yet to be fully explored, but as the first point of contact between the food we eat and the endocrine regulation of adiposity, it could be a promising focus for future metabolic health research.

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Suppl 4.1. Associations with bitter taste and milk fat perception liking groups and plasma blood glucose and cholesterol markers (N= 304§).

	Pacific # (n= 142)	NZ European # (n= 162)	TOTAL ^ (n= 304)
	Bitter likers versus dislikers (OR, 95% C.I.)		
Glucose (mmol/L)	0.67 (0.27, 1.36)	1.59 (0.68, 3.70)	1.00 (0.55, 1.82)
HbA1c (mmol/mol)	0.79 (0.30, 2.07)	1.07 (0.44, 2.64)	0.94 (0.48, 1.83)
HDL (mmol/L)	1.36 (0.54, 3.38)	1.88 (0.74, 4.79)	1.76 (0.93, 3.32)
LDL (mmol/L)	1.00 (0.40, 2.52)	0.44 (0.18, 1.05)	0.60 (0.32, 1.13)
Total cholesterol (mmol/L)	0.98 (0.38, 2.53)	0.76 (0.32, 1.81)	0.80 (0.42, 1.50)
Triglycerides (mmol/L)	1.32 (0.53, 3.27)	1.00 (0.44, 2.30)	1.17 (0.64, 2.15)
	Bitter ranking task incorrect versus correct (OR, 95% C.I.)		
Glucose (mmol/L)	0.74 (0.36, 1.52)	1.47 (0.78, 2.78)	1.10 (0.69, 1.76)
HbA1c (mmol/mol)	**2.20 (1.02, 4.73)***	0.64 (0.32, 1.29)	1.13 (0.67, 1.89)
HDL (mmol/L)	1.03 (0.48, 2.20)	0.90 (0.47, 1.71)	0.94 (0.58, 1.52)
LDL (mmol/L)	1.52 (0.71, 3.22)	0.90 (0.47, 1.74)	1.12 (0.69, 1.82)
Total cholesterol (mmol/L)	1.59 (0.73, 3.46)	0.76 (0.39, 1.47)	1.03 (0.63, 1.69)
Triglycerides (mmol/L)	0.80 (0.39, 1.66)	0.97 (0.52, 1.82)	0.90 (0.56, 1.44)
	Milk fat likers versus dislikers (OR, 95% C.I.)		
Glucose (mmol/L)	1.11 (0.53, 2.31)	1.04 (0.50, 2.12)	1.06 (0.63, 1.76)
HbA1c (mmol/mol)	0.58 (0.25, 1.36)	0.72 (0.33, 1.55)	0.65 (0.37, 1.13)
HDL (mmol/L)	1.09 (0.50, 2.37)	0.80 (0.38, 1.68)	0.94 (0.56, 1.60)
LDL (mmol/L)	1.47 (0.68, 3.18)	1.29 (0.61, 2.73)	1.38 (0.81, 2.34)
Total cholesterol (mmol/L)	1.50 (0.68, 3.34)	1.27 (0.59, 2.72)	1.38 (0.80, 2.37)
Triglycerides (mmol/L)	0.77 (0.36, 1.61)	0.72 (0.35, 1.47)	0.74 (0.44, 1.23)
	Milk fat ranking task incorrect versus correct (OR, 95% C.I.)		
Glucose (mmol/L)	0.58 (0.27, 1.24)	0.71 (0.38, 1.34)	0.63 (0.39, 1.01)
HbA1c (mmol/mol)	1.06 (0.47, 2.43)	0.96 (0.48, 1.92)	1.03 (0.61, 1.72)
HDL (mmol/L)	1.21 (0.54, 2.70)	1.33 (0.70, 2.53)	1.43 (0.87, 2.33)
LDL (mmol/L)	0.77 (0.35, 1.68)	1.43 (0.74, 2.77)	1.01 (0.62, 1.64)
Total cholesterol (mmol/L)	1.25 (0.56, 2.80)	1.34 (0.69, 2.61)	1.18 (0.71, 1.94)
Triglycerides (mmol/L)	1.13 (0.53, 2.41)	1.18 (0.63, 2.20)	1.21 (0.75, 0.94)

Models adjusted for # age and NZDep2013; † age, NZDep2013, and ethnicity; ^ age, NZDep2013, BF% and ethnicity. *P< 0.05, **P< 0.01, ***P< 0.001. OR: odds ratio; CI confidence interval. §Calculated from complete datasets only (i.e. excludes dropouts and participants with missing blood data).

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Chapter V.

Associations between taste perception, dietary intake and eating behaviour in Pacific and NZ European women

5.1. Abstract

Taste perception may influence dietary preferences, potentially contributing to increased dietary intake of energy dense and nutrient poor foods, which may consequently contribute to the development of obesity. The aims of this study are to characterise sweet taste, bitter taste and milk fat perception in two populations with markedly different metabolic disease risk (Pacific and European women) and different levels of body fat (normal and obese) and to explore the association of taste perception with dietary intake and eating behaviour.

Sweet taste perception, assessed as intensity, hedonic liking and discrimination of taste concentration by ranking task was investigated in 304 women (18 − 45 years) selected based on ethnicity (47% Pacific and 53% NZ European) and body fat percentage (body fat %; 51% normal and 49% high, using a cut-point of 35% body fat). Body fat percentage (BF%) was measured by dual-energy x-ray absorptiometry (DXA). Hedonic liking and intensity ratings of taste were investigated using the labelled affective magnitude (LAM) and general labelled magnitude scales (gLMS). Hierarchical cluster analysis was used to obtain taste liking clusters of 'likers' and 'dislikers' from LAM ratings. Dietary intake was assessed using a 5-day estimated food record to measure total energy and macronutrient intake. Eating behaviour was assessed by the three-factor eating questionnaire. Socioeconomic status was measured by deprivation index

(NZDep2013).

In NZ European women, sweet likers had a significantly higher intake of carbohydrates, sugars and starch (199.4 ± 51.1, 87.9 ± 27.4 and 111.1 ± 34.6 g/day) when compared to sweet taste dislikers (165.9 ± 48.7, 71.4 ± 25.2 and 94.1 ± 34.7 g/day, respectively; $P< 0.001$, $P< 0.01$, and $P< 0.01$). In contrast, in Pacific women there was no difference in dietary intake between sweet liking groups. NZ European women in the sweet liking group had an increased disinhibited eating behaviour score (adjusted, $P< 0.01$). Pacific (>35 BF%) and NZ European (<35 BF%) bitter likers had a decreased protein intake (g) compared to bitter dislikers (adjusted, $P< 0.05$). NZ European bitter likers had a higher intake of dietary fibre (% energy) than bitter dislikers (adjusted, $P< 0.01$). Pacific milk fat likers with >35 BF% had a higher intake of protein (g) than milk fat dislikers (adjusted, $P< 0.05$).

The present study provides evidence that sweet taste perception and hedonic liking of taste at suprathreshold concentrations are associated with carbohydrate and sugar intake, which is further associated with disinhibited eating behaviour. It further reveals ethnic group-specific differences in the association between taste perception and dietary intake.

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5.2. Introduction

Obesity is a global pandemic with a multitude of complex interrelated causes. One of the key drivers is the over-consumption of highly palatable, inexpensive, energy-dense and nutrient poor foods (Swinburn et al., 2011; Vandenbroeck, Goossens, and Clemens, 2007). Furthermore, in New Zealand, major health inequities appear to be linked with obesity. Pacific people are disproportionately affected and 2.5 times more likely to be obese than NZ European people (Ministry of Health, 2016; Sundborn et al., 2010).

Taste perception has been proposed to contribute to weight gain and obesity, as taste preferences influence food choice (Loper, La Sala, Dotson, and Steinle, 2015). A number of studies have shown a range of associations between taste perception, dietary intake, appetite regulation and eating behaviour (Han, Keast, and Roura, 2017; Nakamura et al., 2008; Pasquet, Laure Frelut, Simmen, Marcel Hladik, and Monneuse, 2007; Sanematsu, Nakamura, Nomura, Shigemura, and Ninomiya, 2018; Stafford, Tucker, and Gerstner, 2013). Dietary interventions focusing on low fat and sugar intakes have shown an increase in fatty acid taste sensitivity (Costanzo et al., 2018; Stewart and Keast, 2012), and improved sweet taste acuity respectively (Wise, Nattress, Flammer, and Beauchamp, 2016). Cross-sectional studies have found that increased fatty acid taste sensitivity is associated with decreased dietary fat intake (Heinze et al., 2018; Keller et al., 2012; Liang et al., 2012; Martínez-Ruiz, López-Díaz, Wall-Medrano, Jiménez-Castro, and Angulo, 2014; Stevenson et al., 2016) or energy intake (Stewart et al., 2010). However, inconclusive and contradictory results have been observed. Some cross-sectional studies have found increased sweet taste perception to be associated with a reduced intake of sweet-tasting food (Jayasinghe et al., 2017; Low, Lacy, McBride, and Keast, 2016; Martinez-Cordero, Malacara-Hernandez, and Martinez-Cordero, 2015), while other studies have found no association (Cicerale, Riddell, and Keast, 2012; Leong, Forde, Tey, and Henry, 2018). Few studies to date have compared the outcomes of different taste perception measurements (e.g. sweet, bitter, fat) to each other and how they might relate to dietary intake and eating behaviour.

Differences and limitations in study design may contribute to the variations in findings from different studies, including study participants (e.g. age, gender, ethnicity), assessment methods of taste perception (i.e. the concentration of stimuli), and dietary intake assessment methods (Cox, Hendrie, and Carty, 2015). For example, a number of studies measuring taste perception have used food frequency questionnaires (FFQs). Whilst FFQs have low participant and researcher burden, they are prone to inaccurate reporting of quantities consumed due to their reliance on memory (Garneau, Nuessle, Mendelsberg, Shepard, and Tucker, 2018; Low et al., 2016; Methven, Xiao, Cai, and Prescott, 2016; Thompson and Subar, 2013). Food records are more accurate for measuring macronutrient intake (Bingham et al., 1994; Thompson and Subar, 2013). In addition, few studies on taste perception account for the importance of external influences on dietary intake such as socio-cultural and socio-economic factors which may impact on taste-diet associations (Baumann, Szabo, and Johnston, 2017; Low et al., 2016; Overberg, Hummel, Krude, and Wiegand, 2012).

Increasing evidence demonstrates links between higher detection threshold levels (low taste sensitivity) to primary tastes and a higher body mass index (BMI) (Donaldson, Bennett, Baic, and Melichar, 2009; Park et al., 2015). Additionally, studies have shown a link between BMI and the hedonic liking of sweetness (Bartoshuk, Duffy, Hayes, Moskowitz, and Snyder, 2006; Deglaire et al., 2015; Ettinger, Duizer, and Caldwell, 2012; Proserpio et al., 2016). There is increasing interest in the role of sweet, bitter and fatty acid taste perception and together how these mechanisms may impact on energy intake (Burgess, Rao, and Tepper, 2016).

Currently, there are few studies that have compared associations between taste perception and dietary intake by obesity status (Martinez-Cordero et al., 2015; Tucker, Edlinger, Craig, and Mattes, 2014). Investigation of taste perception status in non-obese and obese groups is an important development as weight status may impact taste perception (Tyrovolas, Koyanagi, Stickley, and Haro, 2015). In addition, the investigation of different population groups which have different metabolic disease risk profiles may provide new insights. Taste perception is variable between people for a number of other reasons, including genetics (Behrens and Meyerhof, 2019; Neiers, Canivenc-Lavier, and Briand, 2016; Risso et al., 2017; Sable, Warren, DuFlo,

Bartoshuk, and Skarulis, 2012). For example, differences in taste perception associated with genetics have been observed between different ethnic groups (Sable et al., 2012), however, few studies have investigated ethnic differences in taste perception in relation to dietary intake (Williams, Bartoshuk, Fillingim, and Dotson, 2016).

Taste perception may also influence eating behaviour. The modulation of hunger, satiety and energy balance is associated with eating behaviour, which is further influenced by taste perception (Tomassini Barbarossa et al., 2013). Previous studies have found associations between taste perception and eating behaviour (Kindleysides et al., 2017; Stafford et al., 2013; Tomassini Barbarossa et al., 2013). However, there is a lack of studies which compare taste perception and eating behaviour alongside other influences on energy intake, such as body weight.

This study is the first to characterise sweet taste, bitter taste and milk fat perception in two population groups with markedly different metabolic disease risk, Pacific and NZ European women, and different levels of body fat, obese and non-obese. The specific aims of this study are (1) to characterise in 18 to 45-year-old Pacific and NZ European women sweet taste, bitter taste and milk fat perception, and (2) explore the association of taste perception with dietary intake and eating behaviour. The potential identification of a distinct role for taste perception in modifying dietary intake and eating behaviour will advance our understanding of the aetiology of obesity and contribute to the development of new therapeutic approaches.

5.3. Materials and methods

5.3.1. Participants

Participants for this sub-analysis of the PROMISE (PRedictors linking Obesity and the gut MIcrobiomE) study, which investigated the role of the gut microbiome in obesity, were recruited in Auckland, New Zealand. Details of the study protocol and recruitment strategy have been published elsewhere (Kindleysides et al., 2019). The PROMISE study involved a range of assessments including diet, taste perception, eating behaviour and anthropometric measurements. Inclusion criteria were: women aged 18-45 years, being post menarche and pre-menopausal (as defined by a regular menstrual cycle for the last year), and ethnicity (self-identifying as NZ European and having lived in NZ for at least five years or self-identifying as Pacific and having at least one parent of full Pacific ethnicity). Participants were required to have a BMI categorised as normal (18.5 – 24.9 kg/m^2) or obese (≥ 30 kg/m^2), with the study designed so that approximately half in each ethnic group had a normal BMI, and the other half a BMI defined as obese. Exclusion criteria included pregnancy and lactation, presence of any diagnosed chronic illness (e.g. type 2 diabetes, CVD, cancer, etc), bariatric surgery, severe food allergies, severe dietary restriction or avoidances (e.g. vegan), taking medication that interferes with appetite or the immune system, antibiotic use in the last three months and smoking. Participants were either screened online, in-person or over the phone and self-reported height and weight were used to determine BMI. The protocol for this study was approved by the Southern Health and Disability Ethics Committee (16/STH/32) and written informed consent was provided by each participant. This study complies with the Declaration of Helsinki for medical research involving human subjects. The trial was registered at anzctr.org.nz as ACTRN12618000432213.

5.3.2. Study procedure

The study was conducted at the Massey University Human Nutrition Research Unit (HNRU) between July 2016 and September 2017. All participants attended two visits, with the second visit scheduled 11-14 days after the first. At visit one, participants

arrived at 07:00 am in a fasted state (not eating or drinking anything apart from water since 10pm the previous night) and were asked not to wear perfume, exercise or taste any other products prior to taste testing. Participants completed a health and demographic face-to-face interview, had their height and weight measured, underwent sensory testing (between 07:00 and 09:00) and completed the three-factor eating questionnaire (TFEQ). A 5-day food record was completed at home between visits 1 and 2. At visit 2, participants underwent a whole-body scan using dual-energy x-ray absorptiometry (DXA).

5.3.3. Health and demographic face-to-face interview and deprivation index

General participant information was obtained by a one-on-one interview which included questions about occupation, work patterns, dietary supplement use, frequency of alcohol consumption and recruitment method (not all data reported). The New Zealand index of deprivation 2013 (NZDep2013) was used as a measurement of socioeconomic status based on the participant's home address, which combines census data relating to income, home ownership, employment, qualifications, family structure, housing, access to public transport and communications (Atkinson, Salmond, and Crampton, 2014). NZDep2013 is a scale ranging from 1-10, with 1 representing the least deprived and 10 the most deprived levels (Atkinson et al., 2014).

5.3.4. Body composition and anthropometric measurements

Stretched height (stadiometer) and weight (Sauter platform scale E1200, GmbH, Germany) were measured. BMI was calculated as weight (kg) / height (m^2). A whole-body scan was performed using dual-energy x-ray absorptiometry (DXA) (Hologic QDR Discovery A, Hologic Inc, Bedford, MA with APEX V. 3.2 software) to obtain data on body composition using a standardised protocol.

5.3.5. Taste perception measurements

Three measurements of taste perception were performed by sensory testing; hedonic

liking rating, intensity rating and discrimination of taste concentration by ranking task. All three psychophysical tests were conducted for sweet taste, bitter taste and milk fat perception. All stimuli were evaluated at room temperature (20°C) in individual taste testing booths as whole mouth samples which were evaluated using a sip-and-spit procedure with no solution ingested (Martinez-Cordero et al., 2015; Mattes, 2009). Between each set of tasting stimuli, participants rinsed their palate with water and expelled the water. Prior to sensory testing, a short training procedure was conducted to familiarise each participant with the general labelled magnitude scale (gLMS) and labelled affective magnitude (LAM) scale independently (intensity and hedonic liking, respectively). The blank water solution (labelled "sweet") was tasted first as a control, followed by all sweet taste solutions, all bitter taste and finally all milk fat solutions. Glucose was the chosen tastant for sweet taste, as it has been used in similar studies and cited as having clearly defined links with sweet taste perception being a simple sugar (Jayasinghe et al., 2017). Quinine was the chosen tastant for bitter taste (Rawal, Hoffman, Honda, Huedo-Medina, and Duffy, 2015) and milk and cream were chosen to create a series of milk fat perception tastants (Zhou, Shen, Parker, Kennedy, and Methven, 2016). Milk and cream is a complex tastant due to comprising at least three further elements responsible for the sensory properties of milk, including: (1) mouthfeel and texture due to presence of macromolecules, such as fat globules and colloidal proteins, (2) salt and sweet taste due to milk salts and lactose, respectively, and (3) aroma (Cadwallader, 2010).

5.3.5.1. Preparation of sweet taste, bitter taste and milk fat perception solutions

Solutions for taste testing were prepared on the day of testing. Sweet taste solutions were prepared by dissolving glucose (dextrose monohydrate, Sherratt ingredients, Auckland, New Zealand) in distilled water. Bitter taste solutions were prepared from a stock solution made from distilled water and quinine hydrochloride dehydrate (Sigma-Aldrich, St Louis MO, USA) and refrigerated. Fresh stock was prepared weekly. This was used to create a dilution series from 0.4 mL of 80 mM stock solution added to 200 mL of filtered water (0.16 mM solution/0.06 g/L).

Milk fat perception samples were made from full fat milk (Anchor™ Blue Top standardised milk, New Zealand) and cream (Anchor™ Fresh Cream, New Zealand). Mixtures of milk and cream were prepared on the day of testing and used once only after opening. Each individual sample was mixed by drawing the solution 5-6 times with a 10 mL pipette (Eppendorf Research® pipette, Hamburg, Germany).

5.3.5.2. Intensity and hedonic liking measurements of sweet taste, bitter taste and milk fat perception

Hedonic liking and intensity rating scales were evaluated simultaneously (i.e. LAM scale questions on the same single-sided page) for each of the individual solutions presented. Hedonic liking for each solution was rated using a LAM scale with the extreme anchors of 'strongest imaginable dislike of any kind' (scale score = -50) through to 'strongest imaginable like of any kind' (50). Intermediate labels on the gLMS liking scale were: 'very strongly dislike' (-26.5), 'strongly dislike (-17.5), 'moderately dislike' (-8.5), 'weakly dislike' (-3), 'neutral' (0), 'weakly like' (3), 'moderately like' (8.5), 'strongly like' (17.5), and 'very strongly like' (26.5) (Schutz and Cardello, 2001). Sweet taste intensity for each solution was rated using a gLMS with the extreme anchors 'no sensation' (scale score =0) to 'strongest imaginable sensation of any kind' (200), with intermediate labels 'barely detectable' (1.5), 'weak sensation' (6), 'moderate sensation' (17), 'strong sensation' (35), and 'very strong sensation' (53) (Bartoshuk et al., 2004).

Taste solutions were presented in a randomised manner, with no side-by-side comparisons and identified by a random 3-digit code. Participants initially sampled water (blank), followed by tasting the four individual 10 mL solutions of glucose dissolved in distilled water (30 g/L, 60 g/L, 120 g/L and 240 g/L). These concentrations were selected as they span a range of sweetness levels (Jayasinghe et al., 2017). Quinine samples were evaluated for hedonic liking and intensity of bitter taste, after evaluation of sweet taste samples, at four concentration levels in a randomised order (0.008 g/L, 0.016 g/L, 0.03 g/L and 0.06 g/L) using LAM and gLMS rating scales. Next, milk fat perception samples were evaluated for hedonic liking and intensity in a randomised order (3.3%, 11.8%, 20.3% and 37.3% milk fat).

5.3.5.3. Taste concentration ranking task

After tasting all the individual solutions of the same type, starting with glucose, four new solutions (30 g/L, 60 g/L, 120 g/L and 240 g/L glucose) were presented to perform the taste ranking task. All four solutions were presented at once, randomised and identified with unique 3-digit random codes. The four samples were tasted and placed in order from the highest to the lowest perceived concentration of taste. Similarly, after evaluation of the individual quinine solutions, the ranking task procedure took place for bitter taste (0.008 g/L, 0.016 g/L, 0.03 g/L and 0.06 g/L quinine). To end, milk fat perception ranking task was completed after evaluating the milk fat solutions. The milk fat ranking task solutions (3.3%, 11.8%, 20.3% and 37.3% fat) were covered in foil and tasted through a straw to prevent any visuals cues affecting the taste ranking.

5.3.6. Dietary intake

Participants were given a schedule to ensure that a non-consecutive 5-day food record was kept across both week- and weekend days. Dietary intake was recorded every second day with a minimum of one weekend day, with most participants recording two weekend days and three weekdays of food record data. The 5 days were selected in advance with research staff liaising with each participant to determine suitable data collection days. The 5 days were aligned with the wider PROMISE study at-home data collection protocol (Kindleysides et al., 2019). A 10-minute instructional video was viewed providing in-depth instructions and examples of how to keep a detailed estimated food record. Each participant received further training for estimating and documenting portion sizes. Each food record was reviewed by a registered dietitian at visit 2, in a one-on-one interview to identify any gaps, to clarify reported data, to clarify brands consumed, and to confirm portion size information by using visual aids (e.g. picture guides, measuring cups, spoons, etc). Additional ambiguities in the food record were clarified by the participant providing empty food packages, detailed recipes, food labels, or photographs of meals eaten (e.g. restaurant, takeaway and café meals).

5.3.7. Eating behaviour

Eating behaviour was assessed using the validated three-factor eating questionnaire (TFEQ) to measure cognitive dietary restraint (21 items), disinhibition of control (16 items) and susceptibility to hunger (14 items) (Kruger, De Bray, Beck, Conlon, and Stonehouse, 2016; Stunkard and Messick, 1985). The TFEQ was administered using an online questionnaire hosted on SurveyMonkey© survey software.

5.3.8. Data handling

5.3.8.1. Taste perception measurements

A hierarchical cluster analysis was performed on LAM scale hedonic ratings of 5 variables for sweet taste (5 concentrations), 4 variables for bitter taste (4 concentrations) and 4 variables for milk fat (4 concentrations) to allocate participants into liking clusters. Hierarchical cluster analysis examines the underlying structure of seemingly homogenous data whilst not requiring a *priori* decisions regarding the number of clusters (Garneau et al., 2018). Two clusters for each tastant were derived from the analysis, with one group labelled as ‘likers’ and the other group labelled as ‘dislikers’ based on the average LAM scale rating of liking of the group.

5.3.8.2. Dietary intake measurements

Nutrient analysis of the food record was performed using the Foodworks 9 (Xyris Software (Australia) Pty Ltd, Queensland, Australia) dietary analysis software, which uses FOODfiles 2016 (developed by the NZ Institute for Plant & Food Research and the NZ Ministry of Health) as a reference food composition table for analysis. Additionally, the Xyris database AusFoods 2017 and AusBrands 2017, which are based on the Australian food composition databases AUSNUT 2011-13 (developed by Food Standards Australia New Zealand) were used (Commonwealth Department of Health and Ageing, Ministry of Health NZ, 2018) when food and beverage data were unavailable through the NZ FOODfiles. The data were used to assess dietary adequacy

in terms of energy and nutrient intakes using the current Australia/NZ Nutrient Reference Values (NRV) (Commonwealth Department of Health and Ageing; Ministry of Health NZ, 2018).

Dietary data were assessed for over- and underreporting by assessing the plausibility of intakes and using epidemiological cut-offs; >2,100kJ and <27,000kJ, as the range for under and over reported energy intakes in this population group (Carroll et al., 2012; House, Shearrer, Boisseau, Bray, and Davis, 2018; Willett, 2013). No dietary data were excluded for the primary analysis (N=304). Sensitivity analysis was conducted to verify that removal of under- and over-reporters, as defined by the Goldberg criteria (Black, 2000; Goldberg et al., 1991), did not change key outcomes of the study (Suppl 5.6.). Goldberg cut-offs were calculated using a low-moderate physical activity (PAL) of 1.55 and the Schofield equation to calculate basal metabolic rate. The ratio of reported energy intake (EIrep): estimated basal metabolic rate (BMRest) gave the interval 1.04 – 1.90, which was used to determine plausible reporters.

5.3.8.3. Eating behaviour measurements

TFEQ scores were allocated for each category and associated subscales were calculated under each of the three factors (Bond, McDowell, and Wilkinson, 2001; Westenhoefer, 1991; Westenhoefer, Stunkard, and Pudel, 1999). Higher scores denoted higher levels of restrained eating, disinhibited eating and predisposition to hunger (Stunkard and Messick, 1985).

5.3.8.4. Body composition grouping

An obesity cut-off point for body fat percentage was obtained from the American Association of Clinical Endocrinologists and American College of Endocrinology (AACE/ACE) guidelines (obesity in women >35%) (Dickey et al., 1998; Jo and Mainous, 2018; Oliveros, Somers, Sochor, Goel, and Lopez-Jimenez, 2014). For this analysis, BF% was used instead of BMI to classify obesity status, because previous

studies have shown BMI can grossly misclassify population groups known to have higher metabolic disease risk (Hunma et al., 2016).

5.3.9. Statistical Analysis

Statistical analyses were performed using IBM SPSS software for Windows (version 24.0; SPSS Inc, Armonk, NY: IBM Corp, USA). Data were checked for normality using Shapiro-Wilks test. Descriptive statistics were reported as arithmetic means and standard deviation (SD). Descriptive unadjusted data were presented as medians (25^{th}, 75^{th} percentiles) and compared with Mann-Whitney U independent samples t-tests. Group differences were compared using chi-square tests. Unadjusted dietary intake data in bar graphs were presented as means (95% C.I.) and compared using independent samples t-tests.

Hierarchical cluster analysis was performed using LAM liking ratings for each taste. Distinct taste liker phenotypes were identified. This method derives meaningful groups (clusters) of participants using the agglomerative method with Ward's minimum variance algorithm (Asao et al., 2015). Clusters define those who shared similar liking patterns within each group but were heterogenous in the between-group contrasts. The squared Euclidean distance between pairs of cases or clusters and the between-groups (averages) linkage method were selected to assist with the merging process (Iatridi et al., 2019). These results were checked with the elbow method, silhouette method and gap statistics to observe the optimal number of clusters. Using the majority rule, two liking clusters for each tastant (sweet, bitter and milk fat) were selected (Rousseuw & Kaufman, 1990).

Assessment of linear trends between the concentrations of the different solutions and the taste perception measures was carried out using linear mixed models and random intercepts to account for repeated measurements within each individual. For example, this showed that sweet taste likers rated taste as significantly less intense at 60 g/L, 120 g/L and 240 g/L concentrations when compared to sweet dislikers ($P< 0.001$). Similarly, bitter likers rated all concentrations of taste as significantly less intense than bitter

dislikers ($P< 0.01$). In contrast, milk fat likers rated fat perception intensity as significantly more intense than milk fat dislikers at the two highest concentration levels only (20.3% and 37.3% fat, $P< 0.05$).

Univariate general linear models were used to estimate the effects of age, BF% and NZDep2013 on dietary intake (e.g. macronutrients). For general linear models, unstandardized coefficient estimates (B) were reported along with 95% C.I. Univariate general linear models for eating behaviour scores were stratified by ethnicity and the data were log-transformed where exponential of B (ratio) were reported along with 95% C.I. A P-value of <0.05 was considered statistically significant.

5.4. Results

5.4.1 Participant characteristics and dietary intake

In this study, Pacific women were younger ($P< 0.001$), lived in more socioeconomically deprived areas ($P< 0.001$) and had a higher BMI ($P< 0.01$) than NZ European women. Pacific women had higher intakes of carbohydrate (g), total sugar (g) and starch (g, % energy); and lower intakes of protein (% energy), fat (% energy), polyunsaturated fat (g, % energy), dietary fibre (g, % energy) and alcohol (g, % energy) compared with NZ European women ($P< 0.05$; Table 1). On average, all Pacific and NZ European women consumed more fat, saturated fat and total sugar; and had inadequate carbohydrate intakes ($P< 0.05$) compared with general recommendations (NHMRC, 2006; WHO, 2015). Only NZ European women with <35 BF% met the recommended intake for dietary fibre (Table 5.1).

In Pacific women, there were no differences between BF% groups in demographic or dietary intake measures. In NZ European women, those with >35 BF% had a higher NZDep2013 score ($P< 0.05$) and were older than women with <35 BF% ($P< 0.001$; Table 5.1). NZ European women with <35 BF% had lower saturated fat intake (% energy), higher polyunsaturated fat (% energy), dietary fibre (g, % energy) and alcohol intake (g) ($P< 0.05$) than those with >35 BF%.

Table 5.1. Participant characteristics, dietary intake and body fat percentage groups in Pacific (N=142) and NZ European women (N=162).

		Pacific			*NZ European*		
		Low body fat (<35%) Mean ± SD (n= 68)	**High body fat (>35%)** Mean ± SD (n= 74)	**TOTAL** Mean ± SD (n= 142)	**Low body fat (<35%)** Mean ± SD (n= 87)	**High body fat (>35%)** Mean ± SD (n= 75)	**TOTAL** Mean ± SD (n= 162)
Age (yrs)		24.9 ± 6.74 #	25.2 ± 5.81 #	25.0 ± 6.25‡	29.7 ± 6.53 †	33.5 ± 7.01	31.5 ± 7.00
NZDep2013		7.24 ± 2.55 #	7.86 ± 1.96 #	7.57 ± 2.27‡	3.77 ± 2.44 †	4.61 ± 2.30	4.16 ± 2.41
Total body fat (%)		30.0 ± 3.22 †,#	40.0 ± 3.81	35.2 ± 6.15	27.9 ± 4.42 †	41.1 ± 3.77	34.0 ± 7.80
BMI (kg/m^2)		25.8 ± 3.84 †,#	36.1 ± 6.18 #	31.2 ± 7.29‡	22.5 ± 2.11 †	33.7 ± 3.76	27.7 ± 6.33
Weight (kg)		73.7 ± 11.7 †,#	102.3 ± 19.2 #	88.6 ± 21.5‡	63.1 ± 7.53 †	94.4 ± 13.2	77.6 ± 18.9
Height (cm)		169.0 ± 6.96	168.3 ± 6.92	168.6 ± 6.92	167.3 ± 5.58	167.2 ± 7.0	167.2 ± 6.26
Macronutrient intake	***Reference values***						
Total energy (kJ)	8700 – 8800 kJ [1]	8904.4 ± 2727.8	8557.3 ± 2749.0	8723.5 ± 2734.7	8230.4 ± 1638.1	8245.5 ± 1729.7	8237.4 ± 1675.9
Protein (g)	46 g [2]	83.0 ± 27.3	82.8 ± 25.3	82.9 ± 26.2	83.6 ± 18.0	84.0 ± 17.9	83.8 ± 17.9
Protein (%)	15 – 25% [3]	16.0 ± 3.21 #	16.8 ± 3.35	16.4 ± 3.3‡	17.5 ± 3.61	17.6 ± 3.25	17.6 ± 3.44
Fat (g)	n/a	92.9 ± 30.9	89.6 ± 33.0	91.2 ± 32.0	89.7 ± 26.1	91.0 ± 30.4	90.3 ± 28.1
Fat (%)	20 – 35% [3]	38.6 ± 5.50	38.3 ± 4.84	38.4 ± 5.15‡	40.1 ± 7.59	40.0 ± 7.80	40.1 ± 7.66
Saturated fat (g)	n/a	36.0 ± 13.6	35.0 ± 14.1	35.5 ± 13.8	32.6 ± 11.6	36.2 ± 14.4	34.3 ± 3.06
Saturated fat (%)	<10% [3]	14.9 ± 2.71	14.9 ± 2.89	14.9 ± 2.79	14.6 ± 3.89 †	15.9 ± 4.01	15.2 ± 3.99
Polyunsaturated fat (g)	n/a	12.5 ± 4.62	11.3 ± 4.84	11.9 ± 4.75‡	14.0 ± 6.27	12.3 ± 5.81	13.2 ± 6.10
Polyunsaturated fat (%)	n/a	5.25 ± 1.49 #	4.94 ± 1.54	5.09 ± 1.52‡	6.21 ± 1.97 †	5.44 ± 1.95	5.85 ± 1.99
Monounsaturated fat (g)	n/a	34.9 ± 12.0	34.2 ± 13.6	34.6 ± 12.8	33.7 ± 10.0	33.1 ± 11.4	33.4 ± 10.7
Monounsaturated fat (%)	n/a	14.6 ± 2.52	14.6 ± 2.67	14.6 ± 2.59	15.1 ± 3.44	14.6 ± 3.44	14.9 ± 3.44
Carbohydrate (g)	n/a	223.3 ± 76.7 #	215.6 ± 76.4 #	219.3 ± 76.4‡	178.0 ± 53.1	184.5 ± 51.6	181.0 ± 52.4
Carbohydrate (%)	45 – 65% [3]	42.7 ± 7.05 #	42.9 ± 5.55 #	42.8 ± 6.29‡	36.8 ± 8.28	38.5 ± 8.94	37.6 ± 8.61
Total sugar (g)	n/a	92.3 ± 39.0 #	85.8 ± 40.0	88.9 ± 39.5‡	77.0 ± 26.6	81.0 ± 28.3	78.8 ± 27.4
Total sugar (%)	<10% [4]	17.6 ± 5.30	17.1 ± 5.30	17.3 ± 5.30	16.1 ± 4.90	17.0 ± 6.0	16.5 ± 5.42
Starch (g)	n/a	130.4 ± 46.8 #	129.6 ± 48.7 #	130.0 ± 47.7‡	100.5 ± 38.4	103.2 ± 32.1	101.8 ± 35.6
Starch (%)	n/a	25.0 ± 4.56 #	25.7 ± 4.34 #	25.3 ± 4.45‡	20.6 ± 6.32	21.5 ± 5.81	21.0 ± 6.09
Dietary fibre (g)	25g [5]	19.5 ± 6.96 #	18.6 ± 6.66 #	19.0 ± 6.80‡	25.8 ± 8.98 †	22.0 ± 6.50	24.1 ± 8.13
Dietary fibre (%)	n/a	1.78 ± 0.45 #	1.80 ± 0.52 #	1.79 ± 0.48‡	2.53 ± 0.74 †	2.19 ± 0.63	2.37 ± 0.71
Alcohol (g)	<20 g [6]	4.44 ± 14.0 #	1.50 ± 5.18 #	2.91 ± 10.5‡	9.28 ± 13.1 †	5.53 ± 10.4	7.55 ± 12.0
Alcohol (%)	n/a	1.16 ± 3.27 #	0.50 ± 1.71 #	0.81 ± 2.59‡	3.15 ± 4.38	1.92 ± 3.70	2.58 ± 4.12

Means ± SD. † $P< 0.05$ for t-test comparing body fat groups within the ethnic group. # $P< 0.05$ for t-test comparing between ethnic groups within body fat group. ‡ $P< 0.05$ for t-test comparing between ethnicity only.

1. Estimated energy requirement calculation based on women aged 19 – 50 years and physical activity level 1.6 (minimally active) (NHMRC, 2006).
2. RDI for women between 19 – 50 years, Australian National Health and Medical Research Council & New Zealand Ministry of Health (NHMRC, 2006).

3. AMDR for adults. Macronutrients expressed as a percentage of energy in kJ (NHMRC, 2006).

4. World Health Organisation (WHO) recommendation for ‘free sugar’ intake as a percentage of energy intake (not including intrinsic sugars) (WHO, 2015).

5. Adequate intake of dietary fibre (NHMRC, 2006).

6. Based on guidelines from the Ministry of Health, equivalent to no more than two standard drinks per day (Ministry of Health, 2019)

5.4.2. Participant eating behaviour and taste perception

In Pacific women, disinhibition scores were lower, and hunger scores higher when compared with NZ European women ($P< 0.05$). There was no difference in cognitive restraint scores between ethnicities (Table 5.2). Averages for restraint and disinhibition scores were within the low range in Pacific and NZ European women compared to reference values (Stunkard and Messick, 1985). However, Pacific women had a high hunger score (score 7; high range 7-14) and NZ European women a medium hunger score compared to reference values (score 5; medium range 4-6) (Table 5.2).

Disinhibited eating behaviour was highest in NZ European women with >35 BF% and was in the medium range ($P <0.05$) (score 11; medium range 9-12). In Pacific women, those with >35 BF% had a higher disinhibition score than those with <35 BF% ($P< 0.05$). In NZ European, women with >35 BF% had higher disinhibition, lower restraint and higher hunger score compared to NZ European with <35 BF% ($P< 0.05$; Table 5.2).

There were no significant differences in the number of likers and dislikers of sweet taste, bitter taste or milk fat perception when comparing BF% groups or ethnicity (Table 5.2). Most participants correctly discriminated sweet taste concentration by ranking task (>73%; Table 2). In contrast, less than half of the participants correctly ranked the bitter taste task, and on average less than half correctly ranked the milk fat ranking task (Table 5.2). Pacific women with <35 BF% were more likely to correctly rank the sweet taste ranking task than Pacific women with >35 BF% ($P< 0.05$).

Table 5.2. Eating behaviour scores and taste perception parameters in Pacific (N=142) and NZ European women (N=162).

	Pacific			***NZ European***		
	Low BF (<35%) N=68 Median (25^{th}, 75^{th} percentiles)	**High BF (>35%)** N=74 Median (25^{th}, 75^{th} percentiles)	**TOTAL** N= 142 Median (25^{th}, 75^{th} percentiles)	**Low BF (<35%)** N=87 Median (25^{th}, 75^{th} percentiles)	**High BF (>35%)** N=75 Median (25^{th}, 75^{th} percentiles)	**TOTAL** N= 162 Median (25^{th}, 75^{th} percentiles)
Eating behaviour						
Cognitive dietary restraint[1]	6.0 (4.0, 10) #	7.0 (5.0, 11)	7.0 (4.0, 10.5)	9.0 (6.0, 12) †	7.0 (5.0, 10)	8.0 (5.0, 11.0)
Flexible control restraint	2.0 (1.0, 4.0) #	3.0 (1.0, 4.0) #	2.0 (1.0, 4.0)	3.0 (2.0, 4.0) †	2.0 (1.0, 3.0)	2.0 (1.0, 4.0)
Rigid control restraint	1.0 (0.0, 3.0) #	2.0 (1.0, 4.0)	1.5 (1.0, 3.0)‡	3.0 (2.0, 4.0)	2.0 (1.0, 3.0)	3.0 (2.0, 4.0)
Disinhibition[2]	6.0 (4.0, 9.0) †	8.0 (6.0, 10.0) #	7.0 (5.0, 9.0)‡	7.0 (4.0, 10.0) †	11 (8.0, 13.0)	8.0 (5.0, 12.0)
Habitual disinhibition	1.0 (1.0, 3.0)	2.0 (1.0, 3.0) #	1.5 (1.0, 3.0)	1.0 (0.0, 2.0) †	3.0 (1.0, 4.0)	1.0 (0.0, 3.0)
Emotional disinhibition	0.0 (0.0, 2.0)	1.0 (0.0, 2.0) #	1.0 (0.0, 2.0)‡	1.0 (0.0, 2.0) †	3.0 (2.0, 3.0)	2.0 (0.0, 3.0)
Situational disinhibition	3.0 (1.0, 4.0)	3.0 (2.0, 4.0) #	3.0 (2.0, 4.0)‡	3.0 (2.0, 4.0) †	4.0 (3.0, 5.0)	3.0 (2.0, 4.0)
Hunger[3]	6.0 (3.0, 8.0)	7.0 (5.0, 10)	7.0 (4.0, 9.0)‡	4.0 (3.0, 8.0) †	6.0 (4.0, 9.0)	5.0 (3.0, 9.0)
Internal locus hunger	2.0 (1.0, 4.0) †	3.0 (2.0, 5.0) #	3.0 (1.0, 4.0)‡	2.0 (1.0, 3.0)	2.0 (1.0, 4.0)	2.0 (1.0, 4.0)
External locus hunger	2.0 (1.0, 4.0)	3.0 (1.0, 4.0)	2.0 (1.0, 4.0)	2.0 (1.0, 3.0) †	3.0 (1.0, 4.0)	2.0 (1.0, 4.0)
Taste clusters [§]	*n (%)*	*n (%)*	*n (%)*	*n (%)*	*n (%)*	*n (%)*
Sweet likers (group)	25 (37%)	27 (37%)	52 (37%)	34 (39%)	38 (51%)	73 (45%)
Bitter likers (group)	16 (24%)	9 (12%)	25 (18%)	18 (21%)	10 (13%)	28 (17%)
Milk fat likers (group)	50 (74%)	48 (65%)	98 (69%)	63 (72%)	54 (72%)	117 (72%)
Taste ranking task						
Sweet (correct)	61 (90%) †	54 (73%)	115 (81%)	76 (87%)	58 (78%)	134 (83%)
Bitter (correct)	20 (29%) [#]	26 (35%)	46 (32%)‡	43 (49%)	37 (49%)	80 (49%)
Milk fat (correct)	27 (40%)	20 (27%) [#]	47 (33%)‡	37 (43%)	40 (53%)	77 (48%)

Median (25^{th}, 75^{th} percentiles) Mann-Whitney U test. † $P< 0.05$ for test comparing body fat groups within the ethnic group. [#] $P< 0.05$ for test comparing between ethnic groups within body fat group. ‡ $P< 0.05$ for t-test comparing ethnicity only. [1]Restraint reference values = Low, 4-14; Medium, 15-17; High, 18-21. [2]Disinhibition reference values = Low, 1-8; Medium, 9-12; High, 13-16. [3]Hunger reference values = Low, 0-3; Medium, 4-6; High, 7-14 (Stunkard and Messick, 1985).
[§]Sweet taste likers rated taste as significantly less intense at 60 g/L, 120 g/L and 240 g/L concentrations when compared to sweet dislikers ($P< 0.001$). Bitter likers rated all concentrations of taste as significantly less intense than bitter dislikers ($P< 0.01$). In contrast, milk fat likers rated fat perception intensity as significantly more intense than milk fat dislikers at the two highest concentration levels only (20.3% and 37.3% fat, $P< 0.05$).

5.4.3. Taste perception and dietary intake

Across all participants (N= 304), sweet likers had a higher percentage of carbohydrate intake compared to sweet dislikers (mean 41 versus 39%, $P< 0.05$). Conversely, sweet likers had a decreased intake of fat (% energy) compared to sweet dislikers (mean 38 versus 40%, $P< 0.01$). However, there was no significant difference in the grams of carbohydrate (mean 202.9 versus 196.1 g) or fats (mean 88.3 versus 92.3 g) when comparing sweet likers and dislikers, respectively ($P> 0.05$).

When stratified by ethnic group, NZ European sweet likers had a significantly higher intake of carbohydrates, sugars and starch (199, 88 and 111 g/day respectively) when compared to sweet taste dislikers (166, 71 and 94 g/day, respectively; $P< 0.01$). As a percentage of energy, carbohydrates, sugars and starch intake were also significantly higher in NZ European sweet likers (41, 18 and 23% respectively) than in dislikers (35, 15 and 20% respectively, $P< 0.01$). There were no significant differences between Pacific sweet likers and sweet dislikers when comparing intake of macronutrients in grams or percentage of energy (unadjusted, Figure 1). There were no clear differences found for either bitter or milk fat hedonic liking clusters in relation to macronutrient (grams and percentage) or total energy intake in either ethnic group (Figure 5.1).

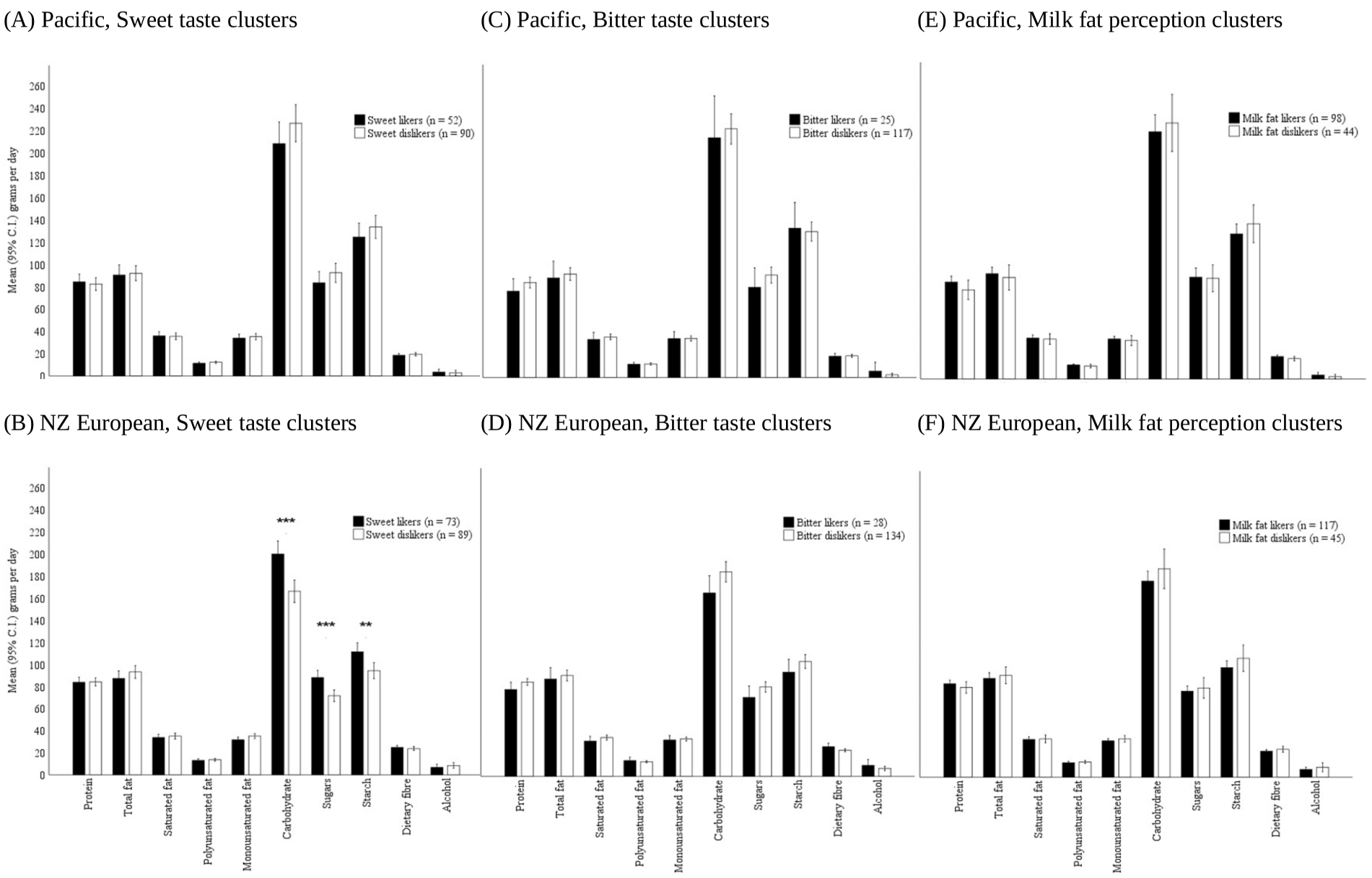

Figure 5.1. Comparison of daily macronutrient intake between likers and dislikers (sweet taste, bitter taste and milk fat perception) in (A, C, E) Pacific (N=142) and (B, D, F) NZ European (N= 162), unadjusted. Means (95% C.I.). C.I., confidence interval. Independent samples t-test. ***P*< 0.01, ****P*< 0.001.

5.4.4. Associations between taste perception, dietary intake and body fat % groups

Linear regression analysis showed that Pacific sweet likers had a significantly higher protein intake (% energy), after adjusting for age, BF% and NZDep2013 ($P< 0.01$) and this effect was most pronounced in Pacific women with <35 BF% (Table 5.3). However, there were no other significant differences in total energy or macronutrient intake between sweet likers and dislikers in Pacific women (Table 5.3). Sensitivity analysis by removal of Pacific under- and over-reporters (n=53), as defined by the Goldberg equation, did not change the significant differences observed between sweet likers and dislikers in Pacific women (Suppl 5.6).

NZ European sweet likers had higher carbohydrate, sugar and starch intake compared to sweet dislikers, after adjustment for age, BF% and NZDep2013 (Table 5.3). NZ European sweet likers had a significantly decreased intake of fat and monounsaturated fat (% energy) (adjusted, $P< 0.01$; Table 3). Sugar (as % energy) and starch intake (in g) were significantly higher for sweet likers versus dislikers in NZ European women with >35 BF% ($P< 0.05$), but not in NZ European women with <35 BF% (Table 5.3). Sensitivity analysis by removal of NZ European under- and over-reporters (n=36), as defined by the Goldberg equation, did not change the significant differences observed between sweet likers and dislikers in NZ European women (Suppl 5.6).

Pacific (>35 BF%) and NZ European (<35 BF%) bitter likers had a decreased protein intake (g) compared to bitter dislikers ($P< 0.05$; Suppl 5.1). NZ European bitter likers had a higher intake of dietary fibre (% energy) than bitter dislikers ($P< 0.01$; Suppl 5.1). Pacific bitter likers (>35% BF%) had a higher alcohol intake (% energy) than bitter dislikers in the same BF% group ($P< 0.05$; Suppl 5.1). There were no other clear associations found in relation to bitter liking clusters in macronutrient or total energy intake within each BF% group in either NZ European or Pacific women (Suppl 5.1).

Pacific milk fat likers with >35 BF% had a higher intake of protein (g) than milk fat dislikers ($P< 0.05$; Suppl 5.2). NZ European milk fat likers with >35 BF% had a lower alcohol intake than milk fat dislikers ($P< 0.05$; Suppl 5.2). There were no other clear

associations found in relation to milk fat liking clusters in macronutrient or total energy intake in either NZ European or Pacific women (Suppl 5.2).

5.4.5. Taste ranking task outcome and dietary intake

There were no associations found in relation to ranking task outcomes and macronutrient or total energy intake (Suppl 5.3).

Table 5.3. Associations between macronutrient intake and sweet liking in Pacific and NZ European women (N=304).

	Pacific (N= 142)			***New Zealand European (N= 162)***		
Macronutrient intake	Sweet liking (groups)§ Low body fat (<35%) (n= 68)	Sweet liking (groups)§ High body fat (>35%) (n= 74)	Sweet liking (groups)§ Pacific (all) (n= 142)	Sweet liking (groups)§ Low body fat (<35%) (n= 87)	Sweet liking (groups)§ High body fat (>35%) (n= 75)	Sweet liking (groups)§ NZ European (all) (n= 162)
	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^
Total energy (kJ)	-608.6 (-2056.7, 839.5)	-313.9 (-1670.1, 1042.3)	-458.8 (-1431.7, 514.1)	509.4 (-248.7, 1267.6)	156.0 (-641.0, 953.0)	271.6 (-260.7, 803.9)
Protein (g)	1.09 (-13.2, 15.4)	1.97 (-10.4, 14.4)	2.11 (-7.15, 11.4)	-1.22 (-9.65, 7.20)	0.32 (-8.12, 8.76)	-0.83 (-6.56, 4.90)
Protein (%)	**1.80 (0.23, 3.38)***	1.06 (-0.58, 2.70)	**1.53 (0.41, 2.66)****	-1.15 (-2.82, 0.53)	-0.47 (-1.95, 1.01)	-0.79 (-1.88, 0.29)
Fat (g)	-4.05 (-20.3, 12.2)	-2.74 (-18.9, 13.4)	-3.40 (-14.7, 7.90)	-2.07 (-14.1, 9.92)	-8.02 (-22.1, 6.06)	-5.09 (-13.9, 3.73)
Fat (%)	1.08 (-1.82, 3.99)	-0.22 (-2.56, 2.11)	0.30 (-1.50, 2.10)	**-3.97 (-7.29, -0.64)***	**-4.43 (-7.90, -0.96)****	**-3.99 (-6.29, -1.69)****
Saturated fat (g)	-0.99 (-8.11, 6.13)	-0.25 (-6.73, 7.23)	-0.20 (-5.11, 4.71)	-0.78 (-4.57, 6.13)	-2.80 (-9.52, 3.91)	-1.17 (-5.26, 2.92)
Saturated fat (%)	0.64 (-0.78, 2.05)	0.56 (-0.87, 1.98)	0.62 (-0.37, 1.61)	-0.93 (-2.70, 0.84)	-1.50 (-3.35, 0.35)	-1.18 (-2.41, 0.04)
Polyunsaturated fat (g)	-0.94 (-3.36, 1.47)	-1.48 (-3.77, 0.82)	-1.36 (-2.98, 0.27)	-0.15 (-3.06, 2.76)	-0.67 (-3.38, 2.04)	-0.38 (-2.29, 1.54)
Polyunsaturated fat (%)	-0.05 (-0.83, 0.72)	-0.41 (-1.14, 0.33)	-0.31 (-0.82, 0.21)	-0.44 (-1.36, 0.47)	-0.42 (-1.33, 0.49)	-0.39 (-1.00, 0.23)
Monounsaturated fat (g)	-2.08 (-8.48, 4.33)	-1.72 (-8.31, 4.87)	-1.95 (-6.50, 2.60)	-2.38 (-7.00, 2.24)	-4.01 (-9.28, 1.26)	-3.14 (-6.48, 0.20)
Monounsaturated fat (%)	0.26 (-1.10, 1.62)	-0.55 (-1.83, 0.73)	-0.23 (-1.15, 0.69)	**-2.14 (-3.66, -0.62)****	**-2.20 (-3.73, 0.67)****	**-2.07 (-3.10, -1.04)*****
Carbohydrate (g)	-26.3 (-66.2, 13.6)	-16.5 (-54.2, 21.3)	-21.4 (-48.3, 5.58)	**33.1 (9.61, 56.6)****	**29.9 (8.29, 51.4)****	**29.8 (14.3, 45.4)*****
Carbohydrate (%)	-2.94 (-6.39, 0.52)	-1.35 (-3.95, 1.26)	-2.04 (-4.13, 0.05)	**4.97 (1.42, 8.52)****	**5.76 (1.87, 9.66)****	**5.42 (2.90, 7.93)*****
Total sugar (g)	-15.5 (-34.4, 3.50)	-6.09 (-25.3, 13.1)	-11.4 (-24.8, 2.04)	**15.9 (4.09, 27.7)****	**15.0 (2.60, 27.3)***	**15.1 (6.88, 23.4)*****
Total sugar (%)	-2.24 (-4.80, 0.33)	-0.00 (-2.42, 2.41)	-1.16 (-2.93, 0.61)	2.18 (-0.01, 4.37)	**3.32 (0.62, 6.02)***	**2.93 (1.27, 4.58)****
Starch (g)	-10.51 (-35.6, 14.6)	-10.5 (-34.2, 13.3)	-9.82 (-26.8, 7.16)	17.0 (-0.56, 34.5)	**14.8 (0.81, 28.8)***	**14.6 (3.69, 25.5)****
Starch (%)	-0.62 (-2.99, 1.75)	-1.36 (-3.42, 0.70)	-0.85 (-2.37, 0.67)	2.75 (-0.07, 5.57)	2.43 (-0.23, 5.08)	**2.48 (0.62, 4.34)****
Dietary fibre (g)	-2.89 (-6.42, 0.65)	-0.01 (-3.20, 3.17)	-1.55 (-3.87, 0.77)	0.46 (-3.74, 4.66)	1.72 (-1.34, 4.78)	1.18 (-1.31, 3.67)
Dietary fibre (%)	-0.16 (-0.38, 0.06)	0.13 (-0.12, 0.38)	-0.01 (-0.18, 0.15)	-0.09 (-0.44, 0.25)	0.16 (-0.14, 0.45)	0.06 (-0.16, 0.28)
Alcohol (g)	-0.34 (-7.85, 7.17)	1.14 (-1.40, 3.68)	0.12 (-3.55, 3.80)	1.58 (-4.44, 7.60)	-2.44 (-7.44, 2.55)	-1.34 (-5.19, 2.52)
Alcohol (%)	0.18 (-1.56, 1.93)	0.37 (-0.46, 1.20)	0.21 (-0.70, 1.11)	0.30 (-1.72, 2.32)	-0.97 (-2.74, 0.79)	-0.65 (-1.96, 0.67)

General linear model coefficients and 95% CI adjusted for # age and NZDep2013 and for ^ age, NZDep2013 and BF% (groups). *P< 0.05, **P< 0.01, ***P< 0.001. BF%, body fat percentage. §Reference category: sweet dislikers, Pacific sweet dislikers n= 90; NZ European sweet dislikers n= 89. Statistically significant observations are highlighted in bold.

5.4.6. Relationships between eating behaviour and sweet taste, bitter taste and milk fat liking and ranking task outcome

NZ European sweet likers had a 19% increase in disinhibition score compared to NZ European sweet dislikers (adjusted, $P< 0.01$; Table 5.4), however, no differences were observed in Pacific women. In NZ European, situational disinhibition was significantly higher in sweet likers than dislikers, after adjusting for age, NZDep2013 and BF% (adjusted, $P< 0.01$; Table 5.4). Emotional disinhibition was 26% higher in NZ European sweet likers compared to NZ European dislikers without adjusting for BF% ($P< 0.05$), however adjustment for BF% attenuated this association (adjusted, $P> 0.05$; Table 5.4). No significant associations were found between sweet taste ranking task outcomes and eating behaviour scores, after adjustment for confounders.

Pacific bitter likers had a 34% higher habitual disinhibition score than Pacific bitter dislikers (adjusted ratio = 1.3, $P< 0.05$; Suppl. 5.4). In addition, Pacific milk fat likers had a 25% higher external locus hunger score compared to Pacific milk fat dislikers (adjusted ratio = 1.3, $P< 0.05$; Suppl 5.5). No other significant associations were found between bitter and milk fat taste liking groups and eating behaviour scores (Suppl. 5.4 and 5.5), or between bitter and milk fat ranking task outcomes and eating behaviour scores.

Table 5.4. Relationships between eating behaviour and sweet taste liking (compared to disliking) (N= 304).

	Unadjusted			***Adjusted***		
	Sweet liking (groups)	Sweet liking (groups)	Sweet liking (groups)	Sweet liking (groups)	Sweet liking (groups)	Sweet liking (groups)
Pacific (N = 142)	Low BF (<35%) (n= 68) Ratio (95% C.I.)	High BF (>35%) (n= 74) Ratio (95% C.I.)	TOTAL ‡ (n= 142) Ratio (95% C.I.)	Low BF (<35%) # (n= 68) Ratio (95% C.I.)	High BF (>35%) # (n= 74) Ratio (95% C.I.)	TOTAL ^ (n= 142) Ratio (95% C.I.)
Cognitive dietary restraint[1]	1.06 (0.77, 1.46)	1.08 (0.84, 1.40)	1.07 (0.88, 1.31)	1.05 (0.75, 1.46)	1.06 (0.82, 1.38)	1.05 (0.85, 1.29)
Flexible control restraint	1.07 (0.79, 1.44)	1.24 (0.95, 1.62)	1.15 (0.95, 1.41)	1.06 (0.78, 1.44)	1.23 (0.93, 1.61)	1.15 (0.94, 1.40)
Rigid control restraint	1.25 (0.89, 1.74)	1.00 (0.74, 1.34)	1.11 (0.89, 1.39)	1.25 (0.89, 1.75)	0.94 (0.70, 1.26)	1.09 (0.87, 1.35)
Disinhibition[2]	0.98 (0.76, 1.27)	0.96 (0.79, 1.17)	0.97 (0.83, 1.14)	0.95 (0.73, 1.25)	0.97 (0.80, 1.19)	0.96 (0.81, 1.12)
Habitual disinhibition	0.87 (0.66, 1.15)	0.86 (0.65, 1.14)	0.87 (0.71, 1.05)	0.85 (0.63, 1.13)	0.86 (0.64, 1.15)	0.86 (0.70, 1.05)
Emotional disinhibition	0.96 (0.74, 1.26)	1.16 (0.89, 1.52)	1.06 (0.88, 1.28)	1.00 (0.75, 1.32)	1.16 (0.88, 1.52)	1.08 (0.89, 1.30)
Situational disinhibition	1.11 (0.84, 1.45)	1.03 (0.84, 1.25)	1.07 (0.90, 1.26)	1.04 (0.79, 1.38)	1.05 (0.86, 1.28)	1.03 (0.87, 1.22)
Hunger[3]	1.28 (0.94, 1.73)	0.89 (0.72, 1.10)	1.06 (0.88, 1.27)	1.25 (0.91, 1.72)	0.90 (0.72, 1.12)	1.05 (0.87, 1.27)
Internal locus hunger	1.67 (0.85, 1.60)	0.84 (0.66, 1.07)	0.99 (0.81, 1.20)	1.13 (0.81, 1.59)	0.84 (0.65, 1.08)	0.96 (0.79, 1.18)
External locus hunger	1.19 (0.91, 1.57)	0.97 (0.76, 1.24)	1.07 (0.90, 1.29)	1.15 (0.87, 1.53)	1.00 (0.78, 1.28)	1.07 (0.89, 1.29)
NZ European (N= 162)	(n= 87)	(n= 75)	(n= 162)	(n= 87)	(n= 75)	(n= 162)
Cognitive dietary restraint[1]	0.92 (0.72, 1.18)	0.96 (0.75, 1.24)	0.94 (0.79, 1.12)	0.90 (0.67, 1.12)	0.95 (0.74, 0.23)	0.91 (0.76, 1.08)
Flexible control restraint	0.92 (0.75, 1.12)	1.04 (0.84, 1.29)	0.97 (0.84, 1.12)	0.86 (0.70, 1.05)	1.04 (0.83, 1.30)	0.94 (0.82, 1.09)
Rigid control restraint	0.91 (0.73, 1.14)	1.04 (0.82, 1.32)	0.97 (0.83, 1.14)	0.92 (0.73, 1.16)	1.02 (0.80, 1.30)	0.95 (0.81, 1.11)
Disinhibition[2]	**1.28 (1.04, 1.57)***	1.15 (0.98, 1.36)	**1.22 (1.07, 1.39)****	1.23 (1.00, 1.53)	1.16 (0.98, 1.38)	**1.19 (1.04, 1.36)****
Habitual disinhibition	1.15 (0.88, 1.50)	1.11 (0.84, 1.46)	1.13 (0.94, 1.37)	1.18 (0.90, 1.55)	1.15 (0.88, 1.52)	1.13 (0.93, 1.37)
Emotional disinhibition	1.16 (0.90, 1.50)	1.18 (0.96, 1.46)	1.17 (0.99, 1.38)	1.15 (0.88, 1.52)	1.19 (0.96, 1.48)	1.17 (0.99, 1.39)
Situational disinhibition	**1.24 (1.04, 1.48)***	1.18 (1.00, 1.39)	**1.21 (1.07, 1.37)****	1.20 (0.99, 1.45)	1.18 (1.00, 1.39)	**1.18 (1.04, 1.32)****
Hunger[3]	1.07 (0.85, 1.35)	1.11 (0.85, 1.46)	1.09 (0.92, 1.30)	1.06 (1.83, 1.34)	1.08 (0.82, 1.43)	1.05 (0.88, 1.25)
Internal locus hunger	1.03 (0.79, 1.34)	1.06 (0.78, 1.43)	1.04 (0.86, 1.27)	1.02 (0.77, 1.35)	1.04 (0.77, 1.42)	1.03 (0.84, 1.25)
External locus hunger	1.14 (0.90, 1.44)	1.18 (0.90, 1.54)	1.16 (0.97, 1.38)	1.12 (0.88, 1.44)	1.15 (0.89, 1.50)	1.10 (0.93, 1.32)

All dependent values log transformed. General linear model exponentiated coefficients and 95% CI adjusted for ‡ BF% group; # age, NZDep; for ^ age, NZDep2013 and BF% group. *P< 0.05, **P< 0.01, ***P< 0.001. C.I., confidence interval. BF%, body fat percentage. Reference category: sweet dislikers, Pacific sweet dislikers n= 90; NZ European sweet dislikers n= 89. [1]Restraint reference values = Low, 4-14; Medium, 15-17; High, 18-21. [2]Disinhibition reference values = Low, 1-8; Medium, 9-12; High, 13-16. [3]Hunger reference values = Low, 0-3; Medium, 4-6; High, 7-14 (Stunkard and Messick, 1985). Statistically significant observations are highlighted in bold.

5.5. Discussion

The present study aimed to increase our understanding of sweet taste, bitter taste and milk fat perception and associations with dietary intake, using a 5-day food record, and eating behaviour, using a three-factor eating questionnaire, in Pacific and NZ European women. These associations were explored in obese and non-obese participants. An adjustment was made for potential factors influencing dietary intake, including age, adiposity and socioeconomic status.

5.5.1. Participant demographics, dietary intake and eating behaviour

Associations between taste perception, dietary intake and eating behaviour were explored separately in Pacific and NZ European women. Pacific women in this study were younger, more likely to reside in a socioeconomically deprived area and had a higher BMI than NZ European women. NZ European women with obesity were older and had a significantly higher deprivation score than non-obese NZ European. However, there were no significant differences between any of the study groups (Pacific and NZ European; obese versus non-obese) for mean energy intakes. Our results are similar to energy intakes reported in previous studies (Pacific: 8700 to 10,300 kJ/day; NZ European: 8100 to 8500 kJ/day) (Metcalf et al., 2008; Ministry of Health, 2011). Participants consumed adequate protein but higher fat, saturated fat and sugar; and lower levels of carbohydrate compared to the acceptable macronutrient distribution ranges (AMDRs) for NZ adults (NHMRC, 2006).

Pacific women had a higher intake of total carbohydrates, sugar and starch; but lower intake of dietary fibre and alcohol in comparison to NZ European women. There are a number of well documented factors contributing to differences in dietary intake between Pacific and NZ European women. For example, some Pacific families have an increased number of social and special occasions where food can play an important role (Tupai-Firestone et al., 2016). Other factors which may influence dietary intake in Pacific

women are socioeconomic status and food availability. For example, it has been observed that takeaway food outlets are more common in socioeconomically deprived areas (Mackay, Vandevijvere, Xie, Lee, and Swinburn, 2017; Oaken, Vaughan, Fa'avale, Ware, and Schubert, 2017) and decreased intake of healthy food options may be due to cost (Pechey and Monsivais, 2016). However, some Pacific women residing in NZ may have a similar dietary intake to that of NZ European women through acculturation (Wall et al., 2016). Therefore, it was considered necessary to adjust for socioeconomic status in the current study to acknowledge how dietary quality can be impacted by socioeconomic factors.

Disinhibited eating behaviour was higher and predisposition to hunger was lower in NZ European compared with Pacific women. This implies NZ European women may be more prone to eating in times of stress or eating in response to heightened palatability, as reported in previous studies (Haynes, Lee, and Yeomans, 2003; Yeomans, 2010). Susceptibility to feelings of hunger may result in impulsive eating, or increased consumption of food outside of mealtimes (Booth, Spronk, Grol, and Fox, 2018). Feelings of increased hunger in Pacific women may be associated with dysregulated reward signalling pathways, which has been associated with the metabolic dysregulation of satiety signals (Barkeling, King, Näslund, and Blundell, 2007). In both Pacific and NZ European women, those with higher body fat had significantly higher disinhibition score than non-obese women. This finding is similar to other studies in New Zealand where disinhibition was positively associated with BMI in premenopausal women (Kruger et al., 2016). Another study showed that individuals who have difficulty maintaining their weight had a higher disinhibition score than those described as resistant to obesity (Brown et al., 2014). Similar associations were also observed in a French population (Lesdéma et al., 2012). NZ European women with high body fat % from the present study had lower restraint and higher hunger scores compared with women with low body fat %. Interestingly, these differences were not observed in Pacific women, and no differences in the other eating behaviour scores were observed in Pacific women when comparing body fat % groups. In summary, there were underlying differences in both dietary intake and eating behaviour observed when comparing Pacific and NZ European women.

5.5.2. Sweet taste liking and dietary intake

This study showed clear associations between sweet taste liking and increased carbohydrate, starch and sugar intakes in NZ European women. However, no significant associations were observed in Pacific women. The results from this study are similar to data published by Jayasinghe et al (2017), where dietary sugar and carbohydrate intake in NZ European women were positively correlated with hedonic liking and negatively correlated with intensity ratings of suprathreshold concentrations of glucose. In both studies, heterogeneity in sweet taste perception was observed between participants and sweet taste was measured using glucose solutions as tastants. In contrast, other studies have used sucrose and found no association between taste intensity perception and diet (Leong et al., 2018; Stevenson et al., 2016).

Sweet taste intensity ratings have been shown to be linked with specific dietary preferences and can change when dietary patterns change, as shown in diet intervention studies, being attributed to the alteration in the expression of associated taste receptors TAS1R2 and TAS1R3 (Wise et al., 2016). In the dietary intervention study by Wise et al, it was shown that during the third month of low-sugar dietary intervention, both low and high in sugar puddings were rated as 40% sweeter than the control group ratings, compared to no difference in the ratings at baseline (Wise et al., 2016). The present study clearly shows that a lower sweet taste perception combined with a higher sweet taste preference is linked with an increase in dietary intake of sweet-tasting food in NZ European women. Previous work has shown that the liking of sweet and fat together may be linked to the overconsumption of corresponding foods (Deglaire et al., 2015). In this study, total fat intake was high (~40% of total energy intake) and saturated fat intake (~15% of total energy intake) exceeded the AMDRs (NHMRC, 2006) together with an excess of total sugar intake. These data suggest a shift in dietary habits, where highly palatable and highly processed foods become a major part of the diet, as opposed to well balanced meals. Further supporting evidence for this concerning relationship comes from intervention studies which have provided evidence that high-fat diets are associated with desensitised fatty acid taste perception in humans (Stewart and Keast, 2012) and in animal models (Zhang et al., 2011).

In the present study, associations between sweet taste perception and dietary intake and eating behaviour that we observed in NZ European women were not observed in Pacific women. It can be speculated that differences in taste-diet associations may be linked to cultural differences in nutrition (Overberg et al., 2012). When comparing Pacific sweet likers and dislikers there was no difference observed in carbohydrate, sugar or starch intakes, but instead, there was a difference in protein intake. There may be a long-term biological adaptation to sugar in NZ European women, which may act to condition inherited taste preferences, influencing life-long dietary habits (Mennella, 2014). For example, traditional diets of the Pacific Islands are high in fresh fruits, complex carbohydrates and fibre (Shintani and Hughes, 1994). On the other hand, traditional European recipes and diets are higher in sugar (Baschetti, 1998). Another study has shown significant differences in maternal child-feeding style between ethnic groups, which may be further linked to long-term eating behaviour (Korani, Rea, King, and Brown, 2018). In addition, another study showed that Hispanics and African Americans rated taste sensations higher than non-Hispanic Whites (Williams et al., 2016).

In this study, however, there was no difference between the number of sweet likers versus dislikers when comparing Pacific and NZ European women, so it is difficult to speculate that differences in the perceived taste of food is associated with variation in food intake. The differences observed between Pacific and NZ European women in sweet taste perception and dietary intake may be indicative of differences in the cognitive response to taste perception instead, in association with appetite and long-term signalling pathways (Rolls, 2016; Veldhuizen et al., 2017). NZ European sweet likers had a higher cognitive disinhibition score than dislikers, which was not observed in Pacific women. This finding supports an association between reward signalling pathways, sweet taste perception and dietary intake. However, further research in this area is warranted, alongside a range of other potential influences on taste and dietary intakes, such as physical activity and sleep (Lenard and Berthoud, 2008; Smith, Ludy, and Tucker, 2016).

5.5.3. Bitter and milk fat perception and dietary intake

Firstly, we established that bitter intensity ratings were associated with hedonic liking, establishing a clear dose-dependent relationship. Milk fat liking and intensity ratings showed a less pronounced relationship, with the hedonic liking rating of bitter taste only corresponding to taste intensity rating at high concentrations (>20.3% fat). However, we found only minor associations between bitter taste and milk fat hedonic liking cluster groups and dietary intake. NZ European bitter likers had an increased intake of dietary fibre compared to bitter dislikers. For both Pacific and NZ European women (obese and non-obese groups, respectively), bitter likers had a decreased protein intake compared to dislikers. It is feasible that bitter liking may influence dietary fibre intake, as this may be indicative of increased cruciferous vegetable intake, due to a less adverse response to bitter-tasting compounds (Ortega et al., 2016), which has been observed in previous studies (Barajas-Ramírez, Quintana-Castro, Oliart-Ros, and Angulo-Guerrero, 2016; Duffy et al., 2010). The basis for an association between dietary protein and bitter taste perception is unclear.

Interestingly, being a milk fat liker was not associated with the increased intake of dietary fat when compared to dislikers. Similar results have been observed in other studies investigating fatty acid taste perception and dietary intake of fat (Costanzo, Orellana, Nowson, Duesing, and Keast, 2017; Stewart and Keast, 2012; Tucker, Nuessle, Garneau, Smutzer, and Mattes, 2015). The present study found that NZ European milk fat likers with higher body fat had decreased alcohol intake compared to milk fat dislikers. The basis for this association is also unclear. Further investigation into food choice and food group intake is warranted in order to substantiate these results.

5.5.4. Taste perception and eating behaviour

Disinhibited eating behaviour, measured by the TFEQ, was linked with sweet liking, however, this relationship was dependent on ethnicity. NZ European sweet likers had a significantly higher disinhibition score than sweet dislikers. Alongside increased disinhibition, NZ European sweet likers had higher situational disinhibition eating behaviour scores than sweet dislikers. Decreased intensity ratings of both sweet and

fatty solutions have been previously associated with a greater reinforcing value of food, or the motivation to get food, as a predictor of energy intake (Panek-Scarborough, Dewey, and Temple, 2012). Therefore, this study supports the hypothesis that people with lower sensitivity to sweet taste, could be vulnerable to overconsumption in order to compensate for a diminished reward that they may experience (Noel, Sugrue, and Dando, 2017). In Pacific women, there were no differences in eating behaviour when comparing sweet likers and dislikers. The reason for a lack of an association between taste perception and eating behaviour in Pacific women is currently unclear, however, it does mirror the lack of an association between taste perception and dietary intake. This indicates that there are important links between taste, diet and eating behaviour that warrants further investigation. A few studies have shown that increased disinhibition is associated with decreased fatty acid taste perception (Kindleysides et al., 2017; Tomassini Barbarossa et al., 2013). To date, few studies which have investigated associations between taste perception and eating behaviour.

5.5.5. Strengths and limitations

One of the strengths of this taste perception study was that the number of participants was relatively high compared to other sensory studies (Tan and Tucker, 2019). In addition, we were able to make comparisons between low and high body fat groups, thus filling a knowledge gap that has been identified in previous studies (Zhou et al., 2016). The sweet taste glucose solutions used in this study provided clear data on taste perception, similar to a recent study that drew comparisons between taste perception and the dietary intake of sweet foods (Jayasinghe et al., 2017). Quinine was chosen to measure bitter taste perception as it has been shown to be representative of broader taste function, as well as being strongly correlated with other bitter taste sensations, such as propylthiouracil (Rawal et al., 2015). Finally, milk and cream solutions were used to measure the perception of fat perception or creaminess, which has been suggested to play a role in influencing satiety (Proserpio et al., 2016).

The results presented in this study need to be considered alongside several limitations, which may have confounded our results. Firstly, all taster groups were described as

either ‘liker’ or ‘disliker’ phenotype groups due to the derivation of only two groups from the hierarchical cluster analysis. However, the group ‘bitter likers’ may be more accurately described as ‘bitter neutral’ and the group ‘milk fat dislikers’ may be more accurately described as ‘milk fat neutral’. In studies where three groups are derived from hierarchical cluster analysis, the naming of the middle group as a taster ‘neutral’ group is clearly appropriate. Response patterns for taste, such as sweet taste, would be best represented by three distinct phenotypes (likers, neutral and dislikers) (Iatridi et al., 2019). In this study it could be argued that taster ‘neutral’ individuals were therefore categorised into either the ‘liker’ or ‘disliker’ group for each taste type. It is acknowledged there is a lack of an intermediate phenotype for each taste type and this may have obfuscated associations and health outcomes of interest.

In the current study glucose was the sweet taste stimulus chosen because glucose is a simple sugar/monosaccharide which is sensed throughout the body, including the gastrointestinal tract (Jayasinghe et al., 2017). However, the majority of previous studies have used the disaccharide sucrose as a sweet taste stimulus instead (Cox et al, 2015, Tan and Tucker, 2019). The choice of the sweet taste stimulus and the concentrations tested (0 g/L – 240 g/L) may in part explain the lack of a neutral or middle phenotype derived from the hierarchical cluster analysis. The testing of higher concentrations of sweetness or the use of sucrose, which is perceived as sweeter than glucose (Peng, Hautus, Oey, and Silcock, 2016), may have also resulted in a decreased number of sweet taste dislikers, which would be more consistent with the proportion of sweet taste likers and dislikers found in recent studies from the US (Garneau et al., 2018), Korea (Kim et al., 2017) and the UK (Iatridi et al., 2019) where 20-25% of participants were sweet dislikers. Future studies will compare different types of sweeteners and sweet taste phenotype.

Secondly, the study was cross-sectional and therefore can only infer associations, but the results do not inform causality (Sedgwick, 2014). Thirdly, a range of dietary assessments can be used to evaluate both actual energy intake (food records, 24-hour recalls) and usual dietary intake (food frequency questionnaires, diet history) (Biro, Hulshof, Ovesen, and Amorim Cruz, 2002). The use of the 5-day non-consecutive food record is considered a limitation, as self-reported measurements of diet are less reliable

than validation methods using biomarkers (i.e. double labelled water). In addition, women with higher body fat are more likely to under report (Gemming, Jiang, Swinburn, Utter, and Mhurchu, 2014). As such, data was further explored using sensitivity analysis by the removal of under- and over reporters as determined by the Goldberg equation (Black, 2000; Goldberg et al., 1991), which verified no differences in key study outcomes. The epidemiological cut-offs used in this study are therefore considered valid and reliable and exclusion of participants by the Goldberg equation did not provide any added advantage, as reported previously (Rhee et al., 2015).

Measurements of milk fat perception (liking, intensity, and ranking task) were not a direct measurement of 'taste', primarily due to the presence of non-taste sensory inputs such as mouthfeel and aroma. In contrast, the measurements of sweet and bitter taste were liquid solutions that are suggested to have less relevance to 'real world' experiences (Cox et al., 2015; Rozin and Tuorila, 1993). This study lacked comprehensive descriptions of sensory attributes, which would be representative of actual foods consumed. Measurement of taste perception using food-based tastants would have provided additional information (Alexy et al., 2011; Lanfer et al., 2012). Therefore, it is recommended that future studies consider incorporating the sensory evaluation of whole foods, alongside the consideration of percepts such as olfaction, to determine the multi-modal sensory impact on dietary intake and food preferences (Cox et al., 2015). Future studies may further investigate differences between sub-populations within an ethnic group (i.e. Pacific peoples of Tongan, Samoan and Fijian descent).

5.5.6. Conclusion

This study has several important findings. The primary finding was that increased hedonic liking of sweet taste is associated with the increased intake of carbohydrates, sugars and starch in NZ European women. This aligned with decreased sweet taste intensity perception being associated with increased hedonic liking of sweet taste. In addition, the increased hedonic liking of sweet taste was associated with disinhibited eating behaviour in the same population group. However, these associations were not observed in Pacific women. Other minor associations were observed. Pacific and NZ

European bitter likers (high and low body fat %, respectively) had a decreased protein intake compared to bitter dislikers. NZ European bitter likers had a higher intake of dietary fibre (% energy) than bitter dislikers and Pacific milk fat likers with high body fat % had a higher intake of protein than milk fat dislikers.

This is the first study to report relationships between taste perception, eating behaviour and dietary intake, adjusting for socioeconomic status and age in non-obese and obese Pacific and NZ European women. These findings may inform future longitudinal and intervention studies. Future longitudinal studies may be able to determine if taste perception changes alongside variations in dietary intake. Future intervention studies may consider targeting the association between taste perception and post-ingestive effects, where taste receptor expression may be targeted to adjust long-term dietary intake. However, a better understanding of the relationship between taste perception and dietary intake is still required, particularly in obesity. Sweet taste perception may be influencing eating behaviour, and therefore, dietary intake; however, the relative degree of this influence is further affected by ethnicity, socio-economic cultural and environmental factors.

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Suppl 5.1. Association between daily macronutrient intake and bitter liking clusters in Pacific and NZ European women (N=304).

	Pacific (N= 142)			***New Zealand European (N= 162)***		
Macronutrient intake	Bitter liking (groups)§ Low body fat (<35%) (n= 68)	Bitter liking (groups)§ High body fat (>35%) (n= 74)	Bitter liking (groups)§ Pacific (all) (n= 142)	Bitter liking (groups)§ Low body fat (<35%) (n= 87)	Bitter liking (groups)§ High body fat (>35%) (n= 75)	Bitter liking (groups)§ NZ European (all) (n= 162)
	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^
Total energy (kJ)	-385.0 (-1262.0, 2032.0)	-1636.8 (-3646.6, 373.1)	-349.2 (-1594.2, 895.8)	-621.0 (-1511.2, 269.2)	-47.2 (-1218.5, 1124.0)	-413.2 (-1112.7, 286.3)
Protein (g)	0.26 (-16.0, 16.5)	**-23.0 (-40.8, -5.09)***	-8.42 (-20.2, 3.33)	**-11.6 (-21.2, -2.03)***	0.61 (-11.8, 13.0)	-7.44 (-14.9, 0.01)
Protein (%)	-0.14 (-2.00, 1.72)	-1.16 (-3.65, 1.32)	-0.62 (-2.09, 0.85)	-1.37 (-3.33, 0.60)	0.19 (-1.98, 2.37)	-0.86 (-2.29, 0.57)
Fat (g)	1.83 (-16.6, 20.3)	-16.2 (-40.3, 7.80)	-5.05 (-19.5, 9.37)	-8.29 (-22.3, 5.69)	2.76 (-18.1, 23.6)	-4.09 (-15.7, 7.55)
Fat (%)	-0.42 (-3.72, 2.88)	0.25 (-3.26, 3.77)	-0.33 (-2.63, 1.97)	-0.21 (-4.25, 3.83)	1.25 (-4.06, 6.57)	0.39 (-2.75, 3.53)
Saturated fat (g)	0.62 (-7.45, 8.69)	-7.00 (-17.4, 3.39)	-2.28 (-8.54, 3.98)	-5.64 (-11.8, 0.53)	1.04 (-8.86, 10.9)	-3.18 (-8.54, 2.18)
Saturated fat (%)	-0.33 (-1.94, 1.28)	-0.47 (-2.63, 1.69)	-0.44 (-1.71, 0.83)	-0.97 (-3.05, 1.11)	0.57 (-2.20, 3.33)	-0.38 (-2.01, 1.25)
Polyunsaturated fat (g)	-0.03 (-2.78, 2.73)	-2.27 (-5.73, 1.19)	-0.84 (-2.93, 1.26)	0.55 (-2.87, 3.97)	0.95 (-3.03, 4.93)	0.83 (-1.70, 3.35)
Polyunsaturated fat (%)	0.07 (-0.81, 0.95)	0.19 (-0.93, 1.30)	-0.10 (-0.57, 0.76)	0.56 (-0.51, 1.63)	0.47 (-0.87, 1.81)	0.58 (-0.24, 1.39)
Monounsaturated fat (g)	1.56 (-5.71, 8.83)	-5.29 (-15.2, 4.58)	-1.06 (-6.88, 4.76)	-2.45 (-7.89, 2.99)	0.52 (-7.35, 8.38)	-1.40 (-5.84, 3.03)
Monounsaturated fat (%)	0.02 (-1.52, 1.56)	0.48 (-1.46, 2.41)	0.13 (-1.05, 1.30)	0.18 (-1.69, 2.05)	0.15 (-2.22, 2.52)	0.14 (-1.29, 1.56)
Carbohydrate (g)	15.5 (-30.1, 61.2)	-42.9 (-99.1, 13.4)	-4.84 (-39.5, 29.9)	-15.5 (-44.2, 13.2)	-11.5 (-44.8, 21.7)	-13.8 (-35.1, 7.44)
Carbohydrate (%)	0.58 (-3.41, 4.58)	-0.33 (-4.28, 3.63)	0.49 (-2.21, 3.19)	-0.50 (-4.86, 3.87)	1.69 (-7.74, 4.36)	-0.83 (-4.32, 2.66)
Total sugar (g)	-0.70 (-22.6, 21.2)	-19.8 (-48.5, 8.82)	-7.26 (-24.5, 10.0)	-6.25 (-20.7, 8.16)	-8.93 (-27.7, 9.84)	-7.05 (-18.3, 4.19)
Total sugar (%)	-1.54 (-4.49, 1.42)	-0.03 (-3.66, 3.61)	-0.82 (-3.10, 1.45)	0.01 (-2.63, 2.65)	-1.97 (-6.08, 2.13)	-0.58 (-2.84, 1.68)
Starch (g)	16.5 (-11.8, 44.8)	-23.3 (-58.9, 12.3)	2.43 (-19.4, 24.2)	-9.41 (-30.3, 11.5)	-2.55 (-23.7, 18.6)	-6.78 (-21.4, 7.87)
Starch (%)	2.18 (-0.45, 4.81)	-0.38 (-3.52, 2.76)	1.31 (-0.63, 3.24)	-0.55 (-3.93, 2.84)	0.28 (-3.71, 4.27)	-0.26 (-2.76, 2.24)
Dietary fibre (g)	-0.23 (-4.32, 3.86)	-3.03 (-7.77, 1.71)	-1.29 (-4.27, 1.68)	1.74 (-3.18, 6.66)	4.25 (-0.17, 8.67)	2.70 (-0.56, 5.96)
Dietary fibre (%)	-0.02 (-0.27, 0.24)	0.07 (-0.31, 0.45)	0.01 (-0.21, 0.22)	0.34 (-0.06, 0.74)	**0.44 (-0.01, 0.86)***	**0.38 (0.10, 0.66)****
Alcohol (g)	1.99 (-6.50, 10.5)	3.71 (-0.04, 7.45)	2.68 (-1.99, 7.35)	4.40 (-2.62, 11.4)	-0.35 (-7.73, 7.04)	2.42 (-2.65, 7.49)
Alcohol (%)	0.01 (-1.96, 1.99)	**1.27 (0.05, 2.49)***	0.50 (-0.65, 1.66)	1.69 (-0.66, 4.03)	-0.35 (-2.96, 2.26)	0.84 (-0.90, 2.57)

General linear model coefficients and 95% CI adjusted for # age and NZDep2013 and for ^ age, NZDep2013 and BF% (groups). *P< 0.05, **P< 0.01, ***P< 0.001. B, unstandardized coefficient; C.I., confidence interval; BF%, body fat percentage. §Reference category: Bitter dislikers, Pacific bitter dislikers n= 117; NZ European bitter dislikers n= 134.

Suppl 5.2. Association between daily macronutrient intake and milk fat liking clusters in Pacific and NZ European women (N=304).

	Pacific (N= 142)			***New Zealand European (N= 162)***		
Macronutrient intake	Milk fat liking (groups)§ Low body fat (<35%) (n= 68)	Milk fat liking (groups)§ High body fat (>35%) (n= 74)	Milk fat liking (groups)§ Pacific (all) (n= 142)	Milk fat liking (groups)§ Low body fat (<35%) (n= 87)	Milk fat liking (groups)§ High body fat (>35%) (n= 75)	Milk fat liking (groups)§ NZ European (all) (n= 162)
	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^
Total energy (kJ)	-733.2 (-2346.8, 880.4)	515.6 (-853.3, 1884.5)	33.4 (-989.4, 1056.2)	-339.4 (-1133.3, 454.4)	-376.1 (-1306.7, 554.5)	-342.3 (-939.8, 255.1)
Protein (g)	-6.04 (-21.9, 9.85)	**12.7 (0.48, 24.8)***	5.58 (-4.09, 15.2)	5.10 (-3.59, 13.8)	-2.32 (-12.2, 7.56)	2.24 (-4.19, 8.68)
Protein (%)	0.36 (-1.47, 2.19)	1.56 (-0.08, 3.19)	1.09 (-0.11, 2.28)	1.67 (-0.06, 3.39)	0.26 (-1.48, 2.00)	1.12 (-0.10, 2.33)
Fat (g)	-9.68 (-27.7, 8.33)	7.65 (-8.60, 23.9)	1.03 (-10.8, 12.9)	-5.33 (-17.8, 7.11)	-0.03 (-16.7, 16.6)	-2.95 (-12.9, 7.00)
Fat (%)	0.11 (-3.14, 3.36)	-1.26 (-1.08, 3.60)	0.82 (-1.06, 2.70)	-1.20 (-4.77, 2.37)	0.86 (-3.38, 5.11)	-0.31 (-2.99, 2.38)
Saturated fat (g)	-4.18 (-12.1, 3.70)	2.41 (-4.64, 9.45)	-0.14 (-5.28, 5.01)	-2.64 (-8.18, 2.90)	2.17 (-5.73, 10.1)	-0.57 (-5.16, 4.03)
Saturated fat (%)	-0.25 (-1.83, 1.34)	0.41 (-1.04, 1.86)	0.15 (-0.90, 1.19)	-0.62 (-2.47, 1.23)	1.42 (-0.76, 3.61)	0.24 (-1.16, 1.63)
Polyunsaturated fat (g)	-1.44 (-4.13, 1.24)	1.74 (-0.57, 4.06)	0.45 (-1.27, 2.17)	-0.50 (-3.53, 2.52)	-0.25 (-3.43, 2.93)	-0.41 (-2.56, 1.75)
Polyunsaturated fat (%)	-0.04 (-0.90, 0.83)	0.40 (-0.34, 1.14)	0.20 (-0.34, 0.74)	-0.09 (-1.04, 0.86)	-0.12 (-1.19, 0.95)	-0.11 (-0.81, 0.59)
Monounsaturated fat (g)	-2.58 (-9.72, 4.56)	2.86 (-3.78, 9.51)	0.89 (-3.89, 5.67)	-2.32 (-7.13, 2.49)	-2.14 (-8.39, 4.12)	-2.12 (-5.90, 1.65)
Monounsaturated fat (%)	0.57 (-0.94, 2.08)	0.31 (-0.98, 1.61)	0.46 (-0.51, 1.41)	-0.71 (-2.36, 0.94)	-0.68 (-2.57, 1.21)	-0.65 (-1.86, 0.57)
Carbohydrate (g)	-23.4 (-68.2, 21.3)	2.63 (-35.8, 41.0)	-7.82 (-36.3, 20.6)	-13.9 (-39.3, 11.5)	-9.97 (-36.5, 16.5)	-12.6 (-30.7, 5.58)
Carbohydrate (%)	-1.56 (-5.48, 2.36)	-2.40 (-4.99, 0.20)	-2.07 (-4.26, 0.12)	-0.82 (-4.68, 3.04)	0.79 (-4.05, 5.63)	-0.31 (-3.30, 2.67)
Total sugar (g)	-3.12 (-24.7, 18.4)	4.68 (-14.8, 24.1)	0.24 (-14.0, 14.4)	-2.54 (-15.3, 10.3)	-2.34 (-17.4, 12.7)	-2.80 (-12.4, 6.84)
Total sugar (%)	-0.12 (-3.05, 2.81)	-0.55 (-2.99, 1.89)	-0.60 (-2.46, 1.27)	0.36 (-1.97, 2.70)	1.25 (-2.03, 4.54)	0.58 (-1.35, 2.51)
Starch (g)	-20.4 (-48.1, 7.27)	-2.09 (-26.3, 22.1)	-8.12 (-25.9, 9.72)	-11.5 (-30.0, 6.94)	-7.43 (-24.2, 9.37)	-9.84 (-22.3, 2.61)
Starch (%)	-1.47 (-4.09, 1.15)	-1.86 (-3.92, 0.20)	-1.49 (-3.07, 0.09)	-1.23 (-4.22, 1.75)	-0.42 (-3.61, 2.76)	-0.91 (-3.04, 1.21)
Dietary fibre (g)	-0.38 (-4.40, 3.65)	2.90 (-0.24, 6.05)	1.45 (-0.99, 3.88)	-3.80 (-8.09, 0.49)	1.28 (-2.32, 4.89)	-1.51 (-4.30, 1.29)
Dietary fibre (%)	0.09 (-0.16, 0.33)	0.14 (-0.11, 0.40)	0.10 (-0.07, 0.28)	-0.30 (-0.65, 0.05)	0.26 (-0.08, 0.60)	-0.05 (-0.30, 0.19)
Alcohol (g)	4.37 (-3.94, 12.7)	-1.87 (-4.41, 0.67)	0.63 (-3.22, 4.48)	0.95 (-5.32, 7.22)	**-6.18 (-11.9, -0.47)***	-1.83 (-6.16, 2.50)
Alcohol (%)	0.99 (-0.94, 2.91)	-0.61 (-1.44, 0.22)	0.02 (-0.93, 0.97)	0.56 (-1.54, 2.66)	**-2.18 (-4.20, -0.16)***	-0.51 (-1.99, 0.97)

General linear model coefficients and 95% CI adjusted for # age and NZDep2013 and for ^ age, NZDep2013 and BF% (groups). *P< 0.05, **P< 0.01, ***P< 0.001. B, unstandardized coefficient; C.I., confidence interval; BF%, body fat percentage. §Reference category: Milk fat dislikers, Pacific milk fat dislikers n= 44; NZ European milk fat dislikers n= 45.

Suppl 5.3. Comparisons of dietary intake by sweet, bitter and milk fat ranking task outcome in (A) Pacific (N=142) and (B) NZ European women (N=162).

Macronutrient intake	Sweet ranking (groups)† Pacific B (95% C.I.)	Sweet ranking (groups)† NZ European B (95% C.I.)	Bitter ranking (groups)‡ Pacific B (95% C.I.)	Bitter ranking (groups)‡ NZ European B (95% C.I.)	Milk fat ranking (groups)# Pacific B (95% C.I.)	Milk fat ranking (groups)# NZ European B (95% C.I.)
Total energy (kJ)	455.0 (-767.3, 1677.4)	150.4 (-559.6, 860.5)	-1.81 (-995.8, 992.1)	49.3 (-474.8, 573.3)	-344.8 (-1395.1, 705.5)	180.7 (-344.2, 705.7)
Protein (g)	2.44 (-9.18, 14.1)	1.49 (-6.15, 9.13)	-0.63 (-10.1, 8.81)	-2.27 (-7.88, 3.35)	-3.90 (-13.9, 6.06)	2.33 (-3.31, 7.96)
Protein (%)	-0.47 (-1.91, 0.98)	0.19 (-1.26, 1.65)	0.13 (-1.05, 1.30)	-0.62 (-1.69, 0.45)	-0.05 (-1.29, 1.19)	0.09 (-0.99, 1.16)
Fat (g)	7.05 (-7.09, 21.2)	2.14 (-9.66, 13.9)	-1.75 (-13.3, 9.76)	0.27 (-8.43, 8.97)	-4.65 (-16.8, 7.51)	3.10 (-5.61, 11.8)
Fat (%)	1.10 (-1.16, 3.35)	-0.15 (-3.33, 3.04)	-0.64 (-2.48, 1.19)	-0.18 (-2.52, 2.16)	-0.70 (-2.64, 1.24)	0.55 (-1.79, 2.90)
Saturated fat (g)	3.00 (-3.14, 9.14)	0.99 (-4.46, 6.43)	-1.17 (-6.16, 3.83)	-1.04 (-5.06, 2.97)	-3.17 (-8.43, 2.09)	0.61 (-3.42, 4.64)
Saturated fat (%)	0.27 (-0.98, 1.52)	-0.08 (-1.73, 1.58)	-0.54 (-1.55, 0.47)	-0.60 (-1.81, 0.61)	-1.01 (-2.07, 0.05)	-0.01 (-1.23, 1.21)
Polyunsaturated fat (g)	0.84 (-1.21, 2.90)	0.10 (-2.46, 2.66)	0.49 (-1.18, 2.16)	0.43 (-1.45, 2.31)	-0.01 (-1.78, 1.76)	1.02 (-0.86, 2.91)
Polyunsaturated fat (%)	0.23 (-0.41, 0.88)	-0.14 (-0.96, 0.69)	0.26 (-0.27, 0.79)	0.19 (-0.42, 0.80)	0.19 (-0.37, 0.74)	0.37 (-0.24, 0.98)
Monounsaturated fat (g)	2.70 (-3.00, 8.40)	0.53 (-3.97, 5.02)	-1.16 (-5.80, 3.48)	1.09 (-2.22, 4.40)	-0.81 (-5.72, 4.10)	1.07 (-2.25, 4.39)
Monounsaturated fat (%)	0.57 (-0.57, 1.72)	-0.09 (-1.53, 1.36)	-0.40 (-1.33, 0.53)	0.36 (-0.70, 1.42)	0.28 (-0.71, 1.27)	0.10 (-0.96, 1.17)
Carbohydrate (g)	10.3 (-23.7, 44.4)	-0.83 (-22.5, 20.8)	6.30 (-21.4, 34.0)	1.80 (-14.1, 17.7)	-7.79 (-37.1, 21.5)	2.78 (-13.2, 18.8)
Carbohydrate (%)	-0.24 (-2.90, 2.42)	-0.68 (-4.22, 2.86)	0.83 (-1.32, 2.98)	0.26 (-2.35, 2.86)	0.32 (-1.97, 2.60)	-0.44 (-3.06, 2.17)
Total sugar (g)	7.09 (-9.87, 24.0)	-1.90 (-13.3, 9.53)	3.18 (-10.6, 17.0)	-0.96 (-9.39, 7.46)	-5.34 (-19.9, 9.23)	-0.83 (-9.28, 7.62)
Total sugar (%)	0.26 (-1.98, 2.49)	-0.37 (-2.66, 1.93)	0.74 (-1.07, 2.55)	-0.21 (-1.90, 1.48)	-0.24 (-2.16, 1.69)	-0.84 (-2.52, 0.85)
Starch (g)	3.29 (-18.1, 24.7)	1.26 (-13.6, 16.2)	3.12 (-14.3, 20.5)	2.93 (-8.03, 13.9)	-2.58 (-21.0, 15.8)	3.64 (-7.34, 14.6)
Starch (%)	-0.49 (-2.40, 1.43)	-0.27 (-2.80, 2.27)	0.09 (-1.47, 1.64)	0.49 (-1.37, 2.36)	0.52 (-1.12, 2.16)	0.40 (-1.47, 2.27)
Dietary fibre (g)	0.57 (-2.36, 3.50)	1.27 (-2.06, 4.60)	-0.15 (-2.52, 2.23)	-0.58 (-3.04, 1.87)	-1.45 (-3.96, 1.05)	0.88 (-1.58, 3.33)
Dietary fibre (%)	-0.03 (-0.24, 0.17)	0.09 (-0.20, 0.38)	-0.00 (-0.17, 0.16)	-0.07 (-0.28, 0.14)	-0.06 (-0.23, 0.12)	0.03 (-0.19, 0.24)
Alcohol (g)	-0.95 (-5.56, 3.66)	1.60 (-3.54, 6.74)	-1.11 (-4.85, 2.63)	2.05 (-1.73, 5.82)	1.24 (-2.72, 5.19)	-1.10 (-4.90, 2.70)
Alcohol (%)	-0.38 (-1.52, 0.75)	0.51 (-1.25, 2.26)	-0.30 (-1.22, 0.63)	0.70 (-0.59, 1.99)	0.50 (-0.48, 1.47)	-0.29 (-1.58, 1.02)

General linear model coefficients and 95% CI adjusted for age, NZDep2013 and BF% (groups). *P< 0.05, **P< 0.01, ***P< 0.001. B, unstandardized coefficient; C.I., confidence interval; BF%, body fat percentage. †Reference category: Sweet ranking incorrect, Pacific n= 27; NZ European n= 27. ‡Reference category: Bitter ranking incorrect, Pacific n= 96; NZ European n= 82. Reference category: #Milk fat ranking incorrect, Pacific n= 95; NZ European n= 85.

Suppl 5.4. Eating behaviour outcome by bitter taste likers (compared to dislikers group) (N= 304).

	Unadjusted			***Adjusted***		
	Bitter liking (grps) Low BF (<35%) (n= 68)	Bitter liking (grps) High BF (>35%) (n= 74)	Bitter liking (grps) TOTAL ‡ (n= 142)	Bitter liking (grps) Low BF (<35%) # (n= 68)	Bitter liking (grps) High BF (>35%) # (n= 74)	Bitter liking (grps) TOTAL ^ (n= 142)
Pacific (N = 142)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)
Cognitive dietary restraint[1]	0.87 (0.61, 1.26)	1.34 (0.93, 1.94)	1.04 (0.80, 1.34)	0.80 (0.55, 1.15)	1.31 (0.90, 1.92)	0.97 (0.74, 1.25)
Flexible control restraint	1.10 (0.79, 1.54)	1.19 (0.80, 1.76)	1.13 (0.88, 1.46)	1.03 (0.73, 1.46)	1.18 (0.78, 1.78)	1.08 (0.83, 1.40)
Rigid control restraint	0.93 (0.64, 1.37)	1.42 (0.93, 2.17)	1.10 (0.83, 1.46)	0.85 (0.58, 1.25)	1.28 (0.83, 1.97)	1.00 (0.75, 1.32)
Disinhibition[2]	1.12 (0.84, 1.50)	0.95 (0.72, 1.26)	1.05 (0.86, 1.28)	1.10 (0.81, 1.50)	0.95 (0.71, 1.26)	1.04 (0.85, 1.28)
Habitual disinhibition	1.26 (0.92, 1.72)	1.33 (0.89, 2.01)	**1.29 (1.01, 1.65)***	1.31 (0.95, 1.82)	1.37 (0.90, 2.10)	**1.34 (1.04, 1.72)***
Emotional disinhibition	1.34 (1.00, 1.81)	0.99 (0.67, 1.45)	1.19 (0.94, 1.51)	1.31 (0.96, 1.79)	0.96 (0.64, 1.44)	1.16 (0.91, 1.48)
Situational disinhibition	0.89 (0.65, 1.21)	0.76 (0.58, 1.01)	0.84 (0.68, 1.03)	0.85 (0.62, 1.16)	0.75 (0.56, 1.01)	0.81 (0.66, 1.00)
Hunger[3]	0.95 (0.67, 1.35)	1.02 (0.75, 1.40)	0.98 (0.77, 1.23)	1.00 (0.69, 1.45)	1.06 (0.77, 1.47)	1.02 (0.80, 1.30)
Internal locus hunger	1.09 (0.76, 1.58)	1.23 (0.79, 1.61)	1.11 (0.86, 1.42)	1.13 (0.77, 1.66)	1.13 (0.77, 1.64)	1.13 (0.87, 1.47)
External locus hunger	0.91 (0.67, 1.24)	0.95 (0.67, 1.35)	0.93 (0.74, 1.16)	0.96 (0.70, 1.32)	1.01 (0.70, 1.45)	0.98 (0.77, 1.23)
NZ European (N= 162)	(n= 87)	(n= 75)	(n= 162)	(n= 87)	(n= 75)	(n= 162)
Cognitive dietary restraint[1]	1.12 (0.83, 1.52)	0.99 (0.68, 1.43)	1.07 (0.85, 1.34)	1.21 (0.89, 1.63)	1.04 (0.71, 0.51)	1.13 (0.90, 1.42)
Flexible control restraint	1.14 (0.90, 1.45)	1.02 (0.74, 1.40)	1.09 (0.90, 1.32)	1.17 (0.92, 1.49)	1.08 (0.78, 1.49)	1.13 (0.93, 1.36)
Rigid control restraint	0.98 (0.75, 1.28)	1.01 (0.71, 1.43)	0.99 (0.80, 1.23)	1.06 (0.81, 1.39)	1.07 (0.74, 1.52)	1.04 (0.85, 1.29)
Disinhibition[2]	1.03 (0.80, 1.33)	1.16 (0.90, 1.48)	1.07 (0.90, 1.28)	1.08 (0.84, 1.39)	1.17 (0.91, 1.51)	1.10 (0.92, 1.32)
Habitual disinhibition	1.11 (0.81, 1.53)	1.10 (0.73, 1.65)	1.11 (0.86, 1.42)	1.23 (0.89, 1.70)	1.08 (0.72, 1.63)	1.12 (0.87, 1.44)
Emotional disinhibition	1.02 (0.75, 1.40)	1.09 (0.80, 1.49)	1.05 (0.84, 1.31)	1.06 (0.77, 1.47)	1.08 (0.78, 1.49)	1.06 (0.85, 1.33)
Situational disinhibition	0.98 (0.78, 1.23)	1.21 (0.95, 1.54)	1.06 (0.90, 1.25)	0.99 (0.79, 1.25)	1.27 (0.99, 1.61)	1.08 (0.92, 1.27)
Hunger[3]	0.89 (0.68, 1.18)	0.81 (0.54, 1.21)	0.86 (0.68, 1.08)	0.95 (0.72, 1.26)	0.82 (0.54, 1.22)	0.88 (0.70, 1.11)
Internal locus hunger	0.81 (0.59, 1.12)	0.81 (0.53, 1.26)	0.81 (0.63, 1.05)	0.83 (0.60, 1.15)	0.82 (0.52, 1.29)	0.82 (0.63, 1.07)
External locus hunger	0.98 (0.73, 1.30)	0.89 (0.60, 1.32)	0.94 (0.75, 1.19)	1.05 (0.78, 1.40)	0.93 (0.63, 1.37)	0.97 (0.77, 1.22)

All dependent values log transformed. General linear model exponentiated coefficients and 95% CI adjusted for ‡ BF% group; # age, NZDep2013; for ^ age, NZDep2013 and BF% group. *P< 0.05, **P< 0.01, ***P< 0.001. C.I., confidence interval. BF%, body fat percentage. Reference category: bitter dislikers, Pacific bitter dislikers n= 117; NZ European bitter dislikers n= 134. [1]Restraint reference values = Low, 4-14; Medium, 15-17; High, 18-21. [2]Disinhibition reference values =

Suppl 5.5. Eating behaviour outcome by milk fat perception likers (compared to dislikers group) (N= 304).

	Unadjusted			***Adjusted***		
Pacific (N = 142)	Milk fat liking (grps) Low BF (<35%) (n= 68)	Milk fat liking (grps) High BF (>35%) (n= 74)	Milk fat liking (grps) TOTAL ‡ (n= 142)	Milk fat liking (grps) Low BF (<35%) # (n= 68)	Milk fat liking (grps) High BF (>35%) # (n= 74)	Milk fat liking (grps) TOTAL ^ (n= 142)
	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)	Ratio (95% C.I.)
Cognitive dietary restraint[1]	0.92 (0.64, 1.31)	1.01 (0.78, 1.31)	0.97 (0.78, 1.19)	0.99 (0.68, 1.44)	1.02 (0.78, 1.32)	0.99 (0.80, 1.23)
Flexible control restraint	0.89 (0.64, 1.24)	0.96 (0.73, 1.26)	0.93 (0.76, 1.15)	0.93 (0.65, 1.32)	0.98 (0.74, 1.29)	0.95 (0.76, 1.18)
Rigid control restraint	1.02 (0.70, 1.49)	1.12 (0.83, 1.50)	1.08 (0.85, 1.36)	1.07 (0.73, 1.59)	1.11 (0.83, 1.49)	1.10 (0.87, 1.39)
Disinhibition[2]	1.05 (0.79, 1.40)	1.15 (0.95, 1.40)	1.10 (0.94, 1.31)	1.08 (0.79, 1.47)	1.13 (0.93, 1.38)	1.10 (0.93, 1.30)
Habitual disinhibition	1.09 (0.80, 1.48)	0.97 (0.73, 1.29)	1.02 (0.83, 1.25)	1.07 (0.76, 1.49)	0.97 (0.72, 1.30)	1.01 (0.81, 1.25)
Emotional disinhibition	0.99 (0.73, 1.34)	1.14 (0.87, 1.49)	1.07 (0.88, 1.30)	1.04 (0.75, 1.44)	1.13 (0.86, 1.49)	1.09 (0.89, 1.34)
Situational disinhibition	1.05 (0.77, 1.42)	1.20 (0.99, 1.47)	1.13 (0.95, 1.35)	1.07 (0.78, 1.47)	1.18 (0.97, 1.44)	1.11 (0.93, 1.33)
Hunger[3]	1.10 (0.78, 1.55)	1.17 (0.95, 1.45)	1.14 (0.94, 1.38)	1.02 (0.70, 1.48)	1.18 (0.95, 1.47)	1.11 (0.91, 1.36)
Internal locus hunger	0.90 (0.63, 1.29)	**1.28 (1.01, 1.63)***	1.10 (0.89, 1.36)	0.88 (0.60, 1.29)	1.28 (1.00, 1.64)	1.10 (0.88, 1.36)
External locus hunger	**1.38 (1.03, 1.86)***	1.15 (0.91, 1.47)	**1.25 (1.04, 1.50)***	1.29 (0.94, 1.78)	1.16 (0.91, 1.48)	1.21 (1.00, 1.46)
NZ European (N= 162)	(n= 87)	(n= 75)	(n= 162)	(n= 87)	(n= 75)	(n= 162)
Cognitive dietary restraint[1]	1.30 (1.00, 1.69)	0.95 (0.72, 1.25)	1.12 (0.93, 1.36)	**1.32 (1.02, 1.71)***	0.96 (0.71, 1.30)	1.15 (0.95, 1.40)
Flexible control restraint	1.08 (0.87, 1.34)	0.92 (0.72, 1.17)	1.00 (0.86, 1.18)	1.08 (0.87, 1.34)	0.91 (0.70, 1.18)	1.00 (0.85, 1.18)
Rigid control restraint	1.20 (0.94, 1.52)	1.04 (0.80, 1.37)	1.13 (0.94, 1.34)	1.23 (0.98, 1.56)	1.06 (0.80, 1.42)	1.17 (0.98, 1.40)
Disinhibition[2]	0.99 (0.78, 1.24)	1.00 (0.83, 1.21)	0.99 (0.85, 1.15)	0.99 (0.79, 1.24)	0.95 (0.77, 1.16)	0.98 (0.84, 1.14)
Habitual disinhibition	1.03 (0.77, 1.37)	1.09 (0.80, 1.48)	1.05 (0.85, 1.30)	1.06 (0.79, 1.41)	0.98 (0.71, 1.36)	1.05 (0.85, 1.31)
Emotional disinhibition	1.01 (0.76, 1.34)	0.89 (0.71, 1.13)	0.96 (0.79, 1.15)	1.02 (0.77, 1.36)	0.89 (0.69, 1.14)	0.96 (0.79, 1.16)
Situational disinhibition	0.96 (0.78, 1.17)	1.04 (0.86, 1.26)	1.00 (0.87, 1.14)	0.96 (0.78, 1.17)	0.98 (0.80, 1.20)	0.97 (0.84, 1.12)
Hunger[3]	0.93 (0.72, 1.19)	1.02 (0.75, 1.39)	0.97 (0.80, 1.18)	0.94 (0.74, 1.21)	0.93 (0.67, 1.29)	0.95 (0.78, 1.16)
Internal locus hunger	0.98 (0.74, 1.31)	0.97 (0.69, 1.35)	0.98 (0.79, 1.21)	0.99 (0.74, 1.33)	0.95 (0.66, 1.36)	0.97 (0.78, 1.22)
External locus hunger	0.93 (0.72, 1.21)	1.04 (0.77, 1.41)	0.98 (0.81, 1.19)	0.95 (0.73, 1.23)	0.92 (0.67, 1.25)	0.95 (0.78, 1.16)

All dependent values log transformed. General linear model exponentiated coefficients and 95% CI adjusted for ‡ BF% group, # age, NZDep2013; ^ age, NZDep2013 and BF% group. *P< 0.05, **P< 0.01, ***P< 0.001. C.I., confidence interval. BF%, body fat percentage. Reference category: milk fat dislikers, Pacific milk fat dislikers n= 44; NZ European milk fat dislikers n= 45. [1]Restraint reference values = Low, 4-14; Medium, 15-17; High, 18-21. [2]Disinhibition reference values = Low, 1-8; Medium, 9-12; High, 13-16. [3]Hunger reference values = Low, 0-3; Medium, 4-6; High, 7-14 (Stunkard and Messick, 1985).

Suppl 5.6. Associations between macronutrient intake and sweet liking in Pacific and NZ European: under- and over-reporters removed (N=215).

	Pacific (N= 89)			***New Zealand European (N= 126)***		
Macronutrient intake	Sweet liking (groups)§ Low body fat (<35%) (n= 46)	Sweet liking (groups)§ High body fat (>35%) (n= 43)	Sweet liking (groups)§ Pacific (all) (n= 89)	Sweet liking (groups)§ Low body fat (<35%) (n= 72)	Sweet liking (groups)§ High body fat (>35%) (n= 54)	Sweet liking (groups)§ NZ European (all) (n= 126)
	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^	B (95% C.I.)#	B (95% C.I.)#	B (95% C.I.)^
Total energy (kJ)	-309.7 (-1325.1, 705.7)	-573.9 (-1794.0, 646.2)	-241.2 (-1003.1, 520.8)	-93.3 (-747.2, 560.5)	-168.0 (-937.3, 601.2)	-64.8 (-542.4, 412.8)
Protein (g)	5.12 (-6.72, 17.0)	4.13 (-7.79, 16.1)	6.91 (-1.12, 14.9)	-3.73 (-12.3, 4.83)	-0.32 (-9.17, 8.53)	-2.00 (-8.01, 4.01)
Protein (%)	**1.76 (0.02, 3.50)***	1.41 (-0.10, 2.91)	**1.65 (0.54, 2.77)****	-0.43 (-2.23, 1.38)	-0.13 (-1.66, 1.92)	-0.26 (-1.49, 0.96)
Fat (g)	-5.48 (-17.7, 6.71)	-5.98 (-21.7, 9.74)	-3.19 (-13.1, 6.73)	-9.65 (-21.3, 2.02)	-13.5 (-27.6, 0.74)	-9.77 (-18.5, 1.03)
Fat (%)	-0.78 (-4.03, 2.47)	-0.45 (-3.20, 2.30)	-0.50 (-2.57, 1.58)	**-4.00 (-7.36, -0.63)***	**-5.06 (-8.63, -1.50)****	**-4.03 (-6.42, -1.64)****
Saturated fat (g)	-1.79 (-7.65, 4.07)	-1.17 (-8.09, 5.74)	-0.34 (-4.75, 4.07)	-3.78 (-8.99, 1.44)	-5.58 (-13.0, 1.89)	-4.06 (-8.26, 0.14)
Saturated fat (%)	-0.31 (-1.81, 1.19)	0.43 (-1.33, 2.19)	0.17 (-0.95, 1.30)	-1.56 (-3.32, 0.20)	-2.03 (-4.07, 0.02)	**-1.62 (-2.90, -0.35)***
Polyunsaturated fat (g)	-1.17 (-3.70, 1.37)	-2.46 (-5.24, 0.33)	-1.72 (-3.55, 0.12)	-0.83 (-3.21, 1.55)	-1.00 (-4.43, 2.43)	-0.61 (-2.51, 1.30)
Polyunsaturated fat (%)	-0.15 (-1.18, 0.89)	-0.72 (-1.48, 0.03)	-0.51 (-1.11, 0.10)	-0.33 (-1.19, 0.52)	-0.33 (-1.55, 0.89)	-0.25 (-0.93, 0.43)
Monounsaturated fat (g)	-2.34 (-7.15, 2.46)	-2.64 (-9.53, 4.26)	-1.43 (-5.70, 2.84)	-4.11 (-9.02, 0.80)	**-5.71 (-11.1, -0.31)***	**-4.23 (-7.76, -0.71)***
Monounsaturated fat (%)	-0.38 (-2.02, 1.27)	-0.40 (-1.76, 0.96)	-0.32 (-1.39, 0.75)	**-1.69 (-3.31, -0.06)***	**-2.29 (-4.00, -0.59)****	**-1.79 (-2.93, -0.65)****
Carbohydrate (g)	-15.0 (-48.1, 18.1)	-25.7 (-64.1, 12.8)	-16.3 (-40.3, 7.65)	16.2 (-3.42, 35.8)	21.0 (-6.10, 48.1)	**19.9 (4.42, 35.4)****
Carbohydrate (%)	-1.73 (-5.69, 2.23)	-1.13 (-4.47, 2.21)	-1.56 (-4.10, 0.98)	**4.03 (0.54, 7.52)***	**5.05 (0.66, 9.44)***	**4.46 (1.83, 7.09)****
Total sugar (g)	-10.5 (-29.3, 8.42)	-12.2 (-37.5, 13.1)	-11.1 (-26.5, 4.41)	9.37 (-2.34, 21.1)	6.56 (-8.33, 21.4)	**9.52 (0.58, 18.5)***
Total sugar (%)	-1.58 (-4.52, 1.36)	-0.55 (-3.87, 2.77)	-1.35 (-3.57, 0.88)	2.31 (-0.03, 4.65)	1.68 (-0.89, 4.25)	**2.17 (0.51, 3.83)****
Starch (g)	-3.82 (-25.3, 17.7)	-13.5 (-38.1, 11.1)	-4.82 (-20.2, 10.6)	6.65 (-7.00, 20.3)	14.2 (-3.40, 31.8)	**10.3 (0.00, 20.5)***
Starch (%)	-0.01 (-3.06, 3.04)	-0.57 (-3.33, 2.19)	-0.13 (-2.10, 1.83)	1.69 (-0.80, 4.18)	**3.32 (0.30, 6.34)***	**2.27 (0.44, 4.10)****
Dietary fibre (g)	-3.05 (-6.35, 0.25)	-1.68 (-5.47, 2.12)	-2.18 (-4.62, 0.27)	0.81 (-4.72, 3.10)	1.74 (-2.15, 5.64)	0.61 (-2.03, 3.24)
Dietary fibre (%)	-0.20 (-0.45, 0.06)	-0.06 (-0.29, 0.18)	-0.15 (-0.31, 0.02)	-0.06 (-0.44, 0.31)	0.23 (-0.10, 0.56)	0.08 (-0.16, 0.32)
Alcohol (g)	-2.94 (-4.11, 9.99)	0.75 (-2.87, 4.37)	1.78 (-2.09, 5.65)	2.09 (-4.80, 8.99)	-1.32 (-6.97, 4.34)	-0.40 (-4.87, 4.08)
Alcohol (%)	0.94 (-1.12, 3.01)	0.22 (-0.72, 1.15)	0.54 (-0.56, 1.65)	0.50 (-1.85, 2.84)	-0.35 (-2.07, 1.37)	-0.22 (-1.70, 1.26)

General linear model coefficients and 95% CI adjusted for # age and NZDep2013 and for ^ age, NZDep2013 and BF% (groups). *P< 0.05, **P< 0.01, ***P< 0.001. BF%, body fat percentage. §Reference category: sweet dislikers, Pacific sweet dislikers n= 55; NZ European sweet dislikers n= 66. Statistically significant observations are highlighted in bold. Removal of n=72 under-reporters and n=17 over-reporters.

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Chapter VI.

Overall discussion and conclusions

6.1. Background and rationale

New Zealand ranks as the third most obese country in the OECD (OECD, 2017). Approximately 32% of the NZ adult population are classified as obese, and, approximately 34% are within the normal weight range (Ministry of Health., 2017). There is an urgent need to build on our current knowledge of the environmental and physiological factors that drive weight gain and its associated metabolic health consequences. The increasing prevalence of obesity appears to be exacerbated by an increase in the availability of energy-dense, highly processed, inexpensive and highly palatable, food and beverage options (Swinburn et al., 2011). Therefore, the physiological factors (e.g. taste perception) which further contribute to the onset of obesity in this setting, are potentially critical targets for future health interventions (Schwartz et al., 2017; Story, Kaphingst, Robinson-O'Brien, & Glanz, 2008).

Taste perception is an important physiological factor, integral to foods that are selected and consumed, thereby mediating energy intake (Breslin, 2013). For example, highly-energy dense and processed food options have an appealing taste, therefore leading to intake in excess of requirements and subsequent weight gain (Drewnowski, 1997). Taste perception has been shown to be dysregulated in an obese state and this dysregulation may reverse with weight loss (Berthoud & Zheng, 2012; Kaufman, Choo, Koh, & Dando, 2018). Therefore, taste perception could be a target for future weight control interventions, with an aim to reduce the intake of energy dense food options. However, determining the clear associations between taste perception and obesity needs to be established first (Kaufman et al., 2018; Newman, Bolhuis, Torres, & Keast, 2016).

This thesis endeavoured to measure multiple associations between taste perception, dietary intake, eating behaviour and measurements of metabolic health. Firstly, the Dessert Taste study investigated the test-retest repeatability and reliability of fatty acid taste perception and compared multimodal fatty acid chemosensory perception (i.e. taste, aroma and mouthfeel) with adiposity, eating behaviour and dietary intake. Secondly, in the PROMISE study, sweet taste, bitter taste and milk fat perception were measured with LAM and gLMS ratings of hedonic liking and intensity, as well as discrimination of taste by ranking task to determine associations between taste perception, body composition, hormonal adiposity signals, dietary intake and eating behaviour. Taste perception was compared with detailed parameters of adiposity which were measured by whole-body DXA scan (body fat %). Measurements of endocrine regulators of energy metabolism, including fasting plasma insulin, leptin, glucose, glycated haemoglobin and blood cholesterol were further investigated. Detailed measurements of dietary intake were conducted to provide robust data on actual dietary intake (5-day non-consecutive food record), alongside eating behaviour characteristics (TFEQ) to measure its associations with taste perception. This research on taste perception contributes to the growing body of knowledge on influences on dietary intake. These advances will support research efforts to improve health outcomes related to poor dietary choices.

6.2. Summary of results

An overview of key associations between and within chapters of this PhD thesis are presented in Figure 6.1. New knowledge was built, and methodology developed across this PhD thesis, with a central focus on taste perception and adiposity (Figure 6.1). In chapter three, detection thresholds of oleic acid taste were shown to be heterogeneous between individuals. This research added to the recently growing body of evidence that there is a perceivable fatty acid taste. Additionally, the taste perception of oleic acid was significantly correlated with olfactory detection of oleic acid (r=0.325; P< 0.02). This was a novel finding that has important implications for future studies that endeavour to establish complex associations between multiple sensory modalities (i.e. taste, aroma, and mouthfeel). Establishing such interactions between each sensory modality may reveal distinct associations between sensory perception, cognitive processing and reward signalling pathways. This study also found that fatty acid taste hyposensitivity was associated with higher BMI values and a higher disinhibited eating behaviour score in premenopausal NZ European women (P< 0.05). In addition, the dietary intake of nuts, nut spreads and seeds was positively correlated with olfactory sensitivity to oleic acid (r_s= -0.410, P< 0.01).

In chapter four, hedonic liking and intensity rating were measured using LAM and gLMS at a range of concentrations for glucose (sweet taste), quinine (bitter taste) and milk fat perception (milk and cream). Discrimination of taste by ranking task was used as a comparative tool for the measurement of taste perception. In Pacific and NZ European women, taster status was derived using cluster analysis from the LAM scale ratings of hedonic liking, which classified the study population into ‘likers’ and ‘dislikers’ for each taste. Important patterns of taster status were found, whereby sweet and bitter likers rated taste as less intense at all concentration levels in comparison to sweet and bitter dislikers, respectively. In contrast, milk fat likers rated milk fat intensity by gLMS as more intense than milk fat dislikers, but at higher concentrations only. Therefore, taste liking status was intimately linked with intensity ratings of taste, so this was used as distinct groups to determine further associations.

NZ European sweet likers were twice as likely to have higher body fat (>35%) compared to NZ European sweet dislikers (adjusted, OR 2.1, $P< 0.01$). In addition, women who incorrectly discriminated sweet taste by ranking task had higher body fat levels (>35%) (adjusted, OR 2.9, $P< 0.01$). Furthermore, increased fasting plasma leptin and insulin concentrations were associated with being a sweet liker (adjusted; OR 1.7, $P< 0.05$). In contrast, bitter taste liking was significantly associated with the decreased likelihood of having body fat >35% (adjusted, OR 0.4, $P< 0.01$). These results have shown differences in sweet taste and bitter taste perception which relate to adiposity.

In chapter five, dietary intake of carbohydrates, starch and sugars were significantly higher in NZ European sweet likers (199.4 ± 51.1, 87.9 ± 27.4 and 111.1 ± 34.6 g/day) when compared to sweet dislikers (165.9 ± 48.7, 71.4 ± 25.2 and 94.1 ± 34.7 g/day, $P< 0.001$, $P< 0.01$, and $P< 0.01$, respectively) and sweet taste hedonic liking was associated with higher disinhibited eating behaviour ($P< 0.01$). However, there were no clear taste-diet or taste-eating behaviour relationships between Pacific sweet likers and dislikers. This study suggests that sweet taste perception in NZ European women may influence dietary intake and eating behaviour. Additional taste-diet associations were minor and will be discussed in more detail below.

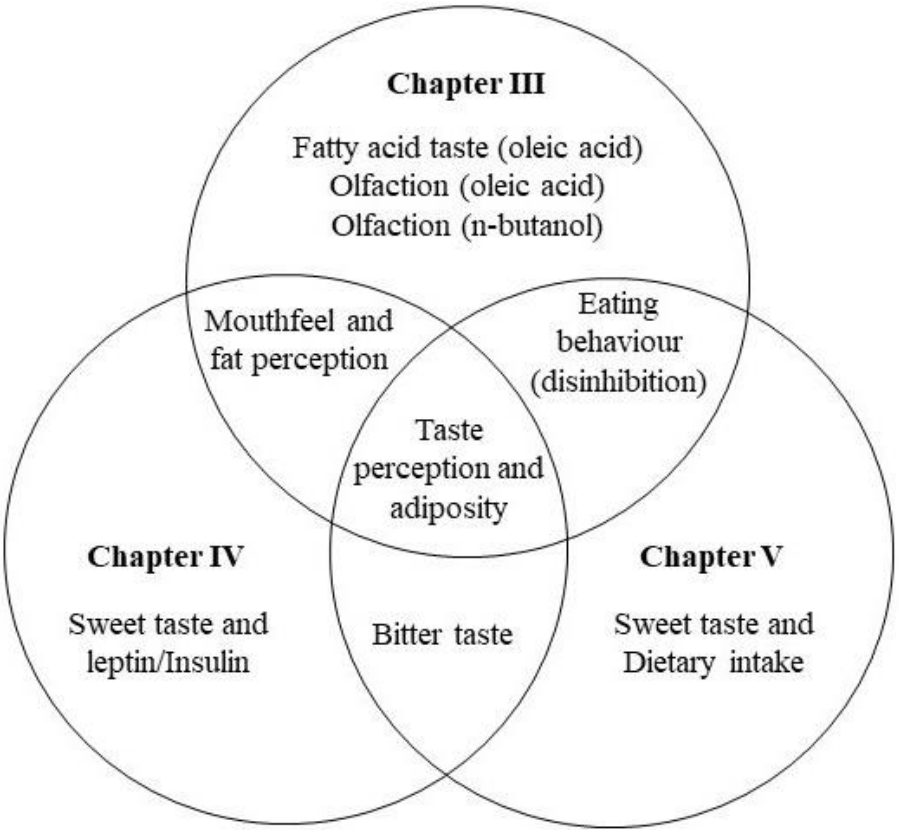

Figure 6.1. Venn diagram and summary of key factors that are associated with taste perception

6.2.1. Associations between fatty acid taste, olfaction, eating behaviour, dietary intake and adiposity

In the study of chapter 3, fatty acid taste was measured and found to be a reproducible and reliable measurement, validating this approach as a quantitative tool. Fatty acid taste detection, alongside olfaction and mouthfeel perception, was found to vary between individuals and was associated with eating behaviour and adiposity (**hypothesis 1 accepted**). We found no clear associations between fatty acid taste perception and dietary intake. It is likely that dietary intake measurement by a FFQ is less suitable for comparing direct taste-diet relationships and that a food record to measure actual intake should be explored in future studies.

In a 'real world' food setting, the perception of fat content would be further enhanced

by the combined perception of taste, olfaction and mouthfeel (Rolls, 2015). Olfactory based discrimination of individual fatty acids supports the notion that humans can detect small concentrations of fat content by odour alone (Kallas & Halpern, 2011). Fatty acid recognition mechanisms are complex, and this study has provided evidence to suggest that multimodal sensory inputs which contribute to fat sensation are interrelated. A recently published *in vitro* study has found that functional olfactory receptors are present in the vicinity of human taste receptors in taste buds of the tongue (Malik, Elkaddi, Turkistani, Spielman, & Ozdener, 2019). To our knowledge, this was the first study to compare oleic acid taste, olfaction and mouthfeel perception alongside dietary intake, eating behaviour and adiposity. Our research suggests that it is feasible for future studies to consider using multimodal classifications of fatty acid hyper- and hyposensitivity. It is conceivable that the multimodal classification of fatty acid sensitivity will have a stronger link with long-term food choice, as the enjoyment and liking of food is based on the experience of the whole food (e.g. taste, aroma, mouthfeel, appearance, etc). Therefore, the future implications of this work are that multiple sensory modalities will continue to be measured and compared, with a view to better understand the associations and non-associations that may exist between each sensation related to dietary fats.

6.2.2. Associations between sweet taste, bitter taste and fatty acid taste perception and obesity

The results of this thesis indicate that some measurable differences in taste perception are associated with obesity. In the PROMISE study, we found that incorrectly ranking the sweet taste task was significantly associated with higher body fat. This finding was further supported by additional measurements of sweet taste liking, whereby NZ European sweet taste likers were twice as likely to have higher body fat % when compared to sweet taste dislikers (**hypothesis 2 accepted; for sweet taste perception**). Bitter taste disliking was associated with having >35% body fat when compared to bitter taste liking (**hypothesis 2 accepted; for bitter taste perception**). In the Dessert Taste study, fatty acid taste hyposensitivity was associated with higher BMI (**hypothesis 2 accepted; for fatty acid taste perception**). However, in the PROMISE study, there were no associations between milk fat perception measurements

(discrimination of taste by ranking task, hedonic liking or intensity rating) and body fat % (**hypothesis 2 rejected; milk fat perception**).

Past studies investigating sweet taste sensitivity have shown varying results, where taste sensitivity was positively correlated with BMI (Hwang et al., 2016; Joseph, Reed, & Mennella, 2015), and negatively correlated with BMI (Bartoshuk, Duffy, Hayes, Moskowitz, & Snyder, 2006; Overberg, Hummel, Krude, & Wiegand, 2012). Furthermore, a large number of studies have reported no association (Cox, Hendrie, & Carty, 2015; Jayasinghe et al., 2017; Tan & Tucker, 2019). In comparison with previous studies, this study used body fat % instead of BMI to draw these comparisons with obesity, nevertheless, the current study supports the notion that a decreased perception of sweet taste is associated with increased adiposity. In addition, an incorrect sweet taste ranking task outcome, indicative of a poor acuteness of broader taste functioning (Kaufman et al., 2018), was associated with increased adiposity in our study. However, these results should be interpreted with caution as complex variations can exist within sub-populations and environmental factors may further confound such associations (Methven, Allen, Withers, & Gosney, 2012; Williams, Bartoshuk, Fillingim, & Dotson, 2016).

Previous studies have suggested that increased sensitivity to bitterness may lead to a decrease in the consumption of healthy, bitter-tasting foods (i.e. cruciferous vegetables) (Turner et al., 2018). Like sweet taste perception, several studies on bitter taste perception have attempted to relate taster status to obesity. However, results are mixed, and to date, a clear link has not been confirmed (Deshaware & Singhal, 2017; Drewnowski, Henderson, & Cockroft, 2007). Bitter taste perception has been regarded as a marker for general taste acuity (i.e. supertaster status, or PROP taster), and that people with a genetic sensitivity to bitter may also be more sensitive to other tastes; however these associations were disputed recently (Garneau et al., 2014). The current study suggests an increased hedonic liking of bitter taste, specifically to that of quinine, is associated with decreased adiposity.

As previously mentioned, (chapter three) fatty acid taste perception was associated with increased adiposity. In healthy weight individuals, maintaining a lower body weight has been previously linked with higher sensitivity to oral fatty acids (Brown, McLay-Cooke,

Gray, & Tey, 2015; Stewart et al., 2010). Chapter three supports a proposed mechanism of fatty acid taste hypersensitivity being an innate predisposition toward improved long-term weight maintenance. Therefore, long-term fat hyposensitivity may have a subtle influence on increasing the dietary intake of fat.

In the PROMISE study (chapter four), the increased hedonic liking of sweet taste was associated with the dose-dependent decrease in gLMS intensity ratings of glucose solutions. Additionally, increased circulating plasma concentrations of the adipogenic hormones leptin and insulin were significantly associated with sweet taste liking status. Important associations have been identified between circulating leptin concentrations and sweet taste perception in previous studies, including that sweet taste recognition matches the diurnal variation in circulating leptin concentration levels (Nakamura et al., 2008). However, this pattern of sweet taste recognition alongside circulating leptin is disassociated in the obese state (Sanematsu, Nakamura, Nomura, Shigemura, & Ninomiya, 2018). Taken together, this information indicates there may be important suppressive effects on the sweet taste receptor by leptin, which has been previously shown to impact on sweet taste perception *in vitro* (Kawai, Sugimoto, Nakashima, Miura, & Ninomiya, 2000; Yoshida et al., 2015). These results further explain the role of leptin and insulin for the maintenance of glucose homeostasis and long-term healthy body weight (Amitani, Asakawa, Amitani, & Inui, 2013). A detailed description of these mechanisms and taste perception associations are described below.

In relation to body weight, the adipocyte hormone leptin is an important regulator of metabolic homeostasis and is able to inhibit food intake and increase energy expenditure (Covey et al., 2006; D'souza, Neumann, Glavas, & Kieffer, 2017). This study has revealed associations which indicate that hyperinsulinemia and hyperleptinemia, appear to be associated with sweet taste suppression via the functionality of the sweet taste receptor. However, these associations were specifically observed in a healthy group of premenopausal women. The sweet taste receptor cell itself has a receptor for leptin (Ob-Rb), but it does not have a receptor for insulin (Behrens & Meyerhof, 2019). The leptin receptor co-localises with T1R3 expressing sweet-sensing taste cells (Kubasova, Burdakov, & Domingos, 2015). The adipoinsular axis is a dual hormonal feedback system involving the hormones insulin and leptin, produced by pancreatic β-cells and adipose tissue, respectively (Kieffer & Habener,

2000). It is therefore likely that the apparent influence of insulin on taste perception, as observed in our present study, is indirect and rather based on insulin's effect of increasing adipose tissues mass and circulating leptin secretion (Kawai et al., 2000; Yoshida et al., 2015). Importantly, the taste-modifying effect of leptin *in vitro* is selective for the taste quality sweet, whereas bitter, umami, salt and sour tastes remain unchanged (Kawai et al., 2000). A lack of an association between leptin and bitter taste or milk fat perception was found in this study, which supports the specificity of the endocrine mechanism described above. These findings indicate that the insulin – leptin – sweet taste receptor feedback system, is an important taste regulating pathway and a potential focus for future research.

In the PROMISE study, higher levels of adiposity were associated with an increased disinhibited eating behaviour score in NZ European women. Disinhibition-BMI relationships have been observed in previous studies (Hays & Roberts, 2008; Kruger, De Bray, Beck, Conlon, & Stonehouse, 2016). It has been previously reported that overweight and obese individuals exhibit increased disinhibited or emotionally disinhibited eating behaviour compared to healthy weight individuals (McLay-Cooke, 2017). In both the Dessert Taste study and the PROMISE study, disinhibited eating behaviour was associated with fatty acid taste hyposensitivity and sweet taste liking, respectively. However, these findings were only found within the NZ European population. These findings indicate that fatty acid taste and sweet taste liking status may influence the type of foods that are consumed, which may be further associated with specific cognitive eating behaviours (Sijtsema, Reinders, Hiller, & Dolors Guàrdia, 2012). For example, psychological factors (i.e. chronic stress, mood) may influence appetite and food selection (Papier, Ahmed, Lee, & Wiseman, 2015) where it has been shown that increased adrenocortical activity is associated with reduced taste intensity (Al'Absi, Nakajima, Hooker, Wittmers, & Cragin, 2012).

6.2.3. Associations between taste perception and dietary intake

In chapter five, sweet likers had a significantly higher intake of carbohydrates, sugars and starch compared to sweet dislikers, in NZ European women only (**hypothesis 3 accepted; for sweet taste perception in NZ European women**). In contrast, in Pacific

women, there were no differences in dietary intake between taster groups (**hypothesis 3 rejected; for sweet taste perception in Pacific women**). Other minor associations were found, for example, Pacific (>35 BF%) and NZ European (<35 BF%) bitter likers had decreased protein intake (grams) compared to bitter dislikers in the same body fat % group. In Pacific women with high body fat %, milk fat likers had higher protein intake (grams) than milk fat dislikers. In addition, NZ European bitter likers had higher dietary fibre intake (% energy) than bitter dislikers. However, it can be concluded that no consistent associations were found when comparing bitter taste and milk fat perception measurements to dietary intake (**hypothesis 3 rejected, for bitter taste perception and milk fat perception**).

The use of hierarchical cluster analysis provides an advanced approach to aid the investigation of the associations between taste perception and dietary intake, eating behaviour and body fat. This approach was important because previous studies have shown substantial variation in the pattern of hedonic responses to increasing concentrations of a taste stimulus. Furthermore, other studies have relied on determining hedonic liking status based on the rating of a single concentration of taste stimulus. In this study we were able to define sweet, bitter and milk fat likers and conversely sweet, bitter and milk fat dislikers from ratings at multiple concentrations. Hierarchical cluster analysis has been used in recent studies (Asao et al., 2015; Garneau, Nuessle, Mendelsberg, Shepard, & Tucker, 2018; Kim, Prescott, & Kim, 2014) and the present study supports the use of this method.

Recent studies have shown a link between suprathreshold sweet taste perception and energy intake (Low, Lacy, McBride, & Keast, 2016), as well as carbohydrate and sugar intake (Jayasinghe et al., 2017) and between carbohydrate taste sensitivity and energy intake (Low, Lacy, McBride, & Keast, 2017). This study has further substantiated these previous results by showing that NZ European women sweet likers consumed more carbohydrates and sugar in comparison to sweet dislikers.

In a recent systematic review, studies that used suprathreshold concentration levels of tastants were more likely to show associations with dietary intake (Tan & Tucker, 2019). For example, salt taste threshold measurements do not predict intake of salt or alcohol while suprathreshold measures do (Duffy, Peterson, & Bartoshuk, 2004; Hayes,

Sullivan, & Duffy, 2010; Lucas, Riddell, Liem, Whitelock, & Keast, 2011). However, a large number of studies to date have used threshold measurements to compare taste perception with dietary intake (Han, Keast, & Roura, 2017; Martinez-Cordero, Malacara-Hernandez, & Martinez-Cordero, 2015; Smith, Ludy, & Tucker, 2016) which may account for the variability in findings. There are still relatively few studies that have compared taste at suprathreshold levels to derive patterns of taste liking by hierarchical cluster analysis (Garneau et al., 2018; Iatridi et al., 2019).

This work has highlighted that there are dietary intake differences between population groups (i.e. Pacific and NZ European). Pacific women had higher intakes of carbohydrates, sugar, and starch and lower intakes of protein, fat, polyunsaturated fat, dietary fibre and alcohol, compared to NZ European women. In NZ, this may in part be due to Pacific people having a higher level of socioeconomic deprivation in comparison to the NZ European population. Socioeconomic factors, such as increased levels of income, have been previously associated with improved diet and food choice (Thiele, Mensink, & Beitz, 2004). In a large study in the USA, people with higher-income had higher diet quality scores than those with lower-income (Zhang et al., 2018). Further evidence of the increasing disparity between high- and low-income groups was evidenced in a study by Leung et al (2012). Lower-income adults were not consuming recommended amounts of whole grains, fruit, vegetables, fish, nuts, seeds and legumes with approximately 13-22% not meeting any of the food and nutrient guidelines (Leung et al., 2012). The conclusion we draw from these previous results is that socioeconomic status has an impact on food choice. Future work should obtain additional information on factors influencing food choice, such as genetic information, to better understand the taste-diet associations found in the present study.

6.2.4. Associations between sweet taste perception, dietary intake and regulators of energy metabolism

New Zealand European women who were sweet likers had higher fasting plasma insulin, plasma leptin, circulating glucose concentration and decreased HDL cholesterol when compared to sweet dislikers (chapter 4). This is the first clear evidence that sweet liking is linked with a range of regulators of energy metabolism and metabolic health status. Further, sweet taste liking (characterised by decreased sweet taste intensity

rating) was associated with increased dietary intake of carbohydrates, starch and sugars (chapter 5). Brought together, there is a taste-diet association regarding carbohydrate intake, and this is related to changes in circulating concentrations of regulators of energy metabolism in NZ European women. This suggests that sweet taste perception is an important indicator of long-term health status driven by peripheral and central effects.

Leptin receptors have been shown to regulate glucose homeostasis by suppressing the production of glucagon and corticosterone, increasing glucose uptake and inhibiting hepatic glucose output (D'souza et al., 2017). The intimate link between sweet-sensing taste cells (T1R3) and the leptin receptor (Ob-Rb) in taste buds suggests an important regulatory pathway for the intake of sugar and carbohydrates (Martin et al., 2010). An important link exists between taste perception and peripheral energy balance, which involves hormonal signalling, impacting on both food intake and peripheral glucose homeostasis (Martin, Maudsley, White, & Egan, 2009). This is supported by decreased sweet taste intensity rating (i.e. sweet liking) being related to increased intake of dietary sugars in this study. The clinical implications of these results are promising, as leptin therapy alongside dietary intervention may have a stronger impact on long-term appetitive and regulatory mechanisms which are driven by the central nervous system (Monteiro & Batterham, 2017).

However, it is important to note that the participants who took part in the PROMISE study were healthy. The reason for the differences observed cannot be determined from a cross-sectional study, however, a higher proportion of the Pacific women in this study had hyperinsulinemia and hyperleptinemia when compared to NZ European women. It can be speculated that there may be differences in the leptin receptor interface (Ob-Rb) which may be associated with the level of taste receptors (T1R3) in this population group (Yoshida et al., 2015). A better understanding of this pathway across different population groups may further explain the differences observed. Furthermore, these associations may be used to design intervention studies that prevent the cascade of events which lead to metabolic disorders such as type 2 diabetes.

6.3. Study strengths and limitations

In this PhD programme, a series of experiments were conducted to investigate associations between taste perception, dietary intake, eating behaviour and circulating concentrations of glucose and endocrine regulators of energy metabolism and body fat. Several previous studies have attempted to identify taste-diet associations, however, consistent relationships have not been found due to the heterogeneity in taste stimuli and variations in dietary intake assessment tools (Cox et al., 2015) used in these studies. Additionally, previous studies typically have used smaller sample sizes and often did not compare representative populations of participants both with, and without obesity (Appleton, Tuorila, Bertenshaw, de Graaf, & Mela, 2018; Cox et al., 2015; Keast, 2016). Key considerations and strengths of this PhD research programme were the use of reliable measurements of taste perception, robust dietary intake methodology and measurement of metabolic markers and endocrine regulators that are functionally linked to body fat. Each of these key factors will be discussed below.

6.3.1. Participant characteristics

Strengths of the studies

All participants who took part in both the Dessert Taste study and the PROMISE study were premenopausal women (aged 18 – 45 years). The women were healthy and lived in Auckland, NZ. The Dessert Taste study was a fatty acid taste test re-test study that involved 50 study participants, which only included NZ European women. The advantages included the homogenous study group to limit biological confounding factors (i.e. gender). Participant retention was high in this study (one dropout only).

The PROMISE study represented a larger study population of both Pacific and NZ European women. This larger study included participants who were representative of four different groups (i.e. Pacific and NZ European women stratified by low and high body fat %). Most of the recruitment was conducted outside of the university setting and

through external contacts. In the PROMISE study, we had recruitment support from our research staff and a senior Pacific research nurse in partnership with the Fono Primary Healthcare Service (Kindleysides et al., 2019). This support framework was highly successful in supporting our engagement with the Pacific women, ensuring that the study was conducted in a culturally appropriate way.

Limitations

This research only included female, premenopausal women of 18 – 45 years of age. This was a limitation as associations in males, or else in older or younger age groups, was not ascertained. In the Dessert Taste study (Chapter 3), many of the participants in this convenience sample were university students or those with a keen interest in nutrition and health. Therefore, a limitation of the Dessert taste study was the convenience sample of participants who took part. However, the study population was adequately powered to meet the primary aims of the study. In the PROMISE study, there were difficulties in recruiting participants that had minimal interest in the research. Therefore, other recruitment strategies (e.g. transport) were required to maintain participant engagement (Kindleysides et al., 2019).

Participants were screened and invited to participate based on self-reported height and weight, based on having either a normal weight profile (BMI: 18.5 – 24.9 kg/m^2) or an obese profile (BMI ≥ 30 kg/m^2). Subsequently, we grouped women into low or high body fat groups, using a cut point of 35% (Dickey et al., 1998; Jo & Mainous, 2018; Oliveros, Somers, Sochor, Goel, & Lopez-Jimenez, 2014). Based on this screening we expected approximately equal proportions of women in each body fat group. However, screening and recruiting women by body fat % initially would have been an advantage. For example, equal separation of the women into groups of <30% body fat, 30.1 – 34.9% body fat, and >35% body fat, may have given more precise information on metabolic risk profile.

In the PROMISE study, the deprivation index was significantly higher in Pacific women in comparison with NZ European women, highlighting the social inequities between the two populations. This is largely due to the current socioeconomic setting in Auckland and the residential areas that we recruited from, being a limitation because the Pacific

and NZ European women typically reside in different areas. There was a range of other complex differences observed between Pacific and NZ European women, which may have been influenced by socio-cultural factors (i.e. dietary intake and eating behaviour). The current health inequalities observed in Pacific youth in NZ (Tupai-Firestone et al., 2016), indicate that socioeconomic status is an important influence on food choice. The associations between taste perception and dietary intake and how these are influenced by environmental drivers of food choice, including affordability and lifestyle, warrants further investigation.

In addition, further heterogeneity in genetics and socio-cultural setting may exist within the subpopulations of the PROMISE study. For example, NZ European women with high body fat % had a higher deprivation index score than NZ European with low body fat %. In our Pacific cohort, approximately half of the women were of Tongan descent, alongside women of Samoan, Fijian, Niuean, Tokelauan, Cook Island Māori and Tuvaluan descent. Similarly, in NZ European women approximately half of the women were born in NZ, alongside women who were born in Europe, South Africa and Australia. We chose to stratify by these broader population groups only (i.e. Pacific and NZ European) as current public health recommendations and reports are based on these classifications (Ministry of Health., 2017). Further associations may have been elucidated by the investigation into variations in single nucleotide polymorphisms (SNPs) and taste inheritability in our study population (Reed, Tanaka, & McDaniel, 2006). Genotype and phenotype comparisons could have provided more precise information on taster status.

6.3.2. Study design

Strengths

Cross-sectional studies allow for multiple comparisons at one point in time, and both the Dessert Taste study and the PROMISE study allowed for this. Biological measurements were compared across groups with approximately even representation in sample sizes. The allocation of participants into distinct categories in the PROMISE study allowed for stratification and effect modification. For example, the recruitment emphasis on obese versus normal body fat % groups in the PROMISE study was an efficient approach to identify and contrast biological parameters that are associated with obesity-related metabolic disease risk.

Limitations

The challenges of a cross-sectional study include the bidirectional nature of some associations. Therefore, the causality of the associations identified in this study cannot be inferred (Sedgwick, 2014). It is useful to first establish the existence of potential associations between taste perception, dietary intake, eating behaviour and markers of metabolic health, prior to determining causation. The multiple comparisons and a large number of assessments required stratification of the data. More research is required in order to establish the cause and effect of these study outcomes.

In the PROMISE study, the selection of obese women who self-reported as healthy (Kindleysides et al., 2019) meant that true random selection of the obese population was not obtained (Tripepi, Jager, Dekker, & Zoccali, 2010). Given that across the general population the number of ‘unhealthy’ women would be higher in those who are obese compared to non-obese (Schwartz et al., 2017), there is some risk of selection bias as only ‘healthy’ obese women were recruited into the PROMISE study. Therefore, we cannot rule out that observed differences may have been influenced by this.

6.3.3. Taste testing methodology

Strengths

A key aim of this research programme was to measure and characterise taste perception. Without reliable evaluation of taste perception, further comparisons with dietary intake or endocrine regulators of energy metabolism and body fat would not be feasible. Firstly, this research further established the test re-test reliability and reproducibility of oral fatty acid threshold testing. The modelling of taste was based on the probability of correctly identifying taste at each concentration level, which measured concentration levels beyond the previously proposed 'stopping point' rule (Mattes, 2007). Binomial regression models of fatty acid taste allowed for the interpolation of an individual's performance across multiple study visits, which classified each person as either hypo- or hypersensitive. Establishing a classification of taste perception based on modelling the probability of success or failure in the identification of oleic acid taste strengthened this data.

In the PROMISE study, the measurement of suprathreshold concentration ratings of intensity and hedonic liking by gLMS and LAM scales were used. This has been cited as an appropriate taste perception measurement for investigating comparisons with dietary intake (Low et al., 2016). In particular, taste perception measurement by suprathreshold concentrations of milk fat allowed for the measurement of hedonic liking at 'real-world' concentration levels (Low et al., 2016). As an additional measurement, non-subjective ranking tasks were conducted with sweet, bitter and milk fat solutions. Data from sweet taste perception measurements by LAM scales can be used to produce distinct hedonic liking clusters. Hierarchical cluster analysis is advantageous because it is a tool for examining the underlying structure of seemingly homogenous data and does not require a *priori* decision regarding the number of clusters (Garneau et al., 2018). Therefore, hierarchical cluster analysis has likely improved the quality of the study and created stronger associations than previously used "cut-off methods" (Methven, Xiao, Cai, & Prescott, 2016).

Limitations

The limitations of taste perception measurements in the PROMISE study included: (1) participant time constraint, (2) no fatty acid taste threshold measurements, and (3) lack of repeated, test re-test taste perception measurements. Fatty acid taste measurements would have been useful in order to substantiate the associations which were found in the previous Dessert Taste study. In the PROMISE study, we did not measure fatty acid taste perception by means of detection threshold (i.e. oleic acid taste) as the participant burden would have been too high. Participants in the PROMISE study did taste testing at one study visit only, in a fasted state, prior to breakfast. Similarly, repeated measurements of taste were not feasible due to the implications of the increased participant burden of repeated study visits. Ideally, future studies will incorporate the evaluation of real-world or as eaten stimuli as a measure of taste hedonics, as these have been defined as a superior measurement compared to liquid or model solutions (Cox et al., 2015). Studies which have used real-world or as eaten stimuli have further concluded that the results are more relevant and valid, particularly when drawing comparisons with dietary intake (Joseph et al., 2015; Mennella, Finkbeiner, Lipchock, Hwang, & Reed, 2014).

6.3.4. Dietary intake and eating behaviour assessment tools

6.3.4.1. Food record and food frequency questionnaire (FFQ)

Strengths

This study employed a 5-day non-consecutive estimated food record. Dietary intake data obtained using this assessment tool was robust. It is the ‘gold standard method’ to assess food intake (De Castro, 1994). Food records were reviewed by a NZ registered dietitian in a one on one interview with each participant. The 220-item validated multi-nutrient, culturally appropriate, semi-quantitative FFQ for use in young adult women (the New Zealand Women’s Food Frequency Questionnaire (NZWFFQ)) was used in the Dessert Taste study (Beck, Houston, McNaughton, & Kruger, 2018). The advantages of using the FFQ was the low participant burden. The FFQ was useful in

obtaining information on usual intake of food groups.

Limitations

In the PROMISE study, associations between taste perception and macronutrient intake measured from the food record were reported. However, additional information on food group data and therefore choices of the types of food consumed would have ideally been reported also. Deriving information on food group intake from food record data is a complex process (Faber et al., 2013). It would be beneficial to expand carbohydrate analysis to include a breakdown of the quantities of added and free sugar contents, or the individual sugars (i.e. glucose, sucrose, fructose, maltose, lactose).

In the Dessert Taste study, the only dietary association found was that usual intake of nuts, nut spreads and seeds, were positively correlated with increased olfactory sensitivity of oleic acid. It is unknown if the lack of taste-diet associations was due to the true lack of any associations, or diluted FFQ measurement (Sandell et al., 2014), or if detection thresholds are less indicative of 'real-world' taste experiences (Low et al., 2016; Mennella et al., 2014). This research suggests that measurements of acute intake (i.e. food record) may strengthen taste-diet comparisons. Therefore, the use of an FFQ may be better for studies that measure long-term food intake, data in large populations, or for the development of dietary patterns (Schrijvers, McNaughton, Beck, & Kruger, 2016; Tan & Tucker, 2019). In addition, a FFQ is a useful tool for ranking individuals based on nutrient intake, rather than deriving absolute intake (Beck et al., 2018).

6.3.4.2. Three-factor eating questionnaire (TFEQ)

Strengths

The TFEQ is a well-known, validated eating behaviour tool which measures the cognitive domains of restraint, disinhibition and hunger, that has been used in a wide range of previous studies. Within the NZ European population, consistent relationships were found between eating behaviour scores. Eating behaviour scores and key associations in this study (i.e. BMI) were consistent with previous studies conducted in

young women (Lesdéma et al., 2012) and young NZ European women (Kruger et al., 2016).

Limitations

It is unknown in this study as to whether the results found in Pacific and NZ European women are comparable. Whilst the TFEQ is a validated tool across other ethnic groups (Chong et al., 2016; Löffler et al., 2015) it is unknown if it is a culturally appropriate tool in Pacific women. Previous studies have used or developed a culturally appropriate TFEQ (i.e. for a Thai population) (Chearskul, Pummoung, Vongsaiyat, Janyachailert, & Phattharayuttawat, 2010; Rosnah, Noor Hassim, & Shafizah, 2013). These previous studies required further adaptation beyond translating from the original English version (Stunkard & Messick, 1985).

6.4. Final conclusions

This PhD research programme presents several key and novel findings. Firstly, in the Dessert Taste study, we have shown that fatty acid taste perception is positively correlated with fatty acid olfaction sensitivity in humans. The eating behaviour factor of disinhibition, which was associated with increased adiposity, was significantly higher in women who were hyposensitive to oleic acid taste. Dietary intake of nuts, nut spreads, and seeds was significantly correlated with high olfactory sensitivity to oleic acid.

In the PROMISE study, participants with higher adiposity were more likely to incorrectly rank the sweet taste task. In support of this, the hedonic liking of sweet taste was associated with higher body fat %. Conversely, bitter taste likers had lower body fat % than dislikers. It was further demonstrated that increased circulating concentrations of plasma leptin and insulin were associated with being a sweet liker. Our findings in NZ European women generate new insights into the possible physiological mechanisms, involving the dual hormonal feedback system insulin and leptin, which may link taste perception and obesity in humans: (i) with increasing obesity, the increased circulating concentrations of leptin suppress sweet taste perception in sweet-sensing taste cells; (ii) sweet liking is significantly associated with decreased intensity rating of sweet taste, which is further indicative of suppressed sweet taste receptor action, (iii) consequently, an increase in the intake of refined carbohydrates and sugar occurs in order to compensate for a lack of sensitivity to sweet taste (Ettinger, Duizer, & Caldwell, 2012), and (iv) disinhibited eating behaviour may increase due to decreased sensitivity towards endocrine regulators of appetite and energy metabolism (Gerspach, Steinert, Schönenberger, Graber-Maier, & Beglinger, 2011). Increasing adiposity exacerbates this cycle due to an increase in plasma leptin, as well as plasma insulin concentrations and the dysregulation of the adipoinsular axis (Kaufman et al., 2018; Kieffer & Habener, 2000). The novel insights from this PhD research suggest that sweet taste perception may mirror a state of leptin sensitivity in healthy individuals. In leptin sensitive individuals, leptin inhibits insulin synthesis and secretion from pancreatic β-cells. In contrast, leptin-resistance, as found in obesity, decreases the permeability of leptin to the brain, resulting in insufficient leptin signalling to the hypothalamus which dysregulates appetite and cognitive mechanisms that drive eating behaviour and appetite

control (Amitani et al., 2013).

Despite having shown that these important mechanistic pathways potentially link dietary intake with sweet taste perception, this cascade of associations was not clearly present in Pacific women. Fasting insulin and leptin concentrations were highest in Pacific women which may suggest that the hyperinsulinemia and hyperleptinemia may reflect an advanced dysregulation within the adipoinsular axis and a re-setting of the feed-back loop with the sweet-taste receptor in Pacific women. Whether this re-setting of the feed-back loop with the sweet-taste receptor is based on genetic or environmental factors (e.g. diet) requires further investigation. However, it suggests that there are other important external drivers of dietary intake which may supersede these innate biological influences on eating behaviour. Additionally, only minor associations were found between bitter taste and milk fat perception with dietary intake, eating behaviour or circulating concentrations of glucose and endocrine regulators of appetite and energy metabolism. The findings of the current study support that there are important associations between taste perception and the long-term regulation of weight maintenance and metabolic health. However, multiple drivers of obesity, including environmental influences on food choice, need to be considered in order to determine the true impact of the biological associations observed in this study (Vandenbroeck, Goossens, & Clemens, 2007).

Finally, in the PROMISE study, comparisons were made between two populations with markedly different metabolic disease risk (Pacific and NZ European women) and different levels of body fat (low and high body fat %). In chapter 5, it was observed that there were differences in dietary intake between Pacific and NZ European women. For example, Pacific women consumed higher proportions of carbohydrates and starch, but lower proportions of dietary fibre, in comparison to NZ European women. However, all Pacific and NZ European women consumed more fat, saturated fat and total sugar; and had inadequate carbohydrate intakes compared with general recommendations (NHMRC, 2006; WHO, 2015). Only NZ European women with low body fat % met the recommended intake for dietary fibre. In addition to this, the associations between sweet taste liking and carbohydrate and sugar intake observed in NZ European women were not observed in Pacific women. The data suggest that in a population with higher metabolic disease risk (i.e. significantly higher insulin), dissimilarities in taste-diet

associations may occur in comparison to a population with lower metabolic disease risk. Future research may be able to further investigate these distinct associations in order to reduce current health inequities, which is a critical public health concern.

6.4.1. Public health implications

Based on the findings in this thesis, some future public health recommendations can be made. Firstly, the information gained through this cross-sectional study can be used to establish future longitudinal and intervention studies, that investigate if alterations in taste perception lead to shifts in preferences for unhealthy foods (Wise, Nattress, Flammer, & Beauchamp, 2016). Furthermore, novel or targeted therapies which reduce adiposity may aid in the control of circulating plasma leptin concentration, to alter sweet taste perception alongside dietary restrictions. Such therapies may improve leptin sensitivity, which would improve the long-term regulation of pathways which control food intake and cognitive signals which drive appetite and satiety (Amitani et al., 2013). Targeted weight loss therapies ideally would mimic the effects of bariatric surgery, which is known to enhance taste perception (Zakeri & Batterham, 2017), or potentially enhance taste acuity itself which may drive the improvements of long-term regulation pathways.

Potentially the strongest impact on public health would come from government strategies to reduce sugar intake, which could include the introduction of a ‘sugar tax’ (Backholer et al., 2016; Nakhimovsky et al., 2016). This would likely result in a population-wide decrease in the intake of food and beverages that are high in added sugar and consequently, would improve long term metabolic health outcomes. More crucially perhaps, this action would target population groups who have a lower socioeconomic status (Cropp, 2017). If taste-diet relationships are associated with habitual dietary intake, this public health action would theoretically improve the biological feedback mechanisms described in this chapter.

Previous studies have indicated that ‘taste learning’ can occur with multiple exposures to a new or novel tastant (i.e. non-esterified fatty acids) (Running, Mattes, & Tucker, 2013; Tucker & Mattes, 2013). This learning indicates that with repeated exposures an

individual may become more attuned to the stimulus. This suggests that 'taste learning' could be an additional tool to enhance taste perception in obesity research. A possible strategy for this would be to repeat exposures to distinct taste sensations that may improve the acceptance and reward signalling pathways associated with that taste (i.e. bitter taste). However, this type of therapy may be difficult to design given the genetic differences that exist for specific taste sensations, particularly for bitter taste, which is associated with food rejection (Keller & Adise, 2016; Yeomans, 2010). The further consideration of 'taste learning' and how this biological mechanism may be advantageous to metabolic health and public health interventions is warranted. It can be speculated that the acceptance of a diverse range of taste and sensory sensations would result in the acceptance of a diverse range of healthy food choices.

It is important that new breakthrough concepts to tackle the obesity epidemic become available, as current health recommendations and dietary guidelines are not being adhered to (Rush, Savila, Jalili-Moghaddam, & Amoah, 2018; Wall et al., 2016). Ideally, recommendations to the general public could become more personalised and informative, which may be based on individual phenotypic and genetic differences in taste perception. The nutrition information presented on food packages could provide further detail on this (i.e. how processed a food is) in comparison to products within the same food category (van Dongen, van den Berg, Vink, Kok, & de Graaf, 2012). For example, a highly processed food product which is high in added sugar could be labelled as a high-risk product for those phenotypically identified as 'taste insensitive to sweet'. This type of personalised system could be more engaging than the currently used health star rating or traffic light front-of-pack nutrition labelling systems (Jones, Thow, Ni Mhurchu, Sacks, & Neal, 2019). In this way, consumers can make informed choices and reduce their energy intake from highly processed foods which are high in added sugar and fat.

6.4.2. Food industry implications

In highly processed foods, the perception of taste and other sensory properties do not always accurately reflect the energy density of the food. This study has provided further

evidence that there is heterogeneity in taste perception across different population groups that further impact on food and beverage product perception. There is potential for the use of taste-modifying products or ingredients such as Synsepalum dulcificum (i.e. ‘miracle fruit’ which modifies sour taste sensation into sweet) which could be used to mask healthy functional ingredients (Swamy, Hadi, Sekaran, & Pichika, 2014). Food reformulation should include clear guidelines and measurements of each basic taste as part of the manufacturing process, to design foods where taste is better perceived to reflect the energy value of the food.

6.4.3. Final statement of the implications of this work

The taste-diet, taste-endocrine and taste-adiposity associations observed in this PhD thesis suggest that the chemosensation of basic taste and subsequent signalling mechanisms can significantly impact on long-term dietary habits and eating behaviour. This work provides a strong foundation for future research on taste perception and the design of longitudinal studies and clinical interventions. Such studies will provide advanced strategies to improve dietary intake and food choice with a view to subsequently reduce the prevalence of obesity and improve metabolic health status globally.

6.5. Recommendations for future research

- Incorporate sensory testing of a wide range of whole foods or ‘real-world’ as eaten stimuli which are representative of dietary intake, by measuring comprehensive descriptions of sensory attributes.
- Further explore if similar taste-diet or adiposity associations exist within other population groups (e.g. males, different ethnic groups, children, adolescents, older adults, people with type 2 diabetes, etc).
- Expand taste testing methodology to include measures of sour, umami and salt taste perception to establish further relationships with the whole diet, endocrine regulators of metabolic health and eating behaviour.
- Future studies need to consider different patterns of taste perception, in order to separate different types of tasters (i.e. sweet likers from dislikers).
- It is recommended that future studies adjust findings with important influences on taste physiology such as age, gender and ethnicity.
- Expand olfactory methodology to establish further flavour-nutrient associations with the whole diet.
- Investigate taste receptor polymorphisms, genotypes and taste inheritability in comparison to taste perception phenotypes.
- Investigate and determine the changes to taste liking phenotype across different seasons or diurnally.
- Develop targeted intervention studies to determine if sweet taste perception can be enhanced or modified, particularly in obese populations.
- Further research and development into clear guidelines for each basic taste as a component of food reformulation, that can be individually tailored to be physically perceived in order to better reflect the energy value of food.
- Investigate longitudinally whether being a sweet liker is associated with any distinctive dietary patterns (i.e. Western-style diet) or long-term carbohydrate intake. Dietary patterns may help to provide a better understanding of the relationship between food choice and taste sensitivity.

- Clarify associations between taste perception and ‘wanting’ (i.e. the motivation and decision making in food consumption) and ‘liking’ (i.e. emotional state) to further elucidate long-term dietary habits.
- Conduct further research on biological associations (i.e. taste perception) and food intake in the setting of food insecurity and deprived socioeconomic settings. This is critical in determining if food intake is influenced by sensory sensitivity in a setting with limited access to nutritious food options.
- Investigate and design a culturally adapted version of the TFEQ for the Pacific population.

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APPENDIX

7.1. List of publications, presentations and awards

7.1.1. Publications and abstracts in international journals

Kindleysides, S.; Kruger, R.; Douwes, J.; Tannock G. W.; Renall, N.; Slater, J.; Lawley, B.; McGill, A-T.; Brennan, N.; Manukia, M.; Richter, M.; Tupai-Firestone, R.; Signal, T.L.; Gander, P.; Stannard, S. R.; Breier, B. H. Predictors Linking Obesity and the Gut Microbiome (the PROMISE Study): Protocol and Recruitment Strategy for a Cross-Sectional Study on Pathways That Affect the Gut Microbiome and Its Impact on Obesity. *JMIR Res Protoc* **2019**, 8(8), e14529.

Kindleysides, S.; Kruger, R.; Corbin, M.; Richter, M.; Douwes, J.; Breier, B.H. Sweet Taste Perception in Pacific and NZ European Women is Associated with Dietary Intake and Eating Behaviour. *Proceedings* **2019**, *8*(1), 35.

Kindleysides, S.; Beck, K.L.; Walsh, D.C.I.; Henderson, L.; Jayasinghe, S.N.; Golding, M.; Breier, B.H. Fat Sensation: Fatty Acid Taste and Olfaction Sensitivity and the Link with Disinhibited Eating Behaviour. *Nutrients* **2017**, *9*, 879.

7.1.2. Presentations (oral and poster presentations)

Kindleysides, S., Kruger, R., Corbin, M., Richter, M., Douwes, J., Breier, BH. *Sweet taste perception in Pacific and NZ European women is associated with dietary intake and eating behaviour*. Presented on Thursday, 29th Nov, 2018. Nutrition Society of New Zealand Conference. (Auckland, New Zealand).

Kindleysides, S., Kruger, R., Singh, S., Breier, BH. *Taste sensitivity to glucose is linked to*

body fat percentage and body mass index in New Zealand European and Pacific women. Presented on Tuesday, 28 Nov, 2017. 10th Asia Pacific Conference on Clinical Nutrition (Adelaide Convention Centre, Adelaide, South Australia).

Kindleysides, S., Kruger, R., Singh, S., Breier, BH. *Taste sensitivity to glucose is linked to body fat percentage and body mass index in New Zealand European and Pacific women.* Presented on Friday, 24 Nov, 2017. 4th Annual Postgraduate and Early Career Nutrition Conference (Tamaki Yacht Club, Auckland, New Zealand).

Kindleysides, S., Beck, KL., Walsh DCI., Henderson, L., Jayasinghe, SN., Golding, M., Breier, BH. Fat Sensation: Are fat taste and olfaction sensitivity linked with eating behaviour? Poster presentation, Presented on Monday, 18th Sep, 2017. Journey through Science Day (The New York Academy of Sciences; New York, USA).

Kindleysides, S., Beck, KL., Walsh, D., Henderson, L., Jayasinghe, S., Golding, M., Breier, BH. Fat sensation: are fat taste and olfaction sensitivity linked with eating behaviour? Oral Presentation, Presented on Friday, 9th Dec 2016. Nutrition Society of New Zealand Conference. (Christchurch, New Zealand).

7.1.3. Awards

2017 New York Academy of Sciences (NYAS) and PepsiCo Travel Award to attend the Journey through Science Day at the NYAS; New York, USA.

2016 Nutrition Society Conference First place (winner) first time oral presentation.

7.1.4. Permission for thesis publication and statements of contribution

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We, the candidate and the candidate's Primary Supervisor, certify that all co-authors have consented to their work being included in the thesis and they have accepted the candidate's contribution as indicated below in the *Statement of Originality*.

Name of candidate:	Sophie Kindleysides
Name/title of Primary Supervisor:	Prof. Bernhard H Breier
Name of Research Output and full reference:	
Kindleysides, et al. Fat Sensation: Fatty Acid Taste and Olfaction Sensitivity and the Link with Disinhibited Eating Behaviour. Nutrients 2017, 9, 879.	
In which Chapter is the Manuscript /Published work:	3
Please indicate:	
• The percentage of the manuscript/Published Work that was contributed by the candidate:	80%
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• Describe the contribution that the candidate has made to the Manuscript/Published Work:	
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Name of candidate:	Sophie Kindleysides
Name/title of Primary Supervisor:	Prof. Bernhard H Breier
Name of Research Output and full reference:	
Taste perception and its association with adiposity and endocrine regulation in Pacific and New Zealand European women	
In which Chapter is the Manuscript /Published work:	4
Please indicate:	
• The percentage of the manuscript/Published Work that was contributed by the candidate:	80%
and	
• Describe the contribution that the candidate has made to the Manuscript/Published Work:	
Design of sensory methodology, data collection, data analysis, interpretation of results, main author of manuscript.	
For manuscripts intended for publication please indicate target journal:	
Nutrients or Journal of Endocrinology	
Candidate's Signature:	
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Name of candidate:	Sophie Kindleysides
Name/title of Primary Supervisor:	Prof. Bernhard H Breier
Name of Research Output and full reference:	
Associations between taste perception, dietary intake and eating behaviour in Pacific and NZ European women	
In which Chapter is the Manuscript /Published work:	5
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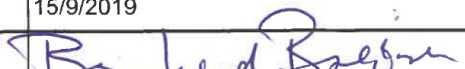
Primary Supervisor's Signature:	
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7.1.5. PROMISE protocol paper

Protocol

Predictors Linking Obesity and the Gut Microbiome (the PROMISE Study): Protocol and Recruitment Strategy for a Cross-Sectional Study on Pathways That Affect the Gut Microbiome and Its Impact on Obesity

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Abstract

Background: The prevalence of obesity has increased substantially over recent decades and is associated with considerable health inequalities. Although the causes of obesity are complex, key drivers include overconsumption of highly palatable, energy-dense, and nutrient-poor foods, which have a profound impact on the composition and function of the gut microbiome. Alterations to the microbiome may play a critical role in obesity by affecting energy extraction from food and subsequent energy metabolism and fat storage.

Objective: We report the study protocol and recruitment strategy of the PRedictors linking Obesity and the gut MIcrobiomE (PROMISE) study, which characterizes the gut microbiome in 2 populations with different metabolic disease risk (Pacific and European women) and different body fat profiles (normal and obese). It investigates (1) the role of gut microbiome composition and functionality in obesity and (2) the interactions between dietary intake; eating behavior; sweet, fat, and bitter taste perception; and sleep and physical activity; and their impact on the gut microbiome, metabolic and endocrine regulation, and body fat profiles.

Methods: Healthy Pacific and New Zealand (NZ) European women aged between 18 and 45 years from the Auckland region were recruited for this cross-sectional study. Participants were recruited such that half in each group had either a normal weight (body mass index [BMI] 18.5-24.9 kg/m^2) or were obese (BMI $_{\geq 30.0}$ kg/m^2). In addition to anthropometric measurements and assessment of the body fat content using dual-energy x-ray absorptiometry, participants completed sweet, fat, and bitter taste perception tests; food records; and sleep diaries; and they wore accelerometers to assess physical activity and sleep. Fasting blood samples were analyzed for metabolic and endocrine biomarkers and DNA extracted from fecal samples was analyzed by shotgun sequencing. Participants completed questionnaires on dietary intake, eating behavior, sleep, and physical activity. Data were analyzed using descriptive and multivariate regression methods to assess the associations between dietary intake, taste perception, sleep, physical activity, gut microbiome complexity and functionality, and host metabolic and body fat profiles.

Results: Of the initial 351 women enrolled, 142 Pacific women and 162 NZ European women completed the study protocol. A partnership with a Pacific primary health and social services provider facilitated the recruitment of Pacific women, involving

direct contact methods and networking within the Pacific communities. NZ European women were primarily recruited through Web-based methods and special interest Facebook pages.

Conclusions: This cross-sectional study will provide a wealth of data enabling the identification of distinct roles for diet, taste perception, sleep, and physical activity in women with different body fat profiles in modifying the gut microbiome and its impact on obesity and metabolic health. It will advance our understanding of the etiology of obesity and guide future intervention studies involving specific dietary approaches and microbiota-based therapies.

Trial Registration: Australian New Zealand Clinical Trials Registry ACTRN12618000432213; https://www.anzctr.org.au/Trial/Registration/TrialReview.aspx?id=370874

International Registered Report Identifier (IRRID): RR1-10.2196/14529

(JMIR Res Protoc 2019;8(8):e14529) doi: 10.2196/14529

KEYWORDS

diet; gut microbiome; body composition; women; overweight; obesity; physical activity; taste perception; sleep; metabolic diseases

Introduction

Background

Obesity is a global health issue of epidemic proportions [1]. The prevalence in New Zealand (NZ) has increased dramatically over the past three decades, with 1.2 million adults (32% of the population) currently being obese [2,3] and NZ ranking as the third most obese country in the Organization for Economic Co-operation and Development [4]. Obesity is related to significant health inequities, that is, Pacific peoples (69%) and Māori (50%) are disproportionately affected compared with the general population in NZ (32%), and rates are highest in the most deprived areas [5]. Recent trends in adult obesity in NZ show a significant rise in overweight (currently 64%) and obesity (currently 34%) in women, with major weight gains between the ages of 18 and 45 years [2,4]. This is of significant concern as increased adiposity in women of child-bearing age is associated with acute maternal, neonatal, and ongoing adverse health outcomes, including the perpetuation of increased obesity risk for the next generation [6]. Interventions to halt the epidemic have been unsuccessful [3,4].

Obesity is a complex, multifactorial condition contributing to a chronic prooxidant and proinflammatory state and to deterioration of glucose and lipid metabolism. It increases the risk of several noncommunicable diseases, including type 2 diabetes (T2D), cardiovascular disease (CVD), and some types of cancer [7,8]. Known contributing factors include imbalances in pathways of glucose and lipid metabolism that occur because of variations in quantity and quality of the diet, sedentary lifestyle, and genetic predisposition [9,10]. Obesity arises as a consequence of how the body regulates energy intake, energy expenditure, and energy storage, and it reflects a state of positive energy balance largely caused by westernized environmental pressures [11] resulting in an energy mismatch. This operates through dietary behaviors that do not trigger strong biological opposition [12]. A vicious cycle ensues, involving a state of excessive insulin secretion and a series of metabolic responses that produce systemic insulin resistance [13]. Desensitization to insulin action is accompanied by increased oxidative stress [14] and increased leptin secretion, inflammation, and a decreased ability to metabolize lipid and default energy storage as adipose tissue [15]. Furthermore, changes in the action of endocrine regulators including insulin, leptin, ghrelin, and glucagon-like peptide-1 (GLP-1) disturb appetite regulation in the obese state, rendering sustained weight loss difficult to achieve [16]. In this setting, the regulation of energy balance is biased toward protection against weight loss, further fat accumulation, and disease progression [12,17].

Current public health research to curb obesity is aimed at developing effective food and nutrition policies [11], promoting healthier food choices [18], and community-based interventions [19]. However, the notion that obesity is caused solely because we consume more energy than we expend does not fully explain the substantial increase in obesity [4,20]. Efforts to reduce obesity by inducing a negative energy balance, by counseling people to either eat less or exercise more, are often ineffective because of multiple physiological, behavioral, and social feedback loops [21]. Commonly cited causes of obesity include major changes in our food environment [3,11], which have led to overconsumption of inexpensive, highly palatable, energy-dense, and nutrient-poor foods. These changes in nutritional habits have been reported to influence the gut microbiome, which comprises the bacterial community of the bowel and its associated genetic endowment [4,22]. Current evidence suggests that the gut microbiome plays an important role in the regulation of energy homeostasis and the development of fat storage and obesity [23]. Proposed mechanisms include the microbiome's capacity to modify energy extraction from food and its ability to influence host signaling pathways that regulate energy metabolism [24]. Furthermore, the gut microbiome can influence satiety and food consumption through gut hormones that trigger endocrine feedback loops regulating appetite [24]. In turn, nondigestible dietary components affect the composition and metabolism of the gut microbiome [25].

The gut microbiome can be viewed as a critical modifiable link between diet and host and may offer new avenues for obesity prevention [26]. Recent studies suggest that people with relatively less diverse microbiomes have higher overall body adiposity and more inflammation-associated characteristics, indicating a higher risk of metabolic diseases [27]. These findings suggest that microbiome complexity and diversity (or richness) may be predictive of the metabolic status of the host and may therefore function as a new biomarker of

metabolic health. Dietary patterns that are associated with gut microbiome composition and dietary interventions can increase microbiome richness [25]. In addition, dietary habits may be the most critical factor influencing microbiome status, and therefore, it is critical to understand diet-microbiome interactions and their effect on human health. Finally, a number of other modifiable biological and behavioral factors in the complex causes of obesity appear to be linked with the gut microbiome: sweet and fat taste perceptions influence appetite and dietary behavior and are linked with body weight [28,29], and sleep duration and quality are linked with changes in appetite regulation and energy metabolism [30,31]. Disruption to the circadian biological clock leads to dysbiosis of the gut microbiome [32], and physical activity increases gut microbiome diversity [33].

Objectives

The PRedictors linking Obesity and the gut MIcrobiomE (PROMISE) study is the first to characterize the gut microbiome in 2 population groups with markedly different metabolic disease risk (Pacific and NZ European women) and different body fat profiles (normal and obese). The potential identification of distinct roles for taste perception, diet, sleep, and physical activity in modifying the gut microbiome and its impact on obesity will greatly advance our understanding of the etiology of obesity and contribute to the discovery of new therapeutic targets. The specific aims of the PROMISE study are to assess in 18- to 45-year-old Pacific and NZ European women: (1) the potential association of gut microbiome complexity and diversity, gene richness, and biochemical endowment in obesity and body fat distribution; (2) interactions between sweet, fat, and bitter taste perception, dietary intake and eating behavior, sleep or physical activity, and their impact on the gut microbiome, metabolic regulation, and body fat profiles; and (3) associations between biomarkers of biological and behavioral risk factors referred to above and specific body fat profiles. This study is designed to test the primary hypothesis that reduced gut microbiome diversity, but high energy-harvest capacity is a key biological driver of obesity and unhealthy body fat distribution in women, whereas greater gut microbiome complexity and gene richness is protective. The secondary hypothesis is that differences in diet, taste perception, sleep, and physical activity affect the associations between the gut microbiome, metabolic regulation, and body fat profiles. This paper reports the outline of the study protocol and the recruitment strategy. Details of the analytical procedures, study outcomes, and clinical measurements will be published elsewhere.

Methods

Conceptual Framework and Study Design

Comparisons between Pacific and NZ European women, who are known to vary the most in terms of physical, ethnic-cultural, and socioeconomic characteristics in NZ, will allow to assess whether findings are different between groups [2]. A further important distinction between the 2 ethnic groups is that a proportion of the participating Pacific women were born in the Pacific Island of origin, retaining their cultural lineage and dietary traditions, [34] and, as a consequence, may also have a distinctly different gut microbiome. This study involved the assessment of diet, taste perception, sleep, and physical activity, and we investigated the complex interactions between the gut microbiome and its impact on obesity, metabolic markers, and endocrine regulators, as described in Figure 1.

Figure 1 describes the central role of the gut microbiome in regulating energy homeostasis and body fat distribution. Aim 1 of this study investigates differential gut microbiome complexity, gene richness, and functionality between obese body fat profile women and normal body fat profile women stratified by ethnicity. For example, we investigate whether specific bacterial phyla are associated with obesity and/or characterized by relative abundances of genes associated with carbohydrate-active enzymes or whether a highly diverse microbiota with lower energy-harvest capacity is protective. Aim 2a investigates the fundamental influence of diet and food intake on body weight and body fat profile. We assess associations with gut microbiome complexity and functionality and explore whether specific microbiota profiles may be associated with specific dietary intakes or cultural dietary traditions. Aim 2b investigates interactions with sweet, bitter, and fat taste perception and Aim 2c explores the influence of sleep and physical activity. We assess independent effects or effect modification of taste perception, sleep, and physical activity and their impact on the gut microbiome and body fat profiles. Aim 3 examines interactions or effect modification with biomarkers and endocrine regulators and their relationships with food consumption, energy metabolism, and a range of risk factors that shape body fat profiles.

Participants

We have conducted a cross-sectional study in 174 Pacific women (who are known to have a high risk of obesity [5]) and 177 NZ European women (who are known to have a moderate risk of obesity [2,5]). Initial screening of body mass index (BMI), based on self-reported weight and height, was conducted on the Web, in person, or via the phone (Figure 2). The 351 participating women, aged 18 to 45 years, were selected such that half in each ethnic group had a normal body fat profile (BMI 18.5-24.9 kg/m^2) and the other half an obese body fat profile (BMI $\geq$30.0 kg/m^2), while recognizing that people with the same BMI can have substantial heterogeneity of body composition and metabolic disease risk factors [35,36].

Figure 1. The central role of the gut microbiome in regulating energy homeostasis and body fat distribution.

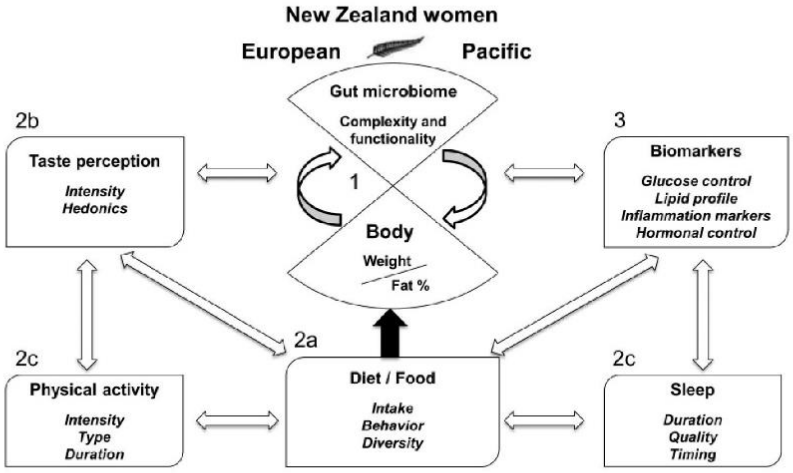
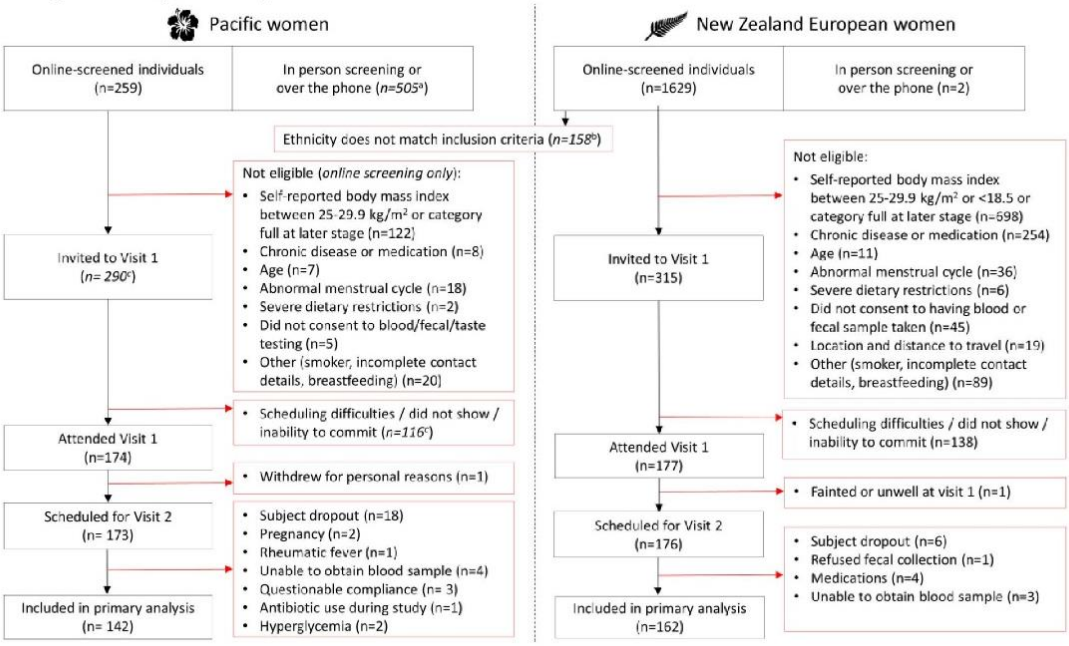


Figure 2. Flowchart describing the recruitment process of the PROMISE study. "a" indicates approximated value for Pacific women in-person screening or over the phone. "b" indicates that ethnicity inclusion was based on Pacific women requiring one parent of full Pacific ethnicity or New Zealand (NZ) European women having lived in NZ for a minimum of 5 years with European parents. "c" indicates approximated values since the majority of study bookings were managed over the phone.



Ethics

The study was approved by the Southern Health and Disability Ethics Committee (16/STH/32). This trial was registered at anzctr.org.au (ACTRN12618000432213). All participants were informed in detail about the procedures and measurements and gave written consent. Access to data is restricted to the immediate research team, and only coded data are used for analysis.

Inclusion and Exclusion Criteria

The inclusion criteria included age 18 to 45 years, being postmenarche and premenopausal (as defined by a regular menstrual cycle for the last year), ethnicity (self-identified Pacific ethnicity and having at least one parent of Pacific ethnicity or self-identified as NZ European ethnicity and having lived in NZ for a minimum of 5 years), written informed consent, willingness to comply with study requirements, and being generally healthy. The exclusion criteria included BMI outside of the predefined normal or

obese BMI ranges, pregnant or lactating, presence of any diagnosed chronic illness (eg, T2D and CVD), previous bariatric surgery, severe food allergies, medication that could interfere with appetite or the immune system (eg, appetite suppressants and corticosteroids), current smoker, severe dietary restrictions or avoidances (eg, vegan), and antibiotic use during the last month.

Participant Recruitment

The Auckland region has a culturally diverse population of 1,534,000, of which approximately 15% are Pacific peoples and 59% NZ European [37]. Health studies sometimes fail to address, or are not appropriately sensitive to, the cultural needs of the participants in terms of the recruitment approach or implementation of the study [38]. We therefore developed a partnership with The Fono, a large Pacific primary health care and social services organization based in Auckland, to integrate culturally appropriate recruitment and research procedures. This included the appointment of a senior Pacific nurse to lead the recruitment and support the data collection and management of the Pacific arm of the study. We also advertised on Pacific Facebook pages and contacted a wide range of special interest and cultural websites; however, we had little uptake from the Pacific organizations or groups that we contacted. As a result, Pacific women were mostly recruited in-person or by phone through the Pacific nurse (see Results). Other recruitment strategies included attending cultural festivals (ie, Pasifika festivals) and other cultural or preorganized events. Another successful strategy was recruiting Pacific students through the University Pacific networks. An additional *Me'a'ofa* (gift or donation) was offered to participants who could successfully enroll another participant who met the inclusion criteria and completed the study, thus enabling recruitment through existing networks. The services of 2 recruitment agencies, PRIME Research Ltd and Consumer Link, were also employed to support the recruitment of Pacific women with a BMI less than or equal to 24.9 kg/m^2. This was a successful strategy because previous networks were exhausted for this group of participants.

NZ European women typically heard of our study through Web-based advertisements such as Facebook community pages or *public figure* pages (well-known NZ nutritionists, etc), work or university email lists, or other social media sources (see Results). A number of Facebook pages, including local public figure pages, shared details of our study and endorsed the research on our behalf. Community Facebook pages (suburbs around Auckland, local businesses, etc) allowed for wide reach across the Auckland region. In contrast to Pacific women, social media (ie, Facebook) was a highly successful method for NZ European recruitment.

Participant gratuity (NZD $100 gift voucher including options for petrol, shopping center, and supermarket vouchers) was handed out at the end of visit 2 to encourage study completion and the return of all home-use devices and data.

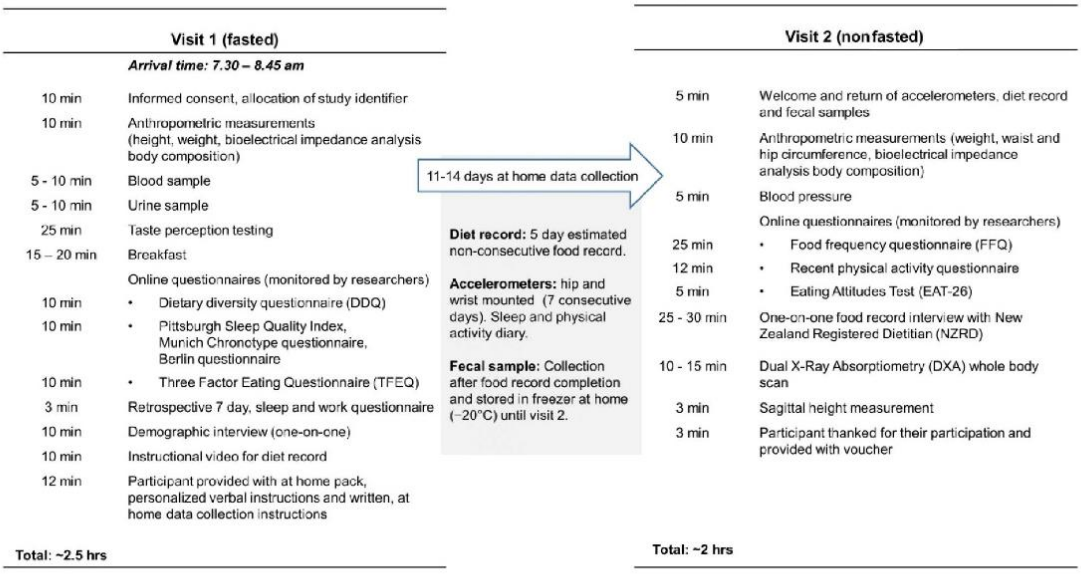
Study Procedures

The PROMISE study was conducted at the Massey University Human Nutrition Research Unit in Auckland, NZ, between July 2016 and September 2017. All eligible participants attended the research unit on 2 occasions, 11 to 14 days apart (Figure 3), where they completed a series of scheduled tasks. Between visits, participants completed the at-home data collection protocols described below. For Pacific women, we ensured that the Pacific nurse was present throughout the duration of Pacific participants' initial visits. She also performed the phlebotomy procedures for all Pacific participants and addressed all concerns about any aspects of the study that were raised. All Pacific participants were offered door-to-door transport to and from the research unit to enable participation in the study and to reduce the likelihood of *dropouts*.

At visit 1, participants were welcomed, asked to carefully read and sign a consent form, and had the opportunity to ask questions before commencing the study. Each participant was then allocated a unique study identifier (ID). Participants completed a one-on-one interview with a researcher to obtain a range of demographic and health information, including the number of biological children, household and personal income, occupation, work patterns, participants' birth weight and delivery method (if known), dietary supplement use, frequency of alcohol consumption, and recruitment method.

Figure 3 presents an overview of the PROMISE study visits and the sequence and timing of procedures. Visit 1 included the consent process, blood and urine sampling, taste perception testing, questionnaires, and instructions for at-home data collection. Participants collected data at home for approximately 11 to 14 days. At-home data collection included a 5-day nonconsecutive estimated food record, measurement of sleep and physical activity, and fecal sample collection. Visit 2 included delivery of all at-home collected data, body composition measurements by DXA, questionnaires, and the one-on-one food record interview.

Figure 3. Overview of the PRedictors linking Obesity and gut MIcrobiomE (PROMISE) study visits and schedule.



Blood Samples

Blood samples were obtained between 7:30 am and 9:00 am (after overnight fast, 10-15 hours), by an experienced phlebotomist. A tourniquet was applied moderately to the arm before venipuncture. Blood was drawn into 4 vacutainers (maximum total blood volume was 30 mL), to obtain serum and plasma for analysis of metabolic markers and endocrine regulators. Ethylenediaminetetraacetic acid (EDTA) 10 mL vacutainers (Becton Dickinson) were used for whole blood and stored immediately in a −80° C freezer before the remainder of the sample was placed on ice. The serum vacutainer was left to stand between 30 and 60 min at room temperature (18° C) to clot before centrifugation (10 mL, Becton Dickinson). For endocrine regulators, an additional plasma sample (2 mL Becton Dickinson vacutainer P800 EDTA, aprotinin, and dipeptidyl peptidase IV) was collected. The latter and all other vacutainers were placed on wet ice immediately after collection until centrifugation. Vacutainer tubes were centrifuged at 1500 g for 15 min at 4° C within 1 hour after blood samples were taken. Aliquots of plasma (23 aliquots, 120-500 µL) and serum (6 aliquots, 500-1300 µL) were transferred into prelabeled 1.5 mL microcentrifuge tubes (Eppendorf safe-lock polymerase chain reaction clean tubes) and cryovials (Cryo.S Greiner Bio-One, GmbH) and stored immediately at −80° C. Blood samples were analyzed for a range of biomarkers (eg, plasma glucose, insulin, glycated hemoglobin, lipids, and liver function tests), inflammation markers (eg, high-sensitivity C-reactive protein, interleukin 6 and tumour necrosis factor alpha), and endocrine regulators (eg, GLP-1, ghrelin, leptin, and adiponectin).

Urine Samples

After completion of blood collection, participants were required to collect a fasting, midstream urine sample. Research staff provided a urine container (BD Vacutainer Urine Collection Cup) labeled with their unique participant ID, cleansing wipes, and gloves. Once urine was collected, participants were asked to place their urine sample container on wet ice inside a clearly labeled polystyrene box before it was further processed by research staff. Midstream urine (14 mL) was pipetted into a prechilled 15 mL falcon tube. Urine samples were centrifuged at 1500 g for 15 min, at 4° C, to remove cellular particles and debris [39]. Ten aliquots of 1000 µL were transferred to each cryovial (Cryo.S, Greiner Bio-One, GmbH) and then transferred to −80° C storage for later metabolomics analysis.

Anthropometric Measurements

At visit 1, anthropometric measurements included stretched height and fasting weight measurements. All anthropometric measurements were conducted using the International Society for the Advancement of Kinanthropometry (ISAK) protocol [40]. All research staff conducting these measurements were level 1 ISAK trained. BMI was calculated using the Quetelet index (weight/height^2). At visit 2, waist and hip circumferences were measured with a Lufkin W600PM flexible steel tape with the participant in a relaxed standing position with their arms folded across their chest. Sagittal abdominal diameter was measured at the umbilicus level using the Holtain-Kahn Abdominal Caliper (Holtain Ltd) and assessed in a supine position. Bioelectrical impedance analysis (InBody230) was used to assess body composition at both visits 1 and 2. Body composition measurements were performed using dual-energy x-ray absorptiometry (DXA; Hologic QDR Discovery A, Hologic Inc with APEX V. 3.2 software) at visit 2 to accurately assess body composition profiles in terms of total and regional fat mass. Before DXA scanning, participants were asked to remove their jewelry and if they were pregnant or had a pacemaker or any metal implants. All staff who conducted DXA scanning procedures had Australian and NZ Bone Mineral Society clinical densitometry accreditation.

Blood Pressure

At visit 2, resting blood pressure was measured with an Omron digital blood pressure monitor (Omron HEM-907, Omron Healthcare Inc) using one of 2 arm cuff sizes (22-32 cm or 32-48 cm). In addition, a record was kept of each participant's pulse. Three measurements were taken consecutively at 1-min intervals. The mean of the second and third measurements was used to calculate systolic and diastolic blood pressure [41].

Dietary Intake and Behavior

Dietary intake was assessed for energy, macro- and micronutrient intakes, distribution of food intake throughout the day, and food choice. Our primary method for obtaining current dietary intake data was the gold-standard prospective, 5-day, food record [42-44]. The 5-day estimated food record included 2 weekend days and 3 weekdays. Estimated rather than weighed food records were used to improve adherence and to reduce participant burden [45,46]. Each participant received training for estimating and documenting portions, and every food record was reviewed by an NZ-registered dietitian before a one-on-one, in-depth discussion with the participant to clarify portions of foods consumed, cooking methods, brands of food products reported, and any other ambiguities. Visual portion book aids, household measures (eg, metric cups and spoons), and Web-based tools were used to confirm specific portion sizes and brands consumed, respectively. During these one-on-one interviews, standardized diet behavior-related questions were asked such as intentional meal skipping, snacking behaviors, and food preferences. These detailed sessions were critical to ensure accurate dietary data were captured [47].

It is often debated that all dietary assessment instruments are vulnerable to measurement errors, and significant improvements of dietary intake data quality can be achieved when different dietary intake recording methods are combined, especially combining food recording with food frequency questionnaires (FFQs) [42]. The validated semiquantitative New Zealand Women's Food Frequency Questionnaire (NZWFFQ) that was adapted from the FFQs used in the National Nutrition Survey NZ [43,44,48,49] was also completed by participants at visit 2. This covered dietary intake retrospectively across the last month, including the days that the 5-day food record was completed, to ensure that the range of actual and usual intakes were captured [42,50]. The 220-item NZWFFQ was administered using a Web-based questionnaire hosted on SurveyMonkey survey software, and live progress was monitored by research staff.

Nutrient analysis of the food record and FFQ data was performed using the Foodworks 9 (Xyris Software Pty Ltd) dietary analysis software, which uses FOODfiles 2016 (developed by the NZ Institute for Plant & Food Research and the NZ Ministry of Health) as a reference food composition table for analysis. In addition, the Xyris database AusFoods 2017 and AusBrands 2017, which are based on the Australian food composition databases AUSNUT 2011-13 (developed by Food Standards Australia New Zealand) were used. The data will be used to assess dietary adequacy in terms of energy and nutrient intakes using the current Australia/New Zealand nutrient reference values [51].

Participants completed the Dietary Diversity Questionnaire to assess food choices and dietary diversity, the Eating Attitudes Test (EAT-26) questionnaire to assess eating disorder risk, and the Three Factor Eating Questionnaire [52] to assess eating behavior in terms of cognitive dietary restraint (restraint), disinhibition of control (disinhibition), and susceptibility to hunger (hunger) by calculating scores for these dimensions and their subcategories.

Taste Perception

At visit 1, taste perception testing was conducted in a fasting state in individual testing booths at room temperature (20° C), before consuming breakfast. Participants were individually trained on the testing procedure and how to use general labeled magnitude scales [53,54] to rate the intensity and hedonic liking of sweet, bitter, and fat taste stimuli. Four distinct concentrations of glucose (30 g/L, 60 g/L, 120 g/L, and 240 g/L), quinine (0.008 g/L, 0.016 g/L, 0.03 g/L, and 0.06 g/L), and milk fat samples (3.3%, 11.8%, 20.3%, and 37.3% fat) were assessed using water as the control measure. Participants rated each solution by tasting the whole 10 mL of sample. They first rated the water stimuli (control, labeled *sweet*), followed by all sweet taste stimuli, before moving on to bitter taste stimuli and then milk fat stimuli. All stimuli were labeled with a 3-digit number, and the order was randomized within the tastant set. Participants had to rinse their mouth with water between samples. A ranking task was also administered for glucose, quinine, and milk fat samples to evaluate taste sensitivity [55], which was administered at the end of each tastant set. Ranking task stimuli were presented as a set of 4 distinct concentrations, labeled with unique 3-digit numbers to be ranked from the lowest to highest concentration (ie, 30 g/L, 60 g/L, 120 g/L, and 240 g/L).

Physical Activity and Sleep

To objectively measure sleep and physical activity, participants were fitted with 2 accelerometers during visit 1, 1 hip-mounted w-GT3X accelerometer (Actigraph), to measure physical activity, and a wrist-worn AW2 actiwatch (Phillips Respironics) to measure sleep. Participants were instructed to wear the accelerometers on their hip and nondominant wrist continuously (24-hour protocol) for the following 7 days, except while bathing or participating in water activities such as swimming. During this 7-day collection period, participants recorded in a provided diary the time they woke up each morning and went to sleep each night, as well as any intentional physical activity they engaged in [56]. At visit 2, participants also completed physical activity and sleep questionnaires, including the Recent Physical Activity Questionnaire [57], the Pittsburgh Sleep Quality Index [58], the Berlin questionnaire for sleep apnea [59], and their chronotype was assessed by the Munich Chronotype Questionnaire [60].

Fecal Samples

Fecal samples were collected by participants at home during the period between visits 1 and 2. At the end of visit 1, participants were briefed on how to collect fecal samples and were provided with a collection kit. This kit contained 2 prelabeled screw-top containers with a scoop in the lid

(LBS3805 25 mL, ThermoFisher NZ), 2 larger prelabeled plastic containers (LBS30130 130mL PP, ThermoFisher NZ), kidney dishes, gloves, zip-lock plastic bags, ice-sheets, chiller carry bag, and detailed written instructions. Participants were given an individualized schedule for food record and accelerometer data collection days, with the fecal sample needing to be collected after completion of the food record. Participants collected 2 separate, *walnut-sized* aliquots, from the same fecal sample and were asked to write down the time and date the aliquots were collected. The outer (larger) container was filled with 2 to 3 cm of cold water and the smaller container was placed inside the larger, to create a water jacket. Samples were then placed immediately in their household freezer (-20° C) and transported inside the chiller bag with ice packs when returning to the research unit at visit 2. Similar fecal sample collection methods have been utilized in a range of previous studies [61].

Gut Microbiome and Bioinformatics

DNA was extracted from fecal samples using methods described in the Human Microbiome Project [62]. In brief, microbial cells in fecal homogenates were physically disrupted by bead-beating, and then DNA was purified using a Mo Bio PowerSoil DNA isolation kit. The DNA was checked for quantity and quality using a combination of gel electrophoresis, nanodrop spectrometry, and Qubit fluorometry. The DNA was shotgun sequenced by NZ Genomics Ltd using Nextera library preparation and pools of 12 barcoded samples run per lane on an Illumina HiSeq 2500 instrument (Illumina).

Gut microbiome sequence data were analyzed using recognized computation (bioinformatics) tools [27]. These tools include the preparation of species-sampling curves that are the classic means of evaluating ecological richness (alpha diversity—biodiversity). As the genomes of all microbial species present in the microbiota were sequenced as small DNA fragments, both phylogenetic (describing what kinds of microbes are there and their relative abundances; MetaPhlAn2, QIIME v2) and functional (the biochemical capacity encoded in the metagenome; HUMAnN2) information are available [61].

Deprivation Index

Deprivation index was assessed in this study as a measurement of socioeconomic status. New Zealand Deprivation Index 2013 (NZDep2013) combines census data relating to home ownership, housing, qualifications, income, employment, access to transport, communications, and family structure [63]. NZDep2013 provides a deprivation score for each meshblock in NZ. Meshblocks are the smallest geographical area defined by Statistics NZ, with a population of around 60 to 110 people. NZDep2013 groups deprivation scores into deciles, where 1 represents the areas with the least deprived scores and 10 the areas with the most deprived scores. Therefore, a value of 10 indicates that a meshblock is in the most deprived 10% of areas in NZ.

Power Calculations

We have based our power calculation on previous studies involving metagenome-based measures of gene richness (gene counts) of fecal microbial communities [25,27] where individuals were categorized into clusters of high or low gene counts. Individuals in the low–gene count cluster may be at increased risk of progressing to obesity-associated comorbidities. On the basis of previous studies [27], we assumed the average gut microbiome complexity and gene richness in normal body fat profile women to equate to 640,000 genes. We then assumed a standard deviation of 350,000 in both normal and obese body fat profile women [25,27]. On the basis of these assumptions, we will have 76% power to detect a difference of 25% between both groups (68 per group). We will have 99% power to detect a difference of 40%, as previously observed in a European study [27]. We assumed that 33% of women have *abnormal* taste perception [28,29] or dietary intake [25], and 33% of women with *normal* taste perception and dietary intake have reduced gut microbiome complexity and functionality. On the basis of these assumptions, we will have 97% power to detect a 2-fold difference between both groups. We would have 83% power to detect the same difference if 25% of women with *normal* sweet and fat taste perception or dietary intake had reduced gut microbiome complexity. All power calculations are based on analyses for each ethnic group separately, as differences in associations between ethnic groups may exist.

Statistical Analyses

Descriptive statistical methods are used to summarize gut microbiome complexity and functionality; dietary intake and behavior; sweet, fat, and bitter taste perception; sleep, physical activity; and biomarkers. Differential gut microbiome complexity and gene richness are analyzed between obese body fat profile women and normal body fat profile women stratified by ethnicity using linear regression analyses. We also assess associations between gut microbiome complexity and functionality and biological and behavioral factors described above. Logistic regression analyses are used to compare reduced versus high gut microbiome complexity and gene richness, based on cut points employed in other studies. We will use multiple regression analysis to assess the independent effects of the biological and behavioral factors and perform stratified analyses to assess effect modification (or interactions). All analyses will be adjusted for potential confounders (socioeconomic position, age, etc).

Results

Recruitment of Pacific and New Zealand European Women

The order of recruitment completion of the main groups of study participants was as follows: (1) NZ European BMI 18.5 to 24.9 kg/m^2; (2) Pacific BMI greater than or equal to 30.0 kg/m^2; (3) NZ European BMI greater than or equal to 30.0 kg/m^2; and (4) Pacific BMI 18.5 to 24.9 kg/m^2. Although the criteria for recruitment of Pacific women generally required that both parents were of Pacific descent, 10% of the Pacific participants recruited for the PROMISE study were accepted if they had only one parent of Pacific descent but identified clearly as being primarily of Pacific ethnicity. The NZ European BMI greater than or equal to 30.0 kg/m^2 group and the Pacific

BMI 18.5 to 24.9 kg/m^2 group required additional advertising and specifically targeted recruitment strategies in comparison with the other 2 groups of women. The specific recruitment methods and the number of participants recruited through each approach are summarized in Table 1.

Researchers made a number of observations during the study visits that characterized some of the logistical challenges during the recruitment period of the study. For Pacific women, key motivations to participate in the study included, but were not limited to, personal contact with the study facilitators through the Pacific community, *Me'a'ofa* (gift or donation), and interest in the outcomes of the study. For NZ European women, key motivations to participate in the study included, but were not limited to, interest in individual measurements such as blood markers (eg, cholesterol) and body composition measurements (eg, DXA scan), interest in the gut microbiome, *Me'a'ofa* (gift or donation), and interest in the nutrition-related measurements in the study.

General Characteristics of the Predictors Linking Obesity and the Gut Microbiome Study

A total of 351 participants were eligible to participate in the study (Figure 2). An overview of basic phenotype characteristics of the PROMISE study participants is presented in Table 2. Although our main target was to recruit participants with a normal BMI (18.5-24.9 kg/m^2) and an obese BMI ($\geq$30.0 kg/m^2), we also recruited an additional 54 participants in the overweight BMI (25.0-29.9 kg/m^2) range. The overweight BMI groups were included in this study because, first, some participants had incorrectly assessed their own height and weight before they arrived at the human nutrition research unit, and second, to offset the enormous difficulties of recruiting normal BMI Pacific women.

Table 1. Recruitment methods, examples, and the number of participants recruited (feedback from enrolled participants only, N=351).

Recruitment method	Examples	Number recruited[a]	
		Pacific	New Zealand European
The Fono Primary Healthcare Service (West Auckland)	Pacific staff members recruited through their database and wider community. Transport to and from research clinic provided. Radio interview (Radio Samoa 1593 FM; Tongan segment discussing the *gut microbiome*)	102	N/A[b]
Participant word of mouth	University students, previous PROMISE[c] participants (interest increased with *Me'a'ofa* [gift or donation])	36	21
Community/University Facebook pages/ Special interest pages	Auckland (New Zealand) city central and surrounding suburbs, (approximately 38 Facebook pages; with repeat posts) and Pacific Heartbeat	4	52
Public figures Facebook pages[d]	Local nutritionists, sports celebrities	2	26
University/work email lists	Massey University (New Zealand), The University of Auckland (New Zealand), New Zealand Police	3	15
Hospital staff newsletter/magazine	Auckland hospital network and email lists	2	15
Job advertisement site or volunteer page	Job search website, volunteer to participate in research website, student job search website	5	5
One-on-one recruitment (handing out flyers)	University orientation week, early childhood centers, schools	1	9
Websites	University website article, The Fono Primary Healthcare Service	1	3
Magazine articles[e]	National magazines (ie, nutrition, current affairs, and lifestyle). Articles mentioned current study and provided contact details	0	9
Festivals	Local Pacific festivals (ie, Polyfest 2017, Pasifika 2017)	4	1
Posters[e]	Universities, hospitals, health clinics, libraries, cafes, leisure centers, public swimming pools, gyms, community boards, supermarket notice boards	0	11
Recruitment companies	Consumer paid market research databases to recruit Pacific women with a body mass index of 18.5-24.9 kg/m^2 only	13	N/A
Instagram	Instagram *story* on local nutritionists/public figure Instagram feeds	1	2
Local newspaper articles/website[e]	Local free newspapers	0	3
Internal database of contacts[e]	List of names recontacted who previously completed a similar trial [43] (note: filtered to contact difficult body mass index categories only)	0	3

Recruitment method	Examples	Number recruited[a]	
		Pacific	New Zealand European
Neighbourly, Twitter[e]	Local suburb page Web noticeboard, PROMISE study twitter account (@promise_study)	0	2

[a]Level of recruitment based on attendance at visit 1 of the PROMISE study.

[b]N/A: not applicable.

[c]PROMISE: PRedictors linking Obesity and the gut MIcrobiomE.

[d]Public figure pages used at the stage of recruitment where NZ European BMI ≤ 24.9 kg/m^2 category was full; eg, one local nutritionist Facebook post resulted in 221 completed screening questionnaires, which resulted in 17 NZ European BMI ≥ 30.0 kg/m^2 participants.

[e]Advertisement method successful across NZ European women only.

Table 2. General phenotype characteristics and New Zealand Deprivation Index 2013 of the PRedictors linking Obesity and the gut MIcrobiomE (PROMISE) study participants who completed the study protocol (N=304).

Variable	Pacific women (n=142)	New Zealand European women (n=162)
Normal (BMI[a] 18.5-24.9 kg/m^2)		
Participants, n	36	79
Age (years), mean (SD)	24 (6)	30 (6)[b,c]
BMI (kg/m^2), mean (SD)	23.0 (1.6)	22.0 (1.5)[b]
Waist circumference (cm), mean (SD)	74.3 (4.3)	72.5 (4.9)
Hip circumference (cm), mean (SD)	100.6 (5.6)	97.0 (5.9)[b]
WHR[d], mean (SD)	0.74 (0.03)	0.75 (0.05)
Systolic BP[e] (mmHg), mean (SD)	109.4 (9.88)	111.7 (10.31)
Diastolic BP (mmHg), mean (SD)	68.5 (5.86)	70.6 (8.86)
NZDep2013[f], mean (SD)	7 (3)	4 (3)[b]
Overweight (BMI range 25.0-29.9 kg/m^2)		
Participants, n	33	13
Age (years), mean (SD)	26 (7)	28 (8)
BMI (kg/m^2), mean (SD)	27.5 (1.6)	26.4 (1.8)
Waist circumference (cm), mean (SD)	83.1 (5.1)	79.6 (5.9)
Hip circumference (cm), mean (SD)	108.6 (4.8)	106.9 (5.8)
WHR, mean (SD)	0.77 (0.05)	0.75 (0.04)
Systolic BP (mmHg), mean (SD)	113.1 (7.27)	115.0 (6.88)
Diastolic BP (mmHg), mean (SD)	70.6 (7.83)	69.5 (3.90)
NZDep2013, mean (SD)	7 (3)	4 (2)[b]
Obese (BMI range ≥ 30.0 kg/m^2)		
Participants, n	73	70
Age (years), mean (SD)	25 (6)	34 (7)[b]
BMI (kg/m^2), mean (SD)	36.9 (5.4)	34.3 (3.0)[b]
Waist circumference (cm), mean (SD)	100.8 (11.4)	99.3 (8.9)
Hip circumference (cm), mean (SD)	123.7 (10.9)	121.6 (7.5)
WHR, mean (SD)	0.81 (0.06)	0.82 (0.06)
Systolic BP (mmHg), mean (SD)	119.6 (11.5)	121.9 (14.2)
Diastolic BP (mmHg), mean (SD)	78.9 (9.99)	81.1 (9.39)

Variable	Pacific women (n=142)	New Zealand European women (n=162)
NZDep2013, mean (SD)	8 (2)	5 (2)[b]

[a]BMI: body mass index.

[b]$P<.01$ was deemed statistically significant.

[c]Independent samples t tests were performed to determine differences between ethnicities.

[d]WHR: waist-to-hip ratio.

[e]BP: blood pressure.

[f]NZDep2013: NZ Deprivation Index 2013.

Discussion

Principal Findings

This paper reports the study protocol and the recruitment strategy of the PROMISE study, the details of the analytical procedures, study outcomes, and clinical and physiological measurements will be published elsewhere. The main objective of the PROMISE study is to characterize the gut microbiome in 2 population groups with markedly different metabolic disease risk (Pacific and European women) and different body fat profiles (normal and obese). The study describes the roles of taste perception, diet, sleep, and physical activity in women with different body fat profiles in modifying the gut microbiome and its impact on obesity and metabolic health. Only healthy participants were included in the study in accordance with strict inclusion and exclusion criteria. We established well-defined protocols of scheduled experimental conditions with standard operating procedures (SOPs) for all domains of the study. All participants followed the same SOPs according to specified timelines, and all specimen samples were treated identically. The main rationale for this approach was to collect high-quality data and to minimize variation related to data acquisition, data analysis, and sampling of biological specimens.

There is a dearth of data in populations at greatest risk of developing obesity. The PROMISE study will help to fill these gaps. Although a cross-sectional design will not infer causality [64], it is a highly efficient approach that will be able to identify distinct roles for diet, taste perception, sleep, and physical activity in modifying the gut microbiome and its impact on obesity and metabolic health. An additional strength of the PROMISE study is the recruitment of women from Pacific and European population groups. It allows us to assess potential differences and commonalities between population groups with markedly different metabolic disease risk profiles and will provide new insights and has the potential to contribute to novel hypotheses. A number of previous studies have presented convincing evidence that the gut microbiome may be a central modifiable link between diet and host and may be likely to offer new avenues to tackle obesity [22,25,26]. However, many important questions remain. First, how important is the ratio of the bacterial phyla *Firmicutes* to *Bacteroidetes*? Highly publicized studies reported low abundances of *Bacteroidetes* and higher abundances of *Firmicutes* as a characteristic of obesity [65]. However, later studies failed to find this association and, in general, meta-analyses show that most obesity-microbiome studies in humans to date have been underpowered to determine valid differences between groups [66]. In addition, there is increasing evidence that whole genome shotgun sequencing, as used in the PROMISE study, has multiple advantages compared with the 16S amplicon method used in many previous studies. The advantages of shotgun metagenomics sequencing include enhanced detection of bacterial species, increased detection of diversity, and increased prediction of the most dominant gene pathways that are present in the particular genes [67]. Furthermore, much of the previous work has been conducted in mice, and the few human studies available were small and need to be replicated in larger studies. Second, how strong is the association between low gene count clusters and obesity? Metagenomic measures of gene richness (gene counts) of gut microbial communities have categorized individuals into clusters of high or low gene count, but the exact nature of this relationship is not known [25,27]. Third, is there an association between relative abundances of bacterial species with obese or lean phenotypes in humans [68]? Fourth, is there an association between abundance of specific gut microbiota with dietary intake of particular food groups or dietary patterns [69,70]? Finally, do ethnic and/or cultural differences, sleep, and physical activity modify associations between the gut microbiome and obesity?

Given the cultural diversity of the participants, it was vital that research staff from the Pacific community were actively involved in performing the study to ensure the study was conducted in a culturally appropriate manner and to support the collection of quality data and successful outcomes [71]. Furthermore, there is convincing evidence that community-based participatory research approaches, involving community members and organizational representatives can overcome recruitment challenges and enhance the quality of a study [71-73]. The participation of a senior Pacific research nurse in the research team and the partnership with The Fono were invaluable in providing support and understanding from a participant perspective. In NZ, the essence of showing respect and kindness is described as *manaakitanga*, which encompasses hosting visitors with care, developing a nurturing relationship, and being a responsible host [74]. Adoption of *manaakitanga* within the framework of the PROMISE study, incorporating socially and emotionally grounded beliefs, enhanced participant engagement in what could commonly be perceived as a formal clinical research setting. Therefore, we recommend that future studies incorporate a range of strategies and culturally appropriate approaches to support community-based engagement throughout all aspects of the research [38].

Strengths and Limitations

A remarkable experience of the recruitment process was the success of using social media as a recruitment tool for NZ European women. Overall, we found that employing multiple recruitment methods, including social media (eg, community Facebook pages), newspaper advertisements, and business circulars (eg, work place email lists) gave this study a wider representation of the general population. It is important to keep in mind that although Auckland city has the highest population of resident Pacific peoples, the total number of Pacific women living in Auckland is much lower than that of NZ European women. In addition, many Pacific communities live in regions that are over 25 km away from the research unit such that transport issues were a significant barrier for some Pacific participants. Therefore, it was not surprising that Pacific women were more challenging to recruit than NZ European women. It has been recognized that barriers to participating in clinical research include fear and lack of trust in the study procedures or in research staff. In contrast, motivating factors include free health care access, feeling connected to the research outcomes that may support family or friends in the future (eg, developing treatments for specific diseases), and monetary incentives [72]. The PROMISE study team embraced these critical factors, paid attention to using appropriate language, and generated a culturally and gender-appropriate setting during all procedures (ie, DXA scanning) to contribute to a positive experience for participants [72].

The recruitment of participants faced a number of challenges. For example, women with a high BMI may have felt less motivated to take part in obesity-related research because of a concern of negative evaluation, as reported previously [75]. Furthermore, difficulties in recruiting Pacific women with a normal BMI in this study were because of their low number within the general Pacific population, only 8% of adult Pacific women have a BMI between 18.5 and 24.9 kg/m^2 [5,37]. To address these barriers, additional efforts were made to advertise and recruit Pacific women into the PROMISE study. Therefore, we extended the recruitment range, especially for Pacific women, to include the overweight BMI range (25.0-29.9 kg/m^2). Furthermore, we increased the *Me'a'ofa* and provided transport to and from the research unit to encourage participation and completion of the study protocol. The disadvantage of such tailored recruitment approaches is the risk of increasing selection bias. Although we are not able to quantify the potential selection bias because of the differences in the success rate of the range of recruitment methods presented in Table 1, we have made every effort to ensure participants are representative of each population group [76]. Furthermore, we tailored our advertising and recruitment strategy in a way that was most culturally appropriate for each population group; it ensured participant engagement and motivation, which is known to enhance data quality and study completion [38,77].

Challenges of a cross-sectional study design include the temporality of single assessments and the potential bidirectional nature of some associations. Furthermore, multiple comparisons and a large number of assessments and outcome variables and the potential for complex interactions may require further stratification. However, the study design and recruitment emphasis on obese *versus* normal BMI categories in the PROMISE study is an efficient approach to identify and contrast biological parameters that are associated with obesity-related metabolic disease risk. Most previous studies have focused on only 1 or 2 aspects that may influence the gut microbiome and obesity. The comprehensiveness of the PROMISE study design and our multidisciplinary approach are a particular strength. It will greatly advance our understanding of the etiology of obesity and will guide future longitudinal studies and interventions involving specific microbiota-based therapies, linking the outcomes of our study with strategies for the design of foods that offer metabolic health benefits through changes of the gut microbiome.

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Authors' Contributions

BB, RK, JD, and GT developed the study concept and design and wrote the grant application for funding support. RF, PG, TLS, SS, BL, and ATMG contributed to the study design. NB, MM, SK, JS, NR, BB, RK, and MR played key roles in participant recruitment, the organization of the study, and data acquisition. BB and SK drafted the manuscript, and all authors contributed to and accepted the final version.

Conflicts of Interest

None declared.

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Abbreviations

BMI: body mass index
BP: blood pressure
CVD: cardiovascular disease
DXA: dual-energy x-ray absorptiometry
EDTA: ethylenediaminetetraacetic acid
FFQ: food frequency questionnaire
GLP-1: glucagon-like peptide-1
ISAK: International Society for the Advancement of Kinanthropometry
NZ: New Zealand
NZDep2013: New Zealand Deprivation Index 2013
NZWFFQ: New Zealand Women's Food Frequency Questionnaire
PROMISE: PRedictors linking Obesity and gut MIcrobiomE
SOP: standard operating procedure
T2D: type 2 diabetes
WHR: waist-to-hip ratio

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PMID:

7.2. Ethics approval, participant information sheet and consent

7.2.1. Low-risk ethics approval (Dessert Taste study, Chapter III)

MASSEY UNIVERSITY
ALBANY

11 May 2015

Sophie Kindleysides
C/O School of Food and Nutrition
Albany Campus

Dear Sophie

Re: Dessert Taste Study: describing the ability to detect fat in food by taste, odour and mouth feel

Thank you for your Low Risk Notification which was received on 7 May 2015.

Your project has been recorded on the Low Risk Database which is reported in the Annual Report of the Massey University Human Ethics Committees.

You are reminded that staff researchers and supervisors are fully responsible for ensuring that the information in the low risk notification has met the requirements and guidelines for submission of a low risk notification.

The low risk notification for this project is valid for a maximum of three years.

Please notify me if situations subsequently occur which cause you to reconsider your initial ethical analysis that it is safe to proceed without approval by one of the University's Human Ethics Committees.

Please note that travel undertaken by students must be approved by the supervisor and the relevant Pro Vice-Chancellor and be in accordance with the Policy and Procedures for Course-Related Student Travel Overseas. In addition, the supervisor must advise the University's Insurance Officer.

A reminder to include the following statement on all public documents:

> *"This project has been evaluated by peer review and judged to be low risk. Consequently, it has not been reviewed by one of the University's Human Ethics Committees. The researcher(s) named above are responsible for the ethical conduct of this research.*
>
> *If you have any concerns about the conduct of this research that you wish to raise with someone other than the researcher(s), please contact Dr Brian Finch, Director (Research Ethics), telephone 06 356 9099, extn 86015, e-mail humanethics@massey.ac.nz".*

Please note that if a sponsoring organisation, funding authority or a journal in which you wish to publish requires evidence of committee approval (with an approval number), you will have to provide a full application to one of the University's Human Ethics Committees. You should also note that such an approval can only be provided prior to the commencement of the research.

Yours sincerely

B T Finch.

Brian T Finch (Dr)
Chair, Human Ethics Chairs' Committee and
Director (Research Ethics)

Cc Professor Bernhard Breier, Dr Kathryn Beck
Professor Matt Golding, Dr Daniel Walsh

School of Food and Nutrition
Albany Campus

Distinguished Professor Harjinder Singh
Head of school. School of Food and Nutrition

Turitea campus

7.2.2. Ethics approval (PROMISE study, Chapter IV and V)

Health and Disability Ethics Committees

Health and Disability Ethics Committees
Ministry of Health
Freyberg Building
20 Aitken Street
PO Box 5013
Wellington
6011

0800 4 ETHICS
hdecs@moh.govt.nz

30 March 2016

Professor Bernhard Breier
Private Bag 102904
North Shore Mail Centre
Auckland 0745

Dear Professor Breier

Re:	**Ethics ref:**	**16/STH/32**
	Study title:	The gut microbiome: a new pathway to obesity prevention and metabolic health

I am pleased to advise that this application has been *approved* by the Southern Health and Disability Ethics Committee. This decision was made through the HDEC-Expedited Review pathway.

Conditions of HDEC approval

HDEC approval for this study is subject to the following conditions being met prior to the commencement of the study in New Zealand. It is your responsibility, and that of the study's sponsor, to ensure that these conditions are met. No further review by the Southern Health and Disability Ethics Committee is required.

Standard conditions:

1. Before the study commences at *any* locality in New Zealand, all relevant regulatory approvals must be obtained.

2. Before the study commences at *a given* locality in New Zealand, it must be authorised by that locality in Online Forms. Locality authorisation confirms that the locality is suitable for the safe and effective conduct of the study, and that local research governance issues have been addressed.

Non-standard conditions:

- Please remove the words 'exciting' and 'greatly' from the opening paragraph.
- Replace the word 'encourage' with 'invite'.
- Data needs to be kept for 10years (says 5years on the consent form).
- Please add the study name to the footer and version number.
- The sections in the information sheet about breastfeeding are confusing – please re-word.
- Currently it is not clear whether participants could not be breastfeeding for longer than 6 months (i.e be breastfeeding but planning to stop within the next 6

months); whether they could not have breastfed for more than 6 months in total any sometime in the past; or whether they could not have breastfeed within the last 6 months.

Non-standard conditions must be completed before commencing your study. Non-standard conditions do not need to be submitted to or reviewed by HDEC before commencing your study.

If you would like an acknowledgement of completion of your non-standard conditions letter you may submit a post approval form amendment. Please clearly identify in the amendment that the changes relate to non-standard conditions and ensure that supporting documents (if requested) are tracked/highlighted with changes.

For information on non-standard conditions please see section 128 and 129 of the Standard Operating Procedures at http://ethics.health.govt.nz/home.

After HDEC review

Please refer to the *Standard Operating Procedures for Health and Disability Ethics Committees* (available on www.ethics.health.govt.nz) for HDEC requirements relating to amendments and other post-approval processes.

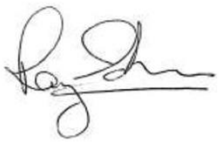
Your next progress report is due by 29 March 2017.

Participant access to ACC

The Southern Health and Disability Ethics Committee is satisfied that your study is not a clinical trial that is to be conducted principally for the benefit of the manufacturer or distributor of the medicine or item being trialled. Participants injured as a result of treatment received as part of your study may therefore be eligible for publicly-funded compensation through the Accident Compensation Corporation (ACC).

Please don't hesitate to contact the HDEC secretariat for further information. We wish you all the best for your study.

Yours sincerely,



Ms Raewyn Idoine
Chairperson
Southern Health and Disability Ethics Committee

Encl: appendix A: documents submitted
appendix B: statement of compliance and list of members

7.2.3. Participant information sheet and consent (Dessert Taste study, Chapter III)

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

DESSERT TASTE STUDY PARTICIPANT INFORMATION SHEET

Taste sensitivity to sweet and fatty flavour has been found to vary considerably between individuals. As variation in taste sensitivity influences food choice, there is much interest to understand the role of taste perception in the way people select food and how much they consume. The ability to identify food by smell may also be related to taste perception and therefore food choice. This project has been designed to better understand the relationship between taste, smell and mouth-feel and furthermore how this may influence diet.

Who can take part?

We are looking for women of:

- New Zealand European ethnicity
- 18-45 years of age
- Not be pregnant or breastfeeding
- Who are non-smokers
- Have had regular menstrual periods for a year
- Not have any chronic illnesses or clinical cause for a dry mouth

Project Procedures

The study requires you to attend **3 sessions** which are approximately 1 – 1.5 hours long at the sensory unit in Massey University, Albany. You will be required to come in for each session after an overnight fast and refrain from brushing your teeth for at least an hour prior to the appointment. These appointments will be conducted between 7.30-11am on weekdays and selected weekends. During your first visit we will obtain your height, weight and basic body composition scale measurement (BIA).

Sensory testing

Part 1: Taste

Taste testing involves tasting food samples to determine your sensitivity to taste. You will be presented with 3 milk based samples at a time. You will then be asked to take the whole cup of each sample (~10 mL), swirl it in your mouth for 3 seconds and then spit it out to a waste cup* (swallowing the sample may affect the results). Two of the three samples will be identical and one is different. You will pick the sample with the 'different' taste. You will be asked to return the tray with the empty cups. In-between evaluation sets you will rinse your mouth with distilled water and wait 20 seconds before you move to the next sample. **Please keep your nose clip on during tasting**.

*Please note: it is important that you spit out the contents of the tasting sample as ingesting all the samples during testing may lead to a laxative effect. One ingredient incorporated (mineral oil) has a maximum daily intake of 100 mg (recommended by the World Health Organisation (WHO)).

Part 2: Smell

Your sense of smell will be tested to determine your sensitivity to aroma. You will receive three samples and you will inhale each sample, and you will be asked to identify which of these is

'different'. You will be asked to return the tray with sniffer bottles or pens. After a short break you will receive more sniffer samples to evaluate.

Part 3: Custard test

Again, your sensitivity to taste will be measured through a short vanilla custard evaluation. You will be presented with 2 samples of vanilla custard. You will need to taste each of these, and in your own time, fill in the corresponding questionnaire.

Part 4: Dietary analysis

In between the study visits you will be required to fill in online questionnaires on dietary habits and eating behaviour. These questionnaires are online and may be filled in at home; however we may have a brief discussion both prior to and after receiving your questionnaire responses in order to ensure we have a genuine dietary evaluation.

What will you receive?

You will be reimbursed for travel expenses with a **$100 MTA petrol voucher** following the completion of testing (voucher received at the end of the third session). You will be given a breakfast after each sensory session. You will also receive a written report containing the main findings of the study once data analysis and interpretation is completed.

Confidentiality

All data collected will be used solely for research purposes and will be prepared for publication in a professional journal. All personal information will be kept confidential by assigning number codes to each participant. No names will be visible on any papers on which you provide information. If you are a student of one of the research teams please note that your academic grades will not be affected whether you decide to complete the study or withdraw at a later time. All data/information will be handled in confidence and will be stored in a secure location for five years on the Massey University Albany campus.

Participant's rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- Decline to answer any particular question;
- Withdraw from the study at any time;
- Ask any questions about the study at any time during participation;
- Provide information on the understanding that your name will not be used unless you give permission to the researcher

Contact information

If you have any further questions or concerns about the project, either now or in the future, please contact the dessert taste study team on desserttastestudy@gmail.com.
Specific contacts:

Professor Bernhard Breier b.breier@massey.ac.nz
Sophie Kindleysides (PhD student) s.j.kindleysides@massey.ac.nz

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

Institute of Food and Nutrition
Massey University
Private Bag 102-904
North Shore Mail Centre
Albany, Auckland
New Zealand
T 09 414 0800

DESSERT TASTE STUDY

Participant Consent Form

I have read the Information Sheet and have had the details of the study explained to me.

My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I understand that I will be trying **milk** and **vanilla custard** samples during this study. By signing this you are declaring that you are happy to taste a number of these samples during the visit.

I agree to participate in this study under the conditions set out in the Information Sheet.

Signature: ..... **Date:** .....

Full Name .....

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

DESSERT TASTE STUDY
Personal information, health and demographic questionnaire

First name: _____

Family name: _____

When did your last period start? (Day / month / year) _____

Do you follow a specific diet for health reasons? Yes □ No □

Please explain

_____

_____

_____

Do you follow a specific diet aimed at weight loss? Yes □ No □

Please explain

_____

_____

_____

Do you follow any diet for cultural or religious reasons? Yes □ No □

Please explain

_____

_____

Questions regarding physical activity

1. Does your job/occupation require any physical activity? If so, please describe:

   _____

   _____

2. Do you play a sport or partake in any other physical activity in addition to any previously described activities? Yes □ No □

   If yes, please describe the activity and how often you partake:

   _____

   _____

I would like to receive a brief report summarizing the main findings of the project:

Yes □ No □

I am willing to be contacted in future research projects within the Institute of Food and Nutrition:

Yes □ No □

7.2.4. Testing documents and SOP for the Dessert Taste Study (Chapter III)

Threshold testing (taste)

C18:1 conc (mM)	Oleic acid (µL)	Base solution (mL)	Mineral oil (mL)
.02	.56	100.0	5
.06	1.9	100.0	5
1	31.5	100.0	5
1.4	44.1	100.0	5
2	63.1	99.9	5
2.8	88.4	99.9	5
3.8	119.9	99.9	5
5	157.8	99.8	5
6.4	202	99.8	5
8	250	99.8	5
9.8	309	99.7	5
12	380	99.6	5
20	631.2	99.4	5

MILK BASE SOLUTION

-100g gum Arabic

-200 mg EDTA

Then add -

2 L non-fat UHT milk

homogenise => allocate 100mL to each of the 13 beakers with C18:1

To the remaining solution (blank) add 35mL mineral oil

Homogenise (do not contaminate threshold series, start with lowest and work up the dilution series)

Odour recognition – threshold series – Oleic acid in mineral oil

C18:1 concentration (mM)	Oleic acid added to mineral oil (g)	Oleic acid added to mineral oil (µL)	Mineral oil (mL)	OA%
6 mM	.01	10	5.0	0.2
12 mM	.02	19	5.0	0.5
24 mM	.03	38	5.0	0.7
48 mM	.07	75	4.9	1.7
95 mM	.13	150	4.9	3.1
190 mM	.27	300	4.7	6.4
380 mM	.54	600	4.4	12.7

Pipette mineral oil into labelled dropper bottles. All blanks to have 5mL mineral oil. Pipette oleic acid after mineral oil.

Custard – concentration series – Coconut oil in vanilla custard

CODE	Coconut oil concentration (%)	Coconut oil added to custard (g)	Vanilla custard (g)
584	0	0	50
634	5	2.5g	47.5
931	10	5g	45
184	15	7.5g	42.5

CUSTARD BASE RECIPE

3 tablespoons cornstarch
500mL UHT non-fat milk
3 tablespoons sugar
½ teaspoon vanilla essence
¼ teaspoon yellow food colouring

=stir well. Microwave for 4 minutes (4 minutes, stir). Microwave for additional 1 + 1 + 1 (ideally 8 minutes total) **but observe carefully and**

ensure does not bubble over.

Bowls labelled (5% CO, 10% CO and 15% CO).
Start with Coconut Oil bowl measure set while custard is hot (so coconut oil melts in). Tare scale to 0.
Spoon coconut oil into bowl.
When correct amount of CO is added to bowl do not tare. At this point add custard to make a total of 50g.
Continue with remaining samples.

Thoroughly mix and then tare portion cups. Add 20g into appropriately labelled custard cups.

Dessert taste study; Researcher questionnaire/checklist. Example:

Name: Date:

	DAY THREE		TASTE sample allocation			✓	x
A	level 1	**0.02mM**	718	**696**	211		
	level 2		**232**	807	682		
	level 3		186	**786**	502		
B	level 1	**0.06mM**	621	839	**017**		
	level 2		**311**	278	869		
	level 3		866	**961**	208		
C	level 1	**1mM**	408	**190**	367		
	level 2		699	**088**	224		
	level 3		**803**	108	916		
D	level 1	**1.4mM**	326	**773**	169		
	level 2		071	628	**382**		
	level 3		198	839	**930**		
E	level 1	**2mM**	**188**	874	992		
	level 2		687	**378**	764		
	level 3		133	289	**091**		
F	level 1	**2.8mM**	439	336	**190**		
	level 2		247	093	**376**		
	level 3		**640**	997	806		
G	level 1	**3.8mM**	972	617	**264**		
	level 2		804	247	**701**		
	level 3		782	**334**	631		
H	level 1	**5mM**	**298**	010	963		
	level 2		**143**	446	893		
	level 3		266	680	**750**		
I	level 1	**6.4mM**	618	240	**170**		
	level 2		429	**667**	883		
	level 3		322	836	**063**		

J	level 1	**8mM**	291	**764**	986		
	level 2		164	**869**	669		
	level 3		**614**	561	786		
K	level 1	**9.8mM**	**391**	268	668		
	level 2		**563**	202	129		
	level 3		644	017	**208**		
L	level 1	**12mM**	879	**284**	346		
	level 2		743	281	**664**		
	level 3		**271**	018	671		
M	level 1	**20mM**	**889**	967	373		
	level 2		609	**964**	409		
	level 3		579	029	**914**		

DAY THREE			**AROMA oleic C18:1 sample allocation**			✓	**x**
A	level 1	**6mM**	402	135	**670**		
	level 2		764	880	**097**		
	level 3		**175**	831	425		
B	level 1	**12mM**	**878**	418	739		
	level 2		350	**957**	843		
	level 3		521	**697**	801		
C	level 1	**24mM**	**751**	641	368		
	level 2		203	**389**	156		
	level 3		**616**	990	146		
D	level 1	**48mM**	**348**	488	677		
	level 2		833	105	**544**		
	level 3		952	**210**	023		
E	level 1	**95mM**	435	**223**	551		
	level 2		**092**	293	730		
	level 3		902	026	**645**		
F	level 1	**190mM**	013	866	**180**		
	level 2		163	715	**966**		

	level 3		774	**926**	637		
G	level 1	**380mM**	**708**	045	182		
	level 2		461	615	**281**		
	level 3		216	**622**	456		

ODD		**AROMA SNIFFIN STICKS sample**			✓	**x**
	15	769	**886**	992		
	13	408	081	**179**		
	11	806	**934**	791		
	9	**592**	172	683		
	7	**021**	440	271		
	5	811	**145**	302		
	3	**653**	723	584		
	1	102	**844**	376		

DAY THREE	**CUSTARD**			
	Hedonic taste 1			
	Hedonic taste 2			

7.2.5. Participant information and consent (PROMISE study, Chapter IV and V)

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE STUDY

(PRedictors linking Obesity and gut MIcrobiomE)

INFORMATION SHEET

We are looking for women aged between 20 and 40 years of age to take part in the PROMISE study. We want to understand how the bacteria in our gut are affected by diet, physical activity, sleep and taste perception, and how this is linked to health and a healthy body weight. Please read this information sheet carefully before deciding whether or not to participate.

What is this research about?

Here in New Zealand we are experiencing an epidemic of obesity and metabolic diseases, such as diabetes. A key contributing factor is our diets and the types of food we eat. Food has a large impact on the number and types of bacteria in our bowel (gut microbiome). There are at least 1000 different types of bacteria. It is thought that these bacteria play a very important role in our health, and may be contributing to obesity in some people. In this study we will investigate how diet, taste perception, sleep and physical activity affect the gut microbiome. This new knowledge will help us understand obesity and how best to prevent it.

Who are we looking for?

We are looking for women to participate in this study. To take part in this study you need to:

- Be between 20 and 40 years of age
- Be of NZ European ethnicity **OR** of Pacific ethnicity
- Not be pregnant or breastfeeding
- Not have any chronic diseases such as heart disease, diabetes or cancer
- Not have any food-allergies
- Not be following a severely restricted diet

What is involved in the study?

If you decide to take part in the study, you will be asked to complete a screening questionnaire (online or over the phone) that will assess your eligibility. If you meet these eligibility criteria you will be invited to take part in the study, which includes 2 visits to the Massey University Human Nutrition Research Unit in Albany and collection of data at home.

Visit 1 (approximately 2.5 hours)

You will have to visit the Human Nutrition Research Unit early in the morning (between 7.30 and 8:30am), without having eaten breakfast. The visit will take approximately 2.5 hours. On the day we will first explain to you what is involved in the study and answer all your questions. We will then ask you to sign a consent form.

The following procedures will take place during this visit:

- Your height, weight and body composition will be measured.
- A blood sample will be taken from a vein in your arm by a trained person (a phlebotomist). For this blood sample you need to have fasted overnight. **You should not eat or drink anything (other than water) from 10pm the previous evening until after the blood sample has been taken.** We will use the blood samples to measure cholesterol and glucose levels, markers of inflammation and small molecules related to fat usage and storage.
- You will be asked to taste a few sweet, fat and bitter solutions by sipping a mouthful (10mL or 2 teaspoons), swirling it in your mouth and swallowing it, and then rating your preference on a scale.
- A buffet-style continental breakfast will be provided prior to further measures to ensure that you break your fasted state.
- You will also be asked to complete some questionnaires regarding your diet and sleep patterns.
- We will ask you to provide a small urine sample.

At home between Visits 1 & Visit 2

You will be asked to:

- Wear an Accelerometer (a small device similar to a step-counter that you wear around your waist to measure physical activity), and an Actiwatch (a watch worn on your wrist which records your sleep patterns) for 7 days. The accelerometer and watch will be provided by the researchers and you will receive detailed instructions on how to use them.
- Record your physical activity for 7 days while you are wearing the small devices above.
- Record your food intake for 5 days while you are wearing the small devices above.
- Provide two small faecal (stool) samples and bring these to Visit 2. It is important that these two small stool samples are frozen (in the containers provided by us) immediately after collection in your freezer. These samples allow us to determine the bacteria (microbes) in your gut.

Visit 2 (approximately 2 hours)

You will be asked to visit the Human Nutrition Research Unit during the day. For this visit we ask that you eat as usual before coming as no food will be provided during Visit 2. The visit will take about 2 hours.

At this visit:

- You will bring back all your devices and the stool samples and your physical activity and food records;
- An in-depth interview will be conducted regarding your data collection tasks including devices and food recording by the research staff.
- Your weight and height will be measured.
- Your blood pressure will be measured.
- You will undergo a full body composition assessment including waist, hip and abdominal height measurements as well as a whole body scan using a DXA machine. These measurements will take place in a private, enclosed room.
- You will also be asked to complete some questionnaires regarding your diet and activity patterns.

DXA machine

What are the benefits and risks of taking part in this study?

There will be no charges made for any of the tests that you undertake. You will receive information regarding your own individual measurements (blood results including blood sugar, cholesterol, blood pressure, % body fat and BMI) and an explanation of the data. In recognition of your participation, you will receive a $100 supermarket or petrol voucher at the end of visit 2. You will also receive a brief report summarising the main findings of the project via mail or e-mail after analysis of the data has been completed. This is also a chance for you to receive some information about your current health status. In addition, this information will help us to provide better recommendations for preventing obesity.

Some people may have a fear of having a blood sample taken or experience discomfort when the blood samples are taken. Occasionally a slight bruise will result. We will take every measure to ensure that you are comfortable and respected. You may also be accompanied by a support person if required.

With the DXA body composition scan you will be exposed to a very low dose of radiation, unlikely to cause any harm. The total dose of radiation will be less than the amount of radiation you are exposed to during a trans-Tasman flight.

Project procedures

Sample Handling and Storage

Samples will be stored in a secure laboratory freezer at the Human Nutrition Research Unit until completion of the study – for a maximum of 10 years. Samples will be analysed by fully-accredited laboratories, either in NZ or overseas, depending on the type of analysis being done. The data will be used only for the purposes of this project and no individual will be identified. Only the investigators and administrators of the study will have access to personal information and this will be kept secure and strictly confidential. Participants will be identified only by a study identification number to ensure anonymity and confidentiality of these samples. Following analysis samples will be destroyed following usual procedures, however, these blood samples can be returned to you upon request.

Data Handling

Results of this project may be published or presented at conferences or seminars. No individual will be able to be identified. At the end of this study the list of participants and their study identification number will be disposed of. Any raw data on which the results of the project depend will be retained in secure storage for 10 years, after which it will be destroyed.

Genetic analysis

Each person has a DNA make-up (their genes) which is different from that of everybody else – except in the case of identical twins. As part of this study we will be performing some DNA-based measures on blood samples that you provide us with. This will allow us to investigate possible links between the gut microbiome and genetic makeup, in particular, links that may relate to diet, body composition, sleep patterns, taste perception and physical activities. This may further our understanding of specific risk factors for obesity. The genetic or DNA-based measures that we will be performing will be research-based and will not have any medical significance for you. Therefore you will not receive the results of these genetic measures as any information gathered will be purely theoretical. As with all other aspects of the study, ID numbers will be used so that your samples are not identifiable and all information will be kept confidential and will only be used for the specific purposes of this study.

Consenting to allow DNA-based measures to be performed on your samples is entirely optional. If you do not wish to participate in this aspect of the research please indicate this on your consent form. You may withdraw your consent to the use of your sample in this research at any time and your samples will be destroyed.

Who is funding the research?

This research is funded by the Health Research Council (HRC) of New Zealand (Grant #15/273).

Participant's rights

You are under no obligation to accept this invitation. If you decide to participate, you have the right to:

- decline to answer any particular question;
- withdraw from the study at any time;
- ask any questions about the study at any time during participation;
- provide information on the understanding that your name will not be used unless you give permission to the researcher;
- be given access to a summary of the project findings when it is concluded.

Project contacts

If you have any further questions or concerns about the project, either now or in the future, please contact the research team on 09 212 7013 or 021 082 74425 or e-mail promise@massey.ac.nz or website www.massey.ac.nz/promise.

The lead researchers for this study are Professor Bernhard Breier and Associate Professor Rozanne Kruger. If you have any concerns please contact Bernhard [b.breier@massey.ac.nz, (09) 213 6652] or Rozanne [r.kruger@massey.ac.nz, (09) 213 6661]

Committee approval statement

This project has been reviewed and approved by the Health and Disability Ethics Committee, Southern Region, Ethics Reference 16/STH/32. If you have any concerns about the conduct of this research, please contact the Southern Health and Disability Ethics Committee on 0800 438442 or hdecs@moh.govt.nz

Compensation for injury

If physical injury results from your participation in this study, you should visit a treatment provider to make a claim to ACC as soon as possible. ACC cover and entitlements are not automatic and your claim will be assessed by ACC in accordance with the Accident Compensation Act 2001.

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE Study

Consent Form for Participants

I have read the Information Sheet* and have had the details of the study explained to me. My questions have been answered to my satisfaction, and I understand that I may ask further questions at any time.

I agree to participate in this study under the conditions set out in the Information Sheet*. This consent form will be held for a period of ten (10) years.

☐ Yes ☐ No

DNA analysis will be performed on parts of the blood samples obtained. Do you consent to parts of your samples being used for DNA analysis?*

☐ Yes ☐ No

The findings of this study may be the basis of further investigations by our team. Would you be happy for us to contact you in future about research related to this study?

☐ Yes ☐ No

*Please see the study Information Sheet for more details

Signature: ..... **Date:** .....

Full Name (printed) .....

Massey Institute of Food Science and Technology – School of Food and Nutrition
Private Bag 102904, North Shore, Auckland 0745, New Zealand **T** +64 9 213 6653 http://sfn.massey.ac.nz

7.2.6. Testing documents and SOP for the PROMISE study (Chapter IV and V)

(A) Glucose sample preparation for intensity and hedonic testing

1. Filtered, bottle water will be required to create glucose/ sweet taste samples
2. Add 57.7g glucose to 200 mL of filtered water. Add the water slowly and dissolve/stir (do not add all the water at once).

    - Formula weight Glucose (Food Grade, Sherratt Ingredients, NZ): 180.16 g/mol
    - Stock molarity required: 1,335 mM
    - Desired final volume of stock: 240 mL
    - Mass of glucose required: 57.7g glucose powder

3. To create serial dilution obtain 100 mL from the stock solution (1,335 mM stock) and add to a new beaker. To this beaker add 100 mL of water.

Molarity	Glucose solution required	Water required
1,335 mM	57.7 g glucose	200ml (final volume =240mL)
668 mM	100 ml from 1,335 mM	100 ml
334 mM	100ml from 668 mM	100ml
167 mM	100ml from 334 mM	100ml

Participants will evaluate 10 mL of each concentration (whole mouth sip-and-spit) and evaluate the intensity and liking for each on a line scale questionnaire. 80 mL of sweet taste samples in total (concentration evaluation and ranking task).

Concentration testing 3-digit codes

Sweet C1	167 mM	**753**
Sweet C2	334 mM	**329**
Sweet C3	668 mM	**018**
Sweet C4	1,335 mM	**837**

Ranking task testing 3-digit codes

Sweet R1	167 mM	**247**
Sweet R2	334 mM	**962**
Sweet R3	668 mM	**443**
Sweet R4	1,335 mM	**516**

Quinine sample preparation for intensity and hedonic testing

1. Filtered, bottle water will be required to create quinine/ bitter taste samples

A super concentrated stock needs to be made in advance. Measure 7g of quinine, add water and dissolve to a total of 220mL. This makes 80mM solution. - Formula weight Quinine hydrochloride dehydrate (Food Grade, Sigma Aldrich): 396.91 g/mol

2. Add 0.4 mL of 80 mM quinine concentrated stock to 200 mL of filtered water (creates a 0.16mM stock).

3. To create serial dilution obtain 100 mL from the stock solution (0.16mM stock) and add to a new beaker. To this beaker add 100 mL of water.

Concentration	Quinine solution required	Water required
0.16mM (0.06 g/L)	0.4 mL high conc stock	200ml
0.08mM (0.03 g/L)	100 ml from stock (0.16mM)	100 ml
0.04mM (0.016 g/L)	100ml from 0.08mM	100ml
0.02mM (0.008 g/L)	100ml from 0.04mM	100ml

Tonic water has 83mg quinine per litre (that is, 0.083 g/L)

Participants will evaluate 10 mL of each concentration (whole mouth sip-and-spit) and evaluate the intensity and liking for each on a line scale questionnaire. 80 ml of bitter taste samples in total (concentration evaluation and ranking task).

Concentration testing 3-digit codes

Bitter C1	0.02 mM	**653**
Bitter C2	0.04 mM	**139**
Bitter C3	0.08 mM	**912**
Bitter C4	0.16 mM	**524**

Bitter R1	0.02 mM	**890**
Bitter R2	0.04 mM	**268**
Bitter R3	0.08 mM	**450**
Bitter R4	0.16 mM	**717**

Fat taste preparation for intensity and hedonic testing

Anchor Blue top milk:	3.3g total fat
Anchor Blue top milk + cream [MIXED]: [ratio 75:25]	11.8g total fat
Anchor Blue top milk + cream [MIXED]: [ratio 50:50]	20.3g total fat
Anchor Cream:	37.3g total fat

Participants will evaluate 10 mL of each concentration (whole mouth sip-and-spit) and evaluate the intensity and liking for each on a line scale questionnaire. 80 ml of milk taste samples in total (concentration evaluation and ranking task).

10 mL of each sample will be pipetted into a portion cup directly. The exception to this is the MIXED samples, where 5 mL of Anchor Blue top milk and 5 mL of Anchor cream will be pipetted separately, then the 10 mL sample will be drawn back and forth into the pipette tip 5 times in order to homogenise the solution thoroughly.

Concentration testing 3-digit codes

Milk C1	3.3% fat	**455**
Milk C2	11.8% fat	**370**
Milk C3	20.3% fat	**284**
Milk C4	37.3% fat	**078**

Milk R1	3.3% fat	**113**
Milk R2	11.8% fat	**609**
Milk R3	20.3% fat	**761**
Milk R4	37.3% fat	**022**

Testing procedure

- Participants will be individually briefed on testing procedure and informed of the 3 key ingredients (milk, quinine and glucose). They will be instructed to spit out all tasting samples
- TOTAL SENSORY TESTING TIME: Less than 30 minutes
- Samples will be accompanied by water and crackers (palate cleansers)
- Samples will be tested one at a time in a randomised order. For the ranking task 4 samples will be given and each tasted and ordered by perceived concentration
- Sweet samples will be tested first, followed by bitter and then milk taste

Protocol for when testing more than 5 participants (max. 9 participants)

SWEET TASTE

Add **115.4g** glucose to 400 mL of filtered water. Add the water slowly and dissolve/stir (do not add all the water at once). Final volume of stock will be 480mL.

To create serial dilution obtain 200 mL from the stock solution (1,335 mM stock) and add to a new beaker. To this beaker add 200 mL of water.

Molarity	Glucose solution required	Water required
1,335 mM	115.4 g glucose	400ml (final volume =480mL)
668 mM	200 ml from 1,335 mM	200 ml
334 mM	200ml from 668 mM	200ml
167 mM	200ml from 334 mM	200ml

BITTER TASTE

Add 0.8 mL of 80 mM quinine from high concentrated stock solution to 400 mL of filtered water (creates 0.16 mM stock).

To create serial dilution obtain 200 mL from the stock solution (0.16mM stock) and add to a new beaker. To this beaker add 200 mL of water.

Concentration	Quinine solution required	Water required
0.16mM (0.06 g/L)	0.8 mL high conc stock	400ml
0.08mM (0.03 g/L)	200 ml from stock (0.16mM)	200 ml
0.04mM (0.016 g/L)	200ml from 0.08mM	200ml
0.02mM (0.008 g/L)	200ml from 0.04mM	200ml

7.3. Questionnaire templates

7.3.1. Food frequency questionnaire (Dessert Taste study, Chapter III)

1. Please read carefully before you begin:

Please make sure when filling out this questionnaire that you:

• Tell us what YOU usually eat (not someone else in your household!).
• Fill in the form YOURSELF.
• Are correct, but don't spend too much time on each food.
• Answer EVERY question; the asterisk symbol (*) at the beginning of each question means that you must answer before moving onto the next question.

This will help us to get the most accurate information about your usual food intake.

Please answer by ticking the box which best describes HOW OFTEN you ate or drank a particular food or drink in the LAST MONTH and HOW MUCH you would usually have.

For example:

1. EXAMPLE: How often do you usually have sugar? (Please do not fill out)

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Sugar - 1 tsp	○	○	○	○	○	○	○	○	○

If every day you have 2 cups of coffee with 1 tsp sugar, 4 cups of tea with 1 tsp sugar, one bowl of cereal with 1 tsp sugar and sugar on pancakes at dinner, you would choose four or more times per day = '4+ x / day'.

Adjust your portion size and frequency of intake to suit your eating habits.

2. EXAMPLE: How often do you usually eat bread? (Please do not fill out)

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Bread - 1 slice	○	○	○	○	○	○	○	○	○

If every day you have two slices of toast for breakfast, and you have a sandwich for lunch three times per week, you would choose two - three times per day = '2-3x / day'.

Adjust your portion size and frequency of intake to suit your eating habits.

1

3. Eating Pattern

* 1. How would you describe your eating pattern? (Please choose one only)

- ◯ Eat a variety of all foods, including animal products
- ◯ Eat eggs, dairy products, fish and chicken but avoid other meats
- ◯ Eat eggs, dairy products and fish, but avoid chicken and other red meats
- ◯ Eat eggs and dairy products, but avoid all meats, chicken and fish
- ◯ Eat eggs, but avoid dairy products, all meats and fish
- ◯ Eat dairy products, but avoid eggs, all meats and fish
- ◯ Eat no animal products
- ◯ None of the above

Other (please state)

3

4. Dairy

* 1. Do you use milk? (e.g. fresh, UHT, powdered)

- ◯ Yes
- ◯ No

* 2. What type(s) of milk do you have most often? (You can choose up to 3 options, but please only choose the ones you usually have)

- ☐ Not applicable
- ☐ Full cream milk (purple top)
- ☐ Standard milk (blue top)
- ☐ Skim milk (light blue top)
- ☐ Trim milk (green top)
- ☐ Super trim milk (light green top)
- ☐ Calcium enriched milk (yellow top) e.g. Xtra, Calci-Trim
- ☐ Calcium and vitamin enriched milk e.g. Mega, Anlene
- ☐ Calcium and protein enriched milk e.g. Sun Latte
- ☐ Standard soy milk (blue)
- ☐ Light soy milk (light blue)
- ☐ Calcium enriched soy milk (purple) e.g. Calci-Forte, Calci-Plus
- ☐ Calcium, vitamin and omega 3 enriched soy milk e.g. Essential
- ☐ Calcium and high fibre enriched soy milk e.g. Calci-Plus High Fibre
- ☐ Rice milk

Other (please state)

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* 3. Choose the one milk you have the most

- [ ] Not applicable
- [ ] Full cream milk (purple top)
- [ ] Standard milk (blue top)
- [ ] Skim milk (light blue)
- [ ] Trim milk (green top)
- [ ] Super trim milk (light green top)
- [ ] Calcium enriched milk (yellow top) e.g. Xtra, Calci-Trim
- [ ] Calcium and vitamin enriched milk e.g. Mega, Anlene
- [ ] Calcium and protein enriched milk e.g. Sun Latte
- [ ] Standard soy milk (blue)
- [ ] Light soy milk (light blue)
- [ ] Calcium enriched soy milk (purple) e.g. Calci-Forte, Calci-Plus
- [ ] Calcium, vitamin and omega 3 enriched soy milk e.g. Essential
- [ ] Calcium and high fibre enriched soy milk e.g. Calci-Plus High Fibre
- [ ] Rice milk

Other (please state)

* 4. On average, how many servings of milk do you have per day? (Please choose one only)
(A 'serving' = 250 mL or 1 cup/glass)
e.g. 5 cups of coffee/tea using 50 mL of milk + ½ cup of milk on cereal = 1 ½ servings per day

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1-2 servings
- ◯ 3-4 servings
- ◯ 5 or more servings

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* 5. How often do you usually have milk?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Flavoured milk (milkshake, iced coffee, Primo, Nesquik) - 250 mL/ 1 cup	○	○	○	○	○	○	○	○	○
Milk as a drink - 250 mL / 1 cup	○	○	○	○	○	○	○	○	○
Milk on breakfast cereals or porridge - 125 mL/ 1/2 cup	○	○	○	○	○	○	○	○	○
Milk added to water-based hot drinks (coffee, tea) - 50 mL / 1/5 cup	○	○	○	○	○	○	○	○	○
Milk-based hot drinks (Latte, Milo) - 250 mL / 1 cup	○	○	○	○	○	○	○	○	○

* 6. How often do you usually eat cheese?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day	4+ x / day
Cheddar (tasty, mild, colby) - 2 heaped Tbsp / matchbox cube	○	○	○	○	○	○	○	○	○	○
Edam, Gouda, Swiss - 2 heaped Tbsp / matchbox cube	○	○	○	○	○	○	○	○	○	○
Feta, Mozarella, Camembert - 1 heaped Tbsp / 1 med wedge	○	○	○	○	○	○	○	○	○	○
Brie, blue and other specialty cheese - 1 heaped Tbsp / 1 med wedge	○	○	○	○	○	○	○	○	○	○
Processed cheese slices - 1 slice	○	○	○	○	○	○	○	○	○	○
Cream cheese - 2 heaped Tbsp	○	○	○	○	○	○	○	○	○	○
Cottage or ricotta cheese - 2 heaped Tbsp	○	○	○	○	○	○	○	○	○	○

* 7. How often do you usually eat these dairy based foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Ice cream - 2 scoops	○	○	○	○	○	○	○	○	○
Custard or dairy food - 1 pottle / ½ cup	○	○	○	○	○	○	○	○	○
Yoghurt, plain or flavour - 1 pottle / ½ cup	○	○	○	○	○	○	○	○	○
Milk puddings (semolina, instant) - ½ cup	○	○	○	○	○	○	○	○	○
Fermented or evaporated milk (buttermilk) - ½ cup	○	○	○	○	○	○	○	○	○

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5. Bread

* 1. Do you eat bread?

- ◯ No
- ◯ Yes

* 2. What type(s) of bread, rolls or toast do you eat most often? (You can choose up to 3 options, but please only choose the ones you usually have)

- ☐ Not applicable
- ☐ White
- ☐ White – high fibre
- ☐ Wholemeal or wheat meal
- ☐ Wholegrain

Other (please state)

* 3. What type of bread slice do you usually have? (Please choose one only)

- ◯ Not applicable
- ◯ Sandwich slice
- ◯ Toast slice
- ◯ Mixture of both sandwich and toast slices

* 4. On average, how many servings of bread do eat per day? (Please choose one only)
(A 'serving' = 1 slice of bread or 1 small roll)

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1–2 servings
- ◯ 3–4 servings
- ◯ 5–6 servings
- ◯ 7 or more servings

* 5. How often do you usually eat these bread based foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Plain white bread - 1 slice	○	○	○	○	○	○	○	○	○
High fibre white bread - 1 slice	○	○	○	○	○	○	○	○	○
Wholemeal or wheat meal - 1 slice	○	○	○	○	○	○	○	○	○
Wholegrain bread - 1 slice	○	○	○	○	○	○	○	○	○
Fruit bread or fruit bun - 1 slice	○	○	○	○	○	○	○	○	○
Wrap - 1 medium	○	○	○	○	○	○	○	○	○
Focaccia, bagel, pita, panini or other speciality breads - 1 medium	○	○	○	○	○	○	○	○	○
Paraoa Parai (fry bread) - 1 slice	○	○	○	○	○	○	○	○	○
Rewena bread - 1 slice	○	○	○	○	○	○	○	○	○
Doughboys or Maori bread - 1 slice	○	○	○	○	○	○	○	○	○

* 6. How often do you usually eat these other bread based foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Crumpet or muffin split - 1 crumpet / 1 whole muffin split	○	○	○	○	○	○	○	○	○
Scone - 1 medium	○	○	○	○	○	○	○	○	○
Bran muffin or savoury muffin - 1 medium	○	○	○	○	○	○	○	○	○
Croissant - 1 medium	○	○	○	○	○	○	○	○	○
Waffle, pancakes or pikelets - 1 medium / 2 small	○	○	○	○	○	○	○	○	○
Iced buns - 1 medium	○	○	○	○	○	○	○	○	○
Crackers (cream crackers, cruskits, corn / rice crackers, vitawheat) - 2 medium	○	○	○	○	○	○	○	○	○

* 7. Do you have butter, margarine or spreads on bread or crackers?

- ○ No
- ○ Yes

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* 8. What type(s) do you have most often? (You can choose up to 3 options, but please only choose the ones you usually have)

- [ ] Not applicable
- [ ] Butter (all varieties)
- [ ] Monounsaturated fat margarine e.g. Olive, Rice Bran, Canola Oil Spreads
- [ ] Polyunsaturated fat margarine e.g. Sunflower Oil Spreads
- [ ] Light monounsaturated fat margarine e.g. Olivio Spread Light
- [ ] Light polyunsaturated fat margarine e.g. Flora Spread Light
- [ ] Plant sterol enriched margarine e.g. Pro Active, Logical Spreads
- [ ] Light plant sterol enriched margarine e.g. Pro Active Spread Light
- [ ] Butter and margarine blend e.g. Country Soft, Butter Lea

Other (please state)

* 9. Choose the one you have the most

- [ ] Not applicable
- [ ] Butter (all varieties)
- [ ] Monounsaturated fat margarine e.g. Olive, Rice Bran, Canola Oil Spreads
- [ ] Polyunsaturated fat margarine e.g. Sunflower Oil Spreads
- [ ] Light monounsaturated fat margarine e.g. Olivio Spread Light
- [ ] Light polyunsaturated fat margarine e.g. Flora Spread Light
- [ ] Plant sterol enriched margarine e.g. Pro Active, Logical Spreads
- [ ] Light plant sterol enriched margarine e.g. Pro Active Spread Light
- [ ] Butter and margarine blend e.g. Country Soft, Butter Lea
- [ ] Other (please state)

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* 10. On average, how many servings of butter, margarine or spreads do you have per day? (Please choose one only)
(A 'serving' = 1 level teaspoon or 5 mL)
e.g. 1 sandwich with butter thinly spread on two pieces of bread = 2 servings

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1–2 servings
- ◯ 3–4 servings
- ◯ 5–6 servings
- ◯ 7 or more servings

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6. Breakfast Cereals and Porridge

* 1. Do you usually eat breakfast cereal and/or porridge?

- ◯ No
- ◯ Yes

* 2. What breakfast cereal(s) do you eat most often? (You can choose up to 3 options, but please only choose the ones you usually have)

- ☐ Not applicable
- ☐ Weetbix
- ☐ Refined cereals e.g. Cornflakes or Rice Bubbles
- ☐ Bran based cereals including fruity varieties e.g. Special K, Muesli, All Bran
- ☐ Sweetened e.g. Nutrigrain, Cocoa Pops
- ☐ Porridge

Other (please state)

* 3. On average, how many servings of breakfast cereal or porridge do you have per week? (Please choose one only)
(A 'serving' = ½ cup porridge, muesli, cornflakes or 2 weetbix)
e.g. ½ cup of porridge 3 times per week + 2 weetbix 4 times a week = 7 servings per week

- ◯ Not applicable
- ◯ Less than 4 servings
- ◯ 4–6 servings
- ◯ 7–9 servings
- ◯ 10–12 servings
- ◯ 13–15 servings
- ◯ 16 or more servings

* 4. How often do you usually eat porridge or these cereal foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Porridge, rolled oats, oat bran, oat meal - ½ cup	○	○	○	○	○	○	○	○	○
Muesli (all varieties) - ½ cup	○	○	○	○	○	○	○	○	○
Weetbix (all varieties) - 2 weetbix	○	○	○	○	○	○	○	○	○
Cornflakes or rice bubbles - ½ cup	○	○	○	○	○	○	○	○	○
Bran cereals (All Bran, Bran Flakes) - ½ cup	○	○	○	○	○	○	○	○	○
Bran based cereals (Sultana Bran, Sultana Bran Extra) - ½ cup	○	○	○	○	○	○	○	○	○
Light and fruity cereals (Special K, Light and Tasty) - ½ cup	○	○	○	○	○	○	○	○	○
Chocolate based cereals (Milo cereal, Coco Pops) - ½ cup	○	○	○	○	○	○	○	○	○
Sweetened cereals (Nutrigrain, Fruit Loops, Honey Puffs, Frosties) - ½ cup	○	○	○	○	○	○	○	○	○
Breakfast drinks (Up and Go) - Small carton / 250 mL	○	○	○	○	○	○	○	○	○

7. Starchy Foods

* 1. Do you eat any type of starchy foods such as rice, pasta, noodles and couscous?

- ◯ No
- ◯ Yes

* 2. On average, how many servings of starchy foods such as rice, pasta, noodles and couscous do you eat per week? (Please choose one only)
(A 'serving' = 1 cup cooked rice / pasta)
e.g. 1 cup of rice + ½ cup of pasta included in a lasagne pasta dish + 1 cup of spaghetti = 2.5 servings

- ◯ Not applicable
- ◯ Less than 4 servings
- ◯ 4–6 servings
- ◯ 7–9 servings
- ◯ 10–12 servings
- ◯ 13–15 servings
- ◯ 16 or more servings

* 3. How often do you usually eat these starchy foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Rice, white - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Rice, brown or wild - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Pasta, white or wholegrain (spaghetti, vermicelli) - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Canned spaghetti (Watties) - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Instant noodles (2 minute noodles) - 1 packet	◯	◯	◯	◯	◯	◯	◯	◯	◯
Egg and rice noodles (hokkien noodles, udon) - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Other grain (quinoa, couscous, bulgar wheat) - 1 cup	◯	◯	◯	◯	◯	◯	◯	◯	◯

8. Meat

* 1. Do you eat beef, mutton, hogget, lamb, or pork

- ◯ No
- ◯ Yes

* 2. Do you trim any excess fat (fat you can see) off these meats? (Please choose one only)

- ◯ Not applicable
- ◯ Always
- ◯ Often
- ◯ Occasionally
- ◯ Never cut the fat off meat

* 3. On average, how many servings of meat e.g. beef, mutton, hogget, lamb or pork do you eat per week? (Please choose one only)
(A 'serving' = palm size or ½ a cup of meat without bone)
e.g. ½ cup of savoury mince + 2 small lamb chops = 2 servings

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1-3 servings
- ◯ 4-6 servings
- ◯ 7 or more servings

* 4. How often do you usually eat meat?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Beef mince dishes (rissoles, meatloaf, hamburger pattie) - 1 slice / patty / ½ cup	○	○	○	○	○	○	○	○	○
Beef or veal mixed dishes (casserole, stir-fry) - ½ cup	○	○	○	○	○	○	○	○	○
Beef or veal (roast, chop, steak, schnitzel, corned beef) - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Lamb, hogget or mutton mixed dishes (stews, casserole, stir-fry) - ½ cup	○	○	○	○	○	○	○	○	○
Lamb, hogget or mutton (roast, chops, steak) - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Pork (roast, chop, steak) - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Canned corned beef - 1 medium slice	○	○	○	○	○	○	○	○	○

* 5. How often do you usually eat these other meats?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Sausage, frankfurter or saveloy - 1 sausage / frankfurter/ 2 saveloys	○	○	○	○	○	○	○	○	○
Bacon - 2 rashers	○	○	○	○	○	○	○	○	○
Ham - 1 medium slice	○	○	○	○	○	○	○	○	○
Luncheon meats or brawn - 1 slice	○	○	○	○	○	○	○	○	○
Salami or chorizo - 1 slice / cube	○	○	○	○	○	○	○	○	○
Offal (liver, kidneys, pate) - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Venison/game - palm size / ½ cup	○	○	○	○	○	○	○	○	○

9. Poultry

* 1. Do you eat poultry e.g. chicken, turkey or duck?

- ◯ No
- ◯ Yes

* 2. Do you remove the skin from chicken? (Please choose one only)

- ◯ Not applicable
- ◯ Always
- ◯ Often
- ◯ Occasionally
- ◯ Never remove the skin from chicken

* 3. On average, how many servings of chicken do you eat per week? (Please choose one only)
(A 'serving' = palm size of chicken or ½ cup)
e.g. 1 chicken breast + 2 chicken drumsticks + 1 chicken thigh = 4 servings per week

- ◯ Not applicable
- ◯ Less than1 serving
- ◯ 1-3 servings
- ◯ 4-6 servings
- ◯ 7 or more servings

* 4. How often do you usually eat poultry?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Chicken legs or wings - palm size / ½ cup / 1 unit (wing, drumstick)	○	○	○	○	○	○	○	○	○
Chicken breast - palm size / ½ cup / ½ breast	○	○	○	○	○	○	○	○	○
Chicken mixed dishes (casserole, stir-fry) - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Crumbed chicken (nuggets, patties, schnitzel) - 1 medium / 4 nuggets	○	○	○	○	○	○	○	○	○
Turkey or quail - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Mutton bird or duck - palm size / ½ cup	○	○	○	○	○	○	○	○	○

10. Fish and Seafood

* 1. Do you eat any type of fish or seafood?

- ◯ No
- ◯ Yes

* 2. On average, how many servings of fish and seafood (all types; fresh, frozen, tinned) do you eat per week? (Please choose one only)
(A 'serving' = 80 - 120g or palm size or small tin (85g))
e.g. 1 fish fillet and 1 small tin of tuna = 2 servings per week.

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1-3 servings
- ◯ 4-6 servings
- ◯ 7 or more servings

* 3. How do you normally cook / eat fish? (You can choose up to 3 options, but please only choose the ones you usually have)

- ☐ Not applicable
- ☐ Raw / I don't cook it
- ☐ Oven baked / Grilled
- ☐ Deep fried
- ☐ Shallow fry
- ☐ Micro waved
- ☐ Steamed
- ☐ Poached
- ☐ Smoked

* 4. How often do you usually eat seafood?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Canned Salmon - 1 small can (85-95g)	○	○	○	○	○	○	○	○	○
Canned Tuna - 1 small can (85-95g)	○	○	○	○	○	○	○	○	○
Canned Mackerel, sardines, anchovies, herring - 1 small can (85-95g)	○	○	○	○	○	○	○	○	○
Frozen crumbed fish (patties, fillets, cakes, fingers, nuggets) - 1 medium / 4 nuggets	○	○	○	○	○	○	○	○	○
Snapper, Tarakihi, Hoki, Cod, Flounder - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Gurnard, Kahawai or Trevally - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Lemon fish or Shark - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Tuna - palm size / ½ cup	○	○	○	○	○	○	○	○	○
Salmon, trout or eel - palm size / ½ cup	○	○	○	○	○	○	○	○	○

* 5. How often do you usually eat seafood?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Shrimp, prawn, lobster or crayfish - ½ cup	○	○	○	○	○	○	○	○	○
Crab or surumi - ½ cup	○	○	○	○	○	○	○	○	○
Scallops, mussels, oysters, paua or clams - ½ cup	○	○	○	○	○	○	○	○	○
Pipi or cockle - ½ cup	○	○	○	○	○	○	○	○	○
Kina - ½ cup	○	○	○	○	○	○	○	○	○
Whitebait - ¼ cup	○	○	○	○	○	○	○	○	○
Roe - ¼ cup	○	○	○	○	○	○	○	○	○
Squid, octopus, calamari, cuttlefish - ½ cup	○	○	○	○	○	○	○	○	○

11. Fats and Oils

* 1. Do you cook meat, chicken, fish, eggs and/or vegetables with fat or oil?

- ○ No
- ○ Yes

* 2. What type(s) do you use most often? (You can choose up to 3 options, but please only choose the ones you usually have)

- ☐ Not applicable
- ☐ Butter (all varieties)
- ☐ Margarines (all varieties)
- ☐ Cooking oils (all varieties)
- ☐ Lard, Dripping, Coconut oil, Ghee (clarified butter)
- ☐ Cooking spray

Other (please state)

* 3. Chose the one you use the most

- ☐ Not applicable
- ☐ Butter (all varieties)
- ☐ Margarines (all varieties)
- ☐ Cooking oils (all varieties)
- ☐ Lard, Dripping, Coconut oil, Ghee (clarified butter)
- ☐ Cooking spray
- ☐ Other (please state)

* 4. When you use fat or oil to cook, how many servings of fat or oil do you use per dish? (Please choose one only)
(A 'serving' = 1 level teaspoon or 5 mL)

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1 serving
- ◯ 2 servings
- ◯ 3 servings
- ◯ 4 servings
- ◯ 5 or more servings

* 5. On average, how many servings of fat or oil do you use to cook per week? (Please choose one only)

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1-3 servings
- ◯ 4-7 servings
- ◯ 8-10 servings
- ◯ 11-14 servings
- ◯ 15 or more servings

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12. Eggs

* 1. Do you eat eggs?

- ◯ No
- ◯ Yes

* 2. On average, not counting eggs used in baking / cooking, how many eggs do you usually eat per week? (Please choose one only)

- ◯ Not applicable
- ◯ Less than 1 egg
- ◯ 1 egg
- ◯ 2 eggs
- ◯ 3 eggs
- ◯ 4 eggs
- ◯ 5 or more eggs

* 3. How often do you usually eat eggs?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Whole eggs (hard-boiled, poached, fried, mashed, omelette, scrambled) - 1 egg	◯	◯	◯	◯	◯	◯	◯	◯	◯
Mixed egg dish (quiche, frittata, other baked egg) - 1 slice	◯	◯	◯	◯	◯	◯	◯	◯	◯

13. Legumes

* 1. Do you eat legumes e.g. chickpeas/dried peas, soybeans, dried/canned beans, baked beans, lentils or Dahl?

- ◯ No
- ◯ Yes

* 2. On average, how many servings of legumes (fresh, frozen, canned, dried) do you eat per week? (Please choose one only)
(A 'serving' = ½ cup or 125g of cooked legumes)

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1 serving
- ◯ 2 servings
- ◯ 3 servings
- ◯ 4-5 servings
- ◯ 6-7 servings
- ◯ 8 or more servings

* 3. How often do you usually eat these legumes?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Soybeans - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Tofu - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Dahl - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Canned or dried legumes, beans (baked beans, chickpeas, lentils, peas, beans) - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Hummus - 2 Tbsp	◯	◯	◯	◯	◯	◯	◯	◯	◯

14. Vegetables

* 1. Do you eat vegetables?

- ◯ No
- ◯ Yes

* 2. On average, how many servings of vegetables (fresh, frozen, canned) do you eat per day? Do NOT include vegetable juices. (Please choose one only)
(A 'serving' = 1 medium potato / kumara or ½ cup cooked vegetables or 1/2 cup of lettuce)
e.g. 2 medium potatoes + ½ cup of peas = 3 servings

- ◯ Not applicable
- ◯ Less than 1 serving
- ◯ 1 serving
- ◯ 2 servings
- ◯ 3 servings
- ◯ 4 or more servings

* 3. How often do you usually eat these vegetables?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Potato (boiled, mashed, baked, roasted) - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Pumpkin (boiled, mashed, baked, roasted) - ½ cup	○	○	○	○	○	○	○	○	○
Kumara (boiled, mashed, baked, roasted) - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Mixed frozen vegetables - ½ cup	○	○	○	○	○	○	○	○	○
Green beans - ½ cup	○	○	○	○	○	○	○	○	○
Silver beet, spinach - ½ cup	○	○	○	○	○	○	○	○	○
Carrots - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Sweet corn - 1 medium cob / ½ cup	○	○	○	○	○	○	○	○	○
Mushrooms - ½ cup	○	○	○	○	○	○	○	○	○
Tomatoes - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Beetroot - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Taro, cassava or breadfruit - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○

* 4. How often do you usually eat these vegetables?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Green bananas (plantain) - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Sprouts (alfalfa, mung) - ½ cup	○	○	○	○	○	○	○	○	○
Pacific Island yams - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Turnips, swedes, parsnip or yams - ½ cup	○	○	○	○	○	○	○	○	○
Onions, celery or leeks - ¼ cup	○	○	○	○	○	○	○	○	○
Cauliflower, broccoli or broccoflower - ½ cup	○	○	○	○	○	○	○	○	○
Brussel sprouts, cabbage, red cabbage or kale - ½ cup	○	○	○	○	○	○	○	○	○
Courgette/zucchini, marrow, eggplant, squash, kamo kamo, asparagus, cucumber - ½ cup	○	○	○	○	○	○	○	○	○
Capsicum (peppers) - ½ medium / ¼ cup	○	○	○	○	○	○	○	○	○
Avocado - ¼ avocado	○	○	○	○	○	○	○	○	○
Lettuce greens (mesculin, cos, iceberg) - ½ cup	○	○	○	○	○	○	○	○	○
Other green leafy vegetables (whitloof, watercress, taro leaves, puha) - ½ cup	○	○	○	○	○	○	○	○	○

15. Fruit

* 1. Do you eat fruit?

- ◯ No
- ◯ Yes

* 2. On average, how many servings of fruit (fresh, frozen, canned or stewed) do you eat per day? Do NOT include fruit juice. (Please choose one only)
(A 'serving' = 1 medium or 2 small pieces of fruit or 1/2 cup of chopped fruit)
e.g. 1 apple + 2 small apricots = 2 servings)

- ◯ Not applicable
- ◯ Less than one serving
- ◯ 1 serving
- ◯ 2 servings
- ◯ 3 or more servings

* 3. How often do you usually eat these fruits?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Apple - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Pear - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Banana - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Orange, mandarin, tangelo, grapefruit - 1 medium / 2 small	○	○	○	○	○	○	○	○	○
Peach, nectarine, plum or apricot - 1 medium / ½ cup / 2 small	○	○	○	○	○	○	○	○	○
Mango, paw-paw or persimmons / ½ cup	○	○	○	○	○	○	○	○	○
Pineapple - ½ cup	○	○	○	○	○	○	○	○	○
Grapes - ½ cup / 8-10 grapes	○	○	○	○	○	○	○	○	○
Strawberries, other berries, cherries - ½ cup	○	○	○	○	○	○	○	○	○
Melon (watermelon, rockmelon) - ½ cup	○	○	○	○	○	○	○	○	○
Kiwifruit - 1 medium / 2 small	○	○	○	○	○	○	○	○	○
Feijoas - 1 medium / 2 small	○	○	○	○	○	○	○	○	○
Tamarillos - 1 medium / ½ cup	○	○	○	○	○	○	○	○	○
Sultanas, raisins or currants - 1 small box	○	○	○	○	○	○	○	○	○
Other dried fruit (apricots, prunes, dates) - 4 pieces	○	○	○	○	○	○	○	○	○

16. Drinks

* 1. On average, how many drinks do you have per day? (Please choose one only)
(A 'serving' = 250 mL or one cup/glass)

- ◯ Less than 1 serving
- ◯ 1-3 servings
- ◯ 4-5 servings
- ◯ 6-8 servings
- ◯ 9-10 servings
- ◯ 11 or more servings

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* 2. How often do you usually have these drinks?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Instant soup (Cup of soup) - 250 mL / 1 cup	○	○	○	○	○	○	○	○	○
Fruit juice (Just Juice, Fresh-up, Charlie's, Rio Gold) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Fruit drink (Choice, Rio Splice) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Vegetable juice (tomato juice, V8 juice) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Iced Tea (Lipton ice tea) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Cordial or Powdered drinks (Thriftee, Raro, Vita-fresh) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Low-calorie cordial - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Energy drinks small-medium can (V, Red Bull) - 250-350 mL	○	○	○	○	○	○	○	○	○
Energy drinks large can (Monster, Mother, Demon, large V) - 450-550 mL	○	○	○	○	○	○	○	○	○
Sugar-free Energy drinks (sugar-free V, Monster, Red Bull) - 1 small can	○	○	○	○	○	○	○	○	○
Diet soft/fizzy/carbonated drink (diet sprite) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Soft/fizzy/carbonated drinks (Coke, Sprite) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○
Sport's drinks (Gatorade, Powerade) - 1 bottle	○	○	○	○	○	○	○	○	○
Flavoured water (Mizone, H2Go flavoured) - 1 bottle	○	○	○	○	○	○	○	○	○
Water (unflavoured mineral water, soda water, tap water) - 250 mL / 1 cup/glass	○	○	○	○	○	○	○	○	○

* 3. How often do you usually have these drinks?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Coffee instant or brewed with or without milk (Nescafe, expresso) - 1 cup	○	○	○	○	○	○	○	○	○
Specialty coffees (flat white, cappuccino, lattes) - 1 small cup	○	○	○	○	○	○	○	○	○
Coffee decaffeinated or substitute (Inka) - 1 cup	○	○	○	○	○	○	○	○	○
Hot chocolate drinks (drinking chocolate, hot chocolate, Koko) - 1 cup	○	○	○	○	○	○	○	○	○
Milo - 1 tsp	○	○	○	○	○	○	○	○	○
Tea (English breakfast tea, Earl Grey) - 1 cup	○	○	○	○	○	○	○	○	○
Herbal tea or Green tea - 1 cup	○	○	○	○	○	○	○	○	○
Soy drinks - 1 cup	○	○	○	○	○	○	○	○	○

* 4. How often do you usually have these alcoholic drinks?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Beer – low alcohol - 1 can or bottle	○	○	○	○	○	○	○	○	○
Beer – ordinary - 1 can or bottle	○	○	○	○	○	○	○	○	○
Red wine - 1 small glass	○	○	○	○	○	○	○	○	○
White wine, champagne, sparkling wine - 1 small glass	○	○	○	○	○	○	○	○	○
Wine cooler - 1 small glass / bottle	○	○	○	○	○	○	○	○	○
Sparkling grape juice - 1 glass / cup	○	○	○	○	○	○	○	○	○
Sherry or port - 100 mL	○	○	○	○	○	○	○	○	○
Spirits, liqueurs - 1 shot or 30 mL	○	○	○	○	○	○	○	○	○
RTD (KGB, Vodka Cruiser, Woodstock bourbon) - 1 bottle / can	○	○	○	○	○	○	○	○	○
Cider - 1 glass / cup / bottle	○	○	○	○	○	○	○	○	○
Kava - 1 glass / cup	○	○	○	○	○	○	○	○	○

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17. Dressings and Sauces

* 1. How often do you usually have these dressings or sauces?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Butter (all varieties) - 1 tsp	○	○	○	○	○	○	○	○	○
Margarine (all varieties) - 1 tsp	○	○	○	○	○	○	○	○	○
Oil (all varieties) - 1 tsp	○	○	○	○	○	○	○	○	○
Cream or sour cream - 1 Tbsp	○	○	○	○	○	○	○	○	○
Mayonnaise or creamy dressings (aioli, tartae sauce) - 1 Tbsp	○	○	○	○	○	○	○	○	○
Low fat/calorie dressing (reduced fat mayonnaise) - 1 Tbsp	○	○	○	○	○	○	○	○	○
Salad dressing (french, italian) - 1 Tbsp	○	○	○	○	○	○	○	○	○
Sauces (tomato, BBQ, sweet chilli, mint) - 1 Tbsp	○	○	○	○	○	○	○	○	○
Mustard - 1 Tbsp	○	○	○	○	○	○	○	○	○
Soy sauce - 1 Tbsp	○	○	○	○	○	○	○	○	○
Chutney or relish - 1 Tbsp	○	○	○	○	○	○	○	○	○
Gravy homemade - ¼ cup	○	○	○	○	○	○	○	○	○
Instant Gravy (e.g. Maggi) - ¼ cup	○	○	○	○	○	○	○	○	○
White sauce/cheese sauce - ¼ cup	○	○	○	○	○	○	○	○	○

18. Miscellaneous - Cakes, Biscuits and Puddings

* 1. How often do you usually eat these baked products?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Cakes, loaves, sweet muffins - 1 slice / 1 muffin	◯	◯	◯	◯	◯	◯	◯	◯	◯
Sweet pies or pastries, tarts, doughnuts - 1 medium	◯	◯	◯	◯	◯	◯	◯	◯	◯
Other puddings or desserts - not including milk-based puddings (sticky date pudding, pavlova) - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Plain biscuits, cookies (Round wine, Ginger nut) - 2 biscuits	◯	◯	◯	◯	◯	◯	◯	◯	◯
Fancy biscuits (chocolate, cream) - 2 biscuits	◯	◯	◯	◯	◯	◯	◯	◯	◯

19. Miscellaneous

* 1. How often do you usually eat these other foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Jelly - ½ cup	○	○	○	○	○	○	○	○	○
Ice blocks - 1 ice block	○	○	○	○	○	○	○	○	○
Lollies - 2 lollies	○	○	○	○	○	○	○	○	○
Chocolate - including chocolate bars (Moro bars) - 1 small bar	○	○	○	○	○	○	○	○	○
Sugar added to food and drinks - 1 level tsp	○	○	○	○	○	○	○	○	○
Jam, honey, marmalade or syrup - 1 level tsp	○	○	○	○	○	○	○	○	○
Vegemite or marmite - 1 level tsp	○	○	○	○	○	○	○	○	○
Peanut butter or other nut spreads - 1 level Tbsp	○	○	○	○	○	○	○	○	○
Brazil nuts or walnuts - 2	○	○	○	○	○	○	○	○	○
Peanuts - 10	○	○	○	○	○	○	○	○	○
Other nuts (almonds, cashew, pistachio, macadamia) - 10	○	○	○	○	○	○	○	○	○
Seeds (pumpkin, sunflower)	○	○	○	○	○	○	○	○	○
Muesli bars - 1 bar	○	○	○	○	○	○	○	○	○
Coconut cream - ¼ cup	○	○	○	○	○	○	○	○	○
Coconut milk - ¼ cup	○	○	○	○	○	○	○	○	○
Lite coconut milk - ¼ cup	○	○	○	○	○	○	○	○	○
Potato crisps, corn chips, Twisties - ½ cup / handful	○	○	○	○	○	○	○	○	○

* 2. Do you use salt in cooking?

- ○ Never
- ○ Rarely
- ○ Sometimes
- ○ Usually
- ○ Always

* 3. Do you use salt at the table?

- ◯ Never
- ◯ Rarely
- ◯ Sometimes
- ◯ Usually
- ◯ Always

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20. Miscellaneous - Takeaways

* 1. On average, how often do you eat takeaways per week? (Please choose one only)

- ◯ Never
- ◯ Less than 1 times
- ◯ 1-2 times
- ◯ 3-4 times
- ◯ 4-6 times
- ◯ More than 7 times

* 2. How often do you usually eat these takeaway foods?

	Never	<1x / month	1-3x / month	1x / week	2-3x / week	4-6x / week	Once / day	2-3x / day	4+ x / day
Meat pie, sausage roll, other savouries - 1 pie / 2 small sausage rolls or savouries	◯	◯	◯	◯	◯	◯	◯	◯	◯
Hot potato chips, kumara chips, french fries, wedges - ½ cup	◯	◯	◯	◯	◯	◯	◯	◯	◯
Chinese - 1 serve	◯	◯	◯	◯	◯	◯	◯	◯	◯
Indian - 1 serve	◯	◯	◯	◯	◯	◯	◯	◯	◯
Thai - 1 serve	◯	◯	◯	◯	◯	◯	◯	◯	◯
Pizza - 1 medium slice	◯	◯	◯	◯	◯	◯	◯	◯	◯
Burgers - 1 medium burger	◯	◯	◯	◯	◯	◯	◯	◯	◯
Battered fish - 1 piece	◯	◯	◯	◯	◯	◯	◯	◯	◯
Fried chicken (KFC, Country fried chicken) - 1 medium piece	◯	◯	◯	◯	◯	◯	◯	◯	◯
Bread based (Kebab, sandwiches, wraps, Pita Pit, Subway) - 1 medium	◯	◯	◯	◯	◯	◯	◯	◯	◯

21. Other

* 1. Are there any other foods or drinks that you can think of that you have on a regular basis that was not covered by this questionnaire?

- ◯ No
- ◯ Yes

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22. Other

1. Please list these foods and drinks including; the serving size, and how many times per week you eat or drink these items (e.g. Pizza, 4 slices, one time per week)

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7.3.2. Three Factor Eating Questionnaire (TFEQ)

PROMISE Three Factor Eating Questionnaire

PROMISE Three Factor Eating Questionnaire

Please answer each question by choosing the the appropriate answer (True or False)

2. 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

- ◯ True
- ◯ False

3. I usually eat too much at social occasions, like parties and picnics

- ◯ True
- ◯ False

4. I am usually so hungry that I eat more than three times a day

- ◯ True
- ◯ False

5. When I have eaten my quota of calories, I am usually good about not eating any more

- ◯ True
- ◯ False

6. Dieting is so hard for me because I just get too hungry

- ◯ True
- ◯ False

7. I deliberately take small helpings as a means of controlling my weight

- ◯ True
- ◯ False

8. Sometimes things just taste so good that I keep on eating even when I am no longer hungry

- ◯ True
- ◯ False

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9. Since I am often hungry, I sometimes wish that while eating, an expert would tell me that I have had enough or that I can have something more to eat

- ◯ True
- ◯ False

10. When I feel anxious, I find myself eating

- ◯ True
- ◯ False

11. Life is too short to worry about dieting

- ◯ True
- ◯ False

12. Since my weight goes up and down, I have gone on reducing diets more than once

- ◯ True
- ◯ False

13. I often feel so hungry that I just have to eat someting

- ◯ True
- ◯ False

14. When I am with someone who is overeating, I usually overeat too

- ◯ True
- ◯ False

15. I have a pretty good idea of the number of calories in common food

- ◯ True
- ◯ False

16. Sometimes when I start eating, I just can't seem to stop

- ◯ True
- ◯ False

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17. It is not difficult for me to leave something on my plate

- ◯ True
- ◯ False

18. At certain times of the day, I get hungry because I have gotten used to eating someting then

- ◯ True
- ◯ False

19. 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

- ◯ True
- ◯ False

20. Being with someone who is eating often makes me hungry enough to eat also

- ◯ True
- ◯ False

21. When I feel blue, I often overeat

- ◯ True
- ◯ False

22. I enjoy eating too much to spoil it by counting calories or watching my weight

- ◯ True
- ◯ False

23. When I see a real delicacy, I often get so hungry that I have to eat right away

- ◯ True
- ◯ False

24. I often stop eating when I am not really full as a conscious means of limiting the amount that I eat

- ◯ True
- ◯ False

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25. I get so hungry that my stomach often seems like a bottomless pit

- ◯ True
- ◯ False

26. My weight has hardly changed at all in the last ten years

- ◯ True
- ◯ False

27. I am always hungry so it is hard for me to stop eating before I finish the food on my plate

- ◯ True
- ◯ False

28. When I feel lonely, I console myself by eating

- ◯ True
- ◯ False

29. I consciously hold back at meals in order not to gain weight

- ◯ True
- ◯ False

30. I sometimes get very hungry late in the evening or at night

- ◯ True
- ◯ False

31. I eat anything I want, any time I want

- ◯ True
- ◯ False

32. Without even thinking about it, I take a long time to eat

- ◯ True
- ◯ False

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33. I count calories as a conscious means of controlling my weight

- ◯ True
- ◯ False

34. I do not eat some foods because they make me fat

- ◯ True
- ◯ False

35. I am always hungry enough to eat at any time

- ◯ True
- ◯ False

36. I pay a great deal of attention to changes in my figure

- ◯ True
- ◯ False

37. While on a diet, if I eat a food that is not allowed, I often then splurge and eat other high calorie foods

- ◯ True
- ◯ False

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PROMISE Three Factor Eating Questionnaire

PROMISE Three Factor Eating Questionnaire

Please answer the following questions by choosing the response that is appropriate to you.

38. How often are you dieting in a conscious effort to control your weight?

- ◯ Rarely
- ◯ Sometimes
- ◯ Usually
- ◯ Always

39. Would a weight fluctuation of 2.5 kg (5 lbs) affect the way you live your life?

- ◯ Not at all
- ◯ Slightly
- ◯ Moderately
- ◯ Very much

40. How often do you feel hungry?

- ◯ Only at mealtimes
- ◯ Sometimes between meals
- ◯ Often between meals
- ◯ Almost always

41. Do your feelings of guilt about overeating help you to control your food intake?

- ◯ Never
- ◯ Rarely
- ◯ Often
- ◯ Always

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42. How difficult would it be for you to stop eating halfway through dinner and not eat for the next four hours?

- ◯ Easy
- ◯ Slightly difficult
- ◯ Moderately difficult
- ◯ Very difficult

43. How conscious are you of what you are eating?

- ◯ Not at all
- ◯ Slightly
- ◯ Moderately
- ◯ Exremely

44. How frequently do you avoid 'stocking up' on tempting foods?

- ◯ Almost never
- ◯ Seldom
- ◯ Usually
- ◯ Almost always

45. How likely are you to shop for low calorie foods?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very likely

46. Do you eat sensibly in front of others and splurge alone?

- ◯ Never
- ◯ Rarely
- ◯ Often
- ◯ Always

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47. How likely are you to consciously eat slowly in order to cut down on how much you eat?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very Likely

48. How frequently do you skip dessert because you are no longer hungry?

- ◯ Almost never
- ◯ Seldom
- ◯ At least once a week
- ◯ Almost every day

49. How likely are you to consciously eat less than you want?

- ◯ Unlikely
- ◯ Slightly likely
- ◯ Moderately likely
- ◯ Very likely

50. Do you go on eating binges though you are not hungry?

- ◯ Never
- ◯ Rarely
- ◯ Sometimes
- ◯ At least once a week

51. On a scale of 0 to 5, where 0 means no restraint in eating (eating whatever you want, whenever you want it) and 5 means total restraint (constantly limiting food intake and never 'giving in'), what number would you give yourself?. Choose the answer which best describes you.

- ◯ 0. Eat whatever you want, whenever you want it
- ◯ 1. Usually eat whatever you want, whenever you want it
- ◯ 2. Often eat whatever you want, whenever you want it
- ◯ 3. Often limit food intake, but often 'give in'
- ◯ 4. Usually limit food intake, rarely 'give in'
- ◯ 5. Constantly limiting food intake, never 'giving in'

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52. To what extent does this statement describe your eating behaviour ?
'I start dieting in the morning, but because of any number of things that happen during the day, by evening I have given up and eat what I want, promising myself to start dieting again tomorrow.'

- ◯ Not like me
- ◯ A little like me
- ◯ Pretty good description of me
- ◯ Describes me perfectly

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7.3.3. Sensory questionnaire for custard (fat mouthfeel sensation) questionnaire (Dessert Taste study, Chapter III)

Vanilla custard Code: 821

Name:

Before you taste, please just smell the custard in front of you…

1. How much do you like or dislike the **aroma** of the vanilla custard?

Dislike extremely *(Strongest imaginable dislike)* — Like extremely *(Strongest imaginable like)*

□ Not applicable

2. Compared to your ideal vanilla custard, what do you think of the **aroma strength**?

Too weak — Just right — Too strong

□ Not applicable

Please try the custard sample…

3. **Overall**, how much do you like or dislike this vanilla custard?

Dislike extremely *(Strongest imaginable dislike)* — Like extremely *(Strongest imaginable like)*

4. How much do you like or dislike the **taste** of this vanilla custard?

Dislike extremely *(Strongest imaginable dislike)* — Like extremely *(Strongest imaginable like)*

5. Compared to your **ideal** vanilla custard, how would you rate the overall **flavour strength**?

Not enough flavour — Just right — Too much flavour

6. How much do you like or dislike the **mouthfeel** of this vanilla custard?

Dislike extremely *(Strongest imaginable dislike)* — Like extremely *(Strongest imaginable like)*

7. Compared to your **ideal** vanilla custard, how would you rate the **mouthfeel**?

Too dry — Just right — Too fatty/oily

8. How much do you like or dislike the **sweetness** of this vanilla custard?

Dislike extremely *(Strongest imaginable dislike)* — Like extremely *(Strongest imaginable like)*

9. Compared to your **ideal** vanilla custard, how would you rate the **sweetness**?

Not sweet enough — Just right — Too sweet

10. How would you rate the **fat content level**?

Very low fat content *(The lowest I have ever tasted in custard)* — Very high fat content *(The highest I have ever tasted in custard)*

11. How would you rate the intensity of **fat taste**?

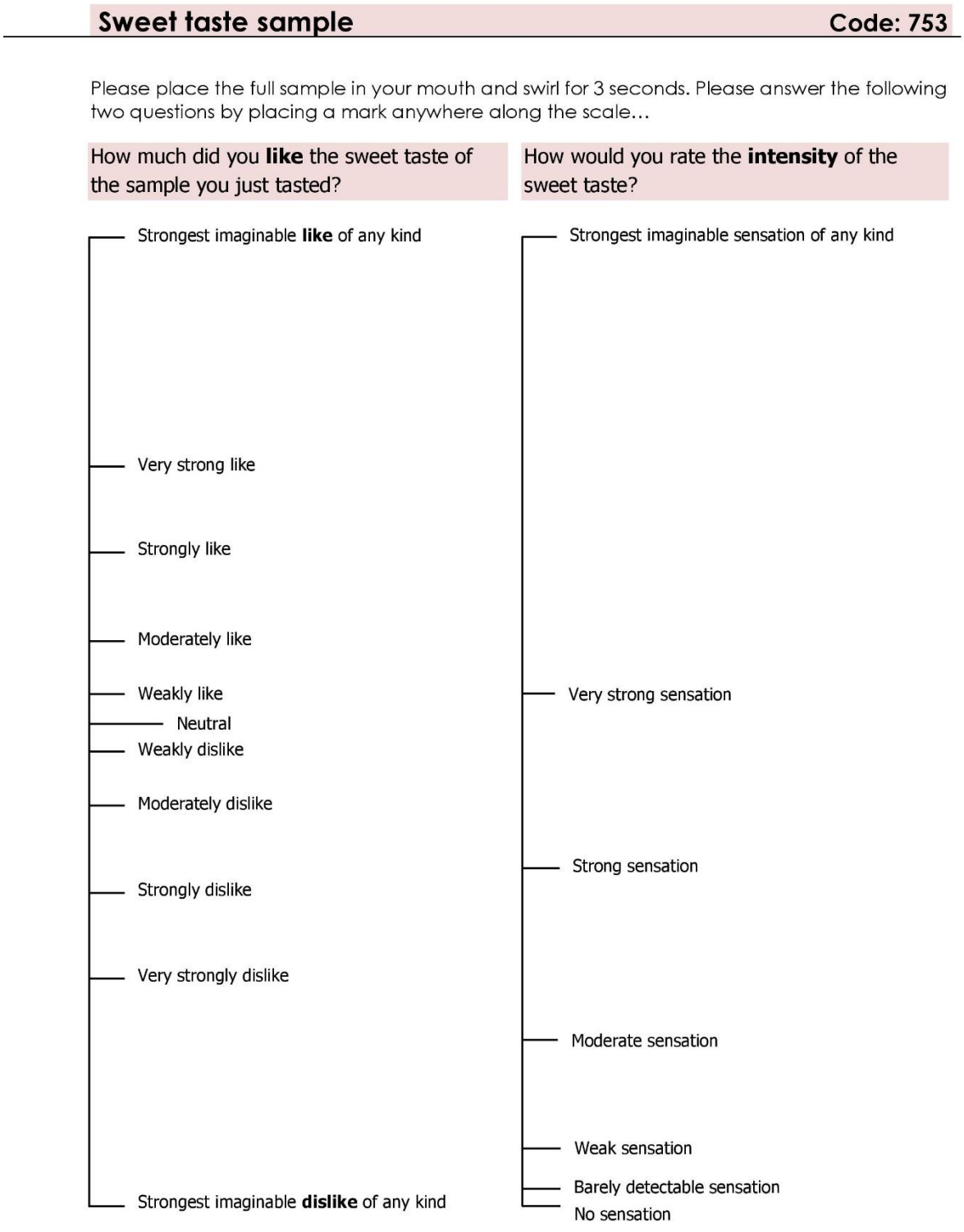
Very low fat taste *(The lowest I have ever tasted in custard)* — Very high fat taste *(The highest I have ever tasted in custard)*

12. Finally, did you notice any unusual taste? If so, can you please describe it? *(Please be specific)*

_____

_____

7.3.4. Hedonic liking and intensity rating LAM scale and gLMS template (PROMISE study, Chapter IV and V)

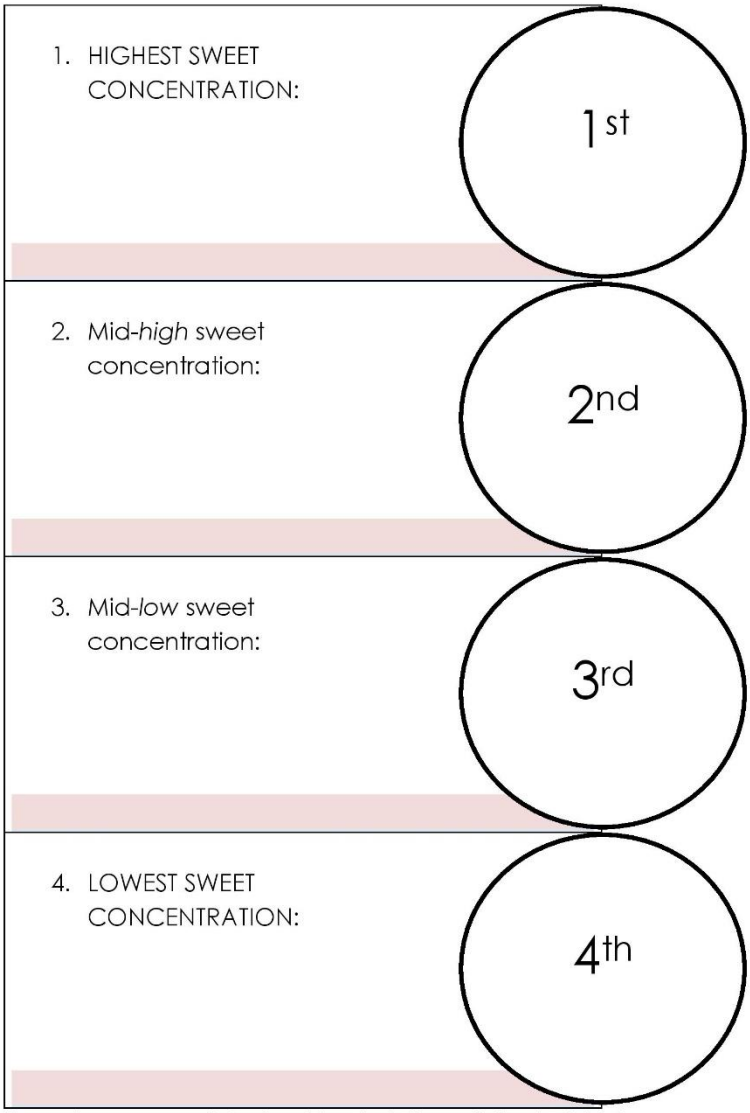


7.3.5. Ranking task questionnaire template (PROMISE study, Chapter IV and V)

Sweet taste ranking task

Please taste **all** four samples in front of you.

Order these 4 samples starting from the **highest** concentration (1) to **lowest** concentration (4).
Please attempt to rank all the samples, by placing the empty portion cups in the 4 circles below. Please rank them all, even if you are uncertain of the correct order:



Please send through your completed ranking task through the hatch door…

MASSEY UNIVERSITY
COLLEGE OF HEALTH
TE KURA HAUORA TANGATA

PROMISE Study

5 Day Food Record

Thank you very much for taking part in the PROMISE Study.
We are extremely grateful for your time, effort and commitment!

If you have any questions, please contact PROMISE staff on:
414 0800 (extn 49013)
email: promise@massey.ac.nz

All information in this diary will be treated with the strictest confidence. No one outside the PROMISE study will have access to this data.

Please bring this food diary with you to visit 2 at the Nutrition Laboratory

What to do?

- Record all that you eat and drink on the following dates.

_____
_____
_____
_____
_____

- If possible record food at the time of eating or just after – try to avoid doing it from memory at the end of the day.

- Include all meals, snacks, and drinks, even tap water.

- Include anything you have added to foods such as sauces, gravies, spreads, dressings, etc.

- Write down any information that might indicate **size or weight** of the food to identify the portion size eaten.

- Use a new line for each food and drink. You can use more than one line for a food or drink. See the examples given.

- Use as many pages of the booklet as you need.

Describing Food and Drink

- Provide as much detail as possible about the type of food eaten. For example **brand names and varieties / types** of food.

General description	**Food record description**
Breakfast example – cereal, milk, sugar	1 cup Sanitarium Natural Muesli 1 cup Pam's whole milk 1 tsp Chelsea white sugar
Coffee	1 tsp Gregg's instant coffee 1 x 200ml cup of water 2 Tbsp Meadow fresh light green milk
Pasta	1 cup San Remo whole grain pasta spirals (boiled)
Pie	Big Ben Classic Mince and Cheese Pie (170g)

- Give details of all the **cooking methods** used. For example, fried, grilled, baked, poached, boiled…

General description	Food record description
2 eggs	2 size 7 eggs fried in 2tsp canola oil 2 size 6 eggs (soft boiled)
Fish	100g salmon (no skin) poached in 1 cup of water for 10 minutes

- When using foods that are cooked (eg. pasta, rice, meat, vegetables, etc), please record the **cooked portion** of food.

General description	Food record description
Rice	1 cup cooked Jasmine rice (cooked on stove top)
Meat	90g lean T-bone steak (fat and bone removed)
Vegetables	½ cup cooked mixed vegetables (Wattie's peas, corn, carrots)

- Please specify the **actual amount of food eaten** (eg. for leftovers, foods where there is waste)

General description	Food record description
Apple	1 x 120g Granny Smith apple (peeled, core not eaten – core equated to ¼ of the apple)
Fried chicken drumstick	100g chicken drumstick (100g includes skin and bone); fried in 3 Tbsp Fern leaf semi-soft butter

- **Record recipes** of home prepared dishes where possible and the proportion of the dish you ate. There are blank pages for you to add recipes or additional information.

Recording the amounts of food you eat

It is important to also record the quantity of each food and drink consumed. This can be done in several ways.

- By using household measures – for example, cups, teaspoons and tablespoons. eg. 1 cup frozen peas, 1 heaped teaspoon of sugar.

- By weight marked on the packages – eg. a 425g tin of baked beans, a 32g cereal bar, 600ml Coke

- For bread – describe the size of the slices of bread (eg. sandwich, medium, toast) – also include brand and variety.

- Using comparisons – eg. Meat equal to the size of a pack of cards, a scoop of ice cream equal to the size of a hen's egg.

- Use the food record instructions provided to help describe portion sizes.

General description	**Food record description**
Cheese	1 heaped tablespoon of grated cheese 1 slice cheese (8.5 x 2.5 x 2mm) 1 cube cheese, match box size Size 10B grated cheese,

- If you go out for meals, describe the food eaten in as much detail as possible.

- ***Please eat as normally as possible - don't adjust what you would normally eat just because you are keeping a diet record and be honest! Your food record will be identified with a number rather than your name.***

Example day

Time food was eaten	Complete description of food (food and beverage name, brand, variety, preparation method)	Amount consumed (units, measures, weight)
7:55am	Sanitarium weetbix	2 weetbix
" "	Anchor Blue Top milk	150ml
" "	Chelsea white sugar	2 heaped teaspoons
" "	Orange juice (Citrus Tree with added calcium – nutrition label attached)	1 glass (275 ml)
10.00am	Raw Apple (gala)	Ate all of apple except the core, whole apple was 125g (core was ¼ of whole apple)
12.00pm	Home made pizza (recipe attached)	1 slice (similar size to 1 slice of sandwich bread, 2 Tbsp tomato paste, 4 olives, 2 rashers bacon (fat removed), 1 Tbsp chopped spring onion, 3 Tbsp mozzarella cheese)
1.00pm	Water	500ml plain tap water
3.00pm	Biscuits, chocolate covered Girl Guide biscuits	6 x (standard size)
6.00pm	Lasagne	½ cup cooked mince, 1 cup cooked Budget lasagne shaped pasta , ½ cup Wattie's creamy mushroom and herb pasta sauce, ½ cup mixed vegetables (Pam's carrots, peas and corn), 4 Tbsp grated Edam cheese
6.30pm	Banana cake with chocolate icing (homemade, recipe attached)	1/8 of a cake (22cm diameter, 8 cm high), 2 Tbsp chocolate icing
" "	Tip Top Cookies and Cream ice cream	1 cup (250g)
7.30pm	Coffee	1 tsp Gregg's instant coffee 1 x 300ml cup of water 2 Tbsp Meadow fresh blue top milk 2 tsp sugar

Date_____ **DAY 1**

Time food was eaten	Complete description of food (food and beverage name, brand, variety, preparation method)	Amount consumed

Recipes (Day 1)

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Figure 1.

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Figure 2.

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Washington, DC 20005

Phone: 202-962-4000
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