# 13 Olfaction

**THE STORY OF THE FINAL TWO CHAPTERS** of this book begins at the dawn of life itself. When single-cell organisms first appeared on Earth, their basic purpose in life was to take in some substances (food) and avoid others (toxins). As these organisms evolved into multicellular creatures, detecting chemicals in the environment continued to be crucial for survival. Systems to detect and analyze environmental molecules were thus the first senses to evolve.

Humans have two chemical detection systems: one for molecules floating in the air, and another for molecules that we put in our mouths. The technical names for these two systems are **olfaction** and **gustation**, respectively. The former, more commonly known as "smell," is the subject of this chapter. Gustation, which you probably know as "taste," will be explored in the next and final chapter (although, as you'll see, the distinction between tastes and smells is not as clear as you probably think).

## Olfactory Physiology

### *Odors and Odorants*

Olfactory sensations are called **odors**. The stimuli for odors are chemical compounds. However, not every chemical is an **odorant** (an odor-inducing substance). To be smelled, a molecule must be volatile (able to float through the air), small (less than about $5.8 \times 10^{-22}$ grams), and hydrophobic (repellent to water). Figure 13.1*a* shows the chemical structures of several odorant molecules. However, many molecules that would seem to meet the basic requirements still don't smell to us. Two examples are natural gas (methane) and a by-product of methane, carbon monoxide (Figure 13.1*b*). Our evolutionary ancestors would have had no reason to detect these substances, which are not dangerous in the concentrations found in nature. But because carbon monoxide buildups in enclosed spaces such as homes with gas furnaces can be fatal, gas companies add a compound (tertiary-butyl mercaptan) that we smell as rotten eggs, to act as a warning signal when your pilot light goes out. We also can't smell the molecules that make up the air we breathe, such as oxygen and nitrogen.

### *The Human Olfactory Apparatus*

Unlike vision and audition, but like touch and taste, the human olfactory system is tacked onto an organ that serves another purpose. The primary function of the nose (Figure 13.2) is to filter, warm, and humidify the air that we breathe. But the inside of the nose has small ridges that add turbulence to incoming air, called turbinates, causing a small puff of each breath to rise upward, pass through a narrow space called the **olfactory cleft**, and settle on a yellowish patch of mucous membrane called the **olfactory epithelium** (Figure 13.3).

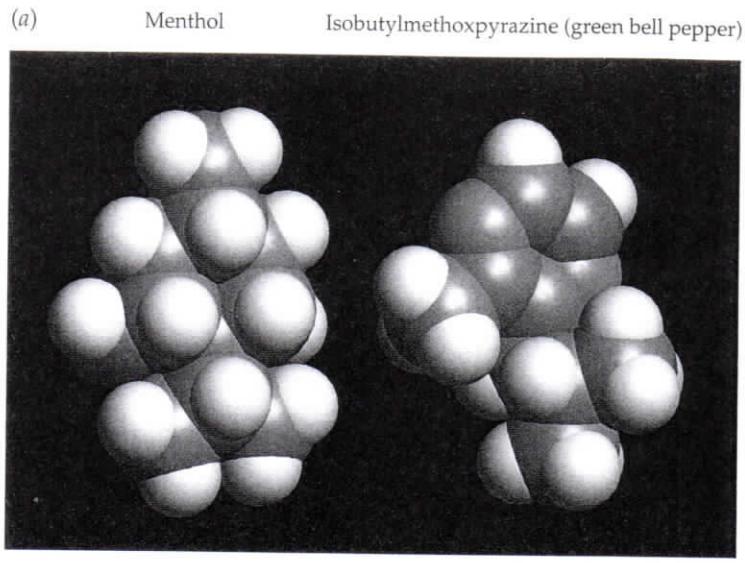



**FIGURE 13.1** Odorants. Most small, volatile and hydrophobic molecules activate the sense of smell (*a*), but there are also notable exceptions to the rule such as (*b*) methane and carbon monoxide.

**olfaction** The sense of smell.

**gustation** The sense of taste.

**odor** A general smell sensation of a particular quality. For example, "The cake had a chocolate *odor*." By contrast, when referring to a specific chemical entity, the term *odorant* should be used.

**odorant** Any specific aromatic chemical. For example, "You were given the *odorant* menthol to smell."

**olfactory cleft** A narrow space at the back of the nose into which air flows, where the main olfactory epithelium is located.

**olfactory epithelium** A secretory mucosa in the human nose whose primary function is to detect odorants in the inspired air. Located on both sides of the upper portion of the nasal cavity and the olfactory clefts, the olfactory epithelium contains three types of cells: olfactory sensory neurons, basal cells, and supporting cells.

**supporting cells** One of the three types of cells in the olfactory epithelium. This cell type performs supportive functions.

**basal cells** One of the three types of cells on the olfactory epithelium. The basal cells are precursor cells to olfactory sensory neurons.

**olfactory sensory neurons (OSNs)** The main cell type on the olfactory epithelium. OSNs are small neurons located beneath a watery mucous layer on the epithelium. The cilia on the OSN dendrites contain the receptor sites for odorant molecules.

This is the "retina of the nose." You have an olfactory epithelium at the back of each nasal passage, about 2.75 inches up from the nostril. Each epithelium measures about 1 to 2 square inches (depending on the size of your nose) and contains three types of cells: (1) **supporting cells**, (2) **basal cells**, and (3) **olfactory sensory neurons** (**OSNs**). (See Web Activity 13.1 Olfactory Anatomy.)

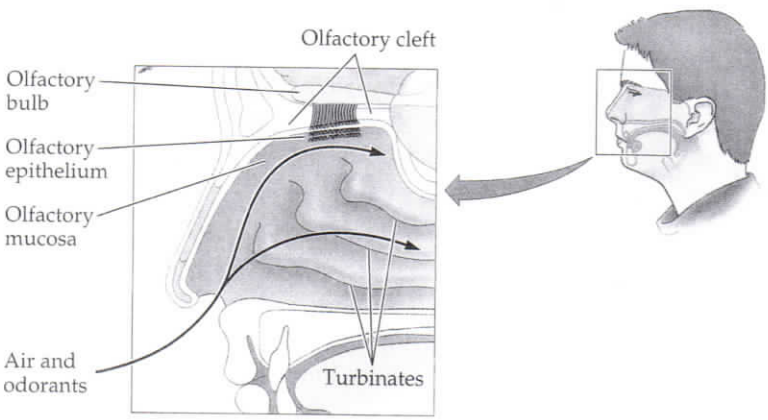



**FIGURE 13.2** The nose. Although the primary function of the nose is to warm and humidify the air that we breathe, it also serves to direct odorants onto the olfactory epithelium.

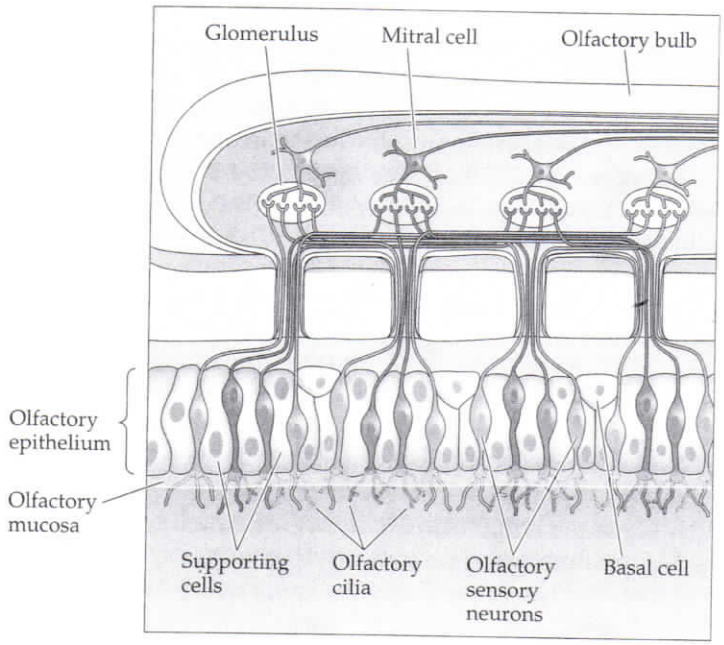



**FIGURE 13.3** The retina of the nose. The olfactory epithelium contains three types of cells: (1) olfactory sensory neurons (OSNs), (2) basal cells, and (3) supporting cells. The basal cells are precursors for the OSNs. The supporting cells have supportive functions. The OSNs are located beneath a watery mucous layer on the epithelium; the hairlike olfactory cilia of the OSN dendrites project through the mucus and are the receptor sites for odorant molecules.

OSNs (Figure 13.4) are small neurons that have cilia protruding into the mucus covering the olfactory epithelium. These cilia, which are actually the OSN's dendrites, have **olfactory receptors** (**ORs**) on their tips. The interaction between an odorant and the OR stimulates a cascade of biochemical events, ultimately producing an action potential that is transmitted along the axon of the OSN to the olfactory bulb (Schild and Restrepo, 1998). In order to initiate

**cilia (s. cilium)** Hairlike protrusions on the dendrites of olfactory sensory neurons. The receptor sites for odorant molecules are on the cilia, which are the first structures involved in olfactory signal transduction.

**olfactory receptor (OR)** The region on olfactory sensory neuron cilia where odorant molecules bind.

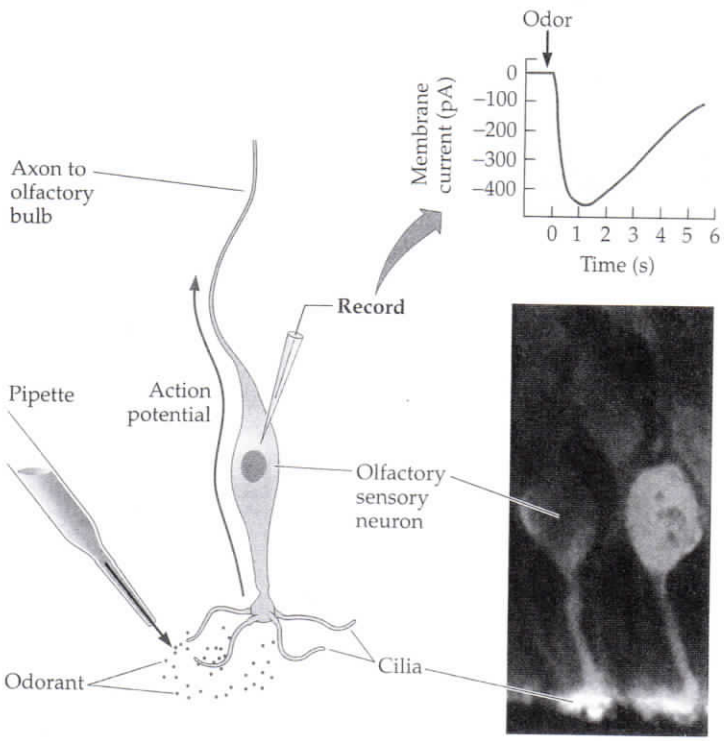



**FIGURE 13.4** Fluorescent image of an olfactory sensory neuron, with a schematic graph of an action potential sequence following odorant application. The OSN collects odorant molecules via receptors on its dendrites and sends action potentials to the brain through its axons. (Scan courtesy of C. Balmer and A. LaMantia.)

**cribriform plate** A bony structure riddled with tiny holes, at the level of the eyebrows, that separates the nose from the brain. The axons from the olfactory sensory neurons pass through the tiny holes of the cribriform plate to enter the brain.

**anosmia** The total inability to smell, most often resulting from sinus illness or head trauma.

an action potential, about 7 to 8 odor molecules must bind to a receptor, and it takes about 40 of these nerve impulses for a smell sensation to be reported.

We have approximately 20 million OSNs, split between the epithelia of our right and left nostrils. This is more receptors than we have in any other sensory system except vision, and these receptors allow us to distinguish thousands of different odors. However, the number of receptors we have is paltry compared to a bloodhound's 220 million OSNs. In addition, a much higher proportion of a dog's brain is dedicated to olfaction: about 5%, compared to 0.1% for humans. Researchers suspect that humans can smell about the same number of scents as dogs (the bloodhounds aren't talking, so we can't be sure), but dogs can sense odors at concentrations nearly 100 million times lower than humans can (Krestel et al., 1984; Willis et al., 2004). Other amazing smellers include pigs, which can smell the scent of truffles (the mushroom, not the chocolate) under 6 inches of soil; and salmon, which use smell to find the waters of their birth from hundreds of miles away (Dittman and Quinn, 1996).

The axons on the ends of OSNs opposite the cilia (dendrites) pass through the tiny sieve-like holes of the **cribriform plate**, a bony structure at the level of your eyebrows that separates the nose from the brain (Figure 13.5). A hard blow to the front of the head can cause the cribriform plate to be jarred back or fractured, slicing off the fragile olfactory axons, and consequently inducing **anosmia** ("smell blindness"), the total absence of a sense of smell. Stem cells in the olfactory epithelium can form new OSNs; indeed, all of your OSNs die and regenerate about once every 28 days. However, fractured cribriform

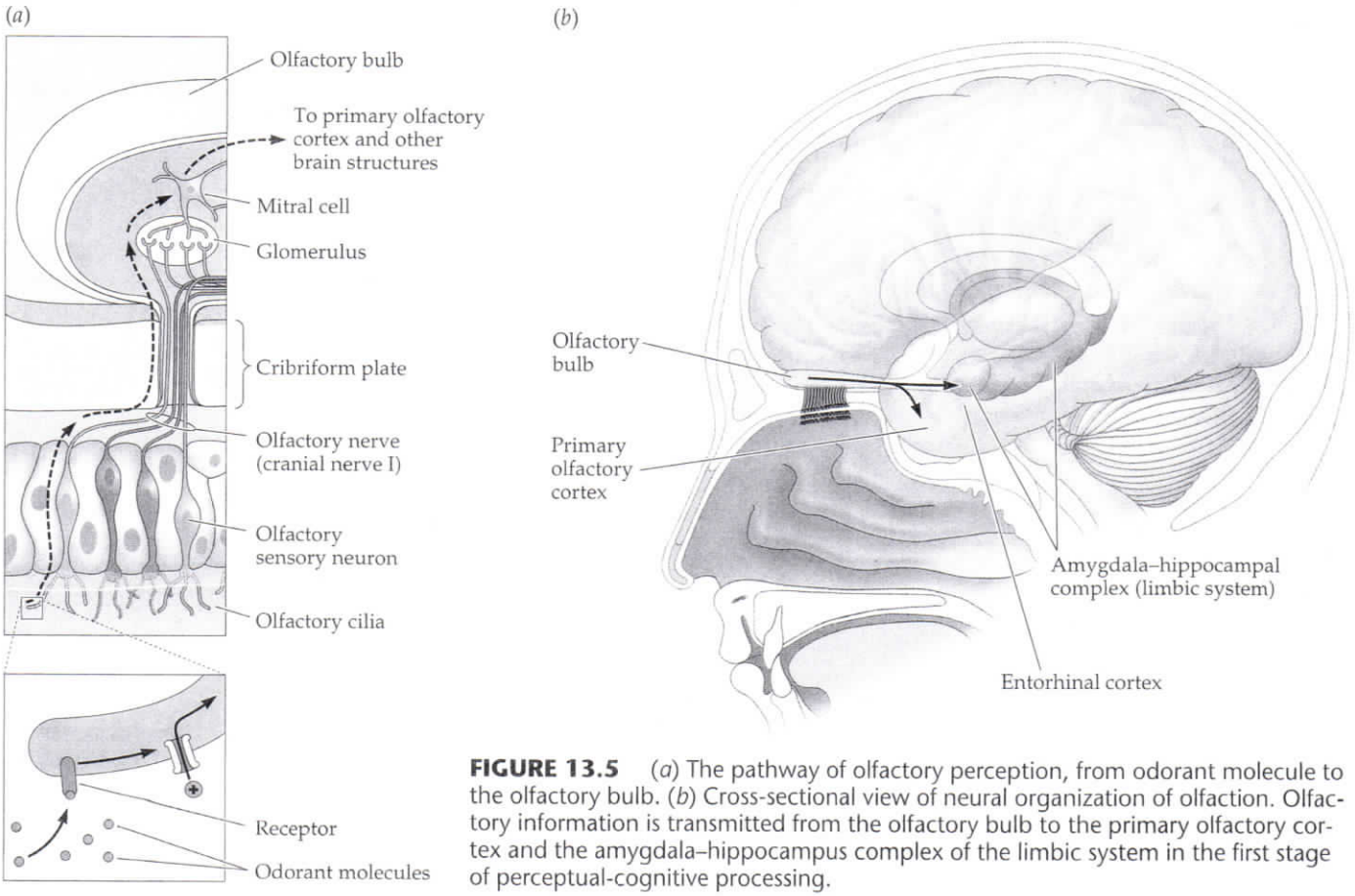



**FIGURE 13.5** (*a*) The pathway of olfactory perception, from odorant molecule to the olfactory bulb. (*b*) Cross-sectional view of neural organization of olfaction. Olfactory information is transmitted from the olfactory bulb to the primary olfactory cortex and the amygdala–hippocampus complex of the limbic system in the first stage of perceptual-cognitive processing.

plates typically scar over, preventing the new OSN axons from passing through to the brain and crippling the sense of smell for life.

Head trauma is not the only way that anosmia can be induced. The most common cause of olfactory loss is upper respiratory tract infection (e.g., sinus infection); the second most common cause is sinonasal disease (such as polyps), followed by head trauma; only 30% of anosmias are caused by head trauma. Certain medications can also cause smell loss or disturbance, particularly those used for treating high blood pressure or elevated cholesterol levels. The rarest form of anosmia is congenital; only 3% of people with anosmia are born anosmic. The likelihood of recovery is best when anosmia is caused by infection or disease, because often when the underlying illness is treated, normal smell function returns.

It is worth commenting here that compared to loss of vision or hearing, olfactory loss is paid little attention by both the medical and the general community. In a questionnaire administered to students at the University of Pennsylvania, loss of the sense of smell was ranked equal to losing one's big toe (unpublished data from Wrzesniewski, McCauley, and Rozin, 1999). However, anosmia can cause great suffering, as it affects many aspects of our lives, most noticeably our sense of taste, as you will discover in the next chapter. Moreover, the incidence of anosmia is surprisingly high. From a survey by the National Institutes of Health in 1994, it was conservatively estimated that 1 out of every 100 people suffers from anosmia. However, more recent estimates indicate that as many as 14 million Americans over the age of 55 have a severely compromised sense of smell. Including younger adults, it appears that about 1 in 20 Americans may have some olfactory dysfunction. Olfactory loss can also be the first symptom of neurological disorders, such as Alzheimer's and Parkinson's diseases. It is therefore very important to investigate olfactory loss when it occurs. Many smell and taste clinics throughout the country administer simple tests to determine the causes and treatment possibilities for olfactory loss and dysfunction.

In someone with a healthily functioning sense of smell, the OSN axons pass through the cribriform plate and bundle together to form the **olfactory nerve** (cranial nerve I) and enter a blueberry-sized extension of the brain just above the nose, called the **olfactory bulb** (see Figure 13.5). We have two olfactory bulbs, one in each brain hemisphere. Unlike the other senses we've studied so far, olfaction is **ipsilateral**, meaning that the right olfactory bulb gets information from the right nostril and the left olfactory bulb gets information from the left nostril.

Within the olfactory bulb are spherical conglomerates called **glomeruli** (singular *glomerulus*), where OSN axons synapse with dendrites of two other types of neurons: **mitral cells** and **tufted cells**. Recent molecular genetic studies in mice have shown that all neurons expressing a particular OR type, *no matter where they are on the nasal epithelium*, converge onto one glomerulus pair (consisting of one medial and one lateral glomerulus) in the olfactory bulb (Mombaerts et al., 1996). Thus, all the ORs detecting methyl salicylate, a synthetic chemical that smells like wintergreen mint, for example, send their axons to a single glomerulus pair. Higher brain structures receiving information from the olfactory bulbs therefore know that a signal coming from this glomerulus pair is consistent with your nose sniffing a mint candy. However, this relatively simple picture is complicated by the fact that each glomerulus may receive axons from several different receptor types.

Central brain structures that process olfactory information from the olfactory bulb include the **olfactory cortex**, the **amygdala–hippocampal complex**, and the **entorhinal cortex** (see Figure 13.5). These are all part of a net-

**olfactory (I) nerves** The first pair of cranial nerves. The axons of the olfactory sensory neurons bundle together after passing through the cribriform plate to form the olfactory nerve.

**olfactory bulb** The blueberry-sized extension of the brain just above the nose, where olfactory information is first processed. There are two olfactory bulbs, one in each brain hemisphere, corresponding to the right and left nostrils.

**ipsilateral** Referring to the same side of the body (or brain).

**glomeruli (s. glomerulus)** Spherical conglomerates containing the incoming axons of the olfactory sensory neurons. Each OSN converges onto two glomeruli (one medial, one lateral).

**mitral cells** The main projective output neurons in the olfactory bulbs.

**tufted cells** A secondary class of output neurons.

**olfactory cortex** The general brain region that processes smell. The olfactory cortex consists of the piriform cortex, the amygdala–hippocampal complex, and the entorhinal cortex.

**amygdala–hippocampal complex** The conjoined regions of the amygdala and hippocampus, which are key structures in the limbic system. This complex is critical for the unique emotional and associative properties of olfactory cognition.

**entorhinal cortex** A phylogenetically old cortical region that provides the major sensory association input into the hippocampus. The entorhinal cortex also receives direct projections from olfactory regions.

**limbic system** A group of neural structures including the olfactory cortex, the amygdala, the hippocampus, the piriform cortex, and the entorhinal cortex. The limbic system is involved in many aspects of emotion and memory. Olfaction is unique among the senses for its direct and intimate connection to the limbic system.

work of brain structures known as the **limbic system**, which is involved in many aspects of emotion and memory. As we will see later, these connections are the key to the unique associative learning and emotional properties of olfaction.

Olfactory receptor cells are different from all other sensory receptor cells in that they are not mediated by any protective barrier and they make direct contact with the brain. By contrast, visual receptors are protected by the cornea, receptors for hearing are protected by the eardrum, and taste buds are buried in papillae. One consequence of the fact that the olfactory sensory neurons are direct conduits into the brain is that many drugs can be inhaled. In spite of their direct linkage into the brain, OSN axons are among the thinnest and slowest in the body. Therefore, even though the nose connects directly to the brain, the time it takes to perceive an odor is long compared to our other sensory experiences. The lag time between sniffing and the brain registering a smell is variable, with a mean of approximately 400 ms (almost half a second); compare this to the 45 ms it takes for the visual cortex to register an image presented to the retina. This half-second duration for odorant registration does not take into account the time it takes for you to react to a scent, which effectively doubles the perceptual time, making olfaction a particularly slow sense. You have probably observed yourself that smells seem to emerge gradually, rather than flashing into your awareness.

These distinctions bring up the subtle differences between *sensation* and *perception* in olfaction. Sensation occurs when a scent is neurally registered; perception occurs when you become aware of detecting it. Similarly, odors tend to linger—because of both ambient airflow and receptor clearance speed. The relatively slow speed and lingering features of olfaction have been the central obstacles for developing effective "smell-o-vision" and smell virtual-reality technologies.

## The Genetic Basis of Olfactory Receptors

In 1991, molecular biologists Linda Buck and Richard Axel (who were rewarded with a Nobel Prize for their efforts) showed that the genome contains about 1000 different olfactory receptor genes, each of which codes for a single type of OR. All mammals appear to have pretty much this same set of 1000 genes, but interestingly, some proportion of the OR genes in each species are nonfunctional "pseudogenes": the genes are there in the chromosomes, but the proteins coded for by the genes never get produced. In dogs and mice, about 20% of the OR genes are pseudogenes; in humans, the proportion is much higher, between 60% and 70%.

Some researchers have recently suggested that the high proportion of OR pseudogenes in humans is the result of an evolutionary trade-off between vision and olfaction. Yoav Gilad and his colleagues (2004) observed that Old World primate species such as gorillas and rhesus monkeys have about 30% OR pseudogenes, whereas most New World species (e.g., squirrel monkeys) have a lower proportion (about 18%). The one New World exception is the howler monkey, which also has about 30% OR pseudogenes. It turns out that the howler monkey has something in common with the Old World primates: trichromatic color vision. Brains can get only so big, so to free up the brain space necessary to house human ancestors' evolving visual analysis tools (including trichromatic vision), we may have dropped the ability to analyze the odorants detected by the OR genes that became pseudogenes. The payoff from superior visual detection must have been better for our ancestors' survival than the disadvantages from diminished olfactory acuity.

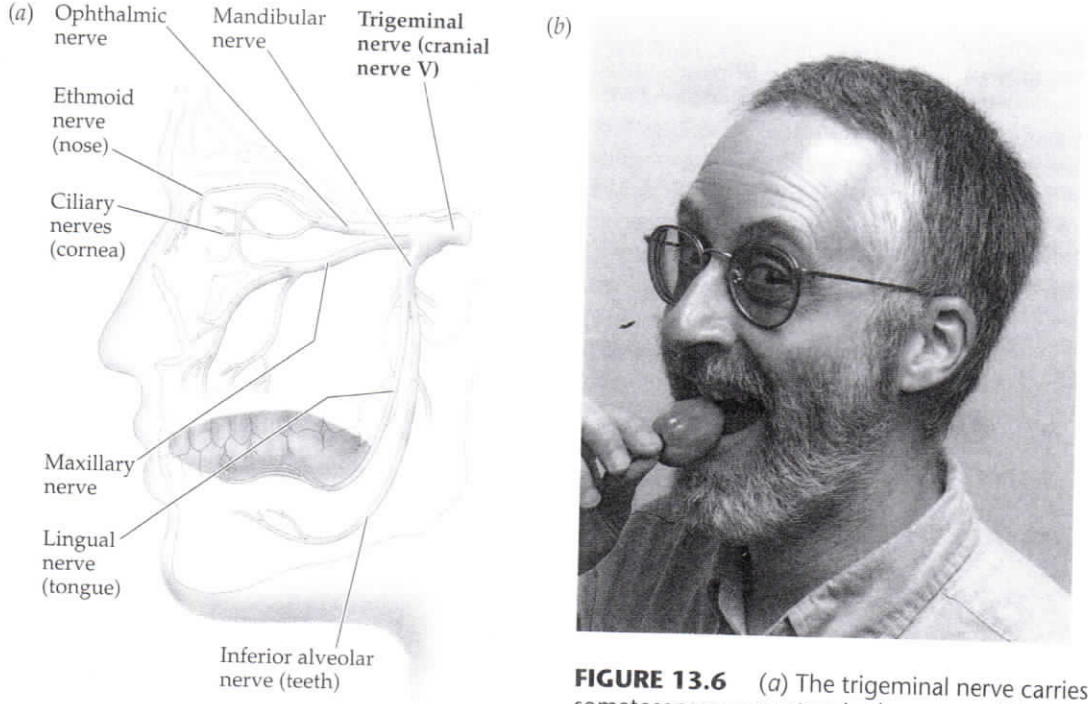



**FIGURE 13.6** (*a*) The trigeminal nerve carries information from somatosensory receptors in the nose and face to the thalamus and then on to the somatosensory cortex. (*b*) The irritation or excitement (depending on your culinary preferences) caused by eating chili peppers is due to trigeminal nerve stimulation.

### *The Feel of Scent*

An important dimension of odorant perception that is often unappreciated is that most odorants stimulate the somatosensory system to some degree through polymodal nociceptors (touch, pain, and temperature receptors) inside of the nose. For example, menthol feels cool and ammonia feels burning. These sensations are mediated by the **trigeminal nerve** (cranial nerve V) (Figure 13.6*a*). In many cases it is impossible to distinguish between the sensations traveling up cranial nerve I from the olfactory receptors and those traveling up cranial nerve V from somatosensory receptors. For example, the nasal irritation and odor associated with the smell of gasoline fuse to produce a holistic sensory experience. Trigeminal stimulation accounts for why we tear when we chop onions and sneeze when we sniff pepper. High levels of trigeminal stimulation (e.g., munching habanero peppers) can produce severe burning sensation (Figure 13.6*b*), and trigeminal activity has been linked to the facial-head pain felt in migraine headaches. "Smelling salts" (made from ammonia combined with eucalyptus oil) revive us because of their trigeminal activation.

**trigeminal (V) nerves** The fifth pair of cranial nerves, which transmit information about an important dimension of our sense of smell: the "feel" of an odorant. For example, menthol feels cool, and ammonia feels burning.

## From Chemicals to Smells

Now that we know something about the physiological basis of olfaction, we can ask the following crucial question: how does the biochemical interaction between an odorant and an OR, and subsequent neurological processing in the olfactory bulbs and later brain structures, result in the psychological perception of a scent such as lemon? Buck and Axel's seminal discovery of olfactory receptor genes has produced an explosion of research surrounding this question over the past decade, but a final theory of how we perceive scents has still not been fully developed.

**shape-pattern theory** The current dominant biochemical theory for how chemicals come to be perceived as specific odorants. Shape-pattern theory contends that odorant molecules have different shapes and olfactory receptors have different shapes, and that an odorant will be detected by a specific OR to the extent that the odorant's molecules fit into the OR. Different scents activate different arrays of olfactory receptors in the olfactory epithelia, producing specific firing patterns of neurons in the olfactory bulb, which then determines the particular scent we perceive.

## *Theories of Olfactory Perception*

At present, the best-accepted biochemical theory (first proposed in its modern form in the 1950s by the British scientist John Amoore) is based on the match between the shapes of odorants and odor receptors. It has been dubbed "shape theory" but is better denoted as "shape-pattern theory." In a nutshell, **shape-pattern theory** contends that odorant molecules have different shapes and olfactory receptors have different shapes, and that an odorant will be detected by a specific OR to the extent that the odorant's molecules fit into the OR (Figure 13.7). Gordon Shepherd and his students pioneered the idea that when a given odorant is sniffed, a particular pattern is generated across the glomeruli. Differences in those spatial patterns provide the basis for the array of odors that we perceive. The most recent molecular research suggests that scents are detected by means of a combinatorial code. Different scents activate different arrays of olfactory receptors in the olfactory epithelia, producing specific firing patterns of neurons in the olfactory bulb. The specific pattern of electrical activity in the olfactory bulb then determines the particular scent we perceive. That is, different patterns are elicited for the perception of vanilla, orange,

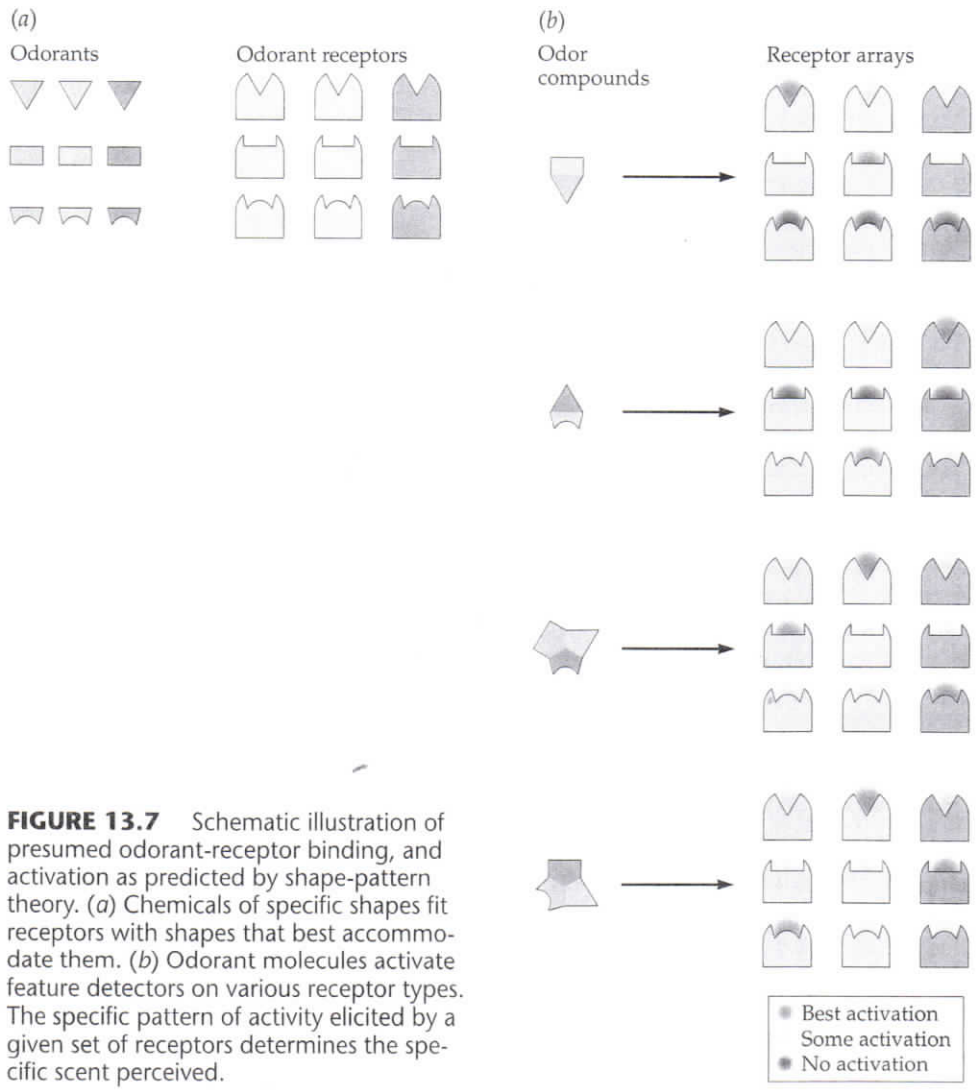



**FIGURE 13.7** Schematic illustration of presumed odorant-receptor binding, and activation as predicted by shape-pattern theory. (*a*) Chemicals of specific shapes fit receptors with shapes that best accommodate them. (*b*) Odorant molecules activate feature detectors on various receptor types. The specific pattern of activity elicited by a given set of receptors determines the specific scent perceived.

urine, and skunk (for example), and the various patterns for specific scents turn out to be highly consistent across individuals (Zou, Li, and Buck, 2005).

However, there are also alternative explanations for how olfaction works. The strongest alternative to shape theory is **vibration theory**, championed most recently by Luca Turin (Turin, 1996). In essence, vibration theory proposes that, because of atomic structure, every smell molecule has a different vibrational frequency, and molecules that produce the same vibrational frequencies have the same smell. Turin reported that various chemicals that have predictably similar vibrations due to their molecular composition also have similar smells. For example, all citrus odors fall into the same class of vibrational frequency. But other independent researchers have not validated Turin's claims (Keller and Vosshall, 2004).

Much less research has focused on the *vibration* theory than on the *shape* theory, so current advances may unfairly bias our understanding. Nevertheless, vibration theory cannot explain several conundrums of olfactory perception, such as *specific anosmias* and the different scents produced by *stereoisomers*, which shape-pattern theory can explain.

A **specific anosmia** is the inability to smell one specific compound when one otherwise has normal smell perception. Most specific anosmias are to steroidal musk compounds, and the condition appears to be genetic. The most studied specific anosmia is an inability to smell the compound androstenone, which is a characteristic chemical in armpit sweat.

Fifty percent of the population has a specific anosmia to androstenone. Interestingly, of the remaining 50% who can smell it, about half describe the smell as a "sweet musky-floral" scent, while the other half describe it as an unpleasant "urinous" odor. Most surprisingly, it was serendipitously observed that through repeated testing, sensitivity to androstenone could be induced in about half of the people who are initially unable to detect it (Wysocki, Dorries, and Beauchamp, 1989). That is, people who were anosmic to this particular chemical developed an ability to smell androstenone through repeated exposure.

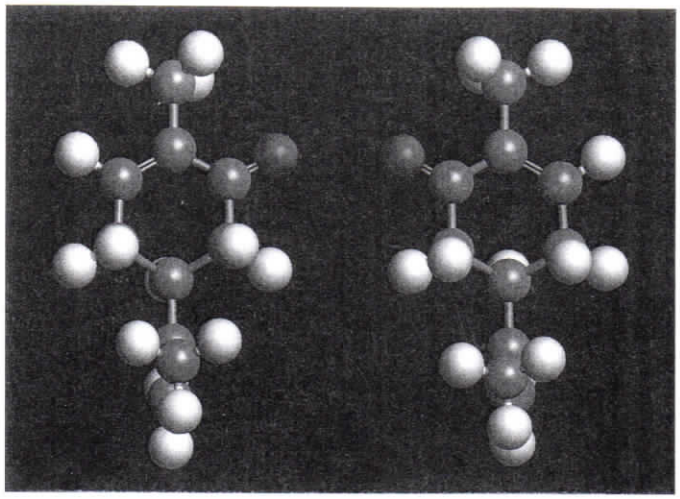
This change in detection ability to a specific chemical cannot be explained by vibration theory. Nor can vibration theory explain why some people perceive the scent as sweet-floral and others as urinous. However, shape-pattern theory can account for these observations. It is presumed that the specific anosmia and the various perceptions of the chemical androstenone are genetically determined. That is, in individuals with the specific anosmia, the receptors that detect androstenone are nonfunctional (coded by pseudogenes), and different receptors detect the compound in those who perceive a floral scent than those who perceive a urinous odor. From other areas of biology, we know that genes can be "turned on" by environmental pressures, and because each olfactory receptor is coded for by a specific gene, it is conceivable that the receptors for detecting androstenone are activated through repeated presentation of the chemical.

Another mark in favor of shape-pattern theory comes from the study of **stereoisomers**. Stereoisomers are molecules that are mirror-image rotations of one another, and although they contain all the same atoms, they can smell completely different. For example, *d*-carvone (the right-handed isomer) smells like caraway (Figure 13.8*a*), and *l*-carvone (the left-handed isomer) smells like spearmint (Figure 13.8*b*). According to shape-pattern theory, this difference arises because the rotated molecules do not fit the same receptors (as if you were trying to put your right hand into your left-hand glove), and thus, different receptors are activated for these two molecules, causing different scents to be perceived. Vibration theory cannot explain why stereoisomers smell different, because the vibration of steroisomers is the same.

**vibration theory** An alternate to *shape-pattern theory*, describing how olfaction works; most recently championed by Luca Turin. Vibration theory proposes that there is a different vibrational frequency for every perceived smell, and that molecules that produce the same vibrational frequencies will produce the same smell.

**specific anosmia** The inability to smell one specific compound with otherwise normal smell perception.

**stereoisomers** Isomers (molecules that can exist in different structural forms) in which the spatial arrangement of the atoms are mirror-image rotations of one another, like a right and left hand. Also called *optical isomers*.



*d*-carvone *l*-carvone

**FIGURE 13.8** These two stereoisomers contain the same atoms yet smell completely different. Shape-pattern theory can account for this fact.

More recent, and more direct, evidence for shape-pattern theory comes from experiments using cloned olfactory receptors, which have revealed chemical-receptor interactions in vitro that show specific odorants binding with specific receptors.

Like so many debates in psychology, it may be that neither shape-pattern nor vibration theory is a complete theory of smell (recall the initial conflicts between the trichromatic and opponent-process theories of color vision, for example). Shape-pattern theory currently has better general explanatory value, but it is also true that all molecules vibrate, and all receptors are made of atoms that vibrate as well. This leaves room for vibrational interactions between odor molecules and receptors to play a role.

## The Importance of Patterns

You may have noticed a potential discrepancy in how we have accounted for odorant perception. We've seen that we can discriminate many thousands of odors, yet our genes code for only about 1000 olfactory receptors, and 600 to 700 of them are nonfunctional. How can we detect so many different scents? To start to see the way out of this conundrum, recall that, in vision, we can tell the difference between thousands of different colors, even though we have only *three* types of cones. Each type of olfactory receptor responds to the structure of certain molecules only. However, a molecule may have features that stimulate several different receptors. Moreover, each receptor appears to have various "feature detectors" that contribute to further specificity in OR activation. Thus a different *set* of feature detectors is activated when you smell chocolate compared to when you smell turpentine (see Figure 13.7*b*).

As with detecting colors, we detect odors by considering the *pattern* of activity across various different receptor types. The intensity of an odorant also changes which receptors (and hence patterns) will be activated, which is why weak and strong concentrations of an odorant do not smell quite the same. The fact that receptor activation is affected by odor intensity probably explains why dogs with many more functional receptors than we have can perceive considerably lower concentrations of odorants. The timing of olfactory receptor activation is probably also important; an odorant that activates several receptors will also stimulate them in a specific time order. Another

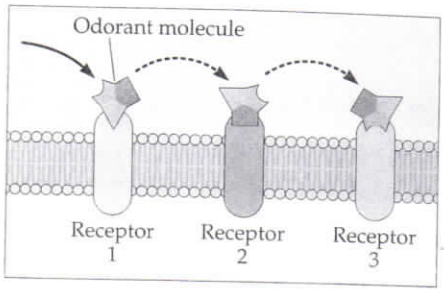



**FIGURE 13.9** Illustration of the hypothetical role of OR receptor activation timing and order. A single molecule binds first to receptor type 1, then a split second later to receptor type 2, then to receptor type 3. Brains are especially well suited to recognize patterns of responses such as this, so the number of odorants we can recognize greatly exceeds the number of receptor types we have available.

odorant might stimulate the same receptors in a different order, and the difference might lead to the perception of a different smell (Figure 13.9).

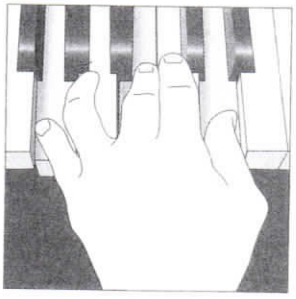
The flip side of the pattern perception mechanism is that if two molecules, or combinations of molecules, activate the same receptors in the same order, you will end up smelling the same thing. For example, the feature detectors for the single molecule phenyl ethyl alcohol and a real live rose in your garden, whose scent is composed of more than 1000 different molecules, might both result in the same basic pattern of activation and hence the same perception of "rose" (this should remind you of metamers in color vision).

## Odor Mixtures

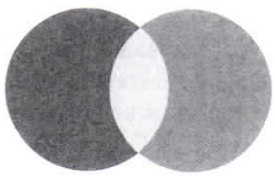
Just as we rarely hear pure tones outside of auditory perception experiments, we rarely smell "pure odorants" outside of an olfactory perception lab. Almost all of the olfactory stimuli that we encounter in the real world are mixtures, like the thousand-molecule rose scent emanating from your flower bed that we discussed in the previous section. How do we process the components in an odorant mixture? There are two broad possibilities: *analysis* and *synthesis*. Auditory mixtures provide the classic example of analysis. A high note and low note played simultaneously on a piano each can be analyzed out of the mix and perceived separately (Figure 13.10*a*). Color mixtures provide the classic example of synthesis. If we mix red and green lights, the resulting color that we see is yellow (Figure 13.10*b*). Red and green cannot be analyzed out, because the two lights have been synthesized into something else. Is olfaction (Figure 13.10*c*) an analytic or a synthetic sense?

Although we are capable of discriminating thousands of different odorants, intuition tells us that most mixtures are perceived as unitary wholes. For example, the smell of bacon is very distinctive, and most people would perceive it to be a unitary sensation. But there is no "bacon" odorant; the sensation we recognize as bacon is made up of a combination of many different volatile chemicals. To test the synthetic/analytic nature of olfactory perception, Laing and his colleagues (Laing and Glemarec, 1992; Laing and Francis, 1989), conducted a series of experiments in which they asked untrained par-

(*a*) Auditory mixture: analysis



(*b*) Color mixture: synthesis



(*c*) Olfactory mixture: analysis and synthesis

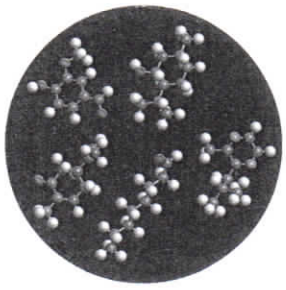


**FIGURE 13.10** We can break the three tones out of the musical chord being played in (*a*), but we cannot separately perceive the high- and medium-wavelength light rays mixing in the center of (*b*). When we mix odorants (*c*), we perceive the mixture primarily synthetically but some degree of analytical perception is possible. Analytical ability varies with prior training and what odorants comprise a mixture.

ticipants, participants who received preliminary "odor training," and experienced perfumers and flavorists to identify the constituents of mixtures containing between one and five common odorants. The average discrimination rate from all the participants combined was no more than three components in a five-component mixture. However, the trained participants did better than the untrained, and the experts did the best overall. Thus it appears that olfaction is primarily a synthetic sense, but that a certain amount of analytical ability can be developed with training.

### *Odor Imagery*

Though visual and auditory imagery is possible, humans appear to have little or no ability to conjure odor "images." For example, you can probably easily generate a visual image of a Hershey's chocolate kiss right now (you might even start salivating). But can you really reproduce the smell in your "mind's nose"? Brain-imaging studies (e.g., Kosslyn et al., 1995) have shown that many of the same parts of the brain that would be involved in actually seeing the kiss are also involved in visually imagining it; however, with olfaction, similar studies suggest that the degree of overlap between smelling an odor and "imaging" it is much weaker. Dreams with olfactory sensations are also very rare (Carskadon et al., 1989; Zadra, Nielson, and Donderi, 1998). Other animals, such as rodents, which rely predominantly on smell as the sense to negotiate the world, may well think and dream in smell. Because we do not think in smell, it is not necessary to have stored representations of olfactory experiences. It is possible, however, that with training, one might be able to develop the capacity to create sensory representations of smells, and many perfumers and chefs insist that they can image olfactorily.

## Olfactory Psychophysics, Identification, and Adaptation

We've come across the subfield of psychology called **psychophysics** a number of times already in this book. The goal of the psychophysicist is to quantify the psychological experience of our sensory world. In this section, we will briefly discuss some of the nuts-and-bolts questions addressed by olfactory psychophysics, then go on to consider the related and more interesting questions of how we identify and adapt to odors.

### *Detection, Discrimination, and Recognition*

Perhaps the most basic question we can ask for any sense is this: how much stimulation is required before we perceive something to be there? As with other senses, our olfactory *detection thresholds* depend on a number of factors. For instance, odorant molecules with longer carbon chains (e.g., vanillin) are easier to detect—have lower detection thresholds—than those with shorter carbon chains (e.g., acetone, otherwise known as nail polish remover). Women also have generally lower olfactory detection thresholds than men, especially during the ovulatory period of their menstrual cycles. But contrary to popular belief, research has shown that olfactory sensitivity is *not* heightened during pregnancy (Hummel et al., 2002; Laska et al., 1996). Taste is another story, as we'll see in the next chapter.

**psychophysics** The science of defining quantitative relationships between physical and psychological (subjective) events.

As we've already noted, a healthy person can *discriminate* thousands of odors. The more "nose" training you have (e.g., professional perfumers and

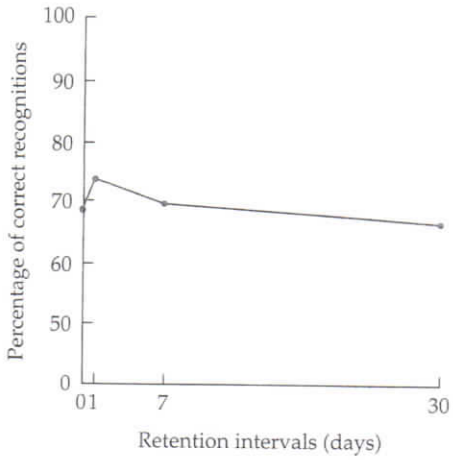



**FIGURE 13.11** The first point on this graph at 70% indicates odor recognition accuracy with only a 30 second delay from learning. Note, however, that this accuracy level is the same after one week and has dropped only about 3% after a retention interval of one month. This retention rate remains relatively constant over intervals at least as long as one year. (From Engen and Ross, 1973.)

wine tasters), the more odors you can discriminate; it has been claimed that professionals can distinguish up to 100,000 odors (Dobb, 1989). Note, however, that discrimination is not the same thing as *recognition*, the ability to remember whether or not you've smelled an odor before. It takes as much as three times as many odorant molecules floating through your nose to recognize an odor than it does to simply detect that the odor is there. You've probably experienced this phenomenon yourself: a smell will often register quite a while before you realize that you know what the smell is.

Another interesting feature of odor recognition is its durability. In controlled experiments, a 30-second delay between odor presentation and testing produces a precipitous drop in recognition accuracy, but what we remember after 30 seconds is very close to what we remember after 3 days, 1 month, or even 1 year (Figure 13.11) (Engen, Kuisma, and Ross, 1973; Engen and Ross, 1973; Murphy et al., 1991; Rabin and Cain, 1984). Again, you probably recognize this phenomenon from your own life. If you smelled a certain flower only once when you were a child, and you came upon that flower again 20 years later, you might very well know that the odor was familiar to you. Our memory for odors is especially potent if emotion is experienced during initial exposure (Herz, 1997).

## *Identification*

Attaching a verbal label to a smell is yet another step beyond odor recognition. Olfaction has been called "the mute sense" because we are often so lost for verbal descriptors for our olfactory experiences (Ackerman, 1990). All of us have had the experience of not being able to come up with the name of something that we know; for example, what was the name of your fourth-grade teacher? This experience is known as the "tip-of-the-tongue" phenomenon. In the olfactory domain, suppose you take a sniff of something from a bottle, where you have no visual clues to what it is, and you immediately know that the scent is extremely familiar, but you just can't come up with the name for it. Borrowing from the verbal scenario, we call this experience the **tip-of-the-nose phenomenon** (Lawless and Engen, 1977), and it can be a very frustrating situation.

**tip-of-the-nose phenomenon** The inability to name an odorant, even though it is very familiar. Contrary to the tip-of-the-tongue phenomenon, one has no lexical access to the name of the odorant, such as first letter, rhyme, number of syllables, and so on, when in the tip-of-the-nose state. This is one example of how language and olfactory perception are deeply disconnected.

There are some important differences between the tip-of-the-nose state and the tip-of-the-tongue state. For one, in the tip-of-the-tongue state we might not know the exact word we're looking for, but we might know its first letter,

its general word configuration, the number of syllables in the word, and so on ("Starts with a *K*, two syllables, sounds like some German word ... Aha, my fourth-grade teacher was Mrs. Kaiser!"). In the tip-of-the-nose state, we typically know nothing about the label we're searching for. But even though we are clueless about the odor's name, we do usually know how to respond to it appropriately. If you smelled maple syrup, even if you couldn't name it you would know it was something that you liked to eat; whereas if you smelled shoe polish, you would know that it wasn't something you'd want to put in your mouth. (These examples might remind you of brain damage patients with visual agnosia, who often know how to use an object even if they don't know what it's called.)

Anthropologists have found that in all languages that have been studied, there are fewer words that refer exclusively to our experience of smells than there are for any other sensation (Classen, Howes, and Synnott, 1994). In English, *aromatic*, *fragrant*, *pungent*, *redolent*, and *stinky* pretty much exhaust the list of adjectives that specifically describe olfactory stimuli and nothing else. More common terms used to describe odors, like *floral* or *fruity*, are references to the odor-producing objects (flowers and fruits), not the odors themselves. We also borrow terms from other senses (chocolate smells *sweet*, grass smells *green*, and so on) (Figure 13.12).

There are various possible explanations of why our sense of smell and language are so disconnected. First, unlike what happens in other sensory systems, olfactory information is not integrated in the thalamus prior to processing in the cortex, and it is argued that the thalamus has relevance for language. Second, a large body of evidence indicates that the majority of olfactory processing occurs in the right hemisphere of the brain, whereas language processing is known to be dominant in the left hemisphere (see Royet and Plailly, 2004, for review). It has even been suggested that odors are hard to name because of competition between odor and language processing for cognitive resources that share the same neural substrates (Lorig, 1999). This latter theory is supported by a recent study using brain-imaging techniques, which showed that the presence of an odor altered the semantic processing of words and degraded word encoding, but did not influence nonsemantic processing (Walla et al., 2003). (See also **Web Essays 13.1 Verbal–Olfactory Interactions** and **13.2 Olfactory Lateralization**.)

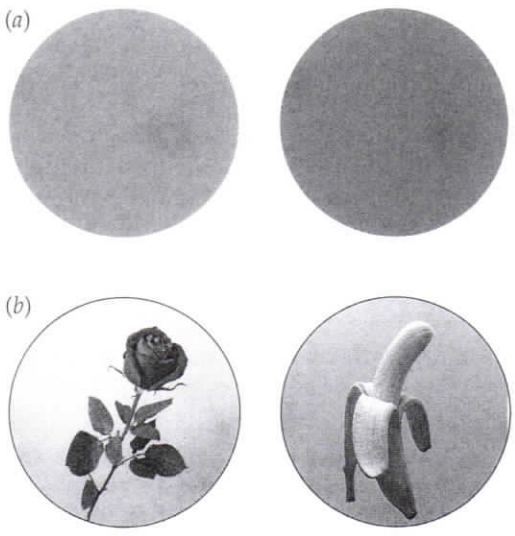



**FIGURE 13.12** It's easy to describe the difference between the two colors in (*a*): one is a greenish blue and the other a purplish blue. Now try describing the difference between the odors of the two objects in (*b*). This is much more difficult, in part because languages provide very few words that refer exclusively to smells.

## *Adaptation*

Have you ever had the experience of noticing coworkers or classmates who seem to be pouring a bottle of cologne over their head every morning, leaving you choking on the overpowering aroma? Can't they smell anything? Or perhaps you've noticed that, now that you've had your bottle of cologne for a few months, you can't smell the fragrance in it anymore. Or you've gone away on vacation and returned home to find that your house seems to have a "funny" smell that it didn't have when you left? What's going on? The answer has two parts: the first involves your nose and the second your mind.

The sense of smell is essentially a change detector. When a new chemical comes along, your olfactory system orients to it, your olfactory receptors fire in response to it, and you perceive a scent. For example, when you first enter a bakery, you notice the mouthwatering aromas of the cakes, cookies, pies, and breads. But if you stand inside inspecting these sweet baked goods for a while, trying to decide which one to get for dessert, the odorant molecules that make up the bakery aroma bind to the corresponding olfactory sensory neurons in your nose and actually make the olfactory receptors bury themselves inside their cell bodies, like an ostrich sticking its head in the sand (Figure 13.13) (Firestein, 2001). The receptors are therefore no longer physically available to respond to the bakery scent molecules. This response is a process in "receptor recycling." Specifically, odorant binding to an OR causes the OR to be internalized into its cell body, where it becomes unbound from the odor-

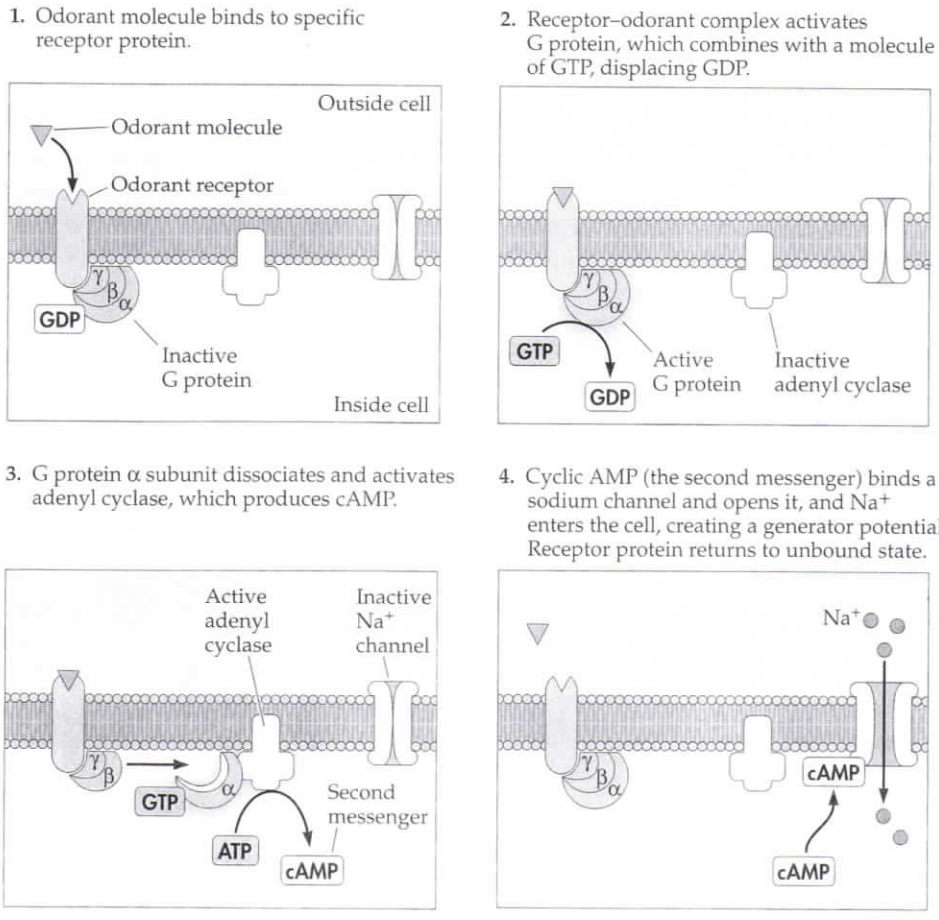



**FIGURE 13.13** The biochemical pathway for odorant molecule-odorant receptor binding, showing the process of receptor recycling that occurs during odor adaptation.

**G protein–coupled receptors (GPCRs)** The class of receptors that are present on the surface of olfactory sensory neurons. All GPCRs are characterized by a common structural feature of seven membrane-spanning α-helices.

**receptor adaptation** The biochemical phenomenon that occurs after continuous exposure to an odorant, whereby the receptors stop responding to the odorant and detection ceases.

**cross-adaptation** The successive reduction in detection of an odorant following exposure to another odorant. Cross-adaptation is presumed to occur because the two odorants share one or more olfactory receptors for their transduction, but the order of odorant presentation also plays a role.

ant and is then recycled through the cell and emerges again in a number of minutes. Receptor recycling is a mechanism common to all receptors in the class to which ORs belong: **G protein–coupled receptors** (**GPCRs**). So if you linger in the bakery for some time, by the time you get to the counter to purchase your dessert, you actually won't be able to smell it anymore!

This process is called **receptor adaptation**. The precise length of time for adaptation to occur varies as a function of both the individual (Dalton, 2002) and the odorant (Pierce et al., 1996). On average it takes about 15 to 20 minutes of continuous exposure to an odorant for the molecules to stop eliciting an olfactory response. Receptor adaptation can also be undone relatively quickly. Stepping outside the bakery for a few minutes will cause the receptors to pop back out, so when you walk back in you can enjoy the appetizing scents again.

One way to prolong the effect of smelling a scent before adaptation kicks in is to dispense an odor intermittently. For example, bursts of air freshener alternated with no scent will draw out the time before your receptors are smothered by the air freshener molecules and duck for cover. Dalton (1996) also showed that psychological processes can have an effect on adaptation rates. In one experiment, half the participants were told that an odorant they were being exposed to was "healthful," while the other half of the participants were told the odorant was "hazardous." Twenty minutes after initial exposure, the participants smelling the supposedly healthful odorant had adapted to it, whereas the participants who thought they were smelling a hazardous molecule actually became sensitized—they reported the smell as even more intense after 20 minutes than at the start of the experiment. This very different reaction in the two sets of participants occurred even though all of them had been smelling *the very same odorant*, isobornyl acetate, which has a balsam woody odor and is completely harmless.

In some cases, exposure to one odorant can raise the odor detection threshold for a second, completely different odorant. For example, when you're picking out a perfume in a department store, your nose may become fairly useless after several samples, despite the salesperson's insistence that all the fragrances are quite different from each other (Figure 13.14). This phenomenon is called **cross-adaptation**, and it is presumed to occur when the two


**FIGURE 13.14** Do these five fragrances smell the same? No, but because of olfactory cross-adaptation, if you've smelled four of them in succession, the subtleties of the fifth one may be lost to your nose.

odorants in question rely on similar sets of olfactory receptors. However, this simple explanation is complicated by the fact that most cross-adaptation relationships are nonreciprocal. For example, pentanol (a chemical used in some paints) seems to have a strong cross-adapting effect on propanol (used as an antiseptic and solvent), whereas smelling propanol first has only a small cross-adapting effect on pentanol (Cain and Engen, 1969). Furthermore, exposure to the first odorant can sometimes *enhance* sensitivity to the second odorant. (See **Web Activity 13.2 Odor Adaptation and Habituation.**)

Regardless of why they occur, cross-adaptation effects, like self-adaptation effects, usually go away after a couple of minutes. Professional perfumers, who may have to smell hundreds of scents per day and thus don't have a couple of minutes to spare, use a trick of sniffing their bare arm or cotton shirt sleeve between smelling odorants, and doing this effectively clears the nose even at a fast pace of odorant presentation. Nobody knows why this works, but it does. The next time you find yourself suffering from numb nose in a department store, try it.

## Cognitive Habituation

Receptor adaptation explains why you lose the delicious aroma of the bakery after you've been in the store for awhile, but can smell it again after a short break outside. However, if you took a job at a bakery, a different process would take place. This is the phenomenon that your friend who can't smell his cologne is experiencing, and it's the reason why you don't smell your house unless you go out of town for a couple of weeks. It is a psychological effect called **cognitive habituation**.

**cognitive habituation** The psychological process by which, after long-term exposure to an odorant, one is no longer able to detect that odorant or has very diminished detection ability.

In short, when you live with an odor, you cognitively habituate to it and no longer react to it, or you show a very diminished response to it. For example, textile workers exposed daily to acetone exhibited acetone detection thresholds that were eight times higher than a comparable group of control subjects, although their thresholds to another chemical (butanol), to which neither group was regularly exposed, were no different from each other (Wysocki et al., 1997). We habituate to some degree to stimuli presented to all our senses (you will eventually learn to sleep through your roommate's snoring, for example), but attention can bring us out of habituation with every sense except smell. Unlike receptor adaptation, which can be undone in a few minutes, cognitive habituation requires weeks to reverse, even for pungent trigeminal stimulants like acetone (Dalton et al., 1997; Wyscoki et al., 1997). That is, if you stopped wearing your cologne for 5 days, you would still *not* be able to smell it again once you put it back on. But if you abstained for 2 weeks or more you would.

Dalton (2002) has suggested that at least three mechanisms could be involved either singularly or in interaction to produce olfactory habituation. First, the olfactory receptors that are internalized into their cell bodies during odor adaptation may be more hindered after continuous exposure and take much longer to recycle than they normally would. Second, from continuous exposure, odorant molecules may be absorbed into the bloodstream, then transported to the olfactory receptors via nasal capillaries when you breathe out through your nose. As long as the odorant chemicals remain in your bloodstream, you will be constantly adapted (Maruniak, Silver, and Moulton, 1983). Finally, cognitive-emotional factors such as the process observed in the experiment in which participants were told that odors were harmful and then did not adapt may be involved (but in the reverse direction) in cognitive habituation.

**odor hedonics** The liking dimension of odor perception; typically measured with scales pertaining to an odorant's perceived pleasantness, familiarity, and intensity.

Another feature of odor perception that highlights the importance of attention is that we cannot smell while we are asleep (Carskadon and Herz, 2004). Unlike responses to auditory stimuli, presenting participants who were in deep (stages 3 and 4) or dreaming (REM) sleep with trigeminally activating odors such as peppermint and pyridine (even at high concentrations) did not awaken them or elicit EEG (electroencephalogram) sleep pattern changes. Recent neuroimaging research on awake subjects during smell tasks indicates that attentional circuits in the brain must be activated if we are to "detect" a scent (Zelano et al., 2005). Therefore, it may be that, because attention is effectively cut off during sleep, so is our ability to respond to odors. Note again the issue of sensation versus perception. Although odors can be registered by the brain without conscious awareness, olfactory perception depends on attention.

## Olfactory Hedonics

The most immediate and basic response we have to an odor is whether we like it or not. Such affective evaluations are known as **odor hedonics**. In tests of odor hedonic evaluation, ratings for how pleasant, familiar, and intense a person finds a given odorant are typically taken. These measures are then used to determine the hedonic value of a specific smell. It is obvious that perceived pleasantness is related to our liking for an odor. But how are familiarity and intensity related to our liking for specific scents?

### *Familiarity and Intensity*

As in many other life situations, we tend to like odors that we've smelled many times before. That is, familiar odors tend to be better liked than unfamiliar odors. Moreover, pleasant odors are often perceived as familiar, even if we haven't smelled them before (Moskowitz, Dravnieks, and Klarman, 1976; Sulmont, Issanchou, and Koster, 2002). Thus there is a linear relationship between ratings of odor pleasantness and familiarity with odor liking.

Intensity has a more complex relationship to odor liking and often shows an inverted-*U* function, but this depends on the odorant. A rose scent may be evaluated as more positive with increasing intensity, up to a point—where the function reverses, and as the scent becomes stronger it is judged to be more disagreeable (Figure 13.15*a*). By contrast, a weak fishy odor may be acceptable, but as intensity increases, its perception becomes more negative (Figure 13.15*b*).

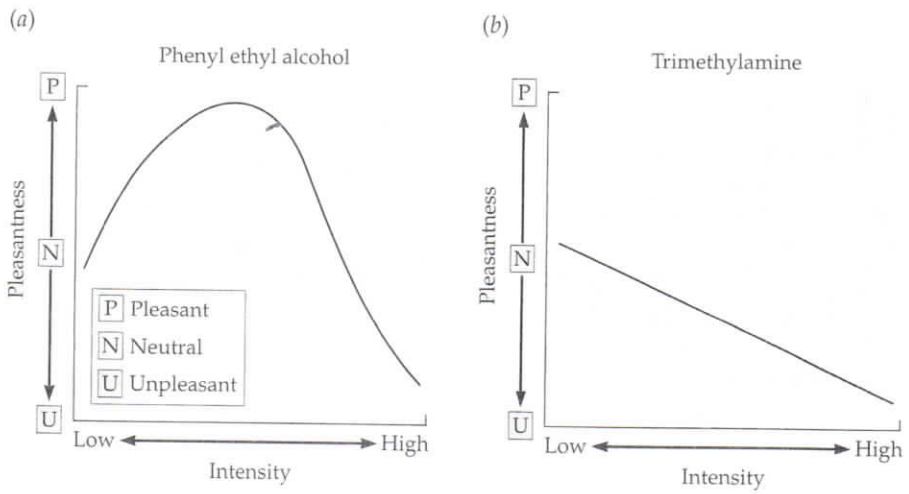



**FIGURE 13.15** Graphs showing pleasantness ratings of odorants plotted against intensity. (*a*) The relationship between odor intensity and pleasantness is often described by an inverted-*U* function, if the odorant is initially considered pleasant, as the synthetic rose scent (phenyl ethyl alcohol) usually is. (*b*) For an odorant that is initially considered tolerable (but not necessarily pleasant), such as fishy smelling trimethylamine, the relationship may more aptly be described by a linear graph like this.

## Nature or Nurture?

A long-standing debate in the olfaction literature centers around whether hedonic responses to odors are innate or learned. Researchers on the *innate* side of the debate claim that we are born with a predisposition to like or dislike various smells. In other words, rose is inherently a good smell and skunk is inherently a bad smell, the way bitter taste is inherently unpleasant to us and sweet inherently pleasant (see Chapter 14). In contrast, researchers taking the *learned* view hold that we are born merely with a predisposition to learn to like or dislike smells, and that whether a smell is liked or not is determined by the emotional value (good or bad) of the experiences that have been associated with it. That is, if you like rose and dislike skunk, it is because you have acquired a good and bad association, respectively, to these two scents. You need not have direct contact with a skunk to form such an association, though, because cultural learning provides meaning to many unencountered stimuli.

If asked to take a position yourself, on the sole basis of your own personal experiences, it's pretty likely you would come down on the innate side of the debate. After all, who could like the smell of skunk, and who wouldn't like the smell of a rose? In fact, however, a great deal of evidence suggests that odor hedonics are almost exclusively learned.

A good place to start looking for such evidence is with infants. If odor preferences are innate, then newborns should display them. However, researchers have repeatedly found that when presented with odorants they have probably never encountered before, infants and children often display very different preferences from those of adults. For instance, infants find the smells of sweat and feces pleasant (Engen, 1982; M. Stein, Ottenberg, and Roulet, 1958), and toddlers do not hedonically differentiate between odorants that adults find either very unpleasant (e.g., butyric acid, found in rancid foods) or pleasant (e.g., amyl acetate, which smells like banana).

One difficulty in doing these types of studies is that the olfactory system is fully functional by the third month of **gestation** (6 months before the baby is born) (Schaal, Marlier, and Soussignan, 1995, 1998; Winberg and Porter, 1998), and odorant molecules do find their way into the womb. So it is difficult to know exactly how much exposure even a newborn infant has had to an odorant. But exposure to in-utero odorants has led to yet another line of evidence in support of the learned view of hedonics: Menella and colleagues found that mothers who consumed distinctive smelling volatiles (e.g., garlic, alcohol, and cigarette smoke) during pregnancy or breast-feeding had infants who showed greater preferences for these smells than infants who had not been exposed to these scents (Mennella and Beauchamp, 1991, 1993; Mennella, Johnson, and Beauchamp, 1995). What we learn about odors prenatally and during infancy and early childhood can also go on to influence food and flavor preferences in adulthood (Haller et al., 1999). This correlation suggests that it might be a good idea for women to eat lots of spinach and liver and other healthy foods during pregnancy if they want their children to accept these dishes later on!

Cross-cultural data provide further support that associative learning, rather than hardwired responses, is responsible for olfactory preferences. No scientific studies to date have found cross-cultural agreement for hedonic responses to common everyday smells, either "good" or "bad" (Ayabe-Kanamura et al., 1998; Schleidt, Hold, and Attila, 1981). And many anecdotal and observational examples illustrate the culturally polarized responses we have to specific odors. For example, Asians consider the smell of cheese to be disgusting, yet Westerners consider it anything from comfort food to an extrav-

**gestation** Fetal development during pregnancy.

**FIGURE 13.16** The Japanese regularly eat *natto* for breakfast whereas Westerners do not equate the smell of this food with eating. In contrast, cheese, which most Westerners enjoy, is considered disgusting by most Japanese eaters.

agant indulgence. In contrast, the Japanese enjoy a meal for breakfast called *natto* (a fermented soybean dish) (Figure 13.16) that most Westerners wouldn't bring near their mouths (a typical American's description of this food's smell is "burning rubber"). Both cheese and natto are high in protein and similarly nutritious; the reason they are preferred, or not, as foods has to do with the associations that we have learned to their scents.

In case you're thinking that the examples of odors given so far aren't really *that* bad, and that there must be consensus on really horrid stenches, this also doesn't seem to be the case. Fecal smells are not high on most North Americans' "best smells list," but the Masai like to dress their hair with cow dung as a cosmetic color treatment. And in a recent study undertaken by the U.S. military to create a stink bomb (to be used for crowd dispersal), it was impossible to find an odor (including "U.S. Army issue latrine scent") that was unanimously considered repellent across ethnic groups (Dilks, Dalton, and Beauchamp, 1999). Recent laboratory studies directly aimed at testing the learning hypothesis have shown that a novel odorant can be made to be perceived as good or bad as a function of the associations (good or bad) made to it (Herz, Beland, and Hellerstein, 2004). Note, however, that from an experimental perspective it is much easer to demonstrate that odor preferences can be learned than to prove that no odors exist to which innate responses may be shown.

## An Evolutionary Argument

The theory that odor preferences are formed through associative learning rather than being innate can be defended on the basis of evolutionary principles (Herz, 2001). *Specialist* animal species live in very specific habitats and thus have a limited number of food sources and predators. For such species, hardwired responses to particular odors are adaptive. Thus, California ground squirrels, for example, exhibit an instinctive defensive response the first time they are exposed to the odor of their natural predator, the Pacific

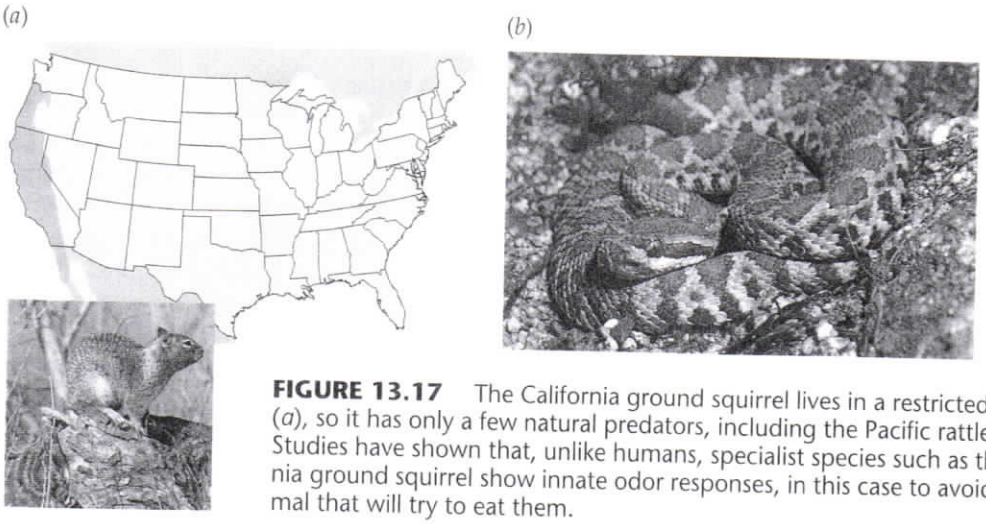



**FIGURE 13.17** The California ground squirrel lives in a restricted habitat (*a*), so it has only a few natural predators, including the Pacific rattlesnake (*b*). Studies have shown that, unlike humans, specialist species such as the California ground squirrel show innate odor responses, in this case to avoid the animal that will try to eat them.

rattlesnake, but they don't show the same response to the scent of Pacific gopher snakes, which are not their natural predators (Figure 13.17) (Coss et al., 1993; Poran and Coss, 1990).

*Generalist* species (including humans, rats, and cockroaches), on the other hand, can exploit many different habitats. Their available resources and potential predators can vary widely across environments, so it is not adaptive for these species to have predetermined olfactory responses to any particular odor. Instead, generalists need to be especially prepared to learn and remember what to approach and what to avoid on the basis of experience.

Clear evidence that learning is a critical mechanism by which generalists acquire odor responses is shown by **learned taste aversions**. Rats and humans can be made to avoid a flavor by being made sick after consumption. See Chapter 14 for an explanation of the critical role of olfaction in flavor perception. For example, presenting a rat with a sweet-tasting banana-smelling drink and then injecting the rat with lithium causes nausea and creates a conditioned avoidance for this flavor in the future. Similarly, children who experienced chemotherapy after ingesting a novel flavor of ice-cream (mapletoff) subsequently showed avoidance to mapletoff ice-cream (Bernstein, 1978). Researchers have shown that in humans the conditioned aversion is to the smell, not the taste, of the substance (Bartoshuk and Wolfe, 1989). In rats, whether there is a discrete role for taste in this type of aversion learning remains unclear. The long-term effects of learned taste aversion for humans are clearly adaptive. If poison is ingested, it is best to learn to avoid it permanently, rather than having to repeat the mistake until it kills you. The key point is that for generalists, banana and mapletoff are not inherently meaningful smells in themselves; rather, it is their association to positive or negative experiences that makes us interpret them as good or bad.

## Caveats

Although overwhelming evidence suggests that odor hedonics are mostly learned, we must note two caveats. First, trigeminally irritating odors may elicit pain responses, and all humans have an innate drive to avoid pain (although even this drive can be overcome by cultural influences, as attested by the popularity of chili peppers in many ethnic cuisines). Second, there is potential variability in the receptor genes and pseudogenes that are expressed

**learned taste aversion** The avoidance of a novel flavor after it has been paired with gastric illness. It is the smell, not the taste, of the substance that is key for the learned aversion response in humans.

**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.

often extremely confident that their recollections are accurate, but research shows that these memories are often incorrect (Busey et al., 2000).

## *Neuroanatomical and Evolutionary Connections between Odor and Emotion*

Neuroanatomy supports the proposition that our olfactory system is especially prepared to learn the affective/emotional significance of odors. The amygdala, which synapses directly with the olfactory nerve, is critical for emotional associative learning (M. Davis and Whalen, 2001). A recent neuroimaging study showed that when participants recalled a significant personal memory connected to the smell of a specific perfume, the amygdala was significantly more activated than when they recalled the same memory connected to the sight of the perfume bottle, or when they smelled or saw a nonmeaningful perfume (Herz et al., 2003).

The **orbitofrontal cortex**, where olfaction is processed, is also the cortical area responsible for assigning affective value—that is, our hedonic judgments to a wide range of stimuli (Davidson, Putnam, and Larson, 2000). Furthermore, the most ancient part of the brain, the rhinencephalon—literally, the "nose-brain"—which comprises the olfactory cortex and limbic areas, developed first from olfactory structures. Only later in evolution did limbic structures such as the amygdala develop. It is interesting to consider that our hedonic and emotional reactions to stimuli in general may have their origin in our sense of smell.

Almost all species of animals use smell or chemical communication for the most basic behaviors necessary for survival: recognizing kin, finding reproductively available mates, locating food, and determining whether an animal or object is dangerous. (See Web Essay 13.3 The Vomeronasal Organ and Pheromones.) It is only in humans that visual and auditory information have mostly replaced smell for imparting this kind of crucial knowledge about the world. Yet our olfactory system has retained some of its basic functions. The most immediate responses we have to odors are simple binary opposites: like or dislike, approach or avoid. Emotions convey similar messages: approach what is good, joyful, loving, and avoid what is bad, fearsome, or liable to cause grief. Thus, emotions and olfaction are functionally analogous. Both enable the organism to react appropriately to its environment, maximizing its chances for basic survival and reproductive success. Viewed in this context, the human emotional system can be seen as a highly evolved, abstract cognitive version of the basic behavioral motivations instigated by the olfactory system in animals (Herz, 2000, 2004).

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

## Summary

1. Olfaction is one of the two chemical senses. The other is taste (see Chapter 14). To be perceived as scent, a chemical must possess certain physical properties; however, even some molecules that possess these characteristics cannot be smelled. Compared to our other senses, olfaction has a number of unique physiological properties. Among the unique features is the fact that only approximately 35% of the genes that code for olfactory receptors in humans are functional. Another unusual feature is that most smells also stimulate the somatosensory system via the trigeminal nerve, and it is often impossible to distinguish the contribution of olfactory sensation from trigeminal stimulation.

2. The dominant biochemical theory of odor perception contends that the fit between a molecule and an olfactory receptor (OR) determines what molecules are detected as scents, and that specific odorant molecules activate arrays of ORs, producing specific patterns of neural activation for each perceived scent (shape-pattern theory). However, this theory is not universally accepted, and alternate explanations exist (e.g., vibration theory).
3. Almost all odors we encounter in the real world are mixtures, and it seems that we are not very good at analyzing the discrete chemical components of a scent mixture. Olfaction is thus primarily a synthetic, as opposed to analytical, sense. However, analytical ability can be developed with training. True odor imagery is also weak (or nonexistent) for most people; however, training may also facilitate this ability.
4. The psychophysical study of smell has shown that different odorant intensity levels and different cognitive functions are required for odor detection, discrimination, and recognition. Identification differs from odor recognition in that, in the former, one must come up with a name for the olfactory sensation. It turns out to be very difficult for us to name even very familiar odors—a phenomenon known as tip-of-the-nose—which illustrates that linguistic processing is highly disconnected from olfactory experience.
5. Another important discrepancy between the physical and psychological experience of odors is receptor adaptation and cognitive habituation. Receptor adaptation occurs after continuous odorant exposure over a number of minutes, can be undone after a few minutes away from the odorant, and is explained by a basic biochemical mechanism. By contrast, cognitive habituation occurs after long-term exposure (e.g., in a living or work environment) to a particular odorant and takes weeks away from the odorant to undo. At present it is not fully understood what mechanisms are responsible for the cognitive–habituation effect.
6. The most immediate and basic response we have to an odor is whether we like it or not; this is hedonic evaluation. Odor hedonics are measured by pleasantness, familiarity, and intensity ratings. Pleasantness and familiarity are linearly related to odor liking; odor intensity has a more complex relationship to hedonic perception. There is substantial evidence that our hedonic responses to odors are learned and not innate, even for so-called stenches. That we have learned to like or dislike various odors rather than being born with hardwired responses is evolutionarily adaptive for generalist species like humans. The caveats to the learned proposition are odors that are highly trigeminally irritating and the potential for individual genetic differences in OR expression to alter odor sensitivity and hence the perception of odor intensity.
7. The key to olfactory associative learning is the emotional value of the context in which the odor is first encountered. If the emotional context is good, the odor will be liked; if it is bad, the odor will be disliked. Emotional associations to odors can also elicit full-blown episodic memories. Odor-evoked memories are distinguished from memories triggered by other sensory cues by their emotional potency. The neuroanatomy of the olfactory and limbic systems and their neuroevolutionary development illustrate how emotional processing and olfactory processing are uniquely and intimately interrelated.