Emotion enriches our lives with, as the writer Virginia Woolf put it, its "buzzing, humming, soaring, and roaring." It also motivates our behavior: Anger intensifies our defensive behavior, fear accelerates flight, and happiness encourages the behaviors that produce it. Emotion adds emphasis to experiences as they are processed in the brain, making them more memorable (A. K. Anderson & Phelps, 2001); as a result, we are likely to repeat the behaviors that bring joy and avoid the ones that produce danger or pain. Although Jane was intelligent, her injury left her unable to learn from her emotional experiences. According to Antonio Damasio (1994), reason without emotion is inadequate for making the decisions that guide our lives and, in fact, make up our lives.

Emotion and the Nervous System

If asked what *emotion* means, you would probably think first of what we call "feelings"—the sense of happiness or excitement or fear or sadness. Then you might think of the facial expressions that go along with these feelings: the curled-up corners of the mouth during a smile, the knit brow and red face of anger. Next you would probably visualize the person acting out the emotion by fleeing, striking, embracing, and so on. Emotion is all these and more; a working definition might be that *emotion* is an increase or a decrease in physiological activity that is accompanied by feelings that are characteristic of the emotion and often accompanied by a characteristic behavior or facial expression. Having said that mouthful, we suspect you will understand why Joseph LeDoux (1996) wrote that we all know what emotion is until we attempt to define it. We will talk about these different facets of emotion in the following pages, along with some practical implications in the form of aggression and health.

Autonomic and Muscular Involvement in Emotion

To the neuroscientist, the most obvious component of emotional response is sympathetic nervous system activation. You may remember from Chapter 3 that the sympathetic system activates the body during arousal; it increases heart rate and respiration rate, increases sweat gland activity, shuts down digestion, and constricts the peripheral blood vessels, which raises the blood pressure and diverts blood to the muscles. As you will see in the section on stress, the sympathetic system also stimulates the adrenal glands to release various hormones, particularly *cortisol*. At the end of arousal, the parasympathetic system puts the brakes on most bodily activity, with the exception that it activates digestion. In other words, the sympathetic nervous system prepares the body for "fight or flight"; in contrast, the parasympathetic system generally reduces activity and conserves and restores energy (Figure 8.1).

? What effect does the autonomic nervous system have during emotions?

Of course, muscular activation is involved in the external expression of emotion, such as smiling or fleeing or attacking. It is also a part of the less obvious responses of emotion, such as the bodily tension that not only prepares us to act but also produces a headache and aching muscles when we try to write a paper the night before it is due. Autonomic and muscular arousal are adaptive, because they prepare the body for an emergency and help it carry out an appropriate response. They are also an important part of the emotion itself, though the exact nature of their contribution has been the subject of controversy. Fortunately, as you will see from the following discussion, competing theories are one of the engines driving research and scientific advancement.

? How do the James-Lange and cognitive theories disagree? What evidence is there for each?

The Role of Feedback From the Body

A bit over a century ago, the American psychologist William James (1893) and Danish physiologist Carl Lange (1885/2010) independently proposed what has come to be known as the *James-Lange theory:* Emotional experience results from the physiological arousal that precedes it, and different emotions are the result of different patterns of arousal. In our discussion of research ethics in Chapter 4, we talked about an experiment by Albert Ax (1953) in which subjects either were made angry by an insulting experimenter or were frightened by the possibility of a dangerous electric shock. Consistent with the James-Lange theory, the two emotions were accompanied by different patterns of physiological activity. Seventy years after James and Lange, Stanley Schacter and Jerome Singer (1962) took a contrary position in their *cognitive theory;* they stated that the identity of the emotion is based on the cognitive assessment of the situation, and physiological arousal contributes only to the emotion's intensity. Their research demonstrated how easily people could misidentify emotions depending on the environmental context. For example, young men who were interviewed by an attractive woman while crossing a swaying footbridge 230 feet above a rocky river included

> "We feel sorry because we cry, angry because we strike, afraid because we tremble."
>
> —William James, 1893

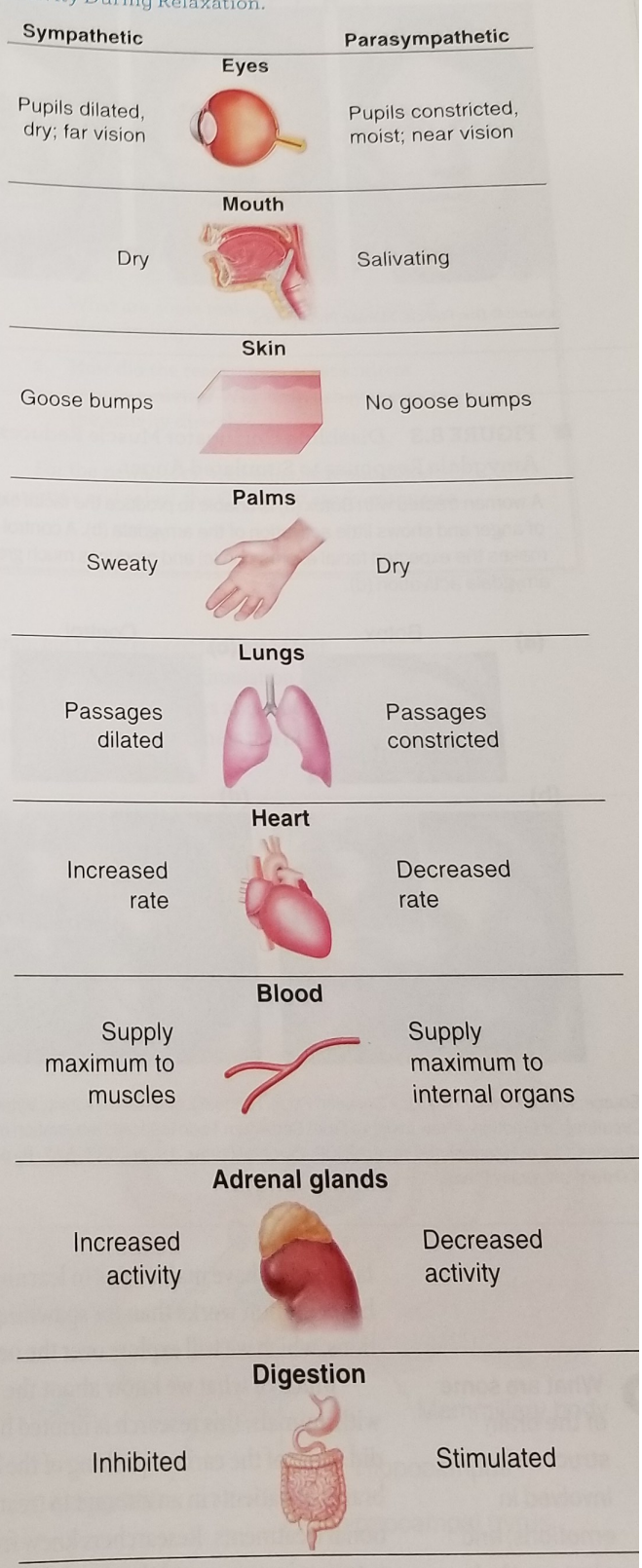
more sexual content in brief stories they wrote later and were more likely to call the phone number the young woman gave them than were men who were interviewed 10 minutes after crossing the bridge (D. G. Dutton & Aron, 1974).

Studies like these have not determined that one theory is right and the other is wrong. Barlassina and Newen (2013) argue that neither is adequate; in their *integrative embodiment theory of emotions* they maintain that bodily sensations are a critical component of emotions, but these perceptions must be integrated with cognitive information. Nevertheless, incorrect theories can be useful if they generate research, and the competition between these two has produced valuable insights into emotion. A good example is the contribution of facial expressions to emotional experience. Experimenters have had to be very inventive in doing facial expression studies; obviously, they can't just tell people to smile or frown and then ask them what emotion they're feeling. Paul Ekman and his colleagues (Levenson, Ekman, & Friesen, 1990) instructed subjects to contract specific facial muscles to produce different expressions (Figure 8.2); for example, to produce an angry expression, subjects were told to pull their eyebrows down and together, raise the upper eyelid, and push the lower lip up with the lips pressed together (p. 365). The posed facial expressions for happiness, fear, anger, disgust, sadness, and surprise each resulted in the experience of the intended emotion, along with a distinct pattern of physiological arousal.

Induced facial poses also influence how the person interprets the environment. Volunteers rate a stimulus as more painful when they are making a sad face than a happy or neutral one (Salomons, Coan, Hunt, Backonja, & Davidson, 2008), and college students rate *Far Side* cartoons as more amusing when they are holding a pen between the teeth, which induces a sort of smile, than when they hold the pen between the lips, producing a frown (Strack, Martin, & Stepper, 1988). More strikingly, women who have had their corrugator muscles paralyzed by injecting botulinum toxin (Botox) to remove frown lines are unable to frown, and they report less negative mood—even if they don't perceive themselves as more attractive (M. B. Lewis & Bowler, 2009). In addition, when these women attempt to imitate angry expressions, they produce less activation of the amygdala than women who have not had Botox treatment (Figure 8.3; Hennenlotter et al., 2009).

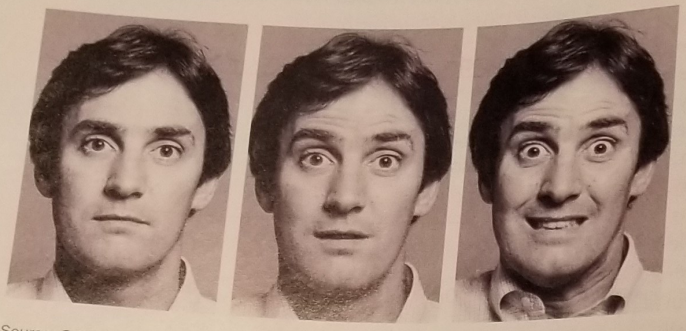
Some researchers suggest that feedback from emotional expressions has another role besides contributing to our emotional experience in that it also helps us understand other people's emotions; this ability is critical to social communication and to societal success. This view is supported by studies of mirror neurons. *Mirror neurons* are neurons that respond both when we engage in a specific act and while observing the same act in others. They were first discovered when researchers noticed that neurons that were active while monkeys reached for food also responded when the monkeys saw the researcher picking up a piece of food (di Pellegrino, Fadiga, Fogassi, Gallese, & Rizzolatti, 1992); similar correlations have

FIGURE 8.1 Comparison of Sympathetic Activity During Emotional Arousal With Parasympathetic Activity During Relaxation.



Source: Created for this book by Epicstudios, Inc.

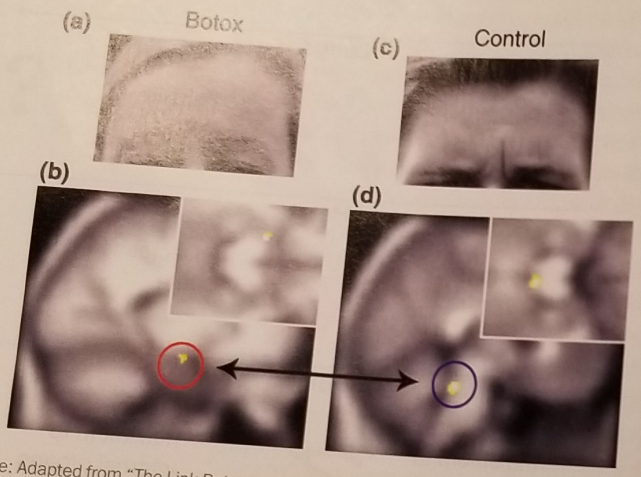
FIGURE 8.2 Emotional Expressions Posed Using Ekman's Instructions.



Source: © Don Francis/Mardan Photography.

FIGURE 8.3 Disabling Corrugator Muscle Reduces Amygdala Response to Simulated Anger.

A woman treated with Botox (a) is unable to produce the facial expression of anger and shows little activation of the amygdala (b). A control subject makes the expected facial expression (c) and produces much greater amygdala activation (d).



Source: Adapted from "The Link Between Facial Feedback and Neural Activity Within Central Circuitries of Emotion—New Insights From Botulinum Toxin-Induced Denervation of Frown Muscles," by A. Hennenlotter et al., 2009, *Cerebral Cortex, 19*, pp. 537–542. By permission of Oxford University Press.

been observed in other brain areas, including those involved in emotions (see review in Bastiaansen, Thioux, & Keysers, 2009). Observing another person's emotional expressions activates emotional areas in our own brains, and the amount of activity is related to scores on a measure of empathy (Chakrabarti, Bullmore, & Baron-Cohen, 2006). The accompanying In the News feature describes research showing that our socioeconomic class affects our empathic ability.

Our observation of other people's emotions is not entirely passive; we also mimic their gestures, body posture, tone of voice, and (often imperceptibly) facial expressions (Bastiaansen et al., 2009). Just as feedback during our own emotional activity adds to our own emotional experience, feedback from imitated expressions may help us empathize with the emotions of others. Indeed, interfering with facial mimicry by engaging the required muscles in other activities (such as chewing gum) impairs subjects' ability to recognize happiness and disgust in photos (Oberman, Winkielman, & Ramachandran, 2007). Women who have Botox treatments also rate facial photos and sentences as less emotional than before treatment (Baumeister, Papa, & Foroni, 2016). People with autism typically have trouble understanding other people's emotions, perhaps in part because their imitation of emotional expressions is delayed by about 160 milliseconds (Oberman, Winkielman, & Ramachandran, 2009).

A system this complex requires an equally complex control system. We will turn our attention now to the brain structures responsible for emotion.

The Emotional Brain

In the late 1930s and 1940s, researchers proposed that emotions originated in the *limbic system*, a network of structures arranged around the upper brain stem (Figure 8.4). As complex as this system is with its looping interconnections, we now know that this hypothesis is an oversimplification; emotion involves structures at all levels of the brain, from the prefrontal area to the brain stem (A. R. Damasio et al., 2000). Also, we know that some of the limbic structures are more involved in non-emotional functions. For example, the hippocampus and mammillary bodies have major roles in learning. The concept of a limbic system is less important as a description of how emotion works than for spawning a tremendous volume of research that has taken us in diverse directions, which we will explore over the next several pages.

? What are some of the brain structures involved in emotions, and what are their functions?

Much of what we know about the brain's role in emotion comes from lesioning and stimulation studies with animals; this research is limited because we do not know what the animal is experiencing. Robert Heath did some of the earliest probing of the limbic system in humans in 1964 when he implanted electrodes in the brains of patients in an attempt to treat epilepsy, sleep disorders, or pain that had failed to respond to conventional treatments. Researchers knew from animal studies that the hypothalamus has primary control over the autonomic system and that it produces a variety of emotional expressions, such as the threatened cat's hissing and bared teeth and claws. Stimulation of the hypothalamus in Heath's patients produced general autonomic

IN THE NEWS

EMOTIONAL PROCESSING DIFFERS BY CLASS

Recent work has confirmed our intuition that others perceive the world in different ways from us. For example, people of higher socioeconomic status (SES) pay less attention to other people and people's faces and show less interest in them than do members of lower classes (Baer, 2017; Dietze & Knowles, 2016). The neural counterpart of these behavioral differences is a difference in mirror neuron activity in response to others' behavior. Researchers at Arizona State University used mu wave suppression—a decrease in 8–13 Hz EEG activity—over the motor cortex as a measure of mirror neuron activation. In their study, lower SES participants responded with more mirror neuron activity to videos of hand movements of others than did the higher SES participants (Varnum, Blais, & Brewer, 2016). According to the authors, their results are consistent with previous studies indicating that lower SES individuals are more empathetic toward others.

THOUGHT QUESTIONS

1. What are some real-world implications of these findings?
2. How did the researchers assess mirror neuron activity? Why didn't they measure the activity directly?

For the news story, visit edge.sagepub.com/garrett5e and select the Chapter 7 study resources.

discharge and sensations such as a pounding heart and feelings of warmth, but it also evoked feelings of fear, rage, or pleasure, depending on the location of the electrode in the hypothalamus. Septal area stimulation also produced a sense of pleasure, but in this case the feeling was accompanied by sexual fantasies and arousal. During septal stimulation one patient went from near tears while talking about his father's illness to a broad smile as he described how he planned to take his girlfriend out and seduce her. When asked why he changed the subject, he replied that the thought just came into his head.

Now researchers are more likely to use one of the scanning techniques to study the brain centers of emotion. Typically, they do MRI scans to determine the location of damage in patients with emotional deficits, or they use positron emission tomography (PET) or functional magnetic resonance imaging (fMRI) while healthy subjects relive an emotional experience, examine facial expressions of emotion, or view an emotional video. Two of the most reliable brain-emotion associations have been the amygdala's role in fear and the location of disgust in the insular cortex and the basal ganglia (Figure 8.5; F. C. Murphy, Nimmo-Smith, & Lawrence, 2003; Phan, Wager, Taylor, & Liberzon, 2002). We will consider the amygdala in some detail later. The insula is the area we identified in Chapter 6 as the cortical projection site for taste; a number of writers have remarked on the fact that taste and disgust share the same brain area and that *dis-gust* means, roughly, "bad taste." In Chapter 3, we identified the basal ganglia as being

FIGURE 8.4 Structures of the Limbic System.

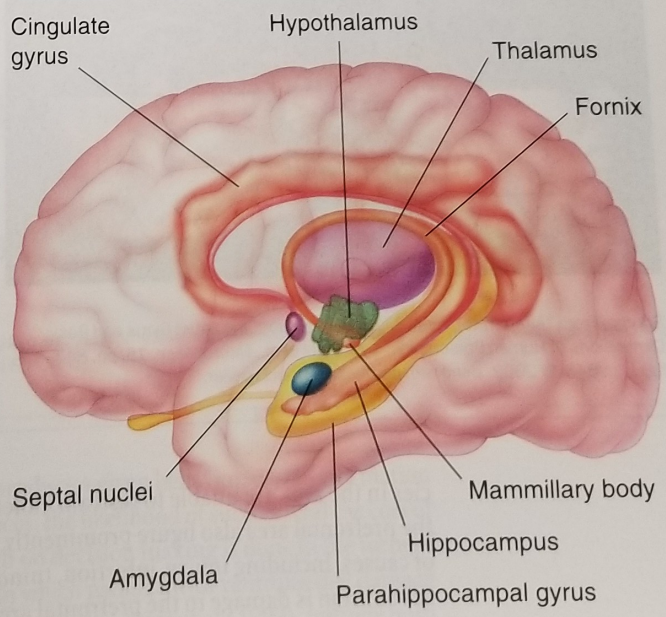
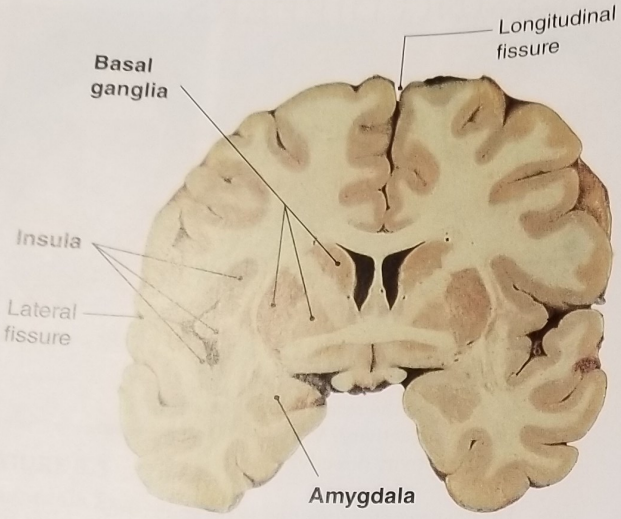


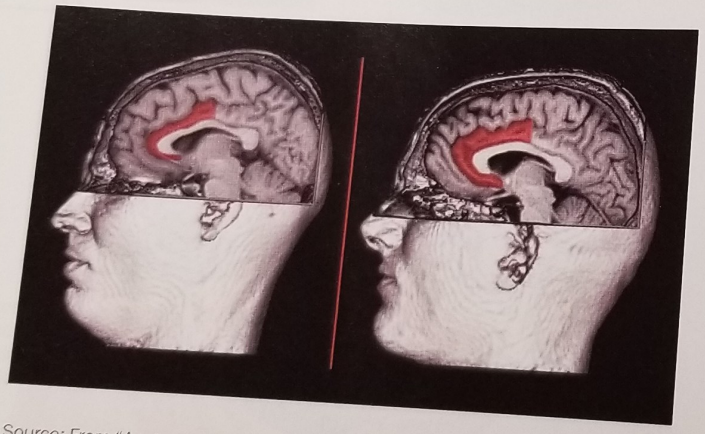
FIGURE 8.5 Location of the Amygdala, Insula, and Basal Ganglia.



Source: Photo courtesy of Dana Copeland.

FIGURE 8.6 Size Differences in the Anterior Cingulate Gyrus.

A larger anterior cingulate gyrus (highlighted in red) is associated with a higher level of the personality characteristic of harm avoidance.



Source: From "Anatomical Variability of the Anterior Cingulated Gyrus and Basic Dimensions of Human Personality," by J. Pujol et al., *Neuroimage, 15*, pp. 847–855, fig. 1, p. 848. © 2002. Used with permission from Elsevier.

involved in motor functions. Interestingly, people with Huntington's disease or obsessive-compulsive disorder, both of which involve abnormalities in the basal ganglia, have trouble recognizing facial expressions of disgust (Phan et al., 2002).

Another significant structure in emotion is the *anterior cingulate cortex*, a part of the cingulate gyrus that is important in attention, cognitive processing, emotion, and possibly consciousness. You can see the cingulate gyrus in Figure 8.4 and the anterior cingulate gyrus in Figure 8.6. The anterior cingulate cortex is believed to combine emotional, attentional, and bodily information to bring about conscious emotional experience (Dalgleish, 2004). Consequently, it is involved in emotional activity regardless of which emotion is being experienced, although some studies have also linked parts of the structure to specific emotions, such as sadness and happiness (F. C. Murphy et al., 2003; Phan et al., 2002). Interestingly, an MRI investigation found that the right anterior cingulate was larger in people with high scores on *harm avoidance*, which involves worry about possible problems, fearfulness in the face of uncertainty, and shyness with strangers (Pujol et al., 2002).

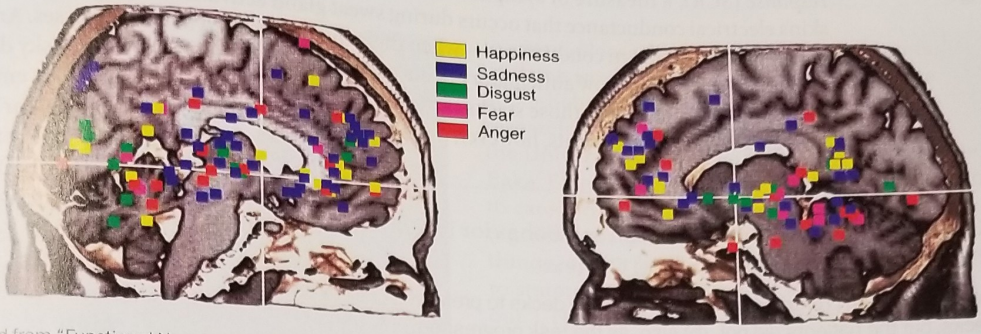
Before we go too far in assigning emotions to specific brain structures, we need to understand that any specific emotion involves activity in a network that includes many brain areas. This is well illustrated in a study that combined the results of 55 PET and fMRI investigations (Phan et al., 2002). As you can see in Figure 8.7, places activated during a specific emotion cluster somewhat in particular areas, but they are also scattered across wide areas of the brain. This is partly due to different methods of inducing the emotions in the studies, but it also reflects the complexity of emotion. With the understanding that no emotion can be relegated to a single part of the brain, we will look more closely at three areas that have particularly important roles in emotional experience and behavior: the prefrontal cortex, the amygdala, and the right hemisphere.

The Prefrontal Cortex

The prefrontal cortex (see Figure 3.8 for location) is the final destination for much of the brain's information about emotion before action is taken. You saw in Chapter 3 that damage to the prefrontal area or severing its connections with the rest of the brain impairs people's ability to make rational judgments. Later in this chapter you will learn that people with deficiencies in the area are unable to restrain violent urges, and in Chapter 14 you will see that abnormalities in the prefrontal area also figure prominently in depression and schizophrenia. These deficits have a variety of causes, including injury, infection, tumors, strokes, and developmental errors. What the victims have in common is damage to the prefrontal area that includes the ventromedial cortex and the orbitofrontal cortex (see Figure 8.8).

FIGURE 8.7 Brain Areas Activated During Different Emotions.

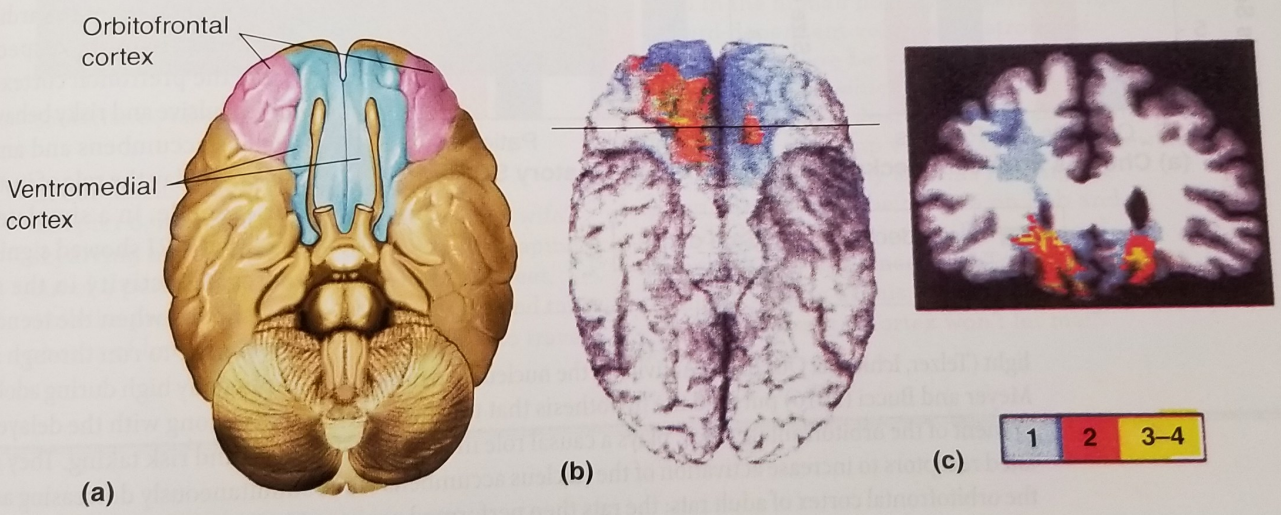
Each colored square represents the location identified in a single study for a particular emotion. In spite of the clustering of control of each emotion in a general area, the scattered dots of the same color indicate that each emotion relies on activity in multiple areas.



Source: Reprinted from "Functional Neuroanatomy of Emotion: A Meta-Analysis of Emotion Activation Studies in PET and fMRI," by K. L. Phan, T. Wager, S. F. Taylor, and I. Liberzon, *Neuroimage, 16*, pp. 331–348. Copyright 2002, with permission from Elsevier.

FIGURE 8.8 Location of Damage That Impairs Emotion-Based Decision Making.

In (a) the location of the ventromedial cortex and the orbitofrontal cortex is shown. In (b) you can see where damage occurred in four patients who showed judgment problems. The horizontal line shows where the scan in (c) was taken. In (b) and (c), the different colors indicate the number of patients with damage in the area, according to the code on the color bar. All shared damage in the ventromedial cortex, but some had damage in the orbitofrontal cortex as well.



Sources: (b) and (c) from "Different Contributions of the Human Amygdala and Ventromedial Prefrontal Cortex to Decision-Making," by A. Bechara, H. Damasio A. R. Damasio, & G. P. Lee, 1999, *Journal of Neuroscience, 19*, pp. 5473–5481.

The prefrontal cortex makes the final decision whether to produce a given behavior, using information supplied by other brain areas about the relative value of the choices, the likelihood of success, and the cost of failure. From this description it is easy to see why most research on decision making is done in the form of studies of risk taking. A good example is a gambling task study in which patients with ventromedial damage and healthy volunteers chose cards from four decks to win play money (Bechara et al., 1999). Initially both groups chose more often from the two "risky" decks, which usually resulted in large rewards but occasionally

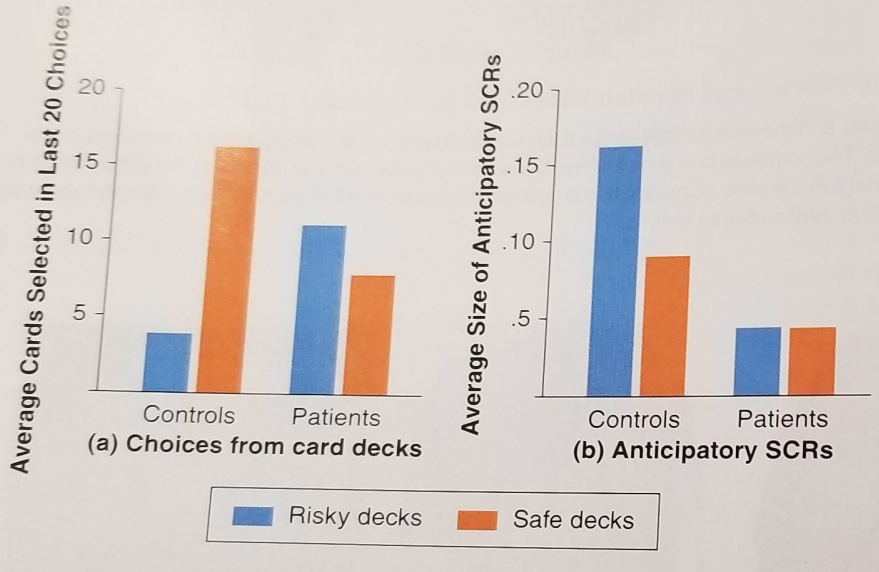
? How does loss of emotion impair "rational" decision making?

led to large penalties, for an overall loss. Over time, the control subjects shifted to the two "safe" decks, whose cards produced lower rewards and smaller penalties for an overall gain; the patients typically did not make the shift, however, even after they figured out how the game worked (Figure 8.9a).

To assess the subjects' emotional responses during the task, the researchers used the *skin conductance response (SCR)*, a measure of sympathetic nervous system activity obtained by recording changes in the skin's electrical conductance that occurs during sweat gland activation. Both groups showed emotional reactions—increases in skin conductance—when their choices resulted in wins or losses. And over time, the control subjects began to show anticipatory SCRs just before drawing a card from a risky deck, even before they were able to verbalize that those stacks were risky. (This is the example of unaware emotional influence we promised earlier.) The patients, however, did not produce anticipatory SCRs to the four decks; although their bad choices were eliciting emotional responses, their prefrontal damage made them unable to process the consequences of risky behavior and use that information to guide their choices (Figure 8.9b).

FIGURE 8.9 Comparison of Gambling Task Behavior in Controls and Patients With Damage to the Prefrontal Cortex.

The controls shifted from preferring cards from the risky decks to preferring cards from the safe decks, but the patients did not. (b) Also, as the task progressed, only the controls showed anticipatory skin conductance responses (SCR) before choosing from the risky decks.



More recently, studies of risk taking have mapped out some of the circuitry involved in decision making. Functional MRIs showed that teenagers identified as risk takers had stronger functional connectivity of the right prefrontal cortex with the nucleus accumbens and the amygdala than did their non-risk-taking age-mates (DeWitt, Aslan, & Filbey, 2014). The nucleus accumbens and amygdala, of course, are involved in the evaluation of rewarding and emotional stimuli, respectively. While the prefrontal cortex dampens impulsive and risky behavior, the nucleus accumbens and amygdala appear to play the role of instigators. For example, in a simulated driving task fMRI showed significantly increased activity in the nucleus accumbens when the teenage subjects chose to run through a yellow light (Telzer, Ichien, & Qu, 2015). Activity in the nucleus accumbens is exceptionally high during adolescence. Meyer and Bucci (2016) pursued the hypothesis that this increased activity, along with the delayed development of the orbitofrontal cortex, plays a causal role in teen impulsiveness and risk taking. They manipulated receptors to increase activation of the nucleus accumbens while simultaneously decreasing activity in the orbitofrontal cortex of adult rats; the rats then performed comparably to adolescent rats, taking twice as long to learn to withhold a conditioned response when a stimulus indicated that it would not be followed by a reward.

Studies have also identified another part of the brain that participates in risk evaluation, the posterior parietal cortex (PPC). Patients with damage to this area had difficulty adjusting their betting according to the chances of winning; they often made large bets when the odds were low and small bets when the chances of winning were strongly favorable (Studer, Manes, Humphreys, Robbins, & Clark, 2015). In two additional studies, willingness to make risky choices was correlated with the amount of gray matter volume in the PPC (Gilaie-Dolan et al., 2014), and across subjects aged 18 to 88, risk taking and PPC gray matter volume declined together (Grubb, Tymula, Gilaie-Dolan, Glimcher, & Levy, 2016).

In the rest of us, who like to think of ourselves as normal, the prefrontal cortex's connections to other parts of the brain determine whether we are novelty-seeking adventurers or are more restrained, as the accompanying Application explains.

APPLICATION

Why Bob Doesn't Jump Out of Airplanes

Source: iStock/vuk8691.

In our discussion of sensation seeking in Chapter 6, we mentioned the first author's wife's skydiving adventure. Why did she enjoy leaping out of a perfectly good airplane at 14,000 feet, while he preferred to stay on the ground and take photos of her descent? Why did she like to travel to exotic places, like Thailand and Patagonia, while he would rather stay at home and write about behavioral neuroscience? Once again, research comes to the rescue. Psychologists at the University of Arizona (M. X. Cohen, Schoene-Bake, Elger, & Weber, 2009) used a questionnaire to categorize 20 volunteers as novelty seekers (agreeing to statements like "I like to try new things just for fun") or reward dependent (agreeing to statements like "I'd rather stay home than go out"). Novelty seekers are higher in exploratory drive and impulsivity, whereas those who are reward dependent are particularly sensitive to rewards and spend a lot of time pursuing activities that have been rewarding in the past.

Next, the researchers used diffusion tensor imaging to measure the density of the subjects' white-matter tracts. Their analysis focused on the hippocampus, amygdala, and striatum, because animal research has indicated that these structures form a looping circuit that is important in novelty seeking. There were strong white-matter connections within this loop in the human novelty seekers, but the reward-dependent volunteers' strongest connections were between the striatum and prefrontal areas, which tend to put the brakes on risky behavior. Just before the skydiving experience, the jump videographer interviewed Bob's wife about why she was making the jump, then turned the camera on Bob and asked why he chose not to. He answered lamely, "I just don't need to." Today, armed with the results of this study, his answer would be, "My prefrontal cortex won't let me!"

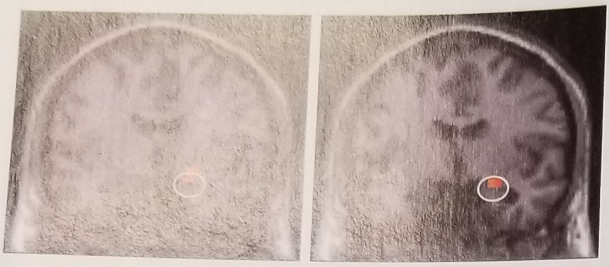
The Amygdala

The prefrontal areas receive much of their emotional input from the amygdala (see Figure 8.4 again), a small limbic system structure in each temporal lobe that is involved in emotions, especially negative ones. The amygdala has other functions as well. In Chapter 7 you learned that the amygdala responds to sexually exciting stimuli, and in Chapter 12 you will see that it participates in memory formation, especially when emotion is involved. Some amygdala neurons fire when the individual judges the facial expression in a photo to be fearful, and others respond to happy faces (S. Wang et al., 2014). So the amygdala's role may involve responding to emotionally significant stimuli in general (Phan et al., 2002).

Although the amygdala participates in other emotions, its role in fear and anxiety has been researched the most thoroughly. *Fear* is an emotional reaction to a specific immediate threat; *anxiety* is an apprehension about a future, and often uncertain, event. Stimulating the amygdala produces fear in human subjects

FIGURE 8.10 Activity in the Right Amygdala While Viewing Facial Expressions of Fear.

The individual on the left has the short allele of the *SLC6A4* gene, which reduces serotonin activity and increases fear responsiveness; the one on the right has two copies of the long allele.



Source: From "Serotonin Transporter Genetic Variation and the Response of the Human Amygdala," by Hariri et al., *Science, 297*, pp. 400–403. Copyright 2002. Reprinted by permission of AAAS.

(Gloor, Olivier, Quesney, Andermann, & Horowitz, 1982). The amygdala contains receptors for benzodiazepines, which are used to treat anxiety, and injection of this type of drug directly into the amygdala reduces signs of both fear and anxiety in animals (M. Davis, 1992). A person with one version of the *SLC6A4* gene has decreased serotonin activity; as a result, the person is prone to fear and anxiety and, as Figure 8.10 shows, the amygdala is hyperreactive to fear stimuli (Hariri et al., 2002).

Rats with amygdala damage are so fearless they will not only approach a sedated cat but also climb all over its back and head (D. C. Blanchard & Blanchard, 1972). One rat even nibbled on the stuporous cat's ear, provoking an attack—and after the attack ended, the rat climbed right back onto the cat. A few humans have sustained damage to both amygdala structures, usually as a result of infection or disease, and they also show a variety of deficits; like the rats, for example, they are unusually trusting of strangers (Adolphs, Tranel, & Damasio, 1998). Bechara's study of prefrontal patients included a group of patients with bilateral amygdala damage (Bechara et al., 1999). The two groups performed similarly in most ways, with one notable exception: While neither group produced anticipatory SCRs when choosing from the risky decks, the amygdala patients also didn't respond to monetary gains and losses. Apparently the ventromedial patients were unable to make use of emotional information from the amygdala, but the amygdala patients couldn't even generate an emotional response to rewards and punishments. As a result, patients with bilateral amygdala damage often live in supervised care, because their actions can easily bring harm to themselves and others. Ventromedial patients are less impaired, which suggests that the amygdala sends its information to additional decision-making areas, suggesting a broad scope to this network.

SM is one of the best characterized patients with bilateral amygdala damage (Figure 8.11); she reports very little feeling of fear and—in spite of having been held up at knifepoint, nearly killed in an act of domestic violence, and threatened with death on other occasions—her behavior never reflected any sense of desperation or urgency (Feinstein, Adolphs, Damasio, & Tranel, 2011). Researchers were unable to find any stimulus that could evoke fear in her; she was undisturbed by horror movies or a haunted house that produced screams in her companions, and she showed an unusual compulsion to touch snakes she had been told were deadly. Interestingly, during a test that involved inhaling carbon dioxide, which produces a feeling of suffocation, she and a similar patient experienced full-blown panic attacks (Feinstein et al., 2013). The researchers' interpretation was that the amygdala monitors external threats from the environment, and that fear triggered internally—in this case by the sense of suffocation—has another neural basis.

Hemispheric Specialization in Emotion

The specialization of the cerebral hemispheres we have seen in other functions is also evident in emotion. Although both hemispheres are involved in the *experience* of emotions, the left frontal area is more active when the person is experiencing positive emotions, and the right frontal area is more active during negative emotion (R. J. Davidson, 1992). This is consistent with a study done with epilepsy patients who had numerous electrodes placed in their brains as part of a presurgical evaluation. Stimulation in the right amygdala evoked negative emotions (identified by the conscious patients as fear, anxiety, or sadness) at 100% of the locations; in the left amygdala the locations were split almost evenly between negative emotions and positive ones (joy and happiness; Lanteaume et al., 2007). People with damage to the left hemisphere often express more anxiety and sadness about their situation, whereas those with right-hemisphere damage are more likely to be unperturbed or even euphoric, even when dealing with an associated paralysis of an arm or a leg (Gainotti, 1972; Gainotti, Caltagirone, & Zoccolotti, 1993; W. Heller, Miller, & Nitschke, 1998). The same difference in emotions occurs when each of the cerebral hemispheres is anesthetized briefly in turn by injecting a short-acting barbiturate into the right or left carotid artery (Rossi & Rosadini, 1967). (This technique is sometimes used in evaluating patients prior to brain surgery.) In fact, when the right hemisphere is anesthetized, individuals can describe negative events in their lives but can barely recall having felt sad or angry or

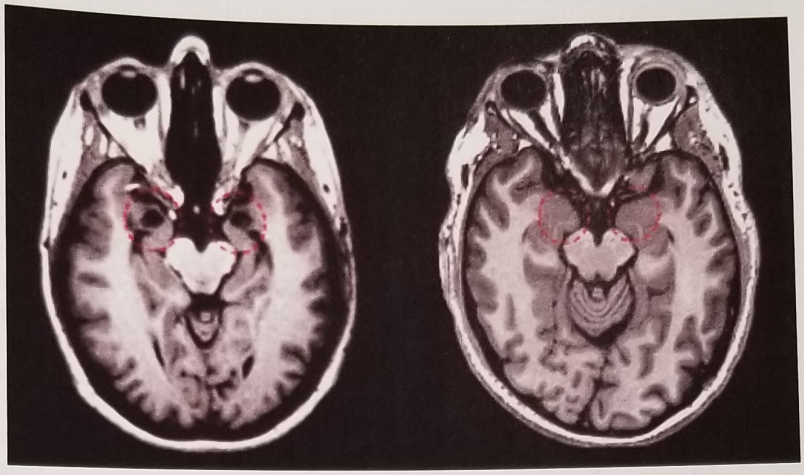
fearful, even with incidents as intense as their mother's death, the discovery of a spouse's affair, or the wife's threatening to kill the individual (E. D. Ross, Homan, & Buck, 1994).

Although both hemispheres are involved in experiencing emotion, the right is more specialized for its expression (Heller et al., 1998). Autonomic responses to emotional stimuli such as facial expressions and emotional scenes are greater when the stimuli are presented to the right hemisphere (using the strategy described by Spence, Shapiro, & Zaidel, 1996, p. 298). Much of the emotional suppression in patients with right-hemisphere damage is due to decreased autonomic response (Gainotti et al., 1993).

Perception of nonverbal aspects of emotion is impaired in patients with right-hemisphere damage; for example, they often have difficulty recognizing emotion in others' facial expressions (Adolphs, Damasio, Tranel, & Damasio, 1996). Verbal aspects are unimpaired, however; the same patients can understand the emotion in a verbal description like "Your team's ball went through the hoop with one second left to go in the game," but they have trouble identifying the emotion in descriptions of facial or gestural expressions such as "Tears fell from her eyes" or "He shook his fist" (Blonder, Bowers, & Heilman, 1991). Patients with right-hemisphere damage also have trouble recognizing emotion from the tone of the speaker's voice (Gorelick & Ross, 1987), and their own speech is usually emotionless as well (Heilman, Watson, & Bowers, 1983). When asked to say a neutral sentence like "The boy went to the store" in a happy, sad, or angry tone, they speak instead in a monotone and often add the designated emotion to the sentence verbally, for example, "... and he was sad."

FIGURE 8.11 SM's Brain, Compared With a Normal Brain.

In SM's brain (left), you see only two dark voids (in the red circles) where each amygdala was before disease caused their deterioration. The brain on the right is normal, for comparison.



Source: From "Human Brain Is Divided on Fear and Panic: New Study Contends Different Areas of Brain Responsible for External Versus Internal Threats," by John Riehl, April 2, 2013, *Iowa Now*, retrieved from http://now.uiowa.edu/2013/01/human-brain-divided-fear-and-panic.

CONCEPT CHECK

Take a Minute to Check Your Knowledge and Understanding

- Describe the role of the autonomic nervous system in emotion (including the possible identification of emotions).
- Organize your knowledge: List the major parts of the brain described in this section that are involved in emotion, along with their functions.
- How are the effects of prefrontal and amygdala damage alike, and how are they different?

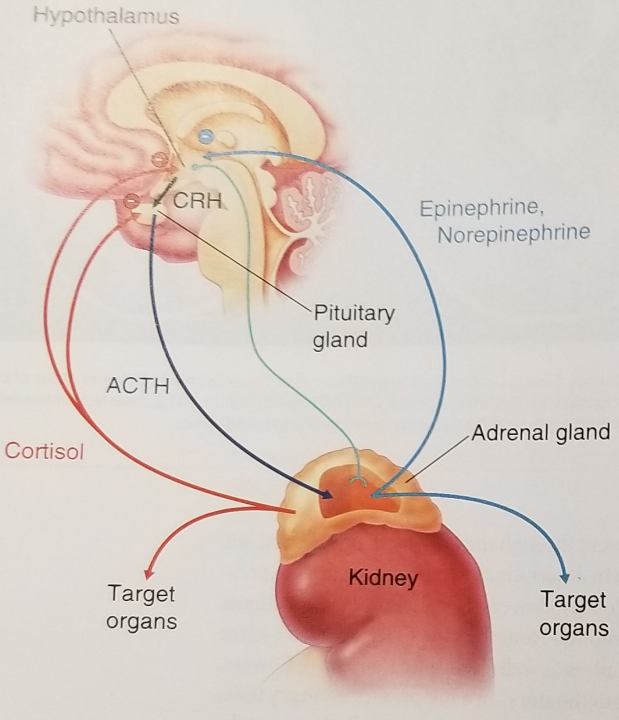
Stress, Immunity, and Health

Stress is a term that has two meanings in psychology. Stress is a condition in the environment that makes unusual demands on the organism, such as threat, failure, or bereavement. Stress is also an internal condition, your response to a stressful situation; you *feel* stressed, and your body reacts in several ways. Whether a situation is stressful to the person is often a matter of individual differences, either in perception of the situation or in physiological reactivity. For some people, even the normal events of daily life are stressful, whereas others thrive on excitement and would feel stressed if they were deprived of regular challenges. In other words, stress in this sense of the term is in the eye of the beholder.

? What are the positive effects of stress?

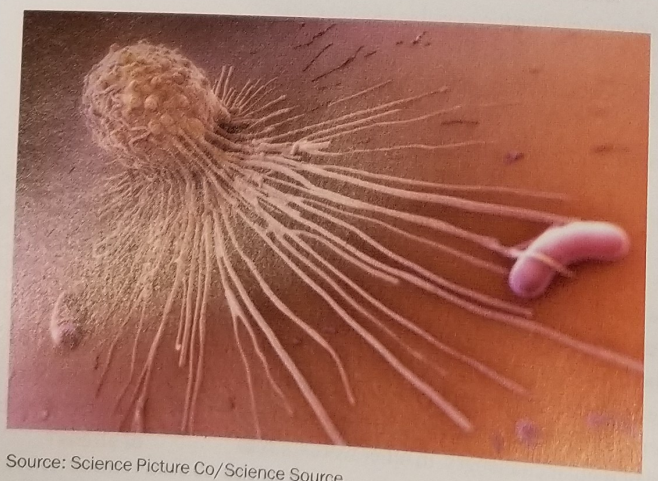
■ FIGURE 8.12 The Hypothalamus-Pituitary-Adrenal Axis.

The hypothalamus releases corticotropin-releasing hormone (CRH), which stimulates the synthesis and release of adrenocorticotropic hormone (ACTH) from the pituitary gland, which then binds to the adrenal cortex and triggers release of several stress hormones (cortisol, epinephrine, and norepinephrine). These hormones trigger a response from target organs, reducing the stress, and decreasing subsequent release of hormones in this pathway.



■ FIGURE 8.13 Macrophages Preparing to Engulf Bacteria.

A macrophage, a type of white blood cell, is stretching out projections which will eventually engulf and digest bacterial cells.



Source: Science Picture Co/Science Source.

Stress as an Adaptive Response

Ordinarily, the body's response to a stressful situation is positive and adaptive. In Chapter 3, you saw that the stress response includes activation of the sympathetic branch of the autonomic nervous system, which is largely under the control of the hypothalamus. The resulting increases in heart rate, blood flow, and respiration rate help the person deal with the stressful situation. Stress also activates the *hypothalamus-pituitary-adrenal axis*, a group of structures that help the body cope with stress (Figure 8.12). The hypothalamus activates the pituitary gland, which in turn releases hormones that stimulate the adrenal glands to release the stress hormones epinephrine, norepinephrine, and cortisol. The first two hormones increase output from the heart and liberate glucose from the muscles for additional energy. The hormone *cortisol* also increases energy levels by converting proteins to glucose, increasing fat availability, and increasing metabolism. Cortisol provides a more sustained release of energy than the sympathetic nervous system does, for coping with prolonged stress.

Brief stress increases activity in the *immune system* (Herbert et al., 1994), the cells and cell products that kill infected and malignant cells and protect the body against foreign substances such as bacteria and viruses. Of course, this is highly adaptive because it helps protect the person from any infections that might result from the threatening situation. The immune response involves two major types of cells. *Leukocytes,* or white blood cells, recognize invaders by the unique proteins that every cell has on its surface and kills them. These proteins in foreign cells are called *antigens.* A type of leukocyte called a *macrophage* ingests intruders (Figure 8.13). Then it displays the intruder's antigens on its own cell surface; this attracts *T cells,* another type of leukocyte that is specific for particular antigens, which kill the invaders. *B cells,* a third type of leukocyte, fight intruders by producing antibodies that attack a particular cell type. *Natural killer cells,* the second type of immune cells, attack and destroy certain kinds of cancer cells and cells infected with viruses; they are less specific in their targets than T or B cells. The brain and spinal cord are considered "immune privileged," in that the central nervous system is protected from most infectious agents by the blood-brain barrier. When these agents do make their way in, they are dealt with by *microglia,* which act in most ways like macrophages. Table 8.1 summarizes the characteristics of these immune cells.

Some antibodies are transferred from mother to child during the prenatal period or postnatally through the mother's milk. Most antibodies, though, result from a direct encounter with invading cells, for example, during exposure to measles. Vaccinations work because injection of a weakened form of the disease-causing bacteria or virus triggers the B cells to make antibodies for that disease.

TABLE 8.1 Major Types of Immune Cells.

LEUKOCYTES				
MACROPHAGES	T CELLS	B CELLS	NATURAL KILLER CELLS	MICROGLIA
Ingest invaders; display antigens, which attract T cells.	Multiply and attack invaders.	Make antibodies, which destroy intruders.	Attack cells containing viruses, certain kinds of tumor cells.	Ingest invaders; display antigens to attract T cells in brain, spinal cord.

The preceding is a description of what happens when all goes well. In the immune deficiency disease AIDS (acquired immune deficiency syndrome), by contrast, T cells fail to detect invaders, and the person dies of an infectious disease. In *autoimmune disorders*, the immune system runs amok and attacks the body's own cells. In the autoimmune disorder multiple sclerosis, for instance, the immune system destroys myelin in the central nervous system.

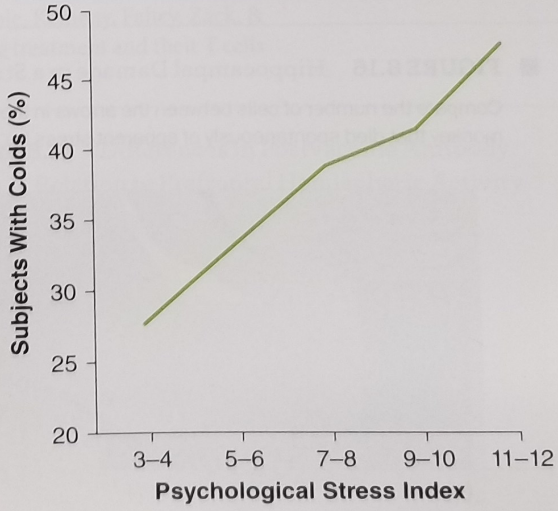
Negative Effects of Stress

We are better equipped to deal with brief stress than with prolonged stress. Chronic stress can interfere with memory, increase or decrease appetite, diminish sexual desire and performance, deplete energy, and cause mood disruptions. Although brief stress enhances immune activity, prolonged stress compromises the immune system. After the nuclear accident at the Three Mile Island electric generating plant, nearby residents had elevated stress symptoms and performed less well on tasks requiring concentration, compared with people who lived outside the area (Baum, Gatchel, & Schaeffer, 1983). Amid concerns about continued radioactivity and the long-term effects of the initial exposure, residents had reduced numbers of B cells, T cells, and natural killer cells as long as six years after the accident (McKinnon, Weisse, Reynolds, Bowles, & Baum, 1989).

Disease symptoms were not measured at Three Mile Island, but other studies have shown that health is compromised when stress impairs immune functioning. Recently widowed women experienced decreased immunity and marked health deterioration in the year following the spouse's death (Maddison & Viola, 1968). Also, students had reduced immune responses, more infectious illnesses, and slower wound healing at exam times than at other times of the year (Glaser et al., 1987; Marucha, Kiecolt-Glaser, & Favagehi, 1998). In a rare experimental study, healthy individuals were given nasal drops containing common cold viruses and then were quarantined and observed for infections. In Figure 8.14, you can see that their chance of catching a cold depended on the level of stress they reported on a questionnaire at the beginning of the study (S. Cohen, Tyrrell, & Smith, 1991). In a follow-up study, it turned out that only stresses that had lasted longer than a month increased the risk of infection (S. Cohen et al., 1998).

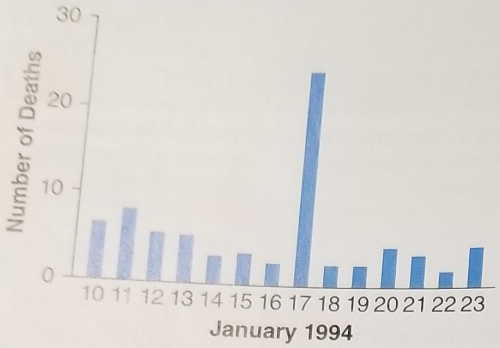
The cardiovascular system is particularly vulnerable to stress. Stress increases blood pressure, and prolonged high blood pressure can damage the heart or cause a stroke. Some people are more vulnerable to health effects from stress than others. Researchers classified young children as normal reactors or excessive reactors based on their blood pressure increases while one hand was immersed in ice water. Forty-five years later, 71% of the excessive reactors had high blood pressure, compared with 19% of the normal reactors (Wood, Sheps, Elveback, & Schirger, 1984).

■ **FIGURE 8.14** Relationship Between Stress and Vulnerability to Colds.



Source: Adapted from "Psychological Stress and Susceptibility to the Common Cold," by S. Cohen, A. D. Tyrrell, and A. P. Smith, *New England Journal of Medicine, 325*, pp. 606–612. © 1991 Massachusetts Medical Society. All rights reserved.

FIGURE 8.15 Increase in Cardiac Deaths on the Day of an Earthquake.



Source: Reprinted from "Sudden Cardiac Death Triggered by an Earthquake," by J. Leor, W. K. Poole, and R. A. Kloner, *New England Journal of Medicine, 334*, pp. 413–419. © 1996 Massachusetts Medical Society. All rights reserved.

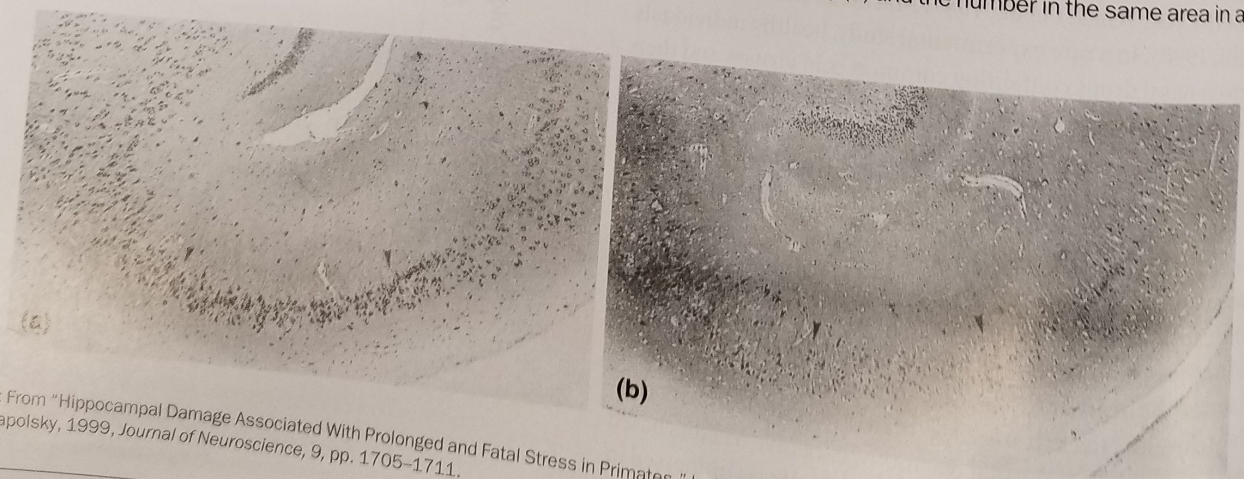
Stress can even produce death. This fact has not always been accepted in the scientific and medical communities, but in 1942 the physiologist Walter Cannon determined that reports of apparent stress-related deaths were legitimate. He even suggested that *voodoo death*, which has been reported to occur within hours of a person being "hexed" by a practitioner of this folk cult, is also due to stress. We now know that fear, loss of a loved one, humiliation, or even extreme joy can result in sudden cardiac death. In *sudden cardiac death*, stress causes excessive sympathetic activity that sends the heart into fibrillation, contracting so rapidly that it pumps little or no blood. When one of the largest earthquakes ever recorded in a major North American city struck the Los Angeles area in 1994, the number of deaths from heart attacks increased fivefold (Figure 8.15; Leor, Poole, & Kloner, 1996). The stress doesn't have to be as extreme as an earthquake: During the 2006 soccer World Cup games in Germany, cardiac emergencies in that country tripled in men and almost doubled in women (Wilbert-Lampen et al., 2008); and when the Los Angeles Rams were defeated in the 1980 Super Bowl, cardiac deaths in the team's hometown increased 15% in men and 27% in women (Kloner, McDonald, Leeka, & Poole, 2011). In 2016, when the Chicago Cubs (Gerald Hough's favorite team) won the World Series after 108 years, a 108-year-old fan died of a heart attack just days after the team's final win (Leavitt, 2016). Heart attack rates even increase during the first three days following the spring change to daylight savings time, as people cope with earlier wake times and minor sleep deprivation, and then drop slightly when the autumn transition gives them an extra hour of sleep (T. S. Janszky & Ljung, 2008).

> "In [emotional] pain there is as much wisdom as in pleasure."
>
> —Friedrich Nietzsche

Extreme stress can also lead to brain damage (Figure 8.16). Hippocampal volume was reduced in Vietnam combat veterans suffering from posttraumatic stress disorder (PTSD; Bremner et al., 1995) and in victims of childhood abuse (Bremner et al., 1997), and cortical tissue was reduced in torture victims (T. S. Jensen et al., 1982). The abused individuals had short-term memory deficits, and some of the torture victims showed slight intellectual impairment. There is some evidence that the damage is caused by cortisol; implanting cortisol pellets in monkeys' brains damaged their hippocampi (Sapolsky, Uno, Rebert, & Finch, 1990), and elderly humans who had elevated cortisol levels over a five-year period had an average 14% decrease in hippocampal volume (Lupien et al., 1998). However, individuals with PTSD have *lowered* cortisol levels. Rachel Yehuda (2001) points out that they also have an increased number and sensitivity of the glucocorticoid

FIGURE 8.16 Hippocampal Damage in a Stressed Monkey.

Compare the number of cells between the arrows in the hippocampus of a control monkey (a) and the number in the same area in a monkey that died spontaneously of apparent stress (b).



Source: From "Hippocampal Damage Associated With Prolonged and Fatal Stress in Primates," by H. Uno, R. Tarara, J. G. Else, M. A. Suleman, and R. M. Sapolsky, 1999, *Journal of Neuroscience, 9*, pp. 1705–1711.

receptors that respond to cortisol. She suggests that PTSD involves increased sensitivity to cortisol rather than an increase in cortisol level. Although there is a compensatory decrease in cortisol release, it is not adequate to protect the hippocampus. The accompanying Research Spotlight describes remarkable evidence of brain damage following recent globally significant disasters.

Several studies suggest that reducing stress can improve health. T cell counts increased in AIDS patients after 20 hours of relaxation training (D. N. Taylor, 1995); similar training was associated with reduced death rates in elderly individuals (C. N. Alexander, Langer, Newman, Chandler, & Davies, 1989) and in cancer patients (Fawzy et al., 1993; Spiegel, 1996). However, evidence that survival rate in these studies is related to immune function improvement is sketchy (Fawzy et al., 1993); it is possible that study participation led the elderly subjects and cancer patients to make lifestyle changes. At any rate, it may be more practical to block stress hormones and bolster immunity chemically. Researchers at Tel Aviv University have found that the psychological and physiological stress of cancer surgery suppresses immunity, allowing the spread of cancer during the postoperative period; combining an anti-anxiety drug with an anti-inflammatory drug greatly increased the survival rate of mice following tumor removal, and clinical trials are under way with humans (Glasner et al., 2010; "New Method to Manage . . . ," 2012).

? In what ways do personality characteristics influence immune functioning?

Social, Personality, and Genetic Factors

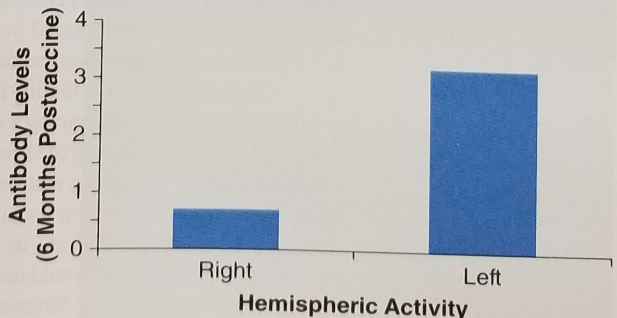
Social support was associated with dramatically lower death rates in several different populations (reviewed in House, Landis, & Umberson, 1988) and with lower stress and reduced stress hormone levels among Three Mile Island residents (Fleming, Baum, Gisriel, & Gatchel, 1982). People who are hostile are at greater risk for heart disease (T. Q. Miller, Smith, Turner, Guijarro, & Hallet, 1996), while cancer patients who have a "fighting spirit" may live longer than patients who accept their illness or have an attitude of hopelessness (Derogatis, Abeloff, & Melisaratos, 1979; Greer, 1991; Temoshok, 1987). Because of the high association between mood disorder and cancer, some observers have suggested that depression is a predisposing factor; however, the opposite is more likely, because animal research indicates that immune system cytokines released by tumors can produce depressive-like behaviors (Pyter, Pineros, Galang, McClintock, & Prendergast, 2009).

Social and personality influences must work through physiological mechanisms, which, unfortunately, are seldom assessed in these studies. An exception is an investigation of individual differences in immune response. Recall that there is a greater association of positive emotion with the left prefrontal area and negative emotion with the right. Six months after volunteers were given influenza vaccinations, the ones with higher EEG activity in the left prefrontal area had a five times greater increase in antibodies than those with higher activation on the right (Figure 8.17; Rosenkranz et al., 2003). In other research, men who had tested positive for human immunodeficiency virus (HIV) infection had HIV levels that were eight times higher if they were introverted (socially inhibited) rather than extroverted (S. W. Cole, Kemeny, Fahey, Zack, & Naliboff, 2003). The introverted patients' HIV levels also decreased less during treatment and their T cells did not increase at all.

The researchers point out that introverted individuals have elevated levels of epinephrine and norepinephrine, which activate the sympathetic nervous system during stress, and that norepinephrine increases the rate at which the HIV virus multiplies in the laboratory. Unfortunately, they didn't measure sympathetic activity specifically, but total autonomic activity (assessed from variability of heart rate, skin conductance, and other measures) was higher among the introverted HIV patients. This correlational study doesn't tell us which among introversion, norepinephrine, and HIV infection is the initial cause, but it does suggest that norepinephrine is an important mediator of the effects.

Personality characteristics such as introversion are moderately heritable, and so is vulnerability to stress; for example, a study of 300 Swedish twins concluded that 32% of workplace stress is genetic (Judge, Ilies, & Zhang, 2012). One gene implicated in stress is *NPY*, which encodes the production of neuropeptide Y (which you know from

■ **FIGURE 8.17** Differences in Postvaccine Antibody Levels in Relation to Prefrontal Hemispheric Activity.



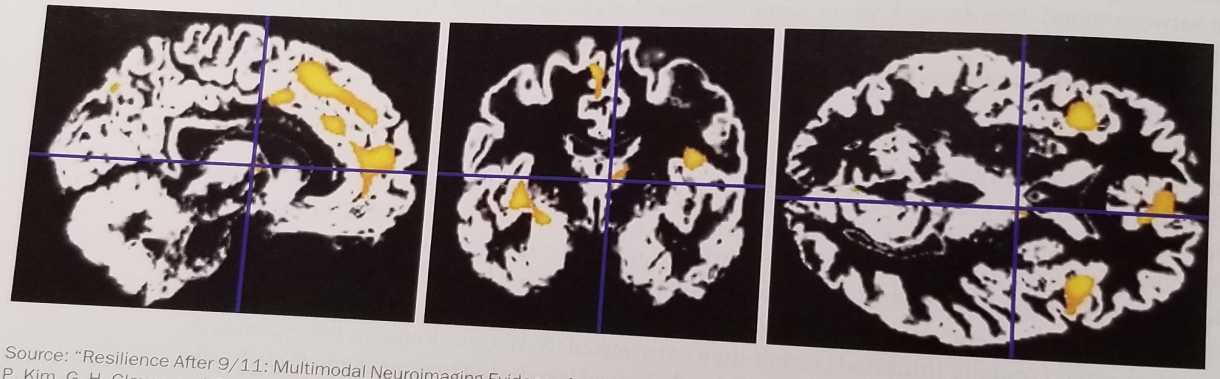
Source: From "Affective Style and In Vivo Immune Response," by M. A. Rosenkranz et al., *PNAS, 100,* pp. 11148–11152. © 2003.

RESEARCH SPOTLIGHT

One Aftermath of Disaster Is Stress-Related Brain Damage

Three years after the 9/11 terrorist attacks on the World Trade Center killed more than 2,600 people, researchers compared fMRI scans of people living within 1.5 miles (2.4 kilometers) of the towers with those of volunteers living 200 miles (322 kilometers) away (Ganzel, Kim, Glover, & Temple, 2008). Although the near residents' symptoms of PTSD were not serious enough to merit diagnosis, they had reduced gray-matter volume in the hippocampus, amygdala, prefrontal cortex, anterior cingulate cortex, and insula; in addition, amygdala activation was greater when they viewed facial expressions of fear (see figure).

Two studies of people affected by the devastating Japanese earthquake of 2011 have the rare advantage of being able to examine before-and-after brain scans. After the earthquake, researchers recruited 42 subjects who had received MRI scans as part of previous research and scanned them again (Sekiguchi et al., 2013). The subjects most likely to have PTSD symptoms had lower gray-matter volume in the anterior cingulate cortex (ACC) prior to the quake and lower gray-matter volume in the orbitofrontal cortex (OFC) after. The researchers believed that, due to the ACC's involvement in processing fear and anxiety, its small volume was a predisposing factor to developing OFC damage and PTSD symptoms. A year later, the 37 original subjects who could be located showed decreased right hippocampal volumes, but they had not developed clinical PTSD and their OFC volumes had increased (Sekiguchi et al., 2014).



Source: "Resilience After 9/11: Multimodal Neuroimaging Evidence for Stress-Related Change in the Healthy Adult Brain," by B. L. Ganzel, P. Kim, G. H. Glover, and E. Temple, 2008, *Neuroimage, 40*, pp. 788–795. Used with permission from Elsevier.

its involvement in appetite); people with a low-functioning version of the gene show greater brain activity in response to negatively charged words, report more negative feelings in anticipation of a painful stimulus, and are more prone to depression (Mickey et al., 2011). But what may be as important as the genes we have is the ability of stress to modify the expression of those genes. German researchers took repeated blood samples from subjects undergoing a stressful interview and found that methylation of the oxytocin receptor gene *OXTR* increased during the first 10 minutes and then decreased below initial levels 90 minutes afterward, presumably increasing and then decreasing the number of oxytocin receptors (Unternaehrer et al., 2012). Besides playing a role in sexual experience and bonding, oxytocin increases during stress and reduces some of the physiological effects, including blood pressure and heart rate. The researchers suggest that the receptor changes observed would mobilize the body's resources initially and then support longer-term coping with the effects of stress. Stress effects are known to sometimes carry over to the offspring, apparently due to epigenetic changes of this sort. Male mice repeatedly separated from their mothers during the first 14 days after birth showed depressive symptoms as adults (passivity in response to stressful situations); the same behaviors were seen in the offspring, along with methylation changes in genes known to be involved in responses to stress (Franklin et al., 2010).

Pain as an Adaptive Emotion

Eighty percent of all visits to physicians are at least partly to seek relief from pain (Gatchel, 1996), and we spend billions each year on nonprescription pain medications. These observations alone qualify pain as a major health problem.

A world without pain might sound wonderful, but in spite of the suffering it causes, pain is valuable for its adaptive benefits. It warns us that the coffee is too hot, that our shoe is rubbing a blister, that we should take our skis back to the bunny slope for more practice. People with *congenital insensitivity to pain* are born unable to sense pain; they injure themselves repeatedly because they are not motivated to avoid dangerous situations, and they die from untreated conditions like a ruptured appendix. Mild pain tells us to change our posture regularly; a woman with congenital insensitivity to pain suffered damage to her spine because she could not respond to these signals, and resulting complications led to her death (Sternbach, 1968).

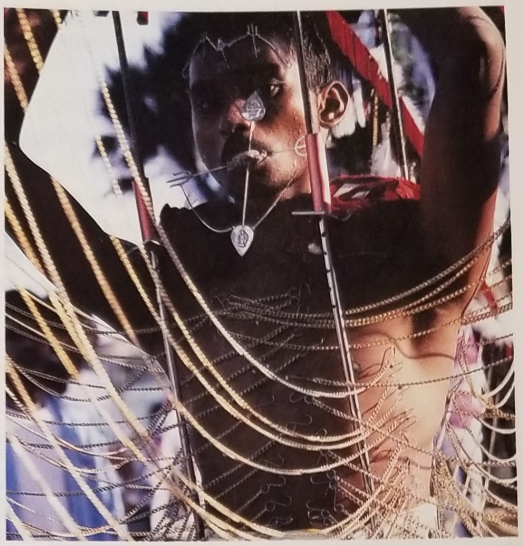
Pain is one of the senses, a point we consider in more detail in Chapter 11. Here we focus on the feature that makes pain unique among the senses: It is so intimately involved with emotion that we are justified in discussing it as an emotional response. In fact, when we tell someone about a pain experience, we are usually describing an emotional reaction; it is the emotional response that makes pain adaptive.

You know it, and Harvard psychologists have confirmed it: Pain someone inflicts on you intentionally hurts more than pain you experience accidentally (K. Gray & Wegner, 2008). As Beecher (1956) observed, "The intensity of suffering is largely determined by what the pain means to the patient" (p. 1609). In our society, childbirth is considered a painful and debilitating ordeal; in other cultures, childbirth is a routine matter, and the woman returns to work in the fields almost immediately. After the landing at the Anzio beachhead in World War II, 68% of the wounded soldiers denied pain and refused morphine; only 17% of civilians with similar "wounds" from surgery accepted their pain so bravely (Beecher, 1956). The soldiers were not simply insensitive to pain, because they complained bitterly about rough treatment or inept blood draws. According to Beecher, who was the surgeon in command at Anzio, the surgery was a major annoyance for the civilians, but the soldiers' wounds meant they had escaped the battlefield alive. Spiritual context can also have a powerful influence on the meaning of pain. Each spring in some remote villages of India, a man is suspended by a rope attached to steel hooks in his back; swinging above the cheering crowd, he blesses the children and the crops. Selection for this role is an honor, and the participant seems not only to be free of pain but also in a "state of exaltation" (Ghosh & Sinha, 2007; Kosambi, 1967). Figure 8.18 shows an example of culturally sanctioned self-torture.

The pain pathway has rich interconnections with the limbic system, where pain becomes an emotional phenomenon. Besides the somatosensory area, pain particularly activates the anterior cingulate cortex, which in turn is intimately connected with other limbic structures (D. D. Price, 2000; Talbot et al., 1991). The brain scan in Figure 8.19 shows increased activity in the anterior cingulate cortex as well as the somatosensory area during painful heat stimulation. But what evidence is there that activity in the anterior cingulate cortex represents the emotional aspect of pain? First of all, in humans and monkeys some of the neurons respond to the anticipation of pain as well as to painful stimulation (Hutchison,

FIGURE 8.18 Voluntary Ritualized Torture in Religious Practice.

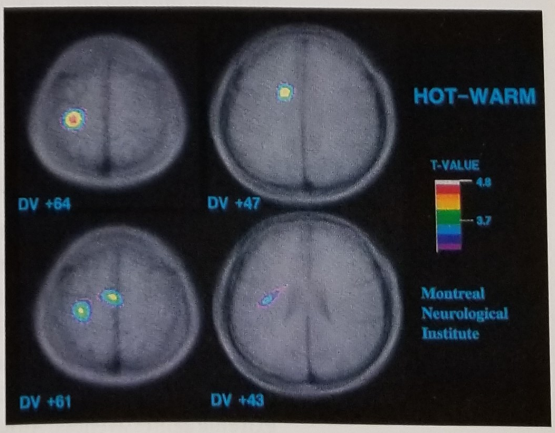
Cultural values help determine a person's reaction to painful stimulation.



Source: ©Alain Evrard/Photo Researchers.

FIGURE 8.19 PET Scan of Brain During Painful Heat Stimulation.

The bright area near the midline is the cingulate gyrus; the one to the left is in the somatosensory area. The four views were taken simultaneously at different depths in the same brain. (The frontal lobes are at the top of the figure.)



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