Excessive consumption of fat and sugar is associated with development of diet related diseases. While there are multiple factors involved with overconsumption of sweet and fatty foods, the taste system is responsible for identifying sugars and fats in foods, and in this way inform consumption of potential foods. Published research linking sweet taste and sugar consumption is limited and conflicting with one recent dietary intervention supporting a link between sweet taste and sugar consumption. Fat taste is an emerging area of interest, and recent research illustrates a direct link with high-fat diet increasing fat taste thresholds while low-fat diets decrease fat taste thresholds.

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Introduction
Diets high in fats, refined sugars or both increase susceptibility to chronic disease including obesity [1–3]. In this review conflicting evidence is presented with one study identifying an association between sweet taste and sugar consumption, and emerging evidence indicating a relationship between dietary fat and fat taste thresholds.

The sense of taste
Taste receptors in the oral cavity give rise to perceptions such as sweet and bitter, which enable organisms to identify nutrients and avoid toxins [4]. Homologous post-oral taste receptors lining the gastrointestinal tract (GIT) also serve physiological functions, such as orchestrating reflexive responses including gastric emptying and hormone secretion to optimise nutrient metabolism [5,6*]. Variations in oral or post-oral taste receptor physiology may therefore influence food choice or metabolism [5].

Psychophysical measures of taste function include detection threshold (DT), the lowest concentration of a stimulus that is perceivable, recognition threshold (RT) the concentration at which the quality of the stimuli can be correctly identified, and the suprathreshold intensity range which increases with increasing stimuli concentration to a terminal threshold [7]. These three measures are all reflective of tastes perceptual domain, but studies have illustrated that the measures are not correlated with each other [8,9] meaning that consumption of sugar or fat may associate with one measure of taste function, such as DT, but not correlate with RT or perceived intensity.

Sweet taste
Sweet taste is mediated by taste cells expressing the heterodimer of T1R2 + T1R3 with variation in gene expression underpinning inter-individual variation in sweet taste perception, which may in-turn influence sugar consumption [10–12,13*]. In support of this, evidence from a genotyping study involving $n = 137$ subjects showed genetic variation in the T1R2 gene was associated with significant difference in total free sugar intake in overweight and obese individuals, but not healthy weight population [14**]. Unfortunately the study did not assess sweet taste function making it impossible to assess if the measured taste gene variance or the consumption of free sugars was associated with perceptual measures of sweet taste.

One small well-controlled dietary intervention has been undertaken with 13 subjects completing a three-month low-sugar diet and a 16 participant control group remaining on their normal diet [15**]. There were no significant changes in sweet taste thresholds during the intervention and no changes in preference for sweet foods in either group or between groups. Sweet taste intensity measures using general labelled magnitude scale (gLMS) were also reported and while there was no difference in intensity ratings during the first month, the low-sugar group rated sweet foods as more intense in months two and three. A one-month post-intervention follow-up showed the low-sugar intervention group had returned to their normal diet and sweet taste intensity returned to baseline. This is direct evidence that a low-sugar diet causes an increase in sweet taste intensity. In contrast to this, one cross-sectional study ($n = 85$ adults) using gLMS intensity ratings failed to find an association between perceived sweetness,
A variety of food in diet, food beliefs, or dietary intake although this study used only one measure of sweet taste [16].

A meta-analyses of randomised controlled trials and co-hort studies reported reduced intake of dietary sugars is associated with a decrease in body weight, and increased sugars intake was associated with a comparable weight increase [17]. This suggests lean and obese will have different intake of sugar and comparable differences in taste function would provide circumstantial evidence of a link between consumption and taste, although the link between sugar intake and obesity remains controversial [18]. Studies have shown that there is a wide range of taste responses to sweet stimuli amongst people with obesity and lean persons with little consistency in results. For example, there was no difference in ability between lean participants ($n = 13$) or participants with obesity ($n = 59$) to identify a 0.175% sucrose solution from water using a two sample forced choice procedure over 175 trials [19,20]. Similarly, a recent study assessing sweet taste threshold using 3-alternate forced choice method in individuals with obesity ($n = 52$) and lean participants ($n = 56$) revealed no significant difference between the populations. In contrast, ascending concentration forced-choice method to determine sucrose DTs in women ($n = 72$) found those with higher body mass index (BMI) and body fat had significantly higher thresholds [21]. Similar results were reported for sucrose DT in a study containing individuals with obesity ($n = 51$) and lean participants ($n = 52$), with obese individuals having significantly higher threshold [22*].

It is possible that sweetness detection or RTs have little to do with intake, and that suprathreshold intensity measures are more appropriate when assessing taste associations with diet [7,9,23]. Measures in the sweetness suprathreshold range using magnitude estimation method also failed to find any differences in intensity between lean ($n = 13$), moderately obese ($n = 14$) and extremely obese ($n = 39$) [20,24**]. However, as Bartoshuk et al. later noted, comparison between these groups may be erroneous as the magnitude estimation method provided standardisation of the scale across people rather than allowing free use of labelled scales [25*]. Using sweetness intensity of candy rated on the gLMS which is appropriate for cross-group evaluation, participants ($n = 3740$) with higher BMIs found the candy less sweet (sr = −0.04) [25*]. In the interpretation of the data it should be noted that the semi partial correlation was small and there was no mention of controlling for odour or other sensory attributes present in the candy that may have influenced the result. Even with large numbers of participants and appropriate psychophysical scaling used, the magnitude of difference in sweet taste intensity between lean participants and those with obesity was minor.

The studies mentioned prior are not intended as a comprehensive review but do nevertheless reflect the discrepancies between studies, thereby ensuring no absolute conclusion can be made on whether sweet taste function and sugar intake are associated.

Hedonics of sweet taste rather than the intensity or threshold of sweetness may influence intake of sweet foods, although as with intensity, much of the data is conflicting. A large 4-survey study by Pangborn and Simone collected liking and preference information from 12 505 participants, all of whom provided anthropometric information [12]. In-home assessment of canned pears ($n = 708$ adults) and apricots ($n = 370$ adults) with 30, 40, 50, and 70% sucrose syrup was performed using 9-point hedonic scale; there was no association between hedonic rating for sweetened apricots and pears with body weight. Pangborn and Simone also used a paired preference method during the 1956 California State Fair to evaluate ice cream ($n = 6093$) with varying levels of sweetness (11, 13, 15, 17 and 19%, w/v, sucrose) and peaches ($n = 5334$) with varying levels of sweetness (45, 55 and 65% sucrose syrup) and reported no significant difference in preference between underweight, healthy weight or overweight participants [12]. In contrast, Bartoshuk and colleagues illustrate the interaction between BMI, sweet intensity and sweetness liking where they plot remembered liking of sugar against actual liking of a candy and group subjects according to BMI. The results show that correlations coefficient increased with each BMI category: underweight ($r = 0.0$), normal ($r = 0.1$), overweight ($r = 0.13$) and obese ($r = 0.25$), meaning for the same perceived sweetness, liking increases as BMI increases [25*]. Other well controlled studies have compared sweet taste DT [19,26,27] and suprathreshold intensities [27–29] with sweetness preference between different BMI groups and failed to find any significant associations.

While data are limited and conflicting, it is important to note that discrepancies between studies may be attributed to type of methodology, or using only one measure of taste function [25*]. It is also probable that the association between sweet taste and sugar intake is minimal due to the multiple other factors that influence diet, one of which will be binary interactions of sugars with other components of food, such as fat. A small study measured liking for fatty-sweet mixtures on a 9-point hedonic scale in healthy weight individuals ($n = 15$), individuals who are formerly obese ($n = 8$) and individuals with obesity ($n = 12$). Three dimensional plots were created using Response Surface method which illustrated differences between the groupings, with obese disliking sweetness without fat compared to healthy weight, while both groups equally liked the sweetened high-fat mixture [30]. Similar methodology using sugar–fat mixtures was employed by Hayes and Duffy, and while not assessing BMI found at high sugar/high fat levels participants with
Fat taste sensitivity in the oral cavity and gastrointestinal tract and proposed differences between lean and obese individuals. Fats in food are broken down into FFAs by lipase enzymes in the mouth (1), and interact with putative receptors within taste cells. Lean individuals may have an increased quantity of these receptors, compared to obese individuals. The presence of fatty acids in the mouth elicits the release of intracellular Ca\(^{2+}\) and neurotransmitter activation, eliciting a taste perception (2). The brain centre talks with GIT via vagus nerve (3). In normal weight individuals (4), fat ingestion triggers the release of satiety hormones including ghrelin, leptin, CCK, PYY, GLP-1, while comparatively, obese individuals (5) have decreased expression of fatty acid specific receptors, impairing fat sensing ability, thereby increasing energy intake. Reproduced by permission from [46].
low fungiform papillae density (n = 41, 15 female) experienced less intensity but retained liking for the mixture, while those participants with high fungiform papillae (n = 38, 21 female), experienced increased intensity and reduced liking for the sweet-fat mixture [31,32]. The role of fat (and other food components) in intensity and liking of sweetness and consumption of sugar may be important factor in food consumption.

**Fat taste**

Fat taste is an emerging area of interest particularly in chemosensory and nutrition research [33,34], and there has been direct evidence of a relationship between fat consumption and fat taste. The impetus for the research is the association between excessive intake of dietary fats and the obese state [2,35,36,37].

The relationship between fat taste thresholds, dietary fat intake, and BMI has recently been investigated by our group and others [5,36,38,39,40,41]. Some studies have found that those who were more sensitive (lower taste thresholds) to the fatty acid C18:1 had lower energy intakes and consumed less total dietary fats, and were also better at detecting the fat content of food (custard) [5,36,39,40,41]. In contrast, some studies show no association between fat taste thresholds and BMI [36,40] and one study investigating fatty acid intensity (not threshold) found no relationship [42]. The conflicting results in the literature may be due to methodological differences including the vehicle solution (water or milk) or type of fatty acid used [34,43]. Another likely explanation is the cross-sectional design of the above research using dietary assessment methods prone to error, whereas weekly weighed food records may provide greater level of accuracy of intake.

Direct evidence of the link between diet and fat taste has been shown in two intervention studies. The first study was a 4-week randomised cross-over design high-fat/low-fat dietary intervention that showed fat taste thresholds can be modified by dietary fat, with a 45% energy from fat diet causing increase in fat taste thresholds (decreased sensitivity) in lean individuals but not participants with obesity, while a 15% energy from fat diet resulted in decrease in fat taste threshold (increased sensitivity) in both lean and participants with obesity [40]. The second study was a 10-week low-fat weight loss diet that resulted in decreased fat taste threshold (increased sensitivity) in subjects who were overweight and obese [44]. The two dietary interventions have been consistent and shown that modifying the fat content of diet does influence fat taste threshold. However, a single meal two hours prior to fat taste threshold has no influence [36], so there appears a critical window between 2 hours and 4 weeks where the level of dietary fat or repeated exposure to high or low fat foods has an effect of fat taste thresholds. Given the influence of diet on fat taste threshold, a single threshold determination without standardising diet and collecting recent dietary history may limit validity of studies investigating the relationship between diet and taste. This is the likely explanation for the conflicting cross-sectional data previously discussed surrounding fat taste, diet and BMI.

The association between fat taste, diet and obesity are probably a result of a coordinated alimentary canal response to dietary fat [45,46] (Figure 1). Indeed, a link between fat taste and GIT responses to fatty acid has been established with individuals with obesity having impaired responses to fatty acid in the oral cavity and the GIT [5,44,47,48] compared to healthy weight subjects. Satiety mechanisms may be impaired in the individuals with obesity with subjects voluntarily consuming twice as much energy from fat products as non-obese [5,49]. Moreover, illustrating the role fat taste sensitivity may have in overconsumption of fatty foods, participants who were classified as orally hyposensitive to C18:1 found fat the least satiating macronutrient, while those who were classified as hypersensitive to C18:1 found fat the most satiating [36]. This result was specific for the high fat meal; this was not observed following a high carbohydrate, high protein or balanced meal [36].

The link between fat taste threshold and ability to identify fat in foods and subsequent preference or liking for fatty foods has also been investigated with conflicting results [38,40,50,52]. Two studies have shown links with diet and preference for fatty foods, with lower fat diets or lower sensory exposure to fats being associated with increased preference for lower fat foods [38,53]. Whereas other cross-sectional studies failed to find associations between habitual diet and preference for fatty foods [39,51]. Low and high fat dietary interventions have modified fat taste thresholds within four weeks as discussed previously, but these same studies that measured fatty food preference before and after the dietary intervention reported no significant or consistent changes in preference for fatty foods [40,44].

**Conclusion**

Inconsistency between studies is to be expected as there are multiple factors influencing sweet and fat taste perceptions/preferences, food preferences, food choices and the type and quantity of food consumed. One consistent is the role of dietary fat influencing fat taste thresholds. As fat taste thresholds are associated with energy consumption, fat taste has major implications for obesity research and development of obesity related pathologies. Sweet taste intensity and sugar consumption may be associated as indicated by a recent dietary intervention, and field would benefit from further well controlled studies including the use of high intensity sweeteners in interventions to gain insights from decoupling sweetness and energy.
Conflict of interest statement
Nothing declared.

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References and recommended reading
Papers of particular interest, published within the period of review, have been highlighted as:

- of special interest
- of outstanding interest


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