Photo credit: Stephen Petegorsky

Joanne Delphia, *Midnight Harvest*, 2004

Chapter 14

14 Taste

CALVIN TRILLIN, A WRITER WHO MAKES wonderful observations about the joys of eating, described his 4-year-old daughter's reaction to "polishing off a particularly satisfying dish of chocolate ice cream." She said, "My tongue is smiling."

What makes tongues smile? As with our noses, the basic answer to this question is "molecules." Olfaction and gustation are often grouped together as the "chemical senses," and in terms of physiology, these two sensory systems are in some ways quite similar. But the chemicals we taste have already entered our mouths and are about to move even farther into our bodies. Thus, taste serves the most specific function of any of the senses: we need to ingest chemicals that are nutritious and spit out chemicals that may be poisonous. Perhaps this is why there seems to be something about our liking or disliking of tastes that is very different from the liking or disliking that one might associate with the color red or the sound of middle C on the piano. Nature has equipped us to care passionately about food because that passion holds the key to our survival.

Taste versus Flavor

Before delving any further into our gustatory system, we need to clear up a very old misunderstanding. According to the early Greeks, sensations perceived from foods and beverages in the mouth were "tastes," whereas sensations perceived by sniffing were "smells." In fact, however, food molecules are almost always perceived by both our gustatory *and* our olfactory systems. The molecules we taste are dissolved in our saliva and passed over the taste receptors on our taste buds, as we'll discuss in this chapter. But when we chew and swallow foods, other molecules are released into the air inside our mouths and forced up behind the palate into the nasal cavity, where they contact the olfactory epithelium and stimulate our olfactory receptors (Figure 14.1). These **retronasal olfactory sensations** are then knitted together with our gustatory sensations by our brains into a kind of metasensation that goes by the name **flavor**.

It is quite easy to prevent the air flow that carries odorants through the retronasal passage. Children do it all the time when they hold their noses while eating spinach. You should try this now—but use a piece of chocolate if you're still not crazy about spinach. Pinch your nose before putting the chocolate in your mouth, then chew it and note the sensation, which will be almost pure taste. Then, just before swallowing, release your nose. Chocolate molecules will immediately be drawn up into the nasal cavity, and you will understand the difference between taste and flavor. Without olfaction, the chocolate should "taste" very similar to raw table sugar. You've probably noticed before that flavor is similarly impoverished when you have a stuffy nose. Web Activity 14.1, Taste without Smell asks you to test this phenomenon with other stimuli.

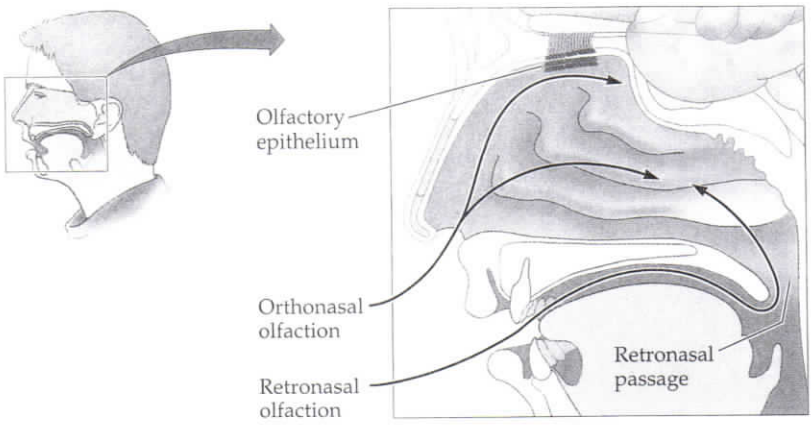


FIGURE 14.1 Molecules released into the air inside our mouths as we chew and swallow food travel up through the retronasal passage into the nose, where they then move upward and contact the olfactory epithelium.

Foods are also perceived by the somatosensory system via touch, temperature, and pain receptors in the tongue and mouth. Some of these sensations have protective functions: the burn of acid (which might damage your internal organs if swallowed), the heat pain from scalding coffee, the pain of biting the tongue, and so on. Somatosensations also provide information about the nature of foods and beverages. For example, we get information about the fat content of foods from tactile sensations such as oily, viscous, thick, and creamy.

Localizing Flavor Sensations

You should have realized something else when you performed the chocolate experiment described in the previous section: even though you now know that the chocolate sensation originates from the olfactory receptors in your nose, you probably still perceived the flavor as coming entirely from your mouth. This perception is due in part to the tactile sensations evoked by chewing and swallowing, and in part to taste. Because you taste and feel the food only in your mouth (not in your nose), your brain concludes that the sensations must have arisen entirely from the mouth. Exceptions include foods such as horseradish, wasabi, and spicy mustard, which give off volatile chemicals that activate pain receptors all the way up through your retronasal passage. But these exceptions prove the rule: when we eat these foods, we experience the sensations as coming from our noses as well as our mouths.

Now consider the following curious case. A patient with normal olfaction but damaged taste and oral touch reported that she could smell lasagna, but when she ate it, it had no flavor. A similar effect was produced in a laboratory using a small amount of lidocaine and a large amount of blueberry yogurt. Subjects in this study had their left **chorda tympani** (one of the **cranial nerves** that carries information from taste receptors to the brain) anesthetized with the lidocaine while they tasted the yogurt. In this situation, the subjects reported that the blueberry sensation—which is entirely due to retronasal olfaction—seemed to come only from the right side of the mouth. Moreover, the intensity of the blueberry sensation was reduced, and this intensity was reduced even further when both taste nerves were blocked (Snyder et al., 2001).

In both the patient and the experimental subjects, the pathway from the mouth to the nasal cavity was completely intact. Why, then, were their retronasal lasagna and blueberry sensations reduced? Recent brain imaging

retronasal olfactory sensation The sensation of an odor that is perceived when chewing and swallowing force an odorant emitted by the mouth up behind the palate into the nose. Such odor sensations are perceived as originating from the mouth, even though the actual contact of odorant and receptor occurs at the olfactory mucosa.

flavor The combination of true taste (sweet, salty, sour, bitter) and retronasal olfaction.

chorda tympani nerve The branch of cranial nerve VII (the facial nerve) that carries taste information from the anterior, mobile tongue (the part that can be stuck out). The chorda tympani nerve leaves the tongue with the lingual branch of the trigeminal nerve (cranial nerve V) and then passes through the middle ear on its way to the brain.

cranial nerves Twelve pairs of nerves (one for each side of the body) that originate in the brain stem and reach sense organs and muscles through openings in the skull.

research by Dana Small appears to answer this question: the brain processes odors differently, depending on whether they come from the mouth or through the nostrils. This distinction makes good sense functionally because the significance of odors in the mouth is very different from that of odors sniffed from the outside world. Without the proper cues to tell us where an odorant is coming from, input from the olfactory receptors apparently cannot be routed to the proper brain area to connect the smell sensation with the food stimulus.

The connections between taste and smell have been understood by the food industry for many years (Noble, 1996). For example, if a company is marketing pear juice and wants to intensify the sensation of pear, it will add sugar. The increase in sweetness (a pure taste sensation) will increase the perceived olfactory sensation of pear. However, this will work only for pairs of taste and retronasal olfaction that are commonly experienced. If our pear juice company were to add salt, the pear sensation would not increase. Thus, learning is playing a role in this phenomenon.

The pervasiveness of food additives such as carageenan, guar gum, and other thickening agents shows that the food industry also has a good handle on the effects of somatosensation on food perception. And the ingredient lists of most processed foods include at least one artificial coloring, testifying to the importance of yet another sense, vision, in how we perceive foods.

Anatomy and Physiology

Figure 14.2 illustrates the structures involved in the perception of tastes, which consists of the following sequence of events: Chewing breaks down food substances into molecules, which are dissolved in saliva. The saliva-borne food molecules flow into the **taste buds** embedded in structures called **papillae** (singular *papilla*) that cover the tongue (if the olfactory epithelium is the retina of the nose, the tongue is the retina of the mouth). Each taste bud, in turn, contains a number of **taste receptor cells**. Each taste receptor cell responds to a limited number of molecule types; when one of its preferred molecules makes contact with it, it may produce action potentials that send information along one of the **cranial nerves** to the brain. See Web Activity 14.2 Gustatory Anatomy for an interactive overview of the system, which is described in greater detail in the sections that follow.

Papillae

Papillae give the tongue its bumpy appearance and come in four major varieties, three of which contain taste buds.

Filiform papillae, the ones *without* any taste function, are located on the anterior portion of the tongue (the part you stick out when giving someone a raspberry) and come in different shapes in different species. In the cat, they are shaped like tiny spoons with sharp edges. The filiform papillae on our tongues do not have these sharp edges, which is why you will find lapping milk from a bowl to be considerably more difficult than your cat does.

Fungiform papillae, so named because they resemble tiny button mushrooms, are also located on the anterior part of the tongue. They are visible to the naked eye, but blue food coloring swabbed onto the tongue makes them particularly easy to see (the blue food coloring stains the filiform papillae much better than the fungiform papillae, so the fungiform papillae appear as pink circles against a blue background). Fungiform papillae vary in diameter, but the maximum is about 1 mm. On average, about six taste buds are buried

taste buds Globular clusters of cells that have the function of creating the neural signals conveyed to the brain by taste nerves. Some of the cells (see *taste receptor cells*) in the taste bud have specialized sites on their apical projections (see *microvilli*) that interact with taste stimuli. Some of the cells form synapses with taste nerve fibers. Current research is just revealing the complex events within taste buds that result in the neural signals.

papillae (s. papilla) Structures that give the tongue its bumpy appearance. From smallest to largest, the papillae types that contain taste buds are fungiform, foliate, and circumvallate; filiform papillae, which do not contain taste buds, are the smallest and most numerous.

taste receptor cells Cells within the taste bud that contain sites on their apical projections that can interact with taste stimuli. These sites fall into two major categories: those interacting with charged particles (e.g., sodium and hydrogen ions), and those interacting with specific structures.

filiform papillae Small structures on the tongue that provide most of the bumpy appearance. Filiform papillae have no taste function.

fungiform papillae Mushroom-shaped structures (maximum diameter 1 mm) that are distributed most densely on the edges of the tongue, especially the tip. Taste buds (an average of six per papilla) are buried in the surface.

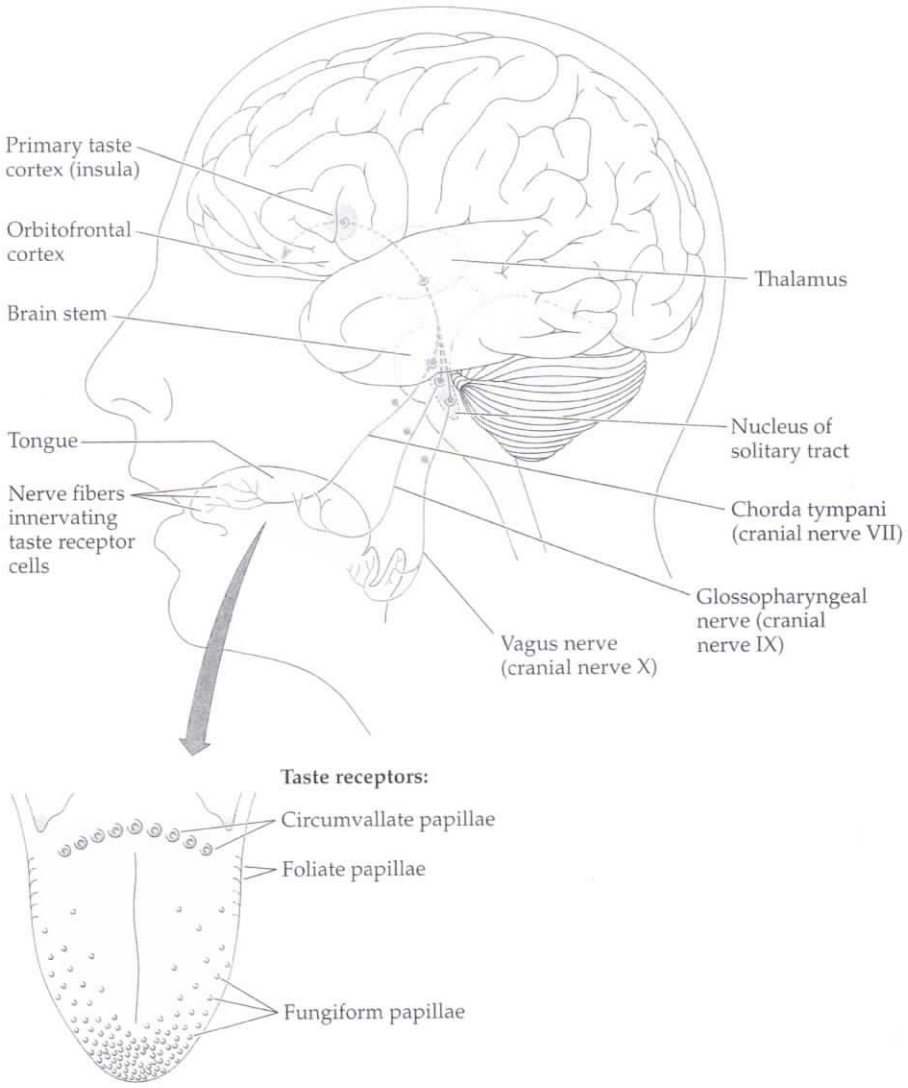


FIGURE 14.2 The locations of each type of taste papillae are shown on the diagram of the tongue shown here. Neural signals from the taste buds in those papillae are transmitted via cranial nerves VII, IX, and X to the brain.

in the surface of each fungiform papilla. If we stain the tongues of a lot of individuals, we see a large amount of variation (Figure 14.3). Some tongues have so few fungiform papillae that their stained tongues appear to have pink polka dots on them. Other tongues have so many that their tongues look as if they have been tiled with pink circles.

Foliate papillae are located on the sides of the tongue at the point where the tongue is attached. Under magnification, they look like a series of folds. Taste buds are buried in the folds.

Finally, **circumvallate papillae** are relatively large circular structures forming an inverted *V* on the rear of the tongue. These papillae look like tiny islands surrounded by moats. The taste buds are buried in the sides of the moats.

Although most people don't realize this, there are also taste buds on the roof of the mouth where the hard and soft palates meet. To demonstrate these, wet your finger and dip it into salt crystals. Touch the roof of your mouth and move your finger back until you feel the bone end (the margin between the hard and soft palates). You will experience a flash of saltiness as you move the salt crystals onto the taste buds arrayed on that margin.

foliate papillae Folds of tissue containing taste buds. Foliate papillae are located on the rear of the tongue lateral to the circumvallate papillae, where the tongue attaches to the mouth.

circumvallate papillae Circular structures that form an inverted *V* on the rear of the tongue (three to five on each side). Circumvallate papillae are moundlike structures surrounded by a trench (like a moat). These papillae are much larger than fungiform papillae.

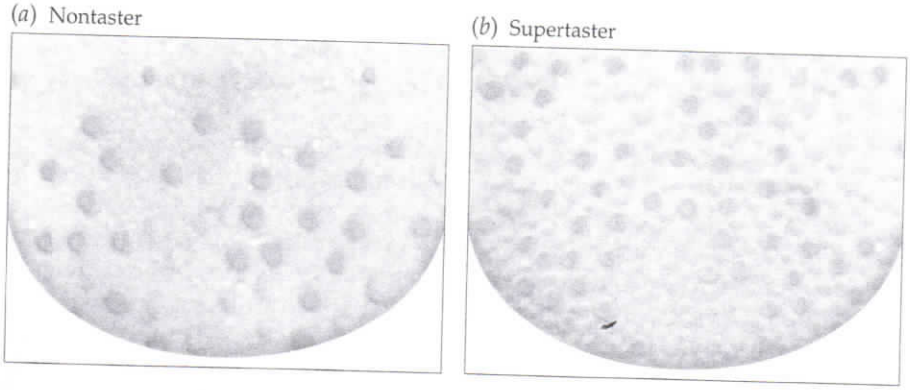


FIGURE 14.3 The tips of the tongues of a nontaster (*a*) and a supertaster (*b*), stained with blue food coloring. The pink circles are fungiform papillae. (Nontasters and supertasters will be discussed later in the chapter.)

In sum, the taste buds are distributed in a line across the roof of the mouth and in papillae distributed in an oval on the tongue. Fungiform papillae make up the front of the oval, and foliate and circumvallate papillae make up its rear. Note that we have no subjective awareness of this distribution of taste buds. (See Web Essay 14.1: Scientific Urban Legend: The Bogus Tongue Map.)

Taste Buds and Taste Receptor Cells

Each taste bud (Figure 14.4) is a cluster of elongated cells, organized much like the segments of an orange. The tips of some of the cells—taste receptor cells—become slender **microvilli** (singular *microvillus*) containing the sites that bind to taste substances. In an earlier era, these microvilli were mistakenly thought to be tiny hairs; we now know that microvilli are extensions of the cell membrane.

It used to be thought that taste axons connected to receptors on one end and then projected into the brain. A considerably more complex series of events is now beginning to emerge. At least some receptors are on cells that do not synapse with taste axons; the information they convey must get to the axons in some other way (Herness et al., 2005).

microvilli (s. microvillus) Slender projections on the tips of some taste bud cells that extend into the taste pore.

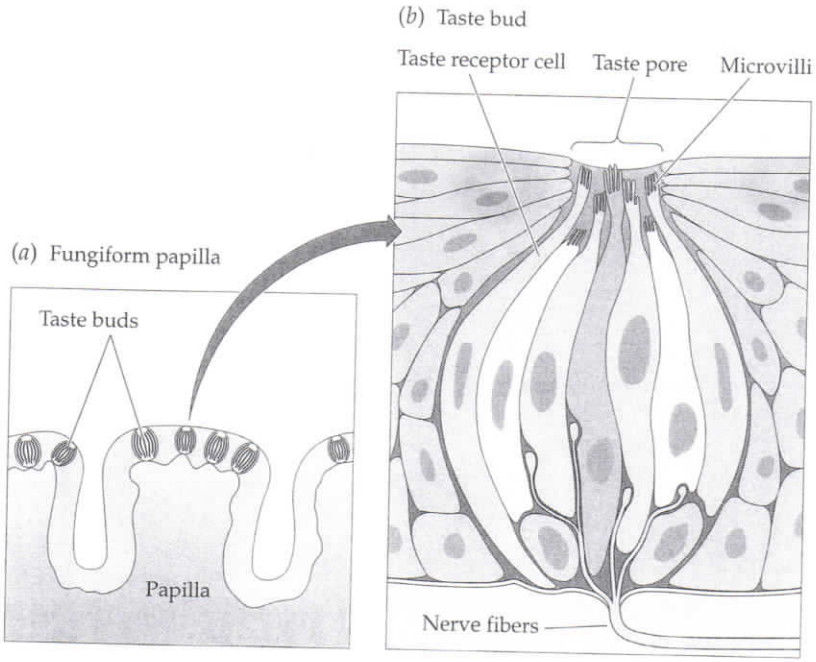


FIGURE 14.4 (*a*) The location of the taste buds buried in the tissue on the tops of the fungiform papillae. (*b*) Cross section of a taste bud.

In fungiform papillae, the taste nerve fibers that enter the taste buds branch so that an individual cell can be innervated by more than one taste fiber and an individual taste fiber can innervate more than one cell. Taste receptors have a limited life span. After about 10 days they die and are replaced by new cells. This constant renewal allows the taste system to recover from a variety of sources of damage, and it explains why our taste systems remain robust even into old age. Recordings from taste nerve fibers show that different receptor cells contacted by branches of a single fiber show similar specificities to taste stimuli. In other words, it appears that the nerve fibers are somehow able to select the cells with which they will synapse so that the message they convey remains stable, even though the receptor cells are continually replaced.

The mechanisms that permit a taste cell to recognize a taste stimulus contacting its microvilli can be divided into two large categories (Figure 14.5). One class of tastants is made up of small, charged molecules that taste salty or sour.

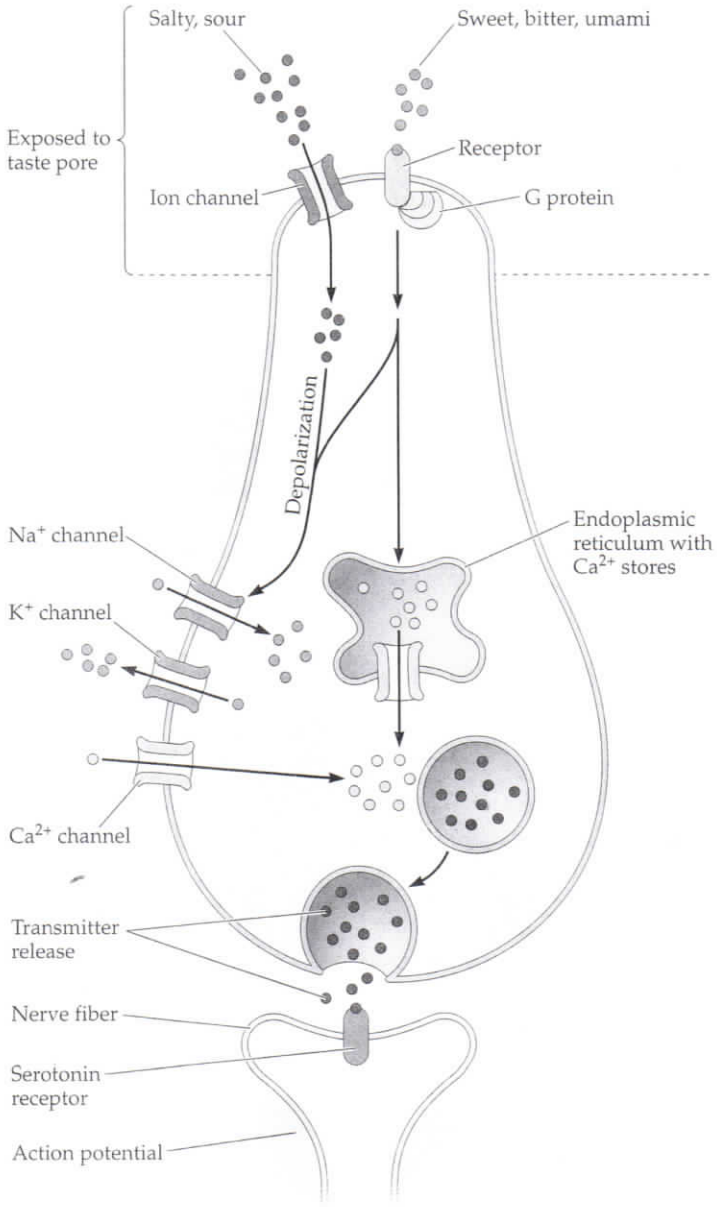


FIGURE 14.5 Diagram of a taste receptor cell illustrating the different receptor mechanisms for ionic stimuli (salty and sour), as well as those using a lock-and-key mechanism (sweet, bitter, umami).

Small openings, called "channels," in microvilli membranes allow some types of charged particles to enter cells but bar others. When the charged particles in salty and sour foods enter salty and sour receptor cells, these cells signal their respective tastes.

Tastants in the second class, which produce sensations that we label as sweet or bitter, are perceived via a mechanism similar to that in the olfactory system, using G protein–coupled receptors (GPCRs). The GPCRs wind back and forth across microvillus membranes, and when a particular tastant molecule "key" is fitted into the "lock" portion of a GPCR on the outside of the membrane, the portion of the GPCR inside the cell starts a cascade of molecular events that eventually leads to an action potential being sent back to the brain.

Central Nervous System

After leaving the taste buds through the cranial nerves, gustatory information travels through way stations in the medulla and thalamus before reaching the cortex (Figure 14.6) (Pritchard and Norgren, 2004). The primary cortical processing area for taste—the part of the cortex that first receives taste information—is the **insular cortex**. The **orbitofrontal cortex** receives projections from the insular cortex. Some orbitofrontal neurons are multimodal. That is, they respond to temperature, touch, and smell, as well as to taste, suggesting that this may be an integration area.

Inhibition plays an important role in the processing of taste information in the brain. One of the functions of this inhibition may be to protect our whole-

insular cortex The primary cortical processing area for taste—the part of the cortex that first receives taste information.

orbitofrontal cortex The part of the frontal lobe of the cortex that lies above the bone (orbit) containing the eyes. The orbitofrontal cortex is responsible for processing olfaction. It is also the area of the brain critical for assigning affective value to stimuli—in other words, determining hedonic meaning.

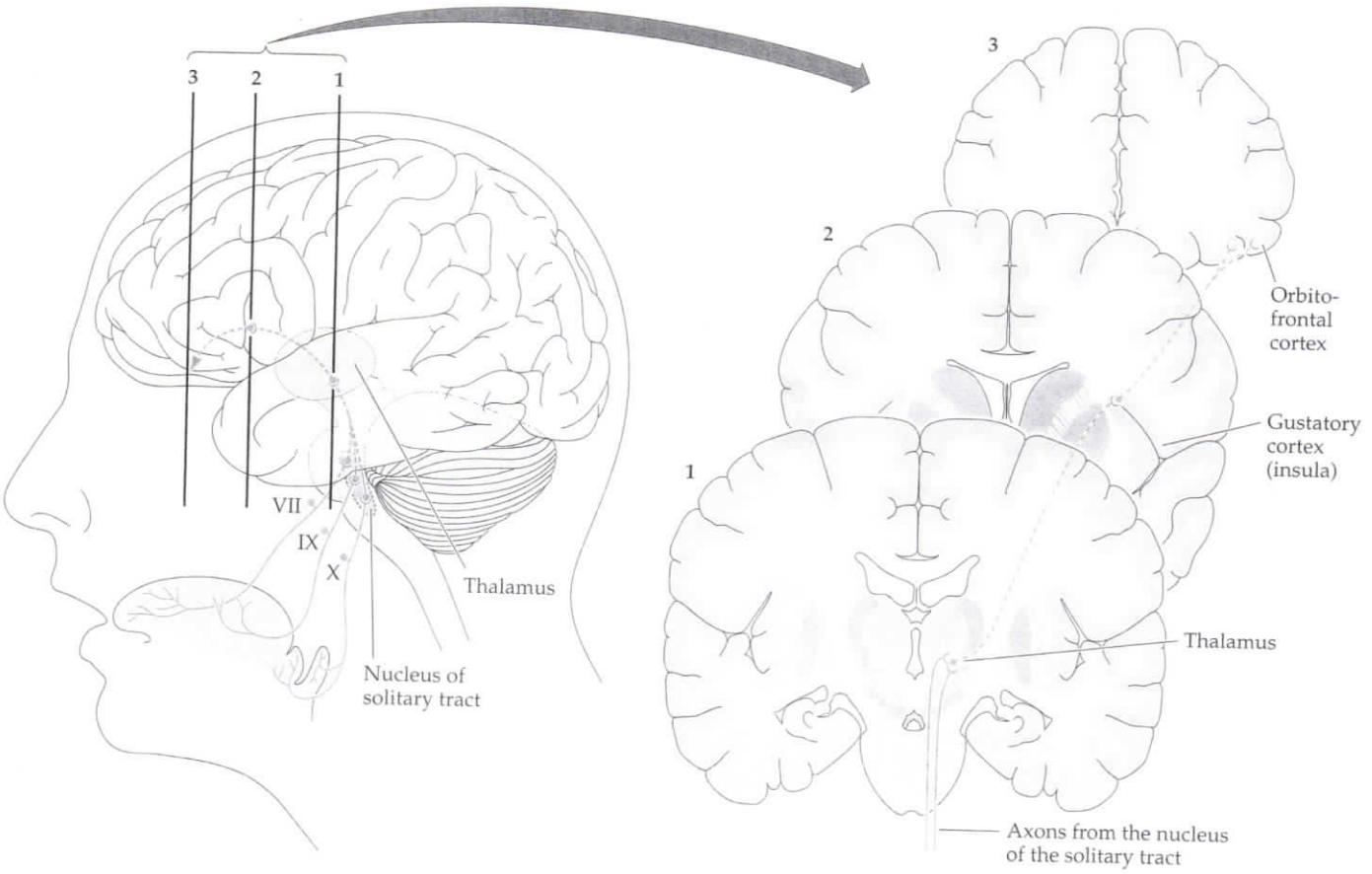


FIGURE 14.6 Taste information projects from the tongue to the medulla, then to the thalamus (shown in cross section 1 of the brain), then to the insula (cross section 2), and finally to the orbitofrontal cortex (cross section 3).

mouth perception of taste in the face of injuries to the taste system. Our brains receive taste input from several nerves (see Figure 14.6). Damage to one of them diminishes its contribution to the whole; however, that damage also releases the inhibition that is normally produced by the damaged nerve. The result is that whole-mouth taste intensities are relatively unchanged. Unfortunately, this preserved whole-mouth perception comes with a cost in some cases. Localized taste damage is often accompanied by "phantom taste" sensations (recall the phantom limbs experienced by many limb amputees), as if the release of inhibition serves to permit even noise in the nervous system to be perceived as a taste.

Descending inhibition from the taste cortex to a variety of other structures (DiLorenzo and Monroe, 1995) may also serve other functions. For example, mouth injuries that lead to oral pain make it harder to eat. The inhibition of such pain perceptions by taste-processing parts of the brain would make eating easier and thus increase the likelihood of survival (because no matter how much your mouth hurts, you still have to eat). Consistent with this principle, patients with a serious oral pain disorder (burning mouth syndrome) were shown to have localized taste damage as well (Grushka and Bartoshuk, 2000). Furthermore, women who have taste damage are more likely to suffer from severe nausea and vomiting during pregnancy (Sipiora et al., 2000), and cancer patients, whose chemotherapy and radiation therapy is known to damage the taste system, are more likely to experience coughing, gagging, and hiccups. In all these cases, inhibitory signals from the taste cortex that normally help prevent eating-disruptive symptoms (oral pain, vomiting, hiccuping, and so on) may have been turned off because of the damage to the taste system.

The Four Basic Tastes

We learned in the previous chapter that we are able to distinguish many different odorants. Already in this chapter, however, we have seen that when olfaction is taken out of the equation, much of the complexity of the sensations evoked by foods vanishes. This suggests that the number of basic taste qualities is quite small. In fact, the current universally accepted list includes only the four **basic tastes** previously mentioned in the section on taste buds and taste receptor cells: **salty**, **sour**, **bitter**, and **sweet**.

basic tastes The four taste qualities that are generally agreed to describe human taste experience: sweet, salty, sour, bitter.

salty The taste quality produced by the cations of salts (e.g., the sodium in sodium chloride produces the salty taste). Some cations also produce other taste qualities (e.g., potassium tastes bitter as well as salty). The purest salty taste is produced by sodium choloride (NaCl), common table salt.

sour The taste quality produced by the hydrogen ion in acids.

bitter The taste quality, generally considered unpleasant, that is produced by substances like quinine or caffeine.

sweet The taste quality produced by some sugars, such as glucose, fructose, and sucrose. These three sugars are particularly biologically useful to us, and our sweet receptors are tuned to them. Some other compounds (e.g., saccharin, aspartame), are also sweet.

Salty

Salts are made up of two charged particles: a cation (positively charged) and an anion (negatively charged). For example, common table salt is NaCl; the sodium is the cation (Na^+) and the chloride is the anion (Cl^-). Although all salts taste at least a little salty to humans, pure NaCl is the saltiest tasting salt around. Sodium must be available in relatively large quantities to maintain nerve and muscle function, and loss of body sodium leads to a swift death.

Our ability to perceive saltiness is not static. Gary Beauchamp and his colleagues (Bertino, Beauchamp, and Engelman, 1982) showed that diet can affect the perception of saltiness. Fortunately for those on low-sodium diets, reduced sodium increases the intensity of saltiness over time. Individuals who are initially successful in reducing their sodium intake will find that foods that they used to love may now taste too salty. This adjustment in perception helps them keep their sodium intake down.

Our liking for saltiness is not static either. Early experiences can modify salt preference. In 1978 and 1979, several hundred infants were fed soy formulas that were accidentally deficient in chloride because of an error in formulation.

Chloride deficiency has effects on our physiologies that mimic the effects of sodium deficiency. Thus the infants who were chloride-deficient offered an important way to study sodium deficiency in humans. The Centers for Disease Control and Prevention in Atlanta monitored these infants, and a variety of studies were done to assess any potential damage. One of the consequences was that the salt preference of the children increased (L. J. Stein et al., 1996). Experiences during gestation can also affect salt preference. Crystal and Bernstein found an increased preference for salty snacks among college students whose mothers had experienced moderate to severe morning sickness during pregnancy (Crystal and Bernstein, 1995). The exact mechanisms by which these abnormal metabolic events enhance salt preference are still not understood.

Sour

As you may remember from high school chemistry, a solution containing hydrogen ions (H^+) and hydroxide ions (OH^-) in equal proportions produces water (HOH, or H_2O). As the relative proportion of H^+ increases (increasing the pH level), the solution becomes more *acidic*.

Why do you need to be reminded of all this? Because sour is the taste of acids. Some individuals like the sourness of acids in relatively low concentrations. Pickles and sauerkraut, both of which get their sour tastes from lactic acid, are enjoyed by many adults, and as the success of sour candies shows, many children like sour tastes that are rejected by adults (Liem and Mennella, 2003). At high concentrations, however, acids will damage both external and internal body tissues.

Bitter

The Human Genome Project revealed a multigene family responsible for about 30 different bitter receptors; quinine (Figure 14.7) is a prototypically bitter-tasting substance. However, each of these bitter receptors does not project via a specific bitter neuron. What this means is that, although a great many different compounds taste bitter, we cannot distinguish between the tastes of these compounds (Mueller et al., 2005). The diversity of receptors for bitterness allows species or even individuals in a given species to have varying responses to an array of bitter compounds. One of the most famous of these is "taste blindness" to PTC (phenylthiocarbamide) found in humans—a phenomenon we will revisit later in this chapter.

Not coincidentally, many compounds that we taste as bitter tend to be poisonous. However, some bitter stimuli are actually good for us. For example, bitter compounds in some vegetables help protect against cancer. We would like to be able to "turn off" these bitter sensations so that people would have an easier time eating their vegetables. Along these lines, Robert Margolskee, a pioneer in studies of bitter transduction, used his understanding of the bitter system to identify a substance than can inhibit some bitter sensations: adenosine monophosphate (AMP) (Ming, Ninomiya, and Margolskee, 1999). In addition to its potential as an artificial bitter inhibitor, AMP may actually function as a natural bitter inhibitor in mother's milk. A number of compounds in milk, such as caseine (milk protein) and calcium salts, taste bitter; and, as we will see later, aversions to bitter tastes are present at birth. The presence of AMP in mother's milk may suppress those bitter tastes enough to allow milk to be palatable to babies.

Bitter sensitivity is also affected by hormone levels in women. Bitter perception intensifies during pregnancy and diminishes after menopause (Duffy

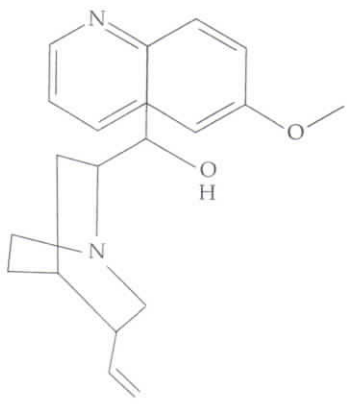


FIGURE 14.7 The molecular structure of quinine ($C_{20}H_{24}N_2O_2$), a prototypically bitter substance.

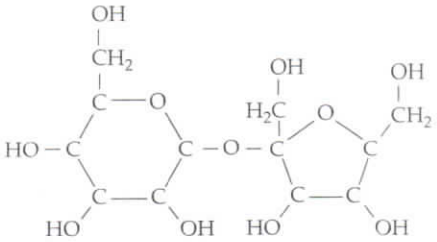


FIGURE 14.8 The molecular structure of sucrose, common table sugar. This disaccharide is formed from a combination of a glucose molecule and a fructose molecule. Glucose, which is easily extracted from sucrose by the digestive system, is the main fuel that powers almost every biological engine (including the human brain currently reading this book).

et al., 1998). This makes sense in the context of the function of bitterness as a poison detection mechanism. Intensifying the perception of bitterness early in pregnancy, when toxins exert their maximum effects, has clear biological value. Consistent with this correlation, some of the aversions during pregnancy occur with foods or beverages that have bitter tastes (e.g., coffee).

Sweet

Sweetness is evoked by sugars, simple carbohydrates that generally conform to the chemical formula $(CH_2O)_n$, where n is between 3 and 7. Glucose, one of the sweetest-tasting sugars, is the principal source of energy in humans (as well as nearly every other living thing on Earth). Common table sugar, sucrose (Figure 14.8)—which is a combination of glucose and yet another sugar, fructose—tastes even sweeter.

As with bitter compounds, there are many different sugars that taste sweet, but the information from all the different sweet detectors is pooled before being sent to the brain. Therefore, we cannot distinguish between the qualities of different sweet tastes. When we said that sucrose tastes sweeter than glucose, what we meant was that a gram of pure sucrose dissolved in a given amount of water tastes more *intensely* sweet than a gram of pure glucose dissolved in the same amount of water.

Artificial sweeteners such as saccharin (discovered in 1879 when a research fellow working on coal tar derivatives failed to wash up before dinner and subsequently noticed that the tar residue on his hands tasted sweet) work by mimicking the chemical structure of sugars well enough to activate sweet receptors. (Saccharin also activates bitter receptors, which is why the search for new artificial sweeteners continues to this day.)

Saccharin is attractive to dieters because its sweet taste comes with essentially no calories. Although countless dieters count on this property to help them watch their weight, a 1986 epidemiological study showed that women who consumed artificial sweeteners actually gained weight (Stellman and Garfinkel, 1986). That same year, John Blundell, an English expert on weight regulation, published a provocative article suggesting that aspartame (the artificial sweetener sold as NutraSweet) increases appetite (Blundell and Hill, 1986). Was the benefit of the reduced calories lost when individuals using aspartame actually increased their caloric intake in subsequent meals? This observation remains controversial, but it points to the complexity of food behavior.

Survival Value of Taste

Like olfactory receptors, taste receptors detect specific features of molecules. But the two senses evolved to serve quite different functions. The sense of smell helps us identify the objects in our environment. Indeed, for many animals (e.g., rodents), olfaction is the primary means for knowing what surrounds them in the world. Consistent with this purpose, the olfactory system is capable of distinguishing between a large number of different molecules, and an individual animal can learn about whatever olfactory stimuli exist in the environment where it happens to live.

On the other hand, we've just seen that the gustatory system responds to a fixed and much smaller set of molecules. This is consistent with the role of taste as a system for detecting nutrients and "antinutrients" (substances that are either helpful or harmful, respectively, to our bodies) before we ingest them.

Each of the four basic tastes is responsible for a different nutrient or antinutrient, and has evolved according to its purpose. For example, the bitter

taste subsystem is nature's poison detector. If we look at the chemical structures of poisons, there is a great deal of chemical diversity. Thus, any built-in poison detector must be able to recognize many different compounds. On the other hand, given that we don't really care if we can discriminate among poisons, since we just want to avoid them all, we could hook all of those receptors up to a few common lines to the brain. As we saw earlier, this is exactly how the bitter subsystem is set up.

Similarly, the sour subsystem is configured to reject any acidic solution without distinguishing exactly what is causing the pH level of the solution to be so high. The other two taste subsystems allow us to detect, and therefore selectively ingest, foods that our bodies need: sodium (salty) and sugars (sweet).

The Special Case of Umami

Umami arose as a candidate for the fifth basic taste as part of advertising claims by manufacturers of **monosodium glutamate** (**MSG**), the sodium salt of glutamic acid. Identified by Japanese chemists in the early 1900s, MSG was initially marketed as a flavor enhancer, said to suppress unpleasant tastes and enhance pleasant ones. When taste experts proved skeptical, MSG manufacturers went on to claim that MSG was a fifth basic taste, speculating that it signaled protein and thus played an important role in the taste perception of many foods. However, the umami taste is not perceptible in many foods containing proteins.

Glutamate is an important neurotransmitter, so receptors for the molecule are common throughout the body. The argument that such receptors might have been harnessed by the taste system to signal umami gained respectability when neuroscientists Nirupa Chaudhari and Steve Roper identified a version of a glutamate receptor in rat taste papillae (Chaudhari, Landin, and Roper, 2000). In spite of the elegant science leading to this discovery, we still do not understand the role of MSG in human taste experience. It is important to remember that taste systems evolved to solve important nutritional problems for different species. Thus, what we have learned from the rat must be tested further to determine what the results mean for human taste. For example, although glutamate is critical for proper cell function, it is not considered an essential nutrient in humans, because our bodies can manufacture it from simpler compounds (unlike a molecule such as sodium, which we cannot manufacture ourselves and therefore must ingest to survive).

Because glutamate is a neurotransmitter, concerns have been raised about its safety in the human diet. MSG became particularly notorious in the 1960s. First it became associated with Chinese restaurant syndrome—a constellation of symptoms including numbness, headache, flushing, tingling, sweating, and tightness in the chest—that was reported by some individuals after consuming MSG (Kwok, 1968). Then Dr. John Olney, a toxicologist, suggested that MSG might induce brain lesions, particularly in infants (Olney and Sharpe, 1969). In response to these concerns, MSG was removed from baby foods in the 1970s. The final conclusion as of now (see Walker and Lupien, 2000) is that MSG in large doses may be a problem for some sensitive individuals, but it does not pose a serious problem for the general population.

Coding of Taste Quality

A major source of historical controversy in the taste literature revolved around whether tastes are coded mainly via **labeled lines**, in which each taste neuron would unambiguously signal the presence of a certain basic taste, or

umami The taste sensation evoked by MSG.

monosodium glutamate (MSG) Sodium salt of glutamic acid (an amino acid).

labeled lines Theory of taste coding in which each taste nerve fiber carries a particular taste quality. For example, the quality evoked from a sucrose-best fiber is sweet, that from an NaCl-best fiber is salty, and so on.

via patterns of activity across many different taste neurons. We've seen examples of both types of coding in other senses. For example, color vision and olfaction use pattern coding. A single type of cone cannot tell us the wavelength of a light ray, but the pattern of activity across our three cone types can give us this information. Hearing, on the other hand, uses a mechanism more akin to the labeled-line approach: certain neurons always respond best to 5000-Hz tones, others always respond best to 5100-Hz tones, and so on. Which scheme is used in the gustatory system?

Given what we've already learned about the functions of the four basic tastes, it is easy to construct an evolutionary argument for labeled-line coding. Recall that in olfaction, which uses pattern coding, mixtures of two different compounds very often produce a third smell sensation that bears no resemblance to the smells of the mixture components. Such a coding system would be disastrous for the purpose of the taste system. For example, poisonous plants contain components with a variety of tastes. If bitterness were to synthesize with these other tastes, we would not be able to parse it out and thus avoid the poison. The functions of the four tastes are well served by their independence from each other. In addition, studies have shown that we are, in fact, very good at analyzing taste mixtures. For example, tonic water, which nowadays contains a combination of quinine and sugar, tastes bittersweet: we have no difficulty identifying its two components, bitter and sweet.

The historical controversy arose because initial research seemed to indicate that most neurons coming from taste buds respond to more than one of the four basic tastes. For example, in Carl Pfaffmann's initial work (Pfaffmann, 1941) employing recording from single cat chorda tympani neurons, some neurons responded to both acid and salt, and others responded to both acid and quinine (adding to the confusion was the fact that Pfaffmann found no

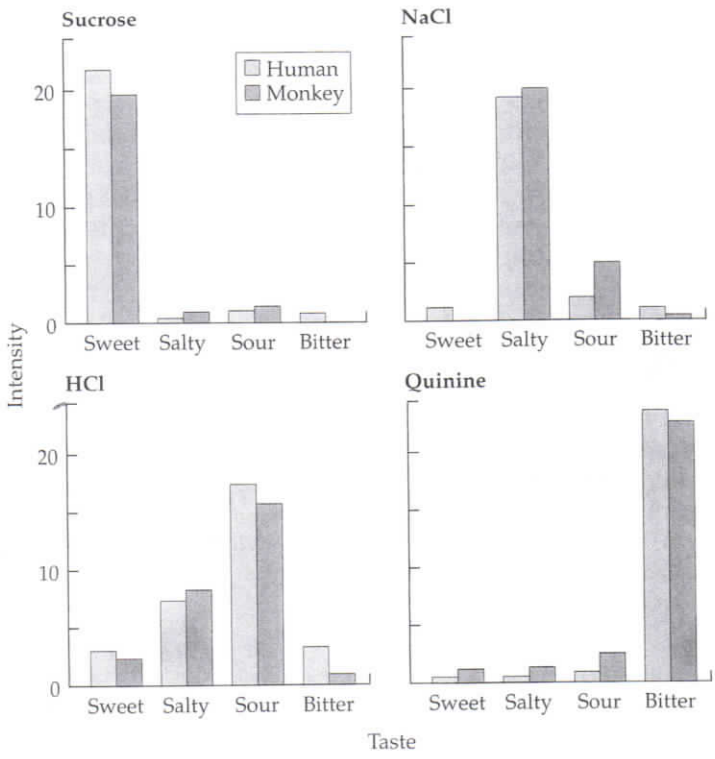


FIGURE 14.9 The tastes that human subjects perceive for each of four stimuli: sucrose, NaCl, HCl, and quinine. The tastes that a monkey would perceive if the monkey's sweet-best fibers coded sweetness, NaCl-best fibers coded saltiness, HCl-best fibers coded sourness, and quinine-best fibers coded bitterness are also shown. (Monkey data from Sato, Ogawa, and Yamashita, 1975.)

neurons that responded to sugar). However, subsequent research showed that although "pure" labeled lines from individual receptor types are rare, most taste nerve fibers do have a clear "favorite" stimulus. Marion Frank (1973) named the neuron types "NaCl-best," "sucrose-best," and so on.

The fact that taste neurons are not exclusively tuned to single basic tastes means that we rarely, if ever, experience "pure tastes." For example, sour tastes are perceived when acid-best neuron fibers are activated, but acids also activate NaCl-best fibers (Figure 14.9). Thus a solution of hydrochloric acid (HCl) will taste primarily sour, but will also taste salty.

Taste Adaptation and Cross-Adaptation

As we've seen throughout this book, all sensory systems show adaptation effects, in which constant application of a certain stimulus temporarily weakens subsequent perception of that stimulus. In taste, we are always adapted to the salt in saliva, and this effects our ability to taste salt; in addition, adaptation to certain components in one food can change the perception of a second food. (See Web Essay 14.2 Water Tastes.)

You've experienced cross-adaptation yourself if you've ever noticed that a beverage like lemonade tastes too sour after you eat a sweet dessert. The sugar in the dessert adapts the sweet receptors so that the subsequent lemonade tastes less sweet and more sour than normal.

Genetic Variation in Taste Experience

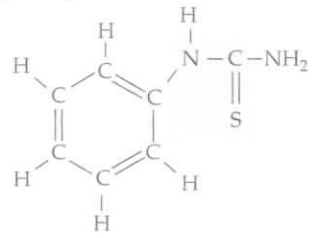
In 1931 a chemist named Fox discovered that we do not all live in the same taste worlds (A. L. Fox, 1931). Fox was synthesizing the compound phenylthiocarbamide (PTC) (Figure 14.10*a*), when some spilled and flew into the air. A colleague nearby noticed a bitter taste, but Fox tasted nothing. A test of additional colleagues revealed a few more **nontasters** like Fox who did not taste the compound, but most tasted it as bitter. The next year Fox and Blakeslee (a famous geneticist of the day) took PTC crystals to a meeting of the American Association for the Advancement of Science and set up a voting booth for attendees to register their perceptions. About one-third of those polled found the crystals to be tasteless, while two-thirds found them to be bitter. These proportions captured the imaginations of many researchers, and for several years the *Journal of Heredity* sold papers impregnated with PTC for further studies. Family studies eventually confirmed that taster status was an inherited trait (e.g., the Dionne quintuplets were all found to be tasters in 1941). Nontasters carried two recessive alleles, whereas tasters had either one or both dominant alleles.

Initially, individuals were simply classified as to whether or not they could taste PTC, but eventually threshold studies came into vogue. In a threshold method invented specifically for PTC studies, subjects were given eight cups, four containing water and four containing a given concentration of PTC. Correct sorting determined the threshold. The distribution of thresholds was bimodal, with nontasters showing very high thresholds and tasters showing low thresholds. This distribution varied by sex and race: women had lower thresholds than men, and Asians had lower thresholds than Caucasians.

In the 1960s, Roland Fischer shifted tests to a chemical relative of PTC that was safer to test—propylthiouracil (PROP) (Figure 14.10*b*)—and focused on the nutritional implications of the genetic variation in taster status (Fischer and Griffin, 1964). Fischer suggested that tasters were more finicky eaters: since bitter tastes are more intense to these individuals, they tend to dislike

nontaster An individual born without receptors for the bitter compound 6-*n*-propylthiouracil (PROP). Nontasters tend to have the fewest fungiform papillae. Thus, they live in a "pastel" taste world.

(*a*) Phenylthiolcarbamide



(*b*) Propylthiouracil

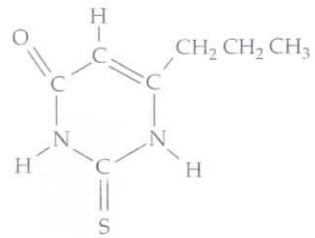


FIGURE 14.10 The chemical structures of PTC (*a*) and PROP (*b*). Both molecules are perceived as bitter by tasters, but they are tasteless to nontasters. The portions shaded in blue are those responsible for the bitter taste.

cross-modality matching The ability to match the intensities of sensations that come from different sensory modalities. This ability allows insight into sensory differences. For example, to a nontaster the bitterness of black coffee roughly matches the pain of a mild headache; for a supertaster, the coffee taste matches the pain of a severe headache.

supertaster An individual born with receptors for the bitter compound 6-*n*-propylthiouracil (PROP) who also has a high density of fungiform papillae.

foods high in bitter compounds, such as many vegetables, that nontasters find more palatable. Fischer also related taster status to body type (e.g., weight) and health. Alcoholics and smokers were found to contain a lower proportion of tasters than would be expected by chance, presumably because unpleasant sensations (e.g., bitterness) produced by alcoholic beverages and tobacco acted as deterrents. The effect of genetic variation in taste was even related to cancer risk, as will be described shortly.

Supertasters

By the 1970s, the "direct" psychophysical methods introduced by Harvard's S. S. Stevens led to a new look at this phenomenon. Instead of measuring thresholds—the dimmest sensations—investigators could look at suprathreshold taste and plot the psychophysical functions showing how perceived taste intensity varies with concentration. Stevens and his students showed that the same relation held for many different sensory modalities, including taste:

$$\psi = \phi^{\beta}$$

where ψ = perceived intensity, ϕ = concentration, and β takes on different values for different sensory modalities (S. S. Stevens and Galanter, 1957). Of special interest for the present purposes, β takes on different values for different taste qualities. For example, bitterness grows more slowly with concentration than sweet does (Figure 14.11). Thus, the value of β for bitter is smaller than the value of β for sweet.

Two of Stevens's students—Joseph Stevens and Lawrence Marks—made another fundamental discovery in this era: humans are very good at **cross-modality matching** (J. C. Stevens, 1959). For example, we can match the loudness of a sound to the brightness of a light, and we can match both of these to the intensity of a taste. This finding led to yet another way to study variability in individuals' perceptions of the bitter taste of PROP (Marks et al., 1988): we could ask subjects to match the bitterness of PROP to other sensations completely unrelated to taste (Figure 14.12). For example, nontasters matched the taste of PROP to very weak sensations (the sound of a watch or a whisper). Tasters proved to be a heterogeneous lot. For some, PROP was likened to very intense sensations, such as the brightness of the sun or the most intense pain ever experienced. These individuals were labeled **supertasters**. "Medium tasters" matched PROP to weaker stimuli, such as the smell of frying bacon or the pain of a mild headache (Bartoshuk, Fast, and Snyder, 2005).

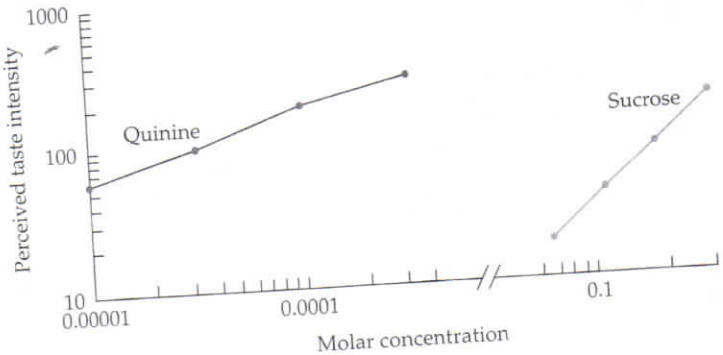


FIGURE 14.11 Psychophysical functions for quinine and sucrose. The logarithm of the perceived taste intensity is plotted against the logarithm of the concentration. In this plot, β is the slope of the function. The value of β for quinine is 0.3; for sucrose, 0.8. (Data from Bartoshuk, 1979.)

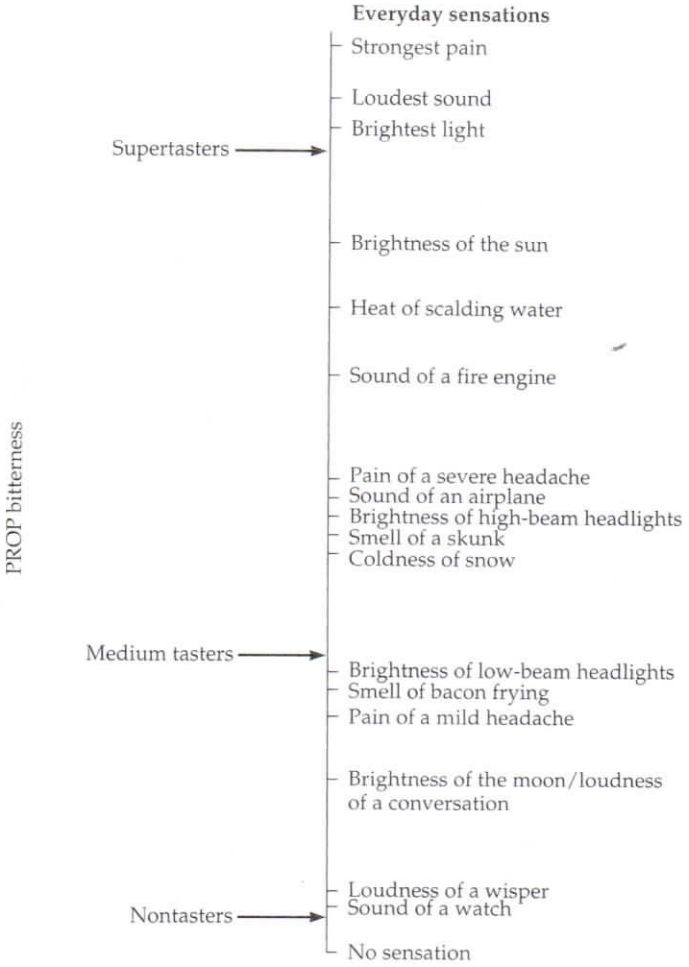


FIGURE 14.12 Cross-modality matching. The levels of bitterness of concentrated PROP perceived by nontasters, medium tasters, and supertasters are shown on the left. The perceived intensities of a variety of everyday sensations, are shown on the right. (Data from Fast, 2004.)

Once the subjects could be grouped in this way, it became clear that the different taste worlds experienced by nontasters, medium tasters, and supertasters are related to the anatomical differences in papillae distribution discussed earlier. Supertasters perceive virtually all taste substances to be more intense than do nontasters because supertasters have many more fungiform papillae, and therefore many more taste buds and taste receptors. In addition, because of the trigeminal innervation of fungiform papillae, supertasters also perceive more intense oral burn/pain sensations from foods such as chili peppers, and more intense oral touch sensations (e.g., the oiliness or thickness of fats in foods).

Health Consequences

With these new insights, the potential links between responsivity to PROP and health have become the focus of renewed interest. Valerie Duffy is a pioneer in the modern movement among nutritionists to evaluate food behavior in terms of the sensory properties of foods instead of only their nutrient content. The new psychophysical methods permitted her to show that variation in the sensory properties of foods and beverages affects food preferences and thus diet. Because diet is a major risk factor for a variety of diseases, genetic variation in taste plays a role in these diseases. For example, some vegetables

produce unpleasant sensations (e.g., bitter) to medium and supertasters, leading these individuals to eat fewer of them. Reduced vegetable intake is in turn a risk factor for colon cancer. Sure enough, Duffy and her colleagues found that, in a sample of older men getting routine colonoscopies at a Department of Veterans Affairs hospital, those tasting PROP as most bitter had the most colon polyps, a precursor to colon cancer (Basson et al., 2005). On the other hand, fats can produce unpleasantly intense sensations to supertasters leading them to eat fewer high-fat foods and thereby lower their risk of cardiovascular disease (Duffy, Lucchina, and Bartoshuk, 2004).

Discovery of the location of the gene for PROP tasting now permits genotyping for this characteristic. As noted earlier, early family studies showed that nontasters must carry two recessive alleles for PROP tasting. Some years ago we thought that supertasters might carry two dominant alleles, but this turned out not to be true. Rather, supertaster status is conferred by at least one dominant allele for PROP tasting, combined with a high density of fungiform papillae. Genotyping has also confirmed the early suggestions of Fischer that nontasters are more likely to smoke and to consume more alcohol (Duffy et al., 2004; Snyder et al., 2005).

The Pleasures of Taste

Pfaffmann wrote a famous paper in 1960 entitled "The Pleasures of Sensation," in which he underlined unique features of the sense of taste with regard to affective experience. Taste not only provides sensory information about certain nutrients but also provides pleasure (sweet, salty, and, for children, sour) and displeasure (bitter). The pleasure or displeasure that these tastes evoke is present at birth. That is, with no experience, an infant will like sweet and dislike bitter and strong sour. Salty receptors are not completely mature at birth, but when they do become functional, infants will like relatively dilute salts.

Some of the most impressive evidence for hardwired affect with taste came from the work of Jacob Steiner on facial expressions in newborn infants (Steiner, 1973). Steiner found that infants responded with stereotyped facial expressions when sweet, salty, sour, and bitter solutions were applied to their tongues. Sweet evoked a "smilelike" expression followed by sucking (Figure 14.13*a*). Sour produced pursing and protrusion of the lips (Figure 14.13*b*). Bitter produced gaping, movements of spitting, and in some cases, vomiting movements. Tragically, infants can be born lacking cerebral hemispheres (a condition known as "anencephaly"). Steiner was able to test a few of these infants, and they showed the same facial expressions; this finding suggests that these expressions are mediated by very primitive parts of the brain.

This "hardwired" affect responds to body need. For example, craving for salt was demonstrated by a dramatic case described in 1940. A 3½-year-old boy with an intense craving for salt died when his salt intake was restricted during a hospital stay. An autopsy revealed a tumor of his adrenal gland that had caused his body to lose sodium. His salt craving had kept enough sodium in his body to keep him alive (Wilkins and Richter, 1940).

specific hungers theory The idea that a deficiency of a given nutrient will produce craving (a specific hunger) for that nutrient. Curt Richter first proposed this idea; cravings for salty or for sweet are associated with deficiencies in those substances. However, the idea proved wrong for other nutrients (e.g., vitamins).

Specific Hungers

The case just described was widely seen as support for the **specific hungers theory** of Curt Richter. According to this view, the need for a nutrient causes the body to crave it. Ingestion of the nutrient reduces the craving and brings the body back into balance. Another source of support for this theory was a treatment for schizophrenia that was popular in the 1940s. At the time, some

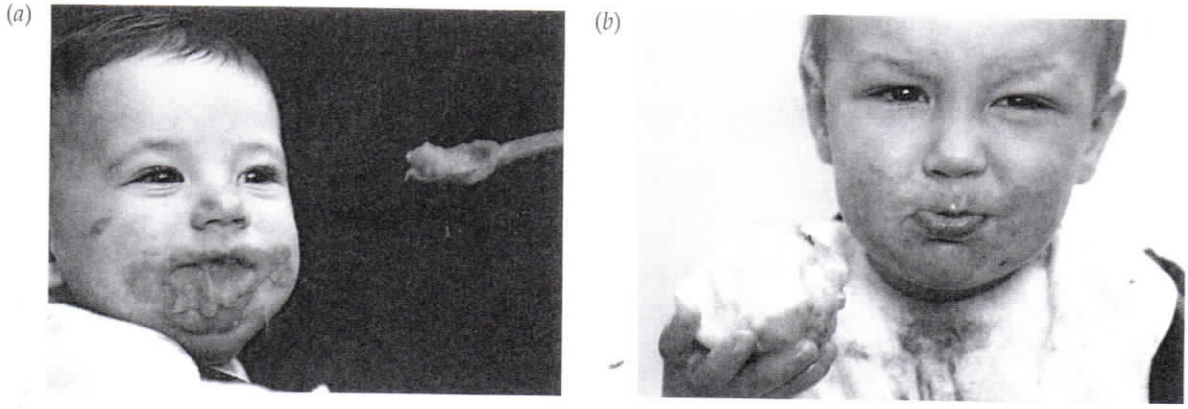


FIGURE 14.13 The two toddlers' facial expressions reveal the taste qualities that they experience. (*a*) Sweet potato produces the typical smile associated with the acceptance of sweet. (*b*) Green apple produces the puckery face associated with sour.

experts believed that the brain, which depends on glucose for fuel, could be forcibly rested if blood glucose were driven to very low values with insulin. Intense cravings for sweet were an unexpected by-product of the therapy. Later laboratory studies confirmed that insulin injections produce increased liking for sweet.

Yet more support for the idea of specific hungers seemed to come from the work of a pediatrician, Clara Davis. She allowed a group of 6-month-old infants to eat whatever they liked to see if they would choose wisely (C. M. Davis, 1928). The infants thrived, leading Dr. Davis to conclude that, when allowed to choose among a variety of healthy foods, infants had the ability to select a healthy diet.

The success of the specific hungers theory spurred further investigations that ultimately proved that the theory was limited only to sweet and salty. In one of the early studies, rats were fed a diet deficient in vitamin B_{12}, which made them sick. When the rats were offered a choice of remaining on the same diet or switching to a diet containing B_{12}, they immediately switched. But Paul Rozin conducted a crucial control. He gave the control rats the choice of the original diet or a different diet that was also deficient in B_{12}. These rats also immediately switched to the different diet. Thus the rats in the original study were not specifically seeking B_{12}; they had simply learned to avoid the diet on which they became ill (Rozin, 1967).

Rozin's work ended belief in specific hungers as an explanation of dietary regulation for anything beyond sugar and salt. In retrospect, we can see that the theory lacked an important ingredient. For craving to cause an animal to seek out and take in a needed nutrient, there would have to be a sensory cue unambiguously associated with the nutrient. The saltiness of salt and the sweetness of sugar could serve as such cues, but the B_{12} molecule does not produce a detectable cue in food (Figure 14.14).

We were left with a problem, though. How did Clara Davis's infants know how to select a healthy diet if specific hungers do not operate for all nutrients? It turned out that they were not selecting a healthy diet at all. They were simply eating a variety of the foods presented because they got bored eating single foods. Because all of the choices were healthy, all the infants needed to do was eat a variety. In the modern world, eating whatever we like will not produce good health, because too many of the available foods are not healthy. In fact, the specific hungers that are genuine can do us considerable harm; just think about our love for sweet and salty junk foods.

If specific hungers don't control all of what we eat, what does? Our likes and dislikes of food depend very much on our likes and dislikes of the

FIGURE 14.14 In our evolutionary past, when food was scarce and we had to expend considerable physical effort to get it, specific hungers for sugar and salt were adaptive. In the current era, in which foods are plentiful and easily obtainable, these specific hungers (combined with the profit motive for the food industry) lead many to consume too much junk food. The nutrients in vegetables are, alas, largely undetectable, so we cannot develop specific hungers for them.

retronasal olfactory sensations associated with foods. As we saw in the previous chapter, these olfactory likes and dislikes are not hardwired as those for taste are. Thus, our affect toward foods is made up of the hardwired affect contributed by taste, combined with the learned affect contributed by retronasal smell.

Pleasure and Retronasal versus Orthonasal Olfaction

There is still much that is not understood about the links between retronasal and orthonasal (through the nostrils) olfaction, including the pleasure or displeasure associated with these sensations. We know that we learn to like or dislike smells, but do we learn this separately for retro- and orthonasal olfaction? David Laing, an Australian expert on the chemical senses, suggested that this may be the case. He noted that many of us like the smell of recently cut grass, but few would like such a sensation if it turned up in food. On the other hand, when an aversion is learned retronasally, it often shows up with orthonasal olfaction as well. Bartoshuk recounts getting carsick on a childhood vacation while simultaneously eating chocolate-covered cherries. She now not only avoids the cherries, but finds cherry-scented soaps disgusting as well.

Chili Peppers

The pleasure that some people experience from chili peppers deserves special attention. We are not born liking chili peppers. Rozin studied the acquisition of chili pepper preference in Mexico and found that the process depended on social influences. Chili is gradually added to the diet of young children begin-

ning at about age 3, and the children observe their family members enjoying it. By age 5 or 6, children voluntarily add chili to their own food. At some point the chili is liked for its own sake.

A variety of arguments based on presumed health benefits have been introduced to account for our love of chili peppers. For example, some have argued that chilis kill microorganisms in food, thus acting as a preservative. Others have argued that chilis contain vitamins A and C, and that this gives them adaptive value (in other words, the pain of the chili serves as a cue for the presence of the vitamins). The pleasure that some people experience from chilis has also been linked to the idea that the resulting burn leads to the release of endorphins, the brain's internal painkillers.

One of the most interesting features of the liking for the burn of chili peppers is its near total restriction to humans. Rozin has documented a few cases on record of animals showing a liking for chilis, but these cases all involved pets fed chili pepper by their human companions. When Rozin tried to produce liking for chilis in rats, he failed. However, one of Rozin's students, Bennett Galef, who is famous for the study of social interactions among rats, was finally able to get rats to like a diet seasoned with a mild level of cayenne pepper by exposing the rats to a "demonstrator" rat that had just eaten the diet. It seems that growing to like chili peppers is a social phenomenon for rats as well.

The burn that we experience from chili peppers is highly variable across individuals (Figure 14.15). The variability comes from two sources. First, as noted earlier, supertasters have more fungiform papillae and therefore more pain fibers, and thus they perceive the most intense oral burn from chilis. Second, capsaicin, the chemical that produces the burn in chilis, desensitizes pain

FIGURE 14.15 Do these images inspire fear or delight in your taste buds?

receptors. This means that individuals who consume chilis quite often (once every 48 hours is sufficient) are chronically desensitized. Chili peppers produce considerably less burn to those who are desensitized.

Desensitization can come to your rescue if you accidentally order a meal that proves to be overspiced for your palate. After the first mouthful, wait until the burn has subsided. The mistake many diners make is to keep trying to eat. As long as the capsaicin continues to be applied, desensitization does not occur. Desensitization occurs during the decline of the burn (B. G. Green, 1993). Once the initial burn has faded, the rest of the meal can be consumed with relative comfort.

Capsaicin desensitization has important clinical value. The ancient Mayan Indians used a concoction made of chilis to treat the pain of mouth sores. Wolffe Nadoolman, then a medical student at Yale working in Bartoshuk's laboratory, created a similar remedy by adding cayenne pepper to a recipe for taffy. Cancer patients often develop painful mouth sores from chemotherapy and radiation therapy, and these patients showed a dramatic reduction in pain from those mouth sores when they used these candies (Berger et al., 1995). Although capsaicin can be used to reduce pain on any body site, it is worth noting that the skin is a potent barrier that prevents capsaicin from contacting pain receptors. Thus, capsaicin remedies for disorders like arthritis are rarely very satisfactory. In the mouth, the mucous membrane permits capsaicin to easily contact pain receptors, so there, desensitization is fast and powerful.

Refer to the ***Sensation and Perception*** website (www.sinauer.com/wolfe) for activities, essays, study questions, and other study aids.

Summary

1. Flavor is produced by the combination of taste and retronasal olfaction (olfactory sensations produced when odorants in the mouth are forced up behind the palate into the nose). Flavor sensations are localized to the mouth, even though the retronasal olfactory sensations are coming from the olfactory receptors high in the nasal cavity.
2. Taste buds are globular clusters of cells (like the segments in an orange). The tips of some of the cells (microvilli) contain sites that interact with taste molecules. Those sites fall into two groups: ion channels that mediate responses to salts and acids, and G protein–coupled receptors that bind to sweet and bitter compounds.
3. The tongue has a bumpy appearance because of structures called papillae. The filiform papillae (most numerous) have no taste buds. Taste buds are found in the fungiform (front of the tongue), foliate (rear edges of the tongue) and circumvallate (rear center of the tongue) papillae, as well as on the roof of the mouth.
4. Taste projects ipsilaterally from the tongue to the medulla, thalamus, and cortex. It projects first to the insula in the cortex, and from there to the orbitofrontal cortex, an area where taste can be integrated with other sensory input (e.g., retronasal olfaction).
5. Taste and olfaction play very different roles in the perception of foods and beverages. Taste is the true nutritional sense; taste receptors are tuned to molecules that function as important nutrients. Bitter taste is a poison detection system. Sweet taste enables us to respond to the sugars that are biologically useful to us: sucrose, glucose, and fructose. Salty taste enables us to identify sodium, a mineral crucial to survival because of its role in nerve conduction and muscle function. Sour taste permits us to avoid acids in concentrations that might injure tissue. Umami, the taste produced by monosodium glutamate, has been suggested as a fifth basic taste; however, the role of the taste of MSG in humans is unclear.
6. The importance of taste to survival requires that we be able to recognize each of the taste qualities, even when it is present in a mixture. By coding taste quality

with labeled lines in much the same way that frequencies are coded in hearing, nature has ensured that we have this important capability. These labeled lines are noisy. For example, acids are able to stimulate fibers mediating saltiness, as well as those mediating sourness. Thus, acids tend to taste both salty and sour.

7. Foods do not taste the same to everyone. Nontasters are born lacking a particular bitter taste receptor (this receptor binds the compounds PTC and PROP). Nontasters tend to have the fewest taste buds and live in a "pastel" taste world. Supertasters are born with the PTC/PROP receptor. They tend to have the largest number of taste buds and live in a "neon" taste world. Psychologists discovered these differences by testing people's ability to match sensory intensities of stimuli from different modalities. For example, the bitterness of black coffee matches the pain of a mild headache to nontasters but resembles a severe headache to supertasters. The way foods taste affects palatability, which, in turn, affects diet. Diet contributes to diseases like cancer and cardiovascular disease. Thus it is not surprising that disease risks vary with the genetic ability to taste.
8. For taste, unlike olfaction, liking and disliking is hardwired; for example, babies are born liking sweet and salty and disliking bitter. When we become deficient in salt or sucrose, liking for salty and sweet tastes, respectively, increases. Junk foods are constructed to appeal to these preferences. Liking the burn of chili peppers, on the other hand, is acquired and, with the exception of some pets, is essentially limited to humans. Because taste buds are surrounded by pain fibers, supertasters perceive much greater burn from chilis than do nontasters.