Objectives

After studying this chapter, you should be able to:

- Describe the basic features of the neural elements in the olfactory epithelium and olfactory bulb.
- Describe signal transduction in odorant receptors.
- Outline the pathway by which impulses generated in the olfactory epithelium reach the olfactory cortex.
- Describe the location and cellular composition of taste buds.
- Name the five major taste receptors and signal transduction mechanisms in these receptors.
- Outline the pathways by which impulses generated in taste receptors reach the insular cortex.

Smell & Taste: Introduction

Smell and taste are generally classified as visceral senses because of their close association with gastrointestinal function. Physiologically, they are related to each other. The flavors of various foods are in large part a combination of their taste and smell. Consequently, food may taste "different" if one has a cold that depresses the sense of smell. Both smell and taste receptors are **chemoreceptors** that are stimulated by molecules in solution in mucus in the nose and saliva in the mouth. Because stimuli arrive from an external source, they are also classified as **exteroceptors.**

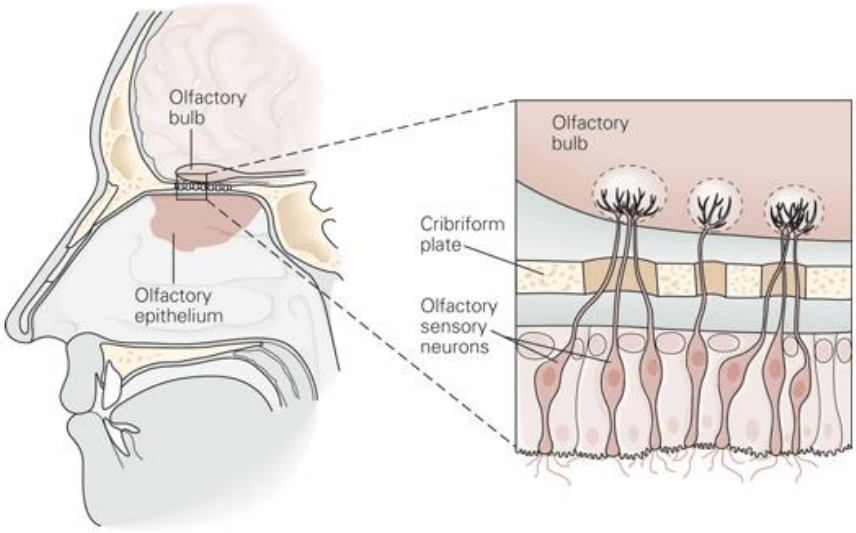
Smell

Olfactory Epithelium

The **olfactory sensory neurons** are located in a specialized portion of the nasal mucosa, the yellowish pigmented **olfactory epithelium.** In dogs and other animals in which the sense of smell is highly developed (macrosmatic animals), the area covered by this membrane is large; in microsmatic animals, such as humans, it is small. In humans, it covers an area of 5 cm^2 in the roof of the nasal cavity near the septum (Figure 14–1). The human olfactory epithelium contains 10 to 20 million bipolar olfactory sensory neurons interspersed with glia-like **supporting (sustentacular) cells** and **basal stem cells.** The olfactory epithelium is said to be the place in the body where the nervous system is closest to the external world. Each neuron has a short, thick dendrite that projects into the nasal cavity where it terminates in a knob containing 10 to 20 **cilia** (Figure 14–2). The cilia are unmyelinated processes about 2 µm long and 0.1µm in diameter and contain specific receptors for odorants **(odorant receptors).** The axons of the olfactory sensory neurons pass through the cribriform plate of the ethmoid bone and enter the olfactory bulbs (Figure 14–1).

Figure 14–1

Figure 14–1

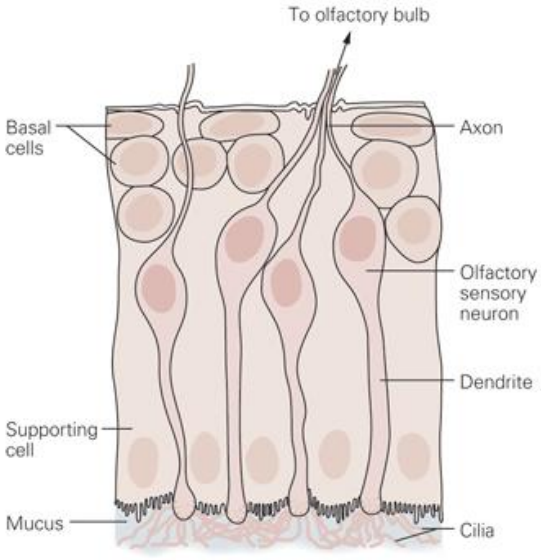


Olfactory sensory neurons embedded within the olfactory epithelium in the dorsal posterior recess of the nasal cavity. These neurons project axons to the olfactory bulb of the brain, a small ovoid structure that rests on the cribriform plate of the ethmoid bone.

(From Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

Figure 14–2

Figure 14–2



Structure of the olfactory epithelium. There are three cell types: olfactory sensory neurons, supporting cells, and basal stem cells at the base of the epithelium. Each sensory neuron has a dendrite that projects to the epithelial surface. Numerous cilia protrude into the mucosal layer lining the nasal lumen. A single axon projects from each neuron to the olfactory bulb. Odorants bind to specific odorant receptors on the cilia and

initiate a cascade of events leading to generation of action potentials in the sensory axon.

(Modified from Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

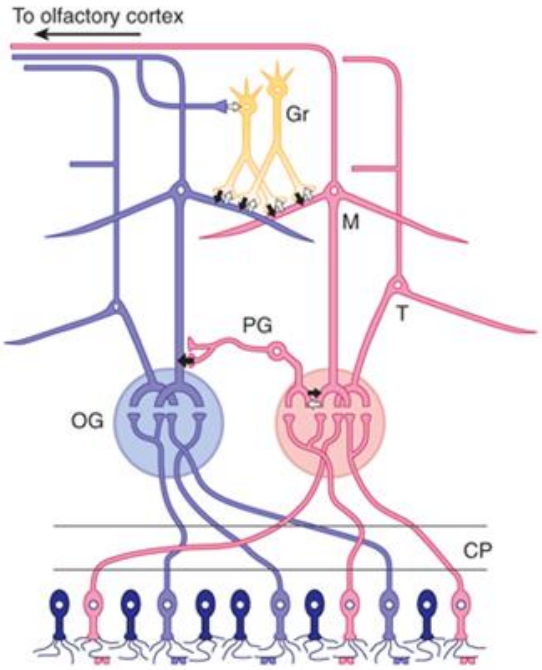
New olfactory sensory neurons are generated by basal stem cells as needed to replace those damaged by exposure to the environment. The olfactory renewal process is carefully regulated, and there is evidence that in this situation a bone morphogenic protein (BMP) exerts an inhibitory effect. BMPs are a large family of growth factors originally described as promoters of bone growth but now known to act on most tissues in the body during development, including many types of nerve cells.

Olfactory Bulbs

In the olfactory bulbs, the axons of the olfactory sensory neurons (first cranial nerve) contact the primary dendrites of the **mitral cells** and **tufted cells** (Figure 14–3) to form anatomically discrete synaptic units called **olfactory glomeruli.** The tufted cells are smaller than the mitral cells and have thinner axons, but both types send axons into the olfactory cortex, and they appear to be similar from a functional point of view. In addition to mitral and tufted cells, the olfactory bulbs contain **periglomerular cells,** which are inhibitory neurons connecting one glomerulus to another, and **granule cells,** which have no axons and make reciprocal synapses with the lateral dendrites of the mitral and tufted cells (Figure 14–3). At these synapses, the mitral or tufted cell excites the granule cell by releasing glutamate, and the granule cell in turn inhibits the mitral or tufted cell by releasing GABA.

Figure 14–3

Figure 14–3



Basic neural circuits in the olfactory bulb. Note that olfactory receptor cells with one type of odorant receptor project to one olfactory glomerulus (OG) and olfactory receptor cells with another type of receptor project to a different olfactory glomerulus. CP, cribriform plate; PG, periglomerular cell; M, mitral cell; T,

tufted cell; Gr, granule cell.

(Modified from Mori K, Nagao H, Yoshihara Y: The olfactory bulb: Coding and processing of odor molecular information. Science 1999;286:711.)

Olfactory Cortex

The axons of the mitral and tufted cells pass posteriorly through the **lateral olfactory stria** to terminate on apical dendrites of pyramidal cells in five regions of the **olfactory cortex: anterior olfactory nucleus, olfactory tubercle, piriform cortex, amygdala,** and **entorhinal cortex** (Figure 14–4). From these regions, information travels directly to the frontal cortex or via the thalamus to the orbitofrontal cortex. Conscious discrimination of odors is dependent on the pathway to the orbitofrontal cortex. The orbitofrontal activation is generally greater on the right side than the left; thus, cortical representation of olfaction is asymmetric. The pathway to the amygdala is probably involved with the emotional responses to olfactory stimuli, and the pathway to the entorhinal cortex is concerned with olfactory memories.

Figure 14–4

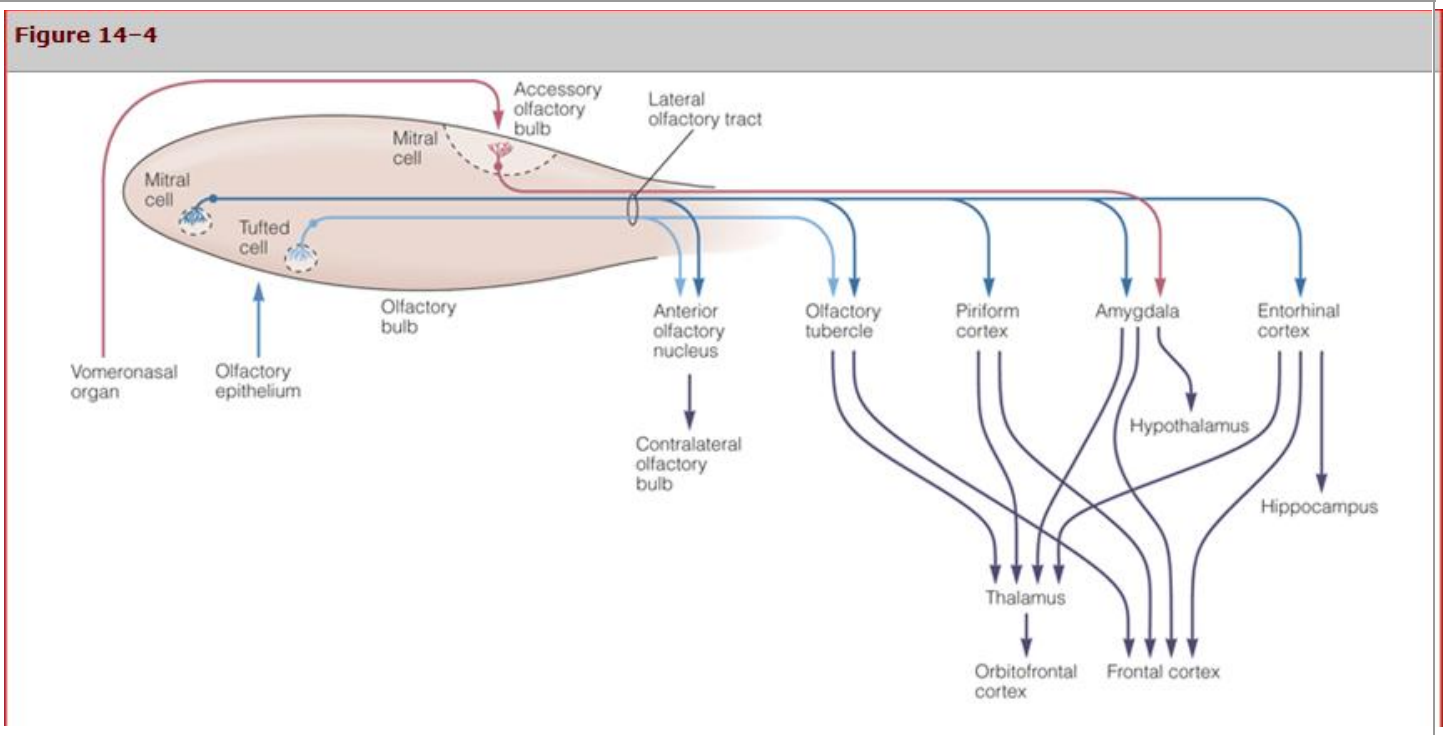


Diagram of the olfactory pathway. Information is transmitted from the olfactory bulb by axons of mitral and tufted relay neurons in the lateral olfactory tract. Mitral cells project to five regions of the olfactory cortex: anterior olfactory nucleus, olfactory tubercle, piriform cortex, and parts of the amygdala and entorhinal cortex. Tufted cells project to anterior olfactory nucleus and olfactory tubercle; mitral cells in the accessory olfactory bulb project only to the amygdala. Conscious discrimination of odor depends on the neocortex (orbitofrontal and frontal cortices). Emotive aspects of olfaction derive from limbic projections (amygdala and hypothalamus).

(From Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

Olfactory Thresholds & Discrimination

The olfactory epithelium is covered by a thin layer of mucus secreted by the supporting cells and Bowman glands, which lie beneath the epithelium. The mucus bathes the odorant receptors on the cilia and provides the appropriate molecular and ionic environment for odor detection.

The olfactory thresholds for substances shown in Table 14–1 illustrate the remarkable sensitivity of the odorant receptors. For example, methyl mercaptan, one of the substances in garlic, can be smelled at a concentration of less than 500 pg/L of air. In addition, olfactory discrimination is remarkable; for example, humans can recognize more than 10,000 different odors. On the other hand, determination of differences in the intensity of any given odor is poor. The concentration of an odor-producing substance must be changed by about 30% before a difference can be detected. The comparable visual discrimination threshold is a 1% change in light intensity. The direction from which a smell comes may be indicated by the slight difference in the time of arrival of odoriferous molecules in the two nostrils.

Table 14–1 Some Olfactory Thresholds.

Substance	**mg/L of Air**
Ethyl ether	5.83
Chloroform	3.30
Pyridine	0.03
Oil of peppermint	0.02
Lodoform	0.02
Butyric acid	0.009
Propyl mercaptan	0.006
Artificial musk	0.00004
Methyl mercaptan	0.0000004

Odor-producing molecules are generally small, containing from 3 to 20 carbon atoms, and molecules with the same number of carbon atoms but different structural configurations have different odors. Relatively high water and lipid solubility are characteristic of substances with strong odors. Some common abnormalities in odor detection are described in Clinical Box 14–1.

Clinical Box 14–1

Abnormalities in Odor Detection

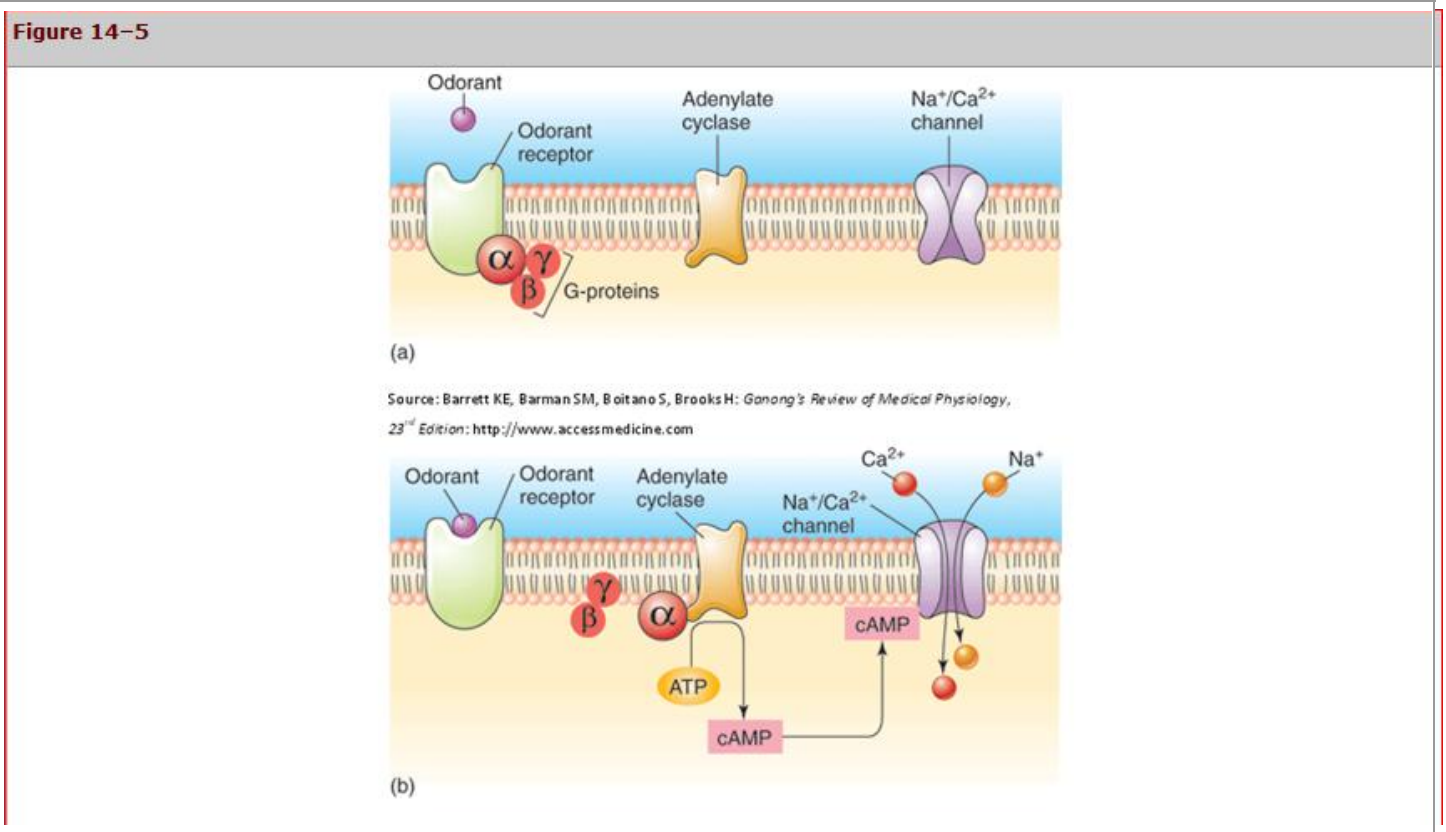
Anosmia (inability to smell) and **hyposmia** or **hypesthesia** (diminished olfactory sensitivity) can result from simple nasal congestion or be a sign of a more serious problem including damage to the olfactory nerves due to fractures of the cribriform plate, tumors such as neuroblastomas or meningiomas, or infections (such as abscesses). Alzheimer disease can also damage the olfactory nerves. Aging is also associated with abnormalities in smell sensation; more than 75% of humans over the age of 80 have an impaired ability to identify smells. **Hyperosmia** (enhanced olfactory sensitivity) is less common than loss of smell, but pregnant women commonly become oversensitive to smell. **Dysosmia** (distorted sense of smell) can be caused by several disorders including sinus infections, partial damage to the olfactory nerves, and poor dental hygiene.

Signal Transduction

The olfactory system has received considerable attention in recent years because of the intriguing biologic question of how a simple sense organ such as the olfactory epithelium and its brain representation, which apparently lacks a high degree of complexity, can mediate discrimination of more than 10,000 different odors. One part of the answer to this question is that there are many different odorant receptors.

The genes that code for about 1000 different types of odorant receptors make up the largest gene family so far described in mammals—larger than the immunoglobulin and T-cell receptor gene families combined. The amino acid sequences of odorant receptors are very diverse, but all the odorant receptors are coupled to heterotrimeric G proteins. When an odorant molecule binds to its receptor, the G protein subunits (α, β, γ) dissociate (Figure 14–5). The α-subunit activates adenylate cyclase to catalyze the production of cAMP, which acts as a second messenger to open cation channels, causing an inward-directed Ca^{2+} current. This produces the graded receptor potential, which then leads to an action potential in the olfactory nerve.

Figure 14–5



Signal transduction in an odorant receptor. Olfactory receptors are G protein-coupled receptors that dissociate upon binding to the odorant. The α-subunit of G proteins activates adenylate cyclase to catalyze production of cAMP. cAMP acts as a second messenger to open cation channels. Inward diffusion of Na^+ and Ca^{2+} produces depolarization.

(From Fox SI: *Human Physiology.* McGraw-Hill, 2008.)

A second part of the answer to the question of how 10,000 different odors can be detected lies in the neural

organization of the olfactory pathway. Although there are millions of olfactory sensory neurons, each expresses only one of the 1000 different odorant receptors. Each neuron projects to one or two glomeruli (Figure 14–3). This provides a distinct two-dimensional map in the olfactory bulb that is unique to the odorant. The mitral cells with their glomeruli project to different parts of the olfactory cortex.

The olfactory glomeruli demonstrate lateral inhibition mediated by periglomerular cells and granule cells. This sharpens and focuses olfactory signals. In addition, the extracellular field potential in each glomerulus oscillates, and the granule cells appear to regulate the frequency of the oscillation. The exact function of the oscillation is unknown, but it probably also helps to focus the olfactory signals reaching the cortex.

Odorant-Binding Proteins

In contrast to the low threshold for olfactory stimulation when the olfactory epithelium is intact, single olfactory receptors that have been patch-clamped have a relatively high threshold and a long latency. In addition, lipophilic odor-producing molecules must traverse the hydrophilic mucus in the nose to reach the receptors. These facts led to the suggestion that the olfactory mucus might contain one or more **odorant-binding proteins (OBP)** that concentrate the odorants and transfer them to the receptors. An 18-kDa OBP that is unique to the nasal cavity has been isolated, and other related proteins probably exist. The protein has considerable homology to other proteins in the body that are known to be carriers for small lipophilic molecules. A similar binding protein appears to be associated with taste.

Vomeronasal Organ

In rodents and various other mammals, the nasal cavity contains another patch of olfactory epithelium located along the nasal septum in a well-developed **vomeronasal organ.** This structure is concerned with the perception of odors that act as **pheromones.** Vomeronasal sensory neurons project to the **accessory olfactory bulb** and from there primarily to areas in the amygdala and hypothalamus that are concerned with reproduction and ingestive behavior. Vomeronasal input has major effects on these functions. An example is pregnancy block in mice; the pheromones of a male from a different strain prevent pregnancy as a result of mating with that male, but mating with a mouse of the same strain does not produce blockade. The vomeronasal organ has about 100 G protein-coupled odorant receptors that differ in structure from those in the rest of the olfactory epithelium.

The organ is not well developed in humans, but an anatomically separate and biochemically unique area of olfactory epithelium occurs in a pit in the anterior third of the nasal septum, which appears to be a homologous structure. There is evidence for the existence of pheromones in humans, and there is a close relationship between smell and sexual function. Perfume advertisements bear witness to this. The sense of smell is said to be more acute in women than in men, and in women it is most acute at the time of ovulation. Smell, and to a lesser extent, taste, have a unique ability to trigger long-term memories, a fact noted by novelists and documented by experimental psychologists.

Sniffing

The portion of the nasal cavity containing the olfactory receptors is poorly ventilated in humans. Most of the air normally moves smoothly over the turbinates with each respiratory cycle, although eddy currents pass some air over the olfactory epithelium. These eddy currents are probably set up by convection as cool air strikes the warm mucosal surfaces. The amount of air reaching this region is greatly increased by sniffing, an action that includes contraction of the lower part of the nares on the septum, deflecting the airstream upward. Sniffing is a semireflex response that usually occurs when a new odor attracts attention.

Role of Pain Fibers in the Nose

Naked endings of many trigeminal pain fibers are found in the olfactory epithelium. They are stimulated by irritating substances and leads to the characteristic "odor" of such substances as peppermint, menthol, and chlorine. Activation of these endings by nasal irritants also initiates sneezing, lacrimation, respiratory inhibition, and other reflexes.

Adaptation

It is common knowledge that when one is continuously exposed to even the most disagreeable odor, perception of the odor decreases and eventually ceases. This sometimes beneficent phenomenon is due to the fairly rapid adaptation, or desensitization, that occurs in the olfactory system. It is mediated by Ca^{2+} acting via calmodulin on **cyclic nucleotide-gated (CNG)** ion channels. When the CNG A4 subunit is knocked out, adaptation is slowed.

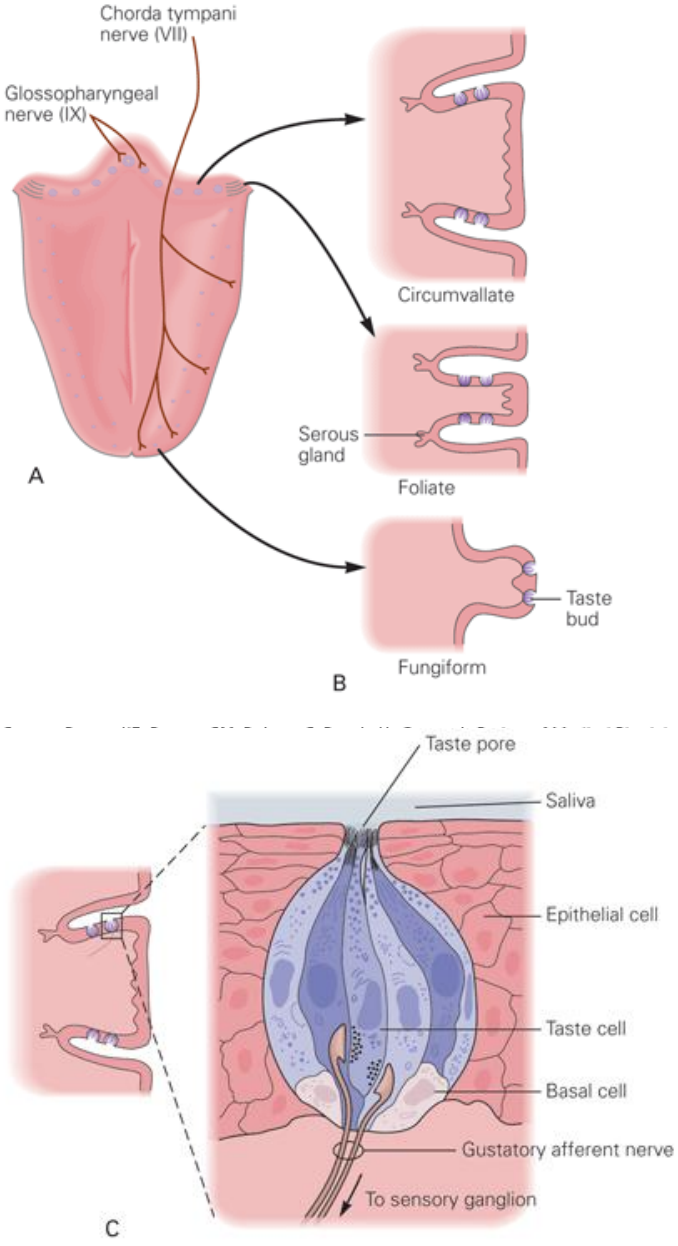
Taste

Taste Buds

The specialized sense organ for taste (gustation) consists of approximately 10,000 **taste buds,** which are ovoid bodies measuring 50–70 μm. There are four morphologically distinct types of cells within each taste bud: basal cells, dark cells, light cells, and intermediate cells (Figure 14–6). The latter three cell types are all referred to as **Type I, II, and III taste cells.** They are the sensory neurons that respond to taste stimuli or **tastants.** The three cell types may represent various stages of differentiation of developing taste cells, with the light cells being the most mature. Alternatively, each cell type might represent different cell lineages. The apical ends of taste cells have microvilli that project into the taste pore, a small opening on the dorsal surface of the tongue where tastes cells are exposed to the oral contents. Each taste bud is innervated by about 50 nerve fibers, and conversely, each nerve fiber receives input from an average of five taste buds. The basal cells arise from the epithelial cells surrounding the taste bud. They differentiate into new taste cells, and the old cells are continuously replaced with a half-time of about 10 days. If the sensory nerve is cut, the taste buds it innervates degenerate and eventually disappear.

Figure 14–6

Figure 14–6



Taste buds located in papillae of the human tongue. A) Taste buds on the anterior two-thirds of the tongue are innervated by the chorda tympani branch of the facial nerve; those on the posterior one-third of the tongue are innervated by the lingual branch of the glossopharyngeal nerve. **B)** The three major types of papillae (circumvallate, foliate, and fungiform) are located on specific parts of the tongue. **C)** Taste buds are composed of basal stem cells and three types of taste cells (dark, light, and intermediate). Taste cells extend from the base of the taste bud to the taste pore, where microvilli contact tastants dissolved in saliva and mucus.

(Modified from Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

In humans, the taste buds are located in the mucosa of the epiglottis, palate, and pharynx and in the walls of **papillae** of the tongue (Figure 14–6). The **fungiform papillae** are rounded structures most numerous near the tip of the tongue; the **circumvallate papillae** are prominent structures arranged in a V on the back of the

tongue; the **foliate papillae** are on the posterior edge of the tongue. Each fungiform papilla has up to five taste buds, mostly located at the top of the papilla, while each vallate and foliate papilla contain up to 100 taste buds, mostly located along the sides of the papillae.

Taste Pathways

The sensory nerve fibers from the taste buds on the anterior two-thirds of the tongue travel in the chorda tympani branch of the facial nerve, and those from the posterior third of the tongue reach the brain stem via the glossopharyngeal nerve (Figure 14–7). The fibers from areas other than the tongue (eg, pharynx) reach the brain stem via the vagus nerve. On each side, the myelinated but relatively slowly conducting taste fibers in these three nerves unite in the gustatory portion of the **nucleus of the solitary tract (NTS)** in the medulla oblongata (Figure 14–7). From there, axons of second-order neurons ascend in the ipsilateral medial lemniscus and, in primates, pass directly to the ventral posteromedial nucleus of the thalamus. From the thalamus, the axons of the third-order neurons pass to neurons in the anterior insula and the frontal operculum in the ipsilateral cerebral cortex. This region is rostral to the face area of the postcentral gyrus, which is probably the area that mediates conscious perception of taste and taste discrimination.

Figure 14–7

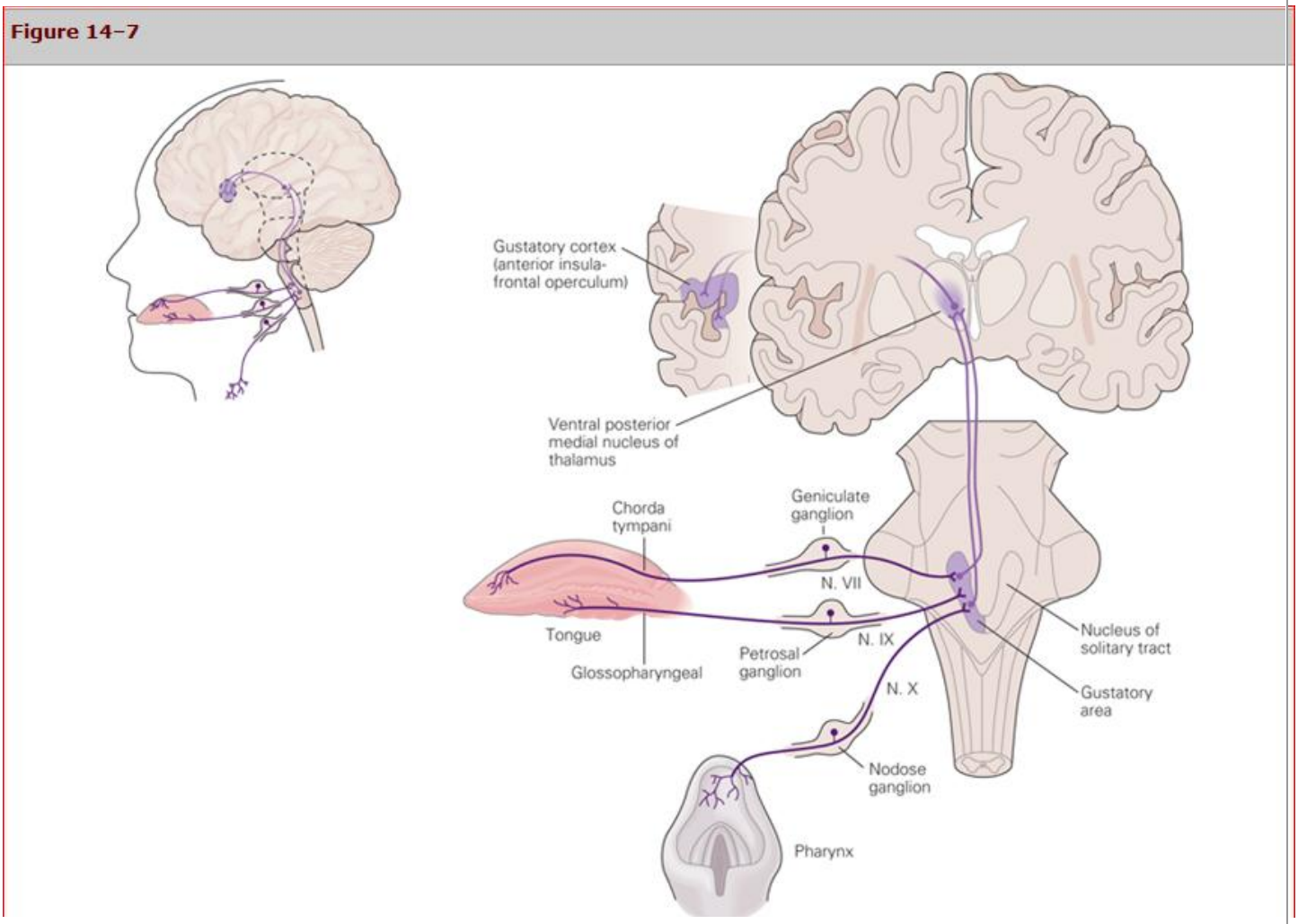


Diagram of taste pathways. Signals from the taste buds travel via different nerves to gustatory areas of the nucleus of the solitary tract which relays information to the thalamus; the thalamus projects to the gustatory

cortex.

(Modified from Kandel ER, Schwartz JH, Jessell TM [editors]: *Principles of Neural Science,* 4th ed. McGraw-Hill, 2000.)

Basic Taste Modalities

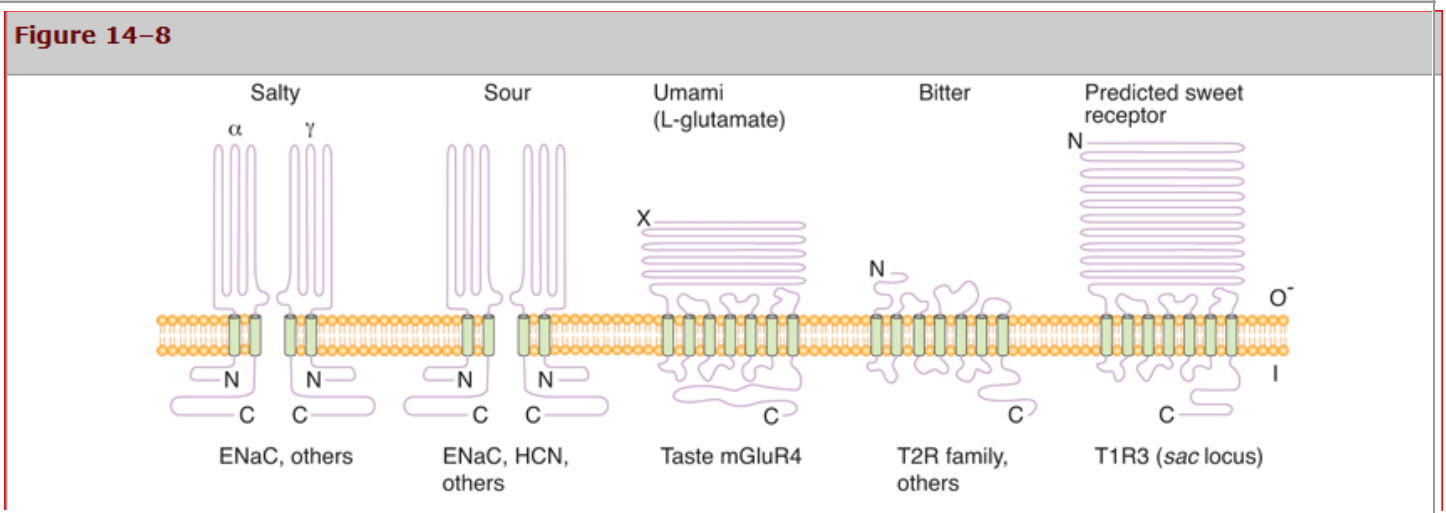
Humans have five established basic tastes: **sweet, sour, bitter, salt,** and **umami.** It used to be thought that the surface of the tongue had special areas for each of the first four of these sensations, but it is now clear that all tastants are sensed from all parts of the tongue and adjacent structures. Afferent nerves to the NTS contain fibers from all types of taste receptors, without any clear localization of types.

The fifth taste sense, umami, was recently added to the four classic tastes. This taste has actually been known for almost 100 years, and it became established once its receptor was identified. It is triggered by glutamate and particularly by the monosodium glutamate (MSG) used so extensively in Asian cooking. The taste is pleasant and sweet but differs from the standard sweet taste.

Taste Receptors & Transduction

The putative receptors for taste are shown diagrammatically in Figure 14–8. The salty taste is triggered by NaCl. Salt-sensitive taste is mediated by a Na^+-selective channel known as **ENaC,** the amiloride-sensitive epithelial sodium channel. The entry of Na^+ into the salt receptors depolarizes the membrane, generating the receptor potential. In humans, the amiloride sensitivity of salt taste is less pronounced than in some species, suggesting that there are additional mechanisms to activate salt-sensitive receptors.

Figure 14–8



Signal transduction in taste receptors. Salt-sensitive taste is mediated by a Na^+-selective channel (ENaC); sour taste is mediated by H^+ ions permeable to ENaCs; umami taste is mediated by glutamate acting on a metabotropic receptor, mGluR4; bitter taste is mediated by the T2R family of G protein-coupled receptors; sweet taste may be dependent on the T1R3 family of G protein-coupled receptors which couple to the G protein gustducin.

(Modified from Lindemann B: Receptors and transduction in taste. Nature 2001;413:219.)

The sour taste is triggered by protons (H^+ ions). ENaCs permit the entry of protons and may contribute to the

sensation of sour taste. The H^+ ions can also bind to and block a K^+-sensitive channel. The fall in K^+ permeability can depolarize the membrane. Also, **HCN,** a hyperpolarization-activated cyclic nucleotide-gated cation channel, and other mechanisms may contribute to sour transduction.

Umami taste is due to activation of a truncated metabotropic glutamate receptor, **mGluR4,** in the taste buds. The way activation of the receptor produces depolarization is unsettled. Glutamate in food may also activate ionotropic glutamate receptors to depolarize umami receptors.

Bitter taste is produced by a variety of unrelated compounds. Many of these are poisons, and bitter taste serves as a warning to avoid them. Some bitter compounds bind to and block K^+-selective channels. Many G protein-linked receptors in the human genome are taste receptors (T2R family) and are stimulated by bitter substances such as strychnine. In some cases, these receptors couple to the heterotrimeric G protein, **gustducin.** Gustducin lowers cAMP and increases the formation of inositol phosphates which could lead to depolarization. Some bitter compounds are membrane permeable and may not involve G proteins; quinine is an example.

Substances that taste sweet also act via the G protein gustducin. The T1R3 family of G protein-coupled receptors is expressed by about 20% of taste cells, some of which also express gustducin. Sugars taste sweet, but so do compounds such as saccharin that have an entirely different structure. It appears at present that natural sugars such as sucrose and synthetic sweeteners act via different receptors on gustducin. Like the bitter-responsive receptors, sweet-responsive receptors act via cyclic nucleotides and inositol phosphate metabolism.

Taste Thresholds & Intensity Discriminations

The ability of humans to discriminate differences in the intensity of tastes, like intensity discrimination in olfaction, is relatively crude. A 30% change in the concentration of the substance being tasted is necessary before an intensity difference can be detected. The threshold concentrations of substances to which the taste buds respond vary with the particular substance (Table 14–2).

Table 14–2 Some Taste Thresholds.

Substance	Taste	Threshold Concentration (µmol/L)
Hydrochloric acid	Sour	100
Sodium chloride	Salt	2000
Strychnine hydrochloride	Bitter	1.6
Glucose	Sweet	80,000
Sucrose	Sweet	10,000
Saccharin	Sweet	23

A protein that binds taste-producing molecules has been cloned. It is produced by **Ebner gland** that secretes mucus into the cleft around vallate papillae (Figure 14–6) and probably has a concentrating and transport function similar to that of the OBP described for olfaction. Some common abnormalities in taste detection are described in Clinical Box 14–2.

Clinical Box 14–2

Abnormalities in Taste Detection
Ageusia (absence of the sense of taste) and **hypogeusia** (diminished taste sensitivity) can be caused by damage to the lingual or glossopharyngeal nerve. Neurological disorders such as vestibular schwannoma, Bell palsy, familial dysautonomia, multiple sclerosis, and certain infections (eg, primary amoeboid meningoencephalopathy) can also cause problems with taste sensitivity. Ageusia can also be an adverse side effect of various drugs including cisplatin and captopril or vitamin B_3 or zinc deficiencies. Aging and tobacco abuse also contribute to diminished taste. **Dysgeusia** or **parageusia** (unpleasant perception of taste) causes a metallic, salty, foul, or rancid taste. In many cases, dysgeusia is a temporary problem. Factors contributing to ageusia or hypogeusia can also lead to abnormal taste sensitivity.

Variation & after Effects

Taste exhibits after reactions and contrast phenomena that are similar in some ways to visual after images and contrasts. Some of these are chemical "tricks," but others may be true central phenomena. A taste modifier protein, **miraculin,** has been discovered in a plant. When applied to the tongue, this protein makes acids taste sweet.

Animals, including humans, form particularly strong aversions to novel foods if eating the food is followed by illness. The survival value of such aversions is apparent in terms of avoiding poisons.

Chapter Summary

- Olfactory sensory neurons, supporting (sustentacular) cells, and basal stem cells are located in the olfactory epithelium within the upper portion of the nasal cavity.
- The cilia located on the dendritic knob of the olfactory sensory neuron contain odorant receptors which are coupled to heterotrimeric G proteins.
- Axons of olfactory sensory neurons contact the dendrites of mitral and tufted cells in the olfactory bulbs to form olfactory glomeruli.
- Information from the olfactory bulb travels via the lateral olfactory stria directly to the olfactory cortex, including the anterior olfactory nucleus, olfactory tubercle, piriform cortex, amygdala, and entorhinal cortex.
- Taste buds are the specialized sense organs for taste and are comprised of basal stem cells and three types of taste cells (dark cells, light cells, and intermediate cells). The three types of taste cells may represent various stages of differentiation of developing taste cells, with the light cells being the most mature. Taste buds are located in the mucosa of the epiglottis, palate, and pharynx and in the walls of papillae of the tongue.
- There are taste receptors for sweet, sour, bitter, salt, and umami. Signal transduction mechanisms include passage through ion channels, binding to and blocking ion channels, and second messenger systems.
- The afferents from taste buds in the tongue travel via the seventh, ninth, and tenth cranial nerves to synapse in the nucleus of the tractus solitarius. From there, axons ascend via the ipsilateral medial lemniscus to the ventral posteromedial nucleus of the thalamus, and on to the anterior insula and frontal operculum in the ipsilateral cerebral cortex.

Chapter Resources

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