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## Review Article

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# How sensory experiences shape our eating behaviour: evidence for individual taste differences affecting weight management

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## Abstract

The modern surge in obesity can be seen as evidence that food choice remains an important area of study. Sensory science has also provided a necessary contribution to this area. This review will describe the latest scientific understanding on flavour perception and how metabolism, weight management, exercise and food design might affect this perception. Taste research has shifted from sensory testing paradigms to a receptor level scientific understanding of gustation with the discovery of the heterodimeric taste receptor type 1 proteins (T1R) for sweet and umami taste, and the family of taste receptor type 2 (T2R) for the detection of bitterness. *TA2R38* has been widely investigated to understand how genetic diversity in bitter taste might influence weight management. Currently there is no substantial evidence that variations in bitter taste affects overall weight status of subjects. However, gustation is highlighted as the key sensory determinant of eating behaviour where there is some evidence that chronic consumption of foods rich in salt and sugar result in a reduced sensitivity to these tastes. Although a strong link between taste sensitivity and BMI appears inconclusive. Human gustatory response to fatty acids is still a matter of debate although dysfunction of this taste maybe seen in cases of obesity. However, gustation is not experienced in isolation and there is an interplay between food structure, oral processing, texture and taste. Therefore, to fully understand the contribution of sensory evaluation in eating behaviour, dynamic sensory techniques need to be applied where facial electromyography has potential.

## Introduction

Foods are a multisensory experience where vision, smell, taste, texture, sound and the trigeminal response combine to create our perception of the foods and drinks we consume. At the peripheral level, these sensory modalities are anatomically and largely neurologically separate where photoreceptors, mechanoreceptors, chemoreceptors, chemosensitive nociceptors and thermoreceptors relay critical information about the condition of the food before, during and after consumption. These modalities however overlap centrally where cross-modal interactions mould our overall perception of the foods we consume[1–3]. These sensory experiences are generally evaluated positively or negatively by consumers through psychological constructs such as affect/liking, preference and wanting with contextual factors such as environment, commensality and other psychological factors such as emotional processing also influencing the final scoring of these measures[4,5]. Considering the complexity of reactions to food cues there have been several methodological approaches taken to try and gain insight into why people make the food decisions they do. One approach taken by scientists (including the authors of this paper) is by studying facial reactions using facial electromyography (FEMG) to provide additional insights into emotional and affective responses to food cues beyond normal hedonic self-reporting techniques[6].

Although the overall picture of food experience is complex, familiar and positive flavour and texture experiences are key determinants in shaping consumer food behaviour. And may if unchecked, drive excessive eating, overweight/obesity and the associated risk of chronic disease[7]. Further, gustation has been proposed as the most dominant modality in shaping our eating behaviour with sweet taste as a key factor in excessive eating[8,9]. However, gustation is rarely experienced in isolation as flavour characteristics are time dependent, relying on the oral processing behaviour of subjects. The mastication processes will initiate and drive the physical and chemical processes releasing the flavour active compounds which is in part dependent on the structure of the food matrix[10].

Therefore, this review will focus on gustation. It will also explore how individual differences in taste perception may affect weight management. In addition, it will briefly investigate the relationship between food structure, texture and sensory perception. Finally, it will review the use and application of FEMG in investigating food behaviour.

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## Flavour chemoreception

Flavour is a combination of gustatory, olfactory and chemesthesis modalities[11,12]. Taste is sensed by chemoreceptor cells found in the mouth whilst olfaction is sensed via chemoreceptor neurons found in the nose. The sensory experiences of odour (orthonasal) and aroma (retronasal) are created by small, volatile chemicals which are released in gaseous form from the foods either naturally or through the process of mastication and interact with the olfactory sensory neurons in the olfactory epithelium. In contrast molecules that possess a taste need to be in part soluble in saliva to enable accession to the receptor cells located mostly but not exclusively in taste buds which in turn are in the circumvallate, fungiform and foliate papillae found on the back, front and sides of the dorsal surface of the tongue[13]. The receptor types for sweet, bitter, umami and aroma are G protein-coupled receptors (GPCRs), whereas the sour and salty tastes are thought to be mediated by ion channels. This is an important distinction as it explains why we have intense sweeteners, for example, saccharin which is 300–500 times sweeter that sucrose[14], but no intense salting or souring agent have yet been discovered. It also part explains why it is generally agreed that common table salt (NaCl) provides the only true salty taste as the Na channels found on the epithelium of taste cells are highly selective for Na ions. Although for the complete picture, we need to understand in more detail the role of the chloride ion in generating this unique salty taste[15].

One of the key taste research breakthroughs was the discovery that GPCRs specific for sweet and umami are heterodimeric proteins made up of the T1R (taste receptor type 1) family where T1R2 and T1R3 combine to form the heterodimer protein complex which mediates sweet taste reception and T1R1 and T1R3 combine to form the heterodimer protein complex which mediates umami (savoury) taste reception[16]. It cannot be ignored that the central nervous system plays a key role in sensory regulation. However, polymorphism of the *TAS1R* gene has been shown to explain mostly the perceptual differences in sweetness between and within species which in turn identifies the importance of peripheral taste mechanisms in the overall perception of the sweet taste modality[17,18].

## Bitter taste, 'supertasters' and the relationship to BMI

In contrast bitter taste is mediated by T2R (taste receptor type 2) receptor proteins and is more complex being recognised by a family of receptor types which are in some types broadly tuned to an array of bitter compounds or in other types specific only to individual bitter compounds[19]. Although there have been extensive genetic investigations identifying about 25 functional bitter taste receptors, the main bulk of this work in terms of understanding individual human taste differences has relied on our understanding of the T2R38 bitter receptor. This receptor is activated by phenylthiocarbamide (PTC) and the less toxic 6-*n*-propylthiouracil (PROP) which makes PROP the preferred chemical for researching this phenomenon. Individual polymorphic differences can be observed with self-reported suprathreshold testing where very roughly a quarter of the population finding PROP/PTC intensely bitter, a similar proportion being non-tasters with the remainder finding PROP/PTC moderately bitter. Genotype categorisation of individuals is based on the *TA2R38* SNP (i.e. tasters PAV/PAV or PAV/AVI and non-tasters AVI/AVI) accounting for 50–85% of the variation in the phenotype[20]. One area that has been explored is the relationship with supertaster status and BMI. The hypothesis for this relationship being that supertasters may find many foods intensely bitter leading to the rejection of a wide array of food items resulting in lower nutrient and caloric intake. In support of this hypothesis the T2R38 receptor has been reported to be broadly tuned to an array of bitter compounds[19]. However, the results from the first author's laboratory found no significant relationship with BMI and supertaster status which, at that time, resulted in adding to the growing conflicting literature on the matter[21]. Notably though, a recent systematic review and meta-analysis judged in favour of our interpretation; finding no clear link between bitter sensitivity and BMI[22]. However, the term supertaster has become entrenched in sensory science and has largely detangled itself from its origins of the PROP/PTC experiments[23]. The term now extends beyond bitterness and encompasses other tastes and odours which subgroups of the general population may have a hypersensitivity towards whilst also encompassing other phenotyping approaches.

## Taste plasticity

The relationship between dietary exposure of foods rich in fat, salt and sugar and changes in taste perception has recently attracted increasing levels of research interest. Research suggests that taste sensitivity tends to be reduced in those with overweight/obesity[24–27]. For example, one relatively recent study found a reduced sensitivity in all tastes (salty, sour, sweet and bitter), apart from fat (rapeseed oil) with increasing BMI[24]. Similarly, another recent study found that sensitivity to all four basic tastes (sweet, sour, salty and bitter) were reduced in those with obesity[25]. A systematic review including 26 studies on weight change and taste function, found taste sensitivity in all basic tastes (especially sweet and salty) to be reduced in those with overweight and obesity[26]. Interestingly, this was more pronounced in men. Longitudinal studies in this analysis showed that sweet and fat preference increased with weight gain in adults[26]. Thus, suggesting a correlation between increased food consumption, namely foods rich in fat, salt and sugar, overweight/obesity and reduced taste sensitivity overtime. This relationship may then perpetuate a destructive cycle of need/desire for increased concentrations of sweet, fat and salty foods to appease desensitisation, while inducing weight gain.

However, some studies find no differences between individuals who were lean and overweight/obese and taste sensitivity[27], and some studies even suggest that overweight/obesity increases taste sensitivity[28]. Many factors may help to explain these mixed results; namely variation in taste measurements (detection, recognition thresholds, pleasantness/liking), biological factors (age, sex, genetics)[29] and possibly varying levels of visceral fat/subcutaneous fat. One study researching 179 women (underweight – morbid obesity) observed that visceral fat was the strongest predictor of both smell and taste function, more so than BMI or total fat mass. Higher visceral fat was linked to a reduced sensitivity to tastes, even after accounting for other factors. The authors recognised that this interesting observation may be indicative that metabolic dysfunction plays some type of role in these changes in taste perception with the search for the signals originating from the visceral adipose tissue as a possible area of further research[30]. Therefore, further work utilising similar methodologies to assess taste and fat mass are needed to fully explore this observation.

In conclusion a strong link between taste sensitivity and BMI appears inconclusive. Although, an association may become more apparent in more extreme cases such as participants with obesity

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and or older participants with metabolic syndrome where basic taste tests show lower sensitivity or perception particularly for sweet and salty tastants. Further there is some inconsistent evidence that significant weight loss may improve sweet taste sensitivity[26]. Taste changes have also been reported after bariatric surgery. In this extreme case, where patients with morbid obesity undergo surgical procedures, that is, the laparoscopic sleeve gastrectomy (LSG) or the Roux-en-Y gastric bypass (RYGB), there is growing evidence that the procedure will change taste perception. Although how taste perception will change is not entirely clear. This may in part be due to the approach taken where studies are split between participants self-reporting their gustatory experiences after surgery and those where psychophysical methodology is applied with the taste stimuli given to participants at a known concentration(s)[31]. One systematic review of twelve studies encompassing 899 patients where 726 patients' data was collected by questionnaire and a further 173 patients by stimuli solutions reported that the type of surgical procedure may influence the type of taste changes seen. In this review the authors reported that LSG shows an increase in sensitivity to all four taste domains whilst RYGB showed a decreased sensitivity to sweetness but increased sensitivity to salty taste. Although still 152 patients out of 411 sweetness intensity increased after RYGB surgery[32]. Another systematic review virtually carried out at the same time concluded that RYGB surgery increased sensitivity to sweet taste[31]. Currently it is safe to conclude that after bariatric surgery sweet taste intensity of patients is sometimes increased finding sweet foods after surgery as 'overly intense' or unpleasant which may in some cases be long lasting not just temporary[31–33]. The proposed mechanisms behind this taste plasticity are still unresolved although studies suggest that more disruption of the digestive system leads to more persistent and greater changes in taste[33] leading to proposed mechanisms focussing on the physiology of hunger, satiation and satiety. Mechanisms proposed therefore include changes in gut hormones in particular PYY, GLP-1 and ghrelin where these hormones may mediate peripheral taste receptor events and/or central processing. Other possible mechanisms proposed also include vagus nerve stimulation and improved metabolic signalling[34,35]. The role of GLP-1 in modulating peripheral taste events has been proposed to involve the localised release of GLP-1 by taste receptor cells which then activates the GLP-1 receptor on the closely aligned gustatory nerve fibres. More sweet ligands binding into sweet taste receptors generate more released GLP-1[36,37]. Research carried out with GLP-1 agonists and its effects on taste perception seems to support this mechanism where recent research indicates that the GLP-1 receptor agonists such as semaglutide can improve overall taste sensitivity including sweet taste[38].

## Acute changes in taste perception

A relatively early study has demonstrated that diurnal sweet taste sensitivity decreases in normal weight subjects in parallel with increasing leptin, postprandial insulin and glucose levels[39]. However, this interesting diurnal variation in sweet taste was not seen in subjects who had a BMI of 25 ($kg/m^2$) and greater which was hypothesised as being indicative of insulin resistance blunting the modulating effect of leptin on peripheral or central taste effects[40].

Acute interventions such as exercise have also been shown to change taste perception. One study (co-authored by the present first author) demonstrated that intensive exercise lasting over 60 min created noticeable reductions in salty taste, increases in sweet taste intensity perception and overall increases in liking of sports drinks with varying concentrations of electrolytes and carbohydrates[41]. The observation that acute exercise can change taste perception and preference has been commonly reported in the literature[42–44]. Although only preference to salty taste reliably increases after acute exercise[42]. One possible explanation for this can be understood through the physiological usefulness theory, where to ensure homeostasis after Na loss through sweating, the appetite for salt increases as the requirement for that compound also increases[45]. Salt seeking behaviour is mediated by hormones such as aldosterone and angiotensin II. Angiotensin II type 1 receptors have been shown in animal experiments to be expressed in taste-bud cells where angiotensin II has been shown to act directly on peripheral taste cells to reduce salty taste and increase sweet taste sensitivity[46]. There is some evidence that chronic exercise is associated with improved taste function[47,48]. Although it is not clear the specific role that regular exercise plays in taste function beyond its association with body composition and lifestyle. These acute intervention studies as discussed in this section therefore provide some important evidence that exercise does induce measurable taste changes independent of other longer-term adaptions.

## Fat as a taste

Over the decades sensory scientists have been considering the possibility of additional tastes beyond the four basic tastes of sweet sour salty and bitter. It has only recently been universally accepted by sensory scientists that umami is a fifth taste with the discovery of the T1R receptor[49]. More controversially, yet particularly relevant to the study of obesity, are investigations into a sixth basic taste, fat. The traditional view is that the sensory experiences of fats are described by subjects using mainly textural attributes relating to viscosity, thickness, mouthfeel, oiliness and olfactory attributes such as fatty smell. However more recently there is mounting evidence that fatty acids specifically linoleic and oleic acid may impart a specific gustatory response with relatively low detection threshold levels for linoleic acid and oleic acid being reported[50]. Based on animal experiments putative mechanisms have been proposed such as a delayed-rectifying potassium channel sensitive to PUFA[51], the CD36 fatty acid transporter[52] and the GPCR GPR 40 and GPR 120[53]. In contrast the human gustatory response to fatty acids is still a matter of debate although evidence exists that the CD36 fatty acid transporter and GPR120 may be functional in humans and are currently prime contenders for this proposed sixth taste[54–56]. Even if the human gustatory response is tuned to fatty acids most fats are in the TAG form. To complete the theory, it has been proposed that lingual lipase aids in the breakdown of TAG into fatty acids[57]. It is still debated if the lingual lipase plays a significant role in creating enough of the free form fatty acids to activate receptors although increased mastication is likely to aid in the conversion of TAG to the free fatty acid form[58]. So why the interest from a nutritional perspective? Previous studies have identified subjects that are hypersensitive and hyposensitive to long-chain fatty acids. Research has shown that participants who were hypersensitive to oleic acid taste (i.e. able to detect fatty acids at a level about 1.4 mM) consumed less energy, fat and carbohydrate and were more perceptive of small differences in fat content in foods. They also had measurable lower BMIs[59]. Further, participants who were hyposensitivity with lower CD 36 expression were likely to be participants with obesity[54].

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One plausible reason why fatty acid taste has not been identified earlier in sensory psychophysical experiments is that the other sensory characteristics of fatty acids need to be carefully masked. That is, the odour, viscosity and mouthfeel that are imparted by fatty acids will be invariably used by subjects to identify the fatty acid in an aqueous solution when applying a forced choice psychometric procedure. This can lead to a high level of false positives – even possibly concluding that a fatty acid taste exists when none does. This is likely to occur if these other sensory cues are not carefully controlled by adding mineral oil, thickeners (e.g. gum acacia), antioxidants (EDTA), appropriate homogenisation (to emulsify) and the wearing of nose clips (to remove odour and aroma cues) for all solutions tasted including the target solution and the control samples. However, a study conducted by the first author's research group showed that 27 male participants out of 40 subjects (BMI range of 19–32 Kg/m$^2$) were able to detect linoleic acid below 29.28 mM. Although, no relationship to BMI and fat taste status was able to be determined[60]. Recent research seems to indicate that fat chemosensory dysfunction may occur with individuals with obesity only and so we may have missed this critical relationship with our study by only having one subject fitting this category[55].

## Modifying foods to increase satiety

From a nutritional perspective, studying the oral processing of solid and semi solid foods are important from the perspective of investigating the role of chewing and bite size in affecting satiety. These studies may employ instrumental techniques such as articulography for measurements of jaw trajectories[61], and/or electromyography for studying the activity of the masseter and temporalis muscles during mastication[62,63]. Subjects report their sensory experience in real time using sensory dynamic techniques such as Temporal Dominance of Sensation (TDS) and/or Time Intensity (TI)[64].

The oral processing of foods can be divided into three phases: the preparatory, reduction and pre swallow phases. These phases follow the food being fractured (in the case of hard foods) with food particles being reduced in size by mastication assisted by the release of saliva to lubricate these mastication processes, followed by the formation of a soft swallowable bolus[10,64].The act of mastication/chewing is a complex process influencing not only perceived sensory changes during the eating process but also digestive processes leading to the possibility of influencing glucose absorption and the satiety signalling cascade (i.e. GLP-1, CCK, GIP and Insulin release) where increased chewing may suppress hunger[65] through increasing the bio-accessibility of nutrients[66]. Although how much chewing influences satiety in comparison to other eating variables (e.g. meal size, plate dimensions, social setting etc) is hard to measure. Taking all things into consideration, it is fair to say that chewing/mastication indirectly affects the Satiety Index of a food[67,68] and warrants more detailed investigations into how structural adaptions to foods can increase chewing effort and time. That is, the structure of food can be modified to increase chewing time by increasing the hardness, elasticity and cohesiveness of the food matrix by adding layers, embedding particles, creating multiple fracture pathways, reducing lubrication and increasing friction between food particles[69,70]. However, dynamic sensory experiences of the food being broken down also controls masticatory behaviour and so an interplay between the food structure and sensory experience determines overall eating trajectory[64]. That is in part oral processing behaviour is determined by pleasantness and enjoyment of the eating process. For example, there is evidence that if we do not like a food, we will consume the product more slowly through cautious eating behaviour[71]. More specifically if the food is matched in terms of its textural characteristics, it is likely that the taste/palatability will become the dominant factor determining eating rate[72].

## Facial electromyography studies

To measure these changes in affect, FEMG can be extended beyond the masseters (which measures chewing activity) to also measure corrugator (the muscle involved in frowning) activity which is associated with negative affect/disliking and the zygomaticus (the muscle involved in smiling) activity which is associated with positive affect/liking. The experimental set up now can measure not only the dynamic self-reported sensory responses of the participant but also objective parallel electromyographic measures of masseter activity and real time dynamic affect during the eating process. FEMG is a relatively new approach with most work being conducted in the last 10–15 year primarily focussed on sensory science rather than nutrition[73]. In addition, the methodology is far from being fully developed for it to be described as a 'standardised technique'. However, FEMG has potential to understand the innate reactions to food without relying on self-reporting techniques – a common limitation in nutrition research[74]. Food intake is often under or over reported (depending on study aim) by individuals due to a myriad of complex factors associated with food consumption. For example, one study investigated the use of FEMG on the masseter muscle to understand self-reporting accuracy. The authors found that eating could be detected with accuracy (>90% sensitivity) compared to trained self-reporting measures[74]. Additionally, another study found that when consuming gel-type food, subjective hedonic experiences (liking/disgust) and FEMG signals were dynamically coupled (i.e. liking ratings and facial response were correlated)[73]. Thus, suggesting that hedonic experiences during food consumption can be measured through FEMG. However, one of the concerns of this technique is how useful this methodology is beyond water-based solutions or gels due to the interference of the masseter muscle from greater chewing activity[75]. In part to answer this question one study (co-authored by the present first author) investigated the same hypothesis with chocolate, where FEMG successfully detected dynamic hedonic processes. In this experiment subjects rated the less bitter sample (36% cocoa solids) higher in liking than the more bitter (85% cocoa solids) sample and subsequently took longer on average to consume with increased corrugator activity[76].

## Conclusion

Taste is the major sensory modality in influencing eating behaviour of foods. Research over the last few decades provides evidence that our taste perception varies between individuals and within individual where, genetic variability (e.g. *TAS2R38*), changes in body weight, surgical procedures, exercise, some medications (e.g. GLP-1 agonists) and time of day all possibly creating various levels of changes in taste perception. There is also mounting evidence that these changes in taste perception are part of the hunger satiation and satiety signalling cascade regulating homeostatic eating behaviour. How much this influences weight changes is unclear and may be disrupted in this modern obesogenic environment by appetite dominating more natural hunger homeostatic signalling.

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In addition, gustation may also moderate the oral processing of foods which may influence the satiety properties of the foods. FEMG is an important method to measure positive and negative valence towards foods. This method can offer important insights into the hedonic value of foods, especially when coupled with other methods, and be of use in nutritional and sensory research.

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