The framework students need to go from inquiry to understanding.
3 BIOLOGICAL PSYCHOLOGY
bridging the levels of analysis

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In the early 21st century, we take for granted the fact that the brain is the seat of psychological activity. When we struggle with a difficult homework problem, we say that “our brains hurt,” when we consult friends for advice about a complicated question, we “pick their brains,” and when we insult others’ intelligence, we call them “bird brains.” Yet throughout much of human history, it seemed obvious that the brain wasn’t the prime location for our thoughts, memories, and emotions.

For example, the ancient Egyptians believed that the heart was the seat of the human soul and the brain was irrelevant to mental life (Finger, 2000; Raulin, 2003). Egyptians often prepared corpses for mummification by scooping their brains out through the nostrils using an iron hook (you’ll be pleased to know that no drawings of this practice survive today) (Leek, 1969). Although some ancient Greeks correctly pinpointed the brain as the source of the psyche, others, like the great philosopher Aristotle, were convinced that the brain functions merely as a radiator, cooling the heart when it becomes overheated. Even today, we can find holdovers of this way of thinking in our everyday language. When we memorize something, we come to know it “by heart” (Finger, 2000). When we’re devastated by the loss of a romantic relationship, we feel “heartbroken.”

Why were so many of the ancients certain that the heart, not the brain, was the source of mental activity? It’s almost surely because they trusted their “common sense,” which as we’ve learned is often a poor signpost of scientific truth (Chapter 1). They noticed that when people become excited, angry, or scared, their hearts pound quickly, whereas their brains seem to do little or nothing. Therefore, they reasoned, the heart must be causing these emotional reactions. By confusing correlation with causation, the ancients’ intuitions misled them.

Today, we recognize that the mushy organ lying between our two ears is by far the most complicated structure in the known universe. Our brain has the consistency of gelatin, and it weighs a mere three pounds. Despite its rather unimpressive appearance, it’s capable of astonishing feats. As poet Robert Frost wrote, “The brain is a wonderful organ. It starts working the moment you get up in the morning and does not stop until you get into the office.”

In recent decades, scientists have made numerous technological strides that have taught us a great deal about how our brains work. Researchers who study the relationship between the nervous system—a communication network consisting of nerve cells, both inside and outside of the brain and spinal cord—and behavior go by the names of biological psychologists or neuroscientists. By linking brain to behavior, these scientists bridge multiple levels of analysis within psychology (see Chapter 1). As we explore what biological psychologists have discovered about the brain, we’ll compare our current state-of-the-art knowledge with misconceptions that have arisen along the way (Aamodt & Wang, 2008). The history of our evolving understanding of the brain provides a wonderful example of the self-correcting nature of science (see Chapter 1). Over time, mistaken beliefs about the brain have gradually been replaced by more accurate knowledge (Finger, 2000).

NERVE CELLS: COMMUNICATION PORTALS

1. Distinguish the parts of neurons and what they do.
2. Describe electrical responses of neurons and what makes them possible.
3. Explain how neurons use neurotransmitters to communicate with each other.
4. Describe how the brain changes as a result of development, learning, and injury.

If we wanted to figure out how a car works, we’d open it up and identify its parts, like its engine, carburetor, and transmission, and then try to figure out how they operate in tandem. Similarly, to understand how our brains works, we first need to get a handle on its key components and determine how they cooperate. To do so, we’ll start with the brain’s most basic unit of communication: its cells. Then, we’ll examine how these cells work in concert to generate our thoughts, feelings, and behaviors.
Neurons: The Brain’s Communicators

The functioning of our brain depends on cross-talk among neurons—nerve cells exquisitely specialized for communication with each other (see FIGURE 3.1). Our brains contain about 100 billion neurons. To give you a sense of how enormous this number is, there are more than 15 times as many neurons in our brains as there are people on Earth. More graphically, 100 billion neurons lined up side to side would reach back and forth from New York to California five times. What’s more, many neurons forge tens of thousands of connections with other neurons, permitting a staggering amount of inter-cellular communication. In total, there are about 160 trillion—that’s a whopping 160,000,000,000,000—connections in the human brain (Tang et al., 2001).
As adults, we lose about 100,000 neurons each day. Although we do lose neurons each day, the actual number is considerably lower, perhaps one-tenth of that (Juan, 2006).

Neurons and their dendrites (shown stained pink) with their nuclei (shown stained blue).

**FICTOID**

**MYTH:** As adults, we lose about 100,000 neurons each day.

**REALITY:** Although we do lose neurons each day, the actual number is considerably lower, perhaps one-tenth of that (Juan, 2006).

**THE CELL BODY.** The **cell body**, also called the soma, is the central region of the neuron. It manufactures new cell components, which consist of small and large molecules (refer to Figure 3.1). Because the cell body contains the nucleus, where proteins are manufactured, damage to this part of the neuron is fatal. The cell body also provides continual renewal of cell components.

**DENDRITES.** Neurons differ from other cells in their branchlike extensions for receiving information from other neurons. These extensions, which we can liken to the receivers on our cell phones, are **dendrites**. Dendrites spread out to “listen in” on information from neighboring neurons and pass it on to the cell body (refer to Figure 3.1).

**AXONS AND AXON TERMINALS.** **Axons** are long tail-like extensions protruding from the cell body. We can liken axons to the transmitters on our cell phones, because they’re specialized for sending messages to other neurons. Unlike dendrites, axons are usually very thin near the cell body. This narrowness creates a **trigger zone**, an area that’s easily activated. The **axon terminal** is a knoblike structure at the far end of the axon (see **FIGURE 3.2**). Axon terminals, in turn, contain **synaptic vesicles**, tiny spheres that contain **neurotransmitters**, chemical messengers that neurons use to communicate with each other. Synaptic vesicles are manufactured in the cell body and travel down the length of the axon. We might think of the synaptic vesicles as similar to gel capsules filled with cold medicine. When we swallow a capsule, its exterior dissolves and the medicine inside it moves down our digestive tracts. Similarly, when the synaptic vesicle reaches the end of the axon terminal, it bursts, releasing neurotransmitters.

**SYNAPSES.** Neurotransmitters then enter the **synapse**, a miniscule fluid-filled space between neurons through which neurotransmitters travel. The synapse consists of a **synaptic cleft**, a gap into which neurotransmitters are released from the axon terminal. This gap is surrounded by small patches of membrane on each side, one on the sending axon of the first neuron and the other on the receiving dendrite of the second neuron. As neurotransmitters are released from the axon of a cell into the synapse, they’re quickly picked up by the dendrites of nearby neurons, just as phone receivers quickly pick up signals from other phones.

British neuroscientist Sir Charles Sherrington was one of the first to hypothesize the existence of synapses. He measured how long it took muscles to become active following nerve stimulation. From these data, he inferred the existence of microscopic spaces between neurons themselves and between neurons and muscle cells (Pearce, 2004). At the time, no microscopes were powerful enough to observe these spaces. Consequently, some scientists believed that all neurons melded together into one giant net. But Sherrington (1906) argued that neurons are separate cells that communicated with each other and with muscle cells. What he hypothesized could have been falsified had he been wrong. Spanish scientist Santiago Ramón y Cajal showed that Sherrington was right using a staining technique that demonstrated the existence of individual neurons. Later studies using powerful **electron microscopes** confirmed that tiny gaps allowing communication between neurons, which we now recognize as synapses, indeed exist (Davis, 2006).
GLIAL CELLS: SUPPORTING ACTORS OR KEY PLAYERS? But neurons aren’t the only players in our nervous systems: Glial cells (glial means glue) are also remarkably plentiful. Scientists once regarded them as nothing more than bit-part actors in the nervous system that surround the synapse and provide protective scaffolding for the neurons they hold in place. Nevertheless, over the past 20 years or so, researchers have realized that glial cells are star performers in their own right (Fields, 2009).

What accounts for their elevated status? It’s more than the star shape of astrocytes (astro means star in Greek), the most abundant of glial cells. A single astrocyte interacts with as many as 300,000–1,000,000 neurons. The well-connected astrocytes communicate closely with neurons, increase the reliability of their transmission, control blood flow in the brain, and play a vital role in the development of the embryo (Metea & Newman, 2006). Astrocytes, in concert with other glial cells, are intimately involved in thought, memory, and the immune system (Gibbs & Bowser, 2009; Koob, 2009). Although researchers once thought that glial cells greatly outnumbered neurons, by as much as 10:1, recent research suggests that the ratio is much lower, and closer to 1:1 (Azevedo et al., 2009).

We can find astrocytes in great supply in the blood–brain barrier, a fatty coating that wraps around tiny blood vessels. As a result, large molecules, highly charged particles, and molecules that dissolve in water but not in fat are blocked from entering the brain. The blood–brain barrier is the brain’s way of protecting itself from infection by bacteria and other intruders. Treatments that target glial cells may assist in treating a variety of conditions related to the number and activity of glial cells, including depression and schizophrenia (Cotter, Pariant, & Everall, 2001; Schroeter et al., 2009), as well as inflammation, chronic pain, and Alzheimer’s disease and other degenerative conditions (Suter et al., 2007).

Another type of glial cell, called an oligodendrocyte, promotes new connections among nerve cells and releases chemicals to aid in healing. In addition, this cell produces an insulating wrapper around axons called the myelin sheath. This sheath contains numerous gaps all the way along the axon called nodes, which help the neuron conduct electricity more efficiently (refer again to Figure 3.1). Much like a person playing hopscotch, the neural signal jumps from node to node, speeding up its transmission. In the autoimmune disease of multiple sclerosis, the myelin sheaths surrounding neurons are “eaten away,” resulting in a progressive loss of insulation of neural messages. As a consequence, these messages become hopelessly scrambled, resulting in a wide variety of physical and emotional symptoms. Glial cells also clear away debris, acting as the brain’s cellular garbage disposals. We hope you’ll agree that if glial cells don’t deserve an academy award they at least merit a nomination.

Neurons respond to neurotransmitters by generating electrical activity (see FIGURE 3.3 on page 88). We know this because scientists have recorded electrical activity from neurons using electrodes, small devices made from wire or fine glass tubes. These electrodes allow them to measure the potential difference in electrical charge inside versus outside the neuron. The basis of all electrical responses in neurons depends on an uneven distribution of charged particles across the membrane surrounding the neuron (see Figure 3.3). Some particles are positively charged, others negatively charged. When there are no neurotransmitters acting on the neuron, the membrane is at the resting potential. In this baseline state, when the neuron isn’t doing much of anything, there are more negative particles inside than outside the neuron. In some large neurons, the voltage of the resting potential can be about one-twentieth that of a flashlight battery, or about –60 millivolts (the negative sign means the inside charge is more negative than outside). While at rest, particles of both types are flowing in and out of the membrane. When the electrical charge inside the neuron reaches a high enough level relative to the outside, called the threshold, an action potential occurs.

ACTION POTENTIALS. Action potentials are abrupt waves of electric discharge triggered by a change in charge inside the axon. When this change occurs, we can describe the neuron as “firing,” similar to the firing of a gun. Much like a gun, neurons obey the “all or none” law:
They either fire or they don’t (you wouldn’t accuse a criminal of “sort of shooting at me”). Action potentials originate in the trigger zone near the cell body and continue all the way down the axon to the axon terminal. During an action potential, positively charged particles flow rapidly into the axon and then just as rapidly flow out, causing a spike in positive charge followed by a sudden decrease in charge, with the inside charge ending up at a slightly more negative level than its original resting value (see FIGURES 3.3 and 3.4). These sudden shifts in charge produce a release of electricity. When the electrical charge reaches the axon terminal, it triggers the release of neurotransmitters—chemical messengers—into the synapse.

**THE ABSOLUTE REFRACTORY PERIOD.** Neurons can fire extremely rapidly, at rates of 100 to 1,000 times per second. At this very moment, energy is traveling down tens of millions of your axons at breakneck speeds of about 220 miles per hour. Each action potential is followed by an absolute refractory period, a brief interval during which another action potential can’t occur. This period limits the maximal firing rate, the fastest rate at which a neuron can fire, much as it takes us awhile to reload some guns after firing them. The rate at which action potentials travel becomes an issue in very long axons, such as the sciatic nerve, which runs from the spinal cord down the leg. Remarkably, in humans this axon extends an average of three feet.

**Chemical Communication: Neurotransmission**

Whereas electrical events transmit information within neurons, chemical events initiated by neurotransmitters orchestrate communication among neurons. After neurotransmitter molecules are released into the synapse, they bind with receptor sites along the dendrites of neighboring neurons. Different receptor sites recognize different types of neurotransmitters. Researchers typically invoke a lock-and-key analogy to describe this specificity (see FIGURE 3.5). We can think of each neurotransmitter as a key that fits only its own type of receptor, or lock.

Neurotransmission can be halted by reuptake of the neurotransmitter back into the axon terminal—a process by which the synaptic vesicle reabsorbs the neurotransmitter. We can think of release and reuptake of the neurotransmitter as analogous to letting some liq-
uid drip out of the bottom of a straw (release) and then sucking it back up again (reuptake). Reuptake is one of nature’s recycling mechanisms.

**NEUROTRANSMITTERS.** Different neurotransmitters are different messengers, each with a slightly different thing to say. Some excite the nervous system, increasing its activity, whereas others inhibit the nervous system, decreasing its activity. Some play a role in movement, others in pain perception, and still others in thinking and emotion. Let’s now meet a few of the more prominent neurotransmitters (see TABLE 3.1).

**Glutamate and GABA.** Glutamate and gamma-aminobutyric acid (GABA) are the most common neurotransmitters in the central nervous system (CNS). Neurons in virtually every brain area use these neurotransmitters to communicate with each other (Fagg & Foster, 1983). Glutamate rapidly excites neurons, increasing the likelihood that they’ll communicate with other neurons. The release of glutamate is associated with enhanced learning and memory (see Chapter 7). When elevated, glutamate may also contribute to schizophrenia and other mental disorders, because in high doses it can be toxic, damaging neural receptors by overstimulating them (Goff & Coyle, 2001; Karlsson et al., 2008).

### TABLE 3.1 Neurotransmitters and Their Major Functional Roles.

<table>
<thead>
<tr>
<th>NEUROTRANSMITTER</th>
<th>SELECTED ROLES</th>
<th>DRUGS THAT INTERACT WITH THE NEUROTRANSMITTER SYSTEM</th>
</tr>
</thead>
<tbody>
<tr>
<td>Glutamate</td>
<td>Main excitatory neurotransmitter in the nervous system; participates in relay of sensory information and learning</td>
<td>Alcohol and memory enhancers interact with N-methyl-D-aspartate (NMDA) receptors, a specific type of glutamate receptor.</td>
</tr>
<tr>
<td>Gamma-aminobutyric acid (GABA)</td>
<td>Main inhibitory neurotransmitter in the nervous system</td>
<td>Alcohol and antianxiety drugs increase GABA activity.</td>
</tr>
<tr>
<td>Acetylcholine (ACh)</td>
<td>Muscle contraction (PNS), Cortical arousal (CNS)</td>
<td>Nicotine stimulates ACh receptors. Memory enhancers increase ACh. Insecticides block the breakdown of ACh. Botox causes paralysis by blocking ACh.</td>
</tr>
<tr>
<td>Norepinephrine (NE)</td>
<td>Brain arousal and other functions like mood, hunger, and sleep</td>
<td>Amphetamine and methamphetamine increase NE.</td>
</tr>
<tr>
<td>Dopamine</td>
<td>Motor function and reward</td>
<td>L-Dopa, which increases dopamine, is used to treat Parkinson’s disease. Antipsychotic drugs, which block dopamine action, are used to treat schizophrenia.</td>
</tr>
<tr>
<td>Serotonin</td>
<td>Mood and temperature regulation, aggression, and sleep cycles</td>
<td>Serotonin-selective reuptake inhibitor (SSRI) antidepressants are used to treat depression.</td>
</tr>
<tr>
<td>Endorphins</td>
<td>Pain reduction</td>
<td>Narcotic drugs—codeine, morphine, and heroin—reduce pain and produce euphoria.</td>
</tr>
<tr>
<td>Anandamide</td>
<td>Pain reduction, increase in appetite</td>
<td>Tetrahydrocannabinol (THC)—found in marijuana—produces euphoria.</td>
</tr>
</tbody>
</table>

(Source: Adapted from Carlson et al., 2007)
Athletes, like this bicyclist, often rely on their endorphins to push them through intense pain.

GABA, in contrast, inhibits neurons, thereby dampening neural activity. That’s why most antianxiety drugs bind to GABA receptors. GABA is a workhorse in our nervous systems, playing critical roles in learning, memory, and sleep (Gottesman, 2002; Jacobson et al., 2007; Wang & Kriegstein, 2009). Scientists are intrigued by the promise of drugs that target GABA to one day treat a variety of conditions, including insomnia, depression, and epilepsy (Gerard & Aybala, 2007; Mann & Mody, 2008; Winkelman et al., 2008).

**Acetylcholine.** The neurotransmitter acetylcholine plays roles in arousal, selective attention, sleep (see Chapter 5), and memory (McKinney & Jacksonville, 2005; Woolf, 1991). In the neurological disorder of Alzheimer’s disease, neurons containing acetylcholine (and several other neurotransmitters) are progressively destroyed, leading to severe memory loss (see Chapter 7). Medications that alleviate some of the symptoms of Alzheimer’s, like the drug Aricept (its generic name is Donezepil), boost acetylcholine levels in the brain. Neurons that connect directly to muscle cells also release acetylcholine, allowing them to trigger movement. That’s how most insecticides work; they limit the breakdown of acetycholine (allowing more acetylcholine to stick around the synapse), causing insects to engage in uncontrolled motor activity that eventually kills them.

**Monoamines.** Norepinephrine, dopamine, and serotonin are the monoamine neurotransmitters (they’re called “monoamines” because they contain only one amino acid, the building block of proteins). Dopamine plays an especially critical role in the rewarding experiences that occur when we seek out or accomplish goals, whether they be sex, a fine meal, or a gambling jackpot. Research even shows that brain areas rich in dopamine become active when we hear a funny joke (Mobbs et al., 2003). Norepinephrine and serotonin activate or deactivate various parts of the brain, influencing arousal and our readiness to respond to stimuli (Jones, 2003).

**Neuropeptides.** Neuropeptides are short strings of amino acids in the nervous system. They act somewhat like neurotransmitters, but their roles are typically more specialized. Endorphins are a type of neuropeptide that play a specialized role in pain reduction (Holden, Jeong, & Forrest, 2005). Endorphins were discovered in the early 1970s by neuroscientists Candace Pert and Solomon Snyder, who hoped to pinpoint the physiological mechanisms of opioids, drugs like morphine and codeine that produce pain relief and euphoria. Remarkably, they discovered that our brains contain their very own receptors for naturally occurring opioids—endorphins (Pert, Pasternak, & Snyder, 1973). So human-made opioids, like morphine, exert their effects by “hijacking” the endorphin system, binding to endorphin receptors and mimicking their effects. Our brains contain a host of other neuropeptides; some regulate hunger and satiety (fullness), and others learning and memory.

**Anandamide.** Just as we knew about opiates long before we knew about endogenous opioids, we knew about marijuana and its active ingredient, tetrahydrocannabinol (THC), long before we knew about anandamide. Cells in our bodies, like neurons, make anandamide, which binds to the same receptors as THC. Anandamide plays roles in eating, motivation, memory, and sleep.

**NEUROTRANSMITTERS AND PSYCHOACTIVE DRUGS.** Scientists have developed specific medications to target the production or inhibition of certain neurotransmitters (refer again to Table 3.1). Drugs that interact with neurotransmitter systems are called psychoactive, meaning they affect mood, arousal, or behavior (see Chapter 5).

Knowing how psychoactive drugs interact with neurotransmitter systems allows us to predict how they’ll affect us psychologically. Opiates, such as codeine and morphine, function as agonists, meaning they increase receptor site activity. Specifically, they reduce our emotional response to painful stimuli by binding with opioid receptors (the receptors discovered by Pert and Snyder) and mimicking endorphins (Evans, 2004). Tranquilizers, like Xanax (whose generic name is Alprazolam), diminish anxiety by stimulating GABA receptor sites, thereby tamping down neuronal activity (Roy-Byrne, 2005). As we’ve already seen with insecticides, still other drugs block the reuptake of neurotransmitters. Many anti-
depressants, like Prozac (whose generic name is Fluoxetine), inhibit the reuptake of certain neurotransmitters, especially serotonin, from the synapse (Schatzberg, 1998). By allowing these neurotransmitters to remain in the synapse longer than usual, these medications enhance these neurotransmitters’ effects on receptor sites—much as we can heighten the pleasurable sensations of a delicious food by keeping it in our mouths a bit longer than usual.

Some drugs work in the opposite way, functioning as receptor antagonists, meaning they decrease receptor site activity. Most medications used to treat schizophrenia—a severe mental disorder we’ll describe more fully in Chapter 15—block dopamine receptors by binding to them and then blocking dopamine from binding to the receptors themselves (Bennett, 1998; Compton & Broussard, 2009).

Neural Plasticity: How and When the Brain Changes

We’ll conclude our guided tour of neurons by looking at the ability of the nervous system to change. Nature—our genetic makeup—influences what kind of changes are possible and when they’ll occur during the long and winding road from birth to old age. Nurture, consisting of learning, life events, injuries, and illnesses, affects our genetically influenced course. Scientists use the term plasticity to describe the nervous system’s ability to change. We can talk about brain circuits being “hardwired” when they don’t change much, if at all.

But in fact, few human behaviors are “hardwired,” even though the popular media frequently use this term to refer to genetically influenced characteristics. That’s because the nervous system is continually changing, by leaps and bounds, as in early development, or more subtly, as with learning. Unfortunately, the nervous system often doesn’t change enough following injury, which can lead to permanent paralysis and disability.

Neural Plasticity over Development. Typically, our brain is most capable of changing during early development, when much of our nervous system has yet to be set in place. Our brains don’t mature fully until late adolescence or early adulthood. This means the period of heightened plasticity in the human brain is lengthy, with some parts maturing faster than others. Watch on mypsychlab.com

The network of neurons in the brain changes over the course of development in four primary ways:

1. growth of dendrites and axons;
2. synaptogenesis, the formation of new synapses;
3. pruning, consisting of the death of certain neurons and the retraction of axons to remove connections that aren’t useful; and
4. myelination, the insulation of axons with a myelin sheath.

Of these four steps, pruning is probably the most surprising. During pruning, as many as 70 percent of neurons die off. This process is helpful, though, because it streamlines neural organization, enhancing communication among brain structures (Oppenheim, 1991). In a real sense, less is more, because with pruning our brains can process information more efficiently with fewer neurons. One theory of infantile autism (see Chapter 15) suggests that this disorder is caused by inadequate pruning (Hill & Frith, 2003), which may explain why individuals with autism tend to have unusually large brains (Herbert, 2005).

Late maturation of certain cortical areas has fueled interest in the brains of teenagers and how their brain maturation—or lack thereof—affects their decision making (Steinberg, 2008). By age 12, the human brain is adult in size and weight. Nonetheless, adolescent brain activity patterns—such as those shown by brain imaging techniques we’ll soon discuss—are still far different from those of adults (see Chapter 10).

Neural Plasticity and Learning. Our brains change as we learn. The simplest change occurs when synapses simply perform better, that is, show stronger and more prolonged excitatory responses. Researchers call this phenomenon potentiation, and when it’s
enduring, long-term potentiation (LTP) (see Chapter 7). Many scientists believe that structural plasticity, in the form of altered neuronal shape, is also critical for learning. A number of investigators have demonstrated learning-related structural changes in both axons and dendrites (Woolf, 2006). In one study, researchers trained rats to swim to a platform hidden in a tub of milky water. By the time the rats became adept at doing so, axons entering a part of their brains relevant to spatial ability had expanded (Holahan et al., 2006). Exposure to enriched environments also results in structural enhancements to dendrites (see Figure 3.6). Two studies compared rats exposed to an enriched environment—such as large cages with multiple animals, toys, and running wheels—with rats exposed to a standard environment of a cage with only two animals and no objects (Freire & Cheng, 2004; Leggio et al., 2005). Enriched environments led to more elaborate dendrites with more branches.

**NEURAL PLASTICITY FOLLOWING INJURY AND DEGENERATION.** In adults, brain plasticity decreases sharply, occurring only on a small scale, such as with learning. The human brain and spinal cord exhibit only limited regeneration following injury or serious illness. Yet certain brain regions can sometimes take over the functions previously performed by others. For example, in blind people, the capacity to read Braille (a system of raised dots that correspond to letters in the alphabet) with the fingers is taken over by brain regions associated with vision in sighted people (Hamilton & Pascual-Leone, 1998).

Not surprisingly, scientists are focused on finding ways to get around the barriers that prevent brain and spinal cord axons from growing back following injury (Maier & Schwab, 2006). Some humans and animals recover sensory and motor function following certain treatments, but the degree of recovery varies greatly (Bradbury & McMahon, 2006; Jones et al., 2001). Because degenerative disorders, such as Alzheimer’s disease and Parkinson’s disease, pose enormous challenges to society, scientists are actively investigating ways of preventing damage or enabling the brain to heal itself.

**Stem Cells.** You’ve probably heard or read about research on stem cells, especially embryonic stem cells, in the news. The reason they’ve garnered so much attention is that they have the potential to become a wide variety of specialized cells (see Figure 3.7). This is akin to being a first-year undergraduate who has yet to declare a major: He or she might become nearly anything. Once the cell begins to specialize, however, the cell type becomes more permanently cast, much like an undergraduate who’s spent three years taking pre-med courses. Stem cells offer several ways of treating diseases marked by neural degeneration (Fukuda & Takahashi, 2005; Miller, 2006; Muller, Snyder, & Loring, 2006). For example, researchers can implant stem cells directly into the host’s nervous system and induce them to grow and replace damaged cells. In addition, researchers can genetically engineer stem cells so that the cells can administer gene therapy—that is, provide the patient with replacement genes.

Yet stem cell research is exceedingly controversial for ethical reasons. Its advocates point to its potential for treating serious diseases, including Alzheimer’s, diabetes, and certain cancers, but its opponents point out that such research requires investigators to destroy lab-created balls of cells that are four or five days old (which at that stage are smaller than the period at the end of this sentence). For stem cell research opponents, these cells are an early form of human life. As we learned in Chapter 1, certain profoundly important questions are metaphysical and therefore lie outside the boundaries of science: Science deals only with testable claims within the realm of the natural world (Gould, 1997). The question of whether
stem cell research may one day cure diseases falls within the scope of science, but the question of whether such research is ethical doesn’t. Nor, in all likelihood, can science ever resolve definitively the question of when human life begins (Buckle, Dawson, & Singer, 1989). As a consequence, reasonable people will continue to disagree on whether stem cell research should be performed.

**Neurogenesis.** There’s another way that researchers may be able to get around the lack of regeneration following injury and neural degeneration. **Neurogenesis** is the creation of new neurons in the adult brain. Less than 20 years ago, most scientists were quite sure that we’re born with all the neurons we’ll ever have. Then Fred Gage (interestingly, a descendant of Phineas Gage, whom we’ll meet later in the chapter), Elizabeth Gould, and their colleagues discovered that in adult monkeys, neurogenesis occurs in certain brain areas (Gage, 2002; Gould & Gross, 2002). The odds are high that neurogenesis occurs in adult human brains, too.

Why does neurogenesis occur in adults? One possibility is that it plays a role in learning (Aimone, Wiles, & Gage, 2006). Another role may be aiding recovery following brain injury. By triggering neurogenesis, scientists may one day be able to induce the adult nervous system to heal itself (Kozorovitskiy & Gould, 2003; Lie et al., 2004).

**assess your knowledge**

**FACT OR FICTION?**

1. Dendrites are the sending portions of neurons. **True** / **False**
2. Positive particles flowing into the neuron inhibit its action. **True** / **False**
3. Neurotransmitters send messages between neurons. **True** / **False**
4. Some antidepressants block the reuptake of serotonin into the axon terminal. **True** / **False**
5. Neurogenesis is the same thing as pruning. **True** / **False**

**THE BRAIN—BEHAVIOR NETWORK**

3.5 Identify what roles different parts of the central nervous system play in behavior.

3.6 Clarify how the somatic and autonomic nervous systems work in emergency and everyday situations.

The connections among neurons provide the physiological bases of our thoughts, emotions, and behaviors. But how do we get from electrical charges and release of neurotransmitters to complex behaviors, like writing a term paper or asking someone out for a date? Let’s say we decide to walk to a vending machine to buy a can of soda. How does our brain, this motley collection of billions of neurons, accomplish this feat? First, our brain makes a conscious decision to do so—or so it would seem. Second, our nervous system propels our body into action. Third, we need to locate and operate the vending machine. We must accurately identify the machine based on how it looks and feels, insert the right amount of money, and finally retrieve our soda to take a well-deserved sip. Communication among neurons in the vast network of connections we call our nervous system allows us to take these complex actions for granted.

We can think of our nervous system as a superhighway with a two-way flow of traffic. Sensory information comes into—and decisions to act come out of—the **central nervous system (CNS)**, composed of the brain and spinal cord. Scientists call all the nerves that extend outside of the CNS the **peripheral nervous system (PNS)** (see **FIGURE 3.8** on page 94). The PNS is further divided into the somatic nervous system, which controls voluntary behavior, and the autonomic nervous system, which controls nonvoluntary, that is, automatic, functions of the body (see Chapter 11).

**neurogenesis**
creation of new neurons in the adult brain

**central nervous system (CNS)**
part of nervous system containing brain and spinal cord that controls the mind and behavior

**peripheral nervous system (PNS)**
nerves in the body that extend outside the central nervous system (CNS)
The Central Nervous System: The Command Center

Scientists divide the CNS into distinct sections or systems (see Table 3.2). The brain and spinal cord are protected by meninges, three thin layers of membranes. Further protection is afforded by the cerebral ventricles, fluid-filled pockets that extend throughout the entire brain that contain cerebrospinal fluid (CSF), which provide the brain with nutrients and cushion against injury.

### Table 3.2 The Organization of the Central Nervous System.

<table>
<thead>
<tr>
<th>Central Nervous System</th>
<th>Function</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cortex</strong></td>
<td>Executive function coordinating other brain areas, motor planning, language, and memory</td>
</tr>
<tr>
<td><strong>Frontal Lobe</strong></td>
<td>Processes touch information, integrates vision and touch</td>
</tr>
<tr>
<td><strong>Parietal Lobe</strong></td>
<td>Processes auditory information, language, and autobiographical memory</td>
</tr>
<tr>
<td><strong>Occipital Lobe</strong></td>
<td>Processes visual information</td>
</tr>
<tr>
<td><strong>Basal Ganglia</strong></td>
<td>Control movement and motor planning</td>
</tr>
<tr>
<td><strong>Midbrain</strong></td>
<td>Tracks visual stimuli and reflexes triggered by sound</td>
</tr>
<tr>
<td><strong>Thalamus</strong></td>
<td>Conveys sensory information to cortex</td>
</tr>
<tr>
<td><strong>Hypothalamus</strong></td>
<td>Oversees endocrine and autonomic nervous system</td>
</tr>
<tr>
<td><strong>Amygdala</strong></td>
<td>Regulates arousal and fear</td>
</tr>
<tr>
<td><strong>Hippocampus</strong></td>
<td>Processes memory for spatial locations</td>
</tr>
<tr>
<td><strong>Brain Stem</strong></td>
<td>Regulates breathing and heartbeats</td>
</tr>
<tr>
<td><strong>Pons</strong></td>
<td>Conveys information between the cortex and cerebellum</td>
</tr>
<tr>
<td><strong>Medulla</strong></td>
<td>Conveys information between the brain and the body</td>
</tr>
<tr>
<td><strong>Cerebellum</strong></td>
<td>Controls balance and coordinated movement</td>
</tr>
<tr>
<td><strong>Spinal Cord</strong></td>
<td>Conveys information between the brain and the body</td>
</tr>
</tbody>
</table>
brain and spinal cord. A clear liquid, called cerebrospinal fluid (CSF), runs through these ventricles and bathes our brain and spinal cord, providing nutrients and cushioning us against injury. This fluid is the CNS’s shock absorber, allowing us to move our heads rapidly in everyday life without sustaining brain damage.

As we review different brain regions, bear in mind that although these regions serve different functions, they cooperate seamlessly with each other to generate our thoughts, feelings, and behaviors (see Figure 3.9). We’ll begin our guided tour of the brain with the part of the brain studied most extensively by psychologists.

THE CEREBRAL CORTEX. The cerebrum or forebrain, is the most highly developed area of the human brain. It gives us our advanced intellectual abilities—which explains why it’s of such keen interest to psychologists.

The cerebrum consists of two cerebral hemispheres (see Figure 3.10). These hemispheres look alike but serve somewhat different functions. Nevertheless, like two figure skaters in a pairs competition, they communicate and cooperate continually. The huge band of fibers connecting the corpus callosum, meaning “colossal body” in Latin, connects the two hemispheres and permits them to communicate (see Figure 3.10).

The largest component of the cerebrum is the cerebral cortex, which contains some 12 to 20 billion neurons. The cortex is the outermost part of the cerebrum. It’s aptly named, because cortex means “bark,” as the cortex surrounds the hemispheres much like bark on a tree. The cerebral cortex analyzes sensory information, helping us to perform complex brain functions, including reasoning and language.

The cortex contains four regions called lobes, each associated with somewhat different functions (see Figure 3.11). Each of our hemispheres contains the same four lobes.

---

**FIGURE 3.9** The Human Brain: A Simple Map. (Source: Modified from Dorling Kindersley)

**FIGURE 3.10** The Cerebral Hemispheres and the Corpus Callosum. The corpus callosum connects the two cerebral hemispheres.

**FIGURE 3.11** The Four Lobes of the Cerebral Cortex. The cerebral cortex consists of four interacting lobes: frontal, parietal, temporal, and occipital.

---

forebrain (cerebrum)
forward part of the brain that allows advanced intellectual abilities

cerebral hemispheres
two halves of the cerebral cortex, each of which serve distinct yet highly integrated functions

corpus callosum
large band of fibers connecting the two cerebral hemispheres

cerebral cortex
outermost part of forebrain, responsible for analyzing sensory processing and higher brain functions
Frontal Lobes. The frontal lobes lie in the forward part of the cerebral cortex. If you touch your forehead right now, your fingers are less than an inch away from your frontal lobes. The frontal lobes assist us in motor function (movement), language, and memory. They also oversee and organize most other brain functions, a process called executive functioning. Just as the U.S. president exerts control over the members of his (and surely one day, her) Cabinet, the brain’s executive function provides a kind of top-level governance over other cognitive functions.

In most people’s brains, a deep groove, called the central sulcus, separates the frontal lobe from the rest of the cortex. The motor cortex is the part of the frontal lobe that lies next to the central sulcus. We owe much of our knowledge of how the motor cortex works to Canadian neurosurgeon Wilder Penfield (1958), who applied mild electrical shocks to the motor cortex of patients who were awake during surgery for epilepsy (because the brain doesn’t contain pain receptors, one can accomplish this procedure without hurting patients). He elicited movements ranging from small muscle twitches to large and complex bodily movements. Penfield found that each part of the motor cortex controlled a specific part of the body, with regions requiring more precise motor control, like our fingers, consuming more cortical space (see Figure 3.12).

In front of the motor cortex lies a large expanse of the frontal lobe called the prefrontal cortex, which is responsible for thinking, planning, and language (see Figure 3.13). One region of the prefrontal cortex, Broca’s area, was named after French surgeon Paul Broca, who discovered that this site plays a key role in language production (Broca, 1861). Broca found that this site was damaged in many patients who were having trouble producing speech. His first patient with this strange condition, named “Tan” in the research literature, responded only with the word “Tan” when asked questions. It didn’t take long for Broca to recognize that brain damage in Tan and other patients with this speech disorder was almost always located in the left cerebral hemisphere. Many researchers have replicated this finding.

Frontal lobe
forward part of cerebral cortex responsible for motor function, language, memory, and planning

motor cortex
part of frontal lobe responsible for body movement

prefrontal cortex
part of frontal lobe responsible for thinking, planning, and language

Broca’s area
language area in the prefrontal cortex that helps to control speech production
The prefrontal cortex, which receives information from many other regions of the cerebral cortex, also contributes to mood, personality, and self-awareness (Chayer & Freedman, 2001; Fuster, 2000). The tragic story of Phineas Gage demonstrates how crucial the prefrontal cortex can be to personality.

Gage was a railroad foreman who experienced a horrific accident in 1848. His job was to build railroad tracks running through rural Vermont. Gage was performing his usual task of filling holes with gunpowder to break up stubborn rock formations. He was pressing gunpowder into one hole with a tamping iron when an explosion suddenly propelled the iron with great thrust through his head. The iron pierced Gage’s face under his cheekbone and destroyed much of his prefrontal cortex. Remarkably, Gage survived but he was never the same. His physician, J. M. Harlow (1848), describes Gage’s personality after the accident as

fitful, irreverent, indulging at times in the grossest proflanity (which was not previously his custom) . . . his mind was radically changed, so decidedly that his friends and acquaintances said he was “no longer Gage.”

Admittedly, we don’t know exactly what Gage was like before the accident, and some scholars have contended that his personality didn’t change as much as is often claimed (Macmillan, 2000). We do know more about the exact location of Gage’s brain damage, however. Hanna Damasio and her colleagues (1994) examined the skull of Phineas Gage with brain imaging techniques and confirmed that both the right and left sides of his prefrontal cortex were seriously damaged.

**Parietal Lobe.** The parietal lobe is the upper middle part of the cerebral cortex lying behind the frontal lobe (refer to Figure 3.11). The region of the parietal lobe lying just behind the central sulcus next to the motor cortex is the somatosensory cortex, which is sensitive to touch, including pressure and pain, and temperature (Figure 3.12). The parietal lobe helps us track objects’ locations (Nachev & Husain, 2006; Shomstein & Yantis, 2006), shapes, and orientations. It also helps us process others’ actions and represent numbers (Gobel & Rushworth, 2004). The parietal lobe communicates visual and touch information to the motor cortex every time we reach, grasp, and move our eyes (Culham & Valyear, 2006). Imagine that you ask your roommate to put a blank CD in your bookbag because you need to copy an assignment for him. You grab your bookbag, head off to school, and forget about it until you’re in the library sitting at the computer terminal and then you reach into your bag. What do you expect to feel? A CD or disk case, or maybe a soft sleeve? You’re probably not sure how, or even if, your roommate packaged the blank CD, but you can construct a mental image of the possibilities. So you can translate what your fingers feel into how the CD will look when you pull it out of your pocket. That’s a parietal lobe function.

**Temporal Lobe.** The temporal lobe is the prime site of hearing, understanding language, and storing memories of our past (look again at Figure 3.11). This lobe is separated from the rest of the cortex by a horizontal groove called the lateral fissure.

The top of the temporal lobe contains the auditory cortex, the part of the cortex devoted to hearing (see Chapter 4). The language area in the temporal lobe is called
Wernicke’s area, although this area also includes the lower parietal lobe (look again at Figure 3.13). It’s located slightly above and behind your left ear (unless you’re a lefty, in which case it might be above your right ear). Damage to Wernicke’s area results in severe difficulties with understanding speech. Moreover, patients with damage to this area tend to speak mostly in gibberish, probably because they don’t realize that the words coming out of their mouths don’t make sense. When asked whether his last name was “Brown,” one patient with damage to this area responded, “What it is here, then let me see. I just don’t know. No, I not going to eat any sigh, no.”

The lower part of the temporal lobe is critical to storing memories of autobiographical events (see Chapter 7). Penfield (1958) discovered that stimulating this region with electrical probes elicited memories, like vivid recollections of “a certain song” or “the view from a childhood window.” Yet psychologists today aren’t certain if stimulating the brain elicits genuine memories of past events or instead altered perceptions, making them closer to hallucinations (Schacter, 1996). Indeed, this alternative hypothesis is difficult to rule out.

Occipital Lobe. At the very back of our brain lies the occipital lobe, containing the visual cortex, dedicated to seeing. Compared with most animals, we human beings are highly dependent on our visual systems—we’ve even been called the “visual primate” (Angier, 2009)—so it stands to reason that we have an awful lot of cortical real estate devoted to seeing. Still, we’re by no means the only highly visual creatures. For each species, the amount of sensory cortex of each type is proportional to the degree to which it relies on that sense. Ghost bats depend highly on sound cues and have proportionally more auditory cortex; the platypus relies heavily on touch cues and has proportionally more touch cortex; and squirrels, like humans, rely strongly on visual inputs and have proportionally more visual cortex (Krubitzer & Kaas, 2005).

Cortical Hierarchies. When information from the outside world is transmitted by a particular sense (like sight, hearing, or touch), it reaches the primary sensory cortex specific to that sense (look at Figure 3.13 again). After the eye, ear, or skin transmits sense information to the primary sensory cortex, it’s passed on to another area for that sense called the association cortex, which is spread throughout all four of the brain’s lobes. The association cortex integrates information to perform more complex functions, such as pulling together size, shape, color, and location information to identify an object (see Chapter 4). The overall organization of the cortex is “hierarchical” because processing becomes increasingly complex as information is passed up the network.

THE BASAL GANGLIA. The basal ganglia are structures buried deep inside the cortex that help to control movement. Damage to the basal ganglia contributes to Parkinson’s disease,
resulting in a lack of control over movement and uncontrollable tremors. After sensory information reaches primary and association areas, it’s transmitted to the basal ganglia, which calculate a course of action and transmit it to the motor cortex.

The basal ganglia also allow us to perform movements to obtain rewards (Graybiel et al., 1994). When we anticipate a pleasurable outcome, such as a tasty sandwich or hot date, we depend on activity in our basal ganglia.

THE LIMBIC SYSTEM. The diverse parts of the brain dedicated to emotion are housed within the **limbic system** (Lambert, 2003; McClean, 1990), a set of highly interconnected brain regions. In contrast to the cortex, which processes information about external stimuli, the limbic system processes information about our internal states, such as blood pressure, heart rate, respiration, and perspiration, as well as our emotions. It’s the latter that we’ll focus on here.

We can think of the limbic system as the brain’s *emotional center* (see **FIGURE 3.14**). Limbic system structures also play roles in smell, motivation, and memory. The limbic system evolved out of the primitive olfactory system (dedicated to smell), that controlled various survival behaviors in early mammals. As anyone who’s walked a dog knows, smell remains vitally important to many animals.

We’ll explore four areas of the limbic system: the thalamus, the hypothalamus, the amygdala, and the hippocampus. Each area plays specific roles, although it cooperates with other regions. The term *thalamus* derives from the Greek word for bedroom or chamber. But the thalamus is more than one room, because it contains many areas, each of which connects to a specific region of the cerebral cortex. We can think of the thalamus as a sensory relay station. The vast majority of sensory information first passes through its doors, undergoing some initial processing, before traveling on to the cortex (refer again to Figure 3.14).

The *hypothalamus*, located on the floor of the brain, regulates and maintains constant internal bodily states. Different areas of the hypothalamus play various roles in emotion and motivation. Some play roles in regulating hunger, thirst, sexual motivation, or other emotional behaviors (see Chapter 11). The hypothalamus also helps control our body temperature, acting much like a thermostat that adjusts our home’s temperature in response to indoor changes in temperature.

The *amygdala* is named for its almond shape (*amygdala* is Greek for “almond”). Excitement, arousal, and fear are all part of its job description. The amygdala kicks into high gear when teenagers play violent video games (Mathews et al., 2006), or when we view fearful faces (Killgore & Yergelen-Todd, 2005). It also plays a key role in fear conditioning, a process by which animals, including humans, learn to predict when something scary is about to happen.

**FIGURE 3.14** The Limbic System. The limbic system consists mainly of the thalamus, hypothalamus, amygdala, and hippocampus. (Left art modified from Dorling Kindersley and right art from Kalat, 2007)
Ralph Adolphs and colleagues verified the role of the amygdala in fear in a 30-year-old woman whose left and right amygdalas were almost entirely destroyed by disease. Although she had no difficulty identifying faces, she was markedly impaired in detecting fear in these faces (Adolphs et al., 1994).

The **hippocampus** plays crucial roles in memory, especially spatial memory—the memory of the physical layout of things in our environment. When we make a mental map of how to get from one place to another, we’re using our hippocampus. This may explain why a portion of the hippocampus is larger in London taxi drivers than in non–taxi drivers and is especially large in experienced taxi drivers (Maguire et al., 2000). This correlation could mean either that people with greater amounts of experience navigating complex environments develop larger hippocampi or that people with larger hippocampi seek out occupations, like taxi driving, that rely on spatial navigation. One study that could help us figure out what’s causing what would be to examine whether cab drivers’ hippocampi become larger as they acquire more driving experience. Although researchers haven’t yet conducted this study, they’ve looked at this issue in people who’ve recently learned to juggle. Sure enough, they’ve found evidence for short-term increases in the size of the hippocampus, suggesting that this brain area can change in size in response to learning (Boyke et al., 2008).

Damage to the hippocampus causes problems with forming new memories, but leaves old memories intact (see Chapter 7). One hypothesis is that the hippocampus stores memories temporarily before transferring them to other sites, such as the cortex, for permanent storage (Sanchez-Andres, Olds, & Alkon, 1993). The **multiple trace theory** is a rival hypothesis of memory storage in the hippocampus (Moscovitch et al., 2005). According to this theory, memories are initially stored at multiple sites. Over time, storage becomes stronger at some sites but weaker at others. The multiple trace theory avoids the need to “transfer” memory from the hippocampus to the cortex. According to this model, memories are already stored in the cortex and merely strengthen over time.

**THE BRAIN STEM.** The **brain stem**, housed inside the cortex and located at the very back of our brains, contains the **midbrain**, **pons**, and the **medulla** (see **FIGURE 3.15**). The brain stem performs some of the basic bodily functions that keep us alive. It also serves as a relay station between the cortex and the rest of the nervous system. The **midbrain**, in turn, plays an important role in movement. It also controls the tracking of visual stimuli and reflexes triggered by sound, like jumping after we’re startled by a car backfiring.

**Reticular Activating System.** The **reticular activating system** (RAS) connects to the forebrain and cerebral cortex; this system plays a key role in arousal. Turn off a dog’s RAS, for example, and it instantly falls asleep. Damage to the RAS can result in a coma. Some scientists even believe that many knockdowns in boxing result from a temporary compression of the RAS following a powerful punch (Weisberg, Garcia, & Strub, 1996).

The pathways emanating from the RAS activate the cortex by jacking up the *signal-to-noise ratio* among neurons in the brain (Gu, 2002). When it’s working well, a cell phone produces sound with a high signal-to-noise ratio so that each caller can understand the other’s messages. When there’s a great deal of background static—resulting in a low signal-to-noise ratio—callers find it difficult to understand each other (see Chapter 4).

A possible example of this problem occurs in attention-deficit/hyperactivity disorder (ADHD), a disorder originating in childhood (see Chapter 15). ADHD is marked by inattention, overactivity, and impulsivity. Stimulant drugs used to treat ADHD, such as methylphenidate (often marketed under the brand name Ritalin), appear to increase the

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**FIGURE 3.15 The Brain Stem.** The brain stem is located at the top of the spinal cord, below the cortex.

**hippocampus**
part of the brain that plays a role in spatial memory

**brain stem**
part of the brain between the spinal cord and cerebral cortex that contains the midbrain, pons, and medulla

**midbrain**
part of the brain stem that contributes to movement, tracking of visual stimuli, and reflexes triggered by sound

**reticular activating system (RAS)**
brain area that plays a key role in arousal
signal-to-noise ratio in the prefrontal cortex (Devilbiss & Berridge, 2006). One hypothesis is that these drugs mimic activity in the RAS and neighboring brain regions, but other explanations are possible. For example, methylphenidate boosts levels of the neurotransmitter dopamine, which may be responsible for increases in attention and decreases in impulsivity (Volkow et al., 2005).

**The Cerebellum, Pons, and Medulla.** Below the midbrain lies the hindbrain, which consists of the cerebellum, pons, and medulla, the last two being part of the brain stem. *Cerebellum* is Latin for “little brain,” and in many respects the cerebellum is a miniature version of the cortex. The cerebellum plays a predominant role in our sense of balance and enables us to coordinate movement and learn motor skills. Among other things, it helps prevent us from falling down. But in recent years, scientists have come to realize that the cerebellum does more: It also contributes to executive, spatial, and linguistic abilities (Schmahmann, 2004). The pons, which as we’ll learn in Chapter 5 plays a crucial role in triggering dreams, connects the cortex to the cerebellum.

The medulla regulates breathing, heartbeat, and other vital functions. Damage to the medulla can cause brain death, which scientists define as irreversible coma. People who are brain dead are totally unaware of their surroundings and unresponsive, even to ordinarily very painful stimuli. They show no signs of spontaneous movement, respiration, or reflex activity.

People often confuse a persistent vegetative state, or cortical death, with brain death, but the two aren’t identical. Terri Schiavo made headlines in 2005 as the woman who had lain in a persistent vegetative state for 15 years. Schiavo collapsed in her Florida home in 1990 following temporary cardiac arrest, depriving her brain of oxygen and resulting in severe brain damage. The deep structures in her brain stem that control breathing, heart rate, digestion, and certain reflexive responses were still operating, so Schiavo wasn’t brain dead, as much of the news media reported incorrectly. Nevertheless, her higher cerebral structures, necessary for awareness of herself and her environment, were damaged permanently. Her doctors knew that much of her cortex had withered away, and an autopsy later showed that she’d lost about half of her brain.

Those who believe that death of the higher brain centers essential for consciousness is equivalent to actual death felt that Schiavo had, in fact, died 15 years earlier. Nevertheless, her death raises difficult and troubling questions that science can’t fully resolve: Should brain death be the true criterion for death, or should this criterion instead be the permanent loss of consciousness?

**THE SPINAL CORD.** The spinal cord extends from our brain stem and runs down the middle of our backs, conveying information between the brain and the rest of the body. Nerves extend from neurons to the body, traveling in two directions much like the traffic on a two-lane highway. Sensory information is carried from the body to the brain by way of sensory nerves; motor commands are carried from the brain to the body by way of motor nerves. The spinal cord also contains sensory neurons that contact interneurons, neurons that send messages to other neurons located nearby. Interneurons connect sensory nerves with motor nerves within the spinal cord without having to report back to the brain. Interneurons explain how reflexes, automatic motor responses to sensory stimuli, can occur.

Consider an automatic behavior called the stretch reflex, which relies only on the spinal cord. We’re carrying our books in our arms, but over time our grasp releases ever so slightly without our even noticing. Our sensory nerves detect the muscle stretch and relay this information to the spinal cord. Interneurons intervene and motor neurons automatically send messages that cause our arm muscles to contract. Without our ever knowing it, a simple reflex causes our arm muscles to tighten, preventing us from dropping our books (see **FIGURE 3.16**).

**The Peripheral Nervous System**

Thus far, we’ve examined the inner workings of the CNS—the central nervous system. Now let’s briefly examine the peripheral nervous system (PNS), the part of the nervous system consisting of the nerves that extend outside of the CNS. The PNS itself contains two branches, somatic and autonomic.
Chapter 3: Biological Psychology

**The Somatic Nervous System.** The somatic nervous system carries messages from the CNS to muscles throughout the body, controlling movement (look back to Figure 3.8). Whenever we stabilize or move our many joints, the CNS cooperates with the somatic nervous system to regulate our posture and bodily movement.

Let’s review what happens when we decide to stroll over to the vending machine to purchase a can of soda. Sensory inputs of all types reach the cortex. Then all parts of the cortex send information to the basal ganglia. The basal ganglia contribute to our decision about what to do and relay that information to the motor cortex. Next up, the motor cortex sends commands to the spinal cord, activating motor neurons. These motor neurons send messages through nerves that reach muscles throughout the body and trigger muscle contractions. We walk, reach, touch, and grasp. Our brain triggers all of these movements, but our somatic nervous system carries them out. After we finish our drink, our somatic nervous system keeps working, enabling us to walk away—ideally, to the nearest recycling container.

**The Autonomic Nervous System.** The brain and spinal cord interact with our somatic nervous system to bring about sensation and behavior. In much the same way, the brain, especially the limbic system, interacts with the autonomic nervous system to regulate emotion and internal physical states. The autonomic nervous system is the part of the nervous system that controls the involuntary actions of our organs and glands; along with the limbic system, it helps to regulate our emotions. The autonomic nervous system, in turn, consists of two divisions: sympathetic and parasympathetic (see Figure 3.17). These two divisions work in opposing directions, so that when one is active, the other is passive. The sympathetic nervous system is active during emotional arousal, especially during crises. This system mobilizes the **fight-or-flight response**, described by Walter Cannon in 1929 (see

**FIGURE 3.17** The Autonomic Nervous System (Female Shown). The sympathetic and parasympathetic divisions of the autonomic nervous system control the internal organs and glands.

**Somatic Nervous System**

part of the nervous system that conveys information between the CNS and the body, controlling and coordinating voluntary movement

**Autonomic Nervous System**

part of the nervous system controlling the involuntary actions of our internal organs and glands, which (along with the limbic system) participates in emotion regulation

**Sympathetic Division**

part of the autonomic nervous system engaged during a crisis or after actions requiring fight or flight
Chapter 12). Cannon noticed that when we encounter threats, like the sight of a huge predator charging toward us, our sympathetic nervous system becomes aroused and prepares us for fighting or fleeing. Sympathetic activation triggers a variety of physical responses helpful for reacting in a crisis, including increased heart rate (allowing more blood to flow into our extremities), respiration, and perspiration. Autonomic nerves that reach the heart, diaphragm, and sweat glands control these reactions. The parasympathetic nervous system, in contrast, is active during rest and digestion. This system kicks into gear when there’s no threat on our mental radar screens.

assess your knowledge

**FACT OR FICTION?**

1. The cortex is divided into the frontal, parietal, temporal, and hippocampal lobes. True / False
2. The basal ganglia control sensation. True / False
3. The amygdala plays a key role in fear. True / False
4. The cerebellum regulates only our sense of balance. True / False
5. There are two divisions of the autonomic nervous system. True / False

Answers:

1. F (p. 95);
2. F (p. 98);
3. T (p. 99);
4. F (p. 101);
5. T (p. 102)

**THE ENDOCRINE SYSTEM**

3.7 Describe what hormones are and how they affect behavior.

The limbic system also cooperates with the endocrine system to regulate emotion. The endocrine system is separate from, but interfaces with, the nervous system, and consists of glands that release hormones, molecules that influence particular organs, into the bloodstream (see **FIGURE 3.18**). Hormones differ from neurotransmitters in that they’re carried through our blood vessels rather than our nerves, so they’re much slower in their actions. We can think of hormonal messages as a bit like regular mail and neurotransmitter messages as a bit like e-mail. But hormones tend to outlast neurotransmitters in their effects, so their eventual impact tends to be more enduring.

**The Pituitary Gland and Pituitary Hormones**

The pituitary gland controls the other glands in the body; for this reason, it was once called the “master gland,” although scientists have now realized that it depends heavily on the actions of other glands, too. The pituitary gland, in turn, is under the control of the hypothalamus. The pituitary releases a variety of hormones that serve numerous functions, ranging all the way from regulating physical growth, controlling blood pressure, and determining how much water we retain in our kidneys. One pituitary hormone called oxytocin is responsible for a several reproductive functions, including stretching the cervix and vagina during birth and aiding milk flow in nursing mothers. Oxytocin also plays essential roles in maternal and romantic love (Esch & Stefano, 2005). Scientists have identified two closely related species of voles (a type of rodent) that differ in their pair bonding: The males of one species are promiscuous, flitting from attractive partner to another, whereas the males of the other remain faithfully devoted to one partner for life. Only in the brains of the loyal voles are oxytocin receptors linked to the dopamine system, which as we’ve learned influences the experience of reward (Young & Wang, 2004). For male voles, at least, remaining faithful isn’t a chore: It’s literally a labor of love. Oxytocin may also influence...
Although these two vole species (the prairie vole on the left and the montane vole on the right) look quite similar, they differ in their “personalities,” at least when it comes to romance. The male prairie vole stays loyal to one partner, but the male montane vole doesn’t. The difference lies in their oxytocin systems.

how much we trust others. In one study, men exposed to a nasal spray containing oxytocin were more likely than others to hand over money to their team partners in a risky investment game (Kosfeld et al., 2005; Rilling, King-Cassas, & Sanfey, 2008).

### The Adrenal Glands and Adrenaline

Psychologists sometimes call the **adrenal glands** the emergency centers of the body. Located atop of the kidneys, they manufacture the hormones **adrenaline** and **cortisol**. Adrenaline boosts energy production in muscle cells, thrusting them into action, while conserving as much energy as possible. Nerves of the sympathetic nervous system signal the adrenal glands to release adrenaline. Adrenaline triggers many actions, including (1) contraction of our heart muscle and constriction of our blood vessels to provide more blood to the body, (2) opening the bronchioles (tiny airways) of the lungs to allow inhalation of more air, (3) breakdown of fat into fatty acids, providing us with more fuel, (4) breakdown of glycogen (a carbohydrate) into glucose (a sugar) to energize our muscles, and (5) opening the pupils of our eyes to enable better sight during emergencies. Adrenaline also inhibits gastrointestinal secretions, explaining why we often lose our appetites when we feel nervous, as when anticipating a big job interview or final exam.

Adrenaline allows people to perform amazing feats in crisis situations, although these acts are constrained by people’s physical limitations. One desperate mother was energized to lift a heavy automobile to save her trapped infant (Solomon, 2002). She probably had evolution to thank, as natural selection has almost surely predisposed the sympathetic nervous system to react to dangerous stimuli to prepare us for counterattack (fight) or escape (flight). But adrenaline isn’t activated only during threatening situations. Pleasurable and exciting activities, like race car driving and skydiving, can also produce adrenaline rushes.

If this rhinoceros suddenly charged at the three people on this African safari, which branch of their autonomic nervous systems would (we hope!) become activated? (See answer upside down at bottom of page.)

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**adrenal gland**

Tissue located on top of the kidneys that releases adrenaline and cortisol during states of emotional arousal.
Like adrenaline, cortisol increases in response to physical and psychological stressors. Not surprisingly, some anxiety disorders are associated with elevated levels of cortisol (Mantello et al., 2008). Cortisol regulates blood pressure and cardiovascular function, as well as the body’s use of proteins, carbohydrates, and fats. The way in which cortisol regulates nutrients has led some researchers to suggest that it regulates body weight, leading to the development of the popular cortisol diet. Proponents of this diet claim that elevated cortisol produced by stress causes weight gain (Talbott, 2002). The solution: Reduce stress, increase exercise, and monitor nutrition—reasonable advice for those of us who want to lose weight. Some people want a quick fix, however, so health food supplement outlets are happy to oblige by selling cortisol blockers. Unfortunately, there’s little scientific evidence that these supplements work better than dieting measures that naturally inactivate the body’s cortisol.

**Sexual Reproductive Glands and Sex Hormones**

The sexual reproductive glands are the testes in males and ovaries in females (refer back to Figure 3.18). Most of us think of sex hormones as either male or female. After all, the testes make the male sex hormone, called testosterone, and the ovaries make the female sex hormone, called estrogen. Although testosterone is correlated with aggression, the interpretation of this association is controversial. Some authors have argued that a certain minimal level of testosterone is needed for humans and other animals to engage in aggression (Dabbs & Dabbs, 2000), but that above that level testosterone isn’t correlated with aggression. Moreover, above that level, these authors contend, aggressive behavior actually causes heightened testosterone rather than the other way around (Sapolsky, 1997).

Although males and females do have more of their own type of sex hormone, both sexes manufacture some amount of the sex hormone associated with the opposite sex. Women’s bodies produce about one-twentieth the amount of testosterone as those of males. That’s because the ovaries also make testosterone, and the adrenal gland makes low amounts of testosterone in both sexes. Conversely, the testes manufacture estrogen, but in low levels (Hess, 2003).

Scientists have long debated the relationship between sex hormones and sex drive (Bancroft, 2005). Most scientists believe that testosterone, which increases sex drive in men, also increases sex drive in women, but to a lesser degree. Australian researchers conducted a survey of 18- to 75-year-old women regarding their sexual arousal and frequency of orgasm (Davis et al., 2005). They found no correlation between the levels of male sex hormone in a woman’s blood and her sex drive. However, the study relied exclusively on self-reports and contained no controls for demand characteristics (see Chapter 2). Most researchers still accept the hypothesis that testosterone influences female sex drive, but additional research from multiple laboratories must be conducted before we can draw firm conclusions.

### FACT OR FICTION?

1. Hormones are more rapid in their actions than neurotransmitters. True / False
2. Adrenaline sometimes allows people to perform amazing physical feats. True / False
3. Cortisol tends to increase in response to stressors. True / False
4. Women have no testosterone. True / False

**FACTOID**

The thrill of watching others win can increase testosterone in sports fans. Males watching World Cup soccer matches showed increased testosterone levels in their saliva if their favorite team won, but decreased testosterone levels if their favorite team lost (Bernhardt et al., 1998).

**correlation vs. causation**

**CAN WE BE SURE THAT A CAUSES B?**

**replicability**

**CAN THE RESULTS BE DUPLICATED IN OTHER STUDIES?**

---

**assess your knowledge**

**FACT OR FICTION?**

1. Hormones are more rapid in their actions than neurotransmitters. True / False
2. Adrenaline sometimes allows people to perform amazing physical feats. True / False
3. Cortisol tends to increase in response to stressors. True / False
4. Women have no testosterone. True / False

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**Study and Review on mypsychlab.com**
Although many questions about the brain remain unanswered, we know far, far more about it today than we did 200, or even 20, years ago. For this, we owe psychologists and related scientists who’ve developed a host of methods to explore the brain and its functioning a major debt of gratitude.

## A Tour of Brain-Mapping Methods

Many advances over the past two centuries have enabled scientists to measure brain activity, resulting in a better understanding of how the most complicated organ in the known universe works. But brain research tools weren’t always grounded in solid science. Some of the earliest methods were fundamentally flawed, but they paved the way for the newer and improved methods used today.

### PHRENOLOGY: AN INCORRECT MAP OF THE MIND

Phrenology—sometimes jokingly called “bumpology”—was one of the first attempts to map mind onto brain. This theory was wildly popular in the 1800s, when phrenologists assessed enlargements of the skull—literally bumps on the head—and attributed various personality and intellectual characteristics to those who sought their “expertise.” Phrenologists assumed that bumps on the skull corresponded to brain enlargements, and that these brain enlargements were linked directly to psychological capacities. From the 1820s through the 1840s, thousands of phrenology shops popped up in Europe and North America. Anyone could go to a phrenology parlor to discover his or her psychological makeup. This popular practice was the origin of the familiar expression “having one’s head examined.”

The founder of phrenology, Viennese physician Franz Joseph Gall (1758–1828), began with some valid assumptions about the brain. He correctly predicted a positive relationship between enlargements in a handful of brain areas and certain traits and abilities, like language. Nevertheless, the up to 37 different traits that phrenologists described—aggressiveness, vanity, friendliness, and happiness among them—are vastly different from the functions scientists studying the brain today assign to different brain areas. What’s more, Gall and others based their hypotheses about the supposed associations between brain areas and personality traits almost entirely on anecdotal observations, which we’ve learned (see Chapter 1) are often subject to a host of errors.

Still, phrenology had one virtue: It was falsifiable. Ironically, this lone asset proved to be its undoing. Eventually, researchers discovered that patients with damage to specific brain areas didn’t experience the kinds of psychological deficits the phrenologists predicted. Even more critically, because the shape of the outer surface of the skull doesn’t closely match that of the underlying brain, phrenologists weren’t even measuring bumps on the brain, as they’d believed. These discoveries ultimately led to the demise of phrenology as an approach.

### BRAIN DAMAGE: UNDERSTANDING HOW THE BRAIN WORKS BY SEEING HOW IT DOESN’T

New methods quickly arose to fill the void left by phrenology. Foremost among them were methods of studying psychological functioning following damage to specific brain regions. We’ve already mentioned the pioneering work of Broca and others that linked specific areas of the cerebral cortex to specific functions. More recently, scientists have created lesions, that is, areas of damage, in experimental animals using stereotaxic methods, techniques that permit them to pinpoint the location of specific brain areas using coordinates, much like those navigators use on a map. Today, neuropsychologists rely on sophisticated psychological tests, like measures of reasoning, attention, and verbal and spatial ability, to infer the location of brain dysfunction in human patients. Neuropsychological tests, which require specialized training to administer, score, and interpret, include laboratory, computer-
ized, and paper-and-pencil measures designed to assess patients’ cognitive strengths and weaknesses (Lezak, Howieson, & Loring, 2004).

**ELECTRICAL STIMULATION AND RECORDING OF NERVOUS SYSTEM ACTIVITY.** Although early studies of function following brain damage provided valuable insights into which brain areas are responsible for which behaviors, many questions remained. Researchers soon discovered that stimulating parts of the human motor cortex in patients undergoing brain surgery produced extremely specific movements (Penfield, 1958). This finding, among others, led to the hypothesis that neurons use electrical activity to send information. But to test that hypothesis, scientists needed to record electrical activity from the nervous system.

To that end, Hans Berger (1929) developed the **electroencephalograph (EEG)**, a device—still widely used today—that measures electrical activity generated by the brain (see **FIGURE 3.19**). Patterns and sequences in the EEG allow scientists to infer whether a person is awake or asleep, dreaming or not, and to tell which regions of the brain are active during specific tasks. To obtain an EEG record, researchers record electrical activity from multiple electrodes placed on the scalp’s surface.

Because the EEG is noninvasive (that is, it doesn’t require us to penetrate bodily tissue), scientists frequently use it in both animal and human studies. EEGs can detect very rapid changes in the electrical activity of the brain occurring in the range of milliseconds (one-thousandths of seconds). Even today, researchers use EEGs to study brain activity in the brains of individuals with schizophrenia, epilepsy, and other psychiatric and neurological disorders as well as those without disorders. But EEGs have a few disadvantages. Because they show averaged neural activity that reaches the surface of the scalp, they tell us little, if anything, about what’s happening inside neurons. In this respect, interpreting EEGs is a bit like trying to understand the mental states of individual people in a stadium with 100,000 football fans by measuring how often they cheer, clap, or boo in response to plays on the field; we’ll certainly do better than chance, but we’ll make lots of mistakes too. EEGs also aren’t especially good for determining exactly where in the brain the activity is occurring.

**BRAIN SCANS.** Although electrical recording and stimulation provided the initial routes for mapping mind functions onto brain areas, a virtual revolution in brain research occurred with the advent of brain scans, or **neuroimaging**. As a group, these imaging methods enable us to peer inside the brain’s structure (that is, its appearance), its function (that is, its activity), and sometimes both.

**CT Scans and MRI Images.** In the mid-1970s, independent teams of researchers developed **computed tomography (CT)** and **magnetic resonance imaging (MRI)**, both of which allow us to visualize the brain’s structure (Hounsfield, 1973; Lauterbur, 1973). The CT scan is a three-dimensional reconstruction of multiple X-rays taken through a part of the body, such as the brain. As a result, it shows far more detail than an individual X-ray. The MRI shows structural detail using a different principle. The MRI scanner measures the release of energy from water in biological tissues following exposure to a magnetic field. MRI images are superior to CT scans for detecting soft tissues, such as brain tumors.

**PET.** CT and MRI scans show only the brain’s structure, not its activity. Therefore, neuroscientists interested in thought and emotion typically turn to **functional imaging** techniques like **positron emission tomography (PET)**, which measures changes in the brain’s...
activity in response to stimuli. PET relies on the fact that neurons, like other cells, increase their consumption of glucose (a sugar) when they’re active. We can think of glucose as the brain’s gasoline. PET requires the injection of radioactive glucose-like molecules into patients. Although they’re radioactive, they’re short-lived, so they do little or no harm. The scanner measures where in the brain most of these glucose-like molecules are consumed, allowing neuroscientists to figure out which brain regions are most active during a task. Clinicians can also use PET scans to see how brain activity changes when patients take a medication. Because PET is invasive, researchers continued to work to develop functional imaging methods that wouldn’t require injections of radioactive molecules.

**fMRI.** In 1990, researchers discovered that as neural activity quickens, there’s an increase in oxygenated blood in response to heightened demand (Ogawa et al., 1990). The discovery of this response, known as the **blood oxygenation level dependent** (BOLD) response, enabled the development of the **functional MRI (fMRI)**. Because fMRI measures the change in blood oxygen level, it’s an indirect correlate of neural activity. Neuroscientists frequently use fMRI to image brain activity in response to specific tasks, like looking at emotional faces or solving math problems (Marsh et al., 2008). The fMRI relies on magnetic fields, as does MRI. fMRI’s strength, especially compared with PET, is its ability to provide detailed images of activity in small brain regions and over brief time intervals. Nevertheless, in contrast to PET and some other imaging techniques, fMRI is extremely sensitive to motion, so researchers often have to toss out fMRI data if participants move too much.

**MAGNETIC STIMULATION AND RECORDING.** Transcranial magnetic stimulation (TMS) applies strong and quickly changing magnetic fields to the skull to create electric fields in the brain. Depending on the level of stimulation, TMS can either enhance or interrupt brain function in a specific region. TMS offers useful insights regarding which brain areas are involved in different psychological processes. For example, if TMS interrupts functioning in the temporal lobe and the subject displays (temporary!) language impairment as a result, we can conclude that the temporal lobe is important for language processing. Because it allows us to manipulate brain areas directly, TMS is the only noninvasive brain imaging technique that allows us to infer causation—all other techniques can only correlate brain activation with psychological processing. Some reports suggest that TMS provides relief for depression and may decrease auditory hallucinations, that is, the hearing of sounds, typically voices (Saba, Schurhoff, & Leboyer, 2006). Repetitive TMS (rTMS) also shows promise as a treatment for depression (Rachid & Bertschy, 2006).

A final imaging technique is **magnetoencephalography (MEG)**, which detects electrical activity in the brain by measuring tiny magnetic fields (Vrba & Robinson, 2001). In this way, MEG reveals patterns of magnetic fields on the skull’s surface, thereby revealing which brain areas are becoming active in response to stimuli. MEG’s strength is its ability to track brain changes over extremely small time intervals. In contrast to PET and fMRI scans, which measure activity changes second by second, MEG measures activity changes millisecond by millisecond.

**How to Interpret—and Misinterpret—Brain Scans.** PET, fMRI, and other functional brain imaging techniques have taught us a great deal about how the brain’s activity changes in response to different stimuli. They’ve also helped scientists to uncover deficits in the brain functioning of people with certain psychiatric disorders. For example, they’ve revealed that schizophrenia, a severe disorder of thought and emotion marked by a loss of contact with reality, is often associated with underactivity of the frontal lobes (Andreasen et al., 1997; see Chapter 15).

Yet it’s extremely easy to misinterpret brain scans, largely because many laypersons and even newspaper reporters hold misunderstandings of how they work (Racine, Barllan, & Illes, 2006). For one thing, many people assume that functional brain images, like the mul-
Ticlor images generated by PET and fMRI scans, are like photographs of the brain in action (Roskies, 2007). They aren’t. In most cases, these images are produced by subtracting brain activity on a “control” task from brain activity on an “experimental” task, which is of primary interest to the researchers. For example, if researchers wanted to find out how people with clinical depression process sad faces, they could subtract the brain’s activity following neutral faces from its activity following sad faces. So although we’re seeing one image, it’s actually one image subtracted from another. Moreover, the pretty colors in these images are arbitrary and superimposed by researchers. They don’t correspond directly to the brain’s activity (Shermer, 2008). Making matters more complicated, when a brain area “lights up” on a brain scan, we know only that neurons in that region are becoming more active. They might actually be inhibiting other neurons rather than exciting them.

Another complexity is introduced by the fact that when researchers conduct the calculations that go into brain scans, they’re typically comparing the activity of hundreds of brain areas across neutral versus experimental tasks (Vul et al., 2009). As a result, there’s a risk of chance findings—those that won’t replicate in later studies. To make this point, one mischievous team of researchers (Bennett et al., 2009) placed a dead salmon in a brain scanner, flashed it photographs of people in social situations, and asked the salmon to guess which emotions the people were experiencing (no, we’re not making this up). Remarkably, the investigators “found” an area in the salmon’s brain that became active in response to the task. In reality, of course, this activation was just a statistical artifact, a result of the fact that they’d computed so many analyses that a few were likely to be statistically significant (see Chapter 2) by chance. This finding is a needed reminder that we should view many brain imaging findings with a bit of caution until other investigators have replicated them.

**How Much of Our Brain Do We Use?**

Despite having so much information available today regarding the relationship between brain and behavior, scores of misconceptions about the brain abound. One widely held myth is that most of us use only 10 percent of our brain (Beyerstein, 1999). What could we do if we could access the other 90 percent? Would we find the cure for cancer, acquire great wealth, or write our own psychology textbook?

The 10-percent myth gained its toehold at around the same time as phrenology, in the late 1800s. William James (1842–1910), one of the fathers of psychology (see Chapter 1), wrote that most people fulfill only a small percent of their intellectual potential. Some people misconstrued James to mean that we only use about 10 percent of our brain. As the 10-percent myth was repeated, it acquired the status of an urban legend (see Chapter 13).

Early difficulties in identifying which brain regions controlled which functions probably reinforced this misconception. In 1929, Karl Lashley showed that there was no single memory area in the brain (see Chapter 7). He made multiple knife cuts in the brains of rats and tested them on mazes. He found that no specific cortical area was more critical to maze learning than any other. Lashley’s results were ripe for misinterpretation.
as evidence for “silent” areas in the cerebral cortex—those that presumably did nothing. In fact, we know today that these supposedly silent areas comprise much of the association cortex, which as we’ve already learned serves invaluable functions. Given how appealing the idea of tapping into our full potential is, it’s no wonder that scores of pop psychology writers and so-called self-improvement experts have assured us they know how to harness our brain’s full potential. Some authors of self-help books who were particularly fond of the 10-percent myth liberally misquoted scientists as saying that 90 percent of the brain isn’t doing anything. Believers in psychic phenomena have even spun the fanciful story that because scientists don’t know what 90 percent of the brain is doing, it must be serving a psychic purpose, like extrasensory perception (ESP) (Clark, 1997).

Today, we now know enough about the brain that we can safely conclude that every brain region has a function. Specialists in clinical neurology and neuropsychology, who deal with the effects of brain damage, have shown that losses of even small areas of certain parts of the brain can cause devastating, often permanent, losses of function (Sacks, 1985). Even when brain damage doesn’t cause severe deficits, it produces some change in behavior, however subtle.

The fatal blow against the 10-percent myth, however, finally came from neuroimaging and brain stimulation studies. No one’s ever discovered any perpetually silent areas, nor is it the case that 90 percent of the brain produces nothing of psychological interest when stimulated. All brain areas become active on brain scans at one time or another as we think, feel, and perceive (Beyerstein, 1999).

Which Parts of Our Brain Do We Use for What?

Scientists refer to localization of function when they identify brain areas that are active during a specific psychological task over and above a baseline rate of activity. We should be careful not to overemphasize localization of function, though, and we need to be especially cautious in our interpretations of neuroimaging results. William Uttal (2001) warned that researchers are too quick to assign narrowly defined functions to specific brain regions. He pointed out that we can’t always dissect higher brain functions into narrower components. Take visual perception, for example: Can we divide it into neat and tidy subcomponents dealing with color, form, and motion, as the cortical localization of functions might imply, or is visual perception a unified experience supported by multiple regions? It’s almost certainly the latter.

Regrettably, much of the popular media hasn’t taken Uttal’s useful cautions to heart. On a virtually weekly basis, we’ll encounter news headlines like “Alcoholism Center in Brain Located” or “Brain Basis of Jealousy Found” (Cacioppo et al., 2003). To take another example, in the late 1990s and as recently as 2009, some newspapers announced the discovery of a “God spot” in the brain when scientists found that certain areas of the frontal lobes become active when individuals think of God. Yet most brain imaging research shows that religious experiences activate a wide variety of brain areas, not just one (Beauregard & Paquette, 2006). As Uttal reminds us, few if any complex psychological functions are likely to be confined to a single brain area.

Just as multiple brain regions contribute to each psychological function, individual brain areas contribute to multiple psychological functions. Broca’s area, well known to play a role in speech, also becomes active when we notice that a musical note is off key (Limb, 2006). There’s enhanced activity in the amygdala and other limbic regions when we listen to inspiring music, even though these regions aren’t traditionally known as “musical areas” (Blood & Zatorre, 2001). The rule of thumb is that each brain region participates in many functions—some expected, some unexpected—so coordination across multiple brain regions contributes to each function.

Which Side of Our Brain Do We Use for What?

As we’ve learned, the cerebral cortex consists of two hemispheres, which are connected largely by the corpus callosum. Although they work together closely to coordinate functions, each hemisphere serves different functions. Many functions rely on one cerebral hemisphere for processing, while other functions rely on the other hemisphere.
hemisphere more than the other; scientists call this phenomenon lateralization (see TABLE 3.3). Many lateralized functions concern specific language and verbal skills.

Roger Sperry (1974) won the Nobel Prize for showing that the two hemispheres serve different functions, such as different levels of language ability. His remarkable studies examined patients who underwent split-brain surgery because their doctors couldn’t control their epilepsy with medication. In this exceedingly rare operation, neurosurgeons separate a patient’s hemispheres by severing the corpus callosum. Split-brain surgery typically offers relief from seizures, and patients behave normally under most conditions.

Nevertheless, carefully designed studies reveal surprising deficits in split-brain patients. Specifically, they experience a bizarre fragmenting of mental functions that we normally experience as integrated. Putting it a bit differently, the two hemispheres of split-brain subjects display somewhat different abilities, even though these individuals experience themselves as unified persons (Gazzaniga, 2000; Zaidel, 1994).

Here’s what Sperry and his colleagues did. They presented stimuli, such as written words, to either patients’ right or left visual field. The right visual field is the right half of information entering each eye, and the left visual field is the left half of information entering each eye. To understand why researchers present stimuli to only one visual field, we need to know that in normal brains most visual information from either the left or right visual field ends up on the opposite side of the visual cortex. The brain’s design also results in crossing over for motor control: The left hemisphere controls the right hand, the right hemisphere controls the left hand.

Because corpus callosum transfers information between the two hemispheres, cutting it prevents most visual information in each visual field from reaching the visual cortex on the same side. As a consequence, we often see a stunning separation of functions. In one extreme case, a split-brain subject complained that his left hand wouldn’t cooperate with his right hand. His left hand misbehaved frequently; it turned off TV shows while he was in the middle of watching them and frequently hit family members against his will (Joseph, 1988).

Split-brain subjects often experience difficulties integrating information presented to separate hemispheres, but find a way to explain away or make sense of their bewildering behaviors. In one study, researchers flashed a chicken claw to a split-brain patient’s left hemisphere and a snow scene to his right hemisphere (see FIGURE 3.20). When asked to match what he saw with a set of choices, he pointed to a shovel with his left hand (controlled by his right hemisphere) but said “chicken” (because speech is controlled by his left hemisphere). When asked to explain these actions, he said, “I saw a claw and I picked the chicken, and you have to clean out the chicken shed with a shovel.”

<table>
<thead>
<tr>
<th>TABLE 3.3 Lateralized Functions</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>LEFT HEMISPHERE</strong></td>
</tr>
<tr>
<td>Fine-tuned language skills</td>
</tr>
<tr>
<td>• Speech comprehension</td>
</tr>
<tr>
<td>• Speech production</td>
</tr>
<tr>
<td>• Phonology</td>
</tr>
<tr>
<td>• Syntax</td>
</tr>
<tr>
<td>• Reading</td>
</tr>
<tr>
<td>• Writing</td>
</tr>
<tr>
<td>Actions</td>
</tr>
<tr>
<td>• Making facial expressions</td>
</tr>
<tr>
<td>• Motion detection</td>
</tr>
<tr>
<td><strong>RIGHT HEMISPHERE</strong></td>
</tr>
<tr>
<td>Coarse language skills</td>
</tr>
<tr>
<td>• Simple speech</td>
</tr>
<tr>
<td>• Simple writing</td>
</tr>
<tr>
<td>• Tone of voice</td>
</tr>
<tr>
<td>Visuospatial skills</td>
</tr>
<tr>
<td>• Perceptual grouping</td>
</tr>
<tr>
<td>• Face perception</td>
</tr>
</tbody>
</table>

(Source: Adapted from Gazzaniga, 2000)

**FIGURE 3.20 Split-Brain Subject.** This woman’s right hemisphere recognizes the snow scene and leads her to point to the shovel, but her left hemisphere recognizes the claw and indicates verbally that the chicken is the matching object.

This man has suffered a stroke that affected the right side of his face. On what side of his brain did his stroke probably occur? And why? (See answer upside down on bottom of page.)

**Legend**
- lateralization: cognitive function that relies more on one side of the brain than the other
- split-brain surgery: procedure that involves severing the corpus callosum to reduce the spread of epileptic seizures
Still, we must guard against taking lateralization of function to an extreme. Remarkably, it’s possible to live with only half a brain, that is, only one hemisphere. Indeed, a number of people have survived operations to remove one hemisphere to spare the brain from serious disease. Their outlook is best when surgeons perform the operation in childhood, which gives the remaining hemisphere a better chance to assume the functions of the missing hemisphere (Kenneally, 2006). The fact that many children who undergo this procedure develop almost normally suggests that functional localization isn’t a foregone conclusion.

**ARE THERE LEFT-BRAINED VERSUS RIGHT-BRAINED PERSONS?**

Despite the great scientific contribution of split-brain studies, the popular notion that normal people are either “left-brained” or “right-brained” is a misconception. According to this myth, left-brained people are scholarly, logical, and analytical, and right-brained people are artistic, creative, and emotional. One Internet blogger tried to explain the differences between people’s political beliefs in terms of the left–right brain distinction; conservatives, he claimed, tend to be left-brained and liberals right-brained (Block, 2006). Yet these claims are vast oversimplifications of a small nugget of truth, because research demonstrates that we use both sides of our brain in a complementary way (Corballis, 1999; Hines, 1987). Furthermore, the corpus callosum and other interconnections ensure that both hemispheres are in continual communication.

We can trace the myth of exaggerated left brain versus right brain differences to misinterpretations of accurate science. Self-help books incorporating the topic have flourished. Robert E. Ornstein was among those to promote the idea of using different ways to tap into our creative right brains versus our intellectual left brains in his 1997 book *The Right Mind: Making Sense of the Hemispheres*. Right brain–oriented educational programs for children sprang up that deemphasized getting the correct answers on tests in favor of developing creative ability. Such programs as the “Applied Creative Thinking Workshop” trained business managers to use their right brain (Herrmann, 1996). For a mere $195, “whole brain learning” supposedly expanded the mind in new ways using “megabliminal messages,” heard only by the left or the right brain (Corballis, 1999). Although there’s nothing wrong with trying to be more creative by using our minds in different ways, using both hemispheres in tandem works far better.

Supposedly, we can also use left-brain, right-brain differences to treat mood disorders or anger. There are even sunglasses with flip-up side panels designed to selectively increase light to either the left or right hemisphere. Nevertheless, there’s little or no scientific support for “goggle therapy” (Lilienfeld, 1999a). The magazine *Consumer Reports* (2006) couldn’t confirm the claim that the sunglasses reduced anger or other negative feelings, with seven out of 12 subjects reporting no change. Surely, more evidence is required before we can interpret an extraordinary claim of this kind as scientifically supported.
DIAGNOSING YOUR BRAIN ORIENTATION

Many online quizzes claim to identify you as either “left-brained” or “right-brained” based on which direction you see an image move, whether you can find an image hidden in an ambiguous photo, or your answers to a series of multiple-choice questions. Other websites and books claim to help you improve your brain’s nondominant side. Let’s evaluate some of these claims, which are modeled after actual tests and products related to brain lateralization.

“Left-brained people are more likely to focus on details and logic and to follow rules and schedules. They do well in math and science. Right-brained people are more likely to be deep thinkers or dreamers, and to act more spontaneously. They excel in the social sciences and the arts.”

The ad implies incorrectly that some people are left-brained and others right-brained, when in fact the left and right hemispheres differ only in emphasis.

NATURE AND NURTURE: DID YOUR GENES—OR PARENTS—MAKE YOU DO IT?

3.10 Describe genes and how they influence psychological traits.
3.11 Explain the concept of heritability and the misconceptions surrounding it.

Up to this point in the chapter, we’ve said relatively little about how what influences shape the development of our brains. Our nervous system, of course, is shaped by both our genes (nature) and our environments (nurture)—everything that affects us after fertilization. But how do nature and nurture operate to shape our physiological, and ultimately our psychological, makeup?

How We Come to be Who We Are

As little as 150 years ago, even the smartest of scientists knew almost nothing about how we humans come to be. Today, the average educated person knows more about the origins of human life and the human brain than did Charles Darwin. We’re remarkably fortunate to be armed with scientific principles concerning heredity, adaptation, and evolution that enable us to understand the origins of many of our psychological characteristics.

THE BIOLOGICAL MATERIAL OF HEREDITY. In 1866, a monk named Gregor Mendel published his classic treatise on inheritance based on his research on pea plants, but Mendel didn’t understand how the characteristics of these plants, like their height, shape, and color, were transmitted across generations. We now know that both plants and animals possess chromosomes (see FIGURE 3.21), slender threads inside the cell’s nucleus that carry genes, the
organisms’ capacity to pass on their genes

fitness

Genes, in turn, are composed of deoxyribonucleic acid (DNA), a remarkable substance shaped like a double helix that stores everything cells need to replicate (reproduce) themselves (see FIGURE 3.22). The genome consists of a full set of chromosomes and the heritable traits associated with them. The monumental Human Genome Project, which characterized all human genes, was completed in 2001. This project has garnered enormous attention and stirred great hopes, as it holds out the promise of treating—and perhaps one day curing—many human disorders, including mental disorders influenced by genes (Plomin & Crabbe, 2000).

FIGURE 3.22 Genetic Expression. The nucleus of the neuron houses chromosomes, which contain strands of DNA. They store codes for constructing proteins needed by the cell.

Our genetic makeup, the set of genes transmitted from our parents to us, is our genotype. In contrast, our phenotype is our set of observable traits. We can’t easily infer people’s genotypes by observing their phenotypes because some genes are dominant, meaning they mask other genes’ effects. In contrast, other genes are recessive, meaning they’re expressed only in the absence of a dominant gene.

Eye, hair, and even skin color are influenced by combinations of recessive and dominant genes. For example, two brown-eyed parents could have a blue-eyed child because the child inherited recessive genes for blue eyes from both parents.

BEHAVIORAL ADAPTATION. Charles Darwin’s classic book On the Origin of Species (1859) introduced the broad brush strokes of his theory of evolution by natural selection (see Chapter 1). Darwin hypothesized that populations of organisms change over time by selective breeding among individuals within the population who possess an adaptive advantage. According to these principles, some organisms possess adaptations that make them better suited to their environments. They survive and reproduce at higher rates than other organisms. Many adaptations are physical changes that enable organisms to better adjust to or manipulate their environments. An opposable thumb—one that can be moved away from the other fingers—for example, greatly enhanced our hand function. Compared with other organisms, those with successful adaptations have heightened levels of fitness, meaning they have a better chance of passing on their genes to later generations.

Other adaptations are behavioral. Indeed, the field of evolutionary psychology (Chapter 1) examines the potential adaptive functions of psychological traits (Buss, 1995). According to most evolutionary psychologists, aggressive behavior is an adaptation, because it enables organisms to obtain more resources. Too much aggression, however, is usually maladaptive, meaning it often decreases organisms’ chances of survival or reproduction, perhaps because they’re likely to be killed in fights or because their aggression scares off potential mates. But evolutionary psychology is controversial, largely because it’s difficult to know whether a psychological trait is a direct product of natural selection (Panksepp & Panksepp, 2000). In contrast to bones and some other physical characteristics, psychological traits don’t leave fossils, so we need to make educated guesses about these traits’ past adaptive functions. For example, is religion an evolutionary adaptation, perhaps because it helps us to cement social ties? It’s difficult to know (Boyer, 2003). Or what about morality, jealousy, artistic ability, and scores of other psychological traits? In all of these cases, we may never know whether they’re direct products of natural selection as opposed to indirect byproducts of other traits that have been selected. Nevertheless, it’s likely that some psychological characteristics, like anxiety, disgust, happiness, and other emotions are adaptations that prepare organisms to react to certain stimuli (Nesse & Elsworth, 2009). Anxiety, for example, predisposes us to attend to potential threats, like predators (see Chapters 11 and 15).

HUMAN BRAIN EVOLUTION. The relationship between the human nervous system and behavior has been finely tuned over millions of years of evolution (Cartwright, 2000). Brain regions with complicated functions, such as the cortex, have evolved the most (Karlen & Krubitzer, 2006). As a result, our behaviors are more complex and
flexible than those of other animals, allowing us to respond in many more ways to a
given situation.

What makes us so distinctive in the animal kingdom? Fossil and genetic evidence
suggests that somewhere between six and seven million years ago, humans and apes split off
from a shared ancestor. After that critical fork in the evolutionary road, we went our separate ways. The human line eventually resulted in our species, *Homo sapiens*, whereas the
ape line resulted in chimpanzees, gorillas, and orangutans (the “great apes”). We often fail
to appreciate that *Homo sapiens* have been around for only about one percent of the total
time period of the human race (Calvin, 2004).

Around the time of our divergence from apes, our brains weren’t that much larger
than theirs. Then, around three to four million years ago, something dramatic happened, although we don’t know why. We do know that within a span of only a few million years—a mere blink of an eye in the earth’s 4.5-billion-year history—one tiny area of the human genome changed about 70 times more rapidly than other areas, resulting in significant changes in the cortex (Pollard et al., 2006). The human brain mushroomed in size, more
than tripling from less than 400 grams—a bit less than a pound—to its present hefty
weight of 1,300 grams—about three pounds (Holloway, 1983). The brains of modern great apes weigh between 300 and 500 grams, even though their overall body size doesn’t differ
that much from humans’ (Bradbury, 2005).

Relative to our body size, we’re proportionally the biggest-brained animals (we
need to correct for body size, because large animals, like whales and elephants, have huge
brains in part because their bodies are also huge). Second in line are dolphins (Marino,
McShea, & Uhen, 2004), followed by chimpanzees and other great apes. Research suggests
that across species, relative brain size—brain size corrected for body size—is associated
with behaviors we typically regard as intelligent (Jerison, 1983). For example, big-brained
animals tend to have especially large and complex social networks (Dunbar, 2003).

**Behavioral Genetics: How We Study Heritability**

Scientists use *behavioral genetics* to examine the influence of nature and nurture on psychological traits, such as intelligence (see Chapter 9). In reality, behavioral genetic designs are
mismarked, because they permit us to look at the roles of both genes and environment in
behavior (Waldman, 2005).

Behavioral genetic designs also allow us to estimate the *heritability* of traits and
diseases. By heritability, we mean the extent to which genes contribute to differences in a
trait among individuals. Typically, we express heritability as a percentage. So, if the heri-
tability of a trait is 60 percent, that means that more than half of the differences among individuals
in their levels of that trait are due to differences in their genes. By definition, the
other 40 percent is due to differences in their environments. Some traits, like height, are
highly heritable; The heritability of height in adults is between 70 and 80 percent (Silven-
toinen et al., 2003). In contrast, other traits, like religious affiliation (which religion we
choose), are due almost entirely to environment and therefore have a heritability of about zero. Our religious affiliation, not surprisingly, is influenced substantially by the beliefs
with which we were raised. Interestingly, though, religiosity, the depth of our religious be-
lief, is moderately heritable (Turkheimer, 1998), perhaps because it stems partly from per-
sonality traits are themselves heritable (see Chapter 14).

**THREE MAJOR MISCONCEPTIONS ABOUT HERITABILITY.** Heritability isn’t as simple a
concept as it appears, and it confuses even some psychologists. So before discussing how
psychologists use heritability in different studies, we’ll first address three misunderstand-
ings about what heritability is—and isn’t:

- **Misconception #1:** Heritability applies to a single individual rather than to dif-
  ferences among individuals. Heritability applies only to groups of people. If someone asks you, “What’s the heritability of your IQ?” you should promptly
  hand that person a copy of this chapter. Heritability tells us about the causes of
differences among people, not within a person.
• **Misconception #2: Heritability tells us whether a trait can be changed.** Many people believe that if a trait is highly heritable, then by definition we can't change it. Yet heritability technically says little or nothing about how malleable (alterable) a trait is. A trait can in principle have a heritability of 100 percent and still be extremely malleable. Imagine 10 plants that differ markedly in height, with some of them only two or three inches tall and others five or six inches tall. Further imagine that they're only a few days old and that since their germination we've exposed them to exactly equal environmental conditions: the same amount of water and identical soil and lighting conditions. What's the heritability of height in this group of plants? It's 100 percent: The causes of differences in their heights must be completely genetic, because we've kept all environmental influences constant. Now imagine we suddenly decide to stop watering these plants and providing them with light. All of the plants will soon die, and their heights will become zero inches. So, even though the heritability of height in these plants was 100 percent, we can easily change their heights by changing their environments.

Behavioral geneticists refer to reaction range as the extent to which genes set limits on how much a trait can change in response to new environments (Gottlieb, 2003; Platt & Sanislow, 1988). Eye color has a limited reaction range, because it won’t change much over our lifetimes, even in the presence of radical environmental changes. In contrast, at least some genetically influenced psychological traits, like intelligence, probably have a larger reaction range, because they can change—in either a positive or negative direction—in response to environmental changes, like early enrichment or early deprivation. As we'll learn in Chapter 9, however, the true reaction range of intelligence is unknown.

• **Misconception #3: Heritability is a fixed number.** Heritability can differ dramatically across different time periods and populations. Remember that heritability is the extent to which differences among people in a trait are due to genetic influences. So if we reduce the range of environmental influences on a trait within a population, the heritability of that trait will increase because more of the differences in that trait will be due to genetic factors. Conversely, if we increase the range of environmental influences on a trait within a population, heritability will go down because fewer of the differences in that trait will be due to genetic factors.

**BEHAVIORAL GENETIC DESIGNS.** Scientists estimate heritability using one of three behavioral genetic designs: *family studies*, *twin studies*, and *adoption studies*. In such studies, scientists track the presence or absence of a trait among different relatives. These studies help them determine how much both genes and environment contribute to that trait.

**Family Studies.** In *family studies*, researchers examine the extent to which a characteristic “runs” or goes together in intact families, namely, those in which all family members are raised in the same home. This information can be useful for estimating the risk of a disorder among the relatives of people afflicted with that disorder. Nevertheless, family studies have crucial drawback: Relatives share a similar environment as well as similar genetic material. As a consequence, family studies don’t allow us to disentangle the effects of nature from nurture. Investigators have therefore turned to more informative research designs to separate these influences and rule out alternative hypotheses about the effects of genes versus environments.

**Twin Studies.** To understand *twin studies*, most of which examine differences between identical and fraternal twins in traits, we first need to say a bit about the birds and the bees. Two different things can happen when a sperm fertilizes an egg. First, a single sperm may fertilize a single egg, producing a *zygote*, or fertilized egg (see Chapter 10). For reasons that scientists still don’t fully understand, that zygote occasionally (in about one in 250 births) splits into two, yielding two identical genetic copies. Researchers refer to these identical twins as *monozygotic* (MZ), because they originate from one zygote. Identical twins are essentially genetic clones of each other because they share 100 percent of their...
genes. In other cases, two different sperm may fertilize two different eggs, resulting in two zygotes. These twins are dizygotic (DZ), or, more loosely, fraternal. In contrast to identical twins, fraternal twins share only 50 percent of their genes on average and are no more alike genetically than ordinary brothers or sisters. Fraternal twins (and triplets, quadruplets, and so on) are more likely to occur in women undergoing fertility treatments to encourage eggs to be produced and released. But fertility treatments have no effect on the frequency of identical twins, because they don’t affect whether a single egg will split.

The logic of twin studies rests on the fact that identical twins are more similar genetically than are fraternal twins. Consequently, if identical twins are more alike on a psychological characteristic, such as intelligence or extraversion, than are fraternal twins, we can infer that this characteristic is genetically influenced, assuming the environmental influences on the characteristic we’re studying are the same in identical and fraternal twins (Kendler et al., 1993).

Adoption Studies. As we’ve noted, studies of intact family members are limited because they can’t disentangle genetic from environmental influences. To address this shortcoming, psychologists have turned to adoption studies, which examine the extent to which children adopted into new homes resemble their adoptive as opposed to their biological parents. Children adopted into other homes share genes, but not environment, with their biological relatives. As a consequence, if adopted children resemble their biological parents on a psychological characteristic, we can typically assume it’s genetically influenced.

One potential confound in adoption studies is selective placement: Adoption agencies frequently place children in homes similar to those of their biological parents (DeFries & Plomin, 1978). This confound can lead investigators to mistakenly interpret the similarity between adoptive children and their biological parents as a genetic effect. In adoption studies, researchers try to control for selective placement by correcting statistically for the correlation between biological and adoptive parents in their psychological characteristics.

As we’ll discover in later chapters, psychologists have come to appreciate that genetic and environmental influences intersect in complex ways to shape our nervous systems, thoughts, feelings, and behaviors. For example, they’ve learned that people with certain genetic makeups tend to seek out certain environments (Plomin, DeFries, & McClearn, 1977) and react differently than people with other genetic makeups to certain environments (Kim-Cohen et al., 2006; see Chapter 10). They’ve also learned that many environmental influences, like life stressors and maternal affection, actually work in part by turning certain genes on or off (Weaver et al., 2004). Nature and nurture, although different sources of psychological influence, are turning out to be far more intertwined than we’d realized.

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**assess your knowledge**

<table>
<thead>
<tr>
<th>FACT OR FICTION?</th>
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<tbody>
<tr>
<td><strong>1.</strong> Brain evolution is responsible for humans’ advanced abilities. True / False</td>
</tr>
<tr>
<td><strong>2.</strong> The fact that the human brain is smaller than an elephant’s shows that brain size is unrelated to intelligence. True / False</td>
</tr>
<tr>
<td><strong>3.</strong> Heritability values can’t change over time within a population. True / False</td>
</tr>
<tr>
<td><strong>4.</strong> Identical twins have similar phenotypes (observable traits) but may have different genotypes (sets of genes). True / False</td>
</tr>
<tr>
<td><strong>5.</strong> Adoption studies are useful for distinguishing nature influences from nurture influences. True / False</td>
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</tbody>
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**Study and Review on mypsychlab.com**

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NERVE CELLS: COMMUNICATION PORTALS 84–93

3.1 DISTINGUISH THE PARTS OF NEURONS AND WHAT THEY DO.
The neuron has a cell body, which contains a nucleus, where proteins that make up our cells are manufactured. Neurons have dendrites, long extensions that receive messages from other neurons and an axon, which extends from the cell body of each neuron and is responsible for sending messages.

1. The central region of the neuron which manufactures new cell components is called the _________ _________. (p. 86)
2. The receiving ends of a neuron, extending from the cell body like tree branches, are known as _________. (p. 86)
3. ________ are long extensions from the neuron at the cell body that ________ messages from one neuron to another. (p. 86)

3.2 DESCRIBE ELECTRICAL RESPONSES OF NEURONS AND WHAT MAKES THEM POSSIBLE.
Neurons exhibit excitatory and inhibitory responses to inputs from other neurons. When excitation is strong enough, the neuron generates an action potential, which travels all the way down the axon to the axon terminal. Charged particles crossing the neuronal membrane are responsible for these events.

4. The space between two connecting neurons where neurotransmitters are released is called the _________. (p. 86)
5. The autoimmune disease multiple sclerosis is linked to the destruction of the glial cells wrapped around the axon, called the _________. (p. 87)

3.3 EXPLAIN HOW NEURONS USE NEUROTRANSMITTERS TO COMMUNICATE WITH EACH OTHER.

6. The electrical charge difference across the membrane of the neuron when it’s not being stimulated is called the _________. (p. 87)
7. Label the image showing the process of action potential in a neuron. Include (a) axon, (b) arrow depicting the direction of the action potential, and (c) neurotransmitters. (p. 88)

3.4 DESCRIBE HOW THE BRAIN CHANGES AS A RESULT OF DEVELOPMENT, LEARNING, AND INJURY.
The brain changes the most before birth and during early development. Throughout the life span the brain demonstrates some degree of plasticity, which plays a role in learning and memory. Later in life, healthy brain plasticity decreases and neurons can show signs of degeneration.

8. Scientists are working to improve ways to encourage neurogenesis, the adult brain’s ability to create new _________. (p. 93)

3.5 IDENTIFY WHAT ROLES DIFFERENT PARTS OF THE CENTRAL NERVOUS SYSTEM PLAY IN BEHAVIOR.
The cerebral cortex consists of the frontal, parietal, temporal, and occipital lobes. Cortex involved with vision lies in the occipital lobe, cortex involved with hearing in the temporal lobe, and cortex involved with touch in the parietal lobe. Association areas throughout the cortex analyze and reanalyze sensory inputs to build up our perceptions. The motor cortex in the frontal lobe, the basal ganglia, and the spinal cord work together with the somatic nervous system to bring about movement and action. The somatic nervous system has a sensory as well as a motor component, which enables touch and feedback from the muscles to guide our actions.

9. The brain and spinal cord combine to form the superhighway known as the _________. (p. 93)

10. Outside of the CNS, the ________ system works to help us control behavior and express emotion. (p. 93)

11. The brain component responsible for analyzing sensory information and our ability to think, talk, and reason is called the _________. (p. 95)

12. Label the various parts of the central nervous system. (p. 94)
15. Fill in the function of each brain component identified in this figure. (p. 96)

16. Parkinson’s disease is the result of damage to the __________ __________, which play a critical role in voluntary movement. (p. 98)

17. The __________ __________ system connects to the forebrain and cerebral cortex and plays a key role in arousal. (p. 100)

3.6 CLARIFY HOW THE SOMATIC AND AUTONOMIC NERVOUS SYSTEMS WORK IN EMERGENCY AND EVERYDAY SITUATIONS.

The somatic nervous system carries messages from the CNS to the body’s muscles. The autonomic nervous system consists of the parasympathetic and sympathetic divisions. Whereas the parasympathetic nervous system is active during rest and digestion, the sympathetic division propels the body into action during an emergency or crisis. Sympathetic arousal also occurs in response to everyday stressors.

18. Our ability to execute messages or commands of our central nervous system, through physical action, is dependent on the __________ __________ system. (p. 102)

19. Our ability to react physically to a perceived threat is dependent on the __________ division of the autonomic system. (p. 103)

20. Sympathetic activation triggers a variety of physical responses, including increased heart rate, __________, and __________. (p. 103)

THE ENDOCRINE SYSTEM 103–105

3.7 DESCRIBE WHAT HORMONES ARE AND HOW THEY AFFECT BEHAVIOR.

Hormones are chemicals released into the bloodstream that trigger specific effects in the body. Activation of the sympathetic nervous system triggers the release of adrenaline and cortisol by the adrenal glands, which energize our bodies. Sex hormones control sexual responses.

21. The limbic system in the brain also cooperates with the __________ __________ in the body to regulate emotion. (p. 103)

22. The gland once called the “master gland” which, under the control of the hypothalamus, directs all other body glands is known as the __________ __________. (p. 103)

23. Label the major endocrine glands in the body. (p. 103)

24. The pituitary hormone called __________ is responsible for a variety of reproductive functions including stretching the cervix and vagina during birth and aiding milk flow in nursing mothers. (p. 103)

25. Psychologists sometimes call the __________ __________ the emergency centers of the body. (p. 104)

26. When under threat or attack, how does the body prepare for fight or flight? (p. 104)

27. Many anxiety disorders are associated with elevated levels of __________. (p. 105)

28. The testes make the male sex hormone, called __________, and the ovaries make the female sex hormone, called __________. (p. 105)

29. Males and females (do/don’t) both manufacture some amount of sex hormone associated with the opposite sex. (p. 105)

30. Most researchers (accept/reject) the hypothesis that testosterone influences female sex drive. (p. 105)
MAPPING THE MIND: THE BRAIN IN ACTION

3.8 IDENTIFY THE DIFFERENT BRAIN-STIMULATING, -RECORDING, AND -IMAGING TECHNIQUES.

Electrical stimulation of the brain can elicit vivid imagery or movement. Methods such as electroencephalography (EEG) and magnetoencephalography (MEG) enable researchers to record brain activity. Imaging techniques provide a way to see the brain’s structure or function. The first imaging techniques included computed tomography (CT) and magnetic resonance imaging (MRI). Imaging techniques that allow us to see how the brain’s activity changes in response to psychological stimuli include positron emission tomography (PET) and functional MRI (fMRI).

31. Franz Joseph Gall made one of the earliest attempts to connect mind and brain by measuring head bumps, a technique known as _________. (p. 106)

32. Early efforts by Hans Berger to measure electrical activity in the brain resulted in the development of the _________. (p. 107)

33. Neuroscientists interested in measuring thought and emotion (would/wouldn’t) employ a CT scan. (p. 107)

34. What do functional MRIs (fMRI), such as the one pictured here, measure? (p. 109)

38. In this experiment, researchers flashed a chicken claw to a split-brain patient’s left hemisphere and a snow scene to his right hemisphere. How can we explain his response? (p. 111)

39. The ________ hemisphere of the brain is related to coarse language skills and visuospatial skills whereas the ________ hemisphere is related to fine-tuned language skills and actions. (p. 111)

40. Artists and other creative thinkers (are/aren’t) able to make use only of their right hemisphere. (p. 112)

3.10 DESCRIBE GENES AND HOW THEY INFLUENCE PSYCHOLOGICAL TRAITS.

Genes are composed of deoxyribonucleic acid (DNA), which are arranged on chromosomes. We inherit this genetic material from our parents. Each gene carries a code to manufacture a specific protein. These proteins influence our observable physical and psychological traits.

41. How many chromosomes do humans have? How many are sex-linked? (p. 114)

42. ________ are the thin threads within a nucleus that carry genes. (p. 113)

43. ________ are made up of deoxyribonucleic acid (DNA), the material that stores everything cells need to reproduce themselves. (p. 114)

44. Our is the set of our observable traits, and our genetic makeup is our _________. (p. 114)

45. (Recessive/Dominant) genes work to mask other genes’ effects. (p. 114)
46. The principle that organisms that possess adaptations survive and reproduce at a higher rate than other organisms is known as ________ _________. (p. 114)

### 3.11 EXPLAIN THE CONCEPT OF HERITABILITY AND THE MISCONCEPTIONS SURROUNDING IT.

Heritability refers to how differences in a trait across people are influenced by their genes as opposed to their environments. Highly heritable traits can sometimes change within individuals and the heritability of a trait can also change over time within a population.

47. Scientists use ________ ________ to examine the roles of nature and nurture in the origins of traits, such as intelligence. (p. 115)

48. Heritability applies only to (a single individual/groups of people). (p. 115)

49. Does high heritability imply a lack of malleability? Why or why not? (p. 116)

50. Analyses of how traits vary in individuals raised apart from their biological relatives are called ________ _________. (p. 117)

### DO YOU KNOW THESE TERMS?

- neuron (p. 85)
- dendrite (p. 86)
- axon (p. 86)
- synaptic vesicle (p. 86)
- neurotransmitter (p. 86)
- synaptic cleft (p. 86)
- synapse (p. 86)
- glial cell (p. 87)
- myelin sheath (p. 87)
- resting potential (p. 87)
- threshold (p. 87)
- action potential (p. 87)
- absolute refractory period (p. 88)
- receptor site (p. 88)
- reuptake (p. 88)
- endorphin (p. 90)
- plasticity (p. 91)
- stem cell (p. 92)
- neurogenesis (p. 93)
- central nervous system (CNS) (p. 93)
- peripheral nervous system (PNS) (p. 93)
- cerebral ventricles (p. 94)
- forebrain (cerebrum) (p. 95)
- cerebral hemispheres (p. 95)
- corpus callosum (p. 95)
- cerebral cortex (p. 95)
- frontal lobe (p. 96)
- motor cortex (p. 96)
- prefrontal cortex (p. 96)
- Broca’s area (p. 96)
- parietal lobe (p. 97)
- temporal lobe (p. 97)
- Wernicke’s area (p. 98)
- occipital lobe (p. 98)
- primary sensory cortex (p. 98)
- association cortex (p. 98)
- basal ganglia (p. 98)
- limbic system (p. 99)
- thalamus (p. 99)
- hypothalamus (p. 99)
- amygdala (p. 99)
- hippocampus (p. 100)
- brain stem (p. 100)
- midbrain (p. 100)
- reticular activating system (RAS) (p. 100)
- hindbrain (p. 101)
- cerebellum (p. 101)
- pons (p. 101)
- medulla (p. 101)
- spinal cord (p. 101)
- interneuron (p. 101)
- reflex (p. 101)
- somatic nervous system (p. 102)
- autonomic nervous system (p. 102)
- sympathetic division (p. 102)
- parasympathetic division (p. 103)
- endocrine system (p. 103)
- hormone (p. 103)
- pituitary gland (p. 103)
- adrenal gland (p. 104)
- electroencephalograph (EEG) (p. 107)
- computed tomography (CT) (p. 107)
- magnetic resonance imaging (MRI) (p. 107)
- positron emission tomography (PET) (p. 107)
- functional MRI (fMRI) (p. 108)
- transcranial magnetic stimulation (TMS) (p. 108)
- magnetoencephalography (MEG) (p. 108)
- lateralization (p. 111)
- split-brain surgery (p. 111)
- chromosome (p. 113)
- gene (p. 113)
- genotype (p. 114)
- phenotype (p. 114)
- dominant gene (p. 114)
- recessive gene (p. 114)
- fitness (p. 114)
- heritability (p. 115)
- family study (p. 116)
- twin study (p. 116)
- adoption study (p. 117)

### APPLY YOUR SCIENTIFIC THINKING SKILLS

Use your scientific thinking skills to answer the following questions, referencing specific common errors in thinking and scientific thinking principles whenever possible.

1. Many websites and magazine articles exaggerate the notion of brain lateralization. Find two examples of products designed for either a “left-brained” or “right-brained” person. Are the claims made by these products supported by scientific evidence? Explain.

2. As we’ve learned in this chapter, scientists still aren’t sure what causes women’s sex drives to increase at certain times, although many view testosterone as a key influence. Locate alternative explanations for this hypothesis in the popular media and evaluate each using your scientific thinking skills.

3. The news media sometimes report functional brain imaging findings accurately, but often report them in oversimplified ways, such as implying that researchers identified a single brain region for Capacity X (like religion, morality, or political affiliation). Locate two media reports on functional brain imaging (ideally using fMRI or PET) and evaluate the quality of media coverage. Did the reporters interpret the findings correctly, or did they go beyond the findings? For example, did the reporters avoid implying that the investigators located a single brain “spot” or “region” underlying a complex psychological capacity?