



Review Article

Cite this article: Shanmugamprema D, Muthuswamy K, and Subramaniam S (2024). Emerging perspectives: the interplay of taste perception and oral microbiota composition in dietary preferences and obesity. *Nutrition Research Reviews*, page 1 of 10. doi: [10.1017/S0954422424000295](https://doi.org/10.1017/S0954422424000295)

Received: 12 June 2024
Revised: 15 October 2024
Accepted: 16 October 2024

Keywords:

microbial diversity; obesity; oral microbiota; taste buds; taste perception; taste receptors

Abbreviations:

BMI, body mass index; GPCR, G protein-coupled receptor; T2R, taste receptor family 2 member; PROP, 6-*n*-propylthiouracil; cAMP, cyclic adenosine monophosphate; PLCB2, phospholipase C beta 2; IP3, inositol 1,4,5-trisphosphate; TRPM5, transient receptor potential cation channel member 5; TBC, taste bud cells; CVP, circumvallate papillae; LCFA, long-chain fatty acids; ATP, adenosine triphosphate; CD36, cluster of differentiation 36; GPCR, G protein-coupled receptor; NCD, non-communicable diseases; LPS, lipopolysaccharides; TNF α , tumour necrosis factor alpha; SNP, single-nucleotide polymorphisms; ENaC, epithelial sodium channels; OTOP1, otopetrin 1; ST, supertasters; NT, non-tasters

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Emerging perspectives: the interplay of taste perception and oral microbiota composition in dietary preferences and obesity

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Abstract

Recent advancements in sensory research have brought to light the intricate relationship between taste perception and the oral microbiota, prompting investigations into their influence on human health, particularly in the context of dietary preferences and obesity. This review aims to update the current understanding of how oral microbiota influence taste perception and dietary choices, elucidating shared metabolic pathways between food processing and oral bacteria. Further, this review outlines the mechanisms underlying taste perception, emphasising the role of taste receptors and taste buds in shaping sensory experiences influenced by genetic and environmental factors. Notably, we explore the bidirectional relationship between oral microbiota and taste sensitivity, highlighting the potential impact of microbial composition on taste perception thresholds and implications for dietary habits and health outcomes, such as obesity and dental caries. However, significant research gaps remain, particularly in the understanding of the molecular mechanisms linking oral microbiota with taste sensitivity, as well as the long-term effects of microbiota-targeted interventions. Future research should focus on longitudinal studies and experimental interventions to explore these connections more deeply, offering insights into potential strategies for promoting healthier dietary behaviours and managing diet-related non-communicable diseases.

Introduction

The significance of food choices for human health is irrefutable, and recent strides in sensory research have unveiled the intricate interplay between taste perception and the oral microbiota.^(1,2) While much attention has been devoted to understanding the role of gut microbiota in health and disease, the potential influence of oral bacteria on conditions such as obesity has emerged as a promising area of investigation.^(3–5) Obesity, a multifaceted condition influenced by genetic, environmental and behavioural factors, including dietary habits, is intricately linked with taste sensitivity, which is shaped by genetic predispositions.^(6,7) Emerging evidence hints at variations in salivary bacterial profiles between individuals with divergent adiposity levels, suggesting a potential link between certain microbial taxa and obesity markers.⁽⁸⁾ Recent studies have shown a relationship between taste sensitivity and body mass index (BMI), with higher BMI being correlated with reduced taste sensitivity.^(9,10) Given the well-established connection between obesity and chronic inflammation or meta-inflammation, there is a growing interest in elucidating the contribution of oral microbiota in this inflammatory milieu.^(11–13)

The sensory experience of food consumption, shaped by taste perception, profoundly influences food acceptability and consumption patterns. Recent investigations have unveiled the role of enzymatic degradation of taste compounds in modulating taste perception, underscoring the impact of salivary disorders on food pleasure and intake.^(14–16) Furthermore, research exploring the influence of oral microbiota on taste perception and food choices has shed light on how specific microorganisms generate fragrant molecules in the mouth, thereby shaping food perception.^(1,17,18) These findings suggest that variations in the microbial composition of the oral cavity may contribute to variations in perception, offering novel insights into the intricate relationship between oral microbiota and sensory experiences. While research has explored differences in taste sensitivity, particularly in the perception of bitterness, between obese and non-obese individuals, investigations into other taste qualities and their relationship to obesity, especially in children, remain scarce and inconclusive. Moreover, food neophobia, characterised by a reluctance to try new foods, may contribute to obesity by limiting dietary diversity to energy-dense options.⁽¹⁹⁾ Additionally, emerging evidence suggests a potential role of oral

microbiota in both obesity development and taste perception, highlighting the need for holistic approaches to address these interconnected factors. This growing field not only promises insights into the mechanisms underlying food preferences, but it also has the potential to update strategies for supporting healthier dietary behaviours.^(2,3,20)

The current review aims to delineate the existing literature on the relationships between oral microbiota and taste perception, and to identify and discuss shared metabolic pathways between food processing strains and oral bacteria. Essentially, we are revealing the significant effects of these elements on human health and wellness as we investigate the molecular mechanisms behind taste perception and the function of oral bacteria in taste modulation. Furthermore, we attempt to address important gaps in our understanding by offering a concise summary of the most recent research on oral microbiome and taste perception.

Understanding taste perception: a multifaceted process

Taste perception serves as a cornerstone in shaping food preferences and aversions. This complex process involves the interaction of several modalities, including vision, audition, kinesthesia and taste perception. Among these, taste stands out as a fundamental driver, with well-understood mechanisms governing basic taste perception.^(21–24) Food-derived compounds activate taste receptors located on the tongue, soft palate, pharynx and gut, stimulating neuronal fibres that transmit signals to the brain. These signals are then processed into sensory experiences encompassing taste quality, intensity and hedonics.^(25,26)

Taste receptors serve as crucial chemosensory receptors found in taste buds and various extra-gustatory tissues.^(24,27) These receptors play critical role in distinguishing five principal tastes: sweet, salt, sour, umami and bitter. Recent studies suggest the existence of potential new basic tastes, such as kokumi and fat (oleogustus).^(28–31) Taste receptors, which belong to the G protein-coupled receptor (GPCR) superfamily and some ionic channels, exhibit different specificities to taste stimuli. Bitter, sweet and umami taste signals are thought to converge on a common intracellular signalling transduction pathway in type II cells.^(32,33) These tastes are activated by GPCR: taste receptor family 1 member (T1R) for sweet and umami stimuli, and taste receptor family 2 member (T2R) for bitter compounds. Upon activation by corresponding stimuli, the G protein coupled to these taste receptors is resolved into G $\beta\gamma$ subunit and G α subunit, which includes G α -gustducin, G α 14 and G α i.^(34,35)

Taste buds, the sensory organs responsible for transmitting taste sensations, are distributed across various locations in the oral cavity, with the highest concentration on the tongue, particularly within three types of papillae: fungiform, foliate and circumvallate. While taste buds are also present in other areas such as the soft palate, epiglottis, larynx and nasopharynx, research in these regions remains limited.^(36,37) Type I cells are predominant and play a supporting role, potentially involved in salt taste perception. Type II cells act as receptor cells, housing receptors for sweet, bitter and umami tastes, while type III cells are considered output cells, responsible for transmitting taste information to the afferent nerve.^(38,39) Despite ongoing research, the exact intercellular signalling within taste buds remains poorly understood. Current models propose that type II cells communicate with type III cells via purinergic signalling mechanisms, and impairment in this signalling leads to diminished gustatory function. The gustatory sensory system, encompassing taste buds, their innervations and

the associated papillae, constitutes the taste system. Although resembling somatosensory and pain pathways more closely than olfaction, gustatory afferent neurons share similarities with olfactory sensory cells, including identical transmission processes and turnover of sensory and supporting cells.^(40–43) Unlike olfactory sensory neurons, which project directly into the brain through nerve fibre bundles called olfactory fila, gustatory sensory neurons first form synapses with taste bud receptor cells before projecting into the brain.

Individual variability in taste perception

Food preferences play a crucial role in determining food intake patterns and are heavily influenced by taste perception and preference. Taste is a fundamental aspect of food choice, as highlighted in the Food Choice Process Model, which identifies taste among the five main values guiding food decisions.^(44,45) Sensory perceptions, including taste sensitivity, vary significantly among individuals and are partly explained by genetic variations in taste perception genes. Genetic factors contribute to individual differences in taste perception and preference, with taste sensitivity and food preferences showing moderate to high heritability estimates.^(46,47) For example, bitter taste sensitivity, as assessed by compounds such as PROP and quinine hydrochloride, has been found to have high heritability estimates, while sweet taste sensitivity shows moderate heritability. Similarly, food preferences for items such as dessert foods, vegetables, fruits and protein foods also exhibit heritability, indicating the influence of genetic factors on taste-related choices.⁽⁴⁸⁾

Significant variability exists in taste perception among individuals, attributable in part to differences in taste receptor expression, abundance and salivary flow rate. Genetic predisposition, life stage and eating behaviour further contribute to taste sensitivity disparities. Age, sex and ethnicity contribute to variability in both oral microbiota composition and taste perception. For example, ageing is associated with reduced salivary flow and changes in oral microbiota diversity, which may lead to decreased taste sensitivity.^(49,50) Women, due to hormonal fluctuations, often exhibit higher sensitivity to certain tastes such as bitterness and sweetness compared with men. Ethnic differences in dietary habits and genetic backgrounds further contribute to the diversity in taste perception and microbial composition.⁽⁵⁰⁾ Single-nucleotide polymorphisms of taste receptors have been implicated in influencing taste perception and food preferences. Notably, lower sensitivity to a taste often correlates with increased preference for that taste. For instance, individuals categorised as supertasters exhibit heightened responsiveness to bitterness, influencing their dietary choices.^(49–52) Activation of bitter and umami receptors reduces intracellular cAMP levels by inhibiting protein kinase A through the activity of the G α subunit. This inhibition of protein kinase A weakens its inhibition on the PLC β 2–IP3 pathway and an increase in Ca²⁺ release from the endoplasmic reticulum.^(53–55) Conversely, activation of sweet receptors increases intracellular cAMP levels through the G α subunit, enhancing protein kinase A activity and inhibiting K⁺ channels, thereby promoting extracellular Ca²⁺ influx. Elevated intracellular Ca²⁺ levels subsequently activate transient receptor potential cation channel subfamily member 5 (TRPM5), an ion channel that induces membrane depolarisation, leading to action potential generation and ATP release.^(56,57)

Food preferences develop during fetal development and evolve over time, shaped by interactions between genetic predispositions

and environmental factors. Early experiences, parental feeding practices, family dynamics and broader environmental contexts all play a role in shaping taste preferences and dietary habits.^(58,59) The sensory properties of food, including taste, are key determinants of dietary patterns, with taste receptors responding to specific compounds in food. These receptors, which comprise G-protein-coupled receptors for sweet, umami and bitter tastes and ion channels for salt and sour tastes, mediate taste perception.^(60,61) The finding that the fatty acid transporter *CD36* is associated with the fat taste modality has expanded our understanding of taste perception.⁽⁶²⁾ Understanding the genetic basis of taste perception and its association with dietary patterns and health outcomes can inform public health strategies aimed at preventing diet-related non-communicable diseases (NCD). Evolutionary mechanisms may have shaped human taste abilities to avoid the consumption of plant-based toxins, which are often associated with bitter tastes. Moreover, single-nucleotide polymorphisms (SNP) in genes such as *TAS2R38* have been shown to significantly influence the perception of bitter taste. These genetic variations not only alter taste receptor expression but also modulate oral microbiota composition, particularly the abundance of Actinobacteria and Bacteroidetes, which interact with taste receptors.^(46,63)

Exploring the role of oral microbiota

The oral microbiota has received interest in healthy individuals, especially with regard to sensory perception, despite being traditionally investigated in the context of oral diseases. The oral microbiome encompasses various niches within the oral cavity, each characterised by unique environmental factors that shape microbial colonisation and community structure.^(64–66) Saliva, in particular, serves as a major reservoir of oral bacteria, reflecting microbial composition on the tongue dorsum. While certain genera, such as *Streptococcus*, *Actinomyces* and *Veillonella*, are frequently identified in the core oral microbiome, the overall composition and diversity of the oral microbiome exhibit considerable variability across individuals and geographic locations.^(65–68) Host-related factors, including personal hygiene, diet, genetics and obesity, further influence oral microbiome composition and dynamics.⁽⁶⁹⁾

The mouth is home to a bustling community of over 700 different microorganisms, including bacteria, fungi, parasites and viruses. Together, they form what is known as the oral microbiota.⁽⁷⁰⁾ These tiny residents settle in various spots within the mouth, such as the tongue, gums and teeth. Some thrive in areas with little oxygen, such as below the gum line. Teeth, in particular, become host to complex biofilms made up of multiple microbial species. These biofilms foster interactions between different types of microorganisms. It is worth noting that fungi, such as *Candida* and others, also play a role in this ecosystem, although our understanding of their impact is still evolving. Within this microbial melting pot, bacteria and fungi interact, and the overall balance can shift with changes in diet and oral hygiene habits. Despite these fluctuations, there is a certain level of consistency in the oral microbiota over time. As we age, external factors continue to shape the composition of our oral microbiota.^(71,72) In infancy, species such as *Streptococcus* and *Lactobacillus* dominate, with limited biofilm formation due to the absence of teeth. As teeth emerge and dietary habits change, the microbiota diversifies, influenced by factors such as breastfeeding and early antibiotic use, which can have long-lasting effects on its development.⁽⁷³⁾

Recent studies have highlighted significant differences in oral microbiome composition between individuals of normal weight and those with obesity. Specifically, obese individuals exhibit microbial signatures in the oral cavity that closely resemble those associated with obesity in the gut.^(74,75) These findings underscore the potential involvement of oral bacteria in pathways leading to obesity and metabolic dysfunction. Evidence suggests that inflammation plays a crucial role in the metabolic disturbances associated with obesity. Dysbiosis of the gut microbiome has been implicated in inflammation and obesity, prompting investigations into the role of the oral microbiome in similar pathways.^(76,77) Studies have highlighted a potential link between oral bacteria and adipose tissue inflammation. For instance, periodontitis-associated systemic inflammation was found to up-regulate inflammatory markers in adipose tissue and liver, exacerbating obesity-related inflammation.^(78,79) Experimental models have further demonstrated that oral bacteria, particularly *Porphyromonas gingivalis*, can induce local white adipose tissue inflammation and systemic insulin resistance, potentially contributing to metabolic disease progression.⁽⁸⁰⁾ Oral microbiota composition may also affect food cravings by modulating taste sensitivity and the hedonic value of foods. Specific strains of *Lactobacillus* and *Bifidobacterium* have been associated with reduced cravings for sugary foods, while an imbalance in microbial diversity may lead to heightened cravings for unhealthy options (Table 1). Furthermore, microbial metabolites, short-chain fatty acids (SCFA), are produced during the fermentation of dietary fibres by gut bacteria and can modulate the release of appetite-regulating hormones such as ghrelin and leptin. This interaction creates a feedback loop where the presence of certain microbial communities may increase cravings for specific food types, particularly those high in sugar and fat, which can perpetuate unhealthy eating habits.

Salivary metabolites, originating from both oral microbiota and host enzymatic activity, influence taste and smell perception through various mechanisms involving the production of flavour-active compounds, which affect the perception threshold and metabolism of food compounds into sensory-active molecules.^(81,82) Additionally, the conversion of taste and smell molecules into new compounds without chemosensory properties alters the quantity of flavour compounds. Acetate and propionate, two common salivary metabolites, are SCFA that are primarily produced by oral bacteria.⁽⁸³⁾ Their concentrations correlate with bacterial load and can influence the perception threshold of NEFA, which interact with both *CD36* and *GPR120* receptors, modulating the sensory perception of fat. Amino acids such as glutamate, a key component in umami taste, are also produced by oral bacteria, impacting the *TIR1/TIR3* receptor, enhancing umami taste perception.

Microbial enzymes also impact olfactory perception by altering the amplitude and kinetics of odorant responses.⁽⁸⁴⁾ Odorant metabolism by microbial enzymes can generate new odorants in the oral cavity, contributing to flavour perception. Glycoside-derived aroma compounds, mainly produced by bacterial enzymes, highlight the role of the oral microbiome in modulating aroma compound formation and perception.⁽⁸⁵⁾ Microbes can regulate the expression of taste receptor genes, influencing host nutrient detection and taste sensitivity. Germ-free mice exhibit altered expression of taste receptors, leading to increased oral nutrient detection.^(86,87) Certain oral bacteria, such as *Streptococcus mutans*, may impact sweet taste sensitivity, potentially affecting dietary choices and oral microbiota composition. Moreover, bacterial lipopolysaccharides induce cytokine production, reducing taste receptor cell numbers and altering taste perception.^(88,89) Understanding these

Table 1. Microbial impact on taste perception

Microbe	Impact on taste perception	Reference
<i>Lactobacillus</i> and <i>Bifidobacterium</i>	Modulation of gut taste receptors, promotion of neurotransmitter production	(137,138)
<i>Streptococcus mutans</i>	Dental cavities may affect taste perception due to damage to oral structures	(139,140)
<i>Helicobacter pylori</i>	Gastritis and ulcers may lead to changes in taste perception and appetite	(141,142)
<i>Candida albicans</i>	Oral thrush can cause bitter or metallic taste, discomfort affecting food sense	(143)
<i>Staphylococcus</i> and <i>Streptococcus</i>	Dampen sweet, salty, sour and bitter and form physical barrier through microbial tongue film	(144,145)
<i>Salmonella</i> and <i>Campylobacter</i>	Foodborne illness symptoms such as nausea and diarrhoea can disrupt taste perception temporarily	(146)
<i>Actinobacteria</i> and <i>Bacteroidetes</i>	Increase taste sensitivity; bacteria produce metabolites precursors of some bitter acids	(90)
<i>Propionibacterium freudenreichii</i>	Production of flavour compounds influencing taste, as in Swiss cheese	(147)
<i>Actinomyces</i> and <i>Oribacterium</i>	Associated with high responsiveness to bitter	(2)
<i>Saccharomyces cerevisiae</i>	Contribution to flavour and aroma in bread, beer and wine through fermentation	(148,149)
<i>Bacillus cereus</i>	Toxins causing foodborne illness can disrupt taste perception and appetite	(150,151)
<i>Clostridia</i>	Associated with protein/fat-rich diets and negatively associated with fibre intake	(152,153)
<i>Escherichia coli</i>	Pathogenic strains can cause foodborne illness, leading to temporary changes in taste	(154)
Bacteroidetes, Bacterolidia	Associated with decreased perception of all tastes in obese children	(155)
<i>Clostridium botulinum</i>	Toxin production in food can lead to paralysis and affect taste perception and appetite	(156,157)
<i>Pseudomonas</i> spp.	Spoilage of food products may result in off-flavours and odours, affecting taste perception	(158,159)

common pathways sheds light on the intricate relationship between oral microbiota and flavour perception, offering insights into aroma modulation in both fermented products and the oral cavity.

Influence of oral microbiota on dietary preferences

Recent studies suggest a bidirectional relationship between the oral microbiota and dietary preferences, indicating that microbes may influence taste sensitivity and, in turn, dietary choices.^(90–92) Investigations in healthy individuals categorised as supertasters or non-tasters revealed intriguing associations between taste

perception, microbial composition and dietary habits. Supertasters exhibited lower taste detection thresholds and higher density of certain bacteria on the tongue, suggesting a potential link between microbial abundance and taste sensitivity.⁽²⁾ Moreover, correlations were observed between bacterial profiles, taste recognition thresholds and dietary intake, shedding light on the intricate interplay between oral microbiota and food preferences.^(17,90) For example, individuals with altered taste sensitivity may exhibit preferences for certain types of foods, such as sweets or fatty foods, leading to imbalanced diets and potential health consequences. Understanding these associations can inform strategies for promoting healthier dietary habits and managing conditions such as obesity and dental caries.

The reduction in taste bud numbers has been observed in individuals with obesity, and inflammatory processes are implicated as the underlying cause. Studies have shown that inflammatory stimuli, such as lipopolysaccharides (LPS), can inhibit the proliferation of progenitor cells in taste buds, leading to a decrease in taste cell turnover.^(93,94) Elevated levels of tumour necrosis factor alpha (TNF α) associated with low-grade inflammation in obesity have been linked to a lower abundance of taste buds. Interestingly, using TNF α -deficient mice, researchers found no changes in taste bud numbers after obesity induction, suggesting that taste bud loss may be a consequence rather than a cause of obesity in these animals.^(95,96) The exact mechanisms by which changes in the oral microbiome affect TNF α levels or contribute to the observed taste bud alterations require further investigation.

Recent studies have focused on how taste receptors engage in immune responses and interact with oral microbiota, providing insights into their role in oral diseases. The ability to perceive sweetness is governed by the *T1R2/T1R3* heterodimer receptor. Genetic variations in *T1R2* and *T1R3* genes influence thresholds for sweet taste perception and food preferences.⁽⁹⁾ These genetic differences can lead to varying levels of sugar intake, impacting the risk of metabolic disorders and oral diseases.^(97,98) Current studies also have demonstrated that SNP in *CD36*, a gene implicated in fat taste perception, influence the interaction between oral microbiota and taste receptors, particularly through microbial metabolites such as SCFA. These variations can alter individual sensitivity to dietary fats, which is further modulated by the composition of oral bacteria. Similarly, SNP in *T1R2* and *T1R3* influence sweet taste perception, with oral microbial composition playing a critical role in mediating taste sensitivity.^(9,46) (Fig. 1).

Research by Kulkarni *et al.* (2013)⁽⁹⁹⁾ and Haznedaroglu *et al.* (2015)⁽¹⁰⁰⁾ revealed a correlation between the *Ile191Val* polymorphism and sugar consumption in relation to dental caries risk. Their findings indicate that individuals with this polymorphism tend to consume less sugar and have a reduced likelihood of developing dental caries. Moreover, increased sugar intake associated with *T1R2/T1R3* SNP can alter the oral microenvironment, affecting the composition of oral microflora and increasing the risk of caries. Research has demonstrated associations between *T2R38* SNP and dental caries risk, with 'supertasters' exhibiting a lower risk of caries. Additionally, other bitter taste receptors, such as *T2R14* and *CA6*, have been implicated in interactions with oral flora and the risk of dental caries.^(101,102) The impact of SNP in other taste receptors, such as ENaC and OTOP1, on taste perception and susceptibility to oral diseases requires further investigation.

Many oral health issues arise from an imbalance in bacterial communities, leading to the buildup of harmful biofilms,

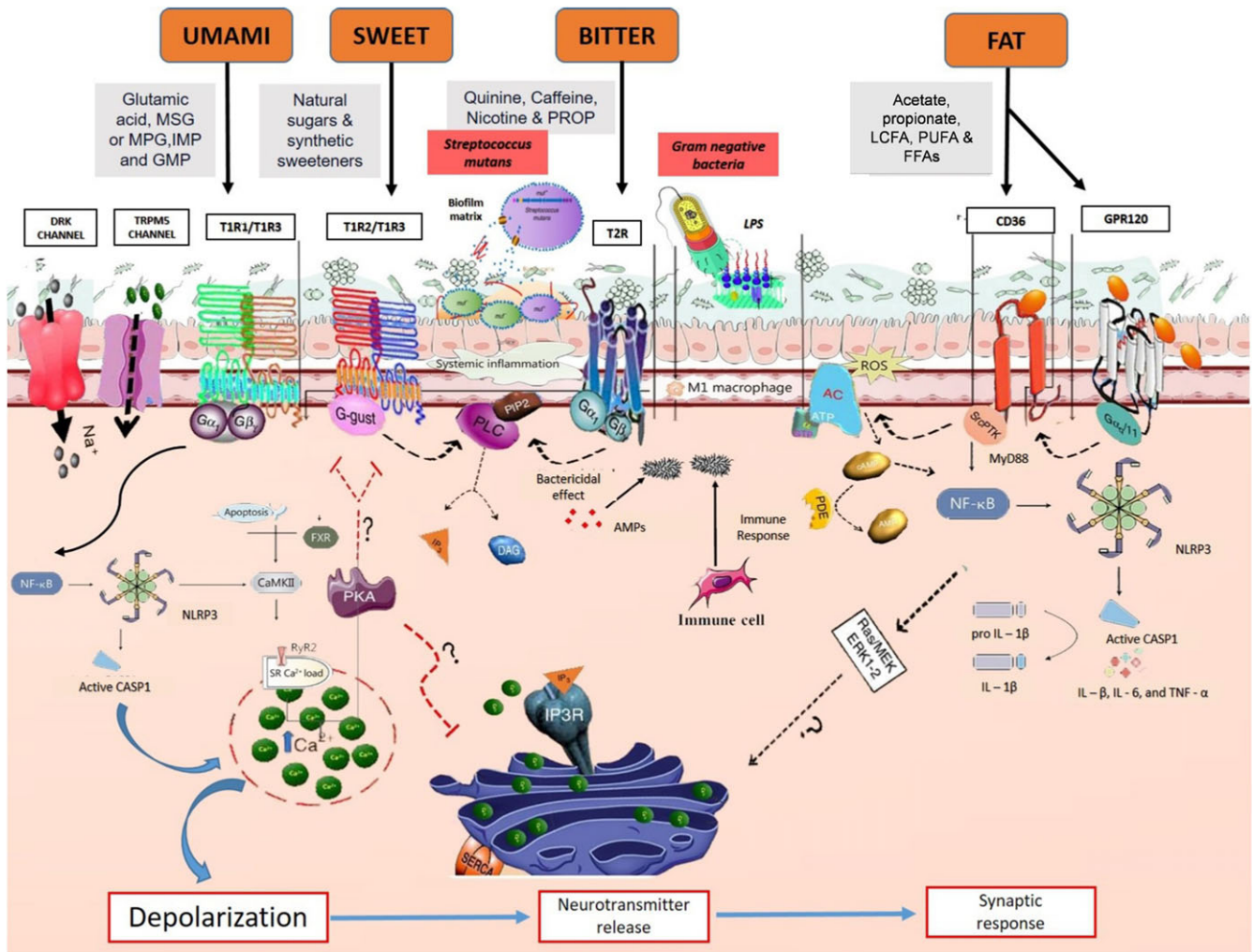


Figure 1. Oral microbiome and metabolite-mediated molecular pathways: interplay with taste receptors, bacterial stimuli and oral immune responses in taste modulation. Taste perception involves the activation of G-protein coupled receptors (GPCR) in taste receptor cells. Bitter taste is detected by *T2R* receptors, while sweet and umami tastes are detected by *T1R1/T1R2/T1R3* receptors. Upon stimulation of these receptors, the downstream $G\beta\gamma$ complex is activated, initiating a cascade of intracellular events. First, phospholipase C isoform $\beta 2$ ($PLC\beta 2$) is activated, leading to the production of inositol 1,4,5-trisphosphate (IP_3). IP_3 then triggers the IP_3 receptors (IP_3R) on the endoplasmic reticulum (ER), causing the release of calcium ions (Ca^{2+}) into the cytoplasm. This increase in intracellular calcium activates transient receptor potential cation channel subfamily M member 5 ($TRPM5$), resulting in sodium ion (Na^+) influx and cell depolarisation. Depolarisation activates calcium homeostasis modulator 1 ($CALHM1$) channels, leading to the release of adenosine triphosphate (ATP) as a neurotransmitter. Furthermore, bacterial stimuli, such as *S. mutans* competence stimulating peptide-1 (CSP-1) and lipopolysaccharides (LPS), can modulate taste receptor signalling pathways and trigger immune responses. CSP-1 primarily activates the $G\beta\gamma$ - $PLC\beta$ pathway, leading to intracellular calcium mobilisation and secretion of cytokines/chemokines, while LPS induces adenylate cyclase (AC) activity, elevating intracellular cyclic adenosine monophosphate (cAMP) levels. This, in turn, activates the $NF-\kappa B$ pathway, promoting the release of pro-inflammatory cytokines. Activation of taste receptors by bacterial metabolites can also stimulate the release of antimicrobial peptides (AMP) from epithelial cells and recruit immune cells to modulate the oral immune response. Additionally, oral flora contributes to M1 macrophage polarisation and modulation of NLRP3 inflammasome activation. SCFA attenuate NLRP3 signalling, preventing disruption of calcium handling and NLRP3 activation.

commonly referred to as dental plaques.⁽¹⁰³⁾ Dental caries, or tooth decay, stem from excessive sugar consumption, fostering the growth of acid-producing bacteria such as *S. mutans* that erode tooth enamel.⁽¹⁰⁴⁾ Halitosis, commonly known as bad breath, results from the proliferation of bacteria on the tongue's surface, particularly those producing volatile sulphur compounds such as *Porphyromonas* and *Fusobacterium*.⁽¹⁰⁵⁾ Beyond these oral afflictions, xerostomia, often associated with conditions such as Sjogren's syndrome, leads to dry mouth sensation due to altered salivary gland function. This condition predisposes individuals to infections from non-oral bacteria, likely due to decreased saliva flow and compromised immune protection.⁽¹⁰⁶⁾ As saliva plays a crucial role in oral health maintenance, its diminished presence exacerbates these disorders.

The microbial community in saliva mirrors that of the oral mucosa and tongue,⁽¹⁰⁷⁾ encompassing over 2000 bacterial proteins across 50 bacterial genera.⁽¹⁰⁸⁾ Environmental factors wield greater influence over microbiota composition than host genetics, with familial upbringing imprinting a lasting mark on salivary microbiome composition over years.⁽¹⁰⁹⁾ Some studies advocate specific diets to deter periodontitis by curbing the proliferation of pathogenic anaerobes.⁽¹¹⁰⁾ Consumption of tea-rich diets amplifies oral microbial diversity, favouring genera such as *Fusobacteriales* and *Clostridiales*.⁽¹¹¹⁾ Conversely, oolong tea consumption diminishes microbial diversity, targeting species such as *Streptococcus* sp., *Prevotella nanceiensis* and *Fusobacterium periodonticum*,⁽¹¹²⁾ owing to antimicrobial flavan-3-ol compounds such as epigallocatechin gallate found in tea.⁽¹¹³⁾

Unravelling the nexus between oral microbiota and taste sensitivity

Among the various niches within the human body, the oral cavity emerges as a focal point, teeming with a bacterial populace nearly equivalent in number to the human cellular constituents, second only to the gastrointestinal tract.⁽¹¹⁴⁾ While these microbial ensembles typically navigate daily challenges with remarkable resilience, disturbances such as frequent sugar intake or compromised immune defences can precipitate dysbiosis, disrupting their delicate regulatory sway over the host. The build-up of oral bacteria on the tongue forms a film that acts as a physical barrier, impeding tastants from accessing taste receptors.⁽¹¹⁵⁾ Studies reveal that individuals with tongue coating often exhibit diminished taste sensitivity across various taste modalities. Notably, the removal of such coating, whether through brushing or scraping, correlates with enhanced taste perception, underscoring the pivotal role of oral bacteria in taste modulation.⁽¹¹⁶⁾

Beyond physical obstruction, oral bacteria contribute to taste perception through the production of metabolites that wield multifaceted effects (Fig. 1). These metabolites can modulate taste perception thresholds, directly activate taste receptors or even degrade flavour compounds, thereby influencing their interaction with taste receptors.⁽⁹²⁾ Consequently, variations in oral bacterial metabolism manifest as differences in taste sensitivity, highlighting the intricate interplay between oral microbiota composition and taste perception.⁽²⁾ Additionally, taste receptors, beyond their canonical role in sensing chemical stimuli, also serve as sentinels for microbial detection, orchestrating immune responses that contribute to oral microbial homeostasis.⁽⁵⁵⁾ Recent investigations into taste perception have spotlighted the PROP phenotype as a pivotal factor influencing individual taste sensitivity and food preferences. Studies have consistently shown that supertasters (ST) rate certain tastes – such as bitterness of caffeine, sweetness of sucrose, saltiness of sodium chloride and sourness of citric acid – as more intense compared with non-tasters (NT). Moreover, our recent report suggests a correlation between the higher density of fungiform papillae (FPD) on ST and their heightened sensitivity.⁽¹¹⁷⁾ While initial hypotheses proposed a connection between higher FPD in ST and increased sensitivity, recent research has offered new perspectives, further expanding our understanding of this association.⁽¹¹⁸⁾

Despite the expanding focus on tongue physiology, genetics and related phenotypes, studies examining specific oral microbial communities and their relationship with taste perception are scant, often employing limited methodologies for taste perception evaluation or oral microbiota analysis. For instance, Solemdal and colleagues⁽¹¹⁹⁾ found that taste perception, particularly sourness, was diminished in acutely hospitalised elderly individuals with high lactobacilli growth, suggesting a potential role of bacterial organic acids in modulating taste thresholds. Conversely, Besnard *et al.*⁽¹²⁰⁾ identified specific bacterial and salivary signatures discriminating between NT and ST but solely assessed the detection threshold for linoleic acid. Analysis of tongue microbiomic profiling data revealed no significant differences in intra- and inter-subject ecological diversity between ST and NT groups, consistent with previous observations of limited microbial community variation among healthy adults. Moreover, the presence of Actinobacteria and Bacteroidetes has been associated with increased taste sensitivity, potentially through the production of secondary metabolites that influence bitterness and astringency perception.^(91,121,122) Collectively, these studies illustrate how

alterations in the microbial community of the tongue can influence taste sensation (Table 1). However, the oro-sensory implications of such changes remain to be fully understood, necessitating further research employing robust analysis techniques on predicted metagenomics data to explore microbial metabolic pathways.

Mechanistic insights into microbiota-mediated taste sensitivity

Understanding the intricate relationship between oral bacteria and taste perception sheds light on how the oral microbiome influences dietary preferences, food intake and potentially weight regulation. While the mechanistic underpinnings of oral microbiota's role in taste sensitivity are still emerging, two predominant mechanisms have been proposed. Firstly, the microbial biofilm development may generate a physical barrier that restricts the contact of tastants to their receptors, thereby modulating taste perception.⁽¹²³⁾ Secondly, the metabolic activity of oral microbiota could influence taste sensitivity through the consumption or production of bioactive metabolites derived from food or saliva. For instance, saccharolytic bacteria can degrade carbohydrates into organic acids, potentially altering taste perception, while proteolytic bacteria may produce metabolites that influence taste sensitivity.⁽¹²⁴⁾ Notably, a novel hypothesis implicating differences in methanogenesis activity of oral microbiota in modulating fatty taste perception in obese individuals has recently emerged, suggesting a nuanced interplay between microbial metabolism and taste perception.⁽¹²⁵⁾

Several mechanisms have also been proposed to elucidate the connection between oral bacteria and obesity-related metabolic disorders. These mechanisms comprise local influences of oral bacteria on taste perception and food preferences, as well as systemic effects on adipose tissue function, gut microbiome composition and systemic inflammation.^(127,126,127) Notably, changes in inflammatory tone, modulation of gut microbiome composition, and alterations in taste perception may contribute to the development and maintenance of obesity and metabolic disease.^(12,128) Two proposed mechanisms connect oral bacteria with inflammatory and metabolic effects in distant organs: the oral–gut axis^(129–131) and the oral–blood axis.^(132–134) The oral–gut axis suggests that oral bacteria can translocate to the gut, influencing gut microbiome composition and metabolic processes. Recent studies support this notion, demonstrating extensive transmission of oral bacteria to the intestine and their potential impact on gut health and metabolism.⁽¹²⁹⁾ Meanwhile, the oral–blood axis posits that oral bacteria and inflammatory molecules can enter the bloodstream, leading to systemic inflammation and local inflammatory responses in distant organs. This pathway has been implicated in various inflammatory diseases, including rheumatoid arthritis and cardiovascular diseases.^(133,134)

Chemical perception arises from the activation of chemoreceptors by a diverse range of compounds across different chemical families. Recent studies indicate that metabolic processes in the mouth can alter both the quality and quantity of compounds activating these receptors.^(82,135) Three mechanisms through which the oral microbiota can modulate host chemosensory perception can be elucidated. Firstly, microbial enzymes generate metabolites that activate or modulate host chemoreceptors.⁽¹³⁶⁾ Secondly, bacterial metabolism of exogenous molecules contributes to terminating their perception.⁽⁸⁴⁾ Thirdly, the microbiota can manipulate the host's chemical senses by altering receptor

density.^(88,89) Understanding these intricate interplay between the oral microbiome and obesity-related metabolic disturbances holds promise for the development of novel therapeutic approaches.

Mapping the current landscape and identifying future directions

As our understanding of how taste perception intertwines with the oral microbiota progresses, it is evident that there are significant gaps in our knowledge, prompting a call for further investigation. While some studies have hinted at links between oral bacteria and taste sensitivity, there are inconsistencies that need addressing, emphasising the necessity for thorough longitudinal studies. Future research should prioritise uncovering fundamental relationships, decoding natural fluctuations and investigating the effects of perturbations. Collaborative efforts across disciplines are vital for fully comprehending the intricate dynamics of human host–microbiome interactions and for exploring the potential manipulation of taste perception through oral microbiota modulation. The association between the oral microbiome and obesity is gaining recognition, with oral bacteria implicated in various mechanisms underlying metabolic diseases. These bacteria contribute to metabolic inflammation in adipose tissue and target different metabolic organs in various pathological conditions. However, further investigations are necessary to fully understand the pathways connecting oral bacteria with distant metabolic tissues and the potential metabolic disruptions they may cause. While both the oral–blood axis and the oral–gut axis show promise as routes of bacterial translocation, their exploration has been uneven. The oral–blood axis has been primarily explored in dental medicine, focusing largely on dental pathogens, while the oral–gut axis, despite its implication in several metabolic disorders, requires deeper investigation, especially concerning its role in obesity and the potential impact of oral bacteria on gut microbiome composition.

Furthermore, investigating the interaction between oral bacteria and taste cells and receptors, as well as potential central effects regulating food preference akin to the gut–brain axis, deserves attention in forthcoming studies. These insights suggest that the oral microbiome may wield a more significant influence on obesity and metabolic diseases than previously acknowledged, emphasising the urgency of understanding these mechanisms and their health implications. Disruptions in taste receptors can interrupt oral microbial balance and exacerbate diseases like periodontitis, suggesting potential alternative therapeutic avenues for oral diseases caused by specific pathogens. Delving into genetic variations in taste receptors could aid in predicting susceptibility to oral diseases and tailoring personalised treatment strategies. In addition to summarising the key points of the review, future research should focus on addressing specific questions such as understanding the molecular mechanisms by which microbial metabolites interact with taste receptors and how these interactions influence long-term dietary behaviour. Methodologies for future studies could include longitudinal assessments and intervention-based research, particularly using probiotics or prebiotics to modulate the oral microbiota. Clinically, these findings pave the way for developing personalised dietary recommendations tailored to an individual's taste perception and oral microbiota profile, offering potential for more effective dietary interventions, especially in the context of obesity and metabolic disorders.

Concluding remarks

In conclusion, our review sheds light on the intricate interplay between oral microbiota and taste sensitivity, expanding our understanding of human taste perception. While previous studies have predominantly focused on genetic and physiological determinants, this review underscores the potential role of oral bacteria in modulating taste perception. Our findings suggest that the composition of oral microbiota may contribute to individual differences in taste sensitivity, with certain bacterial species potentially enhancing or dampening taste perception through their metabolic activities. However, future investigations leveraging advanced analytical techniques on metagenomic data hold promise for uncovering microbial metabolic pathways that may serve as biomarkers for taste sensitivity phenotypes. Ultimately, a deeper understanding of how oral microbiota influence taste perception could have implications for personalised nutrition interventions and the development of novel strategies to promote healthy eating behaviours. As we continue to unravel the complexities of taste perception, incorporating the role of oral microbiota promises to unveil new insights with far-reaching implications for human health and wellbeing.

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