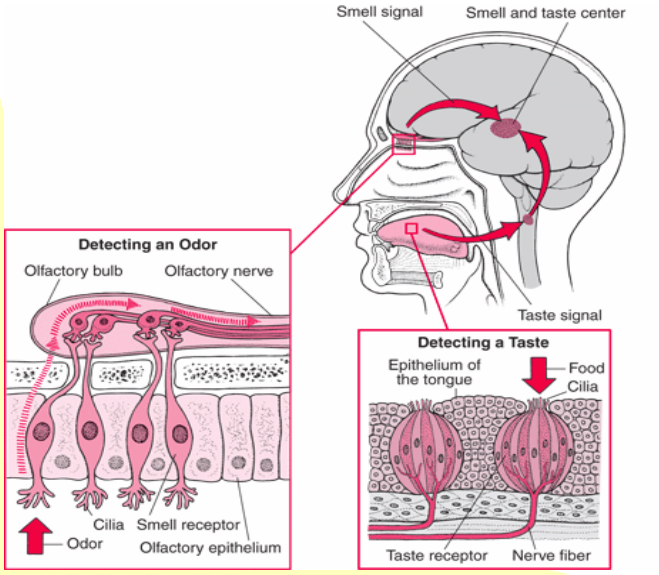
Introduction



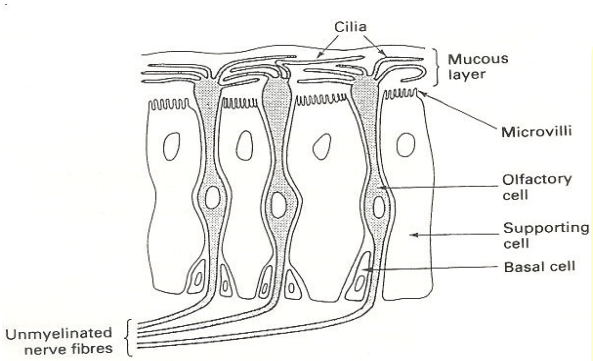
smell **(olfaction)** and taste **(gustation)** are specialized chemical senses. They are closely related, as evidenced by the common observation that the common cold not only depresses the sense of smell but also alters the sense of taste. The two senses are related to food intake: if the smell and taste of a food are agreeable, the food is ingested; but it is rejected if either or both its smell and taste are unacceptable. The combined effect of the smell and taste of a food is included under the *term flavour.* There are, however, differences between the two senses. While smell receptors are telereceptors (distance receivers), i.e. the sensation is coming from a distance outside the body and is projected to the environment, taste is entirely confined to the mouth. Smell pathways do not relay in the thalamus and do not reach the sensory cortex at the postcentral gyrus. Instead, they end in the part of the brain referred to previously as the rhinencephalon but now considered as part of the limbic system. Taste pathways, on the other hand, relay in the thalamus and are finally projected to the postcentral gyrus.

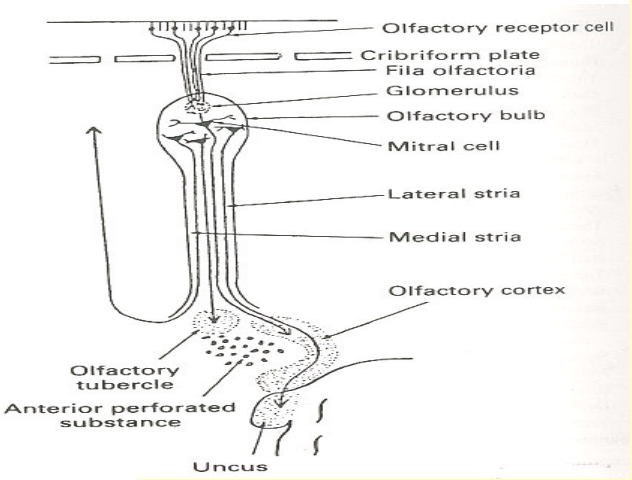
Smell

The sense of smell is developed in some animals, such as the dog and rabbit, which are referred to as macrosmatic, but is greatly reduced in primates, including humans, which are referred to as microsmatic. The olfactory mucosa in humans is located in the roof of the nasal cavity near the septum; it has a yellowish-brown colour. Its total area on both sides is about 5 cm^2. As air enters the nose, it is warmed by the lining epithelium, and convection or eddy currents arise from the airstream, passing through the lower part of the nose to reach the olfactory receptors in the roof.

The olfactory receptors are bipolar neurons, which are about 10-20 million in number. Their dendritic zone is expanded into **olfactory rods,** which end in cilia. Between the receptor neurons there are supporting cells; these end in microvilli and secrete mucus, which overlies the mucosa. The axons of the receptor neurons are unmyelinated and collected in bundles called **fila olfactoria,** which go through the holes in the cribriform plate of the ethmoid bone to enter the olfactory bulb.

The receptor axons synapse with **mitral** and **tufted cells** in the **glomeruli** of the bulbs. A great deal of spatial summation occurs at this step, with over 20 000 receptors synapsing with tens of mitral and tufted cells. From the mitral cells arise the lateral and intermediate **olfactory stria,** which end, respectively, in the ipsilateral **olfactory cortex** and **uncus** and the **olfactory tubercle.** The medial olfactory stria arise from the tufted cells and cross the midline in the anterior commissure to end on granular cells in the opposite bulb. These commissural fibres are concerned with the transfer of olfactory memories from one side to the other.





Physiology of olfaction

Molecules of substances to be smelled dissolve in the mucous layer overlying the olfactory epithelium and combine with receptors on the cilia of the olfactory rods. *The receptor cell is stimulated by means of a specific G* ***protein*** *(Golf).* Adenylate cyclase is activated, thus increasing intracellular cyclic adenosine monophosphate (cAMP). the latter causes opening of Na^+ channels and an influx of Na^+. This is followed by a receptor potential. The receptor potential depolarizes the first segment of the axon, which Pads to action potentials in the olfactory pathways.

There is no generally accepted classification of the basic types of smells recognized by humans. The sense of smell can be very sensitive, sensing minute concentrations of some substances in air. There is also no consistent correlation between strong smell and chemical structure, but substances with strong smells seem to be either highly water-soluble or lipid-soluble. *Discrimination within a certain smell is poor, requiring a change in concentration of at least 30% before a difference can be detected.*

Humans can distinguish about 10 000 different odours. There is considerable individual variation in the acuity of the sense of smell and, in general, women have a keener sense of smell than men. Smell is related to sexual function and in women it becomes more acute during ovulation. Adaptation can occur to pleasant as well as nasty smells and is specific for the odour being smelt. *Adaptation in smell is partly central and partly due to changes in the receptors.*

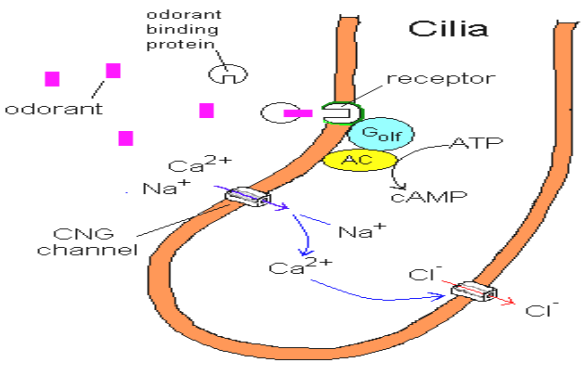
Clinical considerations

Loss of the sense of smell is called **anosmia** and its alteration is referred to as *parosmia or dysosmia.*

Damage to the olfactory epithelium or pathways by trauma or disease may lead to anosmia or parosmia. Patients with adrenal insufficiency develop a more acute sense of taste and especially of smell.

Hyperosmia refers to an increased and **hyposmia** to a decreased acuity in the sense of smell.

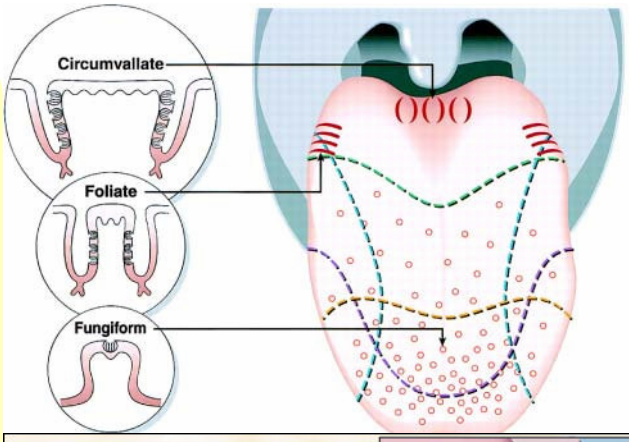
Hyposmia occurs in vitamin A deficiency and hypogonadism.

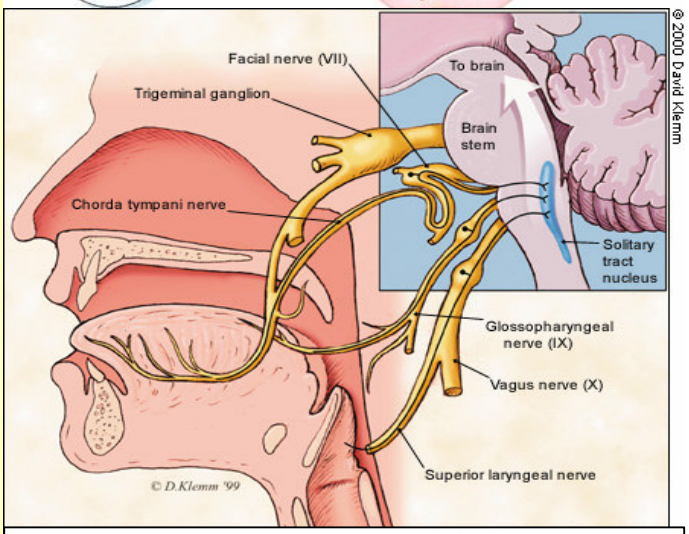


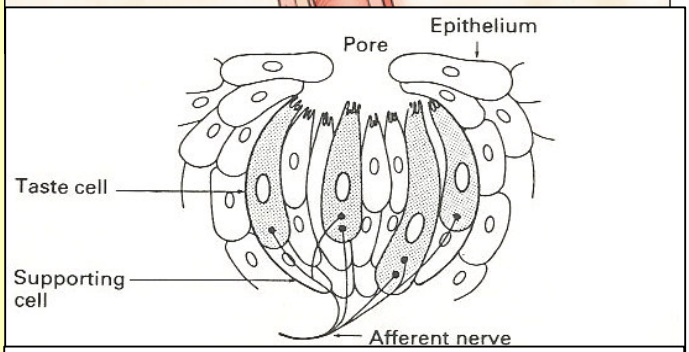
Taste

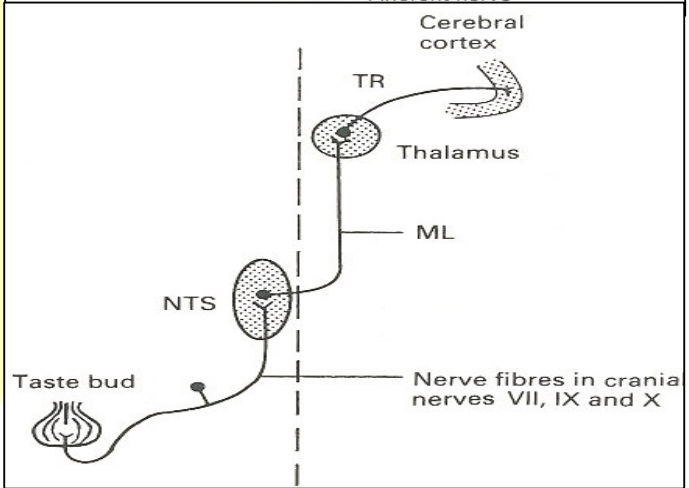
Gustatory receptors are found in the **taste buds.** These number about 10 000 in humans and are found on the tongue, epiglottis, soft palate and pharynx. On the tongue, taste buds are found in the **fungiform, foliate** and **vallate papillae.** *They are absent in the small* ***filiform papillae*** *in the mid-dorsum of the tongue; consequently, the mid-dorsum of the tongue is insensitive to taste.*

In the taste buds the receptor cells end in cilia, which project through the taste pore. Supporting cells are found between the receptors. Nerve fibres probably start as minute filaments within the receptors and form a plexus beneath the basement membrane before they emerge. Taste fibres from the anterior two-thirds of the tongue at first run with the fibres subserving touch, pain and temperature in the *lingual nerve.* They then separate to join the ***chorda tympani nerve***, which enters the medulla as part of the *facial nerve.* Fibres from the posterior one-third of the tongue run in the ***glossopharyngeal nerve*** and those from the epiglottis, palate and pharynx travel in the ***vagus nerve.*** The taste fibres in the three cranial nerves form the **tractus solitarius,** whose nucleus lies in the medulla. After synapse in the nucleus of the tractus solitarius, second-order neurons arise and cross the midline to ascend in the medial lemniscus to the thalamus. Third-order neurons arise after the synapse in the posteroventral nucleus of the **thalamus** and, finally, project to the lower part of the postcentral gyrus, along with other afferents from the face.









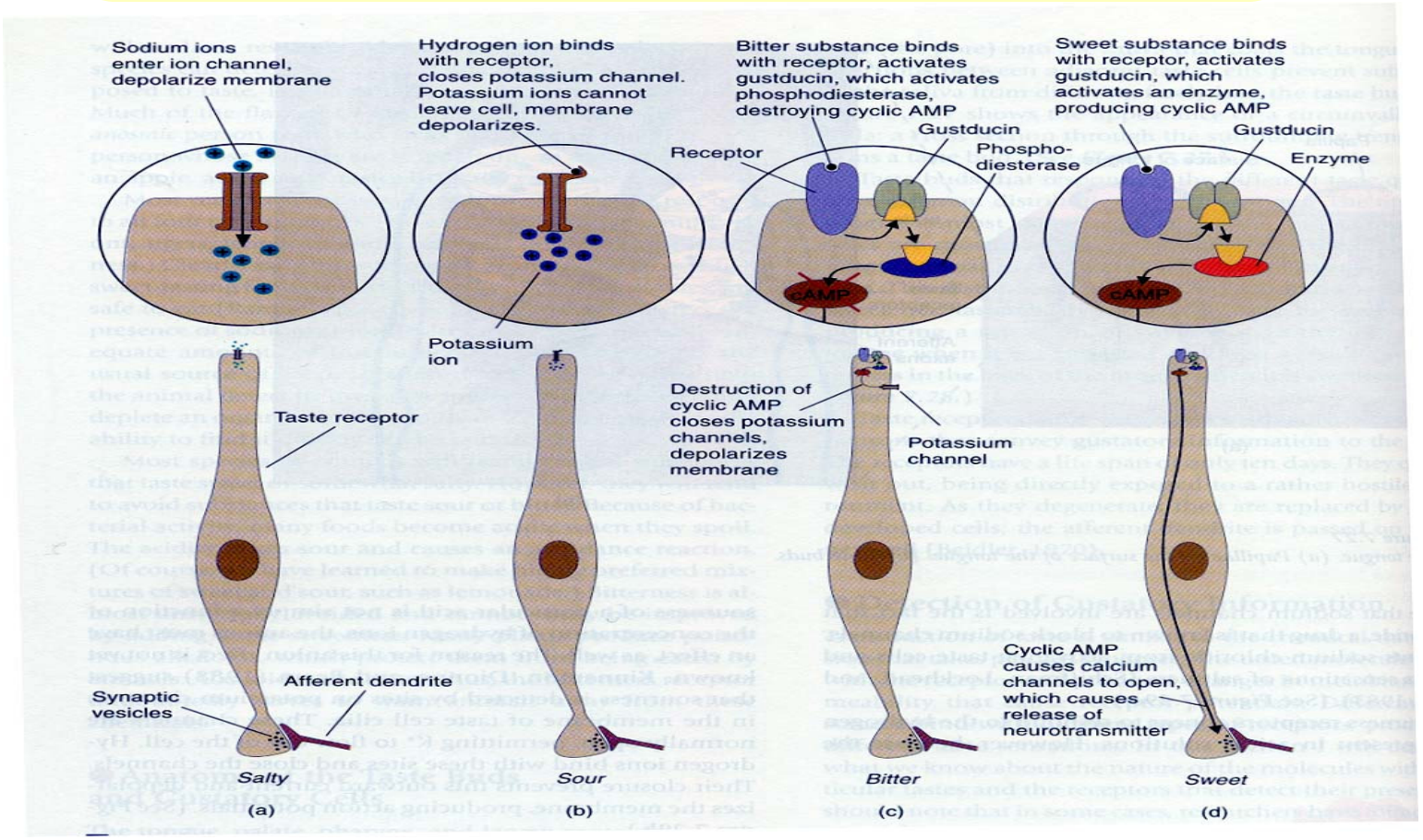
Physiology of taste

In order to be tasted, *substances must be dissolved in saliva*. Their molecules attach to receptors on the cilia of gustatory receptor cells, leading to a generator potential and action potentials in the gustatory pathways. The combination with receptors must be weak, since *taste can be abolished quite easily by washing the mouth with water.*

- In the case of taste, the primary modalities (types) have been identified. They are **sour, salt, sweet** and **bitter.** All other tastes are produced by various combinations of these four primary modalities.

✓ *The mechanisms of stimulation of specific taste receptors are as follows.*

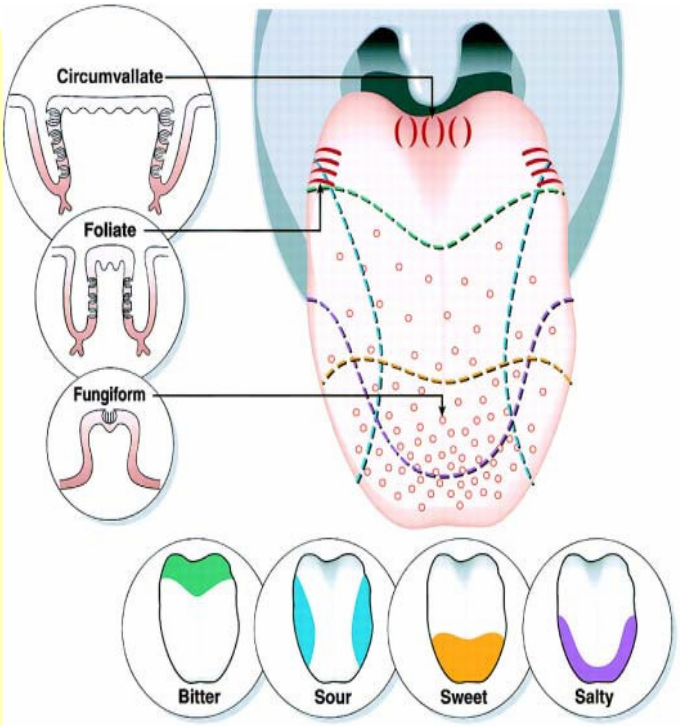
- **Sour** is detected when acids containing H^+ block K^+ channels.
- **Salt** probably acts by depolarization of salt receptors due to influx of Na+.
- **Bitter** stimuli act by a G protein (Go), which activates phospholipase C, leading to an increase of intracellular inositol triphosphate (IP_3), leading to Ca^{2+} release from the endoplasmic reticulum.
- **Sweet** stimuli work through a G protein (Gs), which activates adenylate cyclase, thus increasing cAMP, which leads to decreased K^+ conductance.



✓ *Each of the primary taste modalities is sensed maximally in a specific area of the tongue;* thus,

- ***sweet is best appreciated at the tip,***
- ***bitter at the back,***
- ***sour along the edges and***
- ***salt on the dorsum anteriorly near the edge.***
- In addition, *sour and bitter substances are tasted on the palate*, and all four taste modalities are sensed on the pharynx and epiglottis.

Taste is generally less sensitive than smell. There is also individual variation, and acuity of taste declines in old age. *Discrimination is as poor as in smell, similarly requiring a 30% change.* ***Adaptation occurs with taste and, in humans, is due entirely to the receptors.***



Relationship of chemical structure to taste

Some sapid (taste-producing) substances combine taste modalities but many substances have a taste belonging to one of the primary types.

- A **sour taste** is given by acids, whether organic or inorganic. For any acid, sourness is directly proportional to the hydrogen ion concentration, thereby providing a definite link between chemical structure and the taste sensation. The reference acid is HC1, with a normal threshold of pH 3.5.

- The **salt taste** is typically given by NaCl, which is used as the reference substance, with a threshold of 0.02 mol/litre. In the case of NaCl, the taste is due to the sodium ion but, in salts with other elements or metals, both the cation and the anion contribute and other tastes may be combined with the salty taste.

- The most pleasant taste modality, **sweet**, is given by a variety of substances. The reference substance is sucrose, at a threshold of 0.01 mol/l. Other sugars are sweet, including lactose, glucose and fructose; polysaccharides, glycerol, some alcohols (not ethanol), aldehydes and ketones are also sweet. Chloroform is sweet. The amides of aspartic acid and some recently discovered proteins are sweet, as are the inorganic salts of lead and beryllium. Artificial sweetening substances are used by diabetics and in reducing diets; they include saccharin, dulcin, cyclamates and aspartame.

- Many substances, organic and inorganic, are **bitter.** The reference substance is quinine sulphate, at a threshold of 0.000008 mol/litre. Urea, caffeine, nicotine, strychnine hydrochloride and morphine are bitter. The inorganic salts of magnesium, ammonium and calcium are also bitter and no definite chemical structure can be assigned to the bitter taste.

Clinical considerations

Complete taste blindness is termed ageusia and disturbed taste sensation is **dysgeusia.**

Administration of drugs that have sulph-hydryl groups in their structure, such as penicillamine, may cause temporary ageusia.

Some diseases cause **hypogeusia.**

Hypergeusia occurs in patients with adrenal insufficiency.

Inability to taste phenylthiocarbamide (PTC) in dilute threshold concentrations is a defect which is inherited as an autosomal recessive factor and may be used in human genetic studies.

The end