Taste System Anatomy

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Overview

Taste is a chemical sense. The sensory experience is produced by stimulation of specific receptors in the oral cavity. The gross anatomy (peripheral and central nervous system) of taste, microscopic and ultrastructural morphology of taste buds, physiology of taste (modalities, distribution of taste sensations, electrophysiology of the receptors, mechanism and intensity of stimulation, and taste contrasts), as well as a few clinical applications, are discussed in this article.

The anatomy of the taste system is displayed in the image below.

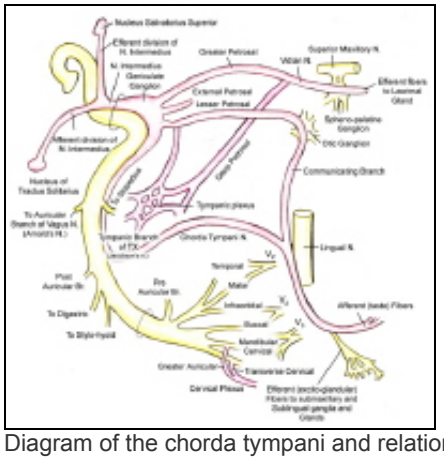


Diagram of the chorda tympani and relations to the petrosal nerves.

Gross Anatomy

Peripheral pathway

Taste is mediated by 3 cranial nerves: the facial (VII), glossopharyngeal (IX), and vagus (X), as displayed in the images below.

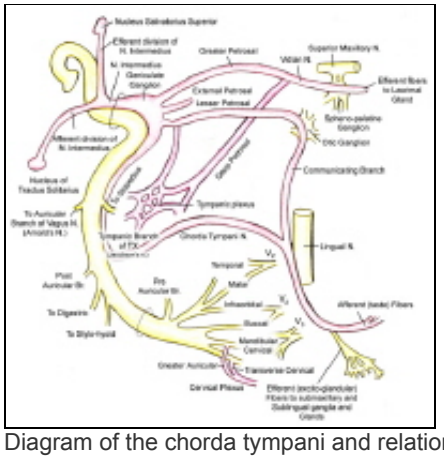
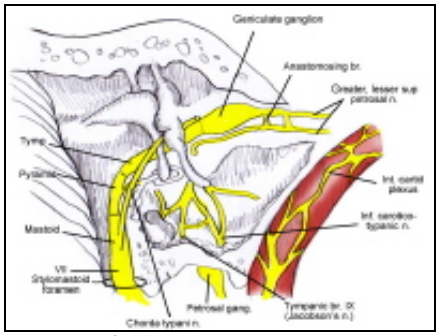
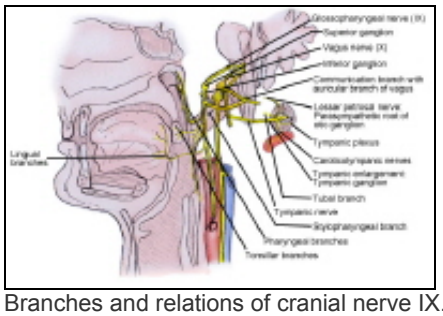


Diagram of the chorda tympani and relations to the petrosal nerves.



Relation of the chorda tympani to the ossicular chain in the middle ear.



Branches and relations of cranial nerve IX.

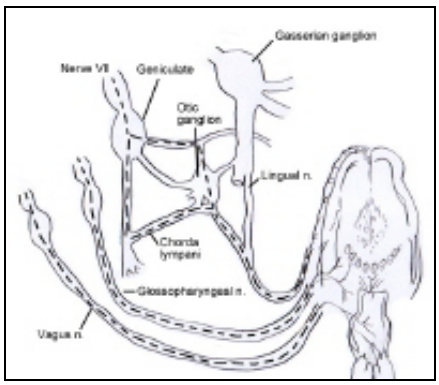


Diagram showing lingual innervation via cranial nerves VII, IX, and X.

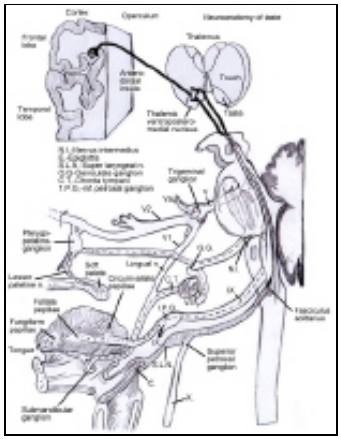


Diagram illustrating the central and peripheral taste pathways.

Glossopharyngeal nerve

The glossopharyngeal (IX) is the most important nerve for the sense of taste. It provides sensory innervation to the base of the tongue and both motor and sensory innervation to part of the pharynx. The lingual branches of the glossopharyngeal nerve mediate taste sensations to the following:

- Circumvallate papillae of the tongue, situated at the junction between the anterior two thirds and posterior third of the tongue
- Foliate papillae, situated at the rear edge of the tongue.

They also provide general afferent neurons to the mucous membrane of the posterior third of the tongue. The lingual branches course along the styloglossus muscle. These branches then course deep in the lower part of the tonsillar fossa. After coursing along the stylopharyngeus muscle and providing its nerve supply, the nerve assumes a medial relation to the external carotid artery and anteromedial to the internal carotid artery.

The inferior glossopharyngeal ganglion is located here, anterior to the superior cervical sympathetic ganglion (SCSG). It contains afferent cell bodies. It communicates with the superior ganglion of the vagus and the SCSG. CN IX then enters the jugular foramen after coursing through a groove in the inferior aspect of the petrous bone. The smaller superior glossopharyngeal ganglion contains unipolar cell bodies and is located in the jugular canal. The glossopharyngeal nerve lies anteriorly in the jugular foramen and exits it to course 10-20 mm in the cerebellopontine angle (CPA). It enters the medulla oblongata in the retro-olivary area. Its fibers proceed to join the upper part of the fasciculus solitarius to join the gustatory nucleus (or nucleus solitarius).[1, 2, 3, 4]

Facial nerve

The components of CN VII that reach the tongue include the chorda tympani and the greater petrosal nerve, which arise from the nervus intermedius (smallest afferent branch of the facial nerve).

The chorda tympani (CT) receives taste information from the anterior two thirds of the tongue. It courses along the lingual nerve, and they both leave the undersurface of the tongue to run beneath the submandibular (Wharton) duct, then ascend after crossing the duct lateral to the hyoglossus and styloglossus muscles.

The CT also supplies the preganglionic parasympathetic fibers to the submandibular ganglion. After entering the infratemporal fossa between the medial pterygoid muscle and mandible, the chorda tympani leaves the lingual nerve and crosses the spine of the sphenoid bone, to proceed to the petrous temporal bone through the petrotympanic fissure (canal of Huguier). The nerve enters the superolateral aspect of the tympanic cavity and lies medial to the neck of the malleus and lateral to the long process of the incus. The CT then exits the middle ear to join the facial nerve in the facial canal. It travels with CN VII in the vertical and horizontal segments to the geniculate ganglion.

The afferent and visceral efferent fibers leave the geniculate ganglion with the facial nerve and are known as the nervus intermedius (nerve of Wrisberg). The nervus intermedius exits the facial nerve at the labyrinthine section. It travels in the internal auditory meatus (IAC) as one nerve or divided in a few fibers between CN VII and VIII. After crossing the CPA, it enters the pons between the motor root of CN VII and vestibular root of CN VIII. Its fibers enter the solitary fasciculus to terminate into the upper part of the nucleus solitarius.

The greater petrosal provides taste for the palate. The palatal taste buds are located at the junction of the soft and hard palate. The palatal fibers travel with the lesser palatine nerve of V2 and ascend the greater palatine canal to

reach the pterygopalatine fossa. These taste fibers unite with the parasympathetic fibers of the greater petrosal nerve, which synapse at the pterygopalatine ganglion and join the deep petrosal nerve to form the nerve of the pterygoid canal. At the exit of the pterygoid canal, it becomes the greater petrosal nerve, which crosses the foramen lacerum lateral to the internal carotid artery. The nerve is then located deep to the trigeminal (semilunar) ganglion and exits the middle cranial fossa at the facial hiatus to join the geniculate ganglion of the VIIth cranial nerve. From the ganglion, the fibers course in the facial nerve in a similar fashion to the nervus intermedius.

Vagus nerve

The superior laryngeal nerve of CN X innervates taste buds on the laryngeal surface of the epiglottis. The role of these fibers in daily taste perceptions is not well understood.

Central pathway

The taste fibers from CNs VII, IX, and X enter the medulla oblongata. After descending in the tractus solitarius, they terminate at different levels in the nucleus solitarius. The fibers from CN VII and IX end in the rostral part, and the fibers from the vagus end in the caudal part of the nucleus (see the image below).

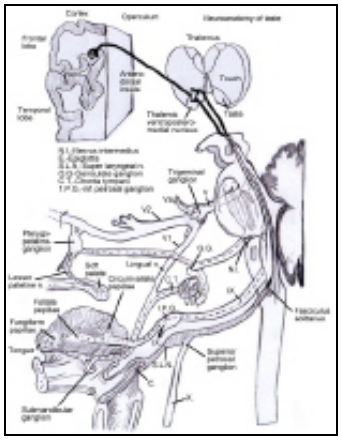


Diagram illustrating the central and peripheral taste pathways.

From this nucleus, 2 sets of fibers project. The first set projects to the preganglionic parasympathetic neurons in the superior and inferior salivary nuclei and the dorsal vagal nuclei. These represent interneurons in the gustatory pathway. A reflex (inborn) response to taste includes an increase in salivary secretion, as well as gastric and pancreatic juice.

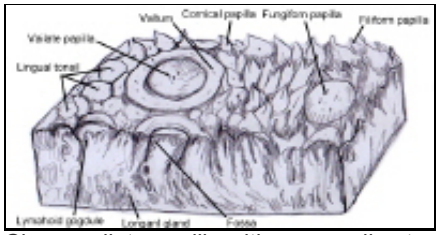
The bulbo-thalamic (second-order ascending neurons) pathway joins the medial lemniscus of the opposite side. These fibers relay in the thalamus close to the fibers of somatic sensations for the face. The third-order neuron projects from the arcuate thalamic nucleus to conduct taste to the cortical taste center. This center is in the inferior part of the parietal lobe cortex adjacent to the somatosensory area of the tongue and face. This area extends into the lateral fissure and on to the insula.

On stimulation of taste receptors, electrophysiological studies have shown potential changes in the inferior part of the postcentral gyrus in the corticosensory area of the tongue and face. Overlap and intermingling exists between the cortical taste area and that of somatic sensations of the face and tongue. Furthermore, evidence exists to support an ipsilateral gustatory system that ascends to the thalamus via the trigeminal tract. Stimulation of the insular region causes taste hallucinations.

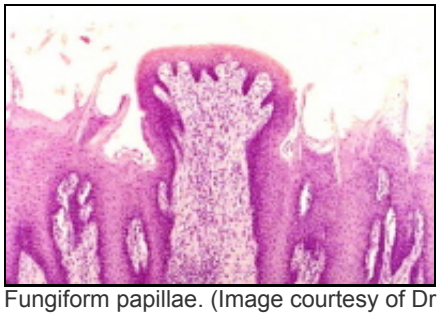
Many diagrams of the tongue depict the different tastes as attributed to specific lingual locations. Sweet is perceived at the tip, bitter in the back, sour at the edges, and salty equal on all locations. However, the idea that all 4 tastes are perceived on all areas that have taste buds is generally accepted.

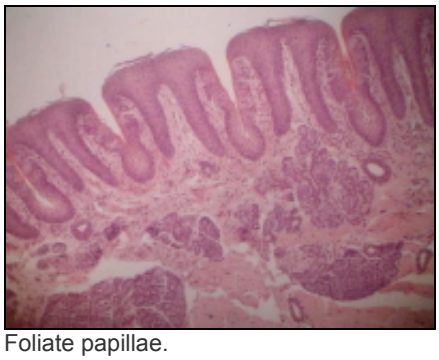
Microscopic Anatomy

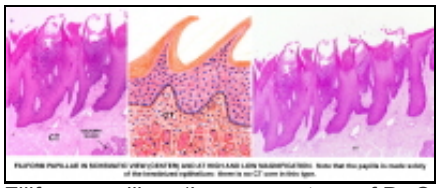
The papillae are projections of a connective tissue core covered with squamous epithelium.[2, 3] The types of papilla are circumvallate (vallate), foliate, fungiform, and filiform (see the images below).



Circumvallate papilla with surrounding trench.


Fungiform papillae. (Image courtesy of Dr Caceci.)


Foliate papillae.


Filiform papillae. (Image courtesy of Dr Caceci and line drawings courtesy of Dr Samir El-Shafey at Cairo University.)

Circumvallate papillae

The circumvallate papillae are the largest; they project slightly over the tongue surface. They vary in number from 8-12 and are situated in front of and parallel to the sulcus terminalis of the tongue and form together the shape of an inverted V. The middle papilla in the center of the V is the largest. They are called circumvallate because they are surrounded by a groove or moatlike trench (vallum). A duct of a serous (Ebner) gland opens in this trench.

Foliate papillae

The foliate papillae are peglike and are also surrounded by trenches. They are studded with taste buds and form ridges on the lateral and posterior surface of the tongue.

Fungiform papillae

The fungiform papillae are more numerous than the vallate type. They are globular and have a slightly constricted stalk. The rich blood supply gives them a bright-red color. They are situated in the tip and sides of the tongue.

Filiform papillae

The filiform papillae are the most numerous. They are abundant on the lingual dorsum and are arranged in rows parallel to the sulcus terminalis. Each papilla consists of a crest of connective tissue and has secondary projections near their summit. Their superficial cells transform into scales, and they are responsible, together with disintegrating lymphocytes, for the “lingual coating” that occurs in some certain general ailments or even normal

persons. These papillae are not gustatory but, rather, function in the tactile aspect of feeding.

Taste buds

The taste buds are the sensory end organs for gustation (see the images below). Each bud is flask-shaped, with a wide base and a short neck opening at the taste pore. In mammals, taste buds contain approximately 50-100 elongated epithelial cells and a small number of proliferative basal cells. The apical ends of the taste cells contain microvilli 2-3 µm in length that connect with the luminal surface through a porelike opening.

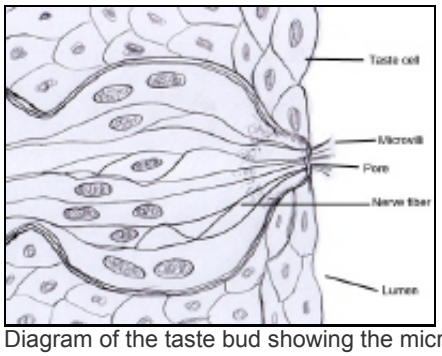
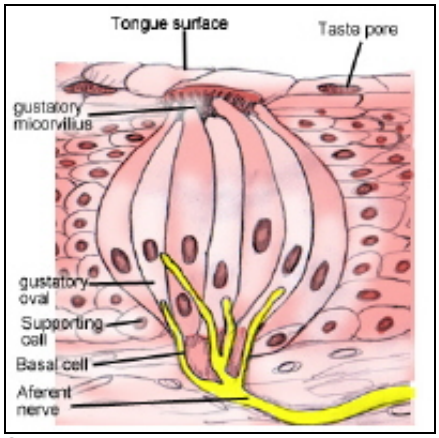


Diagram of the taste bud showing the microvilli.



Section through taste bud depicting the sensory nerve endings.

Taste buds have a life span of about 10-12 days. The taste buds in the anterior two thirds of the tongue are innervated by the chorda tympani and in the posterior third by the lingual branch of the glossopharyngeal nerve. Taste buds in the soft palate are innervated by the greater petrosal nerve of the face. The taste fibers in the epiglottis are innervated by the superior laryngeal branch of the vagus.

Ultrastructurally, 3 types of taste bud cells have been identified: type I, type II, and type III. Many studies have investigated the replacement of these 3 types of cells from the proliferative basal cell population. A recent study (on single lineage theory) postulates that type I cells transform to type III, which then mature to type II. However, the Finger analysis in mice demonstrated that the different cells are not merely different stages of development of one cell type.[2]

The image below illustrates the apical structures of the 3 types.

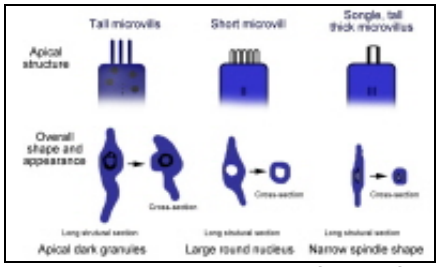


Diagram depicting the types of cells of the taste buds and their apical portions.

Type I cells

Type I cells (or dark cells) extend lamellate processes around other types of taste cells and express a glial glutamate transporter (GLAst).

Type II cells

Type II cells have large, round nuclei and express all the elements of taste transduction cascade for sweet and bitter, including T1R or T2R families of taste receptors, among others. These taste cells (type II) are considered the transducing cells for these taste qualities.

Type III cells

Type III cells are characterized by identifiable synaptic contacts with the gustatory nerve fibers. They express the synaptic membrane protein SNAP25 and the neural cell adhesion molecule (NCAM). The prominent synaptic contact suggests the implication of these cells in the transmission of information to the nervous system.

Physiological Considerations

In this section, aspects of the physiology of taste, its assessment, evaluation of taste intensity, and the difference between taste and olfaction are summarized.[3, 4, 5, 6, 7, 8, 9]

Taste receptors

The microvillous membrane projecting into the taste pore contains the site where the stimulus first occurs with the taste system. Concerning receptor sites and taste stimuli, salt and acid tastes use the ionic channels in the membrane, while sweet and bitter tastes interact with the protein receptors. The sugars of primary use to human physiology are the monosaccharides glucose and fructose and the disaccharide sucrose. The sweet taste receptors are tuned to this small group of sugars.

Regarding bitter taste, many more bitter receptors exist, probably because poisons are generally bitter. Sweet and bitter receptors (T1Rs and T1R2) are expressed on different receptor cells; however, they share the same transduction mechanism. Sodium chloride produces the purest, saltiest taste in humans, and other salts such as potassium chloride taste bitter and salty. A fiber specific to each of the 4 basic types of taste appears to exist, acting as labeled lines for the 4 basic tastes.

Taste assessment

Assessment of taste is not an easy task to perform, because damage may affect taste quality or intensity. The alteration in taste sensation may also be localized if one nerve is injured while the others are not. Assessment of taste sense should involve the following 4 taste qualities:

- Salt: sodium chloride
- Bitter: quinine hydrochloride or quinine sulfate
- Sweet: sucrose
- Sour: citric acid

Testing is done usually with patients as their own control, by testing the right and left sides. Using confocal laser scanning microscopy, Srur et al reported changes in taste bud morphology (shape, diameter) during taste disturbance.[10] However, the fungiform papillae remained unchanged.

Taste-intensity testing

Taste-intensity testing is performed by comparing the patient to normal individuals. Assessment of taste threshold can lead to serious errors. Threshold and suprathreshold perception are dissociated. Clinical evaluation of taste should also consider everyday (or suprathreshold) experience of the patient. Comparing suprathreshold perception between patients is not easy, since we do not have each other’s experience. The use of labeled scales is one attempt to standardize testing.

Electrogustometers are used to test different loci. The basis of the technique is that a weak electric current produces a sour taste when applied to the receptor. Berling et al studied the correlation between electrogustometry and the filter paper disc method for taste assessment.[11] The results indicated that electrogustometry and the filter paper disc method are reliable methods to measure taste with a high degree of reproducibility. The only actions causing significant changes in the electrogustometry readings were having local anesthesia of the tongue and eating a bitter substance.

Difference between taste and olfaction

The difference between taste and olfaction is often confusing. In addition to the 4 sensations mentioned before, metallic taste has been suggested as a fifth sensation. This has not achieved worldwide acceptance. Olfactory qualities are much more numerous. The claim that humans can recognize 10,000 odors has been challenged. Many now believe that discrimination occurs when odors are presented simultaneously rather than one at a time. Experience, of course, plays a role in odor identification.

The current belief regarding age and the senses of olfaction and taste is that taste is much less affected by age than olfaction. When the standard 4-taste stimuli are compared, citric acid and quinine are found to be the most likely to diminish, but sucrose is the most robust. Regarding the evaluation of chronic taste, the patient usually describes it as sweet, salty, etc. However, olfactory sensations are less precise and commonly referred to as “unpleasant or rotten.” In general, if the patient cannot precisely place the sensation, it is most likely olfactory.

Dysgeusia

Dysgeusia, or taste perversion, if genuine, should be investigated regarding the stimulus and how it is gaining access to the mouth. If the dysgeusia is due to tastant, the use of mouth rinse should make it disappear. The application of a local anesthetic and normal cranial nerve examination also validate that dysgeusia is caused by a tastant.

Many medications are excreted through the saliva. Other causes could be gingival fluid, gastroesophageal reflux, postnasal drip, or blood. Blood gives a salty dysgeusia, and medications in general give a bitter taste. Taste phantom may also be produced by medications. Chemotherapeutic agents are examples. Of course, genetic factors play a role in this situation.

PROP

Three phenotypes of individuals have been determined according to their ability to taste PROP (6-n-propylthiouracil). Nontasters perceive no bitterness to PROP, while supertasters and medium tasters perceive it with different degrees of bitterness. Supertasters are known to have more fungiform papillae. They are also more sensitive to pepper, chili, and alcohol. In 2003, the location of the gene controlling the expression of the PROP receptor was discovered.

Taste-sensation variations

Variations in oral sensations have an effect on diet, smoking, and alcohol consumption (see Clinical Applications). Supertasters are more common in women than men and also in certain races (Asian vs white). However, in older women, the ability to taste bitter decreases with age. This is due to hormonal changes after menopause and may contribute to burning mouth syndrome.

Venous taste

Venous taste is a phenomenon that occurs at a receptor level below the microvilli in the taste cell. Saccharine produces a sweet taste 10-15 seconds after intravenous injection. The same occurs with dehydrocholic acid, which is perceived as aftertaste. This may be basically what happens with chemotherapeutic agents.

Other Considerations

Clinical applications

When the taste system is damaged, the result may be alteration or loss of taste. However, phantom taste may occur as well. Different loci in the peripheral or central pathway may be affected by different pathological processes.[9, 10, 11, 12, 13, 14, 15]

Peripheral pathway

Chorda tympani travels with the lingual nerve through the infratemporal fossa with the inferior alveolar nerve. Local anesthesia to this space may damage the chorda tympani. Taste damage may occur with otitis media, Bell palsy, and Ramsay Hunt syndrome; stapedectomy or mastoid surgery may also have the same effect. However, stretching the chorda tympani without cutting it produces a metallic taste in the mouth. Neoplasia of the floor of the mouth produces numbness of the tongue due to lingual nerve involvement. In the absence of a neoplastic process,

tongue numbness may indicate CN V (trigeminal) pathology.

If no oral pathology or numbness exists, loss of taste may be due to involvement of the chorda in the temporal bone or in the cerebellopontine angle. However, Seaberg et al studied chorda tympani function and its relation to otitis media and body mass index in children[15] and did not find any effect of previous acute otitis media history on chorda tympani nerve function. Furthermore, this pediatric study did not demonstrate a relationship between acute otitis media and an increased body mass index.

Injury to the glossopharyngeal nerve may occur during tonsillectomy, or uvulopalatoplatoplasty, if the lingual or the pharyngeal branch of CN IX is severed.

Three syndromes are associated with lesions involving the jugular foramen (eg, glomus jugulare, schwannoma, squamous cell cancer), as follows:

- Vernet syndrome involves CNs IX, X and XI and causes altered taste, ipsilateral paralyzed pharynx, unilateral vocal cord paralysis, and droopy shoulder
- Collet syndrome occurs when CN XII is affected with IX, X, and XI due to inferior extension of the lesion or deeper in the foramen magnum, resulting in ipsilateral deviation of the tongue (or fasciculations)
- Villaret syndrome occurs when the lower 4 cranial nerves are involved with the sympathetic chain (this is usually due to lesions extending below the jugular foramen, giving rise to the above-mentioned signs, plus Horner syndrome [ptosis, myosis, anhydrosis, enophthalmos, and loss of ciliospinal reflex])

Central pathway

Damage to rostral insular cortex or pontine hemorrhage gives rise to ipsilateral loss of taste, due to the fact that the vast majority of taste fibers project ipsilaterally.

Alteration or loss of taste may be due to medications that are excreted through the saliva. Other causes could be gingival fluid, gastroesophageal reflux, postnasal drip, or blood. Blood gives a salty dysgeusia, and medications, in general, give a bitter taste.

Berger et al noted that patients with terminal stages of cancer suffer from cough, nausea, vomiting, and hiccups. They attributed these symptoms to the chemotherapy that damages taste. Also, since taste plays a role in preparing the gastrointestinal tract for the cephalic phase response (arrival of food), taste damage may play a role in GI imbalance that occurs in these conditions.

Burning mouth syndrome was mentioned previously. It is one of a few taste disorders that can be treated satisfactorily in most cases. Clonazepam is useful in treating the condition and eliminates taste phantoms associated with it. Since this medication is a gamma-aminobutyric acid (GABA) agonist, clonazepam increases the level of this inhibitory neurotransmitter, thus stimulating the inhibition of taste.

Various disease states may be associated with decrease in gustatory function. For example an association between 6-N-propylthiouracil (PROP) bitterness and colon cancer was described by Basson et al.[9] They found that PROP bitterness correlated significantly with polyp number. The PROP-polyp relationship was strongest in men older than 66 years, and men who reported PROP as bitter ate fewer vegetables. These data suggested that taste genetics may influence colon cancer risks, through lower consumption of vegetables.

Compared with supertasters, nontasters have fewer taste papillae on the anterior tongue and express less negative (eg, bitterness) and more positive (eg, sweetness) sensations from alcohol. A study by Duffy et al supports taste's genetic effects on alcohol intake.[14] PROP bitterness serves as a marker of these effects,

Neuropathies associated with diabetes may explain the effects on taste associated with this disease. However, the specific deficit for glucose suggests that the receptor mechanism for glucose may be different from that of other sugars.

Taste loss and taste phantoms have been associated with renal failure. Uremic toxins that accumulate in these conditions seem to be the causal factor. Dialysis usually eliminates the bitter or metallic oral taste.

Clinical observations reveal a link between depression and taste change. An association between depression and PROP tasting has been theorized.

Contributor Information and Disclosures

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