

TASTE PERCEPTION- A MATTER OF SENSATION

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ABSTRACT

Taste, gustatory perception is the sensory impression of food or other substances on the tongue and is one of the five traditional senses. Taste is the sensation produced when a substance in the mouth reacts chemically with taste receptor cells located on taste buds. Taste, along with smell and trigeminal nerve stimulation determines flavors of food or other substances. Humans have taste receptors on taste buds and other areas including the upper surface of the tongue and the epiglottis. Taste perception fades with age. On average people lose half their taste receptors by the time they turn twenty. This article review about taste, taste buds, its mechanisms, development and aging, role of saliva and its clinical implications.

Keywords: Taste, Saliva and taste bud

INTRODUCTION

Taste, gustatory perception or gustation is the sensory impression of food or other substances on the tongue and is one of the five traditional senses¹. Single cell animals such as bacteria have surface chemoreceptor sensitive to a variety of chemicals. Invertebrate chemo sensitivity is based on discrete chemosensory organs contained in special appendages. In most air-breathing vertebrates, chemo sensitivity is divided into a contact chemical sense called taste and a distance chemical sense called smell.

Taste Receptors:

Mammalian taste buds are widely distributed throughout the oral cavity pharynx and larynx. Most lingual taste buds are confined to the dorsal and lateral borders of the tongue and are associated with specialized structures called papilla. There are four main types of tongue papilla: the filiform, fungiform, circumvallate and foliate. The most numerous are the filiform, located over the anterior and posterior tongue dorsum, which do not contain taste buds and therefore have no gustatory function. The fungiform papillae contain one or more taste buds on their upper surface and are confined to the papillae. The circumvallate papillae are found at the oral and pharyngeal parts of the tongue. Taste buds are located on the sides of the papillae and sometimes in the wall surrounding the papillae. Humans have 8 to 12 circumvallate papillae arranged in a chevron.

The foliate papillae, found on the posterior, lateral border of the tongue, consists of a series of folds forming clefts in the tongue surface. Taste buds are found in the epithelium of the cleft walls. Not all taste buds are confined to the tongue. Significant

numbers of them are found on the palate and larynx. (Fig. – 1)

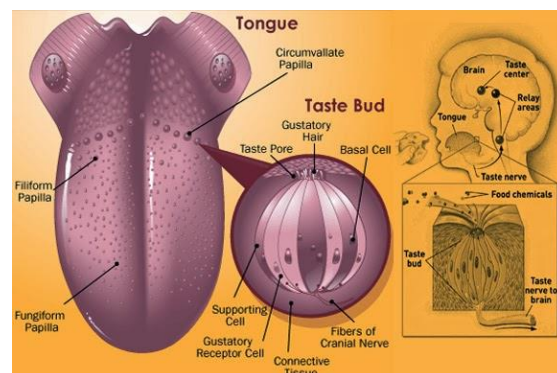


Fig. 1 Tongue Papillae and Taste Bud

Structure:

Taste buds are goblet-shaped structures spanning the depth of the epithelium in which they are situated. Each bud consists of about 50 spindle-shaped, modified, epithelial cells that extend from the basement membrane to the epithelial surface. At the epithelial surface the tapered apical ends of the taste buds are in contact with the fluid environment of the oropharynx and larynx through a narrow channel called taste pore.

Terminal branches of gustatory nerve fibers enter the taste bud at its base and distribute among the taste cells.² The most frequently encountered taste bud cells, Type I cells, are long and narrow, extending from the base of the taste bud to the taste pore, they compose approximately 60% of the total cell population.² These electron dense cells (sometimes called dark cells) are characterized by large, dense-cored vesicles in the apical cytoplasm as well as indented, irregularly shaped nuclei. Type II

cells (often referred to as light cells) also extend from the basement membrane to the taste pore, but they are characterized by electron-lucent cytoplasm and large, round or oval nuclei. Type III cells also have apical specialization that extend into the taste pore and are similar in morphology to Type II cells. However, these cells are infrequently encountered in the taste bud and contain numerous dense-cored vesicles concentrated in the basal portion of the cell.

The initial contact between the taste stimuli and taste receptors takes place in the taste pore. Each of the three taste bud cells that extend into the taste pore has a different apical structure. Type I cells have long, finger like microvilli that arise from a short neck. Type II cells have shorter microvilli, and Type III cells end in a blunt, club shaped structure. (Fig. 2)

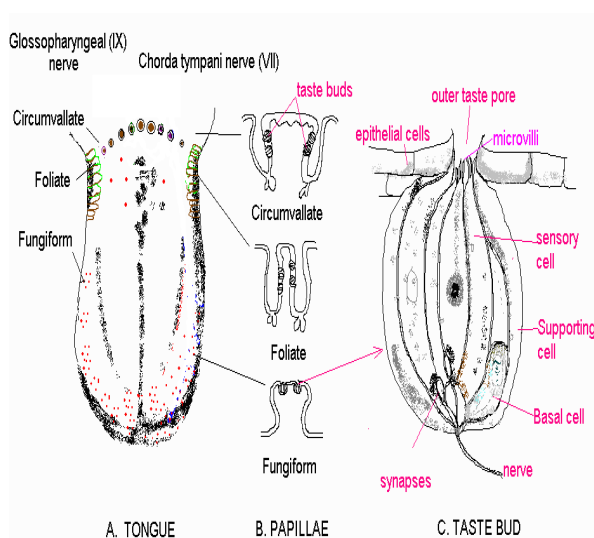


Fig. 2: Structure of Taste Bud

Dynamic Properties of Taste Buds:

Taste bud cells, like surrounding epithelial cells, are rapidly replaced. When the turnover of taste buds is studied with auto radiographic techniques, the basal cells of the taste bud are first labeled, followed by the Type I cells and then by Types III and II cells, suggesting that these cell types are transitional forms of a single cell line. The half-life of a taste bud cell is approximately 10 days. Studies of taste bud turnover also reveal that during turnover taste cells migrate, or move from the periphery to the center of the bud.

Another dynamic property of taste buds is their ability to regenerate. Transection of a gustatory nerve results in disappearance of the taste buds innervated by that nerve. Taste buds reappear once the nerve regenerates and reaches the epithelium. Only gustatory nerves promote regeneration of taste buds. Many experiments support the hypothesis that sensory axons innervating chemoreceptors secrete a substance, called a trophic substance, that causes epithelial basal cells to differentiate into taste bud

cells rather than into stratified squamous epithelial cells.³

Regardless of the source of reinnervation, regenerated taste buds are functional and exhibit neural responses to chemical stimulation of their receptive fields. Moreover, after cross nerve regeneration, response characteristics of the receptive field are identical to those recorded from the normal neural innervations; so the response properties of a particular taste nerve are determined by the receptors that it innervates, and are not inherent characteristics of the sensory innervations.

Receptor Mechanisms

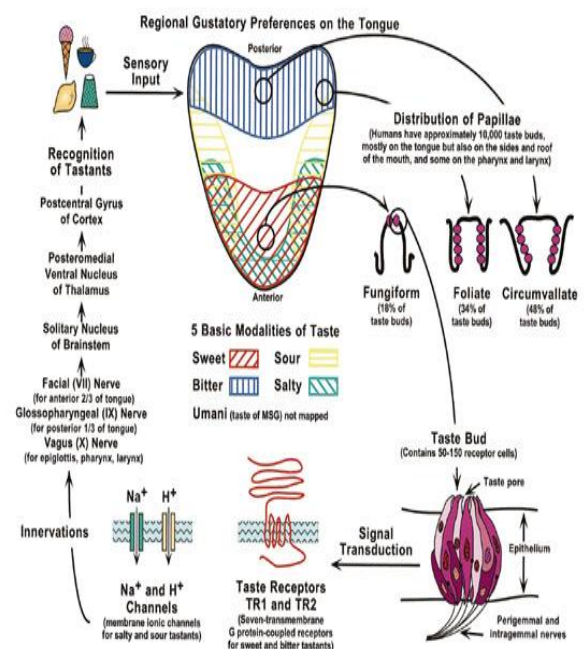


Fig. 3: Taste Receptor Mechanism

In the early 1960s, intracellular recordings techniques were used to study receptor mechanisms in taste buds. Because mammalian taste buds are surrounded by a tough, keratinized epithelium, penetration of the taste cells with glass microelectrodes was made through the taste pore. Because it is not easy to see taste pores in live tongues, the number of successful penetrations was limited in these early studies. Furthermore, taste stimuli interact with the receptor membranes through the taste pore, so that if the seal between the microelectrode and the taste cell membrane is not tight, taste stimuli can leak into the cells, making these experiments vulnerable to artifacts.

This technique makes a very tight seal between the recording electrode and the taste cell membrane, making possible the resolution of small, trans-membrane currents. Therefore, activation of single -ion channels in taste cell membranes in being

investigated so that mechanisms of taste transduction are now emerging.

Another important advance in the study of taste receptor mechanism has resulted from biochemical analysis of taste bud cell membranes. Using techniques that have proved successful in other sensory systems, researchers have made attempts to isolate taste receptors membranes.⁴

Despite various difficulties much has been learned about the membrane properties of taste bud cells. The mean resting membrane potential of mammalian taste cells is about -40 mV when the tongue is adapted to NaCl and -50 mV when the tongue is covered with water. When taste solutions representing the four taste qualities are flowed separately over the tongue, the resting membrane potentials of taste bud cells change slowly. Stimulation with salty (NaCl), bitter (quinine), sour (HCl), and sweet (sucrose) taste solutions results in three patterns of membrane potential change: depolarization, depolarization preceded by hyperpolarization. (Fig. – 3)

Using the patch-clamp recording technique researchers have described a number of different ion channels in the apical membranes of taste bud cells. In a variety of species, including mammals, taste cells have been shown to possess voltage-gated channels for Na⁺, Ca⁺, and K⁺, and also a Ca mediated cation channel. Opening or closing of the ion channels results in either depolarization or hyperpolarization of the cells, depending on the nature of the ions involved. Influx of cations (Na⁺ or Ca⁺) or efflux of anions or stimulus-evoked closure of open K⁺ channels would cause depolarization, whereas influx of anions or efflux of cations as well as closure of open K⁺ channels would cause hyperpolarization, whereas influx of anions or efflux of cations as well as closure of Na⁺ or Ca⁺ channels would hyperpolarize the cell. Taste stimuli interact with the apical membranes of the taste bud cells, so that ion channels located on the apical membranes are important in taste transduction. K⁺ channels are hypothetically involved in transduction mechanisms for both sour and bitter taste, because K⁺ channels are restricted to the apical membranes of taste bud cells and because voltage-gated K⁺ have been shown to be attenuated by acids and bitter stimuli. A major source of K⁺ ions in the extracellular environment of apical taste membranes in saliva. Therefore, salivary K⁺ ions are important in taste transduction. The transduction mechanisms for Na⁺ taste probably involves a voltage-independent Na⁺ channel.

The proportion of this current from the Na⁺ channels can be demonstrated by the use of amiloride, a drug that specifically blocks passive Na⁺ channels. Unlike transduction mechanisms for sour, bitter, and salty tastes, the transduction of sweet tastes appears to involve a specific membrane

receptor-mediated stimulation of adenylatecyclase, which in turn causes closure of K⁺ channels on the basolateral membrane. (Fig. – 4)

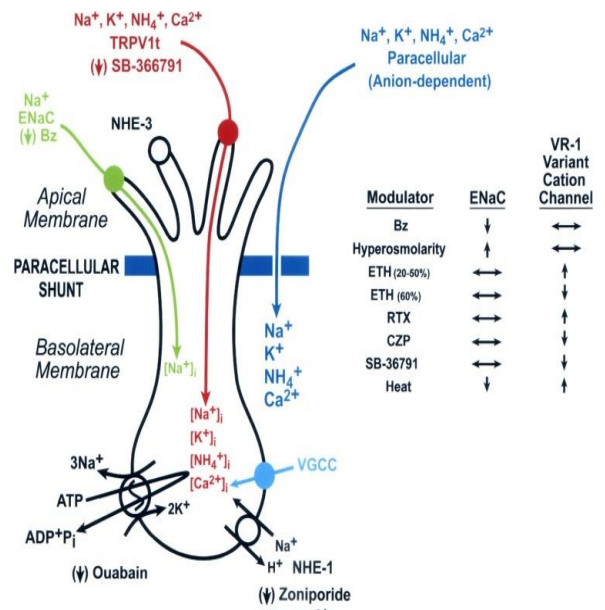


Fig. 4: Na⁺ and K⁺ Channel

Application of these techniques has resulted in rapid progress in the understanding of taste transduction. Despite this progress much remains to be accomplished, in particular determining the mechanisms by which taste stimuli are discriminated by receptors that respond to many different stimuli.

Taste qualities:

In sensory systems such as vision and audition the relationship between sensation and some physical property of the stimulus has been well established. There is usually a physical continuum such as wavelength that correlates with a systematic change in the sensory quality of the stimulus such as hue or pitch. Although taste is a sensory system responding to chemicals there is no clear continuum of physical properties of taste stimuli and taste sensation. Moreover, since the time of the ancient Greeks, taste sensation has been subdivided into different discrete tastes or qualities now commonly described as sweet, sour, salty and bitter. Behavioral thresholds are much lower to sour and bitter stimuli than sweet and salty stimuli. This and much more evidence supports the existence of different taste qualities with different perceptual properties⁵. One of the objectives of the original neurophysiologic analysis of the responses of individual taste fibers by Carl Pfaffmann was to determine if the fibers, and presumably the receptors they innervate, respond specifically to one of the four taste qualities.

Development and Aging:

Taste buds appear very early in the tongue of human fetuses, at 7 to 8 weeks of gestation, but mature appearing taste buds are not observed until later in gestation. Development is not complete at birth and taste bud numbers continue to increase⁶.

Behavioral testing of human newborns reveals that the ability to discriminate between taste stimuli is present at birth, indicating that some attributes of taste preference behavior are innate and do not require any experience for expression. However, other studies have demonstrated that although these taste behaviors are present at birth they can be extensively modified by postnatal experience.

Role of Saliva in Taste Function:

Saliva function is essential for normal taste function. It is usually difficult to taste food with a dry mouth. Saliva not only acts as a solvent for chemical stimuli in food, but also transports these stimuli to the taste stimuli to the taste receptors. At rest gustatory receptors are covered with a layer of fluid that extends into the taste pores and bathes the receptor surface of the microvilli. Little is known about this surface layer. For taste buds in the fungiform papillae it presumably consists of pooled saliva from all the salivary glands. Taste buds in the circumvallate and foliate papillae are bathed in saliva derived from von Ebner's glands. Taste buds on the palate, larynx, and pharynx are covered in fluid secreted by a large number of small salivary glands draining onto the surface of the epithelium. However, the composition of the microenvironment within the taste pore might be different from the layer of fluid that overlies the epithelial surface. Moreover, because gustatory stimulation alters salivary flow and composition, the fluid, environment may alter during transduction. Although this microenvironment, acting as a transport system, may merely play a passive role in taste mechanisms, it may have a more active role. Components within the taste pore may control access and removal of stimuli and interact with tastants during the initial events in receptor stimulus interactions.

Role of saliva in taste transduction:

Of the many organic constituents of saliva, the proline-rich proteins have been associated with the ability to taste bitter compounds such as quinine, raffinoseundecaacetate, and cyclohexamide. Saliva from the major salivary glands bathes taste receptors that are contained in fungiform papillae⁷. However, the majority of the lingual taste buds are situated in the epithelium that lines the clefts of the circumvallate and foliate papillae. The saliva bathing these taste buds is supplied by the lingual salivary (von Ebner's) glands that drain into the base of these

clefts. They provide a diffusion path for stimuli to gain access to taste buds, and they remove stimuli by active secretion.⁸ Taste stimuli must, therefore, transverse this salivary layer before interacting with receptor membranes. Ebner's glands could play an important role in taste transduction in most lingual taste buds.(Fig. – 5)

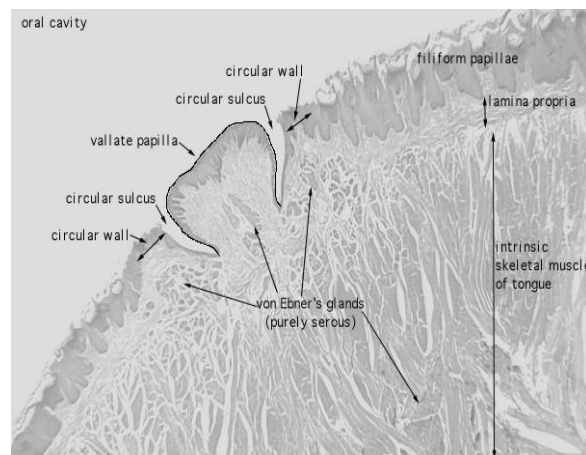


Fig. 5: Von Ebner's Gland

Recently it has been shown that von Ebner's glands secrete a protein that is structurally similar to the odorant-binding proteins isolated in nasal glands. This protein is not found in other salivary glands, and because of the relationship between von Ebner's glands and taste receptors situated in the circumvallate and foliate papillae, it is thought that this protein may be important in transporting lipophilic stimuli to the taste receptor membranes. Because many bitter substances are highly lipophilic, this protein could be important in bitter taste transduction mechanisms. It is possible, therefore, that these glands do not serve merely to rinse the clefts of the foliate and circumvallate papillae but play an active role in the transduction process.

Clinical Considerations:

Disorders of taste and smell are often classified according to the type of sensory loss. Complete loss of the sense of smell is called **anosmia** and loss of taste is called **ageusia**; partial loss is termed **hyposmia** and **hypogeusia** respectively. Often patients have distortions of taste and smell called **dysgeusia** and **parosmia** respectively, in which a chronic taste or smell is present in the absence of any stimulus. When an aberrant taste or smell is experienced because of abnormal stimulation within the central nervous system, the disorder is called a **phantom**.⁹ Although these terms are used by clinicians who diagnose chemosensory disorders, patients usually describe their disorder in different terms.

Patients with a chemosensory disorder complain that "I cannot taste" .often these individuals have no obvious clinical cause for these complaints and treatment is difficult. Until recently, information regarding the cause of chemosensory disorders consisted of an extensive clinical literature with more 200 conditions and many medications linked to alterations in taste and smell.

Olfactory disturbances result most commonly from nasal and/or sinus disease, upper respiratory infection, or head trauma, but they can also be associated with a variety of disease states, chemical exposures, and congenital syndromes. Although true taste disorders are much less common than olfactory dysfunction, they may also arise from upper respiratory infections or head trauma, as well as numerous medications, oral pathology, and radiation therapy. Although these are the most common causes of chemosensory disturbances, other factors can affect taste and olfaction.

Vitamin A deficiency increases keratinization of the tongue, including the pore area of the taste buds and adjacent epithelial and glandular tissue. Rats made deficient of vitamin A gradually lose their normal preference for NaCl and aversion to quinine solutions. Vitamin A repletion tends to restore normal preference or aversion behavior. Significant reductions also occur in the neural response of the chorda tympani to tongue stimulation with NaCl. Loss of gustatory sensitivity from vitamin A deficiency presumably results from blocking of taste pore with keratin plugs, which prevents access of stimuli to the receptors membranes. However, other factors could be involved such as possible alterations in quantity and composition of salivary secretion, since glandular tissue is also affected by the deficiency.

Gustatory hallucinations are sometimes reported by patients with epilepsy as one manifestation of parietal, temporal, or temporoparietal seizures. A brief gustatory hallucination can be induced by electrical stimulation of the parietal area as well as the amygdale and hippocampus in patients with gustatory seizures.

Familial dysautonomia affects children of Jewish ancestry and is transmitted by an autosomal recessive trait. A smooth tongue with decreased papillae and devoid of taste buds is an important feature of familial dysautonomia and is clinically associated with marked reduction in taste sensitivity.¹⁰ Patients with this disorder are unable to taste. Even concentrated sucrose solutions are identified as "not water" by the subject who is uncertain whether it is sweet or sour.

Numerous studies in humans and animals have demonstrated that taste declines after **head and**

neck irradiation. Examination of the tongues of irradiated animals reveals that after a single radiation does the number of taste buds declines. This decrease leads to taste loss as well as reduction in salivary flow rate.

Many patients with **malignancy** becomes anorexic and their food intake declines so that they lose weight; this results in increased mortality. The hypothesis is that this anorexia is possibly caused by alterations in taste sensation, because taste is important in guiding food intake.

Many **drugs** have been reported to alter taste sensitivity. Some drugs influencing the peripheral and central nervous system presumably produce their effect by altering the transmission of neural information through central taste relay. Other drugs may be excreted in saliva and produce their effect by a persistent taste.

The association of taste and smell dysfunction with zinc deficiency has led to studies of zinc therapy in such disorders. Claims for therapeutic benefits in ameliorating taste and smell disorders have been made. The result of this double blind study leave little doubt that at present there is no scientific basis for administering zinc sulfate therapeutically for ordinary taste and smell dysfunction.

A further condition with associated chemosensory disorders is called **burning mouth syndrome**, which is an intraoral pain condition occurring primarily in menopausal females. There is apparent change in sensory perception. The cause of this change is not known but may be a peripheral or central dysfunction of small afferent nerve fibers.

CONCLUSION

Sense of taste affords human being to have ability to evaluate what it eats or drinks. At the most basic level, the evaluation is to promote ingestion of nutritious substances and prevent consumption of potential poisons or toxins. Food preferences and aversions involve the sense of taste and these phenomenon are mediated through central nervous system. Tongue also feels other sensations not generally included in basic tastes. These are largely detected by somatosensory system. Taste perception fades with ageing and there are many disorders which affect the sensation of taste. Thus, sensation of taste plays vital role in maintaining general wellbeing of an individual.

REFERENCES:

1. Fauci, Anthony S., et al. Harrison's Principles of Internal Medicine. 17th ed. United States: McGraw-Hill Professional, 2008.
2. Chaudhari, Nirupa & Roper, Stephen D (2010), "The cell biology of taste", *Journal of Cell Biology* 190(3):285-296.
3. Guyton, Arthur C. (1991). *Textbook of Medical Physiology* (8thed). Philadelphia: W.B. Saunders.
4. Miller I J. Anatomy of the peripheral taste system. In: Doty RL, ed. *Handbook of olfaction and gustation*. New York: Marcel Dekker, 1995. 521-547.
5. Mavi A, Ceyhan O. Bitter taste thresholds, numbers and diameters of circumvallate papillae and their relation with age in a Turkish population. *Gerodontology* 1999. 16:119-122.
6. Bartoshuk LM. Taste. Robust across the age span? *Ann NY AcadSci* 1989. 56:165-75.
7. Lindemann B (September 2001), "Receptors and transduction in taste". *Nature* 413 (6852): 219-25.
8. Mistretta CM. Ageing effect on anatomy and neurophysiology of taste and smell. *Gerodontology* 1984;3:243-248.
9. Guyton, Arthur C. (1991) *Textbook of Medical Physiology* (8thed). Philadelphia: W.B.Saunders.
10. Cowart B J, Young IM, Feldman R.S et al Clinical disorders of smell and taste. *Occup Med* 1997. 12:465-483.