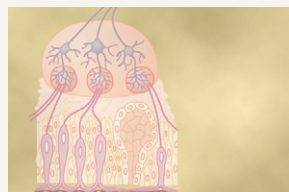


The Chemical Senses— Taste and Smell



The senses of taste and smell allow us to separate undesirable or even lethal foods from those that are pleasant to eat and nutritious. The sense of smell also allows animals to recognize the proximity of other animals or even individuals among animals. Finally, both senses are strongly tied to primitive emotional and behavioral functions of our nervous systems.

Sense of Taste

Taste is mainly a function of the *taste buds* in the mouth, but it is common experience that one's sense of smell also contributes strongly to taste perception. In addition, the texture of food, as detected by tactual senses of the mouth, and the presence of substances in the food that stimulate pain endings, such as pepper, greatly alter the taste experience. The importance of taste lies in the fact that it allows a person to select food in accord with desires and often in accord with the body tissues' metabolic need for specific substances.

Primary Sensations of Taste

The identities of the specific chemicals that excite different taste receptors are not all known. Even so, psychophysiologic and neurophysiologic studies have identified at least 13 possible or probable chemical receptors in the taste cells, as follows: 2 sodium receptors, 2 potassium receptors, 1 chloride receptor, 1 adenosine receptor, 1 inosine receptor, 2 sweet receptors, 2 bitter receptors, 1 glutamate receptor, and 1 hydrogen ion receptor.

For practical analysis of taste, the aforementioned receptor capabilities have also been grouped into five general categories called the *primary sensations of taste*. They are *sour*, *salty*, *sweet*, *bitter*, and “*umami*.”

A person can perceive hundreds of different tastes. They are all supposed to be combinations of the elementary taste sensations, just as all the colors we can see are combinations of the three primary colors, as described in Chapter 50.

Sour Taste. The sour taste is caused by acids, that is, by the hydrogen ion concentration, and the intensity of this taste sensation is approximately proportional to the *logarithm of the hydrogen ion concentration*. That is, the more acidic the food, the stronger the sour sensation becomes.

Salty Taste. The salty taste is elicited by ionized salts, mainly by the sodium ion concentration. The quality of the taste varies somewhat from one salt to another, because some salts elicit other taste sensations in addition to saltiness. The cations of the salts, especially sodium cations, are mainly responsible for the salty taste, but the anions also contribute to a lesser extent.

Sweet Taste. The sweet taste is not caused by any single class of chemicals. Some of the types of chemicals that cause this taste include sugars, glycols, alcohols, aldehydes, ketones, amides, esters, some amino acids, some small proteins,

Table 53–1

Relative Taste Indices of Different Substances

Sour Substances	Index	Bitter Substances	Index	Sweet Substances	Index	Salty Substances	Index
Hydrochloric acid	1	Quinine	1	Sucrose	1	NaCl	1
Formic acid	1.1	Brucine	11	1-Propoxy-2-amino-4-nitrobenzene	5000	NaF	2
Chloracetic acid	0.9	Strychnine	3.1	Saccharin	675	CaCl ₂	1
Acetylacetic acid	0.85	Nicotine	1.3	Chloroform	40	NaBr	0.4
Lactic acid	0.85	Phenylthiourea	0.9	Fructose	1.7	NaI	0.35
Tartaric acid	0.7	Caffeine	0.4	Alanine	1.3	LiCl	0.4
Malic acid	0.6	Veratrine	0.2	Glucose	0.8	NH ₄ Cl	2.5
Potassium H tartrate	0.58	Pilocarpine	0.16	Maltose	0.45	KCl	0.6
Acetic acid	0.55	Atropine	0.13	Galactose	0.32		
Citric acid	0.46	Cocaine	0.02	Lactose	0.3		
Carbonic acid	0.06	Morphine	0.02				

From Pfaffman C: Handbook of Physiology. Sec I, Vol 1. Baltimore: Williams & Wilkins, 1959, p. 507.

sulfonic acids, halogenated acids, and inorganic salts of lead and beryllium. Note specifically that most of the substances that cause a sweet taste are organic chemicals. It is especially interesting that slight changes in the chemical structure, such as addition of a simple radical, can often change the substance from sweet to bitter.

Bitter Taste. The bitter taste, like the sweet taste, is not caused by any single type of chemical agent. Here again, the substances that give the bitter taste are almost entirely organic substances. Two particular classes of substances are especially likely to cause bitter taste sensations: (1) long-chain organic substances that contain nitrogen, and (2) alkaloids. The alkaloids include many of the drugs used in medicines, such as quinine, caffeine, strychnine, and nicotine.

Some substances that at first taste sweet have a bitter aftertaste. This is true of saccharin, which makes this substance objectionable to some people.

The bitter taste, when it occurs in high intensity, usually causes the person or animal to reject the food. This is undoubtedly an important function of the bitter taste sensation, because many deadly toxins found in poisonous plants are alkaloids, and virtually all of these cause intensely bitter taste, usually followed by rejection of the food.

Umami Taste. *Umami* is a Japanese word (meaning “delicious”) designating a pleasant taste sensation that is qualitatively different from sour, salty, sweet, or bitter. Umami is the dominant taste of food containing L-glutamate, such as meat extracts and aging cheese, and some physiologists consider it to be a separate, fifth category of primary taste stimuli.

A taste receptor for L-glutamate may be related to one of the glutamate receptors expressed in neuronal synapses of the brain. However, the precise molecular mechanisms responsible for umami taste are still unclear.

Threshold for Taste

The threshold for stimulation of the sour taste by hydrochloric acid averages 0.0009 N; for stimulation of the salty taste by sodium chloride, 0.01 M; for the sweet taste by sucrose, 0.01 M; and for the bitter taste by quinine, 0.000008 M. Note especially how much more sensitive is the bitter taste sense than all the others, which would be expected, because this sensation provides an important protective function against many dangerous toxins in food.

Table 53–1 gives the relative taste indices (the reciprocals of the taste thresholds) of different substances. In this table, the intensities of four of the primary sensations of taste are referred, respectively, to the intensities of the taste of hydrochloric acid, quinine, sucrose, and sodium chloride, each of which is arbitrarily chosen to have a taste index of 1.

Taste Blindness. Some people are taste blind for certain substances, especially for different types of thiourea compounds. A substance used frequently by psychologists for demonstrating taste blindness is *phenylthiocarbamide*, for which about 15 to 30 per cent of all people exhibit taste blindness; the exact percentage depends on the method of testing and the concentration of the substance.

Taste Bud and Its Function

Figure 53–1 shows a taste bud, which has a diameter of about $\frac{1}{30}$ millimeter and a length of about $\frac{1}{16}$ millimeter. The taste bud is composed of about 50 modified epithelial cells, some of which are supporting cells called *sustentacular cells* and others of which are *taste cells*. The taste cells are continually being replaced by mitotic division of surrounding epithelial cells, so that some taste cells are young cells. Others are mature cells that lie toward the center of the bud; these soon break up and dissolve. The life span of each taste cell

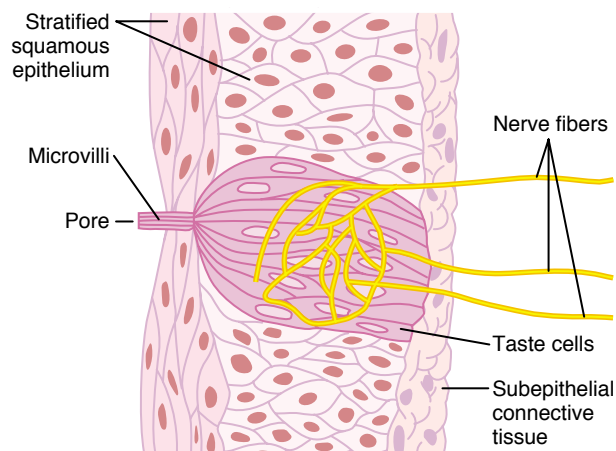


Figure 53-1

Taste bud.

is about 10 days in lower mammals but is unknown for humans.

The outer tips of the taste cells are arranged around a minute *taste pore*, shown in Figure 53-1. From the tip of each taste cell, several *microvilli*, or *taste hairs*, protrude outward into the taste pore to approach the cavity of the mouth. These microvilli provide the receptor surface for taste.

Interwoven around the bodies of the taste cells is a branching terminal network of *taste nerve fibers* that are stimulated by the taste receptor cells. Some of these fibers invaginate into folds of the taste cell membranes. Many vesicles form beneath the cell membrane near the fibers. It is believed that these vesicles contain a neurotransmitter substance that is released through the cell membrane to excite the nerve fiber endings in response to taste stimulation.

Location of the Taste Buds. The taste buds are found on three types of papillae of the tongue, as follows: (1) A large number of taste buds are on the walls of the troughs that surround the circumvallate papillae, which form a V line on the surface of the posterior tongue. (2) Moderate numbers of taste buds are on the fungiform papillae over the flat anterior surface of the tongue. (3) Moderate numbers are on the foliate papillae located in the folds along the lateral surfaces of the tongue. Additional taste buds are located on the palate, and a few are found on the tonsillar pillars, on the epiglottis, and even in the proximal esophagus. Adults have 3000 to 10,000 taste buds, and children have a few more. Beyond the age of 45 years, many taste buds degenerate, causing the taste sensation to become progressively less critical in old age.

Specificity of Taste Buds for a Primary Taste Stimulus. Micro-electrode studies from single taste buds show that each taste bud usually *responds mostly to one of the five primary taste stimuli when the taste substance is in low concentration*. But at high concentration, most buds can be excited by two or more of the primary taste

stimuli, as well as by a few other taste stimuli that do not fit into the “primary” categories.

Mechanism of Stimulation of Taste Buds

Receptor Potential. The membrane of the taste cell, like that of most other sensory receptor cells, is negatively charged on the inside with respect to the outside. Application of a taste substance to the taste hairs causes partial loss of this negative potential—that is, the taste cell becomes *depolarized*. In most instances, the decrease in potential, within a wide range, is approximately proportional to the logarithm of concentration of the stimulating substance. This *change in electrical potential* in the taste cell is called the *receptor potential* for taste.

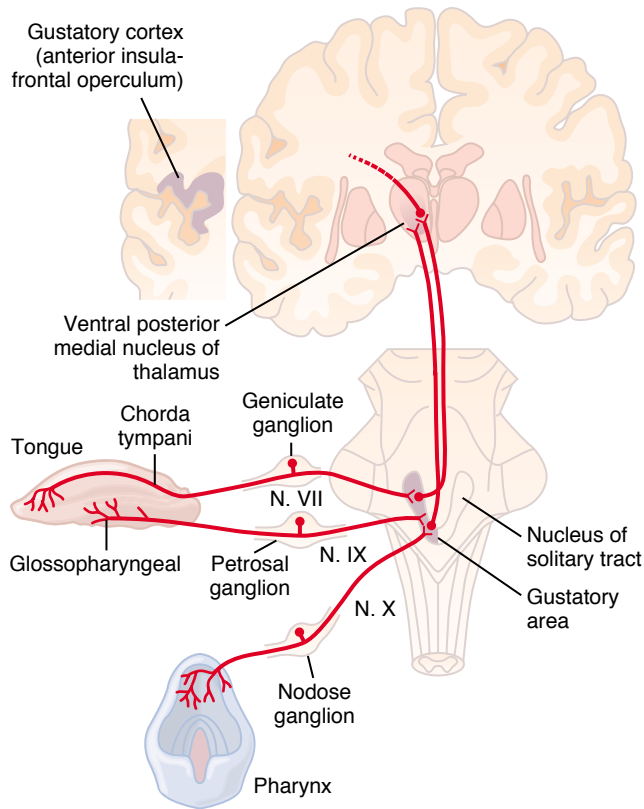
The mechanism by which most stimulating substances react with the taste villi to initiate the receptor potential is by binding of the taste chemical to a protein receptor molecule that lies on the outer surface of the taste receptor cell near to or protruding through a villus membrane. This, in turn, opens ion channels, which allows positively charged sodium ions or hydrogen ions to enter and depolarize the normal negativity of the cell. Then the taste chemical itself is gradually washed away from the taste villus by the saliva, which removes the stimulus.

The type of receptor protein in each taste villus determines the type of taste that will be perceived. For sodium ions and hydrogen ions, which elicit salty and sour taste sensations, respectively, the receptor proteins open specific ion channels in the apical membranes of the taste cells, thereby activating the receptors. However, for the sweet and bitter taste sensations, the portions of the receptor protein molecules that protrude through the apical membranes activate *second-messenger transmitter substances* inside the taste cells, and these second messengers cause intracellular chemical changes that elicit the taste signals.

Generation of Nerve Impulses by the Taste Bud. On first application of the taste stimulus, the rate of discharge of the nerve fibers from taste buds rises to a peak in a small fraction of a second but then adapts within the next few seconds back to a lower, steady level as long as the taste stimulus remains. Thus, a strong immediate signal is transmitted by the taste nerve, and a weaker continuous signal is transmitted as long as the taste bud is exposed to the taste stimulus.

Transmission of Taste Signals into the Central Nervous System

Figure 53-2 shows the neuronal pathways for transmission of taste signals from the tongue and pharyngeal region into the central nervous system. Taste impulses from the anterior two thirds of the tongue pass first into the *lingual nerve*, then through the *chorda tympani* into the *facial nerve*, and finally into the *tractus solitarius* in the brain stem. Taste sensations from the circumvallate papillae on the back of the

**Figure 53-2**

Transmission of taste signals into the central nervous system.

tongue and from other posterior regions of the mouth and throat are transmitted through the *glossopharyngeal nerve* also into the *tractus solitarius*, but at a slightly more posterior level. Finally, a few taste signals are transmitted into the *tractus solitarius* from the base of the tongue and other parts of the pharyngeal region by way of the *vagus nerve*.

All taste fibers synapse in the posterior brain stem in the *nuclei of the tractus solitarius*. These nuclei send second-order neurons to a small area of the *ventral posterior medial nucleus of the thalamus*, located slightly medial to the thalamic terminations of the facial regions of the dorsal column–medial lemniscal system. From the thalamus, third-order neurons are transmitted to the *lower tip of the postcentral gyrus in the parietal cerebral cortex*, where it curls deep into the *sylvian fissure*, and into the adjacent *opercular insular area*. This lies slightly lateral, ventral, and rostral to the area for tongue tactile signals in cerebral somatic area I. From this description of the taste pathways, it is evident that they closely parallel the somatosensory pathways from the tongue.

Taste Reflexes Integrated in the Brain Stem. From the tractus solitarius, many taste signals are transmitted within the brain stem itself directly into the *superior and inferior salivatory nuclei*, and these areas transmit signals to the submandibular, sublingual, and parotid

glands to help control the secretion of saliva during the ingestion and digestion of food.

Adaptation of Taste. Everyone is familiar with the fact that taste sensations adapt rapidly, often almost completely within a minute or so of continuous stimulation. Yet, from electrophysiologic studies of taste nerve fibers, it is clear that adaptation of the taste buds themselves usually accounts for no more than about half of this. Therefore, the final extreme degree of adaptation that occurs in the sensation of taste almost certainly occurs in the central nervous system itself, although the mechanism and site of this are not known. At any rate, it is a mechanism different from that of most other sensory systems, which adapt almost entirely at the receptors.

Taste Preference and Control of the Diet

Taste preference simply means that an animal will choose certain types of food in preference to others, and the animal automatically uses this to help control the type of diet it eats. Furthermore, its taste preferences often change in accord with the body's need for certain specific substances.

The following experiments demonstrate this ability of animals to choose food in accord with the needs of their bodies. First, adrenalectomized, *salt-depleted* animals automatically select drinking water with a high concentration of sodium chloride in preference to pure water, and this is often sufficient to supply the needs of the body and prevent salt-depletion death. Second, an animal given injections of excessive amounts of insulin develops a depleted blood sugar, and the animal automatically chooses the sweetest food from among many samples. Third, calcium-depleted parathyroidectomized animals automatically choose drinking water with a high concentration of calcium chloride.

The same phenomena are also observed in everyday life. For instance, the “salt licks” of desert regions are known to attract animals from far and wide. Also, human beings reject any food that has an unpleasant affective sensation, which in many instances protects our bodies from undesirable substances.

The phenomenon of taste preference almost certainly results from some mechanism located in the central nervous system and not from a mechanism in the taste receptors themselves, although it is true that the receptors often become sensitized in favor of a needed nutrient. An important reason for believing that taste preference is mainly a central nervous system phenomenon is that previous experience with unpleasant or pleasant tastes plays a major role in determining one's taste preferences. For instance, if a person becomes sick soon after eating a particular type of food, the person generally develops a negative taste preference, or *taste aversion*, for that particular food thereafter; the same effect can be demonstrated in lower animals.

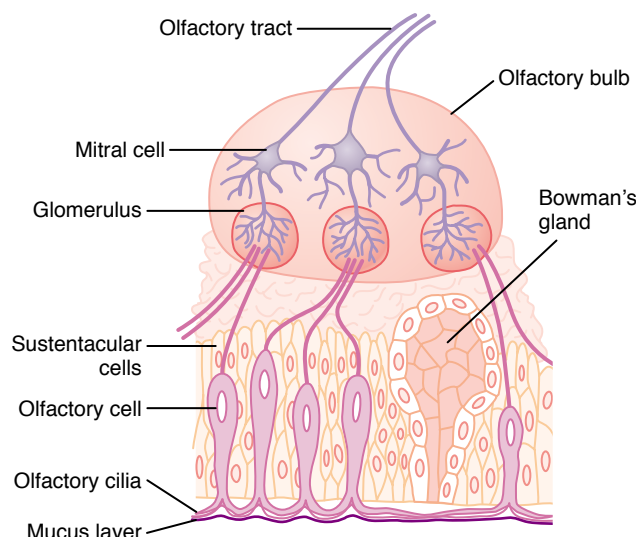


Figure 53–3

Organization of the olfactory membrane and olfactory bulb, and connections to the olfactory tract.

Sense of Smell

Smell is the least understood of our senses. This results partly from the fact that the sense of smell is a subjective phenomenon that cannot be studied with ease in lower animals. Another complicating problem is that the sense of smell is poorly developed in human beings in comparison with the sense of smell in many lower animals.

Olfactory Membrane

The olfactory membrane, the histology of which is shown in Figure 53–3, lies in the superior part of each nostril. Medially, the olfactory membrane folds downward along the surface of the superior septum; laterally, it folds over the superior turbinate and even over a small portion of the upper surface of the middle turbinate. In each nostril, the olfactory membrane has a surface area of about 2.4 square centimeters.

Olfactory Cells. The receptor cells for the smell sensation are the *olfactory cells* (see Figure 53–3), which are actually bipolar nerve cells derived originally from the central nervous system itself. There are about 100 million of these cells in the olfactory epithelium interspersed among *sustentacular cells*, as shown in Figure 53–3. The mucosal end of the olfactory cell forms a knob from which 4 to 25 *olfactory hairs* (also called *olfactory cilia*), measuring 0.3 micrometer in diameter and up to 200 micrometers in length, project into the mucus that coats the inner surface of the nasal cavity. These projecting olfactory cilia form a dense mat in the mucus, and it is these cilia that react to odors in the air and stimulate the olfactory cells, as discussed later. Spaced among the olfactory cells in the olfactory

membrane are many small *Bowman's glands* that secrete mucus onto the surface of the olfactory membrane.

Stimulation of the Olfactory Cells

Mechanism of Excitation of the Olfactory Cells. The portion of each olfactory cell that responds to the olfactory chemical stimuli is the *olfactory cilia*. The odorant substance, on coming in contact with the olfactory membrane surface, first diffuses into the mucus that covers the cilia. Then it binds with *receptor proteins* in the membrane of each cilium. Each receptor protein is actually a long molecule that threads its way through the membrane about seven times, folding inward and outward. The odorant binds with the portion of the receptor protein that folds to the outside. The inside of the folding protein, however, is coupled to a so-called *G-protein*, itself a combination of three subunits. On excitation of the receptor protein, an *alpha* subunit breaks away from the G-protein and immediately activates *adenylyl cyclase*, which is attached to the inside of the ciliary membrane near the receptor cell body. The activated cyclase, in turn, converts many molecules of intracellular *adenosine triphosphate* into *cyclic adenosine monophosphate* (cAMP). Finally, this cAMP activates another nearby membrane protein, a *gated sodium ion channel*, that opens its “gate” and allows large numbers of sodium ions to pour through the membrane into the receptor cell cytoplasm. The sodium ions increase the electrical potential in the positive direction inside the cell membrane, thus exciting the olfactory neuron and transmitting action potentials into the central nervous system by way of the *olfactory nerve*.

The importance of this mechanism for activating olfactory nerves is that it greatly multiplies the excitatory effect of even the weakest odorant. To summarize: (1) Activation of the receptor protein by the odorant substance activates the G-protein complex. (2) This, in turn, activates multiple molecules of adenylyl cyclase inside the olfactory cell membrane. (3) This causes the formation of many times more molecules of cAMP. (4) Finally, the cAMP opens still many times more sodium ion channels. Therefore, even the most minute concentration of a specific odorant initiates a cascading effect that opens extremely large numbers of sodium channels. This accounts for the exquisite sensitivity of the olfactory neurons to even the slightest amount of odorant.

In addition to the basic chemical mechanism by which the olfactory cells are stimulated, several physical factors affect the degree of stimulation. First, only volatile substances that can be sniffed into the nostrils can be smelled. Second, the stimulating substance must be at least slightly water soluble so that it can pass through the mucus to reach the olfactory cilia. Third, it is helpful for the substance to be at least slightly lipid soluble, presumably because lipid constituents of the cilium itself are a weak barrier to non-lipid-soluble odorants.

Membrane Potentials and Action Potentials in Olfactory Cells.

The membrane potential inside unstimulated olfactory cells, as measured by microelectrodes, averages about -55 millivolts. At this potential, most of the cells generate continuous action potentials at a very slow rate, varying from once every 20 seconds up to two or three per second.

Most odorants cause *depolarization* of the olfactory cell membrane, decreasing the negative potential in the cell from the normal level of -55 millivolts to -30 millivolts or less—that is, changing the voltage in the positive direction. Along with this, the number of action potentials increases to 20 to 30 per second, which is a high rate for the minute olfactory nerve fibers.

Over a wide range, the rate of olfactory nerve impulses changes approximately in proportion to the logarithm of the stimulus strength, which demonstrates that the olfactory receptors obey principles of transduction similar to those of other sensory receptors.

Adaptation. The olfactory receptors adapt about 50 per cent in the first second or so after stimulation. Thereafter, they adapt very little and very slowly. Yet we all know from our own experience that smell sensations adapt almost to extinction within a minute or so after entering a strongly odorous atmosphere. Because this psychological adaptation is far greater than the degree of adaptation of the receptors themselves, it is almost certain that most of the additional adaptation occurs within the central nervous system. This seems to be true for the adaptation of taste sensations as well.

A postulated neuronal mechanism for the adaptation is the following: Large numbers of centrifugal nerve fibers pass from the olfactory regions of the brain backward along the olfactory tract and terminate on special inhibitory cells in the olfactory bulb, the *granule cells*. It has been postulated that after the onset of an olfactory stimulus, the central nervous system quickly develops strong feedback inhibition to suppress relay of the smell signals through the olfactory bulb.

Search for the Primary Sensations of Smell

In the past, most physiologists were convinced that the many smell sensations are subserved by a few rather discrete primary sensations, in the same way that vision and taste are subserved by a few select primary sensations. Based on psychological studies, one attempt to classify these sensations is the following:

1. Camphoraceous
2. Musky
3. Floral
4. Pepperminty
5. Ethereal
6. Pungent
7. Putrid

It is certain that this list does not represent the true primary sensations of smell. In recent years, multiple clues, including specific studies of the genes that encode for the receptor proteins, suggest the existence

of at least 100 primary sensations of smell—a marked contrast to only three primary sensations of color detected by the eyes and only four or five primary sensations of taste detected by the tongue. Further support for the many primary sensations of smell is that people have been found who have *odor blindness* for single substances; such discrete odor blindness has been identified for more than 50 different substances. It is presumed that odor blindness for each substance represents lack of the appropriate receptor protein in olfactory cells for that particular substance.

“Affective Nature of Smell.” Smell, even more so than taste, has the affective quality of either *pleasantness* or *unpleasantness*. Because of this, smell is probably even more important than taste for the selection of food. Indeed, a person who has previously eaten food that disagreed with him or her is often nauseated by the smell of that same food on a second occasion. Conversely, perfume of the right quality can wreak havoc with human emotions. In addition, in some lower animals, odors are the primary excitant of sexual drive.

Threshold for Smell. One of the principal characteristics of smell is the minute quantity of stimulating agent in the air that can elicit a smell sensation. For instance, the substance *methylmercaptan* can be smelled when only one 25 trillionth of a gram is present in each milliliter of air. Because of this very low threshold, this substance is mixed with natural gas to give the gas an odor that can be detected when even small amounts of gas leak from a pipeline.

Gradations of Smell Intensities. Although the threshold concentrations of substances that evoke smell are extremely slight, for many (if not most) odorants, concentrations only 10 to 50 times above the threshold evoke maximum intensity of smell. This is in contrast to most other sensory systems of the body, in which the ranges of intensity discrimination are tremendous—for example, 500,000 to 1 in the case of the eyes and 1 trillion to 1 in the case of the ears. This difference might be explained by the fact that smell is concerned more with detecting the presence or absence of odors rather than with quantitative detection of their intensities.

Transmission of Smell Signals into the Central Nervous System

The olfactory portions of the brain were among the first brain structures developed in primitive animals, and much of the remainder of the brain developed around these olfactory beginnings. In fact, part of the brain that originally subserved olfaction later evolved into the basal brain structures that control emotions and other aspects of human behavior; this is the system we call the *limbic system*, discussed in Chapter 58.

Transmission of Olfactory Signals into the Olfactory Bulb. The *olfactory bulb* is shown in Figure 53–4. The olfactory nerve fibers leading backward from the bulb are called *cranial nerve I*, or the *olfactory tract*. However, in reality, both the tract and the bulb are an anterior outgrowth of brain tissue from the base of the brain; the bulbous enlargement at its end, the *olfactory bulb*, lies over the *cribriform plate*, separating the brain cavity from the upper reaches of the nasal cavity. The cribriform plate has multiple small perforations through which an equal number of small nerves pass upward from the olfactory membrane in the nasal cavity to enter the olfactory bulb in the cranial cavity. Figure 53–3 demonstrates the close relation between the *olfactory cells* in the olfactory membrane and the olfactory bulb, showing short axons from the olfactory cells terminating in multiple globular structures within the olfactory bulb called *glomeruli*. Each bulb has several thousand such glomeruli, each of which is the terminus for about 25,000 axons from olfactory cells. Each glomerulus also is the terminus for dendrites from about 25 large *mitral cells* and about 60 smaller *tufted cells*, the cell bodies of which lie in the olfactory bulb superior to the glomeruli. These dendrites receive synapses from the olfactory cell neurons, and the mitral and tufted cells send axons through the olfactory tract to transmit olfactory signals to higher levels in the central nervous system.

Some research has suggested that different glomeruli respond to different odors. It is possible that specific glomeruli are the real clue to the analysis of different odor signals transmitted into the central nervous system.

The Very Old, the Less Old, and the Newer Olfactory Pathways into the Central Nervous System

The olfactory tract enters the brain at the anterior junction between the mesencephalon and cerebrum; there, the tract divides into two pathways, as shown in Figure 53–4, one passing medially into the *medial olfactory area* of the brain stem, and the other passing laterally into the *lateral olfactory area*. The medial olfactory area represents a very old olfactory system, whereas the lateral olfactory area is the input to (1) a less old olfactory system and (2) a newer system.

The Very Old Olfactory System—The Medial Olfactory Area.

The medial olfactory area consists of a group of nuclei located in the midbasal portions of the brain immediately anterior to the hypothalamus. Most conspicuous are the *septal nuclei*, which are midline nuclei that feed into the hypothalamus and other primitive portions of the brain's limbic system. This is the brain area most concerned with basic behavior (described in Chapter 58).

The importance of this medial olfactory area is best understood by considering what happens in animals when the lateral olfactory areas on both sides of the brain are removed and only the medial system remains. The answer is that this hardly affects the more

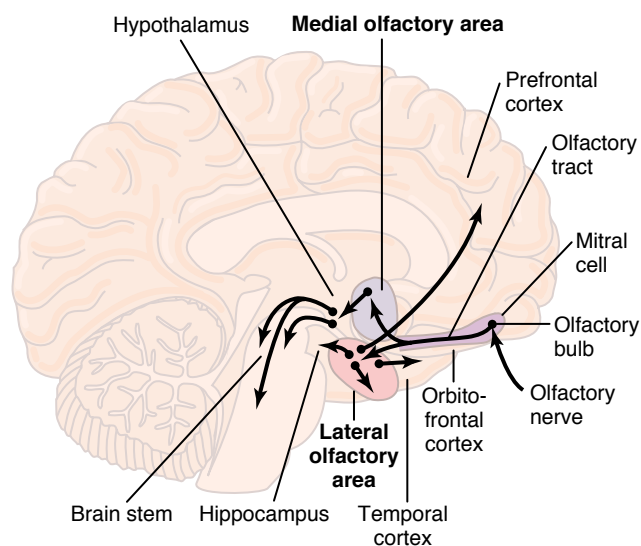


Figure 53–4

Neural connections of the olfactory system.

primitive responses to olfaction, such as licking the lips, salivation, and other feeding responses caused by the smell of food or by primitive emotional drives associated with smell. Conversely, removal of the lateral areas abolishes the more complicated olfactory conditioned reflexes.

The Less Old Olfactory System—The Lateral Olfactory Area.

The lateral olfactory area is composed mainly of the *prepyriform* and *pyriform cortex* plus the *cortical portion of the amygdaloid nuclei*. From these areas, signal pathways pass into almost all portions of the limbic system, especially into less primitive portions such as the hippocampus, which seem to be most important for learning to like or dislike certain foods depending on one's experiences with them. For instance, it is believed that this lateral olfactory area and its many connections with the limbic behavioral system cause a person to develop an absolute aversion to foods that have caused nausea and vomiting.

An important feature of the lateral olfactory area is that many signal pathways from this area also feed directly into an *older part of the cerebral cortex* called the *paleocortex* in the *anteromedial portion of the temporal lobe*. This is the only area of the entire cerebral cortex where sensory signals pass directly to the cortex without passing first through the thalamus.

The Newer Pathway. A newer olfactory pathway has now been found that passes through the thalamus, passing to the dorsomedial thalamic nucleus and then to the lateroposterior quadrant of the orbitofrontal cortex. Based on studies in monkeys, this newer system probably helps in the conscious analysis of odor.

Summary. Thus, there appear to be a *very old* olfactory system that subserves the basic olfactory reflexes, a *less old* system that provides automatic but partially learned control of food intake and aversion to toxic and unhealthy foods, and a *newer* system that is comparable to most of the other cortical sensory systems and is used for conscious perception and analysis of olfaction.

Centrifugal Control of Activity in the Olfactory Bulb by the Central Nervous System. Many nerve fibers that originate in the olfactory portions of the brain pass from the brain in the outward direction into the olfactory tract to the olfactory bulb (i.e., “centrifugally” from the brain to the periphery). These terminate on a large number of small *granule cells* located among the mitral and tufted cells in the olfactory bulb. The granule cells send inhibitory signals to the mitral and tufted cells. It is believed that this inhibitory feedback might be a means for sharpening one’s specific ability to distinguish one odor from another.

References

- Bermudez-Rattoni F: Molecular mechanisms of taste-recognition memory. *Nat Rev Neurosci* 5:209, 2004.
- Dohman HG: G proteins and pheromone signaling. *Annu Rev Physiol* 64:129, 2002.
- Gibson AD, Garbers DL: Guanylyl cyclases as a family of putative odorant receptors. *Annu Rev Neurosci* 23:417, 2000.
- Herness MS, Gilbertson TA: Cellular mechanisms of taste transduction. *Annu Rev Physiol* 61:873, 1999.
- Levine AS, Kotz CM, Gosnell BA: Sugars and fats: the neurobiology of preference. *J Nutr* 133:831S, 2003.
- Lledo PM, Gheusi G: Olfactory processing in a changing brain. *Neuroreport* 14:1655, 2003.
- Lowe G: Electrical signaling in the olfactory bulb. *Curr Opin Neurobiol* 13:476, 2003.
- Margolskee RF: Molecular mechanisms of bitter and sweet taste transduction. *J Biol Chem* 277:1, 2002.
- Matthews HR, Reisert J: Calcium, the two-faced messenger of olfactory transduction and adaptation. *Curr Opin Neurobiol* 13:469, 2003.
- Menini A, Lagostena L, Boccaccio A: Olfaction: from odorant molecules to the olfactory cortex. *News Physiol Sci* 19:101, 2004.
- Mombaerts P: Genes and ligands for odorant, vomeronasal and taste receptors. *Nat Rev Neurosci* 5:263, 2004.
- Mombaerts P: Odorant receptor gene choice in olfactory sensory neurons: the one receptor–one neuron hypothesis revisited. *Curr Opin Neurobiol* 14:31, 2004.
- Montmayeur JP, Matsunami H: Receptors for bitter and sweet taste. *Curr Opin Neurobiol* 12:366, 2002.
- Reed RR: The contribution of signaling pathways to olfactory organization and development. *Curr Opin Neurobiol* 13:482, 2003.
- Ronnett GV, Moon C: G proteins and olfactory signal transduction. *Annu Rev Physiol* 64:189, 2002.
- Sewards TV: Dual separate pathways for sensory and hedonic aspects of taste. *Brain Res Bull* 62:271, 2004.
- Smith DV, Margolskee RF: Making sense of taste. *Sci Am* 284:32, 2001.