Sweet taste and obesity

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\textbf{A R T I C L E I N F O}

\textbf{Keywords:}

Obesity
Bariatric surgery
Taste perception
Psychophysics
Reward-related feeding behavior

\textbf{A B S T R A C T}

For more than 50 years, there has been evidence for greater consumption of sweet foods in overweight humans and animals, relative to those that have a normal weight. Furthermore, it has long been suggested that energy deficit resulting from dieting, while moving the individual from a higher weight set point, would result in heightened susceptibility to palatable tastants, namely to sweet tastants. This was the motivation behind the first studies comparing sweet taste perception between individuals with obesity and those of a normal weight. These studies, using direct measures of taste, have been characterized by significant methodological heterogeneity, contributing towards variability in results and conclusions. Nevertheless, some of these findings have been used to support the theory that patients with obesity have decreased taste perception, particularly for sweet tastants. A similar hypothesis has been proposed regarding evidence for reduced brain dopamine receptors in obesity and, in both cases, it is proposed that increased food consumption, and associated weight gain, result from the need to increase sensory and brain stimulation. However, the available literature is not conclusive on the association between obesity and reduced sweet taste perception, with both negative and contradictory findings in comparisons between individuals with obesity and normal weight control subjects, as well as within-subject comparisons before and after bariatric surgery. Nevertheless, following either Roux-en-Y gastric bypass or sleeve gastrectomy, there is evidence of changes in taste perception, particularly for reward-related measures of sweet tastants, that should be further tested and confirmed in large samples, using consensual methodology.

1. Background

Obesity is associated with significant morbidity and mortality, and currently represents a global health challenge\cite{1}. While it is associated with complex pathophysiology, increased availability of highly palatable foods and beverages, namely those rich in sugar or fat, is thought to be a major determinant of increasing rates of obesity worldwide\cite{2}. Indeed, individuals with obesity have been shown to have altered sensitivity to food reward\cite{3}, which is thought to be related to changes in reward-related brain neurocircuitry, namely decreased striatal availability of dopamine D\(_2\) receptors (D\(_2\)R)\cite{3}. Sugar, through pleasant taste and postingestive value, triggers brain reward circuitries, stimulating consumption of foods that are rich in sugar\cite{4-6}. Another important factor is that, while in non-obese subjects striatal D\(_2\)R availability is inversely associated with sweet preference, in subjects with obesity this association is lacking, through mechanisms that have not yet been clarified\cite{7}. Beyond association between gustatory and reward-related circuits in humans\cite{8}, there is also pre-clinical evidence in rodents\cite{9} and fruit flies\cite{10} that dietary sugar content influences sweet taste perception. For example, in Drosophila melanogaster a high sugar diet led to decreased response of ‘sweet-taste sensing neurons’, resulting in diminished behavioral responses to sweet tastants\cite{10}. Importantly, reduction of sweet taste responses through neural manipulation resulted in overfeeding and obesity, further suggesting that sweet taste perception is a driver of obesity\cite{10}.

Evidence for association between taste, reward and morbidity obesity has also been collected in the context of weight loss. Bariatric surgery is broadly accepted as the most efficient treatment for obesity, leading to significant weight loss and maintenance of weight, as well as improvement of obesity related comorbidities\cite{11-13}. Several mechanisms have been proposed as determinants of changes in ingestive behavior following bariatric surgery, namely a global reduction of appetite, development of conditioned aversions and changes in reward-related feeding behavior\cite{14}, including modulation of taste-related reward circuits in humans\cite{8}.

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https://doi.org/10.1016/j.ejim.2021.01.023
Received 29 September 2020; Received in revised form 8 January 2021; Accepted 20 January 2021
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sensory function. In brief, Type II (receptor) cells have, in their apical
bitter, sour (acid) and umami (savory taste of amino acids) [8,9,34],
allowing for the detection of five distinct taste qualities: salt, sweet,
flavor is better defined as the complex perception resulting from
converging inputs from taste, texture and olfaction, induced by multi-
sensory stimulation from foods during mastication and swallowing [32].
This review will focus primarily in taste, rather than flavor perception.

Taste buds, located in the epithelium of the tongue, palate and
epiglottis, are the peripheral organs of gustation [33]. They are cell
groups shaped similarly to a garlic bulb, embedded in fungiform, foliate
and circumvallate papillae, located on the anterior, lateral, and poste-
rior regions of the tongue, respectively [9]. The taste receptor cells
(TRCs) within taste buds are classified, at least in part, according to their
function. In brief, Type II (receptor) cells have, in their apical
G-protein coupled receptors (GPCR) that are sensitive to bitter,
sweet or umami tastants [34]. Type III (pre-synaptic) cells have ico-
 tropeic receptors to identify acidic stimuli (sour taste) and release GABA,
serotonin and norepinephrine in synapses with neurons from cranial
nerves [34]. Type I cells are glia-like [33,34] while type IV (basal) cells
are undifferentiated cells located in the base of the taste bud [34].
Regarding salt taste, amiloride-sensitive epithelial sodium channels
have been shown to be involved in rodents, although in humans, further
research is needed [9]. Additional taste qualities have been proposed,
such as that occurring via the fatty acid translocase cluster determinant
36 (CD36), that is thought to contribute, with texture perception via the
somatosensory system, for identification of fat [8].

Activation of TRCs leads to neurotransmitter and peptide release
onto affenter fiber terminals of cranial nerves VII, IX and X (facial,
glossopharyngeal and vagus, respectively) that, in turn convey infor-
mation to the central nervous system [35] specifically to the nucleus
tractus solitarius, in the brainstem, that then relays neural information
to the thalamus and insula [8]. The area of the insula receiving taste
sensory information is the primary gustatory cortex, while areas of the
orbitofrontal cortex responding to taste stimulation, as well as to other
flavor-related sensory information (e.g., texture, temperature, and
odor), are sometimes defined as the secondary gustatory cortex [8].

3. Psychophysical measures of taste

For interpretation of the literature involving taste perception in
obesity, it is fundamental to understand the methods used for orosensory
assessment. One of the main challenges of such assessment is to capture
interindividual variability in perceptions of intensity (e.g., the percep-
tion of a soup being too salty or watered down [32]). There are two main
complementary perspectives regarding intensity perception. One
perspective treats intensity as a binary concept (i.e., the tastant is
identified as absent or present) that is thus measured according to the
threshold at which taste stimulation is identified. Both electrical
(Fig. 1A) and chemical methods can be used to assess taste thresholds
[32] (see Supplementary Table 1 for details). The other perspective as-
sumes perceived intensity as a continuous construct [32]. In this case,
chemical tests (e.g., Fig. 1B) can be used with supratreshold scaling,
including the general labeled magnitude scale (gLMS) [37] which is,
currently, the gold standard for this purpose (Fig. 1C; see Supplementary
Table 1 for details). The general labeled hedonic scale (gLHS) [38],
rather than assessing sensory-discriminative domains, assesses the de-
gree of pleasantness, or unpleasantness, of the stimulus [14] (Fig. 1D).
A full summary of methods commonly used for oral sensory assessment
in humans is provided in Supplementary Table 1, highlighting their
complexity and the need to consider methodological specificities in
interpretation of the available data.

4. Studies of taste perception in individuals with obesity

In studies comparing taste perception between individuals with
obesity and control subjects, using direct measures of taste, one of the
most explored outcomes was detection and/or recognition thresholds.
Distinct methods have been used, and overall, the results do not
consistently support that individuals with obesity have altered taste
sensitivity or require different concentrations of a specific tastant (e.g.,
sucrose) to detect taste (Table 1). Detailed inspection of the available
data shows that 3 studies, using the constant stimuli method (see Sup-
plementary Table 1 for details on this and other methods), did not find
differences between individuals with obesity and normal weight control
subjects in detection thresholds for sweet taste [18,19,26]. Another
study, using the 3-stimulus drop method, found no differences relating
to the presence of obesity for both detection and recognition thresholds
for salt, sweet, bitter and sour tastants [24]. Higher detection thresholds
(i.e. lower taste sensitivity) in individuals with obesity relative to
normal weight controls was reported for salt taste using a derivation of
the method of limits among young adults [22], and for umami using the
two-alternative forced-choice (2-AFC) staircase procedure in women
[21]. These studies found no differences for several other tastants,
such as quinine in both cases [21,22], and both quinine and citric acid
in one of the studies [22]. Additionally, one of these studies revealed
higher electrogustometry (EGM) thresholds in individuals with obesity,
despite no correlations with chemical thresholds [22]. While these
studies provide limited evidence for higher detection thresholds for salt
and umami, but not sweet, bitter or sour tastants, two other studies
investigating recognition thresholds with an up-down staircase pro-
cedure found that these were lower, rather than higher, among
individuals with obesity for sweet and salt, but not for bitter and sour
tastants [20,23].

A study assessing acuity scores for supra-threshold concentrations
of salt, bitter and sour tastants, using multiple-alternative forced-choice
tests, found lower acuity among individuals with obesity [28]. This
is consistent with higher detection thresholds for salt [22], but inconsistent
with evidence for unchanged or lower thresholds for salt, bitter and
sour; that have also been reported [20,22–24], as described above. On
the other hand, and in accordance with findings of lower taste recog-
nition thresholds for sweet and salt taste [20,23], the same studies
described higher intensity ratings for these taste qualities in individuals
with obesity when compared with subjects without obesity. In one of

[35, 15]. Indeed, following Roux-en-Y gastric bypass (RYGB) and sleeve
gastrectomy (SG), currently the most commonly performed bariatric
procedures, there is substantial evidence of self-reported decrease in
consumption, cravings and preference for palatable sugar-rich and/or
fat-rich foods (for review see Nance K., et al. [14]). Accordingly, patients
report changes in taste perception of sweet foods after surgery [14].

However, across the several studies exploring taste perception in
obesity [7,16–23], and its changes following bariatric surgery [24–30],
there is a notable heterogeneity in methods and results. This has led to
considerable difficulties in interpreting the available literature
regarding the contribution of taste perception towards obesity. In this
review, following an overview of taste physiology and psychophysical
assessment of taste in humans, we will focus on studies that used direct
measures of taste, using simple tastants rather than mixtures, to compare
between patients with obesity and control subjects, as well as studies
assessing the impact of bariatric surgery on taste perception.

2. Neurobiology of taste

Taste allows for identification and consumption of appetitive sub-
stances, like sucrose (sweet), and avoidance of potentially toxic and
unpleasant compounds, such as quinine (bitter) [9]. In addition to
informing feeding decisions, this system contributes to the physiological
regulation of starch and fat digestion, initiated through salivary secre-
tions [31], as well as to other elements of metabolic regulation through
processes such as the cephalic phase of insulin secretion [9]. In common
terms, taste is frequently used as an equivalent for flavor. However,
flavor better defined as the complex perception resulting from
converging inputs from taste, texture and olfaction, induced by multi-
sensory stimulation from foods during mastication and swallowing [32].
This review will focus primarily in taste, rather than flavor perception.

Neurons in the gustatory system also respond to the postingestive effects
of food, as well as the homeostatic state [36] (for a review on peripheral
somal sensory information (e.g., texture, temperature, and
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soma.
Changes in taste perception following bariatric surgery

In studies conducted to test differences in taste perception following bariatric surgery, threshold estimation was also a common outcome, as shown in Table 2. Here, some[24–26,29], but not all studies[27,28,30], revealed an improvement of taste sensitivity after bariatric surgery, but with inconsistent profiles across taste qualities. Specifically, following RYGB, there are reports of lower detection thresholds (i.e. increase in taste sensitivity) for sweet taste, using the constant stimuli method two months after surgery[26], and also for sweet taste, but not for bitter taste, up to 3 months after surgery, using an up-down/ staircase method[25]. The latter study also reported unaltered recognition thresholds for both sweet and bitter taste[25]. However, another study, using the 3-stimulus drop method, reported reduced detection and recognition thresholds up to 3 months after RYGB for sour and bitter tastants, but not for sweet or salt taste[24]. Furthermore, in two studies using the 2-AFC staircase method, and re-assessing patients after surgery at the point when approximately 20% of weight was lost, changes in detection threshold was not found for sweet, salt or umami taste after RYGB or Laparoscopic Adjustable Gastric Banding (LAGB)[27], nor for sweet or salt taste after RYGB or SG[30]. Finally, somewhat consistently with findings of reduced detection thresholds, taste acuity scores for sour, salt, sweet and bitter tastes improved 3 months after SG in one study[29], but remained unaltered 6 months following SG, LAGB or RYGB in another study[28].

Very few studies assessed suprathreshold intensity or hedonic assessments after bariatric surgery. Pepino et al. 2014[27] showed that, in patients treated with RYGB or LAGB, following ~20% of surgically-induced weight-loss, there were no significant changes in suprathreshold intensity ratings (gLMS) for sweet, salt or umami tastants, while changes in preferences for sweet, but not umami, tastants did occur, as measured by a two-series, forced choice tracking procedure[27]. The authors also found that following RYGB, but not LAGB, the hedonic value of sucrose tasting, as measured by the gLHS, changed from pleasant to unpleasant[27]. The same group repeated this experiment in another bariatric group, following either RYGB or SG, confirming no postoperative change in suprathreshold intensity ratings (gLMS) for sweet, salt or umami taste, nor in preference for umami, but with reduction in preference and hedonic scores for sweet taste after...
Studies that compared individuals with obesity with non-obese controls, using direct measures of taste.

| Reference | Participants | Outcome/Method | Methodology | Taste stimuli/Method of application | Results |
|-----------|--------------|----------------|-------------|-------------------------------------|---------|
| Rodin et al., 1976 | Obese, n=16 | Intensity (9-point scale) | Glucose, 0.125-3 M | | - No differences were found among weight category groups in intensity ratings. |
| | Mildly overweight, n=16 | Pleasantness (9-point scale) | Glucose, 0.125-3 M | | - Individuals with obesity or who were mildly overweight rated higher concentrations of sweet more pleasant than normal weight participants. |
| Thompson et al., 1977 | Obese, n=14 | Intensity and pleasantness | Sucrose, 0.075-1.5 M | | - Individuals with obesity did not differ in responses to sucrose vs. normal weight participants. |
| Grinker et al., 1978 | Obese, n=39 | Detection threshold | Sucrose, 0.175% (w/v) vs. water | | - Individuals with obesity did not differ in sucrose detection thresholds vs. normal weight participants. |
| Frijters & Rasmussen-Conrad et al., 1982 | Obese, n=13 | Detection threshold | Sucrose, 0.0060-0.02 M vs. water | | - No differences between overweight vs. normal weight participants were found in any parameter. |
| Scruggs et al., 1994 | Obese, n=6 | Detection and recognition thresholds | HCl, 0.5-5.00 mMvs. water | | - Individuals with obesity did not differ in detection or recognition thresholds vs. normal weight participants. |
| Pasquet et al., 2007 | Severe early onset obesity, n=39 | Recognition threshold | Sucrose and fructose, 2.0-1000 mM | | - Adolescents with obesity had significantly lower recognition thresholds for sucrose and NaCl vs. non-obese participants. |
| | Non-obese, n=48 | Up-down/staircase | Citric acid, 0.05-0.6 g/mL | | - Adolescents with obesity rated sweet and salty tastants as more intense vs. non-obese participants. |
| | Adolescents | Intensity (9-point scale) | NaCl, 32-1000 mM | | - Adolescents with obesity rated the lowest NaCl solution less pleasant, but not sucrose. |
| | | Pleasantness (9-point scale) | Sucrose, 121-970 mM | | - Adolescents with obesity rated the lowest NaCl solution less pleasant, but not sucrose. |
| Pepino et al., 2010 | Obese, n=23 | Detection threshold, 2-AFC staircase | Sucrose and MSG, 1 - 5.6 × 10^-5 M vs. water | | - Women with obesity had significantly higher detection thresholds for MSG but not sucrose, when compared to normal-weight participants. |
| | Normal-weight, n=34 | Intensity (g/LMS) | Sucrose, 0.1-0.5 M | | - Women with obesity did not differ in intensity ratings given to MSG or sucrose vs. normal weight participants. |
| | All women | Preferred concentration | MSG, 0.09-1.05 M | | - Women with obesity preferred higher concentrations of MSG, but not sucrose, when compared to normal-weight participants. |
| Bueter et al., 2011 | Obese, n=9 | Detection thresholds | Sucrose, 2.1-300 mM vs. water | | - Individuals with obesity did not differ in sucrose detection thresholds vs. normal weight participants. |
| Park et al., 2015 | Obese, n=18 | Detection thresholds | NaCl, 0.016-0.9 g/mL | | - Individuals with obesity had significantly higher detection thresholds for NaCl vs. normal weight participants. |
| | Normal-weight, n=23 | Method of limits (derivation) | Quinine HCl, 10^-10 to 0.3 g/mL | | - Individuals with obesity had significantly higher detection thresholds for NaCl vs. normal weight participants. |
| | Young adults | Electrogestometry (EGM) | Cotton swab (whole mouth test) | | - Individuals with obesity had significantly higher EGM thresholds on both sides of the posterior tongue vs. normal weight participants. |
| Holinski et al., 2015 | Obese, n=44 | Taste acuity (multiple-alternative forced-choice paradigm) | Citric acid, 0.05-0.4 g/mL | | - Individuals with obesity had lower overall taste acuity scores vs. non-obese participants. |
| | Non-obese, n=23 | Taste strips | Quinine HCl, 0.0014-0.006 g/mL | | - Individuals with obesity had lower overall taste acuity scores vs. non-obese participants. |
| Pepino et al., 2016 | Obese, n=24 | Intensity (g/LMS) | Sucrose, 0.0-1.05 M | | - Individuals with obesity did not differ in any parameter vs. non-obese participants. |
| | Non-obese, n=20 | Preferred concentration | Sucrose, 0.09-1.05 M | | - Individuals with obesity did not differ in any parameter vs. non-obese participants. |
| Hardikar et al., 2017 | Obesity, n=5 | Recognition thresholds | Sucrose, 0.01-20 g/100 mL | | - Individuals with obesity did not differ in any parameter vs. non-obese participants. |
| | | Up-down/staircase | NaCl, 0.01-5 g/100 mL | | - Individuals with obesity did not differ in any parameter vs. non-obese participants. |
In this review we provide perspective on the data available to support, or contradict, the general interpretation that individuals with obesity have reduced sweet taste perception, as is frequently proposed [9]. This interpretation has typically been framed jointly with the evidence for decreased availability of striatal D2R in individuals with obesity, with both gustatory and dopaminergic factors proposed to contribute towards compensatory food consumption and weight gain [9]. In fact, in flies there is pre-clinical evidence that a high sugar diet results in diminished sweet perception, via decreased response of ‘sweet-taste sensing neurons’ [10]. Furthermore, in healthy human volunteers, reduction of sugar consumption has been shown to result in increased perception of sweet taste intensity, while pleasantness ratings remained unaltered [39]. Finally, a single nucleotide polymorphism, identified in humans, has been associated with higher perceived intensity of several sweet compounds, as well as with consumption of sweet foods [40], which may contribute towards interindividual differences in weight. As is shown here, however, the available studies directly assessing pure taste function of individuals with obesity, either comparing with normal weight control subjects, or assessing changes following bariatric surgery, do not clearly support the general interpretation regarding reduced sweet taste perception in obesity.

Studies comparing sweet taste detection thresholds between participants with and without obesity have found no differences [18, 19, 21, 24, 26]. On the other hand, some [20, 23], but not all studies [24], have described reduced recognition thresholds among individuals with obesity, that suggest enhanced, rather than reduced, sweet taste sensitivity. Inconsistently, after bariatric surgery there is limited evidence of reduced sweet taste detection thresholds [24–27, 30], thus suggesting enhanced sweet taste sensitivity with weight loss, and no evidence of change in recognition thresholds [24, 25]. Regarding suprathreshold sweet taste intensity assessments, while most studies find no obesity-dependent [7, 16–19, 21] or bariatric surgery-dependent [27, 30] effects, reports of increased intensity ratings among individuals with obesity are available in two studies [20, 23]. For other taste qualities, while there is less evidence that is mostly negative, some studies reveal increased detection thresholds [21, 22], reduced recognition thresholds [20, 23] or higher intensity ratings [20, 23] in individuals with obesity, as well as reduced detection [24] or recognition [24] thresholds after bariatric surgery.

Regarding the hedonic dimension of taste, comparisons between individuals with obesity and normal-weight controls were mostly inconclusive, with no evidence of differences in sweet taste among participants with obesity in 4 studies [7, 19–21], nor in salt, bitter or sour tastes in other studies [20, 23]. Other studies, however, revealed increased [16, 23], or reduced [18], hedonic ratings for sweet taste, reduced hedonic ratings for salt tasters [20] and increased preference for umami taste [21], among individuals with obesity. While studies after bariatric surgery are scarce, two of these studies support adjustments of sweet taste perception leading to lower preferences and hedonic scores [27, 30]. However, confidence in the results of available research is limited, given methodological variability between studies, small sample sizes, and lack of adequate controls in longitudinal studies. More studies, with larger patient samples, are needed to address limitations of previous research.

In fact, we have very recently published results of a multicenter longitudinal cohort, recruiting more than 200 bariatric patients, and showing similar variation in suprathreshold intensity and pleasantness ratings of several tasters, including sweet, between those treated with bariatric surgery and a control group awaiting surgery. While our research is consistent with limited overall effects of bariatric surgery on taste, patients with higher sweet intensity ratings before surgery lost more weight, and reduction of sweet intensity ratings correlated with weight loss [43]. These findings, suggesting that associations between taste and bariatric surgery may be better interpreted at the individual, rather than the group level, are consistent with other recent work testing liking ratings for sucrose-sweetened mixtures containing fat, and showing that, in patients receiving RYGB, but not SG, higher preoperative preference for sucrose-sweetened mixtures, as well as activation of the ventral tegmental area by those mixtures, predicted greater weight loss [15]. Hypotheses considering interactions between sweet preferences and dopamine-related brain neural activity may thus be relevant for weight-loss induced by bariatric surgery, and should be further assessed in future research.

### Notes:
Sucrose, glucose and fructose are tastants for sweet taste; sodium chloride (NaCl) for salty, citric acid and hydrochloric acid (HCl) for sour, quinine hydrochloride (HCl) and 6-N-propylthiouracil (PROP) for bitter and monosodium glutamate (MSG) for umami.

### Abbreviations:
2-AFC staircase - Two-alternative forced-choice staircase procedure; gLMS - general labeled magnitude scale; VAS - Visual Analogue Scale.

### Table 1 (continued)

| Reference | Participants | Methodology | Results |
|-----------|--------------|-------------|---------|
|           |              | Tastant stimuli/Method of application |         |
| Obese, n=23 | Normalweight, n=31 | Quinine HCl, 0.0001–0.025 g/100 mL Citric acid, 0.001–0.9 g/100 mL | Spray dispenser (0.2 mL bolus of each tastant administered in the anterior part of the tongue). |
| Intensity (VAS) | | Sucrose, 0.01–0.20 g/100 mL NaCl, 0.01–5 g/100 mL Quinine HCl, 0.0001–0.025 g/100 mL Citric acid, 0.001–0.9 g/100 mL | Spray dispenser (0.2 mL bolus of each tastant administered in the anterior part of the tongue). |
| Pleasantness (VAS) | | Sucrose, 0.01–0.20 g/100 mL NaCl, 0.01–5 g/100 mL Quinine HCl, 0.0001–0.025 g/100 mL Citric acid, 0.001–0.9 g/100 mL | Spray dispenser (0.2 mL bolus of each tastant administered in the anterior part of the tongue). |

- Individuals with obesity had significantly lower recognition thresholds for sucrose and NaCl vs. normal weight participants.
- Individuals with obesity rated the lower concentrations of sucrose, NaCl and citric acid as more intense vs. normal weight participants.
- Individuals with obesity rated one of the higher concentrations of sucrose as more pleasant vs. normal weight participants.
Studies that followed individuals with obesity before and after bariatric surgery using direct measures of taste.

| Reference | Participants | Surgery/Follow-up | Methodology | Taste stimuli/Method of application | Results |
|-----------|--------------|-------------------|-------------|-------------------------------------|---------|
| Scruggs et al., 1994 [24] | Obese, n=6 | RYGB Pre, 1, 2 and 3 months postoperatively. | Detection/Recognition thresholds 3-stimulus drop | HCL, 0.5-500 mM vs. water Urea, 90-5000 mM vs. water Sucrose, 6-5.8000 mM vs. water NaCl, 6.100 mM vs. water Calibrated drops of the taste solutions/water were placed on the tongue at identical locations. | - Following surgery there was a decrease in detection/recognition thresholds for HCL and urea, but not for sucrose or NaCl. |
| Burge et al., 1995 [25] | Obese, n=9 | RYGB Pre, 1.5Sand 3months postoperatively. | Detection/Recognition thresholds Up-down/staircase | Sucrose, 0.01-0.1 mol/L Urea, 0.01-0.5 mol/L Sucrose, 0.1-0.3 mol/L Urea, 0.1-1.0 mol/L | - Following 1.5 months after surgery, detection thresholds for sucrose significantly decreased and remained so at 3 months. |
| Bueter et al., 2011 [26] | Normal weight, n=9 | RYGB 1-week pre and 2 months postoperatively. | 'Just about right' concentration (200 mm VAS) | Sucrose, 0-400 mM | - Following surgery, patients had decreased detection thresholds for the lowest sucrose concentrations vs. controls. |
| Pepino et al., 2014 [27] | Obese, n=27 - RYGB, n=17 - LAGB, n=10 | Before surgery and after ~20% surgically-induced weight loss | Detection thresholds 2-AFC staircase | | - Following surgery there were no changes in the "just about right" concentration of sucrose vs. controls. |
| Holinski et al., 2015 [28] | Obese, n=44 - Pre-SG, n=37 - Pre-LAGB, n=4 - Pre-RYGB, n=3 Non-obese, n=23 | LAGB Pre, 0.5, 0.75 and 6 months postoperatively. | Preferred concentration Two series, forced choice tracking procedure | Sucrose 24% w/v | - Following surgery, both in RYGB and LAGB groups lower concentrations of sucrose were preferred. |
| Altun et al., 2016 [29] | Obese, n=52 | SG Pre, 1 and 3 months postoperatively. | Tastee acuity (multiple alternative forced-choice paradigm) Taste strips Burghart Messtechnik GmbH, Wedel, Germany Strips were applied to the midline of the anterior third of the tongue. | Citric acid, 0.05-0.3 g/ml NaCl, 0.016-0.25 g/ml Quinine HCl, 0.0004-0.006 g/ml | - Following surgery, but not LAGB, the hedonic value of sucrose (gLHS) changed from pleasant to unpleasant. - Six months after surgery, taste acuity was not significantly different from controls. |
| Nance et al., 2017 [30] | Obese, n=31 - Pre-RYGB, n=23 - Pre-SG, n=8 | RYGB Before surgery and after ~20% surgically-induced weight loss | Detection thresholds 2-AFC staircase | Sucrose, glucose and NaCl, 1 × 10−3 M vs. water | - There was no change in detection thresholds after RYGf or SG. |

Notes: Sucrose, and glucose are tastants for sweet taste; sodium chloride (NaCl) for salty, quinine hydrochloride and urea for bitter; citric acid and hydrochloric acid (HCl) for sour and monosodium glutamate (MSG) for umami.

Abbreviations: 2-AFC staircase - Two-alternative forced-choice staircase procedure; gLHS - general labeled hedonic scale; gLMS - general labeled magnitude scale; LAGB - Laparoscopic Adjustable Gastric Banding; RYGB - Roux-en-Y Gastric Bypass; SG - Sleeve Gastrectomy; VAS - Visual Analogue Scale.
7. Conclusions

Available research suggests that changes of sweet taste with obesity and weight-loss may be more complex than simply considering decreased sweet taste perception. However, there are several indications that sweet taste may be related to weight and, importantly, that it may be a useful marker in the context of bariatric surgery, suggesting the need for further research with refined methods and large sample sizes.

Declaration of Competing Interest

Competing interests are not reported by Ribeiro. Oliveira-Maia is recipient of a grant from Schuhfried GmbH for norming and validation of cognitive tests, and national coordinator for Portugal of a Non-interventional Study (EDMS-ERI-143085581, 4.0) to characterize a Treatment-Resistant Depression Cohort in Europe, sponsored by Janssen-Cilag Ltd, and of a trial of psilocybin therapy for treatment-resistant depression, sponsored by Compass Pathways, Ltd (EuDaCT Number: 2017–003288–36).

Authors Contributions

Ribeiro conducted the literature search. Ribeiro and Oliveira-Maia wrote the manuscript and approved the final version.

Funding/Support

Oliveira-Maia was supported by grants from the BIAL Foundation (176/10), and from Fundação para a Ciência e Tecnologia (FCT) through a Junior Research and Career Development Award from the Harvard Medical School Postdoctoral Program (HMSP/ICI/2020/2011) and grant PTDC-MED/NEU/31331/2017; and is funded by a Starting Grant from the European Research Council (ERC) under the European Union’s Horizon 2020 research and innovation programme (grant agreement No 950357). Ribeiro was funded by doctoral fellowships from Universidade de Lisboa (BD/2015Call) and FCT (SRH/BD/128783/2017).

Role of the Funder/Sponsor

The funding sources did not participate in interpretation of the data, preparation or review of the manuscript.

Supplementary materials

Supplementary material associated with this article can be found, in the online version, at doi:10.1016/j.ejim.2021.01.023.

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[14] Ribeiro conducted the literature search. Ribeiro and Oliveira-Maia wrote the manuscript and approved the final version.

Funding/Support

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