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# How to download R & RStudio

## Mac Users

### To Install R

1. Open an internet browser and go to [www.r-project.org](http://www.r-project.org).
2. Click the “download R” link in the middle of the page under “Getting Started.”
3. Select a CRAN location (a mirror site) and click the corresponding link.
4. Click on the “Download R for (Mac) OS X” link at the top of the page.
5. Click on the file containing the latest version of R under “Files.”
6. Save the .pkg file, double-click it to open, and follow the installation instructions.
7. Now that R is installed, you need to download and install RStudio.

### To Install RStudio

1. Go to [www.rstudio.com](http://www.rstudio.com) and click on the “Download RStudio” button.
2. Click on “Download RStudio Desktop.”
3. Click on the version recommended for your system, or the latest Mac version, save the .dmg file on your computer, double-click it to open, and then drag and drop it to your applications folder.

## Windows Users

### To Install R

1. Open an internet browser and go to [www.r-project.org](http://www.r-project.org).
2. Click the “download R” link in the middle of the page under “Getting Started.”
3. Select a CRAN location (a mirror site) and click the corresponding link.
4. Click on the “Download R for Windows” link at the top of the page.
5. Click on the “install R for the first time” link at the top of the page.
6. Click “Download R for Windows” and save the executable file somewhere on your computer.
7. Run the .exe file and follow the installation instructions. Now that R is installed, you need to download and install RStudio.

## To Install RStudio

1. Go to [www.rstudio.com](http://www.rstudio.com) and click on the “Download RStudio” button.
2. Click on “Download RStudio Desktop.”
3. Click on the version recommended for your system, or the latest Windows version, and save the executable file. Run the .exe file and follow the installation instructions.

# 2

## Ask & Answer

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# Research Methodology

**ADMIT IT. EVEN THOUGH YOU KNEW** it was probably a bad idea at the time, you have used your cell phone to talk or text when it was unwise to do so. Maybe you were in class and could not resist looking at a Snapchat someone just sent you. Or you were walking to class and crossing busy roads while chatting with one of your parents. Or maybe you sent a text message while driving to say you were running late. You are not alone. The risky use of cell phones is common. Various studies have found that 80 percent to 90 percent of college students admit texting while driving on at least one occasion (Harrison, 2011). Unfortunately, texting or phoning while driving can be disastrous.

In 2009, a Boston trolley driver who was texting his girlfriend while on the job rear-ended another trolley, sending 49 people to the hospital and costing the transit system nearly \$10 million. In 2007, five recent high school graduates were killed in an accident in upstate New York. The inexperienced driver had been talking on her cell phone minutes before the accident and might have been responding to a text seconds before she crossed the road and struck a semi-trailer head on. In January 2010, 17-year-old Kelsey Raffaele (**FIGURE 2.1**) was driving after school and decided to pass a slower vehicle in front of her. When she saw an oncoming vehicle in the passing lane, she misjudged the distance and crashed, fatally. Kelsey was talking to a friend on her cell phone while driving. Her last words were “Oh [no], I’m going to crash.”

Talking on the phone while driving is risky, but texting while driving is even worse, dramatically increasing your chances of having an accident (Dingus,



**FIGURE 2.1**  
**Phoning While Driving**  
Phoning while driving is extremely unsafe. Kelsey Raffaele lost her life because she engaged in this dangerous behavior.

Hanowski, & Klauer, 2011). In laboratories, researchers have examined these practices by using driving simulators (**FIGURE 2.2**). In studies that examined the effects of texting on driving, participants had either less than six months of driving experience (Hosking et al., 2009) or on average five years of driving experience (Drews et al., 2009). The participants “drove” either undistracted or while sending and receiving texts. All participants texting while driving missed more landmarks, made more driving errors, and crashed more than participants who were driving undistracted.

Yet in a 2012 survey by the National Highway Traffic Safety Administration (NHTSA), 25 percent of drivers reported that they believed that texting while driving made no difference on driving performance. Why would people hold this belief? As discussed in Chapter 1, we are often poor judges of our own behavior. We feel overconfident about our abilities and fail to see our own weaknesses. Because we tend to overestimate our own driving abilities—seeing ourselves as “good” drivers even when we are not—we also tend to underestimate the dangers we face, such as through texting while driving. In one study, the participants who most overestimated their ability to drive when distracted were the ones who used the cell phone more while driving in everyday life—and they had worse driving records than the other participants (Schlehofer et al., 2010).

So how can we confirm (and convince people) that texting while driving is dangerous? Indeed, how can we confirm (and convince people of) any claim that is made? This chapter will describe how evidence is gathered and verified in psychology. By understanding these processes, you will learn how to interpret information that is being presented to you. And by understanding how to interpret information, you will become an educated consumer and presenter of information.

## Learning Objectives

- Identify the four primary goals of science.
- Describe the scientific method.
- Differentiate among theories, hypotheses, and research.

### data

Measurable outcomes of research studies.

## 2.1 How Is the Scientific Method Used in Psychological Research?

This chapter will introduce you to the science and the practice of psychological research methods. You will learn the basics of collecting, analyzing, and interpreting the **data** of psychological science, the measurable outcomes of research studies. In this way, you will come to understand how psychologists study behavior and mental processes. You will also learn how to effectively evaluate claims so you can become a more educated consumer of information.

### Science Has Four Primary Goals

There are four primary goals of science: *description*, *prediction*, *control*, and *explanation*. Thus, the goals of psychological science are to describe *what* a phenomenon is, predict *when* it will occur, control *what causes* it to occur, and explain *why* it occurs. For example, consider the observation that texting interferes with driving. To understand how this interference happens, we need to address each of the four goals.

We begin by asking: How many people really text while driving? Answering this question can help us describe the phenomenon of texting while driving—as in noting how prevalent this unsafe behavior is. Now, under what circumstances are people likely to text while driving? Answering this question can help us predict when texting while driving may occur—as in which people tend to engage in the behavior. Next, how can we know that texting is the source of the problematic driving? Answering this question can help us be sure that it is texting and not some other factor that is responsible for the observed effects. Ultimately, knowing the answers to each of these questions leads to the question of why texting interferes with driving. Is it because people use their hands to text, or that they take their eyes off the road, or that it interferes with their mental ability to focus on driving?

Careful scientific study also allows us to understand other aspects of texting and driving, such as why people do it in the first place. Understanding how texting interferes with driving skills and why people continue to text while driving, even when they know it is dangerous, will allow scientists, technology developers, and policymakers to develop strategies to reduce the behavior.

## Critical Thinking Means Questioning and Evaluating Information

As you learned in Chapter 1, one important goal of your education is to become a critical thinker. Critical thinking was defined in Chapter 1 as systematically questioning and evaluating information using well-supported evidence. As this definition makes clear, critical thinking is an *ability*—a skill. It is not something you can just memorize and learn, but something you have to practice and develop over time. Most of your courses should provide opportunities for you to practice being a critical thinker. Critical thinking is not just for scientists. It is essential for becoming an educated consumer of information.

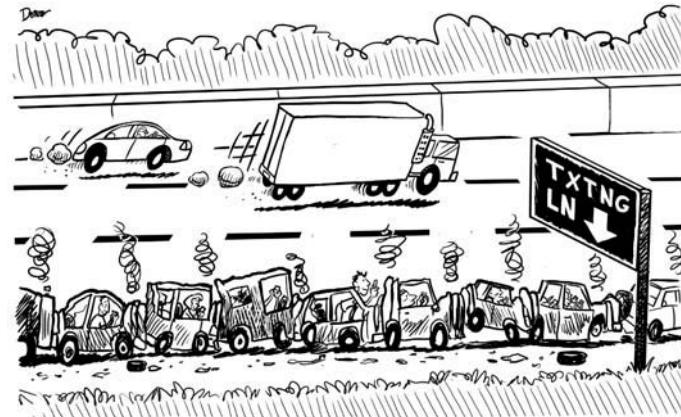
The first step in critical thinking is to question information. What kind of information? To develop the skeptical mindset you need for critical thinking, you should question every kind of information. For any claim you see or hear, ask yourself, “What is the evidence in support of that claim?” For example, in the opening vignette of this chapter, we made the claim that texting while driving is dangerous. What kind of evidence did we present in support of this claim? Was the evidence based on direct, unbiased observation, or did it seem to be the result of rumor, hearsay, or intuition? In fact, think of your own beliefs and behavior. Do you believe that texting while driving is dangerous? If you do, what evidence led you to this belief? If you believe that texting while driving is dangerous, do you still text while driving? If so, why do you do it? Do you think the evidence you have seen or heard is not very good? If so, what makes the evidence not very good?

Another aspect of questioning when thinking critically is to ask for the definition of each part of the claim. For example, imagine you hear the claim that using a cell phone while driving is more dangerous than driving while intoxicated (see “Scientific Thinking: Cell Phone Versus Intoxication,” on p. 36). Upon hearing this claim, a critical thinker immediately asks for definitions. For example, what do they mean by “using a cell phone”? Do they mean talking or texting? Do they mean a handheld or a hands-free device? And what do they mean by “intoxicated”? Would achieving this state require only a little alcohol or a lot of alcohol? Could the person have used another drug?



**FIGURE 2.2**  
**Driving Simulator**

This apparatus enables researchers to study driving skills in the laboratory.



# Scientific Thinking

## Cell Phone Versus Intoxication

**HYPOTHESIS:** Using a cell phone while driving is more dangerous than driving while intoxicated.

**RESEARCH METHOD:** Forty adults, ranging in age from 22 to 34, were recruited by a newspaper advertisement to participate in a research study on driving. In the study, the participants were asked to perform two separate tests in a driving simulator: (a) driving while having verbal conversations via a hand-held or hands-free device, and (b) driving after consuming enough alcohol to achieve a .08 percent blood-alcohol content (BAC), a level that is at or above the legal limit for intoxication in most states (see table). To establish their baseline driving performances, all the participants initially drove in the simulator without talking on the phone and without having consumed alcohol.

The tests occurred on two different days. Half of the participants talked on the phone while driving the first day and drank before driving on the second day. The other half drank before driving on the first day and talked on the phone while driving the second day.

**RESULTS:** Compared to the baseline driving performance, talking on the phone (with either a hand-held or hands-free device) caused a delayed response to objects in the driving scene, including brake lights on the car ahead, and a greater number of rear-end collisions. When they were intoxicated, the participants drove aggressively. They followed other cars more closely and hit the brake pedal much harder than they did in the baseline condition. Talking on a cell phone produced more collisions than driving while intoxicated.

**CONCLUSION:** Both talking on the cell phone and driving while intoxicated led to impaired driving compared to the baseline condition. Talking on the cell phone, whether holding the phone or not, led to more collisions than when the participants were intoxicated.

**SOURCE:** Strayer, D. L., Drews, F. A., & Crouch, D. J. (2006). A comparison of the cell phone driver and the drunk driver. *Human Factors: The Journal of the Human Factors and Ergonomics Society*, 48, 381–391.

### Blood Alcohol Content and Its Effects

In the United States, blood alcohol content is measured by taking a sample of a person's breath or blood and determining the amount of alcohol in that sample. The result is then converted to a percentage. For example, in many states the legal limit is .08 percent. To reach this level, a person's bloodstream needs to have 8 grams of alcohol for every 100 milliliters of blood.

Different blood alcohol levels produce different physical and mental effects. These effects also vary from person to person. This table shows typical effects.

BAC LEVEL	EFFECTS
.01-.06	Feeling of relaxation Sense of well-being Thought, judgment, and coordination are impaired.
.07-.10	Loss of inhibitions Extroversion Reflexes, depth perception, peripheral vision, and reasoning are impaired.
.11-.20	Emotional swings Sense of sadness or anger Reaction time and speech are impaired.
.21-.29	Stupor Blackouts Motor skills are impaired.
.30-.39	Severe depression Unconsciousness Breathing and heart rate are impaired.
>.40	Breathing and heart rate are impaired. Death is possible.

SOURCE: Based on U.S. Department of Transportation, <http://www-nrd.nhtsa.dot.gov/Pubs/811385.pdf>

Answering questions of this kind is the second step in critical thinking: the evaluation of information. To answer our questions, we need to go to the source of the claim.

To get to the source of any claim, you need to think about where you first saw or heard the claim. Did you hear the claim on TV or the radio? Did you read about it in a newspaper? Did you see it on the Internet? Next, you need to think about the evidence offered by the source to support the claim.

Here is where the “well-supported evidence” comes in. Does the evidence at the source of the claim take the form of scientific evidence? Or does it take the form of intuition or simply someone in authority making the claim? Did the source retrieve this information from a news wire? Did it come from an interview with a scientist? Was it summarized from a scientific journal?

In science, well-supported evidence typically means research reports based on empirical data that are published in peer-reviewed journals (**FIGURE 2.3**). “Peer review” is a process by which other scientists with similar expertise evaluate and critique research reports before publication. Peer review ensures that published reports describe research studies that are well designed (using appropriate research and analysis methods, considering all factors that could explain the findings), that are conducted in an ethical manner, and that address an important question.

However, peer review does not mean that flawed studies are never published. Thus, critical thinkers must *always* stay vigilant—always be on the lookout for unreasonable claims and conclusions that may not be valid interpretations of the data. Hone your critical thinking skills by practicing them as often as possible. (At the end of this chapter, the Practice Test includes questions related to designing a scientific study. These questions will both help you practice critical thinking and test the knowledge you have gained from this chapter.)

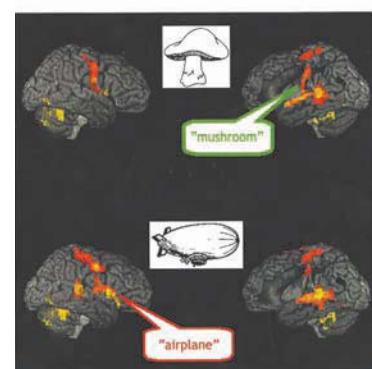
## The Scientific Method Aids Critical Thinking

Critical thinking is determining whether a claim is supported by evidence. Scientific evidence obtained through research is considered the best possible evidence for supporting a claim. **Research** involves the careful collection of data. In conducting research, scientists follow a systematic procedure called the **scientific method**. This procedure begins with the observation of a phenomenon and the question of why that phenomenon occurred.

The scientific method is an interaction among research, theories, and hypotheses (**FIGURE 2.4**). A **theory** is an explanation or model of how a phenomenon works. Consisting of interconnected ideas or concepts, a theory is used to explain prior observations and to make predictions about future events. A **hypothesis** is a specific, testable prediction, narrower than the theory it is based on.

**GOOD THEORIES** How can we decide whether a theory is good? When we talk about a good theory, we do not mean that it is good because it is supported by research findings. In fact, one key feature of a good theory is that it should be *falsifiable*. That is, it should be possible to test hypotheses generated by the theory that prove the theory is incorrect. Moreover, a good theory produces a wide variety of *testable* hypotheses.

For instance, in the early twentieth century, the developmental psychologist Jean Piaget (1924) proposed a theory of infant and child development (see Chapter 9, “Human Development”). According to Piaget’s theory, cognitive development occurs in a fixed series of “stages,” from birth to adolescence. From a scientific standpoint, this theory was good because it led to a number of hypotheses. These



**FIGURE 2.3**  
**Peer-Reviewed Journals**

Research reports in peer-reviewed journals are the most trustworthy source for scientific evidence.

### research

A scientific process that involves the careful collection of data.

### scientific method

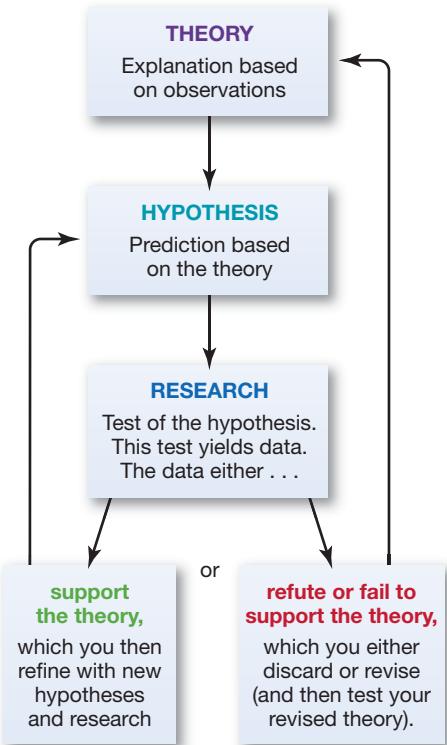
A systematic and dynamic procedure of observing and measuring phenomena, used to achieve the goals of description, prediction, control, and explanation; it involves an interaction among research, theories, and hypotheses.

### theory

A model of interconnected ideas or concepts that explains what is observed and makes predictions about future events. Theories are based on empirical evidence.

### hypothesis

A specific, testable prediction, narrower than the theory it is based on.



**FIGURE 2.4**  
**The Scientific Method**  
The scientific method reflects a cyclical process: A theory is formulated based on evidence from many observations and refined based on hypothesis tests (scientific studies). From the theory, scientists derive one or more testable hypotheses. Scientists then conduct research to test the hypotheses. Findings from the research might prompt scientists to reevaluate and adjust the theory. A good theory evolves over time, and the result is an increasingly accurate model of some phenomenon.

hypotheses concerned the specific kinds of behaviors that should be observed at each stage of development. In the decades since its proposal, the theory has generated thousands of scientific papers. Our understanding of child development has been enhanced both by studies that supported Piaget's stage theory and by those that failed to support it.

In contrast, Piaget's contemporary Sigmund Freud (1900), in his famous treatise *The Interpretation of Dreams*, outlined the theory that all dreams represent the fulfillment of an unconscious wish. From a scientific perspective, Freud's theory was not good, because it generated few testable hypotheses regarding the actual function of dreams. Since the theory lacked testable hypotheses, researchers were left with no way to evaluate whether the wish fulfillment theory was either reasonable or accurate. After all, unconscious wishes are, by definition, not known to anyone, including the person having the dreams. As a result, not only is there no way to prove that dreams do represent unconscious wishes, but there is no way to prove that dreams do not represent unconscious wishes. Thus, the theory is frequently criticized for not being falsifiable.

Good theories also tend toward simplicity. This idea has historical roots in the writings of the fourteenth-century English philosopher William of Occam. Occam proposed that when two competing theories exist to explain the same phenomenon, the simpler of the two theories is generally preferred. This principle is known as *Occam's Razor* or the *law of parsimony*.

**HYPOTHESES NEED TO BE TESTED** In order to test hypotheses generated by good theories, we use the scientific method. After an observation has been made and a theory has been formulated, the scientific method follows a series of six steps (**FIGURE 2.5**):

### Step 1: Form a Hypothesis

From the opening of this chapter, we have been considering cell phone use while driving. Say that you now propose a theory, derived from news accounts and scientific studies. Your theory is that cell phone use impairs driving ability. How can you determine if this theory is true? You design specific tests—that is, specific research studies—aimed at examining the theory's prediction. These specific, testable research predictions are your hypotheses.

If your theory is true, then the tests should provide evidence that using cell phones while driving causes problems. One of your hypotheses therefore might be: "Using a cell phone while driving will lead to more accidents." To test this hypothesis, you might compare people who use a cell phone frequently while driving with people who do not use a cell phone frequently while driving. You would record how often the people in these groups have accidents. If these results do not differ, this finding raises questions about whether the theory is true.

### Step 2: Conduct a Literature Review

Once you form a hypothesis, you want to perform a literature review as soon as possible. A literature review is a review of the scientific literature related to your theory. There are many resources available to assist with literature reviews, including scientific research databases such as PsycINFO and PubMed. You can search these databases by keywords, such as "cell phones and driving" or "cell phones and accidents." The results of your searches will reveal if and how other scientists have been testing your idea. For example, different scientists may have approached this topic at different levels of analysis (see Chapter 1). Their approaches may help guide the direction of your research. For example, you might find a study that compares talking on a cell



**FIGURE 2.5**  
**The Scientific Method in Action**  
This figure lays out the six steps of the scientific method.

phone while driving with texting while driving. You discover that texting is much more likely to cause accidents. You might then narrow your hypothesis to examine the specific action of texting.

### Step 3: Design a Study

Designing a study refers to deciding which research method (and thus, level of analysis) you want to use to test your hypothesis. To test whether texting causes more accidents, you could conduct a survey: Give people a questionnaire that asks how often they text while driving. This method is used widely to gain initial insight into your

hypothesis. In large surveys of high school students and college students, more than 40 percent reported texting while driving at least once in the previous 30 days (Olsen, Shults, & Eaton, 2013).

Instead of a survey, you could conduct a naturalistic observation: Watch a particular group of drivers over time and measure how often they text while driving or talk on a cell phone while driving. To establish how cell phone use affects driving, you could more intensively examine drivers by placing devices in their cars to measure aspects such as driving speed and acceleration. Or you could use video cameras to create an objective record of risky driving behaviors, such as running stop signs. One study of 151 drivers using such methods found that cell phone use, especially texting, was a strong predictor of crashes and near-crashes (Klauer et al., 2013).

Alternatively, you could perform an actual experiment, assigning one group of people to texting while driving and a second group of people to no texting, then comparing the number of accidents they have. Obviously, performing a test of this kind on public roads would be dangerous and unethical. Thus, for research like this, scientists use driving simulators that mimic real-world driving conditions. As you will see later when we discuss the different research methods available to test your hypothesis, there are advantages and disadvantages to each of these methods.

#### **Step 4: Conduct the Study**

Once you choose your research method, you have to conduct the study: Recruit participants and measure their responses. Many people call this step collecting data or gathering data. If you conduct a survey to see whether people who use cell phones while driving have more accidents, your data will include both the frequency with which people use cell phones while driving and how many accidents they have. All the research methods require you to clarify how you are defining “driving while texting” and “accidents.” You must also take care in defining the appropriate size and type of sample of participants. These issues are addressed more completely later in this chapter, under the discussions of operational definitions and sampling.

#### **Step 5: Analyze the Data**

The next step is to analyze your data. There are two main ways to analyze data. First, you want to describe the data. What was the average score? How “typical” is that average? Suppose the average driver in your study has five years of driving experience. Does this statement mean five is the most common number of years of driving experience, or that five is the numerical average if you divide the total number of years driven by the total number of participants, or that about half of drivers have this many years of experience?

Second, you will want to know what conclusions you can draw from your data. You need to know whether your results are meaningful or whether they happened by chance. To determine the usefulness of your data, you analyze the data inferentially. That is, you ask whether you found a significant effect. Asking this question enables you to make inferences about your data—to infer whether your findings might be true for the general population. You accomplish data analyses by using descriptive and inferential statistics, which are described more completely later in the chapter.

#### **Step 6: Report the Results**

Unreported results have no value, because no one can use any of the information. Instead, scientists make their findings public to benefit society, support the scientific culture, and also permit other scientists to build on their work. Various forums are available for distributing the results of scientific research.

Brief reports can be presented at scientific conferences. The most popular formats for presenting data at conferences are talks and poster sessions. At the latter, people create large posters that display information about their study. During these sessions,

researchers stand by their posters and answer questions to those who stop by to read the poster. Conference presentations are especially good for reporting preliminary data or for presenting exciting or cutting-edge results.

Full reports should be published in a peer-reviewed scientific journal (see Figure 2.3). Full reports consist of the background and significance of the research, the full methodology for how the question was studied, the complete results of the descriptive and inferential statistical analyses, and a discussion of what the results mean in relation to the accumulated body of scientific evidence.

Sometimes the results of research are of interest to the general public. People in the media attend scientific conferences and read scientific journals so they can report on exciting findings. Eventually, interesting and important science will reach a general audience.

**THE SCIENTIFIC METHOD IS CYCLICAL** Once the results of a research study are in, the researchers return to the original theory to evaluate the implications of the data. If the study was conducted competently (i.e., used appropriate methods and data analysis to test the hypothesis), the data either support the theory or suggest that the theory be modified or discarded. Then the process starts all over again. Yes, the same sort of work needs to be performed repeatedly. No single study can provide a definitive answer about any phenomenon. No theory would be discarded on the basis of one set of data. Instead, we have more confidence in scientific findings when research outcomes are replicated.

**Replication** involves repeating a study and getting the same (or similar) results. When the results from two or more studies are the same, or at least support the same conclusion, confidence increases in the findings. Ideally, researchers not affiliated with those who produced the original finding conduct replication studies. These independent replications provide more powerful support because they rule out the possibility that some feature of the original setting may have contributed to the findings. Within the last few years, there has been a growing emphasis on replication within psychological science.

Good research reflects the cyclical process shown in Figure 2.5. In other words, a theory is continually refined by new hypotheses and tested by new research methods. In addition, often more than one theory may apply to a particular aspect of human behavior so that the theory needs to be refined to become more precise.

For instance, the theory that using a cell phone while driving impairs driving skills might be accurate, but you want to know more. *How* does using a cell phone impair driving? You might develop new theories that take into account the skills needed to be a good driver. You could theorize that using a cell phone impairs driving because it requires taking your hands off the wheel, or perhaps texting requires taking your eyes off the road, or perhaps using a cell phone at all impairs your ability to think about driving. To understand which theory is best, you can design *critical studies* that directly contrast theories to see which theory better explains the data. Replication is another means of strengthening support for some theories and helping weed out weaker theories.



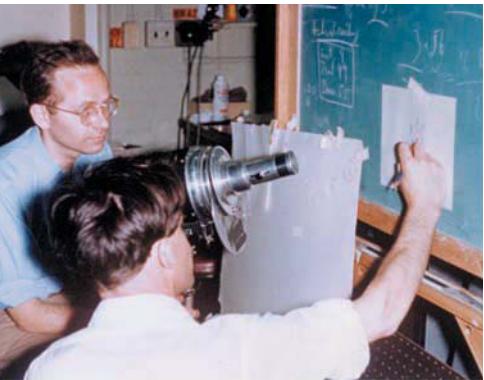
## Unexpected Findings Can Be Valuable

Research does not always proceed in a neat and orderly fashion. On the contrary, many significant findings are the result of *serendipity*. In its general sense, serendipity means unexpectedly finding things that are valuable or agreeable. In science, it means unexpectedly discovering something important.

In the late 1950s, the physiologists Torsten Wiesel and David Hubel recorded the activity of nerve cells in cats' brains. Specifically, they were measuring the

### replication

Repetition of a research study to confirm the results.



**FIGURE 2.6**  
**Wiesel and Hubel's Dot Pattern Experiments**

Torsten Wiesel (**foreground**) and David Hubel are shown with their dot projector, 1958.

activity of cells in brain areas associated with vision. Hubel and Wiesel (1959) were studying how information travels from the eye to the brain (a process explored extensively in Chapter 5, “Sensation and Perception”). They had hypothesized that certain cells in the visual portion of the brain would respond when the cats looked at dots. To test that hypothesis, they showed slides of dot patterns to the cats (**FIGURE 2.6**). After much disappointing work that produced no significant activity in the brain cells being observed, the projector suddenly jammed between slides. The cells in question began to fire at an astonishing rate! What had caused this firing? Wiesel and Hubel realized that the jammed slide had produced a visual “edge” on the screen.

Because of this little accident, Wiesel and Hubel discovered that these specific cells do not respond to simple dots. They eventually received a Nobel Prize for the serendipitous finding that certain brain cells respond specifically to lines and edges. Although their discovery is an example of serendipity, these researchers were not just lucky. They did not stumble onto a groundbreaking discovery that led straight to a Nobel Prize. Rather, they followed up on their unexpected finding. Thanks to their critical thinking abilities, they were open to new ideas. After a lifetime of hard work, they understood the implications of the rapid firing of certain brain cells in response to straight lines but not to other types of visual stimuli.

## Summing Up

### How Is the Scientific Method Used in Psychological Research?

- The four primary goals of science are *description* (describing what a phenomenon is), *prediction* (predicting when a phenomenon might occur), *control* (controlling the conditions under which a phenomenon occurs), and *explanation* (explaining what causes a phenomenon to occur).
- Critical thinking is a skill that helps people become educated consumers of information. Critical thinkers question claims, seek definitions for the parts of the claims, and evaluate the claims by looking for well-supported evidence.
- The scientific method helps psychologists achieve their goals of describing, predicting, controlling, and explaining behavior.
- Scientific inquiry relies on objective methods and empirical evidence to answer testable questions.
- The scientific method is based on the use of theories to generate hypotheses that can be tested by collecting objective data through research. Good theories are falsifiable and will generate several testable hypotheses.
- After a theory has been formulated based on observing a phenomenon, the six steps of the scientific method are forming a hypothesis based on the theory, conducting a literature review to see how people are testing the theory, choosing a research method to test the hypothesis, conducting the research study, analyzing the data, and reporting the results.
- Scientists examine the results to see how well they match the original hypothesis. The theory must be adjusted as new findings confirm or disconfirm the hypothesis.
- Unexpected (serendipitous) discoveries sometimes occur, but only researchers who are prepared to recognize their importance will benefit from them. Although unexpected findings can suggest new theories, these findings must be replicated and elaborated.

## Measuring Up

1. How are theories, hypotheses, and research different?
  - a. Theories ask questions about possible causes of thoughts, emotions, and behaviors. Hypotheses provide the empirical answers. Research is used to examine whether theories are correct.

- b. Theories are broad conceptual frameworks. Hypotheses are derived from theories and are used to design research that will support or fail to support a theory. Research is a test of the hypotheses.
- c. Theories are assumed to be true. Hypotheses need to be tested with appropriate experiments. Research is the final step.
- d. Theories do not require data for their verification because they are abstract. Hypotheses depend on experimental findings. Research uses human participants to test theories and hypotheses.

## 2. Why is critical thinking so important?

- a. Critical thinking is important only for scientists who need to do experiments.
- b. Critical thinking enables us to interpret information and evaluate claims.
- c. Critical thinking is necessary for science and math, but it is not important for other disciplines.

(2) b. Critical thinking enables us to interpret information and evaluate claims.  
used to design research that will support or fail to support a theory. Research is a test of the hypotheses.

**ANSWERS:** (1) b. Theories are broad conceptual frameworks. Hypotheses are derived from theories and are

## 2.2 What Types of Studies Are Used in Psychological Research?

Once a researcher has defined a hypothesis, the next issue to be addressed is the type of research method to be used. There are three main types of research methods: *descriptive*, *correlational*, and *experimental*. These methods differ in the extent to which the researcher has control over the variables in the study. The amount of control over the variables in turn determines the type of conclusions the researcher can draw from the data.

All research involves variables. A **variable** is something in the world that can vary and that the researcher can manipulate (change), measure (evaluate), or both. In a study of texting and driving ability, some of the variables would be number of texts sent, number of texts received, familiarity with the texting device, how coordinated a person is, and driving ability and cell phone experience.

Scientists try to be as specific and as objective as possible when describing variables. Different terms are used to specify whether a variable is being manipulated or measured. An **independent variable** is the variable that gets manipulated. A **dependent variable** is the variable that gets measured, which is why it is sometimes called the *dependent measure*. Another way to think of the dependent variable is as the outcome that gets measured after a manipulation occurs. That is, the value of the dependent variable *depends* on the changes produced in the independent variable. Since independent variables are specific to the experimental research method, independent and dependent variables will be described more completely in that section of this chapter.

In addition to determining what variables will be studied, researchers must define these variables precisely and in ways that reflect the methods used to assess them. They do so by developing an **operational definition**. Operational definitions are important for research. They *qualify* (describe) and *quantify* (measure) variables so the variables can be understood objectively. The use of operational definitions enables other researchers to know precisely what variables were used, how they were manipulated, and how they were measured. These concrete details make it possible for other researchers to use identical methods in their attempts to replicate the findings.

For example, if you choose to study how driving performance is affected by cell phone use, how will you qualify cell phone use? Do you mean talking, texting, reading

## Learning Objectives

- Distinguish between descriptive studies, correlational studies, and experiments.
- List the advantages and disadvantages of different research methods.
- Explain the difference between random sampling and random assignment, and explain when each might be important.

### variable

Something in the world that can vary and that a researcher can manipulate (change), measure (evaluate), or both.

### independent variable

The variable that gets manipulated in a research study.

### dependent variable

The variable that gets measured in a research study.

### operational definition

A definition that *qualifies* (describes) and *quantifies* (measures) a variable so the variable can be understood objectively.



### FIGURE 2.7 Descriptive Methods

Observational studies—such as this one, using a one-way mirror—are a method that researchers use to describe behavior objectively.

#### descriptive research

Research methods that involve observing behavior to describe that behavior objectively and systematically.

#### case study

A descriptive research method that involves the intensive examination of an unusual person or organization.

content, or some combination of these activities? How will you then quantify cell phone use? Will you count how many times a person uses the cell phone in an hour? Then, how will you quantify and qualify driving performance so you can judge whether it is affected by cell phone use? Will you record the number of accidents, the closeness to cars up ahead, the reaction time to red lights or road hazards, speeding? The operational definitions for your study need to spell out the details of your variables.

## Descriptive Research Consists of Case Studies, Observation, and Self-Report Methods

**Descriptive research** involves observing behavior to *describe* that behavior objectively and systematically. Descriptive research helps scientists achieve the goals of describing what phenomena are and (sometimes) predicting when or with what other phenomena they may occur. However, by nature, descriptive research cannot achieve the goals of control and explanation (only the true experimental method, described later in this chapter, can do that).

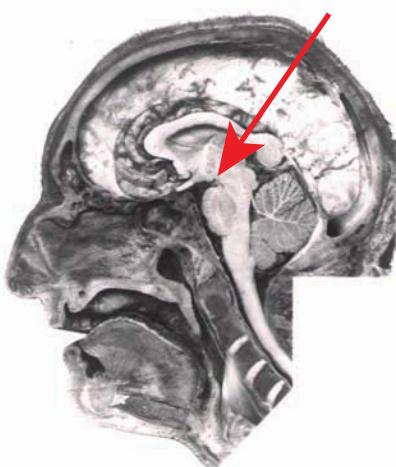
Descriptive methods are widely used to assess many types of behavior. For example, an observer performing descriptive research might record the types of foods that people eat in cafeterias, measure the time that people spend talking during an average conversation, count the number and types of mating behaviors that penguins engage in during their mating season, or tally the number of times poverty or mental illness is mentioned during a presidential debate (FIGURE 2.7). Each of these observations offers important information that can be used to describe current behavior and even predict future behavior. In no case does the investigator control the behavior being observed or explain why any particular behavior occurred.

There are three basic types of descriptive research methods: case studies; observations; and self-report methods and interviews.

**CASE STUDIES** A **case study** is the intensive examination of an unusual person or organization. By intensive examination, we mean observation, recording, and description. An individual might be selected for intensive study if he or she has a special or unique aspect, such as an exceptional memory, a rare disease, or a specific type of brain damage. An organization might be selected for intensive study because it is doing something very well (such as making a lot of money) or very poorly (such as losing a lot of money). The goal of a case study is to describe the events or experiences that lead up to or result from the exceptional aspect.

One famous case study in psychological science involves a young American man whose freak injury impaired his ability to remember new information (Squire, 1987). N.A. was born in 1938. After a brief stint in college, he joined the Air Force and was stationed in the Azores, where he was trained to be a radar technician. One night, he was assembling a model airplane in his room. His roommate was joking around with a miniature fencing foil, pretending to jab at the back of N.A.'s head. When N.A. turned around suddenly, his roommate accidentally stabbed N.A. through the nose and up into his brain (FIGURE 2.8).

Although N.A. seemed to recover from his injury in most ways, he developed extreme problems remembering events that happened to him during the day. He could remember events before his accident, and so he was able to live on his own, keeping his house tidy and regularly cutting his lawn. It was new information that he could not remember. He had trouble watching television because he forgot the storylines, and he had difficulty holding conversations because he forgot what others had just



### FIGURE 2.8 Case Study Data

In this image of Patient N.A., you can see where the miniature foil penetrated brain regions that had not traditionally been seen as involved in memory. This case study provided new insights into how the brain creates memories.

said. Subsequent studies of N.A.'s brain using imaging techniques revealed damage to specific regions not traditionally associated with memory difficulties (Squire, Amaral, Zola-Morgan, Kritchevsky, & Press, 1989). The case study of N.A. helped researchers develop new models of the brain mechanisms involved in memory.

However, not everyone who suffers damage to this brain region experiences the same types of problems as N.A. Such differences highlight the major problem with case studies. Because only one person or organization is the focus of a case study, scientists cannot tell from that study if the same thing would happen to other people or organizations who have the same experience(s). The findings from case studies do not necessarily *generalize*, or apply to the general population.

**OBSERVATIONAL STUDIES** Two main types of observational techniques are used in research: participant observation and naturalistic observation. In **participant observation** (FIGURE 2.9), the researcher is involved in the situation. In **naturalistic observation** (FIGURE 2.10), the observer is passive, separated from the situation and making no attempt to change or alter ongoing behavior.

**CODING** These observational techniques involve the systematic assessment and *coding* of overt behavior. Suppose you hear about a person who was texting while walking, stumbled off a curb, and was killed by an oncoming truck. You develop the hypothesis that using a cell phone while walking can cause problems with walking. How do you operationally define "problems with walking"? Once you have defined your terms, you need to code the forms of behavior you will observe. Your coding might involve written subjective assessments (e.g., "He almost got hit by a car when he walked into traffic"). Alternatively, your coding might use predefined categories (e.g., "1. Walked slowly," "2. Walked into traffic," "3. Stumbled"). Perhaps, after recording your data, you would create an index of impaired walking behavior by adding together the frequencies of each coded category. You might then compare the total number of coded behaviors when people were using a cell phone or not. Studies such as these have shown that cell phone use does impair walking ability (Schwебel et al., 2012; Stavrinos, Byington, & Schwedel, 2011). Pedestrian accidents—not all of them involving cell phones—kill more than 500 college-age students per year and injure more than 12,000 (National Highway Traffic Safety Administration, 2012b).

**REACTIVITY** When conducting observational research, scientists must consider the critical question of whether the observer should be visible. The concern here is that the presence of the observer might alter the behavior being observed. Such an alteration is called **reactivity**. People may feel compelled to make a positive impression on an observer, so they may act differently when they believe they are being observed. For example, drivers who know they were being observed might be less likely to use their cell phones.

Reactivity affected a now-famous series of studies on workplace conditions and productivity. Specifically, the researchers manipulated working conditions and then observed workers' behavior at the Hawthorne Works, a Western Electric manufacturing plant in Cicero, Illinois, between 1924 and 1933 (Olson, Hogan, & Santos, 2006; Roethlisberger & Dickson, 1939). The conditions included different levels of lighting, different pay incentives, and different break schedules. The main dependent variable was how long the workers took to complete certain tasks.

Throughout the studies, the workers knew they were being observed. Because of this awareness, they responded to changes in their working conditions by increasing productivity. The workers did not speed up continuously throughout the various studies. Instead, they worked faster at the start of each new manipulation, regardless of the nature of the manipulation (longer break, shorter break, one of various changes



**FIGURE 2.9**  
**Participant Observation**

The evolutionary psychologist and human behavioral ecologist Lawrence Sugiyama has conducted fieldwork in Ecuadorian Amazonia among the Shiwiar, Achuar, Shuar, and Zaparo peoples. Here, hunting with a bow and arrow, he is conducting a particularly active form of participant observation.

#### **participant observation**

A type of descriptive study in which the researcher is involved in the situation.

#### **naturalistic observation**

A type of descriptive study in which the researcher is a passive observer, separated from the situation and making no attempt to change or alter ongoing behavior.

#### **reactivity**

The phenomenon that occurs when knowledge that one is being observed alters the behavior being observed.



**FIGURE 2.10**  
**Naturalistic Observation**

Using naturalistic observation, the primatologist Jane Goodall observes a family of chimpanzees. Animals are more likely to act naturally in their native habitats than in captivity.

# Scientific Thinking

## The Hawthorne Effect

**HYPOTHESIS:** Being observed can lead participants to change their behavior.

**RESEARCH METHOD (OBSERVATIONAL):**

- 1 During studies of the effects of workplace conditions, the researchers manipulated several independent variables, such as the levels of lighting, pay incentives, and break schedules.
- 2 The researchers then measured the dependent variable, the speed at which workers did their jobs.



**RESULTS:** The workers' productivity increased when they were being observed, regardless of changes to their working conditions.

**CONCLUSION:** Being observed can lead participants to change their behavior because people often act in particular ways to make positive impressions.

**SOURCE:** Roethlisberger, F. J., & Dickson, W. J. (1939). *Management and the worker: An account of a research program conducted by the Western Electric Company, Hawthorne Works, Chicago*. Cambridge, MA: Harvard University Press.

to the pay system, and so on). The *Hawthorne effect* refers to changes in behavior that occur when people know that others are observing them (see “Scientific Thinking: The Hawthorne Effect”).

How might the Hawthorne effect operate in other studies? Consider a study of the effectiveness of a new reading program in elementary schools. Suppose that the teachers know they have been selected to try out a new program. They also know that their students’ reading progress will be reported to the schools’ superintendent. It is easy to see how these teachers might teach more enthusiastically or pay more attention to each child’s reading progress than would teachers using the old program. One likely outcome is that the students receiving the new program of instruction would show reading gains caused by the teachers’ increased attention and not by the new program. Thus, in general, observation should be as unobtrusive as possible.

**OBSERVER BIAS** In conducting observational research, scientists must guard against **observer bias**. This flaw consists of systematic errors in observation that occur because of an observer’s expectations.

Observer bias can especially be a problem if cultural norms favor inhibiting or expressing certain behaviors. For instance, in many societies women are freer to express sadness than men are. If observers are coding men’s and women’s facial expressions, they may be more likely to rate female expressions as indicating sadness because they believe that men are less likely to show sadness. Men’s expressions of sadness might be rated as annoyance or some other emotion. Likewise, in many societies women

### observer bias

Systematic errors in observation that occur because of an observer’s expectations.

are generally expected to be less assertive than men. Observers therefore might rate women as more assertive when exhibiting the same behavior as men. Cultural norms can affect both the participants' actions and the way observers perceive those actions.

**EXPERIMENTER EXPECTANCY EFFECT** There is evidence that observer expectations can even change the behavior being observed. This phenomenon is known as the **experimenter expectancy effect**.

In a classic study by the social psychologist Robert Rosenthal, college students trained rats to run a maze (Rosenthal & Fode, 1963). Half the students were told their rats were bred to be very good at running mazes. The other half were told their rats were bred to be poor performers. In reality, there were no genetic differences between the groups of rats. Nonetheless, when students believed they were training rats that were bred to be fast maze learners, their rats learned the task more quickly! Thus, these students' expectations altered how they treated their rats. This treatment in turn influenced the speed at which the rats learned. The students were not aware of their biased treatment, but it existed. Perhaps they supplied extra food when the rats reached the goal box at the end of the maze. Or perhaps they gave the rats inadvertent cues as to which way to turn in the maze. They might simply have stroked the rats more often (see "Scientific Thinking: Rosenthal's Study of Experimenter Expectancy Effects").

### experimenter expectancy effect

Actual change in the behavior of the people or nonhuman animals being observed that is due to the expectations of the observer.

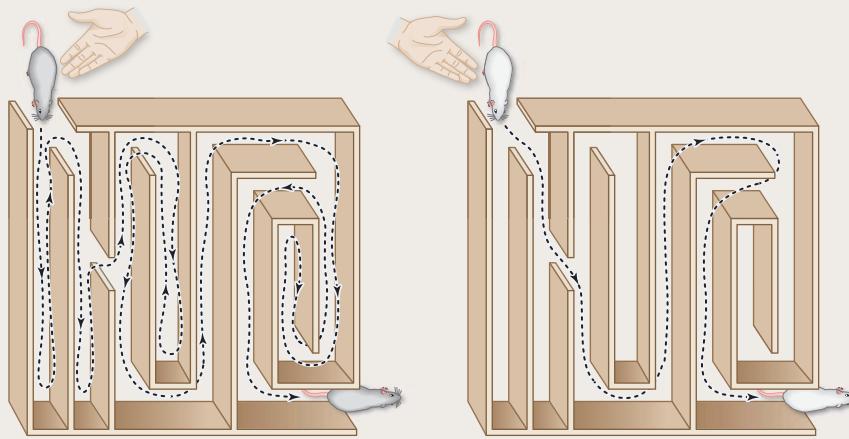
## Scientific Thinking

### Rosenthal's Study of Experimenter Expectancy Effects

**HYPOTHESIS:** Research participants' behavior will be affected by experimenters' biases.

#### RESEARCH METHOD (EXPERIMENT WITH TWO GROUPS):

- 1 One group of college students were given a group of rats and told to train them to run a maze. These students were told their rats were bred to be very poor at running mazes.
- 2 A second group of college students were given a group of rats to train that were genetically the same as the first group of rats. These students were told their rats were bred to be very good at running mazes.



**RESULTS:** The rats trained by the students who believed their rats were bred to be fast maze learners did learn the task more quickly.

**CONCLUSION:** The results for the two groups of rats differed because the students' expectations caused them to give off subtle cues that changed the rats' behavior.

**SOURCE:** Rosenthal, R., & Fode, K. L. (1963). The effect of experimenter bias on the performance of the albino rat. *Behavioral Science*, 8, 183–189.



**FIGURE 2.11**

### Self-Report Methods

Self-report methods, such as surveys or questionnaires, can be used to gather data from a large number of people. They are easy to administer, cost-efficient, and a relatively fast way to collect data.

How do researchers protect against experimenter expectancy effects? It is best if the person running the study is *blind* to, or unaware of, the study's hypotheses. For example, the study just described seemed to be about rats' speed in learning to run through a maze. Instead, it was designed to study experimenter expectancy effects. The students believed they were "experimenters" in the study, but they were actually the participants. Their work with the rats was the subject of the study, not the method. Thus, the students were led to expect certain results so that the researchers could determine whether the students' expectations affected the results of the rats' training.

**SELF-REPORTS AND INTERVIEWS** Ideally, observation is an unobtrusive approach for studying behavior. By contrast, asking people about themselves, their thoughts, their actions, and their feelings is a much more interactive way of collecting data. Methods of posing questions to participants include surveys, interviews, and questionnaires. The type of information sought ranges from demographic facts (e.g., ethnicity, age, religious affiliation) to past behaviors, personal attitudes, beliefs, and so on: "Have you ever used an illegal drug?" "Should people who drink and drive be jailed for a first offense?" "Are you comfortable sending food back to the kitchen in a restaurant when there is a problem?" Questions such as these require people to recall certain events from their lives or reflect on their mental or emotional states.

**Self-report methods**, such as surveys or questionnaires, can be used to gather data from a large number of people in a short time (**FIGURE 2.11**). Questions can be mailed out to a sample drawn from the population of interest or handed out in appropriate locations. They are easy to administer and cost-efficient.

Interviews, another type of interactive method, can be used successfully with groups that cannot be studied through surveys or questionnaires, such as young children. Interviews are also helpful in gaining a more in-depth view of a respondent's opinions, experiences, and attitudes. Thus, the answers from interviewees sometimes inspire avenues of inquiry that the researchers had not planned.

A problem common to all asking-based methods of data collection is that people often introduce biases into their answers. These biases make it difficult to discern an honest or true response. In particular, people may not reveal personal information that casts them in a negative light. We know we are not supposed to use cell phones while driving, and so we might be reluctant to admit regularly doing so. Researchers therefore have to consider the extent to which their questions produce *socially desirable responding*, or *faking good*, in which the person responds in a way that is most socially acceptable.

## Correlational Studies Describe and Predict How Variables Are Related

**Correlational studies** examine how variables are naturally related in the real world, without any attempt by the researcher to alter them or assign causation between them (**FIGURE 2.12**). Correlational studies are used to describe and predict relationships between variables. They cannot be used to determine the causal relationship between the variables.

Consider an example. On your college application, you likely had to provide a score from a standardized test, such as the SAT or ACT. Colleges require these numbers because standardized test scores have been shown to *correlate* with college success. That is, generally, people who score higher on standardized tests tend to perform better in college. However, does this mean that scoring well on a standardized test will *cause* you to do better in college? Or that doing well in school will *cause* you to do



**FIGURE 2.12**

### Correlational Studies

There may be a correlation between the extent to which parents are overweight and the extent to which their children are overweight. A correlational study cannot demonstrate the cause of this relationship, which may include biological propensities to gain weight, lack of exercise, and high-fat diets.

better on standardized tests? Absolutely not. Many people score well on tests but do not perform well in school. Alternatively, many people score poorly on standardized tests but enjoy great success in college.

**DIRECTION OF CORRELATION** When higher or lower values on one variable predict higher or lower values on a second variable, we say there is a **positive correlation** between them. A positive correlation describes a situation where both variables either increase or decrease together—they “move” in the same direction (**FIGURE 2.13A**). For example, people with higher ACT scores generally have higher college GPAs. People with lower ACT scores generally have lower college GPAs. However, remember that correlation does not equal “cause and effect.” Scoring higher or lower on the ACT will *not cause* you to earn a higher or lower GPA.

Remember, too, that *positive* in this case does *not* mean “good.” For example, there is a very strong positive correlation between smoking and cancer. There is nothing good about this relationship. The correlation simply describes how the two variables are related: In general, people who smoke experience higher rates of cancer. The more they smoke, the higher their risk of getting cancer.

Some variables are negatively correlated. In a **negative correlation**, the variables move in opposite directions. An increase in one variable predicts a decrease in the other variable. A decrease in one variable predicts an increase in the other variable (**FIGURE 2.13B**). Here, *negative* does not mean “bad.”

Consider exercise and weight. In general, the more people exercise, the less they weigh. People who take more vitamins experience fewer colds (Meyer, Meister, & Gaus, 2013).

Some variables are just not related. In this case, we say there is a **zero correlation**. That is, one variable is not predictably related to a second variable (**FIGURE 2.13C**). For example, there is a zero correlation between gender and intelligence. As two groups, men and women are equally smart.

**THINKING CRITICALLY ABOUT CORRELATIONS** Now that we have described the types of relationships that can exist, let us try to practice our critical thinking skills by interpreting what these relationships mean. Recall that there is generally a negative correlation between exercise and weight. For some people, however, there

### self-report methods

Methods of data collection in which people are asked to provide information about themselves, such as in surveys or questionnaires.

### correlational studies

A research method that describes and predicts how variables are naturally related in the real world, without any attempt by the researcher to alter them or assign causation between them.

### positive correlation

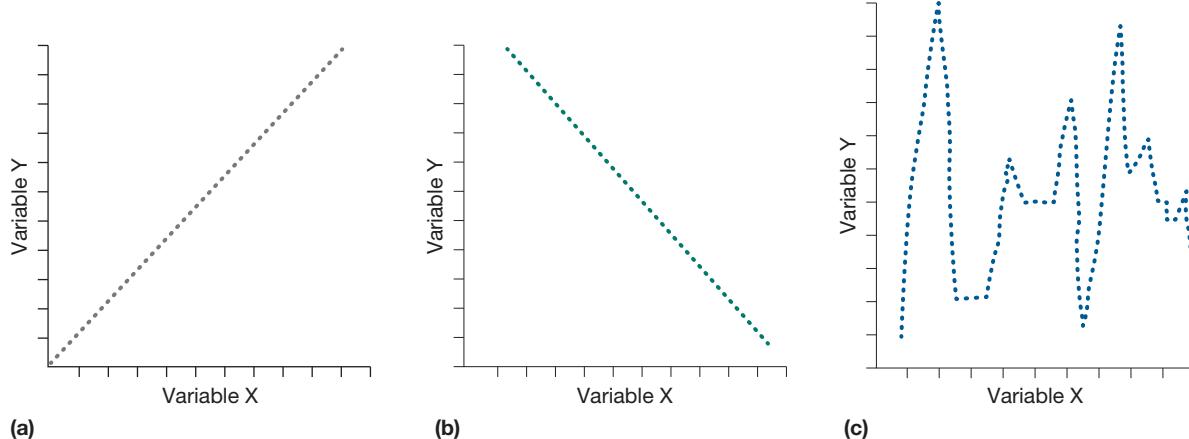
A relationship between two variables in which both variables either increase or decrease together.

### negative correlation

A relationship between two variables in which one variable increases when the other decreases.

### zero correlation

A relationship between two variables in which one variable is not predictably related to the other.



**FIGURE 2.13**  
**Direction of Correlation**

**(a)** In a positive correlation, both variables “move” in the same direction. **(b)** In a negative correlation, the variables move in opposite directions. **(c)** In a zero correlation, one variable is not predictably related to a second variable.

is a *positive* correlation between these variables. The more they exercise, the more weight they *gain*. Why? Because exercise builds muscle mass. So if the gain of muscle mass exceeds the loss of fat, exercise will actually increase weight. Sometimes, the same phenomena can exhibit a negative correlation or a positive correlation, depending on the specific circumstances.

Now consider the positive correlation between smoking and cancer. The more a person smokes, the greater that person's risk of cancer. Does that relationship mean smoking causes cancer? Not necessarily. Just because two things are related, even strongly related, does not mean that one is causing the other. Many genetic, behavioral, and environmental variables may contribute both to whether a person chooses to smoke and to whether the person gets cancer. Complications of this kind prevent researchers from drawing causal conclusions from correlational studies. Two such complications are the directionality problem and the third variable problem.

**DIRECTIONALITY PROBLEM** One problem with correlational studies is in knowing the direction of the relationship between variables. This sort of ambiguity is known as the **directionality problem**. Consider this example. Suppose you survey a large group of people about their sleeping habits and their levels of stress. Those who report sleeping little also report having a higher level of stress. Does lack of sleep increase stress levels, or does increased stress lead to shorter and worse sleep? Both scenarios seem plausible:

- Sleep (A) and stress (B) are correlated.
  - Does less sleep cause more stress? ( $A \rightarrow B$ )  
*or*  
▪ Does more stress cause less sleep? ( $B \rightarrow A$ )

**THIRD VARIABLE PROBLEM** Another drawback with all correlational studies is the **third variable problem**. Instead of variable A causing variable B, as a researcher might assume, it is possible that a third variable, C, causes both A and B. Consider the relationship between texting while driving and dangerous driving. It is possible that people who are risk takers in their daily lives are more likely to text while driving. It is also possible that these people are likely to drive dangerously. Thus, the cause of both texting while driving and dangerous driving is the third variable, risk-taking:

- Texting while driving (A) is correlated with driving dangerously (B).
  - Risk taking (C) causes some people to text while driving. ( $C \rightarrow A$ )  
*and*  
▪ Risk taking (C) causes some people to drive dangerously. ( $C \rightarrow B$ )

Indeed, research has shown that those who text while driving are also likely to engage in a variety of other risky behaviors, such as not wearing seatbelts, riding with a driver who had been drinking, or even drinking alcohol and driving (Olsen, Shults, & Eaton, 2013). Thus, it is possible that both texting while driving and dangerous driving generally result from risk taking, a third variable.

Sometimes the third variable is obvious. Suppose you were told that the more churches there are in a town, the greater the rate of crime. Would you conclude that churches cause crime? In looking for a third variable, you would realize that the population size of the town affects the number of churches and the frequency of crime. But sometimes third variables are not so obvious and may not even be identifiable. It turns out that even the relationship between smoking and cancer is plagued by the third variable problem. Evidence indicates that there is indeed a genetic predisposition—a built-in vulnerability to smoking—that can combine with environmental factors to

#### **directionality problem**

A problem encountered in correlational studies; the researchers find a relationship between two variables, but they cannot determine which variable may have caused changes in the other variable.

#### **third variable problem**

A problem that occurs when the researcher cannot directly manipulate variables; as a result, the researcher cannot be confident that another, unmeasured variable is not the actual cause of differences in the variables of interest.

increase the probability that some people will smoke *and* that they will develop lung cancer (Paz-Elizur et al., 2003; Thorgeirsson et al., 2008). Thus, it is impossible to conclude on the basis of correlational research that one of the variables is *causing* the other.

### ETHICAL REASONS FOR USING CORRELATIONAL DESIGNS

Despite such potentially serious problems, correlational studies are widely used in psychological science. Some research questions require correlational research designs for ethical reasons. For example, as mentioned earlier, it would be unethical to send drivers out into traffic and ask them to text as part of an experiment. Doing so would put the drivers and others at risk.

There are many important real-world experiences that we want to know about but would never expose people to as part of an experiment. Suppose you want to know if soldiers who experience severe trauma during combat have more difficulty learning new tasks after they return home than soldiers who have experienced less-severe trauma during combat. Even if you theorize that severely traumatic combat experiences *cause* later problems with learning, it would be unethical to induce trauma in some soldiers so that you could compare soldiers who had experienced different degrees of trauma. (Likewise, most research on psychopathology—psychological illness—uses the correlational method, because it is unethical to induce psychological disorders in people to study the effects.) For this research question, you would need to study the soldiers' ability to learn a new task after they had returned home. You might, for example, observe soldiers who were attempting to learn computer programming. The participants in your study would have to include some soldiers who had experienced severe trauma during combat and some who had experienced less-severe trauma during combat. You would want to see which group, on average, performed less well when learning the task.

**MAKING PREDICTIONS** Correlational studies can be used to determine that two variables are associated with each other. In the example just discussed, the variables would be trauma during combat and learning difficulties later in life. By establishing such connections, researchers are able to make predictions. If you found the association you expected between severe trauma during combat and learning difficulties, you could predict that soldiers who experience severe trauma during combat will—again, on average—have more difficulty learning new tasks when they return than soldiers who do not experience severe trauma during combat. Because your study drew on but did not control the soldiers' wartime experiences, however, you have not established a causal connection (**FIGURE 2.14**).

By providing important information about the natural relationships between variables, researchers are able to make valuable predictions. For example, correlational research has identified a strong relationship between depression and suicide. For this reason, clinical psychologists often assess symptoms of depression to determine suicide risk. Typically, researchers who use the correlational method use other statistical procedures to rule out potential third variables and problems with the direction of the effect. Once they have shown that a relationship between two variables holds even when potential third variables are taken into account, researchers can be more confident that the relationship is meaningful.

## The Experimental Method Controls and Explains

Scientists ideally want to explain what causes a phenomenon. For this reason, researchers rely on the experimental method. In experimental research, the researcher has maximal control over the situation. Only the experimental method



**FIGURE 2.14**  
**Correlation or Causation?**

According to the players on the 2013 Boston Red Sox baseball team, facial hair causes a person to play better baseball. After two newly bearded players made some game-saving plays, the rest of the team stopped shaving (Al-Khatib, 2013). Did their beards cause the Red Sox to win the World Series that year? The facial hair may have been correlated with winning, but it did not cause an increase in talent. The team won through ability, practice, and luck.

enables the researcher to control the conditions under which a phenomenon occurs and therefore to understand the cause of the phenomenon. In an **experiment**, the researcher manipulates one variable to measure the effect on a second variable.

An experiment also allows researchers to test multiple hypotheses to examine and refine their theory. Suppose researchers initially theorize that using a cell phone while driving impairs driving. This theory does not explain why the effect happens. Researchers can refine the theory to include possible mechanisms and then test hypotheses related to the refined versions of the more general theory.

Suppose the researchers then theorize that using a cell phone while driving impairs driving because drivers need to use their hands both to drive and to use cell phones. One hypothesis to test this theory is that using a hands-free cell phone while driving will cause fewer problems than holding the phone while talking and driving. Another hypothesis to test the same theory is that any use of the hands, such as eating, will impair driving.

An alternative theory is that taking your eyes off the road—to dial a number or read and respond to a text—is the main factor that affects driving. This theory might yield the hypothesis that any action the driver performs that takes his or her eyes off the road—such as reading a map or looking at the radio to change stations—will impair driving.

Yet another theory is that driving requires cognitive resources, such as the ability to pay attention and think about driving. This theory might yield the hypothesis that any activity the driver performs that requires attention or thought—such as thinking about a problem at school—will impair driving. Through experimentation, psychologists test hypotheses about the mechanisms they theorize are responsible for the effect they are studying.

**MANIPULATING VARIABLES** In an experiment, the independent variable (IV) is manipulated. That is, the researchers choose what the study participants do or are exposed to.

In a study on the effects of using a cell phone while driving, the IV would be the type of cell phone use. While in a driving simulator, some participants might simply hold the phone, some might have to answer questions over the phone, and some might have to read and answer text messages.

An IV has “levels,” meaning the different values that are manipulated by the researcher. All IVs must have at least two levels: a “treatment” level and a “comparison” level. In the study of cell phone use and driving ability, the people who actively used the cell phone received the “treatment.” A group of study participants who receive the treatment are the **experimental group**. Since in this hypothetical study, some participants talk on the cell phone and others text, there are actually two experimental groups.

In an experiment, you always want to compare your experimental group with at least one **control group**. A control group consists of similar (or identical) participants who receive everything the experimental group receives except for the treatment. In this example, the experimental group uses a cell phone to talk or text while driving. The control group simply holds a cell phone while driving. This use of a control group includes the possibility that simply the presence of a cell phone is disruptive. To test whether handling a cell phone is disruptive, the control group could be drivers not holding a cell phone.

The dependent variable (DV) is whatever behavioral effect is—or behavioral effects are—measured. For example, the researcher could measure how quickly the participants responded to red lights, how fast they drove, and the distance they maintained behind the car in front of them. The researcher would measure each of these DVs as a function of the IV, the type of cell phone use.

#### **experiment**

A research method that tests causal hypotheses by manipulating and measuring variables.

#### **experimental group**

The participants in an experiment who receive the treatment.

#### **control group**

The participants in an experiment who receive no intervention or who receive an intervention that is unrelated to the independent variable being investigated.

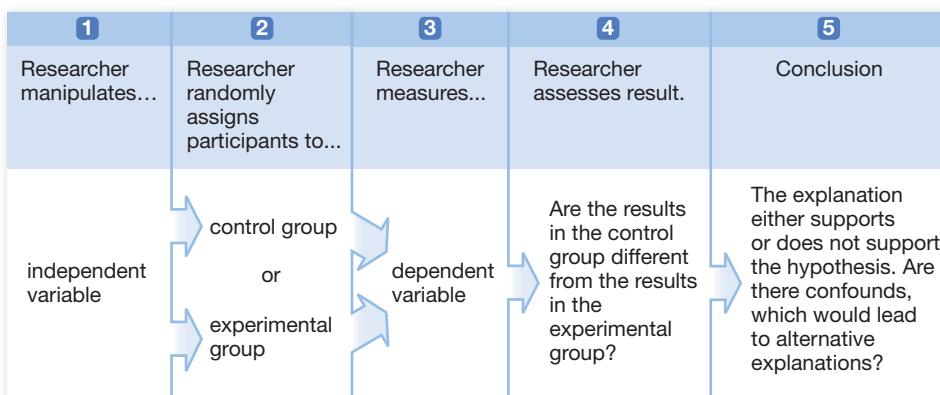
The benefit of an experiment is that the researcher can study the causal relationship between variables. If the IV (such as type of cell phone use) consistently influences the DV (such as driving performance), then the IV is assumed to cause the change in the DV.

**ESTABLISHING CAUSALITY** A properly performed experiment depends on rigorous control. Here, *control* means the steps taken by the researcher to minimize the possibility that anything other than the independent variable could be the cause of differences between the experimental and control groups.

A **confound** is anything that affects a dependent variable and that may unintentionally vary between the study's different experimental conditions. When conducting an experiment, a researcher needs to ensure that the only thing that varies is the independent variable. Control thus represents the foundation of the experimental approach, in that it allows the researcher to rule out alternative explanations for the observed data.

In the study of cell phone use and driving performance, what if a car with an automatic transmission is simulated to assess driving when participants are not using a cell phone, but a car with a manual transmission is simulated to assess performance when participants are texting? Given that manual transmissions require greater dexterity to operate than automatic transmissions, any apparent effect of texting on driving performance might actually be caused by the type of car and the fact that it requires greater use of the hands. In this example, the drivers' skills might be *confounded* with the type of transmission, making it impossible to determine the true effect of the texting.

Other potential confounds in research include changes in the sensitivity of the measuring instruments, such as a systematic change in a scale so that it weighs things more heavily in one condition than in another. Changes in the time of day or the season when the experiment is conducted can also confound the results. Suppose you conducted the texting and driving study so that the cell phone users were tested in snowy winter conditions and control participants were tested during dry, sunny weather. The road conditions associated with the season would be an obvious confound. The more confounds and thus alternative explanations that can be eliminated, the more confident a researcher can be that the change in the independent variable is causing the change (or effect) in the dependent variable. For this reason, researchers have to watch vigilantly for potential confounds. As consumers of research, we all need to think about confounds that could be causing particular results. (For a recap of the experimental method, see **FIGURE 2.15.**)



**FIGURE 2.15 The Experimental Method in Action**

Experiments examine how variables are related when one variable is manipulated by the researchers. The results can demonstrate causal relationships between the variables.

### confound

Anything that affects a dependent variable and that may unintentionally vary between the experimental conditions of a study.

**population**

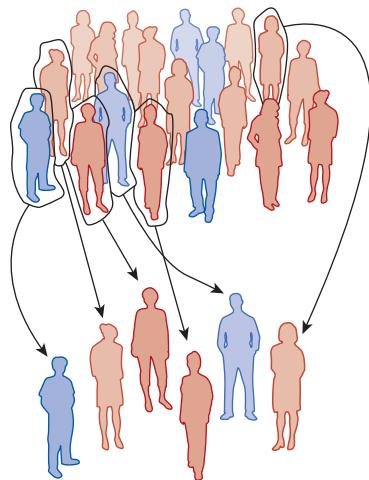
Everyone in the group the experimenter is interested in.

**sample**

A subset of a population.

**FIGURE 2.16****Population**

The population is the group researchers want to know about (e.g., U.S. college students). For the results of an experiment to be considered useful, the participants should be representative of the population.

**FIGURE 2.17****Random Sample**

A random sample is taken at random from the population (e.g., selecting students from schools throughout the United States). The best method for making this happen is random sampling.

## Participants Need to Be Carefully Selected and Randomly Assigned to Conditions

An important issue for any research method is how to select participants for the study. Psychologists typically want to know that their findings *generalize* to people beyond the individuals in the study. In studying the effects of cell phone use on driving skills, you ultimately would not focus on the behavior of the specific participants. Instead, you would seek to discover general laws about human behavior. If your results generalized to all people, that would enable you, other psychologists, and the rest of humanity to predict, in general, how cell phone use would affect driving performance. Other results, depending on the nature of the study, might generalize to all college students, to students who belong to sororities and fraternities, to women, to men over the age of 45, and so on.

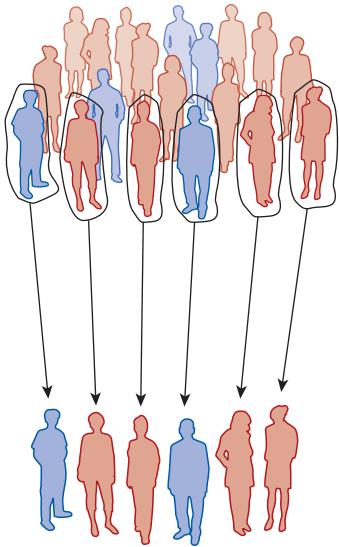
**POPULATION AND SAMPLING** The group you want to know about is the **population** (**FIGURE 2.16**). To learn about the population, you study a subset from it. That subset, the people you actually study, is the **sample**. *Sampling* is the process by which you select people from the population to be in the sample. In a case study, the sample size is one. The sample should represent the population, and the best method for making this happen is *random sampling* (**FIGURE 2.17**). This method gives each member of the population an equal chance of being chosen to participate. Further, larger samples yield more accurate results (**FIGURE 2.18**). However, sample size is often limited by resource constraints, such as time, money, and space in which to work.

Most of the time, a researcher will use a *convenience sample* (**FIGURE 2.19**). As the term implies, this sample consists of people who are conveniently available for the study. However, because a convenience sample does not use random sampling, the sample is likely to be biased. For instance, a sample of students at a small religious school may differ from a sample of students at a large state university. Researchers acknowledge the limitations of their samples when they present their findings.

**FIGURE 2.18 Larger Samples**

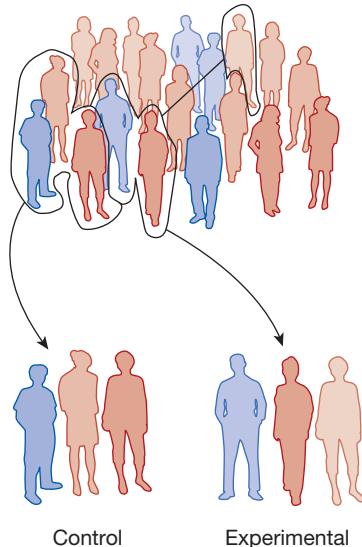
Suppose researchers want to compare how many women go to the beach versus how many men do. Why might the results be more accurate if the researchers use a large sample (such as the big picture here) rather than a small sample (such as the detail)?





**FIGURE 2.19 Convenience Sample**

A convenience sample is taken from an available subgroup in the population (e.g., students at a particular school). Most of the time, circumstances force researchers to use a convenience sample.



**FIGURE 2.20 Random Assignment**

In random assignment, participants are assigned at random to the control group or the experimental group. Random assignment is used when the experimenter wants to test a causal hypothesis.

**RANDOM ASSIGNMENT** Once researchers obtain a representative sample of the population, they use **random assignment** to assign participants to the experimental and control groups (**FIGURE 2.20**). Random assignment gives each potential research participant an equal chance of being assigned to any level of the independent variable.

For your study, there might be three levels: holding a cell phone, answering questions verbally over the phone, and answering questions by texting. First, you would gather participants by taking either a random sample or a convenience sample from the population. Then, to randomly assign those participants, you might have them draw numbers from a hat to determine who is assigned to the control group (holding the phone) and to each experimental group (one talking and the other texting).

Of course, individual differences are bound to exist among participants. For example, any of your groups might include some people with less experience with cell phones and some people who talk or text a great deal, some people with excellent and experienced driving skills and some people with comparably weaker skills. But these differences will tend to average out when participants are assigned to either the control or experimental groups randomly, so that the groups are equivalent *on average*. Random assignment tends to balance out known and unknown factors.

If random assignment to groups is not truly random, and groups are not equivalent because participants in different groups differ in unexpected ways, the condition is known as **selection bias** (also known as *selection threat*). Suppose you have two of the experimental conditions described earlier: a group assigned to hold the phone and a group assigned to respond to text messages. What happens if the group assigned to hold the phone includes many college students with lots of experience using cell phones and the other group includes many older adults who have minimal experience texting? How would you know if the people in the different conditions of the study are equivalent? You could match each group for age, sex, cell phone use habits, and so on, but you can never be sure that you have assessed all possible factors that may differ between the groups. Not using random assignment can create confounds that limit causal claims.

#### random assignment

Placing research participants into the conditions of an experiment in such a way that each participant has an equal chance of being assigned to any level of the independent variable.

#### selection bias

In an experiment, unintended differences between the participants in different groups; it could be caused by nonrandom assignment to groups.



(a)



(b)

### FIGURE 2.21 Cross-Cultural Studies

**(a)** The living space and treasured possessions of a family in Japan, for example, differ from **(b)** those of a family in Mali. Cross-cultural researchers might study how either family would react to crowding or to the loss of its possessions.

**GENERALIZING ACROSS CULTURES** It is important for researchers to assess how well their results generalize to other samples, particularly in cross-cultural research (Henrich, Heine, & Norenzayan, 2010). One difficulty in comparing people from different cultures is that some ideas and practices do not translate easily across cultures, just as some words do not translate easily into other languages. Apparent differences between cultures may reflect such differences in language, or they may reflect participants' relative willingness to report things about themselves publicly. A central challenge for cross-cultural researchers is to refine their measurements to rule out these kinds of alternative explanations (**FIGURE 2.21**).

Some psychological traits are the same across all cultures (e.g., care for the young). Others differ widely across cultures (e.g., behaviors expected of adolescents). **Culturally sensitive research** takes into account the significant role that culture plays in how people think, feel, and act (Adair & Kagitcibasi, 1995; Zebian, Alamuddin, Mallouf, & Chatila, 2007). Scientists use culturally sensitive practices so that their research respects—and perhaps reflects—the “shared system of meaning” that each culture transmits from one generation to the next (Betancourt & Lopez, 1993, p. 630).

In cities with diverse populations, such as Toronto, London, and Los Angeles, cultural differences exist among different groups of people living in the same neighborhoods and having close daily contact. Researchers therefore need to be sensitive to cultural differences even when they are studying people in the same neighborhood or the same school. Researchers must also guard against applying a psychological concept from one culture to another without considering whether the concept is the same in both cultures. For example, Japanese children’s attachment to their parents looks quite different from the attachment styles common among North American children (Miyake, 1993).

## Summing Up

### What Types of Studies Are Used in Psychological Research?

- Three main types of studies are used in psychological research: descriptive, correlational, and experimental.
- Descriptive and correlational designs are useful for describing and predicting behavior, but they do not allow researchers to assess causality.
- Only experiments allow researchers to determine causality.
- In an experiment, a researcher manipulates an independent variable to study how it affects a dependent variable, while controlling all other potential influences.
- When performing research, sampling allows researchers to draw a representative sample of the population and generalize the findings to the population.

## Measuring Up

1. The main reason researchers randomly assign participants to different conditions in an experiment is that
  - a. it is easier to assign participants to different conditions than it is to find people who naturally fit into different conditions.
  - b. random assignment controls for any intuitions the participants may have at the start of the experiment.
  - c. random assignment is used when there are ethical reasons for not using observational or correlational research designs.

### culturally sensitive research

Studies that take into account the role that culture plays in determining thoughts, feelings, and actions.

- d. random assignment helps to ensure that the experimental groups are (on average) equal and that any difference in the dependent variable is due to the participants' being in different experimental groups.
2. Match each of the following with the research method it describes. Choose from among case study, correlational, experimental, naturalistic observation, and survey.
- An end-of-semester course evaluation that asks students to rate the class.
  - Collection of data showing that on average, students who studied more hours for a psychology examination earned higher grades.
  - A study comparing the driving performance between people randomly assigned to text while driving or to drive without distractions.
  - A research report describing a person with an extremely rare psychological disorder.
  - A study comparing voting preferences for people in wealthy neighborhoods compared to people in middle-class neighborhoods.
  - A study describing how 8-year-old children interacted on their school playground.
  - A study comparing tumor size in three groups of mice, each given a different dose of nicotine.
  - A study comparing the rate of cancer in people who are nonsmokers, light smokers, or heavy smokers.

(2) a. survey; b. correlational; c. experimental; d. case study; e. correlational; f. naturalistic observation; g. experimental; h. correlational.

and that any difference in the dependent variable is due to the participants' being in different experimental groups.

**ANSWERS:** (1) d. random assignment helps to ensure that the experimental groups are (on average) equal

## 2.3 What Are the Ethics Governing Psychological Research?

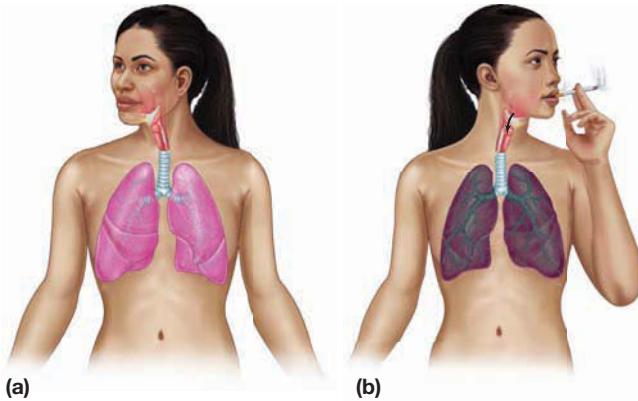
### There Are Ethical Issues to Consider in Research with Human Participants

Psychologists want to know why and how we act, think, feel, and perceive the way we do. In other words, they want to understand the human condition. As a result, it makes sense for psychological studies to involve human participants. As in any science that studies human behavior, however, there are limits to how researchers can manipulate what people do in studies. For ethical and practical reasons, researchers cannot always use the experimental method.

Consider the question of whether smoking causes cancer. To explain why a phenomenon (e.g., cancer) occurs, experimenters must control the conditions under which that phenomenon occurs. And to establish that a cause-and-effect relationship exists between variables, experimenters need to use random assignment. So to determine causality between smoking and cancer, some study participants would have to be randomly “forced” to smoke a controlled number of cigarettes in a specific fashion for a controlled amount of time, while an equal number of different (but similar) participants would have to be randomly “prevented” from smoking for the same amount of time. However, ethics prevent researchers from randomly forcing people to smoke, so researchers cannot experimentally answer this question using human participants (**FIGURE 2.22**).

### Learning Objectives

- Identify ethical issues associated with conducting psychological research on human participants.
- Apply ethical principles to conducting research on animals, identifying the key issues regarding the humane treatment of animal subjects.



**FIGURE 2.22**  
**Research on Smoking and Cancer**

Researchers can compare (a) a nonsmoker's lungs with (b) a smoker's lungs. They can compare the rates of cancer in nonsmokers with the rates of cancer in smokers. Ethically, however, they cannot perform an experiment that entails randomly forcing study participants to smoke, even though such experiments could help establish a link between smoking and cancer.

#### institutional review boards (IRBs)

Groups of people responsible for reviewing proposed research to ensure that it meets the accepted standards of science and provides for the physical and emotional well-being of research participants.

When conducting research, we have to carefully consider ethics. Is the study intended to do good for humanity? What exactly will the participants be asked to do? Are the requests reasonable, or will they put the participants in danger of physical or emotional harm over the short term or long term? Are the burdens of research shared justly among the portions of society that are involved?

**INSTITUTIONAL REVIEW BOARDS (IRBS)** To ensure the health and well-being of all study participants, strict guidelines exist regarding research. These guidelines are shared by all places where research is conducted, including colleges, universities, and research institutes. **Institutional review boards (IRBs)** are the guardians of the guidelines.

Convened at schools and other institutions where research is done, IRBs consist of administrators, legal advisers, trained scholars, and members of the community. At least one member of the IRB must not be a scientist. The purpose of the IRB is to review all proposed research to ensure that it meets scientific and ethical standards to protect the safety and welfare of participants. Most scientific journals today ask for proof of IRB approval before publishing research results. Four key issues are addressed in the IRB approval process: privacy, relative risks, informed consent, and access to data.

**PRIVACY** One major ethical concern about research is the expectation of privacy. Two main aspects of privacy must be considered. One aspect is *confidentiality*. This term means that personal, identifying information about participants absolutely cannot be shared with others. Research participants must be assured that any such information collected in a study will remain private. In some studies, *anonymity* is used. Although this term is often confused with confidentiality, anonymity means that the researchers do not collect personal, identifying information. Without such information, responses can never be traced to any individual. Anonymity helps make participants comfortable enough to respond honestly.

Another important aspect of privacy is participants' knowledge that they are being studied. If behaviors are going to be observed, is it okay to observe people without their knowledge? This question obviously depends on what sorts of behaviors researchers might be observing. If the behaviors tend to occur in public rather than in private, researchers might be less concerned about observing people without their knowledge. For example, even without their knowledge, it would be okay to observe people texting while they walk. The concern over privacy is compounded by the ever-increasing technology for monitoring people remotely. Although it might be useful to compare men's and women's behaviors in public bathrooms, it would not be acceptable to install discreet video cameras to monitor people in restrooms.

**RELATIVE RISKS OF PARTICIPATION** Another ethical issue is the relative risk to participants' mental or physical health. Researchers must always remain aware of what they are asking of participants. They cannot ask people to endure unreasonable amounts of pain or of discomfort, either from stimuli or from the manner in which data measurements are taken.

Fortunately, in the vast majority of studies being conducted, these types of concerns are not an issue. However, even though risk may be low, researchers still have to think carefully about the potential for risk. Therefore, the IRB will evaluate the relative trade-off between risk and benefit for any research study it approves. In some cases, the potential gains from the research may require asking participants to

expose themselves to some risk to obtain important findings. The *risk/benefit ratio* is an analysis of whether the research is important enough to warrant placing participants at risk. If a study has any risk associated with it, then participants must be notified *before* they agree to participate. This process is known as *informed consent*.

**INFORMED CONSENT** Research involving human participants is a partnership based on mutual respect and trust. People who volunteer for psychological research have the right to know what will happen to them during the course of the study. Compensating people either with money or course credit for their participation in research does not alter this fundamental right. Ethical standards require giving people all relevant information that might affect their willingness to become participants (**FIGURE 2.23**).

Informed consent means that participants make a knowledgeable decision to participate. Typically, researchers obtain informed consent in writing (**FIGURE 2.24**). In observational studies of public behavior, the observed individuals remain anonymous to the researchers to protect their privacy, so informed consent is not required. People under the age of 18 and those with severe cognitive disabilities or mental health disorders cannot legally provide informed consent. If such an individual is to participate in a study, a legal guardian must grant permission.

It is not always possible to inform participants fully about a study's details. If knowing the study's specific goals may alter the participants' behavior, thereby rendering the results meaningless, researchers may need to use deception. That is, they might mislead the participants about the study's goals or not fully reveal what will take place. Researchers use deception only when other methods are not appropriate and when the deception does not involve situations that would strongly affect people's willingness to participate. If deception is used, a careful *debriefing* must take place after the study's completion. Here, the researchers inform the participants of the study's goals. They also explain the need for deception, to eliminate or counteract any negative effects produced by the deception.

**ACCESS TO DATA** No matter what research method they use, researchers must also consider who will have access to the data they collect. Participant confidentiality should always be guarded carefully so that personal information is not linked publicly to the study's findings. When participants are told that their information will remain confidential, the implicit promise is that their information will be kept secret or made available to only the few people who need to know it. Often the quality and accuracy of data depend on the participants' certainty that their responses will be kept confidential. When emotionally or legally sensitive topics are involved, people are especially likely to provide valid data after they are promised confidentiality.

## There Are Ethical Issues to Consider in Research with Animals

Many people have ethical concerns about research with nonhuman animals. These concerns involve two questions: Does research threaten the health and well-being of the animals? And is it fair to the animals to study them to improve the human condition?



**FIGURE 2.23**  
**Informed Consent**

The need for informed consent is illustrated by one of the most infamous unethical studies. Between 1932 and 1972, the U.S. Public Health Service and the Tuskegee Institute, in Alabama, studied the natural progression of untreated syphilis in rural African American men. Without their knowledge, 400 impoverished men with the venereal disease were randomly assigned to receive treatment or not. In 1987, the U.S. government publicly apologized to the participants and their families. Here, President Bill Clinton and Vice President Al Gore appear at a news conference with participant Herman Shaw.

## Dartmouth College Brain Imaging Center

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6207 Moore Hall  
Hanover, New Hampshire 03755

### Consent to Participate in Research

#### **Title of Study:** Neural Correlates of Scene Processing

**Introduction:** You are being asked to participate in a research study. Your participation is voluntary. If you are a student your decision whether or not to participate will not have any affect on your academic status. Please feel free to ask questions at any time if there is anything you do not understand.

**Purpose of this fMRI investigation.** The goal of these experiments is to investigate how the brain functions while people are viewing different images or watching different visual scenes (e.g., people, objects, landscapes) and how that relates to responses to various stimuli and behavior. You are being asked to participate because you are a normal healthy adult. Your participation allows us to determine basic principles of brain organization. The data obtained through your participation will be included with that from other subjects as part of a scientific study to appear in the peer-reviewed literature.

**FIGURE 2.24**  
**Informed Consent Form**

This portion of an approved form gives you a sense of how researchers typically obtain informed consent in writing.

# Using Psychology in Your Life



## Should I Participate in Psychological Research?

Someday, perhaps even this term, you will be invited to participate in a psychological research study (**FIGURE 2.25**). Because psychological researchers are a creative lot, they enjoy figuring out clever ways to study the human mind. As a result, participation in research can be a lot of fun. Even studies that simply involve answering self-report questions offer opportunities to reflect on your inner world and behaviors. However, some students in introductory psychology may worry that researchers will trick them into doing something they do not want to do. Others may feel anxious because they have no idea what to expect once they walk through the doors of a psychology laboratory. Understanding the ethical principles that guide psychological research arms potential research participants—like yourself—with insight about what to expect when participating in a study.

Psychologists in the United States conduct their studies according to a set of ethical principles called the Belmont Report. To read the full report, go to <http://www.hhs.gov/ohrp/humansubjects/guidance/belmont.html>. These principles, a few of which are described below, guide many aspects of participants' experiences in research studies.

First, no one can force you to participate in a study. Although many psychology departments "require" students to



**FIGURE 2.25**  
**Student Participation in Psychological Research**

These students are enjoying the opportunity to contribute to scientific knowledge. Join them by participating in a study.

participate in research as part of their course work, they offer students alternatives for fulfilling this requirement. For example, in some departments, students can read and write about articles published in journals in lieu of participating in research. Even if you volunteer for a study, you have the right to discontinue your participation at any time, for any reason, and without penalty. And you can skip any questions you do not care to answer, perhaps because you find them intrusive or offensive. You are in the driver's seat when it comes to choosing if, and to what extent, you would like to participate in a study.

Second, you are legally and ethically entitled to know what you are getting

**HEALTH AND WELL-BEING** Research with animals must always be conducted with regard to the health and well-being of the animals. Federal mandates govern the care and use of animals in research, and these mandates are strictly enforced. An accounting and reporting system is in place for all institutions conducting animal research. Violators of the mandates are prevented from conducting further research.

All colleges, universities, and research institutions conducting research with vertebrate animals must have an Institutional Animal Care and Use Committee (IACUC). This committee is like an institutional review board (discussed earlier), but it evaluates animal research proposals. In addition to scientists and nonscientists, every IACUC includes a certified doctor of veterinary medicine, who must review each proposal to ensure that the research animals will be treated properly before, during, and after the study.

Research facilities must comply with the IACUC's standards. Facilities are given scheduled and surprise inspections. Noncompliance can result in suspended or terminated research, monetary fines, federal charges, and even jail time.



"WHAT IT COMES DOWN TO IS YOU HAVE TO FIND OUT WHAT REACTION THEY'RE LOOKING FOR, AND YOU GIVE THEM THAT REACTION." *V. Harris*

into so you can make an informed decision about participating. Although the researchers will not be able to reveal their exact research questions and hypotheses, they will be able to tell you the general purpose of the study and the kinds of activities you will be asked to complete. You might be asked to answer questions, perform computer tasks, engage in moderate physical activity, navigate a real or imagined social scenario, rate the appeal of different consumer products, and so on. In addition, researchers must tell you about the risks and potential benefits faced by participants. For example, researchers studying ostracism would inform participants they might find the experimental tasks distressing. So even before a study begins, you will actually know a good deal about the research.

Third, after you complete the study, you can expect the researchers to debrief you. During the debriefing, the researchers will tell you if they used deception in the study. For example, if you participate in a study about cooperation, you might learn during the debriefing that the “person” you interacted with online was really a computer program.

Finally, you can expect that the data you provide will remain confidential. To protect confidentiality, the researchers will remove all identifying information, such as your name, from any data you submit.

They will store consent forms separately from data, password-protect electronic files containing sensitive information, and keep all files in a secure location.

While researchers are governed by formal ethical guidelines (in addition to their own moral compasses), good study participants also engage the research process respectfully. When you sign up to participate in a study, record the researcher’s contact information in case an emergency arises and you are unable to fulfill your commitment. Arrive at your session on time, and bring any paperwork your institution might require in order for you to receive class credit for your participation. During the study, minimize potential distractions, such as by turning off your cell phone. And, importantly, ask questions! One of the benefits of volunteering in research is learning firsthand about the research process. Getting answers to your questions helps you derive this benefit.

Study participants are essential to the research enterprise. The principles and procedures described here emerged out of concern for the well-being of participants. Understanding your rights and responsibilities prepares you to contribute meaningfully and confidently, without fear of trickery or unknown risks, to psychologists’ efforts to understand and improve the human condition. On behalf of psychologists everywhere, thank you for joining us in this endeavor.

**FAIRNESS** Animals are not used to study aspects of the human condition because animals are not the same as humans. However, some species share similarities with humans that make them good “models” for particular human behaviors or conditions. For example, as you will learn more about in Chapters 3 and 7, the human brain has a region called the hippocampus, and people with damage to this region suffer from memory loss. It would be unethical for researchers to reproduce hippocampal damage in people in an effort to find treatments for their memory loss. However, many animals also have a hippocampus, and they display similar types of memory loss when this region is damaged. As a way to help humans, researchers thus may find it necessary to conduct animal research. For example, scientists can damage or temporarily “turn off” the hippocampus in rats or mice to test treatments that may help to reverse the resulting memory loss.

Another valuable animal model is the transgenic mouse. Transgenic mice have been produced by manipulating genes in developing mouse embryos—for example, by inserting strands of foreign DNA into the genes. Studying the behavior of mice with



**FIGURE 2.26 Animal Research**

Researchers observe the behaviors of transgenic mice to understand how certain genes affect behavior.

specific genetic changes allows scientists to discover the role that genes play in behavior and disease (**FIGURE 2.26**).

Are such treatments fair to the research animals? Scientists must balance their concern for individual animals' lives with their concern for humanity's future. The pursuit of scientific knowledge and medical advances is noble, and animals' lives are given a kind of nobility—a meaning—when the animals are used respectfully in research.

## Summing Up

### What Are the Ethics Governing Psychological Research?

- Psychological researchers must consider the ethical consequences of their data collection.
- Strict rules govern research with both human participants and research animals.
- Each research study with human participants is evaluated for scientific and ethical validity. The evaluation is done by an Institutional Review Board (IRB), which consists of scientists and nonscientists.
- The four key issues addressed in the IRB approval process are privacy, relative risks, informed consent, and access to data.
- Each animal research study is evaluated by an Institutional Animal Care and Use Committee (IACUC), which consists of scientists, nonscientists, and a veterinarian. The IACUC ensures the ethical treatment of the animals before, during, and after the study.

## Measuring Up

Determine whether each of the following statements is true (T) or false (F).

1. \_\_\_\_\_ Confidentiality is the same as anonymity, because both mean that study results are not revealed to nonscientists.
2. \_\_\_\_\_ Even if research does not involve deception, it still needs to be approved by an IRB.
3. \_\_\_\_\_ Informed consent is required only when a research study poses a risk to safety or health.
4. \_\_\_\_\_ Students who participate in psychological research to receive course credit give up their right to privacy.
5. \_\_\_\_\_ Ethical rules govern research with both human participants and animals.
6. \_\_\_\_\_ Any team of animal researchers must include a veterinarian.
7. \_\_\_\_\_ Violations in the ethical treatment of animals in research may be justified if the study has sufficient scientific merit.
8. \_\_\_\_\_ An IRB reviews proposals for research with humans, whereas an IACUC reviews animal research proposals.

**ANSWERS:** 1. F; 2. T; 3. F; 4. F; 5. T; 6. F; 7. F; 8. T

## 2.4 How Are Data Analyzed and Evaluated?

So far, this chapter has presented the essential elements of scientific inquiry in psychology: thinking critically; asking an empirical question using theories, hypotheses, and research; deciding what type of study to run; considering the ethics of particular research; collecting and presenting data. This section focuses on the data. Specifically, it examines the characteristics that make for good data and the statistical procedures that researchers use to analyze data.

### Good Research Requires Valid, Reliable, and Accurate Data

If you collect data to answer a research question, the data must be *valid*. That is, the data must accurately measure the constructs (concepts) that you think they measure, accurately represent phenomena that occur outside of the laboratory, and accurately reveal effects due specifically and only to manipulation of the independent variable.

**Construct validity** is the extent to which variables measure what they are supposed to measure. For example, suppose at the end of the semester your psychology professor gives you a final examination that consists of chemistry problems. This kind of final examination would lack construct validity—it would not accurately measure your knowledge of psychology (**FIGURE 2.27**).

Now imagine you are a psychological researcher. You hypothesize that “A students” spend more time studying than “C students.” To test your hypothesis, you assess the amount of time students spend studying. However, what if “C students” tended to do other things—such as sleeping, playing video games, or checking their Facebook status—while they claimed to be studying? If this were the case, the data would not accurately reflect studying and would therefore lack construct validity.

**External validity** is the degree to which the findings of a study can be generalized to other people, settings, or situations. A study is externally valid if (1) the participants accurately represent the intended population, and (2) the variables were manipulated and measured in ways similar to how they occur in the “real world.”

**Internal validity** is the degree to which the effects observed in an experiment are due to the independent variable and not to confounds. For data to be internally valid, the experiment must be well designed and well controlled. That is, all the participants must be as similar as possible, and there must be a control group. Only by comparing experimental groups to control groups can you determine that any changes observed in the experimental groups are caused by the independent variable and not something else (for example, practice or the passage of time).

To understand internal validity, suppose you are conducting a study to see if special tutoring causes better grades. You randomly sample 50 students from introductory psychology classes at your university and give them special tutoring for 6 weeks. At the end of the 6 weeks, you find that the students earned an average score of

### Learning Objectives

- Identify three characteristics that reflect the quality of data.
- Describe measures of central tendency and variability.
- Describe the correlation coefficient.
- Discuss the rationale for inferential statistics.

#### construct validity

The extent to which variables measure what they are supposed to measure.

#### external validity

The degree to which the findings of a study can be generalized to other people, settings, or situations.

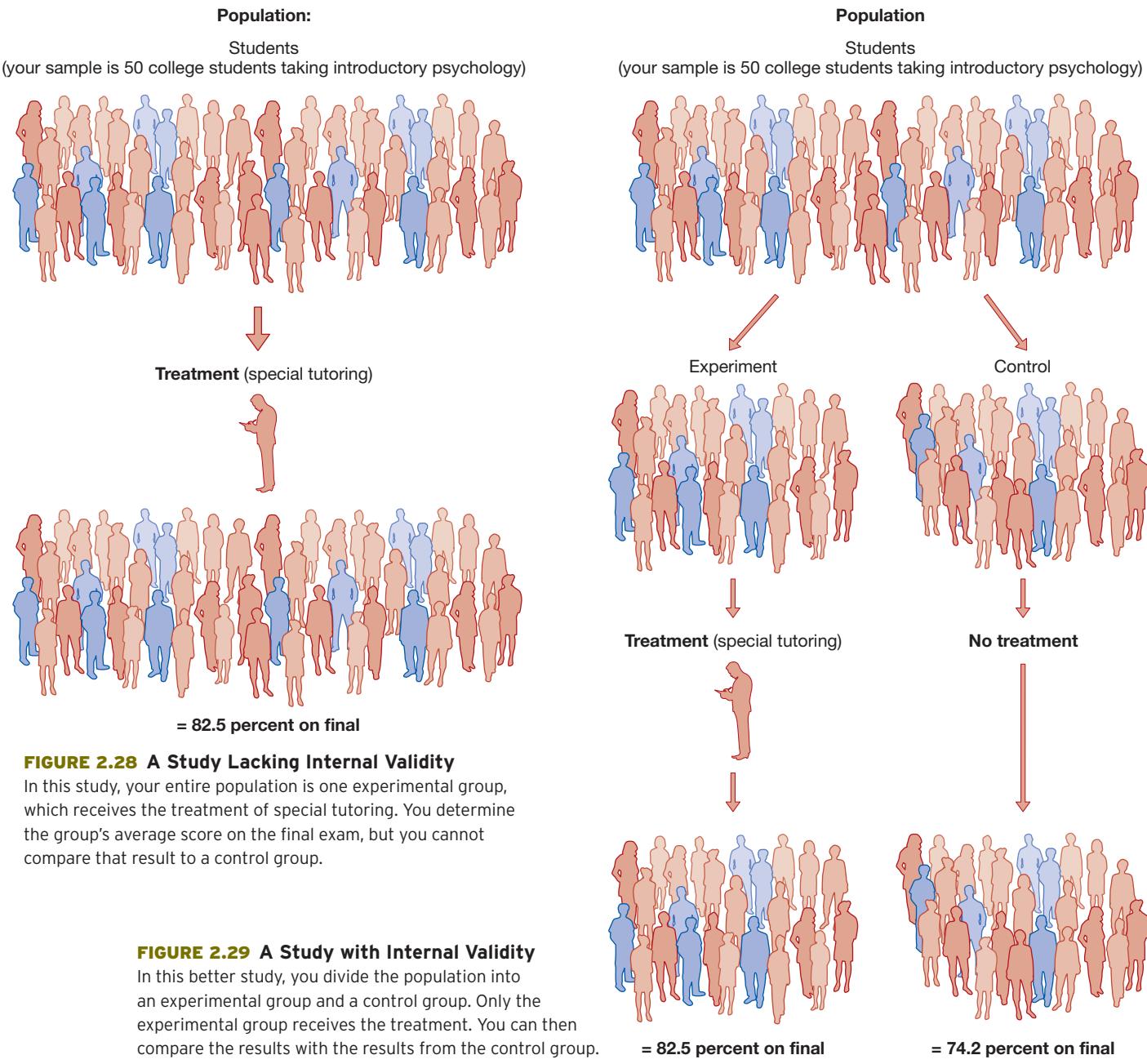
#### internal validity

The degree to which the effects observed in an experiment are due to the independent variable and not confounds.

- PROBLEMS**
- 109.5°      120°      180°
- 5.19. Rank the following molecular geometries in order of increasing bond angles: (a) trigonal planar; (b) linear; (c) tetrahedral.  $c < a < b$
- 5.20. Rank the following molecules in order of increasing bond angles: (a) NH<sub>3</sub>; (b) CH<sub>4</sub>; (c) H<sub>2</sub>O.  $H_2O < NH_3 < CH_4$
- 5.21. Which of the following electron-group geometries is not consistent with a linear molecular geometry, assuming three atoms per molecule? (a) tetrahedral; (b) octahedral; (c) trigonal planar.
- 5.22. How many lone pairs of electrons would there have to be on a SN = 6 central atom for it to have a linear molecular geometry?  
 $6 - 2 = 4$

**FIGURE 2.27**  
**Construct Validity**

Imagine having to answer questions like this on your psychology final. The results would lack construct validity because the course is about psychology, not chemistry.



**FIGURE 2.28 A Study Lacking Internal Validity**

In this study, your entire population is one experimental group, which receives the treatment of special tutoring. You determine the group's average score on the final exam, but you cannot compare that result to a control group.

**FIGURE 2.29 A Study with Internal Validity**

In this better study, you divide the population into an experimental group and a control group. Only the experimental group receives the treatment. You can then compare the results with the results from the control group.

#### reliability

The degree to which a measure is stable and consistent over time.

#### accuracy

The degree to which an experimental measure is free from error.

#### descriptive statistics

Statistics that summarize the data collected in a study.

82.5 percent on the final exam (**FIGURE 2.28**). Can you conclude that the tutoring caused the grade? Wait a minute. How do you know if 82.5 is an improvement over scores typically received on the exam? Maybe all students in introductory psychology “mature” over the semester so that the average final exam grade is about 82, regardless of tutoring. Or perhaps having 6 weeks of practice taking other tests results in higher exam grades, even without tutoring. Only by having an equal comparison group—a control group of students who are otherwise identical to the experimental group except for the treatment—can you determine if your treatment caused the observed effect.

Indeed, a better way to conduct this study would be to sample 50 students from the class, randomly assign 25 of them the special tutoring for 6 weeks (the experimental group), and not give any special treatment to the other 25 (the control group). Say the 25 students in the experimental group average 82.5 percent on the final exam and the 25 students in the control group average 74.2 percent (**FIGURE 2.29**). The control

group was similar in every way to the experimental group. As a result, you are fairly safe to conclude that the tutoring—not something else—led to higher exam grades. Thus, having a true control group can ensure that a study maintains internal validity.

Another important aspect of data is **reliability**, the stability and consistency of a measure over time. If the measurement is reliable, the data collected will not vary substantially over time. For instance, one option for measuring the duration of studying would be to have an observer use a stopwatch. There is likely to be some variability, however, in when the observer starts and stops the watch relative to when the student actually starts studying. As a consequence, the data in this scenario would be less reliable than data collected by an online homework system that measured how much time students spent working on assignments.

The third and final characteristic of good data is **accuracy**, the degree to which the measure is error free. A measure may be reliable but still not be accurate. Psychologists think about this problem by turning it on its head and asking, How do errors creep into a measure?

Suppose you use a stopwatch to measure the duration of studying. The problem with this method is that each measurement will tend to overestimate or underestimate the duration (because of human error or variability in recording times). This type of problem is known as a *random error* or *unsystematic error*. Although an error is introduced into each measurement, the value of the error differs each time (**FIGURE 2.30**). But suppose the stopwatch has a glitch, so that it always overstates the time measured by 1 minute. This type of problem is known as a *systematic error* or *bias*, because the amount of error introduced into each measurement is constant (**FIGURE 2.31**). Generally, systematic error is more problematic than random error because the latter tends to average out over time and therefore is less likely to produce inaccurate results.

## Descriptive Statistics Provide a Summary of the Data

The first step in evaluating data is to inspect the *raw values*. This term refers to data that are as close as possible to the form in which they were collected. In examining raw data, researchers look for errors in data recording. For instance, they assess whether any of the responses seem especially unlikely (e.g., studying for 72 hours or a 113-year-old participant). Once the researchers are satisfied that the data make sense, they summarize the basic patterns using **descriptive statistics**. These mathematical forms provide an overall summary of the study's results. For example, they might show how the participants, on average, performed in one condition compared with another.

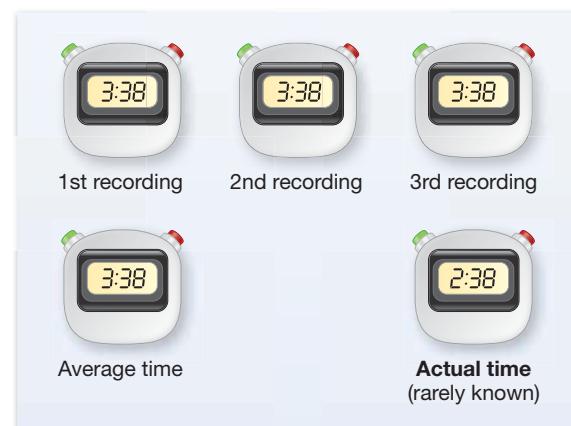
The simplest descriptive statistics are measures of **central tendency**. This single value describes a typical response or the behavior of the group as a whole. The most intuitive measure of central tendency is the **mean**, the arithmetic average of a set of numbers. The class average on an exam is an example of a mean score. Consider our earlier hypothetical study of cell phone use and driving performance. A basic way to summarize the data would be to calculate the means for driving performances using number of seconds they took to travel once around a virtual racetrack in a driving simulator: You would calculate one mean for when participants were simply holding a cell phone and a second mean for when they were texting. If texting affects driving, you would expect to see a difference in the means between those holding cell phones and those using them.



**FIGURE 2.30**

### Random Error

Data accuracy can be affected by random error. For example, say you time the same research participant several times. The stopwatch works accurately. But because your judgment of starting and stopping times differs each time, the degree of error varies each time.



**FIGURE 2.31**

### Systematic Error

Data accuracy can be affected by systematic error. Here, you time the same research participant several times, but the stopwatch is off by 1 minute each time. The degree of error is constant.

#### central tendency

A measure that represents the typical response or the behavior of a group as a whole.

#### mean

A measure of central tendency that is the arithmetic average of a set of numbers.

### median

A measure of central tendency that is the value in a set of numbers that falls exactly halfway between the lowest and highest values.

### mode

A measure of central tendency that is the most frequent score or value in a set of numbers.

A second measure of central tendency is the **median**, the value in a set of numbers that falls exactly halfway between the lowest and highest values. For instance, if you received the median score on a test, half the people who took the test scored lower than you and half the people scored higher.

Sometimes researchers will summarize data using a median instead of a mean because if one or two numbers in the set are dramatically larger or smaller than all the others, the mean will give either an inflated or a deflated summary of the average. This effect occurs in studies of average incomes. Perhaps approximately 50 percent of Americans make more than \$52,000 per year, but a small percentage of people make so much more (multiple millions or billions for the richest) that the mean income is much higher (around \$70,000) than the median and is not an accurate measure of what most people earn. The median provides a better estimate of how much money the average person makes.

A third measure of central tendency is the **mode**, the most frequent score or value in a set of numbers. For instance, the modal number of children in an American family is two, which means that more American families have two children than any other number of children. (For examples of how to calculate all three central tendency measures, see **FIGURE 2.32**.)

You measure the number of seconds that 11 participants take to drive around a simulated racetrack:

- One takes 55 seconds.
- Two take 45 seconds.
- One takes 69 seconds.
- One takes 48 seconds.
- One takes 56 seconds.
- One takes 65 seconds.
- One takes 60 seconds.
- One takes 38 seconds.
- One takes 34 seconds.
- One takes 25 seconds.

Written in ascending order, the number of seconds per participant looks like this:

25 34 38 45 45 48 55 56 60 65 69

### Mean

The arithmetic average of a set of numbers

$$\frac{\text{total # of seconds}}{\text{total # of participants}} = \frac{25+34+38+45+45+48+55+56+60+65+69}{11} = \frac{540}{11} = 49$$

### Median

The value that falls exactly halfway between the lowest and highest values

25 34 38 45 45 48 55 56 60 65 69 = 48

### Mode

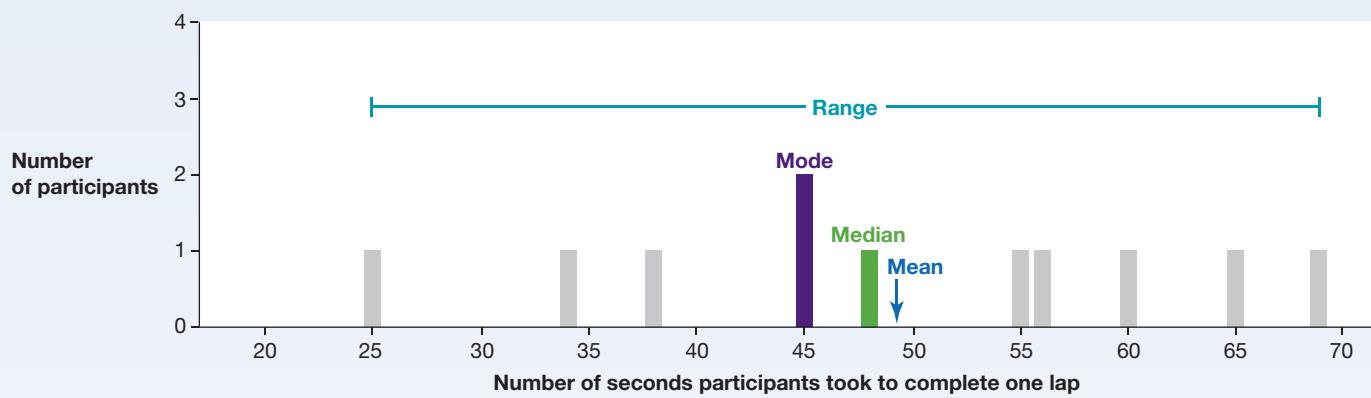
The most frequent score or value in a set of numbers

25 34 38 45 45 48 55 56 60 65 69 = 45

### Range

The distance between the largest and smallest values

25 34 38 45 45 48 55 56 60 65 69 = 69 - 25 = 44



**FIGURE 2.32 Descriptive Statistics**

Descriptive statistics are used to summarize a data set and to measure the central tendency and variability in a set of numbers. The mean, median, and mode are different measures of central tendency. The range is a measure of variability.

In addition to measures of central tendency, another important characteristic of data is the **variability** in a set of numbers. In many respects, the mean is meaningless without knowing the variability. Variability refers to how widely dispersed the values are from each other and from the mean. The most common measure of variability—how spread out the scores are—is the **standard deviation**. This measure reflects how far away each value is, on average, from the mean. For instance, if the mean score for an exam is 75 percent and the standard deviation is 5, most people scored between 70 percent and 80 percent. If the mean remains the same but the standard deviation becomes 15, most people scored between 60 and 90—a much larger spread.

Another measure of how spread out scores are is the **range**, the distance between the largest value and the smallest value. Often the range is not very useful, however, because it is based on only those two scores.

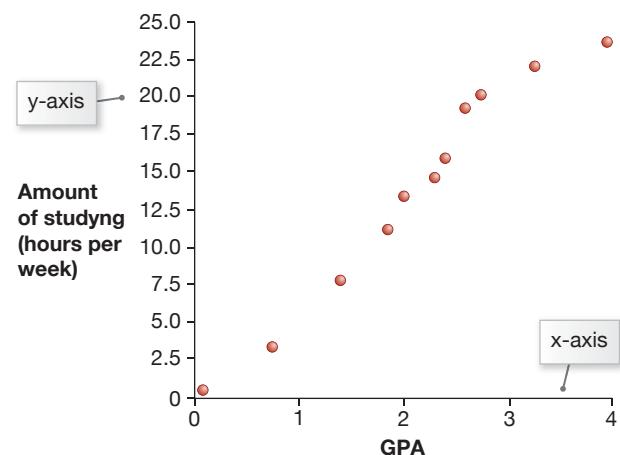
## Correlations Describe the Relationships Between Variables

The descriptive statistics discussed so far are used for summarizing the central tendency and variability in a set of numbers. Descriptive statistics can also be used to summarize how two variables relate to each other. The first step in examining the relationship between two variables is to create a **scatterplot**. This type of graph provides a convenient picture of the data (**FIGURE 2.33**).

In analyzing the relationship between two variables, researchers can compute a **correlation coefficient**. This descriptive statistic provides a numerical value (between -1.0 and +1.0) that indicates the strength of the relationship between the two variables. Some sample scatterplots and their corresponding correlation coefficients can be seen in **FIGURE 2.34**.

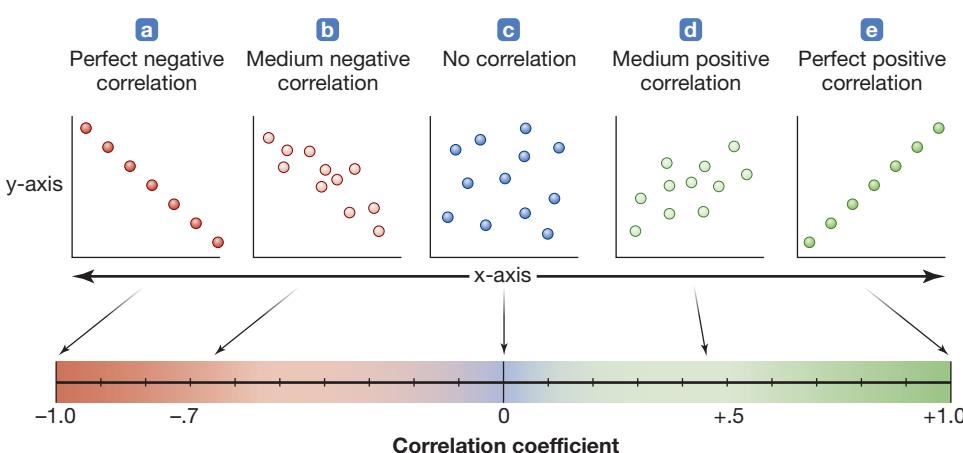
Here we are considering only one type of relationship: a linear relationship. In a linear relationship, an increase or decrease in one variable is associated with an increase or decrease in the other variable. When a linear relationship is strong, knowing how people measure on one variable enables you to predict how they will measure on the other variable. The two types of linear relationship, as discussed in Section 2.2, are positive correlations and negative correlations.

If two variables have a positive correlation, they increase or decrease together. For example, the more people study, the more likely they are to have a higher GPA.



**FIGURE 2.33 Scatterplots**

Scatterplots are graphs that illustrate the relationship between two variables. In general, as this scatterplot indicates, study time is positively correlated with GPA.

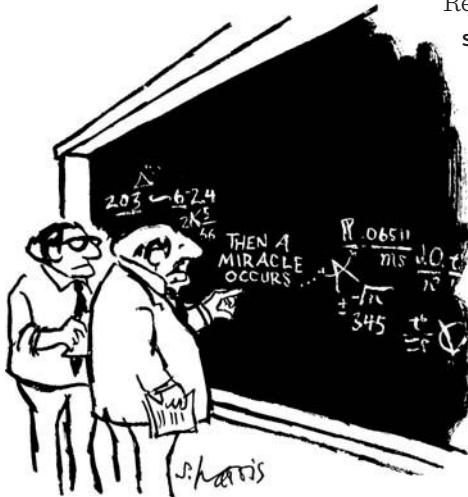


**FIGURE 2.34 Correlation Coefficient**

Correlations can have different values between -1.0 and +1.0. These values reveal different kinds of relationships between two variables. The greater the scatter of values, the lower the correlation. A perfect correlation occurs when all the values fall on a straight line.

A perfect positive correlation is indicated by a value of +1.0 (see Figure 2.34e). If two variables have a *negative correlation*, as one increases in value, the other decreases in value. For example, as people spend more time multitasking, they become less able to study for their exams, so multitasking and GPA have a negative correlation. A perfect negative correlation is indicated by a value of -1.0 (see Figure 2.34a). If two variables show no apparent relationship, the value of the correlation will be a number close to zero (assuming a linear relationship for the purposes of this discussion; see Figure 2.34c).

## Inferential Statistics Permit Generalizations



**"I think you should be more explicit here in step two."**

Researchers use descriptive statistics to summarize data sets. They use **inferential statistics** to determine whether effects actually exist in the populations from which samples were drawn. For instance, suppose you find that the mean driving performance for drivers using cell phones is lower than the mean driving performance for those not using cell phones. How different do these means need to be for you to conclude that using a cell phone reduces people's ability to drive?

A review of 206 studies found that the skills necessary to drive a car can become impaired when people perform a second task (i.e., multitask; Ferdinand & Menachemi, 2014). Pretend for a moment, however, that cell phone use does not influence driving performance. If you measure the driving performances of those using cell phones and those not using them, just by chance there will be some variability in the mean performance of the two groups. The key is that if cell phone use does not affect driving performance, the probability of showing a large difference between the two means is relatively small. Researchers use statistical techniques to determine if the differences among the sample means are (probably) chance variations or if they reflect actual differences in the populations.

When the results obtained from a study would be very unlikely to occur if there really were no differences between the groups of subjects, the researchers conclude that the results are *statistically significant*. According to generally accepted standards, researchers typically conclude there is a significant effect only if the obtained results would occur by chance less than 5 percent of the time.

**META-ANALYSIS** Meta-analysis is a type of study that, as its name implies, is an analysis of multiple analyses. In other words, it is a study of studies that have already been conducted. With meta-analysis, many studies that have addressed the same issue are combined and summarized in one "study of studies." The study we described that looked at 206 studies is an example of a meta-analysis.

Suppose that ten studies have been conducted on men's and women's effectiveness as leaders. Among these ten studies, five found no differences, two favored women, and three favored men. Researchers conducting a meta-analysis would not just count up the numbers of different findings from the research literature. Instead, they would weight more heavily those studies that had larger samples. Large samples are more likely to provide more accurate reflections of what is true in populations (see Figure 2.18). The researchers would also consider the size of each effect. That is, they would factor in whether each study found a large difference, a small difference, or no difference between the groups being compared—in this case, between women and men. (The researchers who conducted such a meta-analysis on men's and women's effectiveness found no overall differences; Eagly, Karau, & Makhijani, 1995.)

Because meta-analysis combines the results of separate studies, many researchers believe that meta-analysis provides stronger evidence than the results of any single study. As discussed earlier in this chapter, we can be more confident about results when the research findings are replicated. Meta-analysis has the concept of replication built into it.

### inferential statistics

A set of assumptions and procedures used to evaluate the likelihood that an observed effect is present in the population from which the sample was drawn.

### meta-analysis

A "study of studies" that combines the findings of multiple studies to arrive at a conclusion.

# What to Believe? Using Psychological Reasoning

## Misunderstanding Statistics: Should You Bet on a Hot Hand?

In 2013, the Miami Heat's LeBron James set a basketball record by scoring over 30 points, while making over 60 percent of his shots, for six straight games (FIGURE 2.35). In the seventh game, James's streak ended, when he scored on just under 60 percent of his shots.

Did James have a "hot hand" during this streak? Are there periods when particular athletes are relaxed, confident, and "in the zone" and play particularly well? Team members try to get the ball to a person who has made several shots in a row, because they think the person's hot hand will increase their chance of winning. Many sports journalists, coaches, athletes, and fans believe in some form of the phenomenon.

The psychologist Tom Gilovich and his colleagues (1985) conducted a series of studies on the hot hand, to assemble beliefs about the phenomenon and to scientifically examine whether it exists. Their first and crucial step was to turn the idea of the hot hand into a testable hypothesis: After a basketball shooter has made two or three shots in a row, that shooter will be more likely to make the next shot than after missing the last two or three shots. When the researchers asked 100 knowledgeable basketball fans, 91 agreed that this outcome was likely. If their belief were accurate, then an analysis of shooting records should show the increased probability of making a shot after previous successes than after previous failures.

To test whether the "hot hand" hypothesis is supported by evidence, Gilovich and colleagues examined the shooting records of the Philadelphia 76ers during the 1980-81 season. The 76ers kept records of the order that shots had been taken as well as the outcome of those shots. The data did not support the hot hand hypothesis.

When events in the world seem to happen in clusters, people develop explanations such as the hot hand to make sense of them.

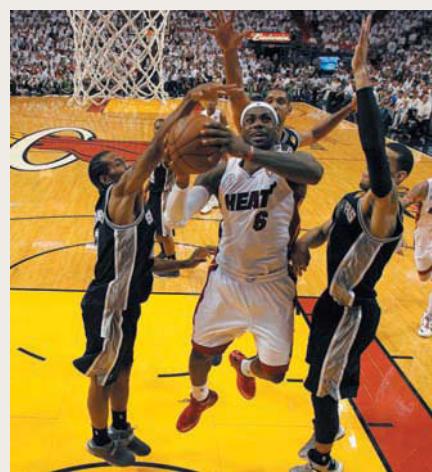
Players made on average 51 percent of their shots after making one previous shot, 50 percent after making two previous shots, and 46 percent after making three in a row. If anything, players were more likely to be successful after prior misses: 51 percent after one prior miss, 53 percent after two prior misses, and 56 percent after missing three in a row.

As a critical thinker, you might wonder whether the defensive team stops the streak by paying more attention to hot shooters and putting in more effort to defend against them. To test this alternative explanation, Gilovich and colleagues examined free throw shooting, where the defense does not matter and players get two free shots. Players made about the same number of second free throws whether they made the first one or not.

Upon hearing the results of this research, the famous coach Red Auerbach, of the Boston Celtics, exclaimed, "Who is this guy? So he makes a study. I couldn't care less" (Gilovich, 1991, p. 17). Any one study might be questionable until other scientists have replicated the findings. Indeed, the occasional study supports the idea of the hot hand for some sports, such as volleyball (Raab, Gula, & Gigerenzer, 2011). However, a meta-analysis of all studies that have examined this phenomenon enables us to consider all the outcomes at the same time. A meta-analysis of 22 published articles found no evidence that the hot hand exists (Avugos, Köppen,

Czienkowski, Raab, & Bar-Eli, 2012). Athletes across various sports were no more likely to be successful after a prior success than after a prior failure.

Why do people believe in shooting streaks? The best answer is that people are bad at recognizing chance outcomes. If a fair coin is flipped, most people intuitively expect there to be a greater alternation of heads and tails than occurs by chance. If you flip a coin 20 times in a row, however, there will be streaks of six heads or tails in a row 10 percent of the time, five in a row 25 percent of the time, and four in a row 50 percent of the time. Players do occasionally sink the shot six, seven, or eight times in a row, but these occurrences do not happen any more often than what we expect from chance, given the number of shots they take in a game.



**FIGURE 2.35 LeBron James**

Did a "hot hand" help James during his six-game streak in 2013?

## Summing Up

### How Are Data Analyzed and Evaluated?

- Data must be valid, reliable, and accurate.
- Data should have construct validity (measure what they are supposed to measure), external validity (apply outside of the laboratory), and internal validity (accurately represent effects of manipulations to the independent variable and not something else).
- Descriptive statistics summarize data. They include measures of central tendency and measures of variability.
- Measures of central tendency—such as the mean, median, and mode—indicate the typical response of a group as a whole.

- Measures of variability, such as standard deviation, indicate how widely numbers are distributed about the mean or average score.
- A correlation coefficient describes the strength and nature of the relationship between two variables.
- Inferential statistics indicate whether the results of a study reflect a true difference between groups or are likely to be due to chance.
- Meta-analysis combines the results of several studies to arrive at a conclusion.

## Measuring Up

1. When researchers want to summarize in a single number all the data they collect, they compute a measure of central tendency. Here are hypothetical data for a study in which 10 people in a sample consumed alcohol. The researchers measured the number of glasses of alcohol each person consumed and assessed her or his motor control after consuming the alcohol. The scores on motor control ranged from 1 (poor motor control) to 10 (good motor control). Compute the mean, median, and mode for the amount of alcohol consumed and the ratings of motor control.

Amount of alcohol consumed	Rating of motor control
3	4
1	9
5	1
2	7
3	5
3	3
1	8
4	2
5	1
2	6

2. Which is an accurate description of the rationale for inferential statistics?
  - a. When the means of two sample groups are significantly different, we still need to compute a mean value for each population before we can conclude that the groups really are different.
  - b. When the means of two sample groups are significantly different, we can be fairly certain that we did not make any mistakes in our research.
  - c. When the means of two sample groups are significantly different, we can be certain that the data are not correlated.
  - d. When the means of two sample groups are significantly different, we can infer that the differences between the two groups are unlikely to be due to chance.

**ANSWERS:** (1) Amount of alcohol consumed: mean = 2.9, median = 3, and mode = 3; rating of motor control: mean = 4.6, median = 4.5, and mode = 1.  
(2) d. When the means of two sample groups are significantly different, we can infer that the populations the groups were selected from are different.

# Your Chapter Review

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## Chapter Summary

### 2.1 How Is the Scientific Method Used in Psychological Research?

- **Science Has Four Primary Goals:** The four primary goals of science are *description* (describing what a phenomenon is), *prediction* (predicting when a phenomenon might occur), *control* (controlling the conditions under which a phenomenon occurs), and *explanation* (explaining what causes a phenomenon to occur).
- **Critical Thinking Means Questioning and Evaluating Information:** Critical thinking is a skill that helps people become educated consumers of information. Critical thinkers question claims, seek definitions for the parts of the claims, and evaluate the claims by looking for well-supported evidence.
- **The Scientific Method Aids Critical Thinking:** Scientific inquiry relies on objective methods and empirical evidence to answer testable questions. The scientific method is based on the use of theories to generate hypotheses that can be tested by collecting objective data through research. After a theory has been formulated based on observing a phenomenon, the six steps of the scientific method are forming a hypothesis based on the theory, reviewing the scientific literature to see how people are testing the theory, choosing a research method to test the hypothesis, conducting the research study, analyzing the data, and disseminating the results.
- **Unexpected Findings Can Be Valuable:** Unexpected (serendipitous) discoveries sometimes occur, but only researchers who are prepared to recognize their importance will benefit from them. Although unexpected findings can suggest new theories, these findings must be replicated and elaborated.

### 2.2 What Types of Studies Are Used in Psychological Research?

- **Descriptive Research Consists of Case Studies, Observation, and Self-Report Methods:** Researchers observe and describe naturally occurring behaviors to provide a systematic and objective analysis. A case study, one kind of descriptive study, examines an individual or an organization. However, the findings of a case study may not generalize. Data collected by observation must be defined clearly and collected systematically. Bias may occur in the data because the participants are aware they are being observed or because of the observer's expectations. Surveys, questionnaires, and interviews can be used to directly ask people about their thoughts and behaviors. Self-report data may be biased by the

respondents' desire to present themselves in a particular way (e.g., smart, honest).

- **Correlational Studies Describe and Predict How Variables Are Related:** Correlational studies are used to examine how variables are naturally related in the real world. These studies cannot be used to establish causality or the direction of a relationship (which variable caused changes in another variable).
- **The Experimental Method Controls and Explains:** Experiments can demonstrate causal relationship between variables. Experimenters manipulate one variable, the independent variable, to determine its effect on another, the dependent variable. Research participants are divided into experimental groups and control groups. The experimental groups experience the independent variable, and the control groups are used for comparison. In evaluating the data, researchers must look for confounds—elements, other than the variables, that may have affected the results.
- **Random Sampling and Random Assignment Are Important for Research:** Researchers sample participants from the population they want to study (e.g., drivers). They use random sampling when everyone in the population is equally likely to participate in the study, a condition that rarely occurs. To establish causality between an intervention and an outcome, random assignment must be used. When random assignment is used, all participants have an equal chance of being assigned to any level of the independent variable, and preexisting differences between the groups are controlled. Culturally sensitive research recognizes the differences among people from different cultural groups and from different language backgrounds.

### 2.3 What Are the Ethics Governing Psychological Research?

- **There Are Ethical Issues to Consider in Research with Human Participants:** Ethical research is governed by principles that ensure fair, safe, and informed treatment of participants. Institutional review boards (IRBs) judge study proposals to make sure the studies will be ethically sound.
- **There Are Ethical Issues to Consider in Research with Animals:** Research involving nonhuman animals provides useful, although simpler, models of behavior and of genetics. The purpose of such research may be to learn about animals' behavior or to make inferences about human behavior. Institutional Animal Care and Use Committee (IACUC) judges study proposals to make sure the animals will be treated properly. Researchers must weigh their concerns for individual animals against their concerns for humanity's future.

## 2.4 How Are Data Analyzed and Evaluated?

- **Good Research Requires Valid, Reliable, and Accurate Data:** Data must be meaningful (valid) and their measurement reliable (i.e., consistent and stable) and accurate.
- **Descriptive Statistics Provide a Summary of the Data:** Measures of central tendency (mean, median, and mode) and variability are used to describe data.
- **Correlations Describe the Relationships Between Variables:** A correlation coefficient is a descriptive statistic that describes the

strength and direction of the relationship between two variables. Correlations close to zero signify weak relationships. Correlations near +1.0 or -1.0 signify strong relationships.

- **Inferential Statistics Permit Generalizations:** Inferential statistics allow us to decide whether differences between two or more groups are probably just chance variations (suggesting that the populations the groups were drawn from are the same) or whether they reflect true differences in the populations being compared. Meta-analysis combines the results of several studies to arrive at a conclusion.

## Key Terms

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accuracy, p. 65	external validity, p. 63	random assignment, p. 55
case study, p. 44	hypothesis, p. 37	reactivity, p. 45
central tendency, p. 65	independent variable, p. 43	reliability, p. 65
confound, p. 53	inferential statistics, p. 68	replication, p. 41
construct validity, p. 63	institutional review boards (IRBs), p. 58	research, p. 37
control group, p. 52	internal validity, p. 63	sample, p. 54
correlation coefficient, p. 67	mean, p. 65	scatterplot, p. 67
correlational studies, p. 48	median, p. 66	scientific method, p. 37
culturally sensitive research, p. 56	meta-analysis, p. 68	selection bias, p. 55
data, p. 34	mode, p. 66	self-report methods, p. 48
dependent variable, p. 43	naturalistic observation, p. 45	standard deviation, p. 67
descriptive research, p. 44	negative correlation, p. 49	theory, p. 37
descriptive statistics, p. 65	observer bias, p. 46	third variable problem, p. 50
directionality problem, p. 50	operational definition, p. 43	variability, p. 67
experiment, p. 52	participant observation, p. 45	variable, p. 43
experimental group, p. 52	population, p. 54	zero correlation, p. 49
experimenter expectancy effect, p. 47	positive correlation, p. 49	

# Practice Test

1. Which of the following is a technique that increases scientists' confidence in the findings from a given research study?
  - a. meta-analysis
  - b. operationalization of variables
  - c. replication
  - d. serendipity

*For the following four questions, imagine you are designing a study to investigate whether deep breathing causes students to feel less stressed. Because you are investigating a causal question, you will need to employ experimental research. For each step in the design process, indicate the most scientifically sound decision.*

2. Which hypothesis is stronger? Why?
  - a. Stress levels will differ between students who engage in deep breathing and those who do not.
  - b. Students who engage in deep breathing will report less stress than those who do not engage in deep breathing.
3. Which sampling method is strongest? Why?
  - a. Obtain an alphabetical list of all students enrolled at the college. Invite every fifth person on the list to participate in the study.
  - b. Post a note to your Facebook page letting friends know you would like their help with the study. Ask your friends to let their friends know about the study, too.
  - c. Post fliers around local gyms and yoga studios inviting people to participate in your study.

4. Which set of conditions should be included in the study? Why?
  - a. All participants should be given written directions for a deep-breathing exercise.
  - b. Some participants should be given written directions for a deep-breathing exercise. Other participants should be given a DVD with demonstrations of deep-breathing exercises.
  - c. Some participants should be given written directions for a deep-breathing exercise. Other participants should be given no instructions regarding their breathing.
5. How should participants be chosen for each condition? Why?
  - a. Once people agree to participate in the study, flip a coin to decide if each will be in the experimental condition or the control condition.
  - b. Let participants select which condition they would like to be in.

The answer key for the Practice Tests can be found at the back of the book.

### Quiz 1. Experimental designs

Listed below are links to several short videos illustrating different topics of psychological research. Topics include effects of emotion on memory, time perception, personality of dogs, etc. Watch the videos and choose one as a topic to develop a testable hypothesis. Then design a research study that tests this hypothesis.

In designing the study, you should come up with an operational definition for the phenomenon of interest, what the population is, and how you intend to sample from that population. You will need to decide what sort of research methodology that you want to use and identify the variables of interest (independent & dependent measures). Identify at least one potential confounding variable that you should consider in designing your study.

Be sure to address the following points in your response:

- 1) The theory and your specific hypothesis
- 2) Population of interest, sample, method of sampling
- 3) Type of experimental design (quasi, true, longitudinal, cross-sectional)
- 4) Conditions of the study, method of assignment of subjects to conditions
- 5) Independent & dependent measures
- 6) Potential confound to consider in the design

Stress & Memory: <https://www.bilibili.com/video/BV13q4y1s7v9/>

Snakes on the Brain: <https://www.bilibili.com/video/BV1FK4y1g7AF/>

Attention Training: <https://www.bilibili.com/video/BV1fo4y1k72a/>

Time Perception: <https://www.bilibili.com/video/BV1wh411h7Ht/>

Humor & Gender: <https://www.bilibili.com/video/BV1CU4y1G7iw/>

Shopper's Mind: <https://www.bilibili.com/video/BV1So4y1k77M/>

Dog Personalities: <https://www.bilibili.com/video/BV1k64y197tc/>

Aspirin & Sex Drive: <https://www.bilibili.com/video/BV1xU4y1G7gT/>

Inherited Obesity: <https://www.bilibili.com/video/BV1if4y1t7u6/>

Virtual Alcohol Control: <https://www.bilibili.com/video/BV1zM4y1u76F/>

## RESEARCH ARTICLE SUMMARY

## PSYCHOLOGY

# Estimating the reproducibility of psychological science

Open Science Collaboration\*

**INTRODUCTION:** Reproducibility is a defining feature of science, but the extent to which it characterizes current research is unknown. Scientific claims should not gain credence because of the status or authority of their originator but by the replicability of their supporting evidence. Even research of exemplary quality may have irreproducible empirical findings because of random or systematic error.

**RATIONALE:** There is concern about the rate and predictors of reproducibility, but limited evidence. Potentially problematic practices include selective reporting, selective analysis, and insufficient specification of the conditions necessary or sufficient to obtain the results. Direct replication is the attempt to recreate the conditions believed sufficient for obtaining a pre-

viously observed finding and is the means of establishing reproducibility of a finding with new data. We conducted a large-scale, collaborative effort to obtain an initial estimate of the reproducibility of psychological science.

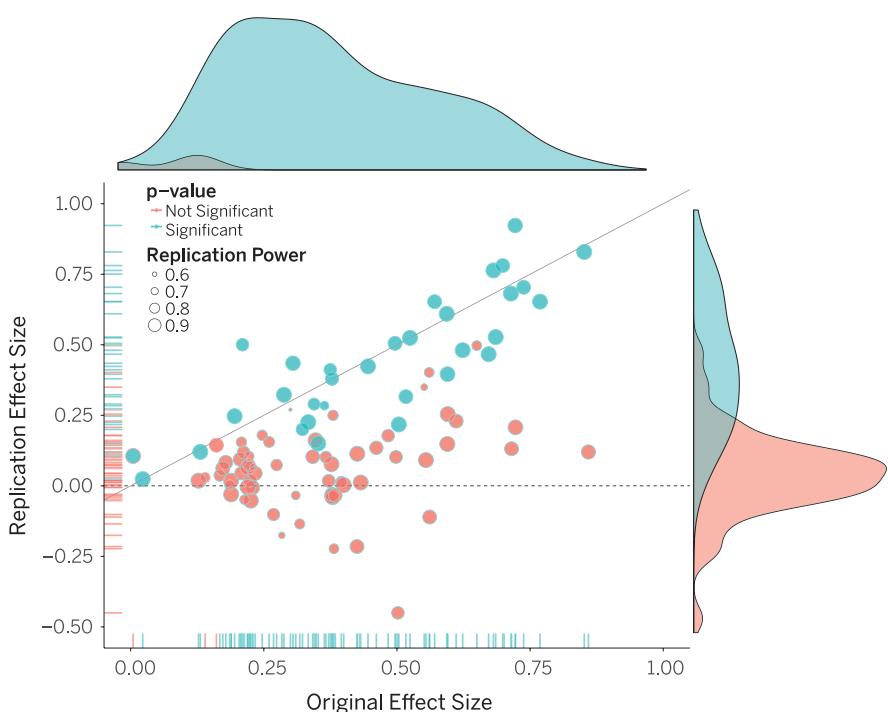
**RESULTS:** We conducted replications of 100 experimental and correlational studies published in three psychology journals using high-powered designs and original materials when available. There is no single standard for evaluating replication success. Here, we evaluated reproducibility using significance and  $P$  values, effect sizes, subjective assessments of replication teams, and meta-analysis of effect sizes. The mean effect size ( $r$ ) of the replication effects ( $M_r = 0.197$ ,  $SD = 0.257$ ) was half the magnitude of the mean effect size of the original effects ( $M_r = 0.403$ ,  $SD = 0.188$ ), representing a

substantial decline. Ninety-seven percent of original studies had significant results ( $P < .05$ ). Thirty-six percent of replications had significant results; 47% of original effect sizes were in the 95% confidence interval of the replication effect size; 39% of effects were subjectively rated to have replicated the original re-

sult; and if no bias in original results is assumed, combining original and replication results left 68% with statistically significant effects. Correlational tests suggest that replication success was better predicted by the strength of original evidence than by characteristics of the original and replication teams.

**CONCLUSION:** No single indicator sufficiently describes replication success, and the five indicators examined here are not the only ways to evaluate reproducibility. Nonetheless, collectively these results offer a clear conclusion: A large portion of replications produced weaker evidence for the original findings despite using materials provided by the original authors, review in advance for methodological fidelity, and high statistical power to detect the original effect sizes. Moreover, correlational evidence is consistent with the conclusion that variation in the strength of initial evidence (such as original  $P$  value) was more predictive of replication success than variation in the characteristics of the teams conducting the research (such as experience and expertise). The latter factors certainly can influence replication success, but they did not appear to do so here.

Reproducibility is not well understood because the incentives for individual scientists prioritize novelty over replication. Innovation is the engine of discovery and is vital for a productive, effective scientific enterprise. However, innovative ideas become old news fast. Journal reviewers and editors may dismiss a new test of a published idea as unoriginal. The claim that “we already know this” belies the uncertainty of scientific evidence. Innovation points out paths that are possible; replication points out paths that are likely; progress relies on both. Replication can increase certainty when findings are reproduced and promote innovation when they are not. This project provides accumulating evidence for many findings in psychological research and suggests that there is still more work to do to verify whether we know what we think we know. ■



**Original study effect size versus replication effect size (correlation coefficients).** Diagonal line represents replication effect size equal to original effect size. Dotted line represents replication effect size of 0. Points below the dotted line were effects in the opposite direction of the original. Density plots are separated by significant (blue) and nonsignificant (red) effects.

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Cite this article as Open Science Collaboration, *Science* 349, aac4716 (2015). DOI: 10.1126/science.aac4716

## RESEARCH ARTICLE

## PSYCHOLOGY

# Estimating the reproducibility of psychological science

Open Science Collaboration\*†

Reproducibility is a defining feature of science, but the extent to which it characterizes current research is unknown. We conducted replications of 100 experimental and correlational studies published in three psychology journals using high-powered designs and original materials when available. Replication effects were half the magnitude of original effects, representing a substantial decline. Ninety-seven percent of original studies had statistically significant results. Thirty-six percent of replications had statistically significant results; 47% of original effect sizes were in the 95% confidence interval of the replication effect size; 39% of effects were subjectively rated to have replicated the original result; and if no bias in original results is assumed, combining original and replication results left 68% with statistically significant effects. Correlational tests suggest that replication success was better predicted by the strength of original evidence than by characteristics of the original and replication teams.

**R**eproducibility is a core principle of scientific progress (1–6). Scientific claims should not gain credence because of the status or authority of their originator but by the replicability of their supporting evidence. Scientists attempt to transparently describe the methodology and resulting evidence used to support their claims. Other scientists agree or disagree whether the evidence supports the claims, citing theoretical or methodological reasons or by collecting new evidence. Such debates are meaningless, however, if the evidence being debated is not reproducible.

Even research of exemplary quality may have irreproducible empirical findings because of random or systematic error. Direct replication is the attempt to recreate the conditions believed sufficient for obtaining a previously observed finding (7, 8) and is the means of establishing reproducibility of a finding with new data. A direct replication may not obtain the original result for a variety of reasons: Known or unknown differences between the replication and original study may moderate the size of an observed effect, the original result could have been a false positive, or the replication could produce a false negative. False positives and false negatives provide misleading information about effects, and failure to identify the necessary and sufficient conditions to reproduce a finding indicates an incomplete theoretical understanding. Direct replication provides the opportunity to assess and improve reproducibility.

There is plenty of concern (9–13) about the rate and predictors of reproducibility but limited evidence. In a theoretical analysis, Ioannidis estimated that publishing and analytic practices make it likely that more than half of research

results are false and therefore irreproducible (9). Some empirical evidence supports this analysis. In cell biology, two industrial laboratories reported success replicating the original results of landmark findings in only 11 and 25% of the attempted cases, respectively (10, 11). These numbers are stunning but also difficult to interpret because no details are available about the studies, methodology, or results. With no transparency, the reasons for low reproducibility cannot be evaluated.

Other investigations point to practices and incentives that may inflate the likelihood of obtaining false-positive results in particular or irreproducible results more generally. Potentially problematic practices include selective reporting, selective analysis, and insufficient specification of the conditions necessary or sufficient to obtain the results (12–23). We were inspired to address the gap in direct empirical evidence about reproducibility. In this Research Article, we report a large-scale, collaborative effort to obtain an initial estimate of the reproducibility of psychological science.

## Method

Starting in November 2011, we constructed a protocol for selecting and conducting high-quality replications (24). Collaborators joined the project, selected a study for replication from the available studies in the sampling frame, and were guided through the replication protocol. The replication protocol articulated the process of selecting the study and key effect from the available articles, contacting the original authors for study materials, preparing a study protocol and analysis plan, obtaining review of the protocol by the original authors and other members within the present project, registering the protocol publicly, conducting the replication, writing the final report, and auditing the process and analysis for quality control. Project coordinators

facilitated each step of the process and maintained the protocol and project resources. Replication materials and data were required to be archived publicly in order to maximize transparency, accountability, and reproducibility of the project (<https://osf.io/ezcuj>).

In total, 100 replications were completed by 270 contributing authors. There were many different research designs and analysis strategies in the original research. Through consultation with original authors, obtaining original materials, and internal review, replications maintained high fidelity to the original designs. Analyses converted results to a common effect size metric [correlation coefficient ( $r$ )] with confidence intervals (CIs). The units of analysis for inferences about reproducibility were the original and replication study effect sizes. The resulting open data set provides an initial estimate of the reproducibility of psychology and correlational data to support development of hypotheses about the causes of reproducibility.

## Sampling frame and study selection

We constructed a sampling frame and selection process to minimize selection biases and maximize generalizability of the accumulated evidence. Simultaneously, to maintain high quality, within this sampling frame we matched individual replication projects with teams that had relevant interests and expertise. We pursued a quasi-random sample by defining the sampling frame as 2008 articles of three important psychology journals: *Psychological Science* (PSCI), *Journal of Personality and Social Psychology* (JPSP), and *Journal of Experimental Psychology: Learning, Memory, and Cognition* (JEP: LMC). The first is a premier outlet for all psychological research; the second and third are leading disciplinary-specific journals for social psychology and cognitive psychology, respectively [more information is available in (24)]. These were selected a priori in order to (i) provide a tractable sampling frame that would not plausibly bias reproducibility estimates, (ii) enable comparisons across journal types and subdisciplines, (iii) fit with the range of expertise available in the initial collaborative team, (iv) be recent enough to obtain original materials, (v) be old enough to obtain meaningful indicators of citation impact, and (vi) represent psychology subdisciplines that have a high frequency of studies that are feasible to conduct at relatively low cost.

The first replication teams could select from a pool of the first 20 articles from each journal, starting with the first article published in the first 2008 issue. Project coordinators facilitated matching articles with replication teams by interests and expertise until the remaining articles were difficult to match. If there were still interested teams, then another 10 articles from one or more of the three journals were made available from the sampling frame. Further, project coordinators actively recruited teams from the community with relevant experience for particular articles. This approach balanced competing goals: minimizing selection bias by

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having only a small set of articles available at a time and matching studies with replication teams' interests, resources, and expertise.

By default, the last experiment reported in each article was the subject of replication. This decision established an objective standard for study selection within an article and was based on the intuition that the first study in a multiple-study article (the obvious alternative selection strategy) was more frequently a preliminary demonstration. Deviations from selecting the last experiment were made occasionally on the basis of feasibility or recommendations of the original authors. Justifications for deviations were reported in the replication reports, which were made available on the Open Science Framework (OSF) (<http://osf.io/ezcuj>). In total, 84 of the 100 completed replications (84%) were of the last reported study in the article. On average, the to-be-replicated articles contained 2.99 studies ( $SD = 1.78$ ) with the following distribution: 24 single study, 24 two studies, 18 three studies, 13 four studies, 12 five studies, 9 six or more studies. All following summary statistics refer to the 100 completed replications.

For the purposes of aggregating results across studies to estimate reproducibility, a key result from the selected experiment was identified as the focus of replication. The key result had to be represented as a single statistical inference test or an effect size. In most cases, that test was a *t* test, *F* test, or correlation coefficient. This effect was identified before data collection or analysis and was presented to the original authors as part of the design protocol for critique. Original authors occasionally suggested that a different effect be used, and by default, replication teams deferred to original authors' judgments. Nonetheless, because the single effect came from a single study, it is not necessarily the case that the identified effect was central to the overall aims of the article. In the individual replication reports and subjective assessments of replication outcomes, more than a single result could be examined, but only the result of the single effect was considered in the aggregate analyses [additional details of the general protocol and individual study methods are provided in the supplementary materials and (25)].

In total, there were 488 articles in the 2008 issues of the three journals. One hundred fifty-eight of these (32%) became eligible for selection for replication during the project period, between November 2011 and December 2014. From those, 111 articles (70%) were selected by a replication team, producing 113 replications. Two articles had two replications each (supplementary materials). And 100 of those (88%) replications were completed by the project deadline for inclusion in this aggregate report. After being claimed, some studies were not completed because the replication teams ran out of time or could not devote sufficient resources to completing the study. By journal, replications were completed for 39 of 64 (61%) articles from *PSCI*, 31 of 55 (56%) articles from *JPSP*, and 28 of 39 (72%) articles from *JEP:LMC*.

The most common reasons for failure to match an article with a team were feasibility constraints for conducting the research. Of the 47 articles from the eligible pool that were not claimed, six (13%) had been deemed infeasible to replicate because of time, resources, instrumentation, dependence on historical events, or hard-to-access samples. The remaining 41 (87%) were eligible but not claimed. These often required specialized samples (such as macaques or people with autism), resources (such as eye tracking machines or functional magnetic resonance imaging), or knowledge making them difficult to match with teams.

### Aggregate data preparation

Each replication team conducted the study, analyzed their data, wrote their summary report, and completed a checklist of requirements for sharing the materials and data. Then, independent reviewers and analysts conducted a project-wide audit of all individual projects, materials, data, and reports. A description of this review is available on the OSF (<https://osf.io/xtine>). Moreover, to maximize reproducibility and accuracy, the analyses for every replication study were reproduced by another analyst independent of the replication team using the R statistical programming language and a standardized analytic format. A controller R script was created to regenerate the entire analysis of every study and recreate the master data file. This R script, available at <https://osf.io/fkmwg>, can be executed to reproduce the results of the individual studies. A comprehensive description of this reanalysis process is available publicly (<https://osf.io/a2eyg>).

### Measures and moderators

We assessed features of the original study and replication as possible correlates of reproducibility and conducted exploratory analyses to inspire further investigation. These included characteristics of the original study such as the publishing journal; original effect size, *P* value, and sample size; experience and expertise of the original research team; importance of the effect, with indicators such as the citation impact of the article; and rated surprisingness of the effect. We also assessed characteristics of the replication such as statistical power and sample size, experience and expertise of the replication team, independently assessed challenge of conducting an effective replication, and self-assessed quality of the replication effort. Variables such as the *P* value indicate the statistical strength of evidence given the null hypothesis, and variables such as "effect surprisingness" and "expertise of the team" indicate qualities of the topic of study and the teams studying it, respectively. The master data file, containing these and other variables, is available for exploratory analysis (<https://osf.io/5wup8>).

It is possible to derive a variety of hypotheses about predictors of reproducibility. To reduce the likelihood of false positives due to many tests, we aggregated some variables into summary indicators: experience and expertise of original team, experience and expertise of replication team, challenge of replication, self-assessed quality of repli-

cation, and importance of the effect. We had no a priori justification to give some indicators stronger weighting over others, so aggregates were created by standardizing [ $\text{mean } (M) = 0, \text{ SD} = 1$ ] the individual variables and then averaging to create a single index. In addition to the publishing journal and subdiscipline, potential moderators included six characteristics of the original study and five characteristics of the replication (supplementary materials).

### Publishing journal and subdiscipline

Journals' different publishing practices may result in a selection bias that covaries with reproducibility. Articles from three journals were made available for selection: *JPSP* ( $n = 59$  articles), *JEP:LMC* ( $n = 40$  articles), and *PSCI* ( $n = 68$  articles). From this pool of available studies, replications were selected and completed from *JPSP* ( $n = 32$  studies), *JEP:LMC* ( $n = 28$  studies), and *PSCI* ( $n = 40$  studies) and were coded as representing cognitive ( $n = 43$  studies) or social-personality ( $n = 57$  studies) subdisciplines. Four studies that would ordinarily be understood as "developmental psychology" because of studying children or infants were coded as having a cognitive or social emphasis. Reproducibility may vary by subdiscipline in psychology because of differing practices. For example, within-subjects designs are more common in cognitive than social psychology, and these designs often have greater power to detect effects with the same number of participants.

### Statistical analyses

There is no single standard for evaluating replication success (25). We evaluated reproducibility using significance and *P* values, effect sizes, subjective assessments of replication teams, and meta-analyses of effect sizes. All five of these indicators contribute information about the relations between the replication and original finding and the cumulative evidence about the effect and were positively correlated with one another ( $r$  ranged from 0.22 to 0.96, median  $r = 0.57$ ). Results are summarized in Table 1, and full details of analyses are in the supplementary materials.

### Significance and *P* values

Assuming a two-tailed test and significance or  $\alpha$  level of 0.05, all test results of original and replication studies were classified as statistically significant ( $P \leq 0.05$ ) and nonsignificant ( $P > 0.05$ ). However, original studies that interpreted nonsignificant *P* values as significant were coded as significant (four cases, all with *P* values  $< 0.06$ ). Using only the nonsignificant *P* values of the replication studies and applying Fisher's method (26), we tested the hypothesis that these studies had "no evidential value" (the null hypothesis of zero-effect holds for all these studies). We tested the hypothesis that the proportions of statistically significant results in the original and replication studies are equal using the McNemar test for paired nominal data and calculated a CI of the reproducibility parameter. Second, we compared the central tendency of the distribution of *P* values of original and

**Table 1. Summary of reproducibility rates and effect sizes for original and replication studies overall and by journal/discipline.** df/N refers to the information on which the test of the effect was based (for example, df of t test, denominator df of F test, sample size –3 of correlation, and sample size for z and  $\chi^2$ ). Four original results had P values slightly higher than 0.05 but were considered positive results in the original article and are treated that way here. Exclusions (explanation provided in supplementary materials, A3) are “replications P < 0.05” (3 original nulls excluded; n = 97 studies); “mean original and replication effect sizes” (3 excluded; n = 97 studies); “meta-analytic mean estimates” (27 excluded; n = 73 studies); “percent meta-analytic (P < 0.05)” (25 excluded; n = 75 studies); and, “percent original effect size within replication 95% CI” (5 excluded, n = 95 studies).

	Effect size comparison						Original and replication combined				
	Replications P < 0.05 in original direction	Percent	Mean (SD)	Median original df/N	Mean (SD) replication effect size	Median replication df/N	Average replication power	Meta- analytic mean (SD) estimate	Percent meta- analytic (P < 0.05)	Percent original effect size within replication 95% CI	Percent subjective “yes” to “Did it replicate?”
Overall	35/97	36	0.403 (0.188)	54	0.197 (0.257)	68	0.92	0.309 (0.223)	68	47	39
JPSP, social	7/31	23	0.29 (0.10)	73	0.07 (0.11)	120	0.91	0.138 (0.087)	43	34	25
JEP-LMC, cognitive	13/27	48	0.47 (0.18)	36.5	0.27 (0.24)	43	0.93	0.393 (0.209)	86	62	54
PSCI, social	7/24	29	0.39 (0.20)	76	0.21 (0.30)	122	0.92	0.286 (0.228)	58	40	32
PSCI, cognitive	8/15	53	0.53 (0.2)	23	0.29 (0.35)	21	0.94	0.464 (0.221)	92	60	53

**Table 2. Spearman’s rank-order correlations of reproducibility indicators with summary original and replication study characteristics.** Effect size difference computed after converting  $r$  to Fisher’s z. df/N refers to the information on which the test of the effect was based (for example, df of t test, denominator df of F test, sample size –3 of correlation, and sample size for z and  $\chi^2$ ). Four original results had P values slightly higher than 0.05 but were considered positive results in the original article and are treated that way here. Exclusions (explanation provided in supplementary materials, A3) are “replications P < .05” (3 original nulls excluded; n = 97 studies), “effect size difference” (3 excluded; n = 97 studies); “meta-analytic mean estimates” (27 excluded; n = 73 studies); and, “percent original effect size within replication 95% CI” (5 excluded, n = 95 studies).

	Replications P < 0.05 in original direction	Effect size difference	Meta-analytic estimate	Original effect size within replication 95% CI	Subjective “yes” to “Did it replicate?”
<b>Original study characteristics</b>					
Original P value	–0.327	–0.057	–0.468	0.032	–0.260
Original effect size	0.304	0.279	0.793	0.121	0.277
Original df/N	–0.150	–0.194	–0.502	–0.221	–0.185
Importance of original result	–0.105	0.038	–0.205	–0.133	–0.074
Surprising original result	–0.244	0.102	–0.181	–0.113	–0.241
Experience and expertise of original team	–0.072	–0.033	–0.059	–0.103	–0.044
<b>Replication characteristics</b>					
Replication P value	–0.828	0.621	–0.614	–0.562	–0.738
Replication effect size	0.731	–0.586	0.850	0.611	0.710
Replication power	0.368	–0.053	0.142	–0.056	0.285
Replication df/N	–0.085	–0.224	–0.692	–0.257	–0.164
Challenge of conducting replication	–0.219	0.085	–0.301	–0.109	–0.151
Experience and expertise of replication team	–0.096	0.133	0.017	–0.053	–0.068
Self-assessed quality of replication	–0.069	0.017	0.054	–0.088	–0.055

replication studies using the Wilcoxon signed-rank test and the  $t$  test for dependent samples. For both tests, we only used study-pairs for which both P values were available.

#### Effect sizes

We transformed effect sizes into correlation coefficients whenever possible. Correlation coefficients have several advantages over other effect size measures, such as Cohen’s  $d$ . Correlation coefficients are bounded, well known, and therefore more readily interpretable. Most important for our purposes, analysis of correlation coefficients is straightforward because, after ap-

plying the Fisher transformation, their standard error is only a function of sample size. Formulas and code for converting test statistics  $z$ ,  $F$ ,  $t$ , and  $\chi^2$  into correlation coefficients are provided in the appendices at <http://osf.io/ezum7>. To be able to compare and analyze correlations across study-pairs, the original study’s effect size was coded as positive; the replication study’s effect size was coded as negative if the replication study’s effect was opposite to that of the original study.

We compared effect sizes using four tests. We compared the central tendency of the effect size distributions of original and replication studies using both a paired two-sample  $t$  test and the

Wilcoxon signed-rank test. Third, we computed the proportion of study-pairs in which the effect of the original study was stronger than in the replication study and tested the hypothesis that this proportion is 0.5. For this test, we included findings for which effect size measures were available but no correlation coefficient could be computed (for example, if a regression coefficient was reported but not its test statistic). Fourth, we calculated “coverage,” or the proportion of study-pairs in which the effect of the original study was in the CI of the effect of the replication study, and compared this with the expected proportion using a goodness-of-fit  $\chi^2$  test. We carried

out this test on the subset of study pairs in which both the correlation coefficient and its standard error could be computed [we refer to this data set as the meta-analytic (MA) subset]. Standard errors could only be computed if test statistics were  $r$ ,  $t$ , or  $F(d_1, d_2)$ . The expected proportion is the sum over expected probabilities across study-pairs. The test assumes the same population effect size for original and replication study in the same study-pair. For those studies that tested the effect with  $F(d_1 > 1, d_2)$  or  $\chi^2$ , we verified coverage using other statistical procedures (computational details are provided in the supplementary materials).

#### Meta-analysis combining original and replication effects

We conducted fixed-effect meta-analyses using the R package metafor (27) on Fisher-transformed correlations for all study-pairs in subset MA and on study-pairs with the odds ratio as the dependent variable. The number of times the CI of all these meta-analyses contained 0 was calculated. For studies in the MA subset, estimated effect sizes were averaged and analyzed by discipline.

#### Subjective assessment of "Did it replicate?"

In addition to the quantitative assessments of replication and effect estimation, we collected subjective assessments of whether the replication provided evidence of replicating the original result. In some cases, the quantitative data anticipate a straightforward subjective assessment of replication. For more complex designs, such as multivariate interaction effects, the quantitative analysis may not provide a simple interpretation. For subjective assessment, replication teams answered "yes" or "no" to the question, "Did your results replicate the original effect?" Additional subjective variables are available for analysis in the full data set.

#### Analysis of moderators

We correlated the five indicators evaluating reproducibility with six indicators of the origi-

nal study (original  $P$  value, original effect size, original sample size, importance of the effect, surprising effect, and experience and expertise of original team) and seven indicators of the replication study (replication  $P$  value, replication effect size, replication power based on original effect size, replication sample size, challenge of conducting replication, experience and expertise of replication team, and self-assessed quality of replication) (Table 2). As follow-up, we did the same with the individual indicators comprising the moderator variables (tables S3 and S4).

## Results

#### Evaluating replication effect against null hypothesis of no effect

A straightforward method for evaluating replication is to test whether the replication shows a statistically significant effect ( $P < 0.05$ ) with the same direction as the original study. This dichotomous vote-counting method is intuitively appealing and consistent with common heuristics used to decide whether original studies "worked." Ninety-seven of 100 (97%) effects from original studies were positive results (four had  $P$  values falling a bit short of the 0.05 criterion— $P = 0.0508, 0.0514, 0.0516$ , and 0.0567—but all of these were interpreted as positive effects). On the basis of only the average replication power of the 97 original, significant effects [ $M = 0.92$ , median ( $Mdn$ ) = 0.95], we would expect approximately 89 positive results in the replications if all original effects were true and accurately estimated; however, there were just 35 [36.1%; 95% CI = (26.6%, 46.2%)], a significant reduction [McNemar test,  $\chi^2(1) = 59.1$ ,  $P < 0.001$ ].

A key weakness of this method is that it treats the 0.05 threshold as a bright-line criterion between replication success and failure (28). It could be that many of the replications fell just short of the 0.05 criterion. The density plots of  $P$  values for original studies (mean  $P$  value = 0.028) and replications (mean  $P$  value = 0.302) are shown in Fig. 1, left. The 64 nonsignificant

$P$  values for replications were distributed widely. When there is no effect to detect, the null distribution of  $P$  values is uniform. This distribution deviated slightly from uniform with positive skew, however, suggesting that at least one replication could be a false negative,  $\chi^2(128) = 155.83$ ,  $P = 0.048$ . Nonetheless, the wide distribution of  $P$  values suggests against insufficient power as the only explanation for failures to replicate. A scatterplot of original compared with replication study  $P$  values is shown in Fig. 2.

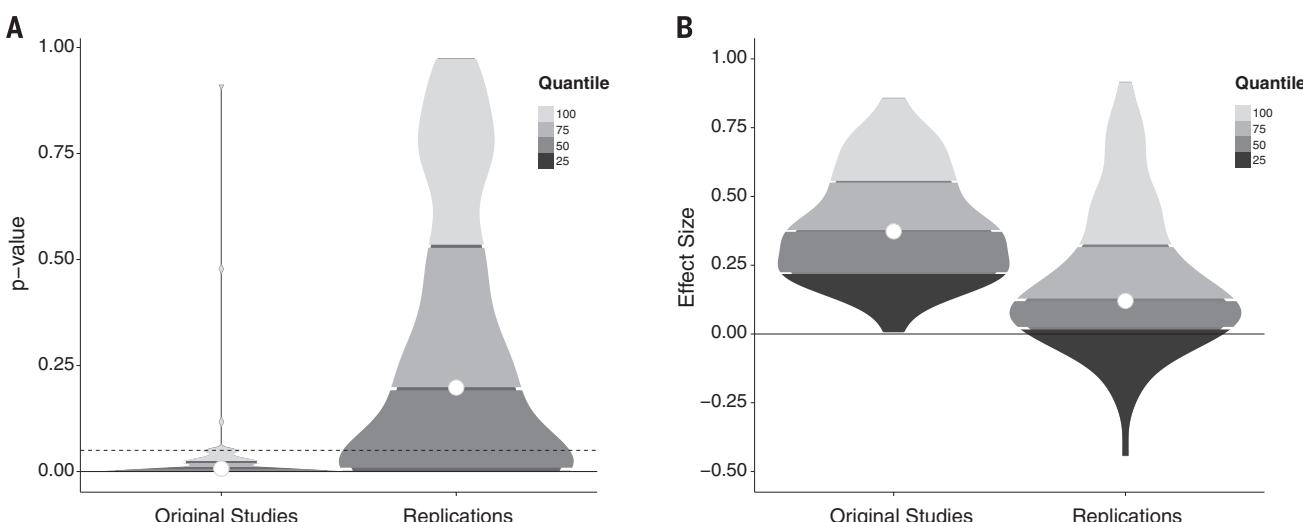
#### Evaluating replication effect against original effect size

A complementary method for evaluating replication is to test whether the original effect size is within the 95% CI of the effect size estimate from the replication. For the subset of 73 studies in which the standard error of the correlation could be computed, 30 (41.1%) of the replication CIs contained the original effect size (significantly lower than the expected value of 78.5%,  $P < 0.001$ ) (supplementary materials). For 22 studies using other test statistics [ $F(d_1 > 1, d_2)$  and  $\chi^2$ ], 68.2% of CIs contained the effect size of the original study. Overall, this analysis suggests a 47.4% replication success rate.

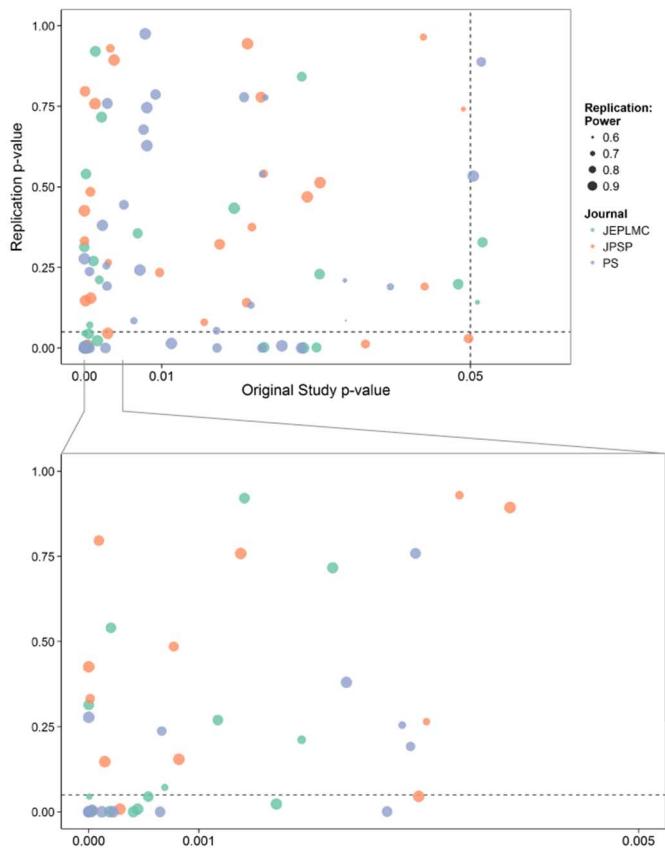
This method addresses the weakness of the first test that a replication in the same direction and a  $P$  value of 0.06 may not be significantly different from the original result. However, the method will also indicate that a replication "fails" when the direction of the effect is the same but the replication effect size is significantly smaller than the original effect size (29). Also, the replication "succeeds" when the result is near zero but not estimated with sufficiently high precision to be distinguished from the original effect size.

#### Comparing original and replication effect sizes

Comparing the magnitude of the original and replication effect sizes avoids special emphasis on  $P$  values. Overall, original study effect sizes



**Fig. 1. Density plots of original and replication  $P$  values and effect sizes.** (A)  $P$  values. (B) Effect sizes (correlation coefficients). Lowest quantiles for  $P$  values are not visible because they are clustered near zero.



**Fig. 2. Scatterplots of original study and replication  $P$  values for three psychology journals.** Data points scaled by power of the replication based on original study effect size. Dotted red lines indicate  $P = 0.05$  criterion. Subplot below shows  $P$  values from the range between the gray lines ( $P = 0$  to 0.005) in the main plot above.

( $M = 0.403$ ,  $SD = 0.188$ ) were reliably larger than replication effect sizes ( $M = 0.197$ ,  $SD = 0.257$ ), Wilcoxon's  $W = 7137$ ,  $P < 0.001$ . Of the 99 studies for which an effect size in both the original and replication study could be calculated (30), 82 showed a stronger effect size in the original study (82.8%;  $P < 0.001$ , binomial test) (Fig. 1, right). Original and replication effect sizes were positively correlated (Spearman's  $r = 0.51$ ,  $P < 0.001$ ). A scatterplot of the original and replication effect sizes is presented in Fig. 3.

#### Combining original and replication effect sizes for cumulative evidence

The disadvantage of the descriptive comparison of effect sizes is that it does not provide information about the precision of either estimate or resolution of the cumulative evidence for the effect. This is often addressed by computing a meta-analytic estimate of the effect sizes by combining the original and replication studies (28). This approach weights each study by the inverse of its variance and uses these weighted estimates of effect size to estimate cumulative evidence and precision of the effect. Using a fixed-effect model, 51 of the 75 (68%) effects for which a meta-analytic estimate could be computed had 95% CIs that did not include 0.

One qualification about this result is the possibility that the original studies have inflated effect sizes due to publication, selection, reporting, or other biases (9, 12–23). In a discipline with low-powered research designs and an emphasis on positive results for publication, effect sizes will be systematically overestimated in the published literature. There is no publication bias in the replication studies because all results are reported. Also, there are no selection or reporting biases because all were confirmatory tests based on pre-analysis plans. This maximizes the interpretability of the replication  $P$  values and effect estimates. If publication, selection, and reporting biases completely explain the effect differences, then the replication estimates would be a better estimate of the effect size than would the meta-analytic and original results. However, to the extent that there are other influences, such as moderation by sample, setting, or quality of replication, the relative bias influencing original and replication effect size estimation is unknown.

#### Subjective assessment of “Did it replicate?”

In addition to the quantitative assessments of replication and effect estimation, replication teams

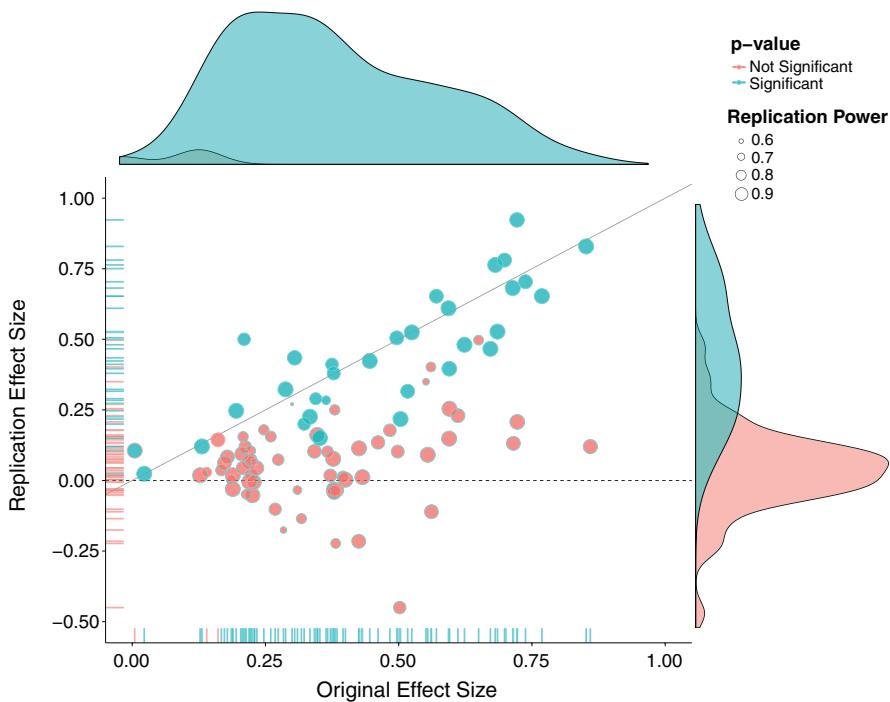
provided a subjective assessment of replication success of the study they conducted. Subjective assessments of replication success were very similar to significance testing results (39 of 100 successful replications), including evaluating “success” for two null replications when the original study reported a null result and “failure” for a  $P < 0.05$  replication when the original result was a null.

#### Correlates of reproducibility

The overall replication evidence is summarized in Table 1 across the criteria described above and then separately by journal/discipline. Considering significance testing, reproducibility was stronger in studies and journals representing cognitive psychology than social psychology topics. For example, combining across journals, 14 of 55 (25%) of social psychology effects replicated by the  $P < 0.05$  criterion, whereas 21 of 42 (50%) of cognitive psychology effects did so. Simultaneously, all journals and disciplines showed substantial and similar [ $\chi^2(3) = 2.45$ ,  $P = 0.48$ ] declines in effect size in the replications compared with the original studies. The difference in significance testing results between fields appears to be partly a function of weaker original effects in social psychology studies, particularly in *JPS*, and perhaps of the greater frequency of high-powered within-subjects manipulations and repeated measurement designs in cognitive psychology as suggested by high power despite relatively small participant samples. Further, the type of test was associated with replication success. Among original, significant effects, 23 of the 49 (47%) that tested main or simple effects replicated at  $P < 0.05$ , but just 8 of the 37 (22%) that tested interaction effects did.

Correlations between reproducibility indicators and characteristics of replication and original studies are provided in Table 2. A negative correlation of replication success with the original study  $P$  value indicates that the initial strength of evidence is predictive of reproducibility. For example, 26 of 63 (41%) original studies with  $P < 0.02$  achieved  $P < 0.05$  in the replication, whereas 6 of 23 (26%) that had a  $P$  value between  $0.02 < P < 0.04$  and 2 of 11 (18%) that had a  $P$  value  $> 0.04$  did so (Fig. 2). Almost two thirds (20 of 32, 63%) of original studies with  $P < 0.001$  had a significant  $P$  value in the replication.

Larger original effect sizes were associated with greater likelihood of achieving  $P < 0.05$  ( $r = 0.304$ ) and a greater effect size difference between original and replication ( $r = 0.279$ ). Moreover, replication power was related to replication success via significance testing ( $r = 0.368$ ) but not with the effect size difference between original and replication ( $r = -0.053$ ). Comparing effect sizes across indicators, surprisingness of the original effect, and the challenge of conducting the replication were related to replication success for some indicators. Surprising effects were less reproducible, as were effects for which it was more challenging to conduct the replication. Last, there was little evidence that perceived importance of the effect, expertise of the original or replication teams, or self-assessed quality of the replication accounted for meaningful variation



**Fig. 3. Original study effect size versus replication effect size (correlation coefficients).**

Diagonal line represents replication effect size equal to original effect size. Dotted line represents replication effect size of 0. Points below the dotted line were effects in the opposite direction of the original. Density plots are separated by significant (blue) and nonsignificant (red) effects.

in reproducibility across indicators. Replication success was more consistently related to the original strength of evidence (such as original  $P$  value, effect size, and effect tested) than to characteristics of the teams and implementation of the replication (such as expertise, quality, or challenge of conducting study) (tables S3 and S4).

## Discussion

No single indicator sufficiently describes replication success, and the five indicators examined here are not the only ways to evaluate reproducibility. Nonetheless, collectively, these results offer a clear conclusion: A large portion of replications produced weaker evidence for the original findings (31) despite using materials provided by the original authors, review in advance for methodological fidelity, and high statistical power to detect the original effect sizes. Moreover, correlational evidence is consistent with the conclusion that variation in the strength of initial evidence (such as original  $P$  value) was more predictive of replication success than was variation in the characteristics of the teams conducting the research (such as experience and expertise). The latter factors certainly can influence replication success, but the evidence is that they did not systematically do so here. Other investigators may develop alternative indicators to explore further the role of expertise and quality in reproducibility on this open data set.

## Insights on reproducibility

It is too easy to conclude that successful replication means that the theoretical understanding of

the original finding is correct. Direct replication mainly provides evidence for the reliability of a result. If there are alternative explanations for the original finding, those alternatives could likewise account for the replication. Understanding is achieved through multiple, diverse investigations that provide converging support for a theoretical interpretation and rule out alternative explanations.

It is also too easy to conclude that a failure to replicate a result means that the original evidence was a false positive. Replications can fail if the replication methodology differs from the original in ways that interfere with observing the effect. We conducted replications designed to minimize *a priori* reasons to expect a different result by using original materials, engaging original authors for review of the designs, and conducting internal reviews. Nonetheless, unanticipated factors in the sample, setting, or procedure could still have altered the observed effect magnitudes (32).

More generally, there are indications of cultural practices in scientific communication that may be responsible for the observed results. Low-power research designs combined with publication bias favoring positive results together produce a literature with upwardly biased effect sizes (14, 16, 33, 34). This anticipates that replication effect sizes would be smaller than original studies on a routine basis—not because of differences in implementation but because the original study effect sizes are affected by publication and reporting bias, and the replications are not. Consistent with this expectation, most replication effects were

smaller than original results, and reproducibility success was correlated with indicators of the strength of initial evidence, such as lower original  $P$  values and larger effect sizes. This suggests publication, selection, and reporting biases as plausible explanations for the difference between original and replication effects. The replication studies significantly reduced these biases because replication preregistration and pre-analysis plans ensured confirmatory tests and reporting of all results.

The observed variation in replication and original results may reduce certainty about the statistical inferences from the original studies but also provides an opportunity for theoretical innovation to explain differing outcomes, and then new research to test those hypothesized explanations. The correlational evidence, for example, suggests that procedures that are more challenging to execute may result in less reproducible results, and that more surprising original effects may be less reproducible than less surprising original effects. Further, systematic, repeated replication efforts that fail to identify conditions under which the original finding can be observed reliably may reduce confidence in the original finding.

## Implications and limitations

The present study provides the first open, systematic evidence of reproducibility from a sample of studies in psychology. We sought to maximize generalizability of the results with a structured process for selecting studies for replication. However, it is unknown the extent to which these findings extend to the rest of psychology or other disciplines. In the sampling frame itself, not all articles were replicated; in each article, only one study was replicated; and in each study, only one statistical result was subject to replication. More resource-intensive studies were less likely to be included than were less resource-intensive studies. Although study selection bias was reduced by the sampling frame and selection strategy, the impact of selection bias is unknown.

We investigated the reproducibility rate of psychology not because there is something special about psychology, but because it is our discipline. Concerns about reproducibility are widespread across disciplines (9–21). Reproducibility is not well understood because the incentives for individual scientists prioritize novelty over replication (20). If nothing else, this project demonstrates that it is possible to conduct a large-scale examination of reproducibility despite the incentive barriers. Here, we conducted single-replication attempts of many effects obtaining broad-and-shallow evidence. These data provide information about reproducibility in general but little precision about individual effects in particular. A complementary narrow-and-deep approach is characterized by the Many Labs replication projects (32). In those, many replications of single effects allow precise estimates of effect size but result in generalizability that is circumscribed to those individual effects. Pursuing both strategies across disciplines, such as the ongoing effort in cancer biology (35), would yield insight about common and distinct

challenges and may cross-fertilize strategies so as to improve reproducibility.

Because reproducibility is a hallmark of credible scientific evidence, it is tempting to think that maximum reproducibility of original results is important from the onset of a line of inquiry through its maturation. This is a mistake. If initial ideas were always correct, then there would hardly be a reason to conduct research in the first place. A healthy discipline will have many false starts as it confronts the limits of present understanding.

Innovation is the engine of discovery and is vital for a productive, effective scientific enterprise. However, innovative ideas become old news fast. Journal reviewers and editors may dismiss a new test of a published idea as unoriginal. The claim that “we already know this” belies the uncertainty of scientific evidence. Deciding the ideal balance of resourcing innovation versus verification is a question of research efficiency. How can we maximize the rate of research progress? Innovation points out paths that are possible; replication points out paths that are likely; progress relies on both. The ideal balance is a topic for investigation itself. Scientific incentives—funding, publication, or awards—can be tuned to encourage an optimal balance in the collective effort of discovery (36, 37).

Progress occurs when existing expectations are violated and a surprising result spurs a new investigation. Replication can increase certainty when findings are reproduced and promote innovation when they are not. This project provides accumulating evidence for many findings in psychological research and suggests that there is still more work to do to verify whether we know what we think we know.

## Conclusion

After this intensive effort to reproduce a sample of published psychological findings, how many of the effects have we established are true? Zero. And how many of the effects have we established are false? Zero. Is this a limitation of the project design? No. It is the reality of doing science, even if it is not appreciated in daily practice. Humans desire certainty, and science infrequently provides it. As much as we might wish it to be otherwise, a single study almost never provides definitive resolution for or against an effect and its explanation. The original studies examined here offered tentative evidence; the replications we conducted offered additional, confirmatory evidence. In some cases, the replications increase confidence in the reliability of the original results; in other cases, the replications suggest that more investigation is needed to establish the validity of the original findings. Scientific progress is a cumulative process of uncertainty reduction that can only succeed if science itself remains the greatest skeptic of its explanatory claims.

The present results suggest that there is room to improve reproducibility in psychology. Any temptation to interpret these results as a defeat for psychology, or science more generally, must contend with the fact that this project demon-

strates science behaving as it should. Hypotheses abound that the present culture in science may be negatively affecting the reproducibility of findings. An ideological response would discount the arguments, discredit the sources, and proceed merrily along. The scientific process is not ideological. Science does not always provide comfort for what we wish to be; it confronts us with what is. Moreover, as illustrated by the Transparency and Openness Promotion (TOP) Guidelines (<http://cos.io/top>) (37), the research community is taking action already to improve the quality and credibility of the scientific literature.

We conducted this project because we care deeply about the health of our discipline and believe in its promise for accumulating knowledge about human behavior that can advance the quality of the human condition. Reproducibility is central to that aim. Accumulating evidence is the scientific community’s method of self-correction and is the best available option for achieving that ultimate goal: truth.

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## ACKNOWLEDGMENTS

In addition to the coauthors of this manuscript, there were many volunteers who contributed to project success. We thank D. Acup, J. Anderson, S. Anzellotti, R. Araujo, J. D. Arnal, T. Bates, R. Battleday, R. Bauchowitz, M. Bernstein, B. Blohowiak, M. Boffo, E. Bruneau, B. Chabot-Hanowell, J. Chan, P. Chu, A. Dalla Rosa, B. Deen, P. DiGiocomo, C. Dogulu, N. Dufour, C. Fitzgerald, A. Foote, A. Garcia, E. Garcia, C. Gautreau, L. Germine, T. Gill, L. Goldberg, S. D. Goldfinger, H. Gweon, D. Haile, K. Hart, F. Hjorth, J. Hoenig, Å. Innes-Ker, B. Jansen, R. Jersakova, Y. Jie, Z. Kaldy, W. K. Vong, A. Kenney, J. Kingston, J. Koster-Hale, A. Lam, R. LeDonne, D. Lumian, E. Luong, S. Man-pui, J. Martin, A. Mauk, T. McElroy, K. McMae, T. Miller, K. Moser, M. Mullarkey, A. R. Munoz, J. Ong, C. Parks, D. S. Pate, D. Patron, H. J. M. Pennings, M. Penuliar, A. Pfammatter, J. P. Shanoltz, E. Stevenson, E. Pichler, H. Raudszus, H. Richardson, N. Rothstein, T. Scherndl, S. Schrager, S. Shah, Y. S. Tai, A. Skerry, M. Steinberg, J. Stoeterau, H. Tibboel, A. Tooley, A. Tullett, C. Vaccaro, E. Vergauwe, A. Watanabe, I. Weiss, M. H. White II,

P. Whitehead, C. Widmann, D. K. Williams, K. M. Williams, and H. Yi. Also, we thank the authors of the original research that was the subject of replication in this project. These authors were generous with their time, materials, and advice for improving the quality of each replication and identifying the strengths and limits of the outcomes. The authors of this work are listed alphabetically. This project was supported by the Center for Open Science and the Laura and John Arnold Foundation. The authors declare no financial conflict of interest with the reported research.

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29 April 2015; accepted 28 July 2015

10.1126/science.aac4716

## Estimating the reproducibility of psychological science

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Science 349 (6251), aac4716.  
DOI: 10.1126/science.aac4716

### Empirically analyzing empirical evidence

One of the central goals in any scientific endeavor is to understand causality. Experiments that seek to demonstrate a cause/effect relation most often manipulate the postulated causal factor. Aarts *et al.* describe the replication of 100 experiments reported in papers published in 2008 in three high-ranking psychology journals. Assessing whether the replication and the original experiment yielded the same result according to several criteria, they find that about one-third to one-half of the original findings were also observed in the replication study.

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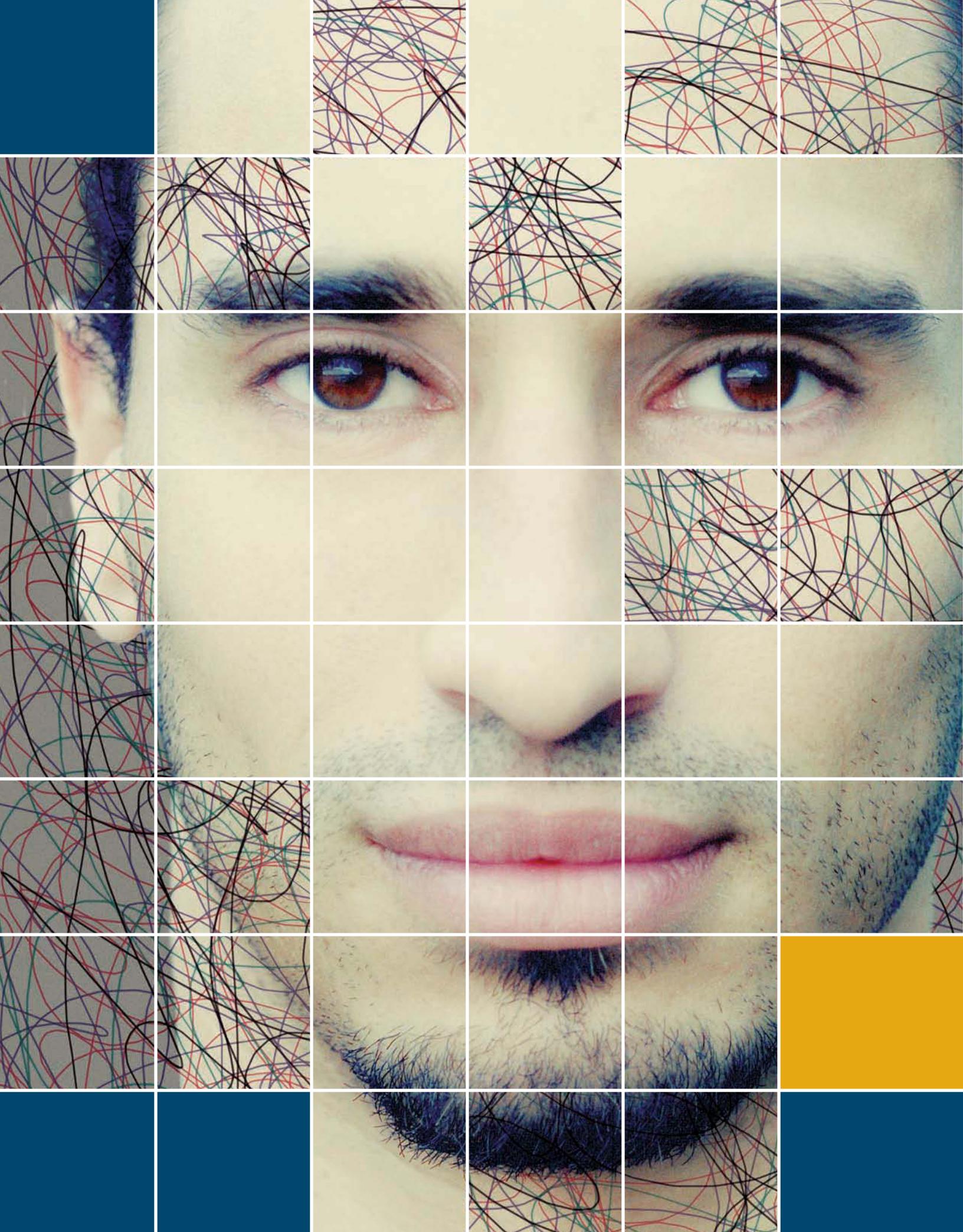
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# 14

# Psychological Disorders

## Ask & Answer

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**MOST PEOPLE EXPERIENCE UPS AND DOWNS.** For people with bipolar disorder, mood and energy swings are much more intense, quickly changing from episodes of extreme listlessness and depression to excited states of extraordinary joyfulness known as mania. This condition was previously called *manic-depressive disorder* because of the dramatic shifts between mania and depression. A number of celebrities have been diagnosed with bipolar disorder, including Britney Spears, Demi Lovato, Russell Brand, and Catherine Zeta-Jones.

One of the world's foremost authorities on bipolar disorder has unique insight among psychologists—she has suffered from the disorder since childhood. Kay Redfield Jamison acknowledged her struggles with bipolar disorder in her award-winning memoir, *An Unquiet Mind* (1995; **FIGURE 14.1**). Her work has helped shape the study of the disorder, and her 1990 textbook, coauthored with Frederick Goodwin, is considered the standard for the field (Goodwin & Jamison, 1990).

In *An Unquiet Mind*, Jamison details how as a child she was intensely emotional and occasionally obsessive. When she was 17, she had her first serious bout of what she describes as profoundly suicidal depression. Jamison experienced deepening swings from wild exuberance to paralyzing depression throughout her undergraduate years. In 1975, after obtaining her Ph.D. in clinical psychology, she joined the UCLA Department of Psychiatry, where she directed the Affective Disorders Clinic.



**FIGURE 14.1**

**Kay Redfield Jamison**

Jamison was able to overcome her crippling bipolar disorder to succeed as a teacher, researcher, and author.

## Learning Objectives

- Understand what is meant by the term *psychological disorder*.
- Explain how psychological disorders are classified.
- Identify assessment methods for psychological disorders.
- Describe the diathesis-stress model.
- Identify biological, environmental, and cognitive-behavioral causes of psychological disorders.
- Discuss sex differences and cultural differences in psychological disorders.

Within months after she began this job, her condition deteriorated dramatically. She began hallucinating and feared that she was losing her mind. This state so terrified her that she sought out a psychiatrist, who quickly diagnosed her as having manic-depressive disorder (i.e., bipolar disorder) and prescribed a drug called lithium. (You will learn more about lithium and other treatments for bipolar disorder in Chapter 15, “Treatment of Psychological Disorders.”)

People with bipolar disorder experience profoundly enjoyable highs during their manic phases. One of the unfortunate side effects of lithium is that it blunts positive feelings. Even though these patients know that lithium helps them, they often resent the drug and refuse to take it. Because people in manic episodes have impaired judgment, they often engage in dangerous behavior or make disastrous decisions. Lithium has helped Jamison, but she also credits the support of her psychiatrist, as well as of her family and friends.

Jamison has made the point that lithium can rob people of creative energy. In her book *Touched with Fire* (1993), she asks whether lithium would have dampened the genius of those major artists and writers who may have had mood disorders, such as Michelangelo, Vincent van Gogh, Georgia O’Keeffe, Emily Dickinson, and Ernest Hemingway. Jamison demonstrates the strong association between bipolar disorder and artistic genius, and she raises the disturbing question of whether eradicating the disorder would rob society of much great art. She embodies this irony: Her early career benefited from the energy and creativity of her manic phases even as her personal life was threatened by devastating depression.

Jamison provides a good example of both the ravages caused by psychological disorders and the effective methods available to help people live with them. In this chapter, you will learn about the various psychological disorders. In the next chapter, you will learn the most effective treatments for each disorder.

## 14.1 How Are Psychological Disorders Conceptualized and Classified?

Those who have psychological disorders display symptoms of **psychopathology**. This term means sickness or disorder of the mind. From the writings of Aristotle to those of Freud, accounts exist of people suffering from various forms of psychopathology. Although considerable progress has been made over the last century, we are still struggling to determine the causes of psychopathology. To understand any disorder, psychologists investigate its **etiology**: the factors that contribute to its development. For example, they investigate commonalities among people such as Kay Redfield Jamison and Demi Lovato to identify factors that might explain why they (and others) developed bipolar disorders.

The earliest views of psychopathology explained apparent “madness” as resulting from possession by demons or evil spirits (**FIGURE 14.2**). The ancient Babylonians believed a demon called Idta caused madness. Similar examples of demonology can be found among the ancient Chinese, Egyptians, and Greeks. This view of psychopathology continued into the Middle Ages. At that time, there was greater emphasis on possession as having resulted from the wrath of God for some sinful moral transgression. During any of these periods, someone like Kay Redfield Jamison might have been persecuted and subjected to an array of methods to cast out her demons. Such “treatments” included exorcism, bloodletting, and the forced ingestion of magical potions.

In the latter half of the Middle Ages and into the Renaissance, people with psychopathology were removed from society so they would not bother others. In the 1700s, Jamison likely would have been left in an understaffed, overcrowded mental institution called an asylum. Even there, the small staff would have made little attempt to understand Jamison’s disorder and even less of an attempt to treat her. Indeed, people housed in asylums were often chained up and lived in incredibly filthy conditions, treated more like nonhuman animals than humans. “Treatments” included starvation, beatings, bloodletting, and isolation.

In 1793, Philippe Pinel, a French physician who believed that medical treatments should be based on empirical observations, became the head physician at Bicêtre Hospital, in Paris. At that time, among the hospital’s 4,000 patients were about 200 with psychopathology being cared for by a former patient, Jean-Baptiste Pussin. Pussin treated his patients with kindness and care rather than violence. Impressed by the positive therapeutic results, Pinel removed patients from their chains and banished physical punishment. He instituted what came to be known as *moral treatment*, a therapy that involved close contact with and careful observation of patients. Pinel’s benevolent treatment gained a foothold in Europe, and later, through the efforts of a Massachusetts schoolteacher, Dorothea Dix, in America.

As far back as ancient Greece, some people had a sense that there was a physical basis to psychopathology. Hippocrates (c. 460–377 BCE), often credited as the founder of modern medicine, classified psychopathologies into *mania*, *melancholia*, and *phrenitis*, the latter characterized by mental confusion. Hippocrates believed that such disorders resulted from the relative amount of “humors,” or bodily fluids, a person possessed (Maher & Maher, 1994). For instance, having too much black bile led to melancholia, or extreme sadness and depression. From this term, we get the word *melancholy*, which we often use to describe people who are sad. The idea that bodily fluids cause mental illness was abandoned long ago, however. Increasingly throughout the nineteenth and twentieth centuries, psychopathology was viewed more as a medical condition than as a demonic curse caused by sin. The medical model viewed psychopathology as resulting from disease. During the last 200 years, recognition has grown that psychopathology reflects dysfunction of the body, particularly of the brain.

At various points in recent history, researchers and clinicians would have focused solely on environmental factors that contributed to Jamison’s psychological disorder. For example, was she abused as a child? Although environmental factors are important, we now understand that biology plays a critical role in many psychological disorders, especially disorders such as bipolar disorder or schizophrenia. Indeed, an important lesson in this chapter is that environment and biology interact to produce psychological disorders. As noted throughout the book, it is meaningless to state that a condition is caused by just biology or just environment. Both factors affect all psychological disorders to some extent.



**FIGURE 14.2**  
**Historical View of Psychological Disorders**

Throughout history, people believed that the gods, witches, or evil spirits caused psychological disorders.

### psychopathology

Sickness or disorder of the mind.

### etiology

Factors that contribute to the development of a disorder.

## Psychopathology Is Different from Everyday Problems

Psychological disorders are common around the globe, in all countries and all societies. These disorders account for the greatest proportion of disability in developed countries, surpassing even cancer and heart disease (Centers for Disease Control and Prevention, 2011a). Indeed, about 1 in 4 Americans over age 18 has a diagnosable psychological disorder in a given year (Kessler, Chiu, Demler, & Walters, 2005a). About 1 in 5 American adults receives treatment over any two-year period (Kessler et al., 2005b). Nearly half of Americans will have some form of psychological disorder at some point in life, most commonly a depressive disorder, an attention-deficit/hyperactivity disorder, an anxiety disorder, or a substance-related and addictive disorder (Kessler & Wang, 2008). Of course, psychological disorders range in severity. Only about 7 percent of the U.S. population is severely affected, and this group also tend to suffer from multiple disorders (Kessler et al., 2005a).

As you read this chapter, you may realize that you have experienced some of the symptoms of many psychological disorders. Even if particular symptoms seem to describe you (or anyone you know) perfectly, resist the urge to make a diagnosis. Just like medical students who worry they have every disease they learn about, you need to guard against overanalyzing yourself and others. At the same time, what you learn in this chapter and the next one may help you understand the mental health problems you or others might experience.

**PSYCHOLOGICAL DISORDERS AS MALADAPTIVE** How do you know if someone has a psychological disorder? It can be challenging to decide if a given behavior is caused by psychopathology. Keep in mind that behavior, especially unusual behavior, must always be reviewed in the context of the situation. A woman running through the streets screaming, sobbing, and grabbing and hugging people might have some form of psychological disorder—or she might be celebrating because she just won the lottery. Many behaviors considered normal in one setting may be considered deviant in other settings. Some Native American and East Asian cultures consider it a great honor to hear the voices of spirits. In urban America, hearing spirits would be seen as evidence of auditory hallucinations.

In determining whether behavior represents psychopathology, it is important to consider certain criteria: (1) Does the person act in a way that deviates from cultural norms for acceptable behavior? (2) Is the behavior maladaptive? That is, does the behavior interfere with the person's ability to respond appropriately in different situations? For example, a person who is afraid to leave the house may avoid feeling anxious by staying inside, and that behavior might prevent the person from working, having a social life, or both. (3) Is the behavior self-destructive, does it cause the individual personal distress, or does it threaten other people in the community? (4) Does the behavior cause discomfort and concern to others, thus impairing a person's social relationships?

It may be hard to draw the line between normal and abnormal. As a result, psychopathology is increasingly defined in terms of *maladaptiveness*. That is, a person with psychopathology exhibits thoughts, feelings, and behaviors that are maladaptive rather than deviant. For example, people concerned about germs may wash their hands more than average and therefore be deviant, but that behavior may be beneficial in many ways and therefore adaptive—after all, it is the best way of avoiding contagious disease. The same behavior, however, can be maladaptive when people cannot stop until they have washed their hands raw. Indeed, the diagnostic criteria for all the major disorder categories stipulate that the symptoms of the disorder must interfere

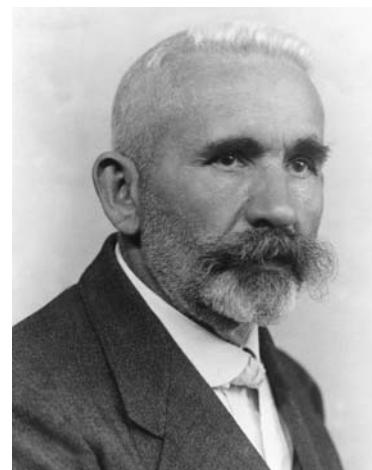
with at least one aspect of the person's life, such as work, social relations, or self-care. This component is critical in determining whether given thoughts, emotions, or behaviors represent psychopathology or are simply unusual.

## Psychological Disorders Are Classified into Categories

In the late 1800s, the psychiatrist Emil Kraepelin recognized that not all patients with psychological disorders suffer from the same disorder (**FIGURE 14.3**). Kraepelin separated disorders into categories based on what he could observe: groups of symptoms that occur together. For instance, he separated disorders of mood (emotions) from disorders of cognition. He called the latter disorder *dementia praecox*. It is now better known as *schizophrenia* and is discussed fully in this chapter and the next one.

The idea of categorizing psychological disorders systematically was not officially adopted until 1952, when the American Psychiatric Association published the first edition of the *Diagnostic and Statistical Manual of Mental Disorders (DSM)*. Since then, the *DSM* has undergone several revisions. It remains the standard in psychology and psychiatry. The guiding principle of the *DSM* is that if disorders can be grouped based on similar etiologies and symptoms, then figuring out how to treat those disorders should be easier.

In the current edition, *DSM-5* (released in 2013), disorders are described in terms of observable symptoms. A client must meet specific criteria to receive a particular diagnosis. The *DSM-5* consists of three sections: (1) an introduction with instructions for using the manual; (2) diagnostic criteria for all of the disorders, which are grouped so that similar categories of disorders are located near each other (**TABLE 14.1**); and



**FIGURE 14.3**  
**Emil Kraepelin**

Kraepelin was one of the first researchers to propose a classification system for psychological disorders.

**Table 14.1 DSM-5 Disorders**

CATEGORY	EXAMPLES
<b>Neurodevelopmental disorders</b>	Autism spectrum disorder
<b>Schizophrenia spectrum and other psychotic disorders</b>	Schizophrenia
<b>Bipolar and related disorders</b>	Bipolar I disorder
<b>Depressive disorders</b>	Major depressive disorder
<b>Anxiety disorders</b>	Panic disorder
<b>Obsessive-compulsive and related disorders</b>	Body dysmorphic disorder
<b>Trauma- and stressor-related disorders</b>	Posttraumatic stress disorder
<b>Dissociative disorders</b>	Dissociative amnesia
<b>Somatic symptom and related disorders</b>	Conversion disorder
<b>Feeding and eating disorders</b>	Anorexia nervosa
<b>Elimination disorders</b>	Enuresis (bed wetting)
<b>Sleep-wake disorders</b>	Narcolepsy
<b>Sexual dysfunctions</b>	Erectile disorder
<b>Gender dysphoria</b>	Gender dysphoria
<b>Disruptive, impulse-control, and conduct disorders</b>	Pyromania
<b>Substance-related and addictive disorders</b>	Alcohol use disorder
<b>Neurocognitive disorders</b>	Delirium
<b>Personality disorders</b>	Borderline personality disorder
<b>Paraphilic disorders</b>	Exhibitionist disorder

SOURCE: Based on American Psychiatric Association (2013).



**FIGURE 14.4**  
**Dimensional Nature of Psychopathology**

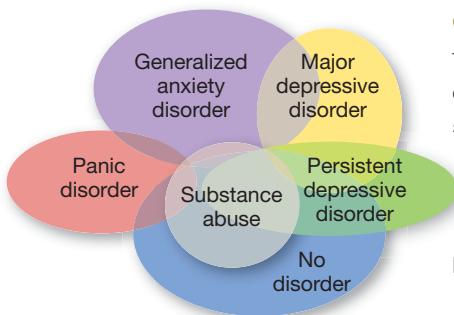
Symptoms of psychological disorders occur along continuums. They are not absolute states. A person who falls below the cut-off level may not meet the diagnostic criteria but may still experience symptoms that interfere with his or her life and will therefore benefit from treatment.

(3) a guide for future psychopathology research, which also includes conditions not yet officially recognized as disorders, such as excessive Internet gaming and misuse of caffeine.

### DIMENSIONAL NATURE OF PSYCHOPATHOLOGY

One problem with the *DSM* approach is that it implies that a person either has a psychological disorder or does not, which is known as a *categorical approach* (Bernstein, 2011). That is, the diagnosis is categorical, and a person is either in the category or not. This approach fails to capture differences in the severity of a disorder.

An alternative type of evaluation, called a *dimensional approach*, is to consider psychological disorders along a continuum in which people vary in degree rather than in kind (FIGURE 14.4). With categorization, the approach can be compared to a simple switch that turns a light on or off. By contrast, the dimensional approach is like a dimmer switch, which can provide light in varying amounts. A dimensional approach recognizes that many psychological disorders are extreme versions of normal feelings. We are all a little sad at times, and sometimes we feel more sad than usual. But no specific amount of sadness passes a threshold for depressive disorders. In the third section of *DSM-5*, researchers are encouraged to examine whether a dimensional approach might be helpful for understanding many psychological disorders, particularly personality disorders. Indeed, research indicates that personality disorders can be viewed as maladaptive extremes of the Big Five personality traits, which were described in Chapter 13.



**FIGURE 14.5**  
**Comorbidity**

Psychological disorders commonly overlap. For instance, substance abuse is common across psychological disorders, and people with depression (or a milder form known as persistent depressive disorder) often also have anxiety disorders (such as panic disorder or generalized anxiety disorder).

**COMORBIDITY** Another problem with the *DSM-5* is that people seldom fit neatly into the precise categories provided. Indeed, many psychological disorders occur together even though the *DSM-5* treats them as separate disorders—for example, depression and anxiety, or depression and substance abuse. This state is known as *comorbidity*

(FIGURE 14.5). Accordingly, people who are found to be depressed should also be examined for comorbid conditions. Though people may be diagnosed with two or more disorders, a dual diagnosis offers no advantages in terms of treatment because both conditions will usually respond to the same treatment.

It is possible that psychological disorders are comorbid because of common underlying factors. Although the *DSM-5* separates disorders involving anxiety from those involving depression, both types involve the trait *neuroticism*, the tendency to experience frequent and intense negative emotions (Barlow, Sauer-Zavala, Carl, Bullis, & Ellard, 2014). In fact, it has recently been proposed that psychopathology reflects a common general factor, analogous to general intelligence (or g, discussed in Chapter 8). Avshalom Caspi and colleagues (2014) examined symptoms of psychopathology in a large sample of individuals who were studied for more than 30 years, from childhood to middle adulthood. They found that one underlying factor, which they called the *p factor*, was involved in all types of psychological disorders. Higher scores on the p factor were associated with more life impairment, such as suicide attempts, psychiatric hospitalizations, and criminal behaviors. High p scores also predicted a worsening of impairments over time. Just as the g dimension of intelligence reflects low to high cognitive abilities, the p dimension reflects low to high psychopathology severity.

**RESEARCH DOMAIN CRITERIA (RDoC)** The U.S. National Institute of Mental Health (NIMH) has proposed an entirely new way of classifying and understanding psychological disorders (Insel et al., 2010). Whereas the *DSM* approach classifies disorders by observable symptoms, the **Research Domain Criteria (RDoC)** method defines basic *domains* of functioning (such as attention, social communication, anxiety) and considers them across multiple levels of analysis, from genes to brain

### Research Domain Criteria (RDoC)

A method that defines basic aspects of functioning and considers them across multiple levels of analysis, from genes to brain systems to behavior.

systems to behavior. For example, researchers might study attention problems for people with anxiety disorders, depression, schizophrenia, and posttraumatic stress disorder.

The RDoC initiative is initially meant to guide research rather than classify disorders for treatment. The goal of the initiative is to understand the processes that give rise to disordered thoughts, emotions, and behaviors. Identifying the cause of these symptoms ultimately may provide insight into treating them.

The RDoC approach capitalizes on recent advances in genetics, neuroscience, and psychology in understanding adaptive behavior, as well as how functioning can be disrupted by various disorders (National Institute of Mental Health, 2011). For example, as you will learn later in this chapter, the same genetic mutation may be involved in a number of apparently different psychological disorders. This mutation may affect how neurotransmitters function and cause similar impairments in thought processes across those disorders. In other instances, people diagnosed with the same *DSM* disorder can show radically different behaviors or responses, which might indicate that two different disorders share the same *DSM* diagnosis. Because of such problems, RDoC examines psychopathology without regard to *DSM* diagnoses. The ultimate aim of the RDoC initiative is for the classification and treatment of psychological disorders to be based on the underlying biological and psychosocial causes (Insel, 2014).

Some critics of RDoC argue that it is moving too quickly and that an abrupt shift in diagnostic criteria is no guarantee of better treatment results (Marder, 2014). Others worry that this initiative is focused on neuroscience at the expense of understanding personal experience (Parnas, 2014). At least within the United States, however, because the NIMH funds the majority of research, RDoC will be a driving force for research on psychopathology.

## Psychological Disorders Must Be Assessed

Physical disorders can generally be detected by medical tests, such as blood tests or biopsies. Determining whether a person has a mental disorder is not as straightforward. Examining a person's mental functions and psychological condition to diagnose a psychological disorder is known as **assessment**. This process often includes self-reports, psychological testing, observations, and interviews. It may also involve neuropsychological testing.

In the neuropsychological method, the client performs actions such as copying a picture; drawing a design from memory; sorting cards that show various stimuli into categories based on size, shape, or color; placing blocks into slots on a board while blindfolded; and tapping fingers rapidly (**FIGURE 14.6**). Each task requires an ability such as planning, coordinating, or remembering. By highlighting actions that the client performs poorly, the assessment might indicate problems with a particular brain region. For instance, people who have difficulty switching from one rule to another for categorizing objects, as in sorting by shape rather than by color, may have impairments in the frontal lobes. Subsequent assessment with MRI or PET (brain imaging techniques discussed in Chapter 3, "Biology and Behavior") might indicate brain damage caused by a tumor or by an injury. Often a medical evaluation is indicated. For instance, the symptoms of depression or anxiety disorder can be similar to those of hypothyroidism, an endocrine disorder that should be ruled out before the psychological disorder is treated (Giynas Ayhan, Uguz, Askin, & Gonen, 2014).

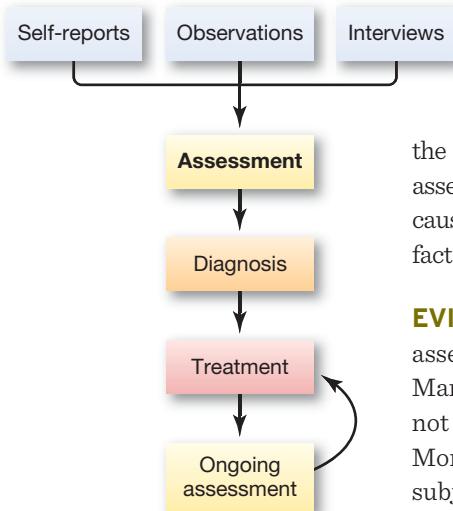
### assessment

In psychology, examination of a person's cognitive, behavioral, or emotional functioning to diagnose possible psychological disorders.



**FIGURE 14.6**  
**Neuropsychological Testing**

The assessment depicted here uses a neuropsychological test to examine mental function. In this timed test, a researcher watches a client fit wooden blocks into a corresponding template to test for signs of Alzheimer's disease.



**FIGURE 14.7**

### Assessing a Client

Clinical psychologists examine a person's mental functions and psychological health to diagnose a psychological disorder and determine an appropriate treatment. This flowchart shows the factors that lead to treatment.

### diathesis-stress model

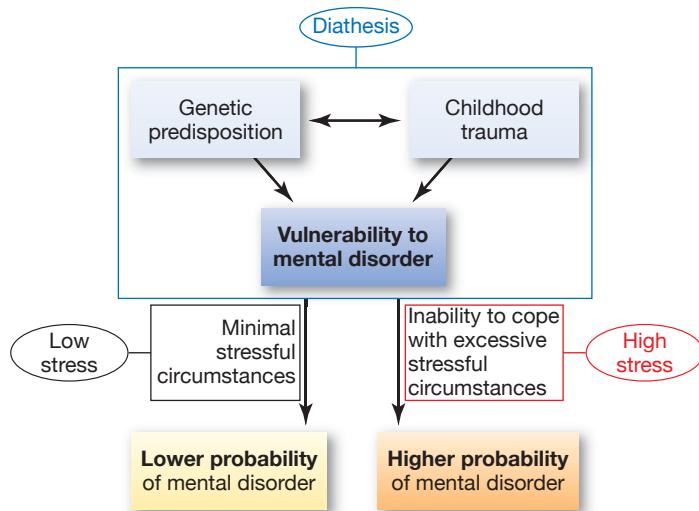
A diagnostic model that proposes that a disorder may develop when an underlying vulnerability is coupled with a precipitating event.

The primary goal of assessment is to make a diagnosis so that appropriate treatment can be provided. The course and probable outcome, or *prognosis*, will depend on the particular psychological disorder that is diagnosed. Therefore, a correct diagnosis will help the client—and perhaps the client's family—understand what the future might bring. Assessment does not stop with diagnosis, however. Ongoing assessment helps mental health workers understand whether specific situations might cause a worsening of the disorder, whether progress is being made in treatment, and other factors that might help in understanding unique aspects of a given case (FIGURE 14.7).

**EVIDENCE-BASED ASSESSMENT** A key question is whether psychological assessments provide information that is useful for treating psychological disorders. Many popular methods of assessment, such as projective tests (see Chapter 13), have not been shown to be helpful in predicting the kinds of treatments that are useful. Moreover, individual clinicians often choose assessment procedures based on their subjective beliefs and training rather than on scientific studies. For instance, when making diagnoses, some clinicians use their clinical judgment rather than a formal method—such as a structured interview that consists of standardized questions, derived from *DSM* criteria, in the same order each time.

*Evidence-based assessment* is an approach to clinical evaluation in which research guides the evaluation of psychopathology, the selection of appropriate psychological tests and neuropsychological methods, and the use of critical thinking in making a diagnosis (Hunsley & Mash, 2007; Joiner, Walker, Pettit, Perez, & Cukrowicz, 2005). For instance, as noted earlier, scientific research indicates that many disorders are comorbid. Research also indicates that people who are depressed often have substance abuse disorders. Therefore, an evidence-based assessment approach would indicate that people found to be depressed should also be assessed for comorbid conditions, such as substance abuse.

## Psychological Disorders Have Many Causes



**FIGURE 14.8**

### Diathesis-Stress Model

The onset of psychological disorders can be seen as resulting from the interactions of a diathesis and stress. The diathesis may be biological (e.g., genetic predisposition), environmental (e.g., childhood trauma), or both.

Psychologists do not completely agree about the causes of most psychopathology. Still, some factors are thought to play important developmental roles. As discussed throughout this book, both nature and nurture matter, and it is futile to try to identify biology or environment as solely responsible for a given disorder. The **diathesis-stress model** (presented as a flowchart in FIGURE 14.8) provides one way of thinking about the onset of psychopathology (Monroe & Simons, 1991).

In this model, an individual can have an underlying vulnerability (known as *diathesis*) to a psychological disorder. This diathesis can be biological, such as a genetic predisposition to a specific disorder, or it can be environmental, such as childhood trauma. The vulnerability may not be sufficient to trigger a disorder, but the addition of stressful circumstances can tip the scales. If the stress level exceeds an individual's ability to cope,

the symptoms of psychological disorder will occur. In this view, a family history of psychopathology suggests vulnerability rather than destiny.

**BIOLOGICAL FACTORS** The biological perspective focuses on how physiological factors, such as genetics, contribute to psychological disorders (Kandel, 1998).

Chapter 3 describes how comparing the rates of psychological disorders between identical and fraternal twins and studying individuals who have been adopted have revealed the importance of genetic factors (Kendler, Prescott, Myers, & Neale, 2003; Krueger, 1999). Genetic factors can affect the production and levels of neurotransmitters and their receptor sites. Research has provided insights on the role of neurotransmitters in psychopathology. In some cases, based on what is known about the neurochemistry of psychological disorders, medications have been developed. In other cases, the unexpected effects of medications have led to discoveries about the neurotransmitters involved in psychological disorders.

Genetic factors can also affect the size of brain structures and their level of connectivity. Structural imaging and postmortem studies have revealed differences in brain anatomy, perhaps due to genetics, between those with psychological disorders and those without. Functional neuroimaging is currently at the forefront of research into the neurological components of mental disorders: PET and fMRI have revealed brain regions that may function differently in individuals with mental disorders (**FIGURE 14.9**).

Environmental effects on the body also influence the development and course of psychological disorders. The fetus is particularly vulnerable to other biological factors—such as malnutrition, exposure to toxins (such as drugs and alcohol), and maternal illness—that because of their effects on the central nervous system may contribute to psychological disorders (Salum, Polanczyk, Miguel, & Rohde, 2010). Similarly, during childhood and adolescence, environmental toxins and malnutrition can put an individual at risk for psychological disorders. Again, biological factors often reflect the vulnerabilities that occur in individuals. As the diathesis-stress model reminds us, single explanations (nature *or* nurture, rather than nature *and* nurture) are seldom sufficient for understanding psychological disorders.

**ENVIRONMENTAL FACTORS** The first edition of the *DSM* was influenced heavily by Freudian psychoanalytic theory. Freud believed that psychopathology was mostly due to unconscious conflicts, often sexual in nature, dating back to childhood. Consistently with this perspective, the first edition of the *DSM* described many disorders as reactions to environmental conditions or as involving various defense mechanisms. Although Freud made important historical contributions in shaping psychology, most of his theories—particularly his theories on the causes of psychopathology—have not stood the test of time. Environmental factors clearly play an important role, however, in the expression and treatment of psychological disorders.

Thoughts and emotions shaped by an environment can profoundly influence behavior, including disordered behavior. Not only traumatic events but also less extreme circumstances, such as constantly being belittled by a parent, can have long-lasting effects. The **family systems model** proposes that an individual's behavior must be considered within a social context, particularly within the family (Kazak, Simms, & Rourke, 2002). According to this model, problems that arise within an individual are manifestations of problems within the family (Goodman & Gotlib, 1999). Thus, developing a profile of an individual's family interactions can be important for understanding the factors that may be contributing to the disorder. A profile can also be important for determining whether the family is likely to be helpful or detrimental to the client's progress in therapy.

Similarly, the **sociocultural model** views psychopathology as the result of the interaction between individuals and their cultures. For example, disorders such as schizophrenia appear to be more common among those in lower socioeconomic classes (**FIGURE 14.10**). From the sociocultural perspective, these differences in occurrence are due to differences in lifestyles, in expectations, and in opportunities among classes. There may be biases in people's willingness to ascribe disorders to different social classes, however.



**FIGURE 14.9**  
**Biological Factors in Psychopathology**

Although these men are twins, the one on the right has schizophrenia and the one on the left does not. In the MRI of the twin with schizophrenia, note the larger ventricles (these fluid-filled cavities appear dark in the image). This same pattern has emerged in the study of other twin pairs in which one has schizophrenia and the other does not. Thus, the brain may be deteriorating over time for those with schizophrenia, and this finding tells us that biological factors may be important for understanding schizophrenia.

#### family systems model

A diagnostic model that considers problems within an individual as indicating problems within the family.

#### sociocultural model

A diagnostic model that views psychopathology as the result of the interaction between individuals and their cultures.



**FIGURE 14.10** **Sociocultural Model of Psychopathology**  
According to the sociocultural model, psychopathology results from the interaction between individuals and their cultures. This homeless man with schizophrenia lives on the streets in Notting Hill, a fashionable area of London.

### cognitive-behavioral approach

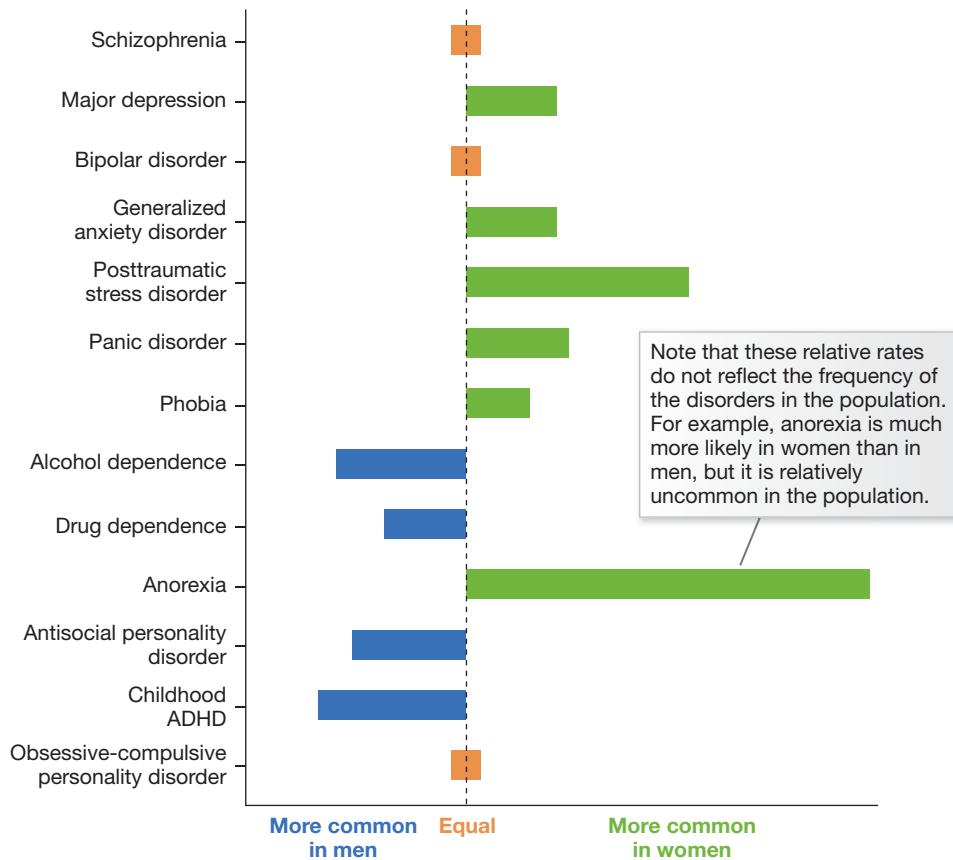
A diagnostic model that views psychopathology as the result of learned, maladaptive thoughts and beliefs.

Eccentric behavior among the wealthy elite might be tolerated or viewed as amusing. The same behaviors observed among those living in poverty might be taken as evidence of psychopathology. Moreover, people who develop schizophrenia may have trouble finding work, and so their lower socioeconomic status may be a result of their disorder.

**COGNITIVE-BEHAVIORAL FACTORS** The central principle of the **cognitive-behavioral approach** is that abnormal behavior is learned (Butler, Chapman, Forman, & Beck, 2006). As discussed in Chapter 6, through classical conditioning an unconditioned stimulus produces an unconditioned response. For example, a loud noise produces a startled response. A neutral stimulus paired with this unconditioned stimulus can eventually by itself produce a similar response. As was the case with Little Albert, if a child is playing with a fluffy white rat and is frightened by a loud noise, the white rat alone can later cause fear in the child. In fact, this process is how John B. Watson, the founder of behaviorism, demonstrated that many fears are learned rather than innate.

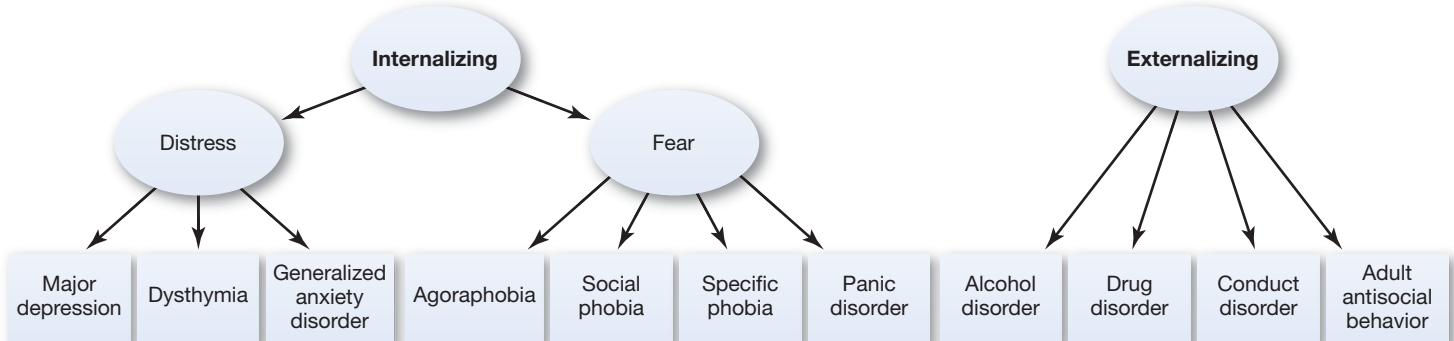
According to the cognitive-behavioral perspective, thoughts and beliefs are learned and therefore can be unlearned through treatment. The premise of this approach is that thoughts can become distorted and produce maladaptive behaviors and maladaptive emotions. In contrast to the psychologists who subscribe to the psychoanalytic perspective, cognitive-behavioral psychologists believe that thought processes are available to the conscious mind. Individuals are aware of, or easily can be made aware of, the thought processes that give rise to maladaptive emotions and behaviors.

**SEX DIFFERENCES IN PSYCHOLOGICAL DISORDERS** Some disorders, such as schizophrenia and bipolar disorder, are equally likely in both sexes (**FIGURE 14.11**). Rates of other disorders vary between the sexes. For example, dependence on alcohol is much more likely in males, whereas anorexia nervosa is much more likely in females. The reasons for these differences are both biological and environmental.



**FIGURE 14.11**  
**Sex Differences in Psychological Disorders**

The bars in this graph represent how common particular psychological disorders are for men and for women.



**FIGURE 14.12**

### Internalizing and Externalizing Model of Psychological Disorders

This diagram divides disorders into two basic categories, internalizing and externalizing. It also divides internalizing disorders into those related to fear and those related to distress.

SOURCE: Krueger & Markon, 2006.

One way of categorizing psychological disorders is to divide them into two major groups: *Internalizing disorders* are characterized by negative emotions, and they can be grouped into categories that reflect the emotions of distress and fear. These disorders include major depressive disorder, generalized anxiety disorder, and panic disorder. *Externalizing disorders* are characterized by disinhibition. These disorders include alcoholism, conduct disorders, and antisocial personality disorder (FIGURE 14.12). In general, the disorders associated with internalizing are more prevalent in females, and those associated with externalizing are more prevalent in males (Krueger & Markon, 2006).

**CULTURE AND PSYCHOLOGICAL DISORDERS** Increasingly, psychologists and other mental health professionals are recognizing the importance of culture in many aspects of our lives. Most psychological disorders show both universal and culture-specific symptoms. That is, the disorders may be very similar around the world, but they still reflect cultural differences. A disorder with a strong biological component will tend to be more similar across cultures. A disorder heavily influenced by learning, context, or both is more likely to differ across cultures. For example, depression is a major mental health problem around the world, but the manifestations of depression differ by culture.

Since the 1994 edition, the *DSM* has included a number of disorders that tend to occur within specific cultural settings. The *DSM-5* incorporates a greater consideration of cultural factors for each psychological disorder and updates criteria to reflect cross-cultural variations in how people exhibit symptoms. For example, the fear of “offending others” has been added as a possible symptom of social anxiety disorder to reflect the collectivist cultural concept that not harming others is as important as not harming the self. *DSM-5* also provides more details about cultural concepts of distress, such as encouraging clinicians to consider the specific words and phrases used by different cultural groups to describe their distress, as well as the cultural explanations for the cause of psychopathology. Finally, *DSM-5* provides examples of *cultural syndromes*, disorders that include a cluster of symptoms that are found in specific cultural groups or regions (TABLE 14.2 presents examples of common cultural syndromes, and FIGURE 14.13 illustrates one of them).

Clinicians and researchers need to be sensitive to cultural issues to avoid making mistakes in their diagnoses and treatments (Marsella & Yamada, 2007). Cultural factors can be critical in determining how a disorder is expressed and how an individual will respond to different types of therapies.



**FIGURE 14.13**

### Taijin Kyofusho

This Japanese woman may be exhibiting symptoms of a psychological syndrome unique to her culture.

**Table 14.2** Cultural Syndromes

NAME	DEFINITION AND LOCATION
<b>Ataque de nervios</b>	Uncontrollable shouting and/or crying; verbal and physical aggression; heat in chest rising to head; feeling of losing control; occasional amnesia for experience (Caribbean and South American Latinos).
<b>Dhat syndrome</b>	Anxiety, fatigue, weakness, weight loss, and other bodily complaints; typically observed in young males who believe their symptoms are due to loss of semen (South Asia).
<b>Khyâl cap</b>	Belief that a “windlike” substance may rise in the body and cause serious effects; acute panic, autonomic arousal, anxiety; catastrophic cognitions (Cambodians in the United States and Cambodia).
<b>Kufungisia</b>	Belief that thinking too much can damage the mind and body; an explanation for anxiety, depression, and somatic problems indicating distress (Zimbabwe).
<b>Maladi moun</b>	A cultural explanation that sickness has been sent by people to harm their enemies; visible success makes one vulnerable to attack; causes various illnesses, including psychosis, depression, and social failure (Haiti).
<b>Nervios</b>	A phrase used to refer to a general state of vulnerability to stressful life experience; common symptoms include headaches and “brain aches” as well as irritability and nervousness (Latinos in the United States and Latin America).
<b>Shenjing shuairuo</b>	A weakness in the nervous system; mental fatigue, negative emotions, excitement, nervous pain, and sleep disturbances; caused by stress, embarrassment, or acute sense of failure (China).
<b>Susto</b>	An illness attributed to a frightening event that causes the soul to leave the body; sadness, somatic complaints, lack of motivation, and difficulty functioning in daily living (Latinos in the United States and Latin America).
<b>Taijin kyofusho</b>	Intense fear of interpersonal relations; belief that parts of the body give off offensive odors or displease others (Japan).

SOURCE: Based on American Psychiatric Association (2013).

## Summing Up

### How Are Psychological Disorders Conceptualized and Classified?

- Because psychopathology takes many forms, psychological disorders are difficult to define and categorize. The behavioral manifestations vary widely, but people diagnosed with these disorders have two things in common: Their behavior deviates from cultural norms and is maladaptive.
- The *DSM-5* is used by clinicians to classify and diagnose psychological disorders.
- Psychological disorders are often comorbid—that is, they occur together.
- Due to comorbidity, it has been proposed that all psychological disorders involve a common underlying factor, p. High scores on the p factor have been found to be associated with more-severe psychopathology.
- Rather than classifying disorders, the Research Domain Criteria (RDoC) method strives to understand the processes that give rise to psychopathology. The RDoC defines basic domains of functioning such as attention and social communication and considers them across multiple levels of analysis, from genes to brain systems to behavior.

- Clinical assessments help with the diagnosis and treatment of psychological disorders. Assessments may include interviews, behavioral assessments, psychological tests, and neuropsychological tests. The diathesis-stress model suggests that psychological disorders result from an underlying vulnerability coupled with a stressful, precipitating event.
- The causes of most psychopathology are unknown and may result from complex interactions between biological, environmental, and cognitive-behavioral factors.
- In general, females are more likely to suffer from internalizing disorders, such as major depressive disorder and generalized anxiety disorder. Males are more likely to suffer from externalizing disorders, such as alcohol use disorder and conduct disorder.
- Most psychological disorders show both universal and culture-specific symptoms. Disorders that are largely biologically determined tend to be more similar across cultures than disorders that are strongly influenced by learning and context. The *DSM* includes a number of cultural syndromes—that is, disorders that occur in specific cultures or regions.

## Measuring Up

1. Which of the following statements are true?
  - \_\_\_\_\_ Neuropsychological assessments are useful for determining brain regions that may be impaired.
  - \_\_\_\_\_ The primary goal of psychological assessment is diagnosis.
  - \_\_\_\_\_ Psychological disorders are assessed only through client self-report of maladaptive thoughts, maladaptive behaviors, or both.
  - \_\_\_\_\_ Neuropsychological testing might involve asking patients to sort cards or draw pictures from memory.
2. Mitch has struggled with psychological problems for most of his adult life. In high school and college, he experimented with alcohol and drugs and suffered through periodic bouts of depression and anxiety. As he has gotten older, Mitch's symptoms and subsequent problems have escalated, including his substance abuse. Mitch would most likely score high on which of the following factors?
  - g factor
  - p factor
  - unconscious conflicts
  - the neuropsychological inventory
3. While at a conference on psychological disorders, you attend a symposium titled "Understanding the Origins of Mental Health." Excerpts from three of the presentations appear below. Match each excerpt with one of the etiological models discussed in this chapter: diathesis-stress model, biological model, family systems model, sociocultural model, and cognitive-behavioral approach. (Not all the models will have matches.)
  - "By understanding the mechanisms by which neurotransmitters affect behavior and cognition and emotion, we gain insight into the underlying causes of psychological disorders."
  - "Children are not raised in vacuums; they are raised in families. Therefore, to understand the origins of psychological disorders, we must understand the dynamics of the client's family."
  - "Some individuals are predisposed—whether as a function of their biology or of their past experiences—to develop psychological disorders. Stressful circumstances amplify these predispositions, making the individual more likely to evidence symptoms of psychopathology."

(2) b. p factor; (3) a. biological; b. family systems model; c. diathesis-stress model.  
**ANSWERS:** (1) Choices a, b, and d are true.

## Learning Objectives

- Differentiate the various anxiety disorders.
- Understand the various causes of obsessive-compulsive disorder.
- Understand the role of trauma in posttraumatic stress disorders.
- Discuss cultural and sex differences in depressive disorders.
- Distinguish between bipolar I and bipolar II disorder.

## 14.2 Which Disorders Emphasize Emotions or Moods?

People often feel emotional—down, anxious, and so on. Such feelings can be useful. They can prepare people for dealing with future events, motivating them to learn new ways of coping with challenges. For example, being anxious about tests reminds people to keep up with their homework and study. Being slightly anxious when meeting strangers helps people avoid doing bizarre things and making bad impressions. For some people, however, feelings such as anxiety can become debilitating and interfere with every aspect of life.

Likewise, moods color every aspect of people's lives. When people are happy, the world seems like a wonderful place, and they feel boundless energy. When people are sad, they view the world in a decidedly less rosy light, feeling hopeless and isolated. Few people, however, experience the same strong moods day after day.

When emotions or moods go from being a normal part of daily living to being extreme enough to disrupt people's ability to work, learn, and play, these states are considered symptoms of psychological disorders. Most forms of psychopathology influence how people feel as well as how they think, but emotional experiences or moods are more central to some disorders and thought disturbances are more central to others. In this section, we consider the most common disorders involving emotions or moods.

### Anxiety Disorders Make People Apprehensive and Tense

Imagine that you are about to make your first parachute jump out of an airplane. If you are like most people, your heart will be racing, and you will be sweating. You might feel queasy. While under these circumstances you may find such sensations thrilling, people who have an anxiety disorder experience these feelings all the time and are unhappy about them.

**Anxiety disorders** are characterized by excessive fear and anxiety in the absence of true danger. Those with anxiety disorders feel tense and apprehensive. They are often irritable because they cannot see any solution to their anxiety. Constant worry can make falling asleep and staying asleep difficult, and attention span and concentration can be impaired. By continually arousing the autonomic nervous system, chronic anxiety also causes bodily symptoms such as sweating, dry mouth, rapid pulse, shallow breathing, increased blood pressure, and increased muscular tension. Chronic arousal can also result in hypertension, headaches, and other health problems. More than 1 in 4 Americans will have some type of anxiety disorder during their lifetimes (Kessler & Wang, 2008).

Because of their high levels of autonomic arousal, people who have anxiety disorders also exhibit restless and pointless motor behaviors. Exaggerated startle response is typical, and behaviors such as toe tapping and excessive fidgeting are common. Problem solving and judgment may suffer as well. Research has shown that chronic stress can produce atrophy in the hippocampus, a brain structure involved in learning and memory (McEwen, 2008). Because chronic stress can damage the body, including the brain, it is very important to identify and effectively treat disorders that involve chronic anxiety. The various anxiety disorders share some emotional, cognitive, somatic, and motor symptoms, even though the behavioral manifestations of these disorders are quite different (Barlow, 2002). These disorders include specific phobia, social anxiety disorder, generalized anxiety disorder, and panic disorder.

#### anxiety disorder

A psychological disorder characterized by excessive fear and anxiety in the absence of true danger.

**Table 14.3 Some Unusual Specific Phobias**

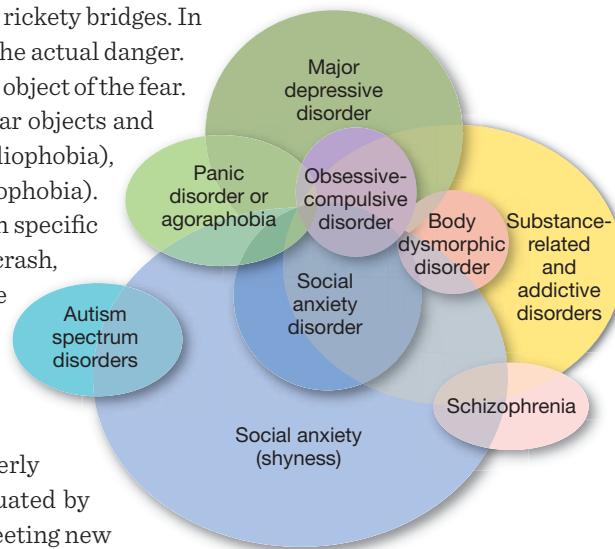
- **Arachibutyrophobia:** fear of peanut butter sticking to the roof of one's mouth
- **Automatonophobia:** fear of ventriloquists' dummies
- **Barophobia:** fear of gravity
- **Dextrophobia:** fear of objects at the right side of the body
- **Geliophobia:** fear of laughter
- **Gnomophobia:** fear of garden gnomes
- **Hippopotomonstrosesquipedaliophobia:** fear of long words
- **Ochophobia:** fear of being in a moving automobile
- **Panophobia:** fear of everything
- **Pentheraphobia:** fear of mothers-in-law
- **Triskaidekaphobia:** fear of the number 13

**SPECIFIC PHOBIA** As discussed in Chapter 6, a phobia is a fear of a specific object or situation. Of course, some fear can be a good thing. As an adaptive force, fear can help people avoid potential dangers, such as poisonous snakes and rickety bridges. In phobias, however, the fear is exaggerated and out of proportion to the actual danger.

In *DSM-5*, people are diagnosed with *specific phobia* based on the object of the fear. Specific phobias, which affect about 1 in 8 people, involve particular objects and situations. Common specific phobias include fear of snakes (*ophidiophobia*), fear of enclosed spaces (*claustrophobia*), and fear of heights (*acrophobia*). (**TABLE 14.3** lists some unusual specific phobias.) Another common specific phobia is fear of flying. Even though the odds of dying in a plane crash, compared with a car crash, are extraordinarily small, some people find flying terrifying. For those who need to travel frequently for their jobs, a fear of flying can cause significant impairment in daily living.

**SOCIAL ANXIETY DISORDER** *Social anxiety disorder*, formerly sometimes called *social phobia*, is a fear of being negatively evaluated by others. It includes fears of public speaking, speaking up in class, meeting new people, and eating in front of others. About 1 in 8 people will experience social anxiety disorder at some point in their lifetime, and around 7 percent are experiencing social anxiety disorder at any given time (Ruscio et al., 2008). It is one of the earliest forms of anxiety disorder to develop, often beginning at around age 13. The more social fears a person has, the more likely he or she is to develop other disorders, particularly depression and substance abuse problems. Indeed, assessment must consider the overlap between social anxiety disorder and related disorders to make an informed diagnosis (Stein & Stein, 2008; **FIGURE 14.14**).

**GENERALIZED ANXIETY DISORDER** The anxiety in specific phobia has a focus. By contrast, the anxiety in **generalized anxiety disorder (GAD)** is diffuse and always present. People with this disorder are constantly anxious and worry incessantly about even minor matters (Sanderson & Barlow, 1990). They even worry about being worried! Because the anxiety is not focused, it can occur in response to almost anything, so the sufferer is constantly on the alert for problems. This hypervigilance results in distractibility, fatigue, irritability, and sleep problems, as



**FIGURE 14.14**  
**Comorbidity of Social Anxiety Disorder**

As this diagram illustrates, social anxiety disorder is comorbid with many other psychological disorders. If a client has social anxiety disorder, all of these disorders need to be considered to make an accurate and complete diagnosis.

#### **generalized anxiety disorder (GAD)**

A diffuse state of constant anxiety not associated with any specific object or event.

### **panic disorder**

An anxiety disorder that consists of sudden, overwhelming attacks of terror.

### **agoraphobia**

An anxiety disorder marked by fear of being in situations in which escape may be difficult or impossible.

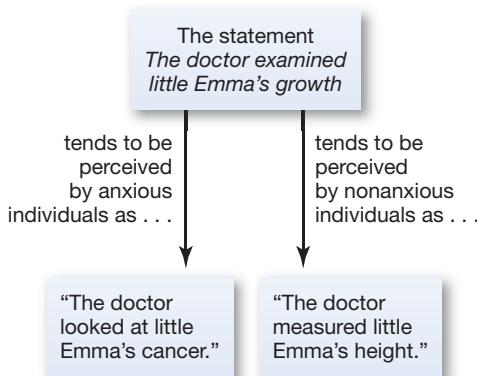
well as headaches, restlessness, light-headedness, and muscle pain. Just under 6 percent of the United States population is affected by this disorder at some point in their lives, though women are diagnosed more often than men (Kessler et al., 1994; Kessler & Wang, 2008).

**PANIC DISORDER** Panic disorder consists of sudden, overwhelming attacks of terror and worry about having additional panic attacks. The attacks seemingly come out of nowhere, or they are cued by external stimuli or internal thought processes. Panic attacks typically last for several minutes, during which the person may begin to sweat and tremble; feels his or her heart racing; feels short of breath; feels chest pain; and may feel dizzy and light-headed, with numbness and tingling in the hands and feet. People experiencing panic attacks often believe that they are going crazy or that they are dying, and those who suffer from persistent panic attacks attempt suicide much more often than those in the general population (Fawcett, 1992; Korn et al., 1992; Noyes, 1991). People who experience panic attacks during adolescence are especially likely to develop other anxiety disorders—such as generalized anxiety disorder—in adulthood (Goodwin et al., 2004). Panic disorder affects an estimated 3 percent of the population in a given year, and women are twice as likely to be diagnosed as men (Kessler & Wang, 2008).

A related disorder is **agoraphobia**. People with this disorder are afraid of being in situations in which escape is difficult or impossible. For example, they may fear being in a crowded shopping mall or using public transportation. Their fear is so strong that being in such situations causes panic attacks. As a result, people who suffer from agoraphobia avoid going into open spaces or to places where there might be crowds. In extreme cases, sufferers may feel unable to leave their homes. In addition to fearing the particular situations, they fear having a panic attack in public:

Ms. Watson began to dread going out of the house alone. She feared that while out she would have an attack and would be stranded and helpless. She stopped riding the subway to work out of fear she might be trapped in a car between stops when an attack struck, preferring instead to walk the 20 blocks between her home and work. She also severely curtailed her social and recreational activities—previously frequent and enjoyed—because an attack might occur, necessitating an abrupt and embarrassing flight from the scene. (Spitzer, Skodol, Gibbon, & Williams, 1983)

This description demonstrates the clear links between panic attacks and agoraphobia. Indeed, agoraphobia without panic is quite rare (Kessler & Wang, 2008).



**FIGURE 14.15**  
**Anxiety Disorders**

As this example illustrates, anxious individuals tend to perceive ambiguous situations as threatening.

**DEVELOPMENT OF ANXIETY DISORDERS** The behavioral manifestations of anxiety disorders can be quite different, but all share some causal factors (Barlow, 2002). The first factor is biased thinking. When presented with ambiguous or neutral situations, anxious individuals tend to perceive them as threatening, whereas nonanxious individuals assume they are nonthreatening (Eysenck, Mogg, May, Richards, & Matthews, 1991; FIGURE 14.15). Anxious individuals also focus excessive attention on perceived threats (Rinck, Reinecke, Ellwart, Heuer, & Becker, 2005). They thus recall threatening events more easily than nonthreatening ones, exaggerating the events' perceived magnitude and frequency.

A second factor is learning. As discussed in Chapter 6, monkeys develop a fear of snakes if they observe other monkeys responding to snakes fearfully. Similarly, a person could develop a fear of flying by observing another person's fearful reaction to the closing of cabin doors. Such a fear might then generalize to other enclosed spaces, resulting in claustrophobia.

# Scientific Thinking

## Inhibition and Social Anxiety

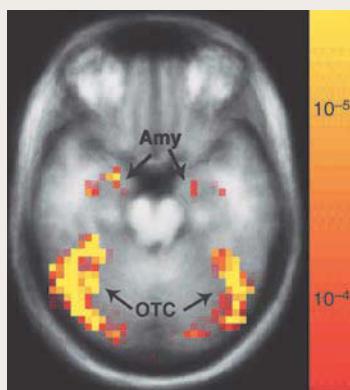
**HYPOTHESIS:** People who had an inhibited temperamental style as children are more likely to show signs of social anxiety later in life.

### RESEARCH METHOD:

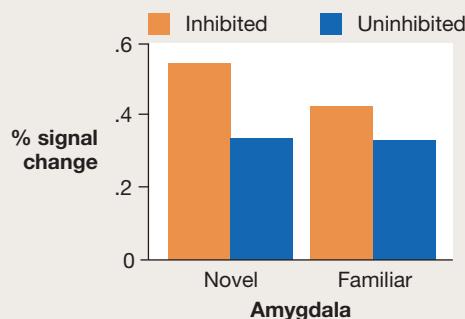
- 1 Adults received brain scans while viewing pictures of familiar faces and of novel faces. One group of these adults had been categorized as inhibited before age 2. The other group had been categorized as uninhibited before age 2.



- 2 Two regions of the brain were more activated by novel faces. These areas were the amygdala (marked "Amy" in the brain scan below) and the occipitotemporal cortex (marked "OTC"). The amygdala is normally active when people are threatened. The occipitotemporal cortex is normally active when people see faces, whether the faces are novel or familiar.



**RESULTS:** Compared with the uninhibited group, the inhibited group showed greater activation of the amygdala while viewing novel faces. That activation indicated that, when seeing novel faces, the inhibited group showed greater brain activity associated with threat.



**CONCLUSION:** The results suggest that some aspects of childhood temperament are preserved in the adult brain. In particular, biological factors seem to play an important role in social anxiety.

**SOURCE:** Schwartz, C. E., Wright, C. I., Shin, L. M., Kagan, J., & Rauch, S. L. (2003, June 20). Inhibited and uninhibited infants "grown up": Adult amygdalar response to novelty. *Science*, 300, 1952–1953.

There is also a biological factor. As noted in Chapter 13, children who have an inhibited temperamental style are usually shy and tend to avoid unfamiliar people and novel objects. These inhibited children are more likely to develop anxiety disorders later in life (Fox, Henderson, Marshall, Nichols, & Ghera, 2005). They are especially at risk for developing social anxiety disorder (Biederman et al., 2001). In one study, adults received brain scans while viewing pictures of familiar faces and of novel faces (Schwartz, Wright, Shin, Kagan, & Rauch, 2003). One group of these adults had been categorized as inhibited before age 2. The other group had been categorized as uninhibited before age 2. Compared with the uninhibited group, the inhibited group showed greater activation of the amygdala—a brain region involved when people are threatened—while viewing the novel faces. That is, after the passage of so many years, the inhibited group still seemed to show a threat response to novel faces. Although this study did not involve those with diagnosed anxiety disorders, it does show that childhood temperaments are preserved in the adult brain (see "Scientific Thinking: Inhibition and Social Anxiety").

## Unwanted Thoughts Create Anxiety in Obsessive-Compulsive Disorders

We have seen that many psychological disorders involve both emotional and cognitive impairments. In some cases, having unwanted thoughts leads to emotional distress and anxiety. *DSM-5* categorizes a number of disorders together that involve

### **obsessive-compulsive disorder (OCD)**

A disorder characterized by frequent intrusive thoughts and compulsive actions.



"Is the Itsy Bitsy Spider obsessive-compulsive?"



**FIGURE 14.16 Howie Mandel**

The comedian Howie Mandel has been diagnosed with obsessive-compulsive disorder. Like many people with OCD, Mandel suffers from mysophobia, or the fear of germs. His trademark shaved head helps him with this problem, as it makes him feel cleaner. Mandel even built a second, sterile house, to which he can retreat if he feels he might be contaminated by anyone around him. Here, Mandel promotes his autobiography, *Here's the Deal: Don't Touch Me* (2009), in which he "comes clean" about suffering from OCD and other disorders.

experiencing unwanted thoughts or the desire to engage in maladaptive behaviors (see Table 14.1). The commonality is an obsession with an idea or thought or the compulsion to repeatedly act in a certain way. These compulsive actions temporarily reduce anxiety. Related disorders in this category include people chronically pulling at their hair or picking at their skin, people being obsessed with deficiencies in their physical appearance, and *hoarding disorder*, in which people have persistent difficulty parting with their possessions and end up accumulating clutter and garbage that can make their living conditions seem like disaster zones.

**OBSSESSIVE-COMPULSIVE DISORDER** The most common disorder in this *DSM-5* category is **obsessive-compulsive disorder (OCD)**, which involves frequent intrusive thoughts and compulsive actions (Kessler & Wang, 2008). Affecting 1–2 percent of the population, OCD is more common in women than men, and it generally begins in early adulthood (Robins & Regier, 1991; Weissman et al., 1994).

*Obsessions* are recurrent, intrusive, and unwanted thoughts or ideas or mental images. They often include fear of contamination, of accidents, or of one's own aggression. The individual typically attempts to ignore or suppress such thoughts but sometimes engages in particular behaviors to neutralize his or her obsessions (**FIGURE 14.16**).

*Compulsions* are particular acts that the OCD patient feels driven to perform over and over. The most common compulsive behaviors are cleaning, checking, and counting. For instance, a person might continually check to make sure a door is locked, because of an obsession that his or her home might be invaded, or a person might engage in superstitious counting to protect against accidents, such as counting the number of telephone poles while driving. The compulsive behavior or mental act, such as counting, is aimed at preventing or reducing anxiety or preventing something dreadful from happening.

Those with OCD anticipate catastrophe and loss of control. However, as opposed to those who suffer from anxiety disorders—who fear what might happen to them—those with OCD fear what they might do or might have done. Checking is one way to calm the anxiety:

While in reality no one is on the road, I'm intruded with the heinous thought that I *might* have hit someone . . . a human being! God knows where such a fantasy comes from. . . . I try to make reality chase away this fantasy. I reason, "Well, if I hit someone while driving, I would have *felt* it." This brief trip into reality helps the pain dissipate . . . but only for a second. . . . I start ruminating, "Maybe I did hit someone and didn't realize it. . . . Oh my God! I might have killed somebody! I have to go back and check." (Rapoport, 1990, pp. 22–27)

**CAUSES OF OBSESSIVE-COMPULSIVE DISORDER** A paradoxical aspect of OCD is that people are aware that their obsessions and compulsions are irrational, yet they are unable to stop them. One explanation is that the disorder results from conditioning. In the person with OCD, anxiety is somehow paired to a specific event, probably through classical conditioning. As a result, the person engages in behavior that reduces anxiety and therefore is reinforced through operant conditioning. This reduction of anxiety is reinforcing and increases the person's chance of engaging in that behavior again.

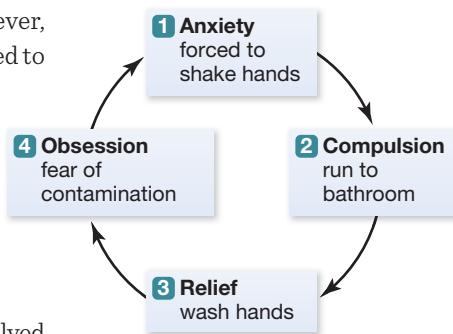
Suppose you are forced to shake hands with a man who has a bad cold. You have just seen him wiping his nose with his right hand. Shaking that hand might cause you to be anxious or uncomfortable because you do not want to get sick. As soon as the pleasantries are over, you run to the bathroom and wash your hands. You feel relieved. You have now paired handwashing with a reduction in anxiety, thus increasing the

chances of handwashing in the future (**FIGURE 14.17**). If you develop OCD, however, the compulsive behavior will reduce your anxiety only temporarily, so you will need to perform the behavior over and over.

There is also good evidence that the etiology of OCD is in part genetic (Crowe, 2000). Indeed, various behavioral genetics methods, such as twin studies, have shown that OCD runs in families. The specific mechanism has not been identified, but the OCD-related genes appear to control the neurotransmitter glutamate (Pauls, 2008). As noted in Chapter 3, glutamate is the major excitatory transmitter in the brain, causing increased neural firing.

Brain imaging has provided some evidence regarding which brain systems are involved in OCD. The caudate, a brain structure involved in suppressing impulses, is smaller and has structural abnormalities in people with OCD (Baxter, 2000). Moreover, brain imaging studies show abnormal activity in the caudates of people with OCD compared with the caudates of controls (Maia, Cooney, & Peterson, 2008). Because the caudate is involved in impulse suppression, dysfunction in this region may result in the leak of impulses into consciousness. The prefrontal cortex, which is involved in conscious control of behavior, then becomes overactive in an effort to compensate (Whiteside, Port, & Abramowitz, 2004; Yucel et al., 2007). As discussed in Chapter 15, deep brain electrical stimulation of the caudate has been successful in alleviating the symptoms of OCD, providing additional evidence that this brain structure is involved in OCD (Aouizerate et al., 2004).

There is also growing evidence that OCD can be triggered by environmental factors. In particular, a streptococcal infection apparently can cause a severe form of OCD in some young children. Originally identified in 1998 by Susan Swedo and her colleagues at the National Institute of Mental Health, this syndrome strikes virtually overnight. The affected children suddenly display odd symptoms of OCD, such as engaging in repetitive behaviors, developing irrational fears and obsessions, and having facial tics. Researchers have speculated that an autoimmune response damages the caudate, thereby producing the symptoms of OCD (Snider & Swedo, 2004). Treatments that enhance the immune system have been found to diminish the symptoms of OCD in children with this syndrome. Why some children are susceptible to this autoimmune response is unknown.



**FIGURE 14.17**

### OCD Cycle

This flowchart illustrates the operations of conditioning for the example given in the text. Classical conditioning (step 1) and operant conditioning (steps 2 and 3) reinforce behavior. Continued reinforcement may contribute to a person's developing OCD (step 4).

## Posttraumatic Stress Disorder Results from Trauma

*DSM-5* categorizes a number of disorders together that result from trauma or excessive stress. This category describes *trauma- and stressor-related disorders* (see Table 14.1). For example, a person who cries continually, has difficulty studying, and avoids social settings six months after a romantic breakup may have an *adjustment disorder*. This person is having difficulty adjusting to the stressor. When people experience severe stress or emotional trauma—such as having a serious accident, being raped, fighting in active combat, or surviving a natural disaster—they often have negative reactions long after the danger has passed. In severe cases, people develop **posttraumatic stress disorder (PTSD)**, a psychological disorder that involves frequent and recurring unwanted thoughts related to the trauma, including nightmares, intrusive thoughts, and flashbacks. People with PTSD often try to avoid situations or stimuli that remind them of their trauma. The lifetime prevalence of PTSD is around 7 percent, with women being more likely to develop the disorder (Kessler et al., 2005b).

An opportunity to study susceptibility to PTSD came about because of a tragedy at Northern Illinois University in 2008. On the campus, in front of many observers, a lone gunman killed 5 people and wounded 21. Among a sample of female students, those with certain genetic markers related to serotonin functioning were much more likely to show PTSD symptoms in the weeks after the shooting (Mercer et al., 2011).

### posttraumatic stress disorder (PTSD)

A disorder that involves frequent nightmares, intrusive thoughts, and flashbacks related to an earlier trauma.

### **major depressive disorder**

A disorder characterized by severe negative moods or a lack of interest in normally pleasurable activities.

This finding suggests that some individuals may be more at risk than others for developing PTSD after exposure to a stressful event.

Those with PTSD often have chronic tension, anxiety, and health problems, and they may experience memory and attention problems in their daily lives. PTSD involves an unusual problem in memory: the inability to forget. PTSD is associated with an attentional bias, such that people with PTSD are hypervigilant to stimuli associated with their traumatic events. For instance, soldiers with combat-induced PTSD show increased physiological responsiveness to pictures of troops, sounds of gunfire, and even words associated with combat. Exposure to stimuli associated with past trauma leads to activation of the amygdala (Rauch, van der Kolk, Fisler, & Alpert, 1996). It is as if the severe emotional event is “overconsolidated,” burned into memory (see Chapter 7 for a discussion of consolidation of memory).

## **Depressive Disorders Consist of Sad, Empty, or Irritable Mood**

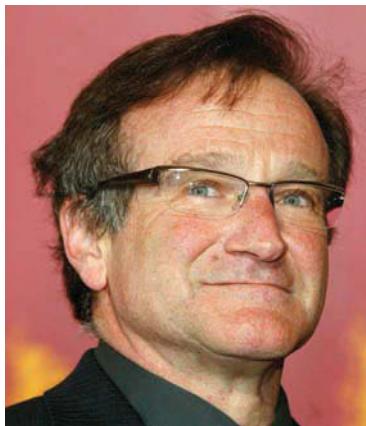
When we feel down or sad about something happening in our life, we often say we are depressed. Although this experience is relatively common, it often does not last very long. For some people, however, the negative feelings persist and turn into a psychological disorder. The *DSM-5* categorizes a number of disorders as *depressive disorders*. The common feature of all depressive disorders is the presence of sad, empty, or irritable mood along with bodily symptoms and cognitive problems that interfere with daily life.

**MAJOR DEPRESSIVE DISORDER** The classic disorder in this category is major depressive disorder. According to *DSM-5* criteria, to be diagnosed with **major depressive disorder**, a person must experience a *major depressive episode*, during which he or she experiences a depressed mood or a loss of interest in pleasurable activities every day for at least two weeks. In addition, the person must have other symptoms, such as appetite and weight changes, sleep disturbances, loss of energy, difficulty concentrating, feelings of self-reproach or guilt, and frequent thoughts of death, perhaps by suicide. The following excerpt is from a case study of a 56-year-old woman diagnosed with depression:

She described herself as overwhelmed with feelings of guilt, worthlessness, and hopelessness. She twisted her hands almost continuously and played nervously with her hair. She stated that her family would be better off without her and that she had considered taking her life by hanging herself. She felt that after death she would go to hell, where she would experience eternal torment, but that this would be a just punishment. (Andreasen, 1984, p. 39)

Feelings of depression are relatively common, but only long-lasting episodes that impair a person’s life are diagnosed as depressive disorders. Major depression affects about 6–7 percent of Americans in a given 12-month period, whereas approximately 16 percent of Americans will experience major depression at some point in their lives (Kessler & Wang, 2008). Although major depressive disorder varies in severity, those who receive a diagnosis are highly impaired by the condition, and it tends to persist over several months, often lasting for years (Kessler, Merikangas, & Wang, 2007).

Depression is the leading risk factor for suicide, which claims approximately a million lives annually around the world and is among the top three causes of death for people between ages 15 and 35 (Insel & Charney, 2003). The suicide of the comedian and actor Robin Williams shocked many, but Williams reportedly had long battled depression and substance abuse (**FIGURE 14.18**). You will learn more about suicide in this chapter’s “Using Psychology in Your Life” feature (p. 622).



**FIGURE 14.18**

### **Robin Williams**

In 2014, the actor and comedian Robin Williams hanged himself. Although he built his career on making people laugh, Williams appears to have struggled for years with depression, along with drug and alcohol problems.

**PERSISTENT DEPRESSIVE DISORDER** Unlike major depressive disorder, **persistent depressive disorder**, sometimes called *dysthymia*, is of mild to moderate severity. Most individuals with this disorder describe their mood as “down in the dumps.” People with persistent depressive disorder have many of the same symptoms as people with major depressive disorder, but those symptoms are less intense. People diagnosed with this disorder—approximately 2–3 percent of the population—must have a depressed mood most of the day, more days than not, for at least 2 years. Periods of depressed mood last from 2 to 20 or more years, although the typical duration is about 5 to 10 years. Because the depressed mood is so long-lasting, some psychologists consider it a personality disorder rather than a mood disorder.

The distinctions between a depressive personality, persistent depressive disorder, and major depressive disorder are unclear. In keeping with a dimensional view of psychological disorders, these states may be points along a continuum rather than distinct disorders (Lewinsohn, Allen, Seeley, & Gotlib, 1999; Lewinsohn, Rodhe, Seeley, & Hops, 1991).

### THE ROLES OF CULTURE AND GENDER IN DEPRESSIVE DISORDERS

Depression is so prevalent that it is sometimes called the common cold of psychological disorders. In its most severe form, depression is the leading cause of disability in the United States and worldwide (Worley, 2006). The stigma associated with this disorder has especially dire consequences in developing countries, where people do not take advantage of the treatment options because they do not want to admit to being depressed. One way to combat the stigma of psychological disorders is to focus attention on their high incidence and to educate more people about effective treatments (discussed in Chapter 15, “Treatment of Psychological Disorders”; **FIGURE 14.19**).

Gender also plays a role in the incidence of depression. Across multiple countries and contexts, twice as many women as men suffer from depressive disorders (Kessler et al., 2003; Ustün, Ayuso-Mateos, Chatterji, Mathers, & Murray, 2004). In fact, suicide is the leading cause of death among young women in India and China (Khan, 2005), and the highest rates of depression are found in women in developing countries, with especially high rates reported for women in rural Pakistan (Mumford, Saeed, Ahmad, Latif, & Mubbashar, 1997).

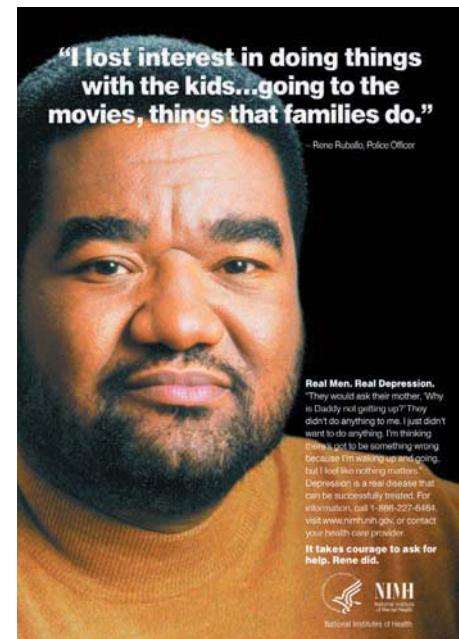
Why are the rates of depressive disorders so much higher for women than for men? Some researchers have theorized that women’s multiple roles in most societies—as wage earners and family caregivers—cause stress that results in increased incidence of depression, but other researchers have pointed out the health benefits of having multiple roles, such as wife, mother, and employee (Barnett & Hyde, 2001). Thus, it is not multiple roles per se but more likely overwork and lack of support that contribute to the high rate of depression in women. Research in India, Brazil, and Chile shows that low income, lack of education, and difficult family relationships contribute to psychological disorders in women (Blue & Harpham, 1996).

## Depressive Disorders Have Biological, Situational, and Cognitive Components

Depression can be devastating. The sadness, hopelessness, and inability to concentrate that characterize major depressive disorder can result in the loss of jobs, of friends, and of family relationships. Because of this disorder’s profound effects, particularly the danger of suicide, much research has focused on understanding the causes of depression and treating it.

### persistent depressive disorder

A form of depression that is not severe enough to be diagnosed as major depressive disorder.



**FIGURE 14.19**  
**Informing the Public**

Public-service ads may help “normalize” the treatment of psychological disorders. The more people hear about talking to doctors about problems, the more inclined they may be to visit doctors when problems arise.

**BIOLOGICAL COMPONENTS** Studies of twins, families, and adoptions support the notion that depression has a genetic component. Although there is some variability among studies, concordance rates (i.e., the percentage who share the same disorder) for identical twins are generally around two to three times higher than rates for fraternal twins (Levinson, 2006). The genetic contribution to depression is somewhat weaker than the genetic contribution to schizophrenia or to bipolar disorder (Belmaker & Agam, 2008).

The existence of a genetic component implies that biological factors are involved in depression. In fact, there is evidence that major depressive disorder may involve one or more monoamines. (As discussed in Chapter 3, monoamines are neurotransmitters that regulate emotion and arousal and motivate behavior.) For instance, medications that increase the availability of norepinephrine, a monoamine, may help alleviate depression. Medications that decrease levels of this neurotransmitter can cause symptoms of depression. Medications such as Prozac are known as *selective serotonin reuptake inhibitors (SSRIs)*. SSRIs selectively increase another monoamine, serotonin, and are often used to treat depression (Barton et al., 2008; SSRIs and other medications are discussed in Chapter 15). Yet depression is not simply due to a lack of norepinephrine or serotonin. For example, research has found that medications that reduce serotonin can also alleviate depression (Nickel et al., 2003). At this time, there is not a clear understanding of the role of neurotransmitters in the development of depressive disorders.

In addition, studies of brain function have suggested that certain neural structures may be involved in mood disorders. Damage to the left prefrontal cortex can lead to depression, but damage to the right hemisphere does not. The brain waves of people with depression show low activity in the left prefrontal cortex (Allen, Coan, & Nazarian, 2004), irrespective of the person's current mood (Stewart, Coan, Towers, & Allen, 2011). Interestingly, this pattern persists in patients who have been depressed but are currently in remission (Henriques & Davidson, 1990). The pattern may therefore be a biological marker of a predisposition to depression.

Biological rhythms have also been implicated in depression. Depressed patients enter REM sleep more quickly and have more of it. In fact, one symptom of depression is excessive sleeping and tiredness. In addition, many people show a cyclical pattern of depression, depending on the season. This condition, *seasonal affective disorder (SAD)*, results in periods of depression that correspond to the shorter days of winter in northern latitudes.

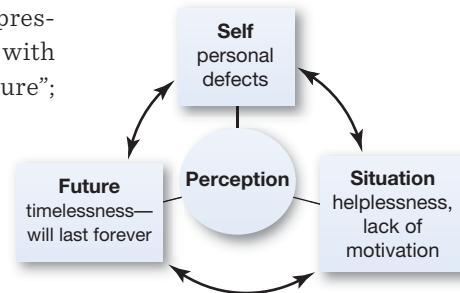
**SITUATIONAL COMPONENTS** Situational factors also play a role in depression. A number of studies have implicated life stressors in many cases of depression (Hammen, 2005). Particularly relevant for depression is interpersonal loss, such as the death of a loved one or a divorce (Paykel, 2003). Depression is especially likely in the face of multiple negative events (Brown & Harris, 1978), and patients with depression have often experienced negative life events during the year before the onset of their depression (Dohrenwend, Shrout, Link, Skodol, & Martin, 1986).

How an individual reacts to stress, however, can be influenced by interpersonal relationships, which play an extremely important role in depression (Joiner, Coyne, & Blalock, 1999). Regardless of any other factors, relationships contribute to the development of depression, alter people's experiences when depressed, and ultimately may be damaged by the constant needs of the person with depression. Many people report negative reactions to people with depression, perhaps because of their frequent complaining. Over time, people may avoid interactions with those suffering from depression, thus initiating a downward spiral by making the sufferers even more depressed (Dykman, Horowitz, Abramson, & Usher, 1991). By contrast, a person who has a close friend or group of friends is less likely to become depressed when faced with stress. This protective factor is not related to the number of friends. It is related to the quality of the friendships. One good friend is more protective than a large number of casual acquaintances.

**COGNITIVE COMPONENTS** Finally, cognitive processes play a role in depressive disorders. The psychologist Aaron Beck has hypothesized that people with depression think negatively about themselves (“I am worthless”; “I am a failure”; “I am ugly”), about their situations (“Everybody hates me”; “the world is unfair”), and about the future (“Things are hopeless”; “I can’t change”). Beck refers to these negative thoughts about self, situation, and the future as the *cognitive triad* (Beck, 1967, 1976; Beck, Brown, Seer, Eidelson, & Riskind, 1987; Beck, Rush, Shaw, & Emery, 1979; **FIGURE 14.20**).

People with depression blame misfortunes on personal defects while seeing positive occurrences as the result of luck. People who are not suffering from depression do the opposite. Beck also notes that people with depression make errors in logic. For example, they overgeneralize based on single events, magnify the seriousness of bad events, think in extremes (such as believing they should either be perfect or not try), and take responsibility for bad events that actually have little to do with them.

A second cognitive model of depression is based on **learned helplessness** (Seligman, 1974, 1975). “Learned helplessness” means that people come to see themselves as unable to have any effect on events in their lives. The psychologist Martin Seligman based this model on years of animal research. When animals are placed in aversive situations they cannot escape (such as receiving unescapable shock), the animals eventually become passive and unresponsive. They end up lacking the motivation to try new methods of escape when given the opportunity. Similarly, people suffering from learned helplessness come to expect that bad things will happen to them and believe they are powerless to avoid negative events. The *attributions*, or explanations, they make for negative events refer to personal factors that are stable and global, rather than to situational factors that are temporary and specific. This attributional pattern leads people to feel hopeless about making positive changes in their lives (Abramson, Metalsky, & Alloy, 1989). According to the scientific evidence, dysfunctional cognitive patterns are a cause rather than a consequence of depression.



**FIGURE 14.20**  
**Cognitive Triad**

According to Beck, people suffering from depression perceive themselves, their situations, and the future negatively. These perceptions influence each other and contribute to the disorder.

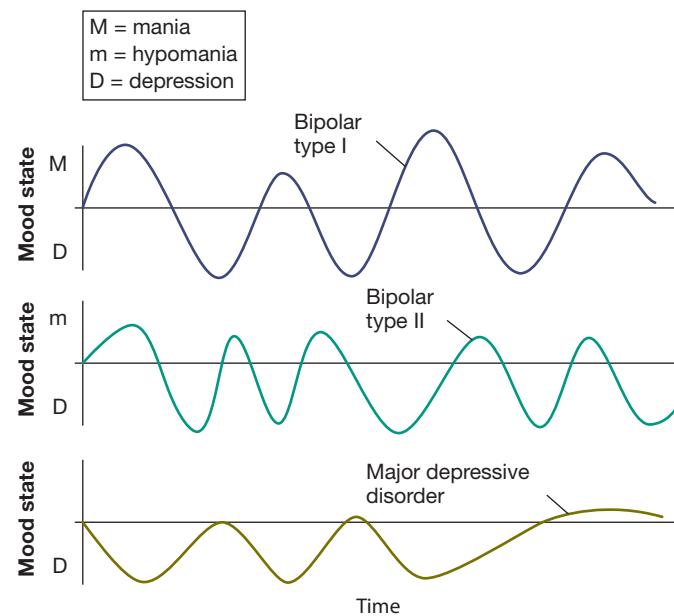
#### learned helplessness

A cognitive model of depression in which people feel unable to control events in their lives.

## Bipolar Disorders Involve Depression and Mania

We all experience variations in mood. Our normal fluctuations from sadness to exuberance seem minuscule, however, compared with the extremes experienced by people with *bipolar disorders*. Recall the discussion, at the opening of this chapter, of Kay Redfield Jamison’s extreme depression and episodes of mania. *Mania* refers to an elevated mood that feels like being “on the top of the world.” This positive mood can vary in degree and is accompanied by major shifts in energy level and physical activity (**FIGURE 14.21**).

True *manic episodes* last at least one week and are characterized by abnormally and persistently elevated mood, increased activity, diminished need for sleep, grandiose ideas, racing thoughts, and extreme distractibility. During episodes of mania, heightened levels of activity and extreme happiness often result in excessive involvement in pleasurable but foolish activities. People may engage in sexual indiscretions, buying sprees, risky business ventures, and



**FIGURE 14.21**  
**Bipolar I, Bipolar II, and Major Depressive Disorders**

These graphs compare the mood changes over time for three disorders that involve mood states.

# Using Psychology in Your Life

## I Think My Friend Might Be Suicidal. What Should I Do?

### bipolar I disorder

A disorder characterized by extremely elevated moods during manic episodes and, frequently, depressive episodes as well.

### bipolar II disorder

A disorder characterized by alternating periods of extremely depressed and mildly elevated moods.

Many people contemplate suicide at some point in their lives. Tragically, as of 2007, suicide was the third leading cause of death among Americans ages 10 to 24 (American Association of Suicidology, 2011). As a result, many college students will be or have been touched by suicide. Perhaps you know someone who died by suicide. Perhaps a friend of yours talks about wanting to die. Or maybe you have considered taking your own life. Understanding the risk factors associated with suicide is an important step toward preventing suicide. Knowing where and how to find support can save lives.

In his book *Why People Die by Suicide* (2005), the clinical psychologist Thomas Joiner considers two key questions about suicide: Who *wants* to commit suicide? And who *can* commit suicide? In answering the first question, Joiner argues that "people desire death when two fundamental needs are frustrated to the point of extinction" (p. 47). The first of these fundamental needs is the need to belong, to feel connected with others. We all want to have positive interactions with others who care about us. If we do not perceive those things in our lives, our need to belong is thwarted. The second of these fundamental needs is the need for competence. We all want to be capable agents in the world. If we do not perceive ourselves as able to do the things we think we should be able to do, our need

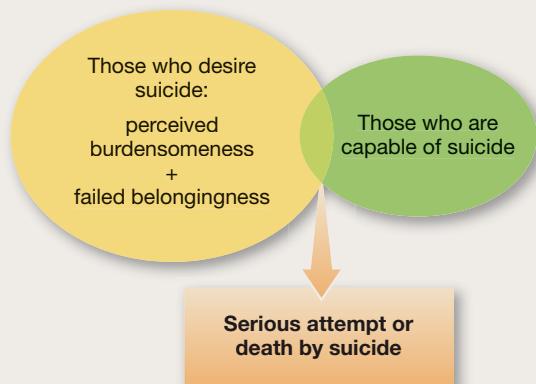
for competence is thwarted. Joiner says that when the need to belong and the need for competence are frustrated, we desire death.

But as Joiner points out, just because a person wants to commit suicide does not mean she or he will be able to do so. Evolution has hardwired us with a tremendously strong self-preservation instinct. What makes a person able to endure the tremendous physical pain or overwhelming psychological fear many of us would experience if we tried to kill ourselves? Joiner presents a straightforward answer: practice. He writes that "those prone to serious suicidal behavior have reached that status through a process of exposure to self-injury and other provocative experiences" (pp. 85–86) and "when people get used to dangerous behavior . . . the groundwork for catastrophe is laid" (p. 48). For example, a person who drives recklessly, engages in self-cutting, and/or experiments with drugs is more practiced at self-harm than someone who does not engage in any of these behaviors. Thus, the person who engages in dangerous behavior is more likely to have the capacity to carry out lethal self-injury.

Take a look at **FIGURE 14.22**. The larger oval represents the people in the world who desire suicide. These individuals perceive themselves to be burdens on others and do not perceive themselves as having frequent and positive interactions with others who care about them.

similar "out of character" behaviors that they regret once the mania has subsided. They might also have severe thought disturbances and hallucinations. This form of the condition is known as **bipolar I disorder**. Bipolar I disorder is based more on the manic episodes than on depression. Although those with bipolar I disorder often have depressive episodes, these episodes are not necessary for a *DSM-5* diagnosis. The manic episodes in bipolar I disorder cause significant impairment in daily living and can often result in hospitalization.

Whereas people with bipolar I disorder experience true manic episodes, those with **bipolar II disorder** may experience less extreme mood elevations called *hypomania* (Phillips & Kupfer, 2013). These episodes are often characterized by heightened creativity and productivity, and they can be extremely pleasurable and rewarding. As mentioned in the chapter opener, the singer Demi Lovato and the actor Catherine Zeta-Jones have both revealed that they have been diagnosed with



**FIGURE 14.22**

### The Risk of Suicide: Desire + Capability = Attempt?

According to Joiner, the individuals who are *most* at risk of dying by suicide both want to do so and are able to do so.

In other words, these are the people who may want to commit suicide. The smaller oval represents the people who, over time, have developed the ability to lethally injure themselves. The overlap between the ovals represents a small fraction of the people who want to commit suicide and are able to do so. It also represents, conversely, the small fraction of the people who are well practiced at endangering themselves and want to die. Again, Joiner posits that the individuals who are *most* at risk of dying by suicide both want to do so and are able to do so.

Of course, like so many other topics you have learned about in this book,

suicide is a very complex psychological phenomenon. Perhaps you have heard that suicide tends to run in families or that everyone who commits suicide has a psychological disorder. Indeed, the data support a genetic risk factor for suicide (Roy, 1992), and the majority of people who commit suicide seem to suffer from psychological disorders (Cavanagh, Carson, Sharpe, & Lawrie, 2003). How do these factors figure into Joiner's model? He points out: "Genes, neurobiology, impulsivity, childhood adversity, and mental disorders are interconnected strands that converge and influence whether people acquire the ability for lethal self-injury, feel a burden on others, and fail to feel they belong" (Joiner, 2005, p. 202).

In other words, many factors might lead someone to want to commit suicide. In addition, many factors might prompt someone to arm himself or herself with the ability to endure self-harm.

With such risk factors in mind, we can now turn to the important question of what to do if you think a friend might be suicidal. First and foremost, take suicidal threats seriously. Second, get help. Someone who is considering suicide should be screened by a trained professional. Contact a counselor at

your school, ask a religious leader for help, or speak to someone at the National Suicide Prevention Lifeline: 1-800-273-TALK (8255). These individuals can help you get your friend the support he or she needs. Third, let your friend know you care.

Remember, suicide risk is particularly high when people do not feel a sense of connection with others and when they feel a lack of competence. You can remind the suicidal person that you value your relationship, that you care about her or his well-being, that you would be devastated if that person were no longer in your life. These forms of support can challenge the suicidal person's sense that she or he lacks belongingness. To challenge the person's perceived incompetence, you can remind your friend about the reasons you admire her or him, or you can ask for help on a project or issue you are genuinely struggling with.

And remember, suicide is forever. The problems that prompt a person to feel suicidal, however, are often temporary. If you ever find yourself or a friend feeling that suicide offers the best way out of an overwhelming or hopeless situation, know that other options exist. You or your friend might not be able to see those options right away. Reach out to someone who can help you or your friend see the ways out of current problems and into the future.

bipolar II disorder. Although these less extreme positive moods may be somewhat disruptive to a person's life, they do not cause significant impairment in daily living or require hospitalization. However, the bipolar II diagnosis does require at least one major depressive episode, and therefore the depression might cause significant impairments. Thus, the impairments to daily living for bipolar I disorder are the manic episodes, but the impairments for bipolar II disorder are the major depressive episodes.

Bipolar disorders are much less common than depression. The lifetime prevalence for any type is estimated at around 3–4 percent (Kessler & Wang, 2008). In addition, whereas depression is more common in women, bipolar disorders are equally prevalent in women and men. Bipolar disorders emerge most commonly during late adolescence or early adulthood, with bipolar I disorder typically first diagnosed at a younger age than bipolar II disorder.

**CAUSE OF BIPOLAR DISORDERS** A family history of a bipolar disorder is the strongest and most consistent risk factor for bipolar disorders (Craddock & Sklar, 2013). The concordance rate for bipolar disorders in identical twins is more than 70 percent, versus only 20 percent for fraternal, or dizygotic, twins (Nurnberger, Goldin, & Gershon, 1994).

In the 1980s, the Amish community was involved in a genetic research study. The Amish were an ideal population for this sort of research because they keep good genealogical records and few outsiders marry into the community. In addition, substance abuse is virtually nonexistent among Amish adults, so psychological disorders are easier to detect. The research results revealed that bipolar disorders ran in a limited number of families and that all of those afflicted had a similar genetic defect (Egeland et al., 1987).

Genetic research suggests, however, that the hereditary nature of bipolar disorders is complex and not linked to just one gene. Current research focuses on identifying several genes that may be involved (Wray, Byrne, Stinger, & Mowry, 2014). In addition, it appears that in families with bipolar disorders, successive generations have more-severe disorders and earlier ages of onset (Petronis & Kennedy, 1995; Post et al., 2013). Research on this pattern of transmission may help reveal the genetics of the disorder, but the specific nature of the heritability of bipolar disorders remains to be discovered.

## Summing Up

### Which Disorders Emphasize Emotions or Moods?

- Anxiety disorders are characterized by excessive fear and anxiety in the absence of true danger.
- Common anxiety disorders include specific phobia, social anxiety disorder, generalized anxiety disorder, and panic disorder.
- Obsessive-compulsive disorder (OCD) involves frequent intrusive thoughts and compulsive actions. OCD may involve learned behaviors or may be caused by biological factors.
- Posttraumatic stress disorder (PTSD) is a trauma- or stressor-related disorder that appears after exposure to a traumatic event. PTSD affects women more than men.
- PTSD is characterized by frequent and recurring unwanted thoughts related to the trauma, nightmares, intrusive thoughts, flashbacks, and avoidance of situations related to the event.
- Major depressive disorder is characterized by sad, empty, or irritable mood or a loss of interest in pleasurable activities, among other symptoms. Persistent depressive disorder is less disruptive but leaves a person feeling sad on more days than not for at least two years.
- Depressive disorders have biological components, including dysfunction of the monoamine neurotransmitters norepinephrine and serotonin, low left frontal lobe function, and disrupted biological rhythms.
- Situational factors (such as poor relationships and stress) and cognitive factors (such as the cognitive triad and learned helplessness) also contribute to the occurrence of depression.
- Bipolar disorders include episodes of both mania and depression. The impairments to daily living for bipolar I disorder are the manic episodes, whereas the impairments for bipolar II disorder are the major depressive episodes.
- The best predictor of bipolar disorder is a family history of the disorder, suggesting that genetic factors are an important cause.

## Measuring Up

1. Indicate whether each of the following empirical findings supports a cognitive, situational, or biological underpinning of anxiety disorders.
  - a. Adults who were categorized as inhibited versus uninhibited during childhood show differential patterns of amygdala activation when viewing novel faces (Schwartz et al., 2003).
  - b. Anxious individuals tend to focus excessive attention on perceived threats (Rinck et al., 2005).
  - c. Monkeys tend to develop a fear of snakes if they observe other monkeys responding to snakes fearfully (Mineka, Davidson, Cook, & Keir, 1984).
  - d. When presented with ambiguous or neutral situations, anxious individuals generally perceive them as threatening, whereas nonanxious individuals perceive them as nonthreatening (Eysenck et al., 1991).
2. Which of the following statements represent dysfunctional cognitive patterns believed to cause depression? If a statement is an example of dysfunctional cognition, briefly describe why.
  - a. "I didn't make the soccer team. I fail at everything."
  - b. "I didn't get a raise because I was late to work a number of times over the past quarter."
  - c. "There's nothing I can do about the fact that my boss is so mean to me."
  - d. "This assignment is really hard. I need to see my instructor during office hours."

to avoid negative events.)

(2) a. Shows evidence of overgeneralizing based on a single event; c. shows a perceived powerlessness

ANSWERS: (1) a. biological; b. situational; c. cognitive; d. cognitive.

## 14.3 Which Disorders Emphasize Thought Disturbances?

As we have seen, many psychological disorders include emotional impairments that influence how people think. For example, those with depression can have distorted thoughts about themselves or their futures. By contrast, disorders that revolve around thinking involve disruptions in the connection between thoughts and experiences, such as people losing their sense of identity or feeling that external forces are controlling their thoughts. Many disorders of thought involve *psychosis*, which is a break from reality in which the person has difficulty distinguishing what thoughts or perceptions are real versus what are imagined. People experiencing this disorder have extreme difficulty functioning in everyday life.

### Dissociative Disorders Are Disruptions in Memory, Awareness, and Identity

As noted in Chapter 5, we sometimes get lost in our thoughts or daydreams, even to the point of losing track of what is going on around us. Many of us have had the experience of forgetting what we are doing while in the middle of an action ("Why was I headed to the kitchen?"). When we wake up in an unfamiliar location, we may momentarily be disoriented and not know where we are. In other words, our thoughts and experiences can become dissociated, or split, from the external world.

**Dissociative disorders** are extreme versions of this phenomenon. These disorders involve disruptions of identity, of memory, or of conscious awareness (Kihlstrom,

### Learning Objectives

- Describe dissociative amnesia, dissociative fugue, and dissociative identity disorder.
- Discuss the controversy regarding dissociative identity disorder.
- Describe the five symptoms of schizophrenia.
- Distinguish between negative and positive symptoms of schizophrenia.
- Identify biological and environmental factors that contribute to schizophrenia.

#### dissociative disorders

Disorders that involve disruptions of identity, of memory, or of conscious awareness.



**FIGURE 14.23**

### The Two Forms of Amnesia

Jeff Ingram, pictured here, developed retrograde amnesia after leaving his home, in Washington state. When he arrived in Denver, Colorado, four days later, he had no memory of his previous life. He was recognized two months later, when he appeared on the news pleading for help from anyone who knew who he was. Though he did not remember his three-year relationship with his fiancée (here seated next to him), the two eventually married.

2005). The commonality among dissociative disorders is the splitting off of some parts of memory from conscious awareness. Dissociative disorders are believed to result from extreme stress. That is, the person with a dissociative disorder has split off a traumatic event in order to protect the self. Some researchers believe that people prone to dissociative disorders are also prone to PTSD (Cardeña & Carlson, 2011).

**DISSOCIATIVE AMNESIA** In *dissociative amnesia*, a person forgets that an event happened or loses awareness of a substantial block of time. For example, the person with this disorder may suddenly lose memory for personal facts, including his or her identity and place of residence. These memory failures cannot be accounted for by ordinary forgetting (such as momentarily forgetting where you parked your car) or by the effects of drugs or alcohol.

Consider the case of Dorothy Joudrie, from Calgary, Canada. In 1995, after suffering years of physical abuse from her husband, Joudrie shot her husband six times. Her husband survived, and he described her behavior during the shooting as very calm, as if she were detached from what she was doing. When the police arrived, however, Joudrie was extremely distraught. She had no memory of the shooting and told the police that she simply found her husband shot and lying on the garage floor, at which time she called for help. Joudrie was found not criminally responsible for her actions because of her dissociative state (Butcher, Mineka, & Hooley, 2007).

**DISSOCIATIVE FUGUE** The rarest and most extreme form of dissociative amnesia is *dissociative fugue*. The disorder involves a loss of identity. In addition, it involves travel to another location (the French word *fugue* means “flight”) and sometimes the assumption of a new identity. The fugue state often ends suddenly, with the person unsure how she or he ended up in unfamiliar surroundings. Typically, the person does not remember events that occurred during the fugue state.

Consider the case of Jeff Ingram, who developed retrograde amnesia, a form of dissociative amnesia (**FIGURE 14.23**). After Ingram found himself in Denver not knowing who he was, his fiancée brought him home to Washington state. Ingram did not recognize his fiancée’s face, but she felt familiar to him, as did his home.

**DISSOCIATIVE IDENTITY DISORDER** According to *DSM-5*, **dissociative identity disorder (DID)** consists of the occurrence of two or more distinct identities in the same individual, along with memory gaps in which the person does not recall everyday events. It used to be known as *multiple personality disorder*. Consider the strange case of Billy Milligan, who in 1978 was found innocent of robbery and rape charges on the grounds that he had dissociative identity disorder. Milligan clearly committed the robberies and rapes, but his lawyers successfully argued that he had multiple personalities and that different ones committed the crimes. Therefore, Billy could not be held responsible.

In his book *The Minds of Billy Milligan* (1981), Daniel Keyes describes the 24 separate personalities sharing the body of 26-year-old Billy Milligan. One is Arthur, who at age 22 speaks with a British accent and is self-taught in physics and biology. He reads and writes fluent Arabic. Eight-year-old David is the keeper of the pain. Anytime something physically painful happens, David experiences it. Christene is a 3-year-old dyslexic girl who likes to draw flowers and butterflies. Regan is 23 and Yugoslavian, speaks with a marked Slavic accent, and reads, writes, and speaks Serbo-Croatian. He is the protector of the “family” and acknowledges robbing his victims, but he denies raping them. Adalana, a 19-year-old lesbian who writes poetry, cooks, and keeps house for the others, later admitted to committing the rapes.

After his acquittal, Milligan spent close to a decade in various mental hospitals. In 1988, psychiatrists declared that Milligan’s 24 personalities had merged into one and that he was no longer a danger to society. Milligan was released and reportedly has lived

### dissociative identity disorder (DID)

The occurrence of two or more distinct identities in the same individual.

quietly since then. Many people respond to reports such as this with astonishment and incredulity, believing that people such as Milligan must be faking. To judge the facts, we need to examine what is known about this condition and how it is diagnosed.

Most people diagnosed with DID are women who report being severely abused as children. According to the most common theory of DID, children cope with abuse by pretending it is happening to someone else. They enter a trancelike state in which they dissociate their mental states from their physical bodies. Over time, this dissociated state takes on its own identity. Different identities develop to deal with different traumas. Often the identities have periods of amnesia, and sometimes only one identity is aware of the others. Indeed, diagnosis often occurs only when a person has difficulty accounting for large chunks of his or her day. The separate identities usually differ substantially, such as in gender identity, sexual orientation, age, language spoken, interests, physiological profiles, and patterns of brain activation (Reinders et al., 2003). Even their handwritings can differ (**FIGURE 14.24**).

#### Participant 1

Sister opens that hot door for the people  
that wronged me!!!

I was sitting in a jail cell! Sitting in a jail  
cell for a crime someone else has done. I guess that

Happened. Just as the name that I really  
belong to people I know...  
By M.L.

By Johnny7

Freelind

#### Participant 2

Keith (51-year-old in New Jersey—see *Atlanta Journal*  
of 11-10) in prison for bank robbery during first 4:  
killed 61 yr. old Anna Mae Chicago on Dec. 19, 1979; in prison from  
Aug. 24-Dec. 1, 1979; What about rest??

I also have the letter  
from the YMCA in Rochester,  
and the Bureau of Vital Statistics

Place him in a cell in the back side of prison having him  
with him is being submitted April 17/11/16, the court is in it.  
The court was denied in the hearing, his right under the 6th

#### FIGURE 14.24

#### Handwriting Samples of Three People Diagnosed with Dissociative Identity Disorder

When researchers studied 12 murderers diagnosed with DID, writing samples from 10 of the participants revealed markedly different handwriting in each of their identities. Here handwriting samples from three of the participants show different identities expressing themselves.

#### Participant 3

Vincent

Jeff

Sophie

### **schizophrenia**

A psychological disorder characterized by a split between thought and emotion; it involves alterations in thoughts, perceptions, or consciousness.

Despite this evidence, many researchers remain skeptical about whether DID is a genuine psychological disorder or whether it exists at all (Kihlstrom, 2005). Moreover, some people may have ulterior motives for claiming DID. A diagnosis of DID often occurs after someone has been accused of committing a crime. This timing raises the possibility that people are pretending to have multiple identities to avoid conviction. Other skeptics point to the sharp rise in reported cases as evidence that the disorder might not be real or that it is diagnosed far too often. The 1980s and 1990s saw a surge of therapists who believed that childhood trauma frequently was repressed and that it needed to be uncovered during treatment. These therapists tended to use hypnosis, and they might have suggested DID symptoms to the patients they were assessing while the patients were hypnotized.

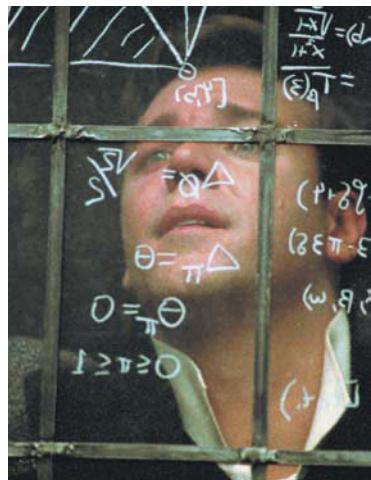
Ultimately, how can we know whether a diagnosis of DID is valid? As mentioned earlier, most often there is no objective, definitive test for diagnosing a psychological disorder. It can be difficult to tell if a person is faking, has come to believe what a therapist said, or has a genuine psychological disorder. Individuals who fake DID tend to report well-publicized symptoms of the disorder but neglect to mention the more subtle symptoms that are extremely common, such as major depressive episodes or PTSD (American Psychiatric Association, 2013). Those faking it seem indifferent or even proud of the disorder. Those truly afflicted are ashamed of or overwhelmed by their symptoms.

## **Schizophrenia Involves a Split Between Thought and Emotion**

The term *schizophrenia* literally means “splitting of the mind.” The psychological disorder **schizophrenia** is characterized by a split between thought and emotion (**FIGURE 14.25**). In popular culture, schizophrenia is often confused with dissociative identity disorder, or split personality, but the two disorders are unrelated. With DID, the “self” is split. Schizophrenia involves alterations in thought, perceptions, or consciousness. The essence of schizophrenia is a disconnection from reality, or psychosis.

According to current estimates, between 0.5 percent and 1.0 percent of the population has schizophrenia (Tandon, Keshavan, & Nasrallah, 2008). A meta-analysis of 188 studies from 46 countries found similar rates for men and women, roughly 4 to 7 per 1,000 people (Saha, Chant, Welham, & McGrath, 2006). These researchers also found that the rate of schizophrenia was slightly lower in developing nations. Interestingly, the prognosis is better in developing than in developed cultures (Kulhara & Chakrabarti, 2001). Perhaps there is more tolerance for symptoms or greater sympathy for unusual or different people in developing countries (Waxler, 1979). It is also possible that methods of defining and assessing recovery vary across countries, thereby exaggerating recovery in developing nations (Jääskeläinen et al., 2013).

Schizophrenia is arguably the most devastating disorder for the people who have it and the relatives and friends who support them. It is characterized by a combination of motor, cognitive, behavioral, and perceptual abnormalities. These abnormalities result in impaired social, personal, or vocational functioning. According to *DSM-5*, to be diagnosed with schizophrenia a person has to have shown continuous signs of disturbances for at least six months. There are five major *DSM-5* symptoms for schizophrenia, and a diagnosis requires a person to show two or more of the symptoms. At least one of those symptoms has to be among the first three listed in criterion A of **TABLE 14.4** (i.e., delusions, hallucinations, and disorganized speech). By tradition, researchers tend to group symptoms into two categories: positive and negative.



**FIGURE 14.25**  
**Schizophrenia**

In the 2001 film *A Beautiful Mind*, Russell Crowe plays the real-life Princeton mathematics professor and Nobel laureate John Forbes Nash, who has suffered from schizophrenia.

**Table 14.4 DSM-5 Diagnostic Criteria for Schizophrenia**

- A. Two (or more) of the following, present for a significant portion of time during a 1-month period. At least one of these must be (1), (2), or (3).
1. Delusions
  2. Hallucinations
  3. Disorganized speech (e.g., frequent incoherence)
  4. Grossly disorganized or catatonic behavior
  5. Negative symptoms (i.e., diminished emotional response or lack of motivation)
- B. For a significant portion of time since the onset of the disturbance, level of functioning in one or more major areas, such as work, interpersonal relations, or self-care, is markedly below the level achieved prior to the onset.
- C. Continuous signs of the disturbance persist for at least 6 months. This 6-month period must include at least 1 month of symptoms that meet criteria A (i.e., active phase symptoms) and may include periods where the symptoms are less extreme.
- D. Other disorders and conditions have been ruled out (e.g., bipolar disorder, reactions to drugs, or other medical condition).

SOURCE: Based on American Psychiatric Association (2013).

*Positive symptoms* are excesses. They are not positive in the sense of being good or desirable, but in the sense of adding abnormal behaviors. The first four *DSM-5* criteria in Table 14.4 are considered positive symptoms.

As you will see, *negative symptoms* are deficits in functioning, such as apathy, lack of emotion, and slowed speech and movement.

**DELUSIONS** One of the positive (i.e., excessive) symptoms most commonly associated with schizophrenia is **delusions**. Delusions are false beliefs based on incorrect inferences about reality. (Common types of delusions are listed in **TABLE 14.5**.) Delusional people persist in their beliefs despite evidence that contradicts those beliefs.

Delusions are characteristic of schizophrenia regardless of the culture, but the type of delusion can be influenced by cultural factors (Tateyama et al., 1993). When the delusions of German and Japanese patients with schizophrenia were compared, the two groups had similar rates of grandiose delusions, believing themselves much more powerful and important than they really were. The two groups differed significantly, however, for other types of delusions. The German patients had delusions that involved guilt and sin, particularly as these concepts related to religion. By contrast, the Japanese patients had delusions of harassment, such as the belief that they were being slandered by others. The types of delusions that people with schizophrenia have can also be affected by current events:

**delusions**

False beliefs based on incorrect inferences about reality.

**Table 14.5 Delusions and Associated Beliefs**

<b>Persecutory</b>	Belief that others are persecuting, spying on, or trying to harm one
<b>Referential</b>	Belief that objects, events, or other people have particular significance to one
<b>Grandiose</b>	Belief that one has great power, knowledge, or talent
<b>Identity</b>	Belief that one is someone else, such as Jesus Christ or the president of the United States
<b>Guilt</b>	Belief that one has committed a terrible sin
<b>Control</b>	Belief that one's thoughts and behaviors are being controlled by external forces

In summer, 1994, mass media in the U.S. reported that North Korea was developing nuclear weapons. At that time, in New York, a middle-age woman with schizophrenia told me that she feared a Korean invasion. In fall, 1995, during a psychiatric interview a young woman with psychotic disorder told me that she had secret connections with the United Nations, the Pope, and O. J. Simpson, and they were helping her. The celebration of the 50th Anniversary of the United Nations, the visit of the Pope to the U.S., and the O. J. Simpson criminal trial were the highly publicized events in the United States at that time. (Sher, 2000, p. 507)

**HALLUCINATIONS** Another positive symptom commonly associated with schizophrenia is **hallucinations**. Hallucinations are false sensory perceptions that are experienced without an external source. They are vivid and clear, and they seem real to the person experiencing them. Frequently auditory, they can also be visual, olfactory, or somatosensory:

I was afraid to go outside and when I looked out of the window, it seemed that everyone outside was yelling, “Kill her, kill her.” . . . Things continued to get worse. I imagined that I had a foul body odor and I sometimes took up to six showers a day. I recall going to the grocery store one day, and I imagined that the people in the store were saying “Get saved, Jesus is the answer.” (O’Neal, 1984, pp. 109–110)

Auditory hallucinations are often accusatory voices. These voices may tell the person with schizophrenia that he or she is evil or inept, or they may command the person to do dangerous things. Sometimes the person hears a cacophony of sounds with voices intermingled.

The cause of hallucinations remains unclear. Neuroimaging studies suggest, however, that hallucinations are associated with activation in areas of the cortex that process external sensory stimuli. For example, auditory hallucinations accompany increased activation in brain areas that are normally activated when people engage in inner speech (Stein & Richardson, 1999). This finding has led to speculation that auditory hallucinations might be caused by a difficulty in distinguishing normal inner speech (i.e., the type we all engage in) from external sounds. People with schizophrenia need to learn to ignore the voices in their heads, but doing so is extremely difficult and sometimes impossible.

**DISORGANIZED SPEECH** Another key positive symptom of schizophrenia is **disorganized speech**. It is disorganized in the sense that it is incoherent, failing to follow a normal conversational structure. A person with schizophrenia may respond to questions with tangential or irrelevant information. It is very difficult to follow what those with schizophrenia are talking about because they frequently change topics, which is known as a *loosening of associations*. These shifts make it difficult or impossible for a listener to follow the speaker’s train of thought:

They’re destroying too many cattle and oil just to make soap. If we need soap when you can jump into a pool of water, and then when you go to buy your gasoline, my folks always thought they could get pop, but the best thing to get is motor oil, and money. May as well go there and trade in some pop caps and, uh, tires, and tractors to car garages, so they can pull cars away from wrecks, is what I believed in. (Andreasen, 1984, p. 115)

In more extreme cases, speech is so disorganized that it is totally incomprehensible, which is described by clinicians as *word salad*. This jumbling can also involve *clang associations*: the stringing together of words that rhyme but have no other

#### hallucinations

False sensory perceptions that are experienced without an external source.

#### disorganized speech

Speaking in an incoherent fashion that involves frequently changing topics and saying strange or inappropriate things.

apparent link. Those with schizophrenia might also display strange and inappropriate emotions while talking. Such strange speaking patterns make it very difficult for people with schizophrenia to communicate (Docherty, 2005).

**DISORGANIZED BEHAVIOR** Another common symptom of schizophrenia is **disorganized behavior**. People with schizophrenia often act strangely, such as displaying unpredictable agitation or childish silliness. People exhibiting this symptom might wear multiple layers of clothing even on hot summer days, walk along muttering to themselves, alternate between anger and laughter, or pace and wring their hands as if extremely worried. They also have poor hygiene, failing to bathe or change clothes regularly. They have problems performing many activities, which interferes with daily living.

Sometimes those with schizophrenia may display *catatonic behavior*, where they show a decrease in responsiveness to the environment. For example, they might remain immobilized in one position for hours. Some have speculated that catatonic behavior may be an extreme fear response, akin to how animals respond to sudden dangers—the person is literally “scared stiff” (Moskowitz, 2004). Catatonic features can also include a rigid, masklike facial expression with eyes staring into the distance. In addition, people exhibiting catatonic behavior might mindlessly repeat words they hear, which is called *echolalia*.

**NEGATIVE SYMPTOMS** A number of behavioral deficits, called **negative symptoms**, associated with schizophrenia result in patients’ becoming isolated and withdrawn. People with schizophrenia often avoid eye contact and seem apathetic. They do not express emotion even when discussing emotional subjects. Their speech is slowed, they say less than normal, and they use a monotonous tone of voice. Their speech may be characterized by long pauses before answering, failure to respond to a question, or inability to complete an utterance after initiating it. There is often a similar reduction in overt behavior: Patients’ movements may be slowed and their overall amount of movement reduced, with little initiation of behavior and no interest in social participation. These symptoms, though less dramatic than delusions and hallucinations, can be equally serious. Negative symptoms are more common in men than in women (Raesaenen, Pakaslahti, Syvaelahti, Jones, & Isohanni, 2000). They are associated with a poorer prognosis.

Although the positive symptoms of schizophrenia (i.e., delusions, hallucinations, and disorganized speech and behavior) can be dramatically reduced or eliminated with antipsychotic medications, the negative symptoms often persist. Because negative symptoms are more resistant to medications, researchers have speculated that positive and negative symptoms have different organic causes. Since positive symptoms respond to a class of medications (known as *antipsychotics*) that act on neurotransmitter systems, these symptoms are thought to result from neurotransmitter dysfunction. In contrast, negative symptoms may be associated with abnormal brain anatomy, since structural brain deficits are not affected by changes in neurochemistry. The apparent differences in biological causality lead some researchers to believe that schizophrenia with negative symptoms is in fact a separate disorder from schizophrenia with positive symptoms (Messias et al., 2004).

**BIOLOGICAL CAUSES** The etiology of schizophrenia is complex and not well understood. Early theories attributed this disorder to the patients’ mothers. According to these theories, the mothers simultaneously accepted and rejected their children. This contradictory behavior caused the children to develop schizophrenia. There is no evidence

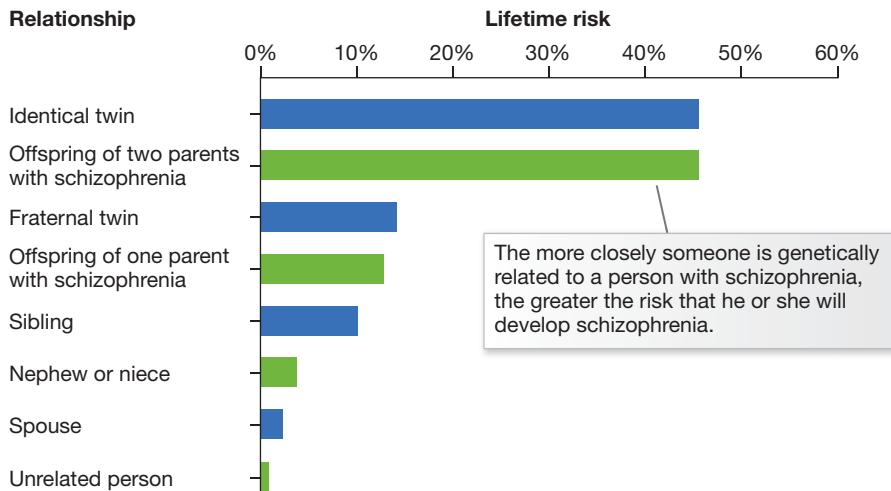
#### **disorganized behavior**

Acting in strange or unusual ways, including strange movement of limbs, bizarre speech, and inappropriate self-care, such as failing to dress properly or bathe.

#### **negative symptoms**

Symptoms of schizophrenia that are marked by deficits in functioning, such as apathy, lack of emotion, and slowed speech and movement.

**FIGURE 14.26**  
**Genetics and Schizophrenia**



to support this belief. Schizophrenia runs in families, however, and it is clear that genetics plays a role in the development of the disorder (**FIGURE 14.26**). If one twin develops schizophrenia, the likelihood of the other twin's developing it is almost 50 percent if the twins are identical but only 14 percent if the twins are fraternal. If one parent has schizophrenia, the risk of a child's developing the disease is 13 percent. If, however, both parents have schizophrenia, the risk jumps to almost 50 percent (Gottesman, 1991).

People with schizophrenia have rare mutations of their DNA about three to four times more often than healthy individuals do, especially in genes related to brain development and to neurological function (Fromer et al., 2014; Walsh et al., 2008). These mutations may result in abnormal brain development, which might lead to schizophrenia. No single gene causes schizophrenia. Instead, it is likely that multiple genes or gene mutations contribute in subtle ways to the expression of the disorder (Purcell et al., 2014). More than 100 candidate genes might modestly influence the development of schizophrenia (Schizophrenia Working Group of the Psychiatric Genomics Consortium, 2014).

Schizophrenia is primarily a brain disorder (Walker, Kestler, Bollini, & Hochman, 2004). As seen in imaging that shows the structure of the brain, the ventricles are enlarged in people with schizophrenia (see Figure 14.8). In other words, actual brain tissue is reduced. Moreover, greater reductions in brain tissue are associated with more negative outcomes (Mitelman, Shihabuddin, Brickman, Hazlett, & Buchsbaum, 2005), and longitudinal studies show continued reductions over time (Ho et al., 2003; van Haren et al., 2011). This reduction of tissue occurs in many regions of the brain, especially the frontal lobes and medial temporal lobes. In addition, as seen in imaging that shows the functioning of the brain, activity is typically reduced in the frontal and temporal regions in people with schizophrenia (Barch, Sheline, Csernansky, & Snyder, 2003). Given that abnormalities occur throughout many brain regions in people with schizophrenia, some researchers have speculated that schizophrenia is more likely a problem of connection between brain regions than the result of diminished or changed functions of any particular brain region (Walker et al., 2004).

One possibility is that schizophrenia results from abnormality in neurotransmitters. Since the 1950s, scientists have believed that dopamine may play an important role. Drugs that block dopamine activity decrease symptoms, whereas drugs that increase the activity of dopamine neurons increase symptoms. There is now also evidence that a number of other neurotransmitter systems are involved. More recently, researchers have suggested that schizophrenia might involve abnormalities in the glial cells that make up the myelin sheath (Davis et al., 2003; Moises & Gottesman, 2004). Such abnormalities would impair neurotransmission throughout the brain.

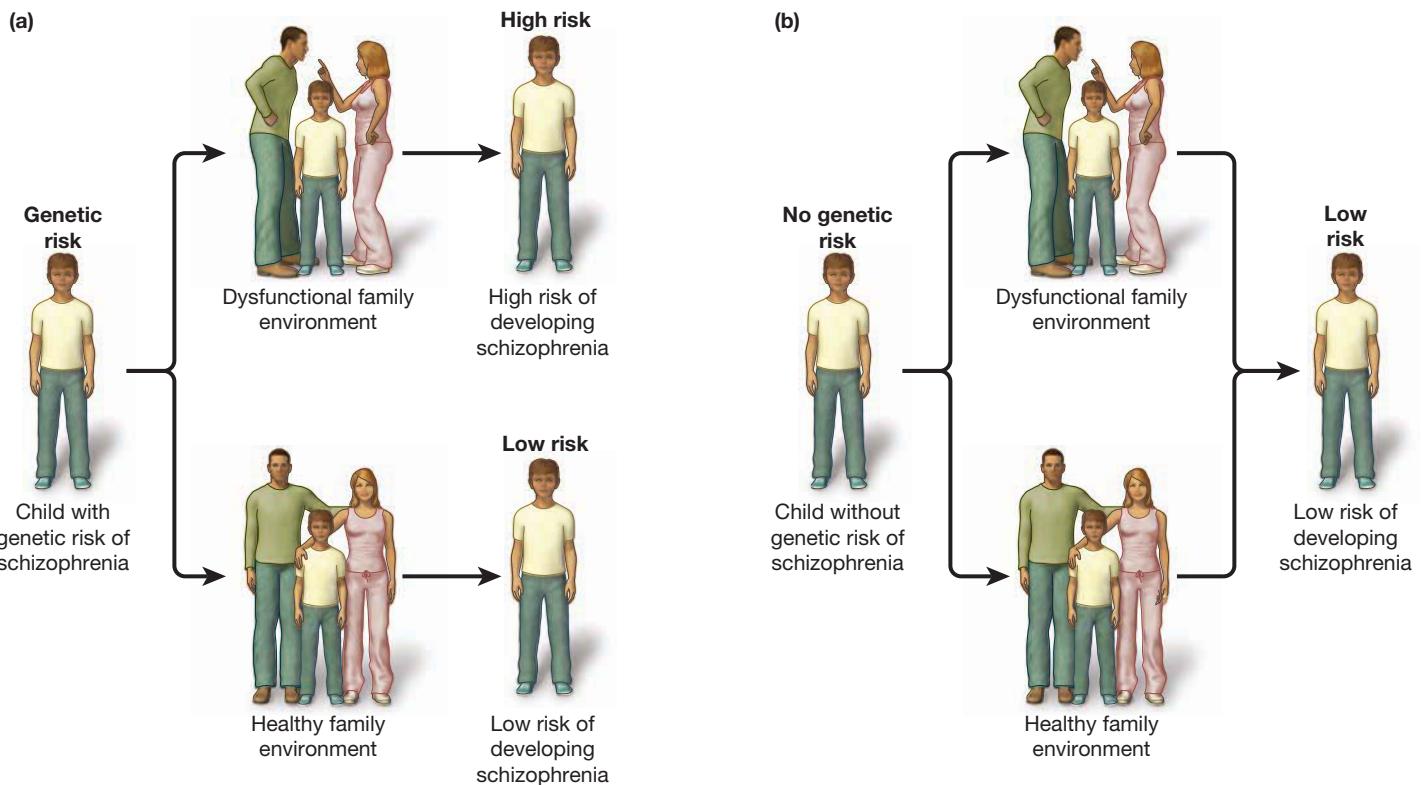
If schizophrenia is a brain disorder, when do these brain abnormalities emerge? Because schizophrenia is most often diagnosed when people are in their 20s or 30s, it is hard to assess whether brain impairments occur earlier in life. There is evidence that some neurological signs of schizophrenia can be observed long before the disorder is diagnosed. Elaine Walker and colleagues (2004) have analyzed home movies taken by parents whose children later developed schizophrenia. Compared with their siblings, those who developed the disorder displayed unusual social behaviors, more-severe negative emotions, and motor disturbances. All of these differences often went unnoticed during the children's early years.

One study followed a group of children at risk for developing psychopathology because their parents suffered from a psychological disorder (Amminger et al., 1999). Adults who developed schizophrenia were much more likely to have displayed behavioral problems as children—such as fighting or not getting along with others—than those who developed mood disorders or drug abuse problems or did not develop any disorders in adulthood. Children at risk for schizophrenia display increasingly abnormal motor movements, such as strange facial expressions, as they progress through adolescence (Mittal, Neumann, Saczawa, & Walker, 2008).

In another study, Walker and colleagues followed a group of children, ages 11 to 13, with a high genetic risk of schizophrenia (Schiffman et al., 2004). These children were videotaped eating lunch in 1972. Those who later developed schizophrenia showed greater impairments in social behavior and motor functioning than those who developed other psychological disorders or those who developed no problems. Another team of researchers followed 291 high-risk youths (average age 16) over 2.5 years (Cannon et al., 2008). These psychologists determined that five factors predicted the onset of psychotic disorders: a family history of schizophrenia, greater social impairment, higher levels of suspicion/paranoia, a history of substance abuse, and higher levels of unusual thoughts. When youths had two or three of the first three factors, nearly 80 percent of them developed full-blown psychosis. Studies such as these suggest that schizophrenia develops over the life course but that obvious symptoms often emerge by late adolescence. Hints of future problems may even be evident in young children.

**ENVIRONMENTAL FACTORS** Since genetics does not account fully for the onset and severity of schizophrenia, other factors must also be at work. In those at risk for schizophrenia, environmental stress seems to contribute to its development (Walker et al., 2004). One study looked at adopted children whose biological mothers were diagnosed with schizophrenia (Tienari et al., 1990, 1994). If the adoptive families were psychologically healthy, none of the children became psychotic. If the adoptive families were severely disturbed, 11 percent of the children became psychotic and 41 percent had severe psychological disorders. More generally, growing up in a dysfunctional family may increase the risk of developing schizophrenia for those who are genetically at risk (Tienari et al., 2004; **FIGURE 14.27**).

Some researchers have theorized that the increased stress of urban environments can trigger the onset of the disorder, since being born or raised in an urban area approximately doubles the risk of developing schizophrenia later in life (Torrey, 1999). Others have speculated that some kind of *schizovirus* exists. If so, the close quarters of a big city increase the likelihood of the virus spreading. In support of the virus hypothesis, some researchers have reported finding antibodies in the blood of people with schizophrenia that are not found in those without the disorder (Waltrip et al., 1997). Moreover, people with schizophrenia are more likely to have been born during late winter and early spring (Mednick, Huttunen, & Machon, 1994; Torrey, Torrey, & Peterson, 1977). Consider that mothers of children born in late winter and early spring were in their second trimester of pregnancy during flu season. Retrospective studies suggest



**FIGURE 14.27**

#### Effects of Biology and Environment on Schizophrenia

**(a)** If a child has a genetic risk for schizophrenia and is raised in a dysfunctional family environment, he or she will have a high risk of developing schizophrenia. **(b)** By contrast, if a child has no genetic risk for schizophrenia, the child will have a low risk of developing the disorder whether raised in a dysfunctional family environment or a healthy family environment.

that the mothers of people with schizophrenia are more likely than other mothers to have contracted influenza during this critical period (Limosin, Rouillon, Payan, Cohen, & Strub, 2003; Mednick et al., 1994). During the second trimester, a great deal of fetal brain development occurs. At that time, trauma or pathogens can interfere with the organization of brain regions.

## Summing Up

### Which Disorders Emphasize Thought Disturbances?

- Dissociative disorders involve disruptions of identity, memory, or conscious awareness.
- Dissociative amnesia involves forgetting that an event happened or losing awareness of a substantial block of time. Dissociative fugue involves a loss of identity.
- Dissociative identity disorder involves the occurrence of two or more distinct identities in the same individual, along with memory gaps for everyday events.
- Dissociative identity disorder is believed to emerge as a consequence of severe abuse—through repeated dissociation, different identities develop to cope with different traumas. However, dissociative identity disorder remains a controversial diagnosis for two reasons: The condition is often diagnosed after someone has been accused of a crime, and a sharp rise in reported cases has occurred in recent years.

- Schizophrenia is characterized by a split between thought and emotion.
- The positive symptoms associated with schizophrenia reflect excesses and include delusions, hallucinations, disorganized speech, and disorganized behavior. The negative symptoms of schizophrenia reflect deficits and include apathy, lack of emotion, and slowed speech.
- Research suggests that schizophrenia is largely a biological disorder. Twin, adoption, and family studies have highlighted the critical role of genetics in the development of schizophrenia, and recent advances in genetic analysis have indicated that multiple genes may contribute to this disorder. Research has also shown that schizophrenia is associated with abnormalities in brain anatomy and neurotransmitters.
- Most researchers agree that environmental factors play a role in schizophrenia. In particular, environmental stressors such as dysfunctional family dynamics, urban stress, and exposure to pathogens may contribute to the genesis of schizophrenia.

## Measuring Up

1. Which of the following statements about fugue are true?
  - a. It is a form of dissociative amnesia.
  - b. It involves a loss of identity.
  - c. It is also referred to as dissociative identity disorder.
  - d. It may occur as a result of alcohol or drug abuse.
  - e. It occurs concurrently with posttraumatic stress disorder.
2. Indicate whether each of the following phenomena is a negative or a positive symptom of schizophrenia.
  - a. social withdrawal
  - b. flat affect
  - c. delusions
  - d. hallucinations
  - e. slowed motor movement
  - f. loosening of associations

(2) a. negative; b. negative; c. positive; d. positive; e. negative; f. positive.

**ANSWERS:** (1) Choices a and b are true.

## 14.4 What Are Personality Disorders?

As discussed in Chapter 13, personality reflects each person's unique response to his or her environment. Although individuals change somewhat over time, the ways they interact with the world and cope with events are fairly fixed by the end of adolescence. For example, some people interact with the world in maladaptive and inflexible ways. When this style of interaction is long-lasting and causes problems in work and in social situations, it becomes a *personality disorder*.

Most people are likely to exhibit symptoms of personality disorders. At times anyone might be indecisive, self-absorbed, or emotionally unstable. In fact, true personality disorders are relatively common, affecting just under 1 in 10 people (Lenzenweger, Lane, Loranger, & Kessler, 2007). People with personality disorders consistently behave in maladaptive ways, show a more extreme level of maladaptive behavior, and experience more personal distress and more problems as a result of their behavior.

## Learning Objectives

- Distinguish between the clusters of personality disorders.
- Understand controversies related to defining personality disorders.
- Identify the symptoms and possible causes of borderline personality disorder and antisocial personality disorder.

## Personality Disorders Are Maladaptive Ways of Relating to the World

*DSM-5* divides personality disorders into three clusters, as listed in TABLE 14.6. Disorders in the Cluster A group are characterized by odd or eccentric behavior. Paranoid, schizoid, and schizotypal personality disorders make up this group. People with these disorders are often reclusive and suspicious, and they have difficulty forming personal relationships because of their strange behavior and aloofness. As you might expect, people with personality disorders in this category show some similarities to people with schizophrenia, but their symptoms are far less severe.

Disorders in the Cluster B group are characterized by dramatic, emotional, or erratic behaviors. *Histrionic, narcissistic, borderline, and antisocial* personality disorders make up this group. Borderline and antisocial personality disorders have been the focus of much research, and they are considered in more detail in the following sections.

Disorders in the Cluster C group are characterized by anxious or fearful behavior. *Avoidant, dependent, and obsessive-compulsive* personality disorders make up this group. These disorders share some characteristics of anxiety disorders such as social anxiety disorder or generalized anxiety disorder. However, the personality disorders in this group are different from anxiety disorders in that they refer more to general ways of interacting with others and of responding to events. For instance, a person with an obsessive-compulsive personality disorder may be excessively neat and orderly. The person might always eat the same food at precisely the same time or perhaps read a newspaper in a particular order each time. This pattern becomes problematic only when it interferes with the person's life, as in making it impossible to travel or to maintain relationships.

**Table 14.6** Personality Disorders and Associated Characteristics

CLUSTER A: ODD OR ECCENTRIC BEHAVIOR	
<b>Paranoid</b>	Tense, guarded, suspicious; holds grudges
<b>Schizoid</b>	Socially isolated, with restricted emotional expression
<b>Schizotypal</b>	Peculiarities of thought, appearance, and behavior that are disconcerting to others; emotionally detached and isolated
CLUSTER B: DRAMATIC, EMOTIONAL, OR ERRATIC BEHAVIOR	
<b>Histrionic</b>	Seductive behavior; needs immediate gratification and constant reassurance; rapidly changing moods; shallow emotions
<b>Narcissistic</b>	Self-absorbed; expects special treatment and adulation; envious of attention to others
<b>Borderline</b>	Cannot stand to be alone; intense, unstable moods and personal relationships; chronic anger; drug and alcohol abuse
<b>Antisocial</b>	Manipulative, exploitative; dishonest; disloyal; lacking in guilt; habitually breaks social rules; childhood history of such behavior; often in trouble with the law
CLUSTER C: ANXIOUS OR FEARFUL BEHAVIOR	
<b>Avoidant</b>	Easily hurt and embarrassed; few close friends; sticks to routines to avoid new and possibly stressful experiences
<b>Dependent</b>	Wants others to make decisions; needs constant advice and reassurance; fears being abandoned
<b>Obsessive-compulsive</b>	Perfectionistic; overconscientious; indecisive; preoccupied with details; stiff; unable to express affection

SOURCE: Adapted from American Psychiatric Association (2013).

In modern clinical practice, personality disorders are controversial for several reasons. First, personality disorders appear to be extreme versions of normal personality traits, demonstrating the continuum between what is considered normal versus abnormal (Clark & Ro, 2014; Widiger, 2011). For example, indecisiveness is characteristic of obsessive-compulsive personality disorder, but the *DSM* does not define the degree to which someone must be indecisive to be diagnosed as obsessive-compulsive. Second, there is overlap among the traits listed as characteristic of different personality disorders, so the majority of people diagnosed with one personality disorder also meet the criteria for another (Clark, 2007). This overlap suggests that the categories may not be mutually exclusive and that fewer types of personality disorders may exist than are listed in the *DSM*. Indeed, there is evidence that personality disorders can be conceptualized and organized as extreme versions of the Big Five personality traits, described in Chapter 13.

Acknowledging this weakness, but wanting to preserve continuity in current clinical practice, *DSM-5* describes an alternative model for personality disorders in Section III that aims to address many of the shortcomings of the traditional *DSM* approach. In this alternative model, personality disorders are viewed as impairments in personality functioning and the existence of pathological personality traits. That is, the person with the disorder shows extreme personality traits that interfere with successful functioning in society.

Personality disorders may not seem to affect daily life as much as do some of the other disorders discussed in this chapter, such as schizophrenia or bipolar disorders. Although people with personality disorders do not hallucinate or experience radical mood swings, their ways of interacting with the world can have serious consequences. The following in-depth considerations of borderline personality disorder and antisocial personality disorder illustrate the devastating effect of these disorders on the individual, family and friends, and society.

## Borderline Personality Disorder Is Associated with Poor Self-Control

**Borderline personality disorder** is characterized by disturbances in identity, in affect, and in impulse control. This disorder was officially recognized as a diagnosis in 1980. The term *borderline* was initially used because these patients were considered on the border between normal and psychotic (Knight, 1953). As presented in **TABLE 14.7**, the wide variety of clinical features of this disorder reflects its complexity. Approximately 1–2 percent of adults meet the criteria for borderline personality disorder, and the disorder is more than twice as common in women as in men (Lenzenweger et al., 2007; Swartz, Blazer, George, & Winfield, 1990; Torgerson, Kringlen, & Cramer, 2001).

People with borderline personality disorder seem to lack a strong sense of self. They cannot tolerate being alone and have an intense fear of abandonment. Because they desperately need an exclusive and dependent relationship with another person, they can be very manipulative in their attempts to control relationships, as shown in the following example:

A borderline patient periodically rented a motel room and, with a stockpile of pills nearby, would call her therapist's home with an urgent message. He would respond by engaging in long conversations in which he "talked her down." Even as he told her that she could not count on his always being available, he became more wary of going out evenings without detailed instructions about how he could be reached. One night the patient couldn't reach him due to a bad phone connection. She fatally overdosed from what was probably a miscalculated manipulation. (Gunderson, 1984, p. 93)

### borderline personality disorder

A personality disorder characterized by disturbances in identity, in affect, and in impulse control.

## **Table 14.7 DSM-5 Diagnostic Criteria of Borderline Personality Disorder**

A pervasive pattern of instability of interpersonal relations, self-image, and affects, along with marked impulsivity, beginning by early adulthood and present in a variety of contexts, as indicated by five (or more) of the following:

1. Frantic efforts to avoid real or imagined abandonment
2. A pattern of unstable and intense interpersonal relationships
3. Identity disturbance: markedly and persistently unstable self-image or sense of self
4. Impulsiveness in at least two areas that are potentially self-damaging (e.g., spending, sex, substance abuse, reckless driving, binge eating)
5. Recurrent suicidal behavior, gestures, or threats, or self-mutilating behavior
6. Affective instability due to a marked reactivity of mood, with periods of extreme depression, irritability, or anxiety usually lasting a few hours and only rarely more than a few days
7. Chronic feelings of emptiness
8. Inappropriate intense anger or difficulty controlling anger (e.g., displays of temper, constant anger, recurrent physical fights)
9. Transient, stress-related paranoid thoughts or severe dissociative symptoms

SOURCE: Based on American Psychiatric Association (2013).

In addition to problems with identity, borderline individuals have affective disturbances. Emotional instability is paramount. Episodes of depression, anxiety, anger, irritability, or some combination of these states can last from a few hours to a few days. Shifts from one mood to another usually occur with no obvious precipitating cause. Consider the therapist Molly Layton's description of her patient Vicki:

She had chronic and debilitating feelings of emptiness and paralyzing numbness, during which she could only crawl under the covers of her bed and hide. On these days, she was sometimes driven to mutilate her thighs with scissors. Although highly accomplished as a medical student and researcher, who had garnered many grants and fellowships, she would sometimes panic and shut down in the middle of a project, creating unbearable pressures on herself to finish the work. While she longed for intimacy and friendship, she was dismally shy around men. (Layton, 1995, p. 36)

The third hallmark of borderline personality disorder is impulsivity, which may explain the much higher rate of the disorder in prison populations (Conn et al., 2010). This characteristic can include sexual promiscuity, physical fighting, and binge eating and purging. As was the case with Vicki, however, self-mutilation is also commonly associated with this disorder. Cutting and burning of the skin are typical, as well as a high risk for suicide. Some evidence indicates that those with borderline personality disorder have diminished capacity in the frontal lobes, which normally help control behavior (Silbersweig et al., 2007).

In addition, people with borderline personality disorder often show sleep abnormalities characteristic of depression. One possible reason that borderline personality disorder and affective disorders such as depression may be linked is that both appear to involve the neurotransmitter serotonin. Evidence has linked low serotonin levels to the impulsive behavior seen in borderline personality disorder (Skodol et al., 2002).

Borderline personality disorder may also have an environmental component, as a strong relationship exists between the disorder and trauma or abuse (Lieb, Zanarini, Schmahl, Linehan, & Bohus, 2004). Some studies have reported that 70–80 percent

of those with borderline personality disorder have experienced physical or sexual abuse or observed some kind of extreme violence. Other theories implicate early interactions with caretakers. Clients with borderline personality disorder may have had caretakers who did not accept them or who were unreliable or unavailable. The constant rejection and criticism made it difficult for the individuals to learn to regulate emotions and understand emotional reactions to events (Linehan, 1987). An alternative theory is that caregivers encouraged dependence, preventing the individuals in their charge from adequately developing a sense of self. As a result, the individuals became overly sensitive to others' reactions: If rejected by others, they reject themselves.

## Antisocial Personality Disorder Is Associated with a Lack of Empathy

In the 1800s, the term *psychopath* was coined to describe people who seem willing to take advantage of and to hurt others without any evidence of concern or of remorse (Koch, 1891). In his classic book *The Mask of Sanity* (1941), the psychiatrist Hervey Cleckley described characteristics of psychopaths from his clinical experience. For example, such individuals could be superficially charming and rational; be insincere, unsocial, and incapable of love; lack insight; and be shameless. In 1980, the *DSM* dropped the label *psychopath*, which was seen as pejorative, and adopted the term **antisocial personality disorder (APD)**. This change has led to confusion because *psychopath* is still widely used to refer to a related but not identical type of personality disorder as defined by *DSM-5*.

APD is the catchall diagnosis for individuals who behave in socially undesirable ways, such as breaking the law, being deceitful and irresponsible, and feeling a lack of remorse for their behavior. People with this disorder tend to be hedonistic, seeking immediate gratification of wants and needs without any thought of others.

True psychopaths display more extreme behaviors than those with APD. They also tend to have other personality characteristics not found in those with APD, such as glibness, a grandiose sense of self-worth, shallow affect, and cunning/manipulativeness. Psychopaths would be classified as APD under *DSM-5*, but they are an extreme version of the disorder. Psychopaths are pathological in their degree of callousness and are particularly dangerous. For instance, one study of murderers found that those with psychopathic tendencies nearly always kill intentionally. They want to gain something, such as money, sex, or drugs. People without psychopathic tendencies are much more likely to commit murder impulsively, when provoked or angry (Woodworth & Porter, 2002). Psychopaths fit the stereotype of cold-blooded killers. Infamous examples include Dennis Rader—the BTK strangler, who bound, tortured, and killed 10 victims—and Gary Gilmore (**FIGURE 14.28**). In 1977, Gilmore was executed for the murder he describes here:

I went in and told the guy to give me the money. I told him to lay on the floor and then I shot him. I then walked out and was carrying the cash drawer with me. I took the money and threw the cash drawer in a bush and I tried to push the gun in the bush, too. But as I was pushing it in the bush, it went off and that's how come I was shot in the arm. It seems like things have always gone bad for me. It seems like I've always done dumb things that just caused trouble for me. I remember when I was a boy I would feel like I had to do things like sit on a railroad track until just before the train came and then I would dash off. Or I would put my finger over the end of a BB gun and pull the trigger to see if a BB was really in it. Sometimes I would stick my finger in water and then put my finger in a light socket to see if it would really shock me. (Spitzer et al., 1983, pp. 66–68)

### antisocial personality disorder (APD)

A personality disorder in which people engage in socially undesirable behavior, are hedonistic and impulsive, and lack empathy.



**FIGURE 14.28**  
**Gary Gilmore After His Arrest**

Under *DSM-5*, Gilmore would have been given a diagnosis of antisocial personality disorder. He also showed psychopathic traits.

**ASSESSMENT AND CONSEQUENCES** It is estimated that 1–4 percent of the population have antisocial personality disorder (Compton, Conway, Stinson, Colliver, & Grant, 2005). People with this condition who also show more-extreme psychopathic traits are less common (Lenzenweger et al., 2007). Both APD and psychopathy are much more common in men than in women (Robins & Regier, 1991).

Much of what psychologists know about the traits associated with antisocial personality disorder was discovered by the psychologist Robert Hare (1993). Hare also developed many of the assessment tools to identify people with psychopathic tendencies. He and colleagues have shown that the disorder (including its extreme version) is most apparent in late adolescence and early adulthood, and it generally improves around age 40 (Hare, McPherson, & Forth, 1988), at least for those without psychopathic traits. According to the *DSM-5* diagnostic criteria, APD cannot be diagnosed before age 18, but the person must have displayed antisocial conduct before age 15. This stipulation ensures that only those with a lifetime history of antisocial behaviors can be diagnosed with antisocial personality disorder. They also must meet other criteria, such as repeatedly performing illegal acts, repeatedly lying or using aliases, and showing reckless disregard for their own safety or the safety of others. Because many such individuals are quite bright and highly verbal, they can talk their way out of bad situations. In any event, punishment seems to have very little effect on them (Lykken, 1957, 1995), and they often repeat the problem behaviors a short time later.

Perhaps as many as 50 percent of prison inmates meet the criteria for antisocial personality disorder (Hare, 1993; Widiger & Corbitt, 1995). Because of the prevalence of the disorder in the prison population, much of the research on APD has been conducted in this setting. One researcher, however, came up with an ingenious way of finding research participants outside of prison. She put the following advertisement in a counterculture newspaper: “Wanted: charming, aggressive, carefree people who are impulsively irresponsible but are good at handling people and at looking after number one. Send name, address, phone, and short biography proving how interesting you are to . . .” (Widom, 1978, p. 72). Seventy-three people responded, and about one-third of them met the criteria for antisocial personality disorder. These individuals were then interviewed and given a battery of psychological tests. Their characteristics proved very similar to those of prisoners diagnosed with APD, except that the group responding to the ad had avoided imprisonment. Indeed, these findings fit Cleckley’s view of people with psychopathic traits as often being charming and intelligent. Lacking remorse, willing to lie or cheat, and lacking empathy, some psychopaths manage to be successful professionals and to elude detection for crimes they may commit. Their psychopathic traits may even provide advantages in some occupations, such as business and politics (**FIGURE 14.29**).



**FIGURE 14.29**  
**American Psychopath**  
In the 2000 movie *American Psycho*, Christian Bale plays Patrick Bateman, who appears to be a suave man-about-town, a successful professional, and a serial killer. The movie, like the Bret Easton Ellis novel it is based on, raises questions about the connections between a slick presentation of self, a knack for making and spending money, and psychopathy.

**THE ETIOLOGY OF ANTISOCIAL PERSONALITY DISORDER** Various physiological abnormalities may play a role in antisocial personality disorder. In 1957, David Lykken reported that true psychopaths do not become anxious when they are subjected to aversive stimuli. He and other investigators have continued this line of work, showing that such individuals do not seem to feel fear or anxiety (Lykken, 1995).

Electroencephalogram (EEG) examinations have demonstrated that criminals who meet the criteria for antisocial personality disorder have slower alpha-wave activity (Raine, 1989). This finding indicates a lower overall level of arousal. It is possible that low arousal prompts people with APD to engage in sensation-seeking behavior. In addition, because of low arousal, these individuals do not learn from punishment because they do not experience punishment as particularly aversive. This pattern of reduced psychophysiological response in the face of punishment also occurs in adolescents at risk for developing psychopathy (Fung et al., 2005).

There is also evidence of amygdala abnormalities in those with antisocial tendencies, such as having a smaller amygdala and being less responsive to negative stimuli (Blair, 2003; Marsh et al., 2011). Deficits in frontal lobe functioning have also been found and may account for the lack of forethought and the inability to consider the implications of actions, both characteristic of antisocial personality disorder (Seguin, 2004).

Genetic and environmental factors appear to play roles in antisocial personality disorder. Genetics may be more important for the extreme psychopathic version, however. Identical twins have a higher concordance rate for criminal behavior than fraternal twins do (Lykken, 1995), although the research just cited did not rule out the role of a shared environment. A study of 14,000 adoptions found that adopted male children have a higher rate of crime if their biological fathers have criminal records (Mednick, Gabrielli, & Hutchings, 1987). In addition, the greater the criminal record of the biological father, the more likely it is that the adopted son will engage in criminal behavior.

Although genes may be at the root of antisocial behaviors and psychopathy, factors such as low socioeconomic status, dysfunctional families, and childhood abuse may also be important. Indeed, malnutrition at age 3 has been found to predict antisocial behavior at age 17 (Liu, Raine, Venables, & Mednick, 2004). An enrichment program for children that included a structured nutrition component was associated with less criminal and antisocial behavior 20 years later (Raine, Mellingen, Liu, Venables, & Mednick, 2003). This finding raises the possibility that malnutrition or other, similar environmental factors might contribute to the development of antisocial personality disorder.

## Summing Up

### What Are Personality Disorders?

- Ten personality disorders, clustered in three groups, are identified in the *DSM*: paranoid, schizoid, schizotypal (odd or eccentric cluster), histrionic, narcissistic, borderline, antisocial (dramatic, emotional, or erratic cluster), and avoidant, dependent, obsessive-compulsive (anxious or fearful cluster).
- Borderline personality disorder is characterized by disturbances in identity, in affect, and in impulse control.
- Research has shown that people with borderline personality disorder often have diminished frontal lobe capacity, low levels of serotonin, and a history of abuse or rejection by caregivers.
- Those with antisocial personality disorder engage in socially undesirable behavior, are hedonistic and impulsive, and lack empathy. Psychopaths have an extreme version of APD.
- Antisocial personality disorder is associated with lower levels of arousal, a smaller amygdala, and deficits in frontal lobe functioning.
- Twin and adoption studies suggest that genes play a role in antisocial personality disorder. However, environmental factors (such as low socioeconomic status, dysfunctional families, abuse, and malnutrition) also contribute to the development of this disorder.

## Measuring Up

1. Which of the following characteristics are *DSM-5* diagnostic criteria of borderline personality disorder?
  - a. frantic efforts to avoid real or imagined abandonment
  - b. a pattern of unstable and intense relationships
  - c. a lack of guilt or remorse

- d. a lack of empathy
  - e. an unstable self-image or sense of self
  - f. self-mutilating behavior
  - g. chronic feelings of emptiness
2. Why would punishment be an ineffective means of treating those with antisocial personality disorder?
- a. The disorder is genetically based, and you cannot change genes.
  - b. Most people with the disorder have already been effectively punished by imprisonment.
  - c. People with the disorder do not find punishment aversive.
  - d. The best treatment is to provide a nutritionally balanced diet.

**ANSWERS:** (1) Choices a, b, e, f, and g apply. (2) c. People with the disorder do not find punishment aversive.

## Learning Objectives

- Understand the childhood context of neurodevelopmental disorders.
- Identify the symptoms and possible causes of autism spectrum disorder.
- Identify the symptoms and possible causes of attention-deficit/hyperactivity disorder.

## 14.5 Which Psychological Disorders Are Prominent in Childhood?

In his classic text on the classification of psychological disorders, published in 1883, Emil Kraepelin did not mention childhood disorders. The first edition of the *DSM*, published 70 years later, essentially considered children small versions of adults. Consequently, the manual did not consider childhood disorders separately from adulthood disorders. The current version of the manual includes a wide range of childhood disorders (**TABLE 14.8**). Some of these conditions—such as specific learning disorders—affect only limited and particular areas of a child’s world. Other

**Table 14.8 DSM-5 Neurodevelopmental Disorders**

DISORDER	DESCRIPTION
<b>Intellectual disabilities</b>	Deficits in general mental abilities (e.g., reasoning, problem solving, planning, academic learning, learning from experience) and in adaptive functioning (e.g., independent living, working, social participation); begins during childhood or adolescence
<b>Communication disorders</b>	Deficits in language, speech, or communications, such as difficulty learning a language, stuttering, or failure to follow social rules for communication; begins in childhood
<b>Autism spectrum disorder</b>	Persistent impairment in social interaction; characterized by unresponsiveness; impaired language, social, and cognitive development; and restricted and repetitive behavior; begins during early childhood
<b>Attention-deficit/hyperactivity disorder</b>	A pattern of hyperactive, inattentive, and impulsive behavior that causes social or academic impairment; begins before age 12
<b>Specific learning disorders</b>	Difficulty learning and using academic skills; much lower performance in reading, mathematics, or written expression with regard to what is expected for age, amount of education, and intelligence; begins during school-age years
<b>Motor disorders</b>	Recurrent motor and vocal tics that cause marked distress or deficits in developing or being able to show coordinated motor skills; begins in childhood

SOURCE: Based on American Psychiatric Association (2013).

conditions—such as autism spectrum disorder, attention-deficit/hyperactivity disorder, and others listed in Table 14.8—affect every aspect of a child’s life. Some of these disorders, such as autism spectrum disorder, usually do not get better over time. Others, such as attention-deficit/hyperactivity disorder, usually do improve over time.

All of the disorders in this category should be considered within the context of normal childhood development. Some symptoms of childhood psychological disorders are extreme manifestations of normal behavior or are actually normal behaviors for children at an earlier developmental stage. For example, bedwetting is normal for 2-year-olds but not for 10-year-olds. Other behaviors, however, deviate significantly from normal development. Two disorders of childhood, autism spectrum disorder and attention-deficit/hyperactivity, are explored here as illustrations.

## Autism Spectrum Disorder Involves Social Deficits and Restricted Interests

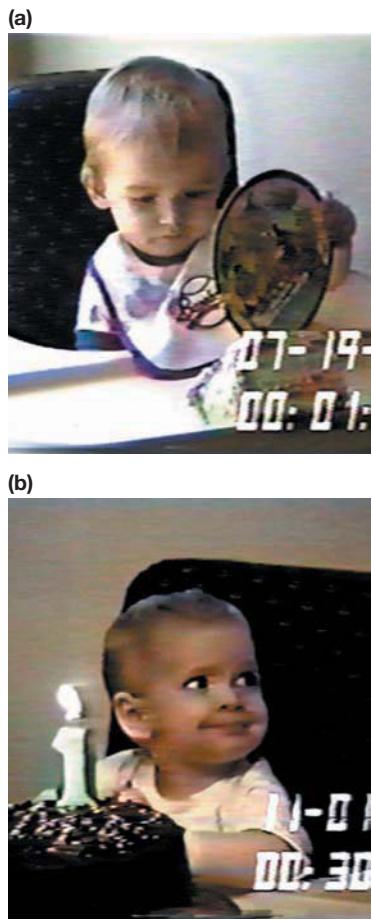
Prior to *DSM-5*, a number of similar disorders were considered variants of *autistic disorder*, commonly known as *autism*, which is characterized by deficits in social interaction, by impaired communication, and by restricted interests (Volkmar, Chawarska, & Klin, 2005). The disorder was first described in 1943, by the psychiatrist and physician Leo Kanner. Struck by the profound isolation of some children, Kanner coined the term *early infantile autism*. Researchers and clinicians recognized that autism varied considerably in severity, from mild social impairments to severe social and intellectual impairments. For example, those with high-functioning autism were considered to have *Asperger's syndrome*, named after the pediatrician who first described it. A child with Asperger's has normal intelligence but deficits in social interaction. These deficits reflect an underdeveloped theory of mind. As discussed in Chapter 9, theory of mind is both the understanding that other people have mental states and the ability to predict their behavior accordingly.

Based on the *DSM-IV* diagnosis of autistic disorder, approximately 3 to 6 children out of 1,000 showed signs of autism, and males with the disorder outnumbered females 3 to 1 (Muhle, Trentacoste, & Rapin, 2004). From 1991 to 1997, a dramatic escalation—of 556 percent—occurred in the number of children diagnosed with autism (Stokstad, 2001). This increase was likely due to a greater awareness of symptoms by parents and physicians and a willingness to apply the diagnosis to a wider spectrum of behaviors (Rutter, 2005). For example, a study of all children born between 1983 and 1999 in Western Australia found that the apparent growth in the diagnosis of autistic disorder was due to changes in how it was diagnosed as well as expanded funding for psychological services for children showing signs of autism (Nassar et al., 2009). In other words, the notion that autism was epidemic was somewhat misleading because what changed was how it was defined, not how many new cases developed (Gernsbacher, Dawson, & Goldsmith, 2005).

**Autism spectrum disorder** is a new *DSM-5* disorder that groups together all the variants in symptoms of autism, including Asperger's syndrome. This diagnosis classification is an excellent example of the dimensional approach to psychopathology, in that there is clear recognition that the disorder varies along a single continuum from mild to severe impairment. In *DSM-5*, the two essential features of autism spectrum disorder are impairments in social interactions along with restrictive or repetitive behaviors, interests, or activities. These symptoms are present in early childhood and limit or impair everyday functioning. In the following sections, we use the terms *autism spectrum disorder* and *autism* interchangeably because most of the research to date has not used the *DSM-5* criteria for diagnosis. Most of our discussion focuses on the classic severe end of the autism spectrum, which definitely meets the *DSM-5* criteria.

### autism spectrum disorder

A developmental disorder characterized by deficits in social interaction, by impaired communication, and by restricted interests.



**FIGURE 14.30**  
**Scenes from Videotapes of Children's Birthday Parties**  
**(a)** This child focused more on objects than on people. The child was later diagnosed with autism. **(b)** This child focused appropriately on objects and on people. The child developed normally.

**CORE SYMPTOMS OF AUTISM SPECTRUM DISORDER** Children on the more extreme end of the autism spectrum are seemingly unaware of others. As babies, they do not smile at their caregivers, do not respond to vocalizations, and may actively reject physical contact with others. Children with autism do not establish eye contact and do not use their gazes to gain or direct the attention of those around them. Although they show attention to the eyes before 2 months of age, they stop making eye contact by 6 months of age (Jones & Klin, 2013). One group of researchers had participants view video footage of the first birthdays of children with autistic disorder to see if characteristics of autism could be detected before the children were diagnosed (Osterling & Dawson, 1994). By considering only the number of times a child looked at another person's face, the participants correctly classified the children as having or not having autism 77 percent of the time (**FIGURE 14.30**).

Deficits in communication are the second major cluster of behaviors characteristic of autism spectrum disorders. Such deficits are evident by 14 months of age among children who are subsequently diagnosed with autism (Landa, Holman, & Garrett-Mayer, 2007). Children with autism show severe impairments in verbal and nonverbal communication. Even if they vocalize, it is often not with any intent to communicate. Children with autism who develop language usually exhibit odd speech patterns, such as echolalia (the mindless repeating of words or phrases that someone else has spoken that is also observed in those with schizophrenia). The repeater may imitate the first speaker's intonation or may use a high-pitched monotone. Those who develop functional language also often interpret words literally, use language inappropriately, and lack verbal spontaneity.

A third category of deficits includes restricted activities and interests. Children with autism spectrum disorder appear oblivious to people around them, but they are acutely aware of their surroundings. Although most children automatically pay attention to the social aspects of a situation, those with autism may focus on seemingly inconsequential details (Klin, Jones, Schultz, & Volkmar, 2003; **FIGURE 14.31**).

Any changes in daily routine or in the placement of furniture or of toys are very upsetting for children with autism. Once they are upset, the children can become extremely agitated or throw tantrums. In addition, the play of children with autism tends to be repetitive and obsessive, with a focus on objects' sensory aspects. They may smell and taste objects, or they may spin and flick them for visual stimulation. Similarly, their own behavior tends to be repetitive, with strange hand movements, body rocking, and hand flapping. Self-injury is common, and some children must be forcibly restrained to keep them from hurting themselves.

**BIOLOGICAL BASIS OF AUTISM SPECTRUM DISORDER** Kanner, one of the first scientists to study autism, believed the disorder was innate in some children but exacerbated by cold and unresponsive mothers, whom he called "ice box mothers" or "refrigerator mothers." He described the parents of children with autism as insensitive, meticulous, introverted, and highly intellectual. This view is given little credence today, as it is now well established that autism spectrum disorder is the result of biological factors. For example, there is evidence for a genetic component to autism. A number of studies have found concordance rates to be as high as 70–90 percent for identical twins (Holmboe et al., 2013; Hyman, 2008; Ronald & Hoekstra, 2011; Steffenburg et al., 1989).

In addition to autism being heritable, it also appears that gene mutations may play a role (Ronemus, Iossifov, Levy, & Wigler, 2014). An international study that compared 996 children with autism to 1,287 control children found a number of rare gene abnormalities (Pinto et al., 2010). These rare mutations involve cells having an abnormal number of copies of DNA segments. An independent study of over 1,000

individuals with autism spectrum disorders who had an unaffected sibling found that these mutations were much more common in the children with autism (Levy et al., 2011). The mutations may affect the way neural networks are formed during childhood development (Gilman et al., 2011). There is growing evidence that autism and schizophrenia share the same gene mutations (Fromer et al., 2014; McCarthy et al., 2014). There are also some similarities in the symptoms for the two disorders, including social impairment and avoiding eye contact. Recall the RDoC initiative, discussed earlier in the chapter, that integrates findings across multiple disorders rather than classifying by *DSM* diagnostic categories. The RDoC approach suggests that schizophrenia and ASD may be related disorders or involve similar deficits in core psychological domains.

Research into the causes of autism also points to prenatal and/or early childhood events that may result in brain dysfunction. The brains of children with autism grow unusually large during the first two years of life, and then growth slows until age 5 (Courchesne et al., 2007; Courchesne, Redcay, & Kennedy, 2004). The brains of children with autism also do not develop normally during adolescence (Amaral, Schumann, & Nordahl, 2008). Researchers are investigating genetic factors, such as mutations, and nongenetic factors that might explain this overgrowth/undergrowth pattern.

Some recent work suggests that exposure to antibodies in the womb may affect brain development. Investigators found abnormal antibodies in the blood of the mothers of 11 percent of children with autism but not in a large sample of mothers with healthy children or mothers of children with other developmental disorders (Braunschweig et al., 2008). Following up on this study, researchers injected four pregnant rhesus monkeys with the antibodies from the mothers of children with autism. All the offspring of these monkeys demonstrated unusual behaviors characteristic of autism, such as repetitive movements and hyperactive limb movements (Martin et al., 2008). None of the offspring of monkeys injected with normal antibodies from mothers of healthy children showed this unusual behavior.

In addition, there is evidence that the brains of people with autism have faulty wiring in a large number of areas (Minshew & Williams, 2007). Some of those brain areas are associated with social thinking, and others might support attention to social aspects of the environment (Minshew & Keller, 2010).

One line of research examined the possibility that those with autism have impairments in the mirror neuron system. (Recall from Chapter 6 that mirror neurons are involved in observational learning and are activated when someone watches other people performing actions.) This connection between mirror neurons and autism was suggested by an imaging study that found weaker activation in the mirror neuron system for those with autism than for those without (Dapretto et al., 2006). Other researchers, however, have not found impairments in mirror neuron activity for gestures and movements (Dinstein et al., 2010; Southgate & Hamilton, 2008). What might these apparently contradictory findings mean?

It is possible that impairments in the mirror neuron system prevent the person with autism from understanding the *why* of actions, not the *what* of actions (Rizzolatti & Fabbri-Destro, 2010). For example, suppose that the person with autism knows that another person is lifting a pair of scissors. The person with autism may have little insight into what the person intends to do with the scissors.



**FIGURE 14.31**  
**Toddler Viewer with Autism**

As shown in these combined video images from a 1994 study of autism, a 2-year-old with autism will focus on the unimportant details in the scene rather than on the social interaction.

# What to Believe? Using Psychological Reasoning

## Seeing Relationships That Do Not Exist: Do Vaccinations Cause Autism Spectrum Disorder?

What if you heard about a study in which researchers found that moving to Florida or Arizona is a leading cause of death? Or that wearing dentures is another leading cause of death, along with retiring, wearing bifocals, or moving to a nursing home? As a critical thinker, you probably noticed that these things are all associated with aging. It is getting older, rather than moving to Florida or buying bifocals, that is associated with dying. As you have been reminded throughout this book, correlation does not equal causation. We need to be especially vigilant for lurking third variables that might explain apparent correlations between unrelated variables.

Recognizing the third variable problem is especially important when trying to understand claims about causes of psychological disorders. In 1998, the British physician Andrew Wakefield published a study in the prestigious journal *Lancet* claiming to find a connection, in 12 children, between receiving vaccinations to prevent measles, mumps, and rubella (MMR) and developing autism (Wakefield et al., 1998). This finding was widely reported in the media even though most scientists were skeptical and urged people to be patient until the result could be replicated with larger samples. But many people panicked. In 2007, the celebrity Jenny McCarthy publicly blamed the MMR vaccine for her son's autism. She became a prominent spokesperson for the anti-vaccine movement, appearing on television shows such as *Oprah* to warn people about "the autism shot" (**FIGURE 14.32**). Deirdre Imus, the wife of the outspoken radio host Don Imus, joined the publicity war against vaccinations, claiming that the chemical thimerosal in the solutions used to administer vaccines is responsible

for autism. Thimerosal is a preservative that contains small amounts of mercury and was widely used before 2000. Since then, it has been removed in all childhood vaccines except for one type of flu shot.

Unfortunately, the Wakefield study was fraudulent. Wakefield altered medical records and lied about several aspects of his study, including a financial conflict of interest (Godlee, Smith, & Marcovitch, 2011). His coauthors had earlier retracted the paper when they had developed doubts about the data and conclusions (Murch et al., 2004). Wakefield has subsequently been banished by the British medical community, and his license to practice medicine has been taken away.

The original *Lancet* report prompted several large international studies to examine the possibility of a link between autism spectrum disorders and the MMR vaccine. A thorough review of these studies by the Institute of Medicine found no evidence of any link between MMR vaccinations and autism (Immunization Safety Review Committee, 2004). Recent studies have continued to find no evidence of any link between childhood vaccinations and ASD (e.g., DeStefano, Price, &

Weintraub, 2013). The results of dozens upon dozens of carefully designed studies have provided a firm conclusion: Vaccines do not cause ASD.

But the fear of ASD led many parents around the globe to forgo vaccinating their children. As one researcher noted, "Unfortunately, the media has given celebrities who comment on an autism-MMR link far more attention than they deserve, and the public, unfamiliar with the background science, has confused celebrity status with authority" (Poland, 2011, p. 870). Even today, with overwhelming scientific evidence that vaccines do not cause ASD, many parents refuse to vaccinate their children because of worries that it might do so (Opel et al., 2014).

As a consequence of the decline in childhood immunizations, there has been an increase in outbreaks of diseases that had become quite rare because of successful vaccine programs. In 2011, France had 14,000 cases of measles, 6 of them fatal. In 2012, the Centers for Disease Control reported the largest number of cases of whooping cough in 60 years. In the first four months of 2013, rubella cases in Japan jumped from a few a year to more than 5,000. The reemergence of these diseases is occurring in many European nations (Eisenstein, 2014). Meanwhile, researchers at the CDC estimate that for children born between 1994 and 2013, vaccinations prevented an estimated 322 million illnesses, 21 million hospitalizations, and 732,000 deaths over their lifetimes (Whitney, Zhou, Singleton, & Schuchat, 2014).

Wakefield originally conducted his study because the parents of the 12 children with autism told him that they remembered the autism starting right after their children were immunized. Jenny McCarthy told Oprah that



**FIGURE 14.32**  
**Anti-Vaccination Statements**

Jenny McCarthy speaks to the audience at a Green Our Vaccines press conference outside the U.S. Capitol in 2008.

immediately after her son received the vaccine, "Boom—the soul's gone from his eyes" (September 18, 2007). Many have disputed her account, but the bottom line is that vaccines are given to children at about the same developmental period that symptoms of ASD become apparent. Think about the other characteristics that emerge at the same time in development. For example, lower molars emerge in children's mouths during early childhood. However, few people would suggest that being vaccinated causes molars to grow. Children

start speaking at about this age, but no one thinks vaccines cause this ability.

People see an apparent connection between vaccines and ASD, but the lurking third variable is age.

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The results of dozens upon dozens of carefully designed studies have provided a firm conclusion: Vaccines do not cause ASD.

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Since Wakefield's 1998 publication, cases of ASD have increased even though thimerosal is no longer used in vaccines and the number of children being immunized has dropped. These facts would indicate that vaccination and ASD are negatively correlated! As noted in the text, however, definitional changes in the diagnostic criteria are likely a better explanation for the increase in ASD.

## Attention-Deficit/Hyperactivity Disorder Is a Disruptive Impulse Control Disorder

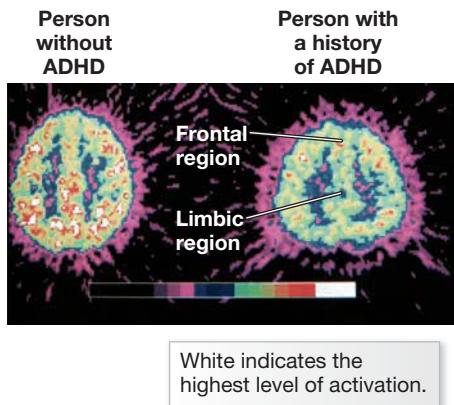
Suppose you are a child who exhibits hyperactivity. At home, you might have difficulty remembering not to trail your dirty hand along the clean wall as you run from the front door to the kitchen. While playing games with your peers, you might spontaneously change the rules. At school, you might ask what you are supposed to do immediately after the teacher has presented detailed instructions to the entire class. You might make warbling noises or other strange sounds that inadvertently disturb anyone nearby. You might seem to have more than your share of accidents—for example, knocking over the tower your classmates are erecting, spilling your juice, or tripping over the television cord while retrieving the family cat, thereby disconnecting the set in the middle of the Super Bowl (Whalen, 1989).

Symptoms such as these can seem humorous in the retelling, but the reality is a different story. Children with **attention-deficit/hyperactivity disorder (ADHD)** are restless, inattentive, and impulsive. They need to have directions repeated and rules explained over and over. Although these children are often friendly and talkative, they can have trouble making and keeping friends because they miss subtle social cues and make unintentional social mistakes. Many of these symptoms are exaggerations of typical toddler behavior, and thus the line between normal and abnormal behavior is hard to draw. The *DSM-5* requires at least six or more symptoms of inattention (e.g., careless mistakes, not listening, losing things, easily distracted) and six or more symptoms of hyperactivity or impulsiveness (e.g., fidgeting, running about when inappropriate, talking excessively, difficulty waiting) that last for at least six months and interfere with functioning or development. Several of these symptoms must be prior to age 12 and occur in multiple settings. Estimates of the prevalence of ADHD vary widely. The best available evidence for children in the United States is that 11 percent of boys and 4 percent of girls have the disorder (Bloom & Cohen, 2007).

**THE ETIOLOGY OF ADHD** The causes of this disorder are unknown. One of the difficulties in pinpointing the etiology is that ADHD is most likely a heterogeneous disorder. In other words, the behavioral profiles of children with ADHD vary, so the causes of the disorder most likely vary as well. Children with ADHD may be more likely than other children to come from disturbed families. Factors such as poor parenting and social disadvantage may contribute to the onset of symptoms,

### **attention-deficit/hyperactivity disorder (ADHD)**

A disorder characterized by restlessness, inattentiveness, and impulsivity.



**FIGURE 14.33**  
**ADHD and the Brain**

The brain image of a person with a history of ADHD shows less overall activation (at the red and white levels), especially in the frontal and limbic regions.

as is true for all psychological disorders. Still, ADHD clearly has a genetic component: Concordance is estimated at 55 percent in identical twins and 32 percent in dizygotic twins (Goodman & Stevenson, 1989; Sherman, McGue, & Iacono, 1997).

In an early imaging study, Alan Zametkin and colleagues (1990) found that adults who had been diagnosed with ADHD in childhood had reduced metabolism in brain regions involved in the self-regulation of motor functions and of attentional systems (**FIGURE 14.33**). These researchers theorized that the connection between the frontal lobes and the limbic system is impaired in ADHD patients. In fact, the symptoms of ADHD are similar to those seen in patients with frontal lobe damage: problems with planning, sustaining concentration, using feedback, and thinking flexibly. Other imaging studies have found that when adolescents with ADHD perform tasks that require them to inhibit motor responses, greater impairments in performance on the tasks are associated with abnormal activation of prefrontal regions (Schulz et al., 2004).

Researchers have also demonstrated differences in the basal ganglia in the brains of some ADHD patients (Aylward, Reiss, Reader, & Singer, 1996; Castellanos, Giedd, Eckberg, & Marsh, 1998; Fillipek et al., 1997). Because this structure is involved in regulating motor behavior and impulse control, dysfunction in the basal ganglia could contribute to the hyperactivity characteristic of ADHD.

**ADHD ACROSS THE LIFE SPAN** Children generally are not given diagnoses of ADHD until they enter structured settings in which they must conform to rules, get along with peers, and sit in their seats for long periods. In the past, these things happened when children entered school, between ages 5 and 7. Now, with the increasing prevalence of structured day care settings, the demands on children to conform are occurring much earlier.

According to longitudinal studies, children do not outgrow ADHD by the time they enter adulthood (McGough & Barkley, 2004). Adults with ADHD symptoms, about 4 percent of the population (Kessler et al., 2006), may struggle academically and vocationally. They generally reach a lower-than-expected socioeconomic level and change jobs more often than other adults (Bellak & Black, 1992; Mannuzza et al., 1991). At the same time, many adults with ADHD learn how to adapt to their condition, such as by reducing distractions while they work (**FIGURE 14.34**).



**FIGURE 14.34**  
**Living with ADHD**  
Paula Luper, of North Carolina, was diagnosed with ADHD in elementary school. Here, as a senior in high school, she is taking a quiz in the teachers lounge to avoid distraction.

## Summing Up

### Which Psychological Disorders Are Prominent in Childhood?

- Disorders in children are considered within the context of normal development.
- In some cases, psychological disorders identified in childhood have lasting impacts on the individual, and the problems apparent early in life continue throughout maturation.
- Autism spectrum disorder is characterized by impaired social interaction, deficits in communication, and restricted interests.
- Research suggests that autism is heritable.
- Causes of autism include gene mutations, unusual patterns of brain growth, exposure to unusual antibodies in the womb, faulty brain wiring, and impairments in mirror neuron activity.
- ADHD is characterized by inattentiveness, restlessness, and impulsivity.
- Environmental and genetic factors contribute to the development of ADHD.
- Abnormalities associated with the frontal lobes, limbic system, and basal ganglia have been identified in individuals with ADHD.

## Measuring Up

1. Identify whether each of the following characteristics is observed in children with autism spectrum disorder or observed in children with attention-deficit/hyperactivity disorder.
  - a. impulsivity and restlessness
  - b. restricted activities and interests
  - c. focus on objects rather than on people
  - d. easily distracted
  - e. failure to make and maintain eye contact
  
2. Compared with people without ADHD, people with ADHD show \_\_\_\_\_ activation in the \_\_\_\_\_ of the brain.
  - a. less; frontal lobes and limbic regions
  - b. less; temporal lobes and Broca's area
  - c. more; frontal lobes and limbic regions
  - d. more; temporal lobes and Broca's area

(2) a. less; frontal lobes and limbic regions.

**ANSWERS:** (1) a. ADHD; b. autism; c. autism; d. ADHD; e. autism.

# Your Chapter Review

## Chapter Summary

### 14.1 How Are Psychological Disorders Conceptualized and Classified?

- **Psychopathology Is Different from Everyday Problems:** Psychological disorders are common in all societies. Individuals with psychological disorders behave in ways that deviate from cultural norms and that are maladaptive.
- **Psychological Disorders Are Classified into Categories:** The *Diagnostic and Statistical Manual of Mental Disorders* is a system for diagnosing psychological disorders. The current version is *DSM-5*. Psychological disorders are often comorbid—that is, they occur together. Due to comorbidity, it has been proposed that all psychological disorders reflect a common factor, p. High scores on the p factor have been found to be associated with more-severe psychopathology. Rather than classifying disorders, the Research Domain Criteria (RDoC) method strives to understand the processes that give rise to disordered thoughts, emotions, and behaviors. The RDoC defines basic domains of functioning, such as attention and social communication, and considers them across multiple levels of analysis, from genes to brain systems to behavior.
- **Psychological Disorders Must Be Assessed:** Assessment is the process of examining a person's mental functions and psychological health to make a diagnosis. Assessment is accomplished through interviews, behavioral observations, psychological testing, and neuropsychological testing.
- **Psychological Disorders Have Many Causes:** According to the diathesis-stress model, mental health problems arise from a vulnerability coupled with a stressful precipitating event. Psychological disorders may arise from biological factors, psychological factors, or cognitive-behavioral factors. Females are more likely than males to exhibit internalizing disorders (such as major depressive disorder and generalized anxiety disorder). Males are more likely than females to exhibit externalizing disorders (such as alcohol use disorder and conduct disorders). Most psychological disorders show some universal symptoms, but the *DSM* recognizes a number of cultural syndromes related to mental health problems.

### 14.2 Which Disorders Emphasize Emotions or Moods?

- **Anxiety Disorders Make People Apprehensive and Tense:** Specific phobias are exaggerated fears of specific stimuli. Generalized anxiety disorder is diffuse and omnipresent. Social anxiety disorder is a fear of being negatively evaluated by others. Panic attacks cause sudden overwhelming terror and may lead to agoraphobia. Cognitive, situational, and biological factors contribute to the onset of anxiety disorders.
- **Unwanted Thoughts Create Anxiety in Obsessive-Compulsive Disorders:** Obsessive-compulsive disorder involves frequent intrusive thoughts and compulsive behaviors. OCD occurs in approximately

1–2 percent of the population and affects women more than men. OCD may involve learned behaviors or be caused by biological factors.

- **Posttraumatic Stress Disorder Results from Trauma:** Post-traumatic stress disorder involves frequent and recurring nightmares, intrusive thoughts, and flashbacks related to an earlier trauma. PTSD occurs in approximately 7 percent of the population and affects women more than men.
- **Depressive Disorders Consist of Sad, Empty, or Irritable Mood:** Major depressive disorder is characterized by a number of symptoms, including depressed mood and a loss of interest in pleasurable activities. Persistent depressive disorder is less severe, with people being sad on more days than not for at least two years.
- **Depressive Disorders Have Biological, Situational, and Cognitive Components:** Depressive disorders have biological components, including possible dysfunction of the monoamine neurotransmitters norepinephrine and serotonin, low left frontal lobe function, and disrupted biological rhythms. Situational factors (such as poor relationships and stress) and cognitive factors (such as the cognitive triad and learned helplessness) also contribute to the occurrence of depression.
- **Bipolar Disorders Involve Depression and Mania:** Bipolar disorder is characterized by depression and manic episodes—that is, episodes of increased activity and euphoria. The impairment in bipolar I disorder is due to manic episodes, whereas the impairment in bipolar II disorder is due to depressive episodes. Genes may play a role in bipolar disorders.

### 14.3 Which Disorders Emphasize Thought Disturbances?

- **Dissociative Disorders Are Disruptions in Memory, Awareness, and Identity:** Dissociative disorders involve disruptions of identity, memory, or conscious awareness. Dissociative amnesia involves forgetting that an event happened or losing awareness of a substantial block of time. Dissociative fugue involves a loss of identity. Dissociative identity disorder involves the occurrence of two or more distinct identities in the same individual, along with memory gaps for everyday events. Dissociative identity disorder is believed to emerge as a consequence of severe abuse—through repeated dissociation, different identities develop to cope with different traumas. Dissociative identity disorder remains a controversial diagnosis for two reasons: The condition is often diagnosed after someone has been accused of a crime, and a sharp rise in reported cases has occurred in recent years.
- **Schizophrenia Involves a Split Between Thought and Emotion:** Schizophrenia is characterized by a split between thought and emotion. The positive symptoms associated with schizophrenia reflect excesses and include delusions, hallucinations, disorganized speech, and disorganized behavior. The negative symptoms of schizophrenia reflect deficits and include apathy, lack of emotion, and slowed speech. Research suggests that schizophrenia is largely a biological disorder. Environmental factors also play a

role in the development of schizophrenia, including dysfunctional family dynamics, urban stress, and exposure to pathogens.

lobe functioning. Both genetics and environment seem to contribute to the development of antisocial personality disorder.

## 14.4 What Are Personality Disorders?

- **Personality Disorders Are Maladaptive Ways of Relating to the World:** The *DSM* identifies 10 personality disorders clustered in three groups. Paranoid, schizoid, and schizotypal make up the odd and eccentric cluster. Histrionic, narcissistic, borderline, and antisocial make up the dramatic, emotional, and erratic cluster. Avoidant, dependent, and obsessive-compulsive make up the anxious and fearful cluster.
- **Borderline Personality Disorder Is Associated with Poor Self-Control:** Borderline personality disorder involves disturbances in identity, affect, and impulse control. Borderline personality disorder is associated with reduced frontal lobe capacity, low levels of serotonin, and a history of trauma and abuse.
- **Antisocial Personality Disorder Is Associated with a Lack of Empathy:** Antisocial personality disorder is characterized by socially undesirable behavior, hedonism, sensation seeking, and a lack of remorse. Antisocial personality disorder is associated with lower levels of arousal, a smaller amygdala, and deficits in frontal

## 14.5 Which Psychological Disorders Are Prominent in Childhood?

- **Autism Spectrum Disorder Involves Social Deficits and Restricted Interests:** Autism spectrum disorder emerges in infancy and is marked by impaired social functioning and communication and restricted interests. Autism is heritable and may result from genetic mutations. Autism has been linked to abnormal brain growth, exposure to antibodies in the womb, faulty brain wiring, and mirror neuron impairment.
- **Attention-Deficit/Hyperactivity Disorder Is a Disruptive Impulse Control Disorder:** Children with ADHD are restless, inattentive, and impulsive. The causes of ADHD may include environmental factors such as poor parenting and social disadvantages; genetic factors; and brain abnormalities, particularly with regard to activation of the frontal lobes, limbic system, and basal ganglia. ADHD continues into adulthood, presenting challenges to academic work and to career pursuits.

## Key Terms

agoraphobia, p. 614	delusions, p. 629	negative symptoms, p. 631
antisocial personality disorder (APD), p. 639	diathesis-stress model, p. 606	obsessive-compulsive disorder (OCD), p. 616
anxiety disorder, p. 612	disorganized behavior, p. 631	panic disorder, p. 614
assessment, p. 605	disorganized speech, p. 630	persistent depressive disorder, p. 619
attention-deficit/hyperactivity disorder (ADHD), p. 647	dissociative disorders, p. 625	posttraumatic stress disorder (PTSD), p. 617
autism spectrum disorder, p. 643	dissociative identity disorder (DID), p. 626	psychopathology, p. 600
bipolar I disorder, p. 622	etiology, p. 600	Research Domain Criteria (RDoC), p. 604
bipolar II disorder, p. 622	family systems model, p. 607	schizophrenia, p. 628
borderline personality disorder, p. 637	generalized anxiety disorder (GAD), p. 613	sociocultural model, p. 607
cognitive-behavioral approach, p. 608	hallucinations, p. 630	
	learned helplessness, p. 621	
	major depressive disorder, p. 618	

## Practice Test

1. Which of the following questions would a clinician consider in order to determine whether a behavior represents psychopathology? Select all that apply.
    - a. Does the behavior deviate from cultural norms?
    - b. Is the behavior causing the individual personal distress?
    - c. Is the behavior maladaptive?
    - d. Is the behavior unusual?
    - e. Is the behavior upsetting to members of the client's social network?
  2. Two students visit the campus health center. Student A describes feeling constantly fearful and anxious. Student B describes feeling persistently agitated and often exhibiting violent outbursts. Student A's symptoms are consistent with an \_\_\_\_\_ disorder, which is more common in \_\_\_\_\_; student B's symptoms are consistent with an \_\_\_\_\_ disorder, which is more common in \_\_\_\_\_.
    - a. externalizing, females; internalizing, males
- b. externalizing, males; internalizing, females**
- c. internalizing, females; externalizing, males**
- d. internalizing, males; externalizing, females**
3. True or false: The *DSM-5* is proven to offer definitive and accurate diagnoses coupled with the best recommended treatment options for all accepted psychological disorders.
  4. Which of the following are examples of neuropsychological assessments?
    - a. the patient's self-report of symptoms
    - b. reports from interviews with people who know the patient well
    - c. blood tests
    - d. card-sorting tasks
    - e. having the person copy a picture by hand
    - f. having the person place blocks on a mat while blindfolded
    - g. having the person draw designs from memory

The answer key for the Practice Tests can be found at the back of the book.

# Beliefs About Self-Compassion: Implications for Coping and Self-Improvement

Personality and Social Psychology Bulletin  
I-16  
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DOI: 10.1177/0146167220965303  
[journals.sagepub.com/home/pspb](http://journals.sagepub.com/home/pspb)  


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## Abstract

Self-compassion—treating oneself with care and understanding during difficult times—promotes adaptive coping and self-improvement. Nonetheless, many people are not self-compassionate. We examined a key barrier people face to treating themselves self-compassionately: their negative beliefs about self-compassion (i.e., that it leads to complacency, indulgence, or irresponsibility). Across three studies, the more people held these negative beliefs, the less self-compassionately they reported responding to a real-world event (Study 2) and hypothetical emotional challenges (Studies 1 and 3). Self-compassionate responding, in turn, predicted adaptive coping strategies and intentions for self-improvement. Experimentally inducing people to hold positive, as opposed to negative, beliefs about self-compassion predicted self-compassionate responding 5 to 7 days later (Study 3). By recognizing and targeting peoples' beliefs, our findings highlight the importance of reducing such beliefs that are barriers to practicing self-compassion, as a means to improve the way people respond to difficult times.

## Keywords

self-compassion, mindset, lay theories, coping, self-improvement

Received September 19, 2019; revision accepted September 17, 2020

The “Golden Rule” prescribes that people should treat others how they want to be treated. However, this maxim is seldom accompanied by advice on how individuals should first treat themselves. This reflects an emphasis to be kind to others, which is not always extended to oneself (Neff, 2003b). Such self-compassion fundamentally involves giving oneself care and support in difficult times—much like we would extend to a loved one (Neff, 2011). This phenomenon is typically conceptualized as involving three key processes: (a) self-kindness, as opposed to self-criticism (i.e., offering words of comfort rather than berating); (b) mindfulness, as opposed to overidentification (i.e., observing emotions nonjudgmentally and openly, rather than dwelling on them); and (c) a sense of common humanity, as opposed to isolation (i.e., acknowledging that all humans are imperfect, rather than feeling alone in failings and suffering; Neff, 2003a, 2003b).

Although self-compassion confers numerous benefits, many people do not treat themselves self-compassionately when encountering challenges. For example, in a previous research, the majority of 391 undergraduates sampled reported treating others with more kindness than which they treated themselves (Neff, 2003b). One important, yet underresearched reason for this could be that some people hold negative beliefs about self-compassion, believing it leads to complacency, self-indulgence, or irresponsibility (e.g., Germer & Neff, 2019;

Gilbert et al., 2011; Neff & Germer, 2018). Such “negative self-compassion beliefs” hinder people from practicing self-compassion, especially when they might need it most (e.g., during emotionally challenging situations). In turn, this may affect how adaptively they cope with such situations and strive to improve themselves thereafter. In this article, our goal was to examine how these negative self-compassion beliefs relate to peoples’ practice of self-compassion, and in turn, adaptive coping and self-improvement outcomes.

## The Benefits of Self-Compassion

Self-compassion has consistently been linked with many benefits, including lower levels of anxiety and depression, greater psychological well-being, better physical health, and

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positive interpersonal relationships (Dunne et al., 2018; Hall et al., 2013; MacBeth & Gumley, 2012; Neff & Beretvas, 2013; Neff et al., 2007; Neff & McGehee, 2010; Yarnell & Neff, 2013; Zessin et al., 2015). People who are self-compassionate tend to experience these numerous benefits, in part, because self-compassion is associated with how adaptively people cope with a wide range of emotionally challenging events, including those that are not necessarily their fault (e.g., Germer & Neff, 2013; Leary et al., 2007; Neff, 2003a; Sirois et al., 2015). In addition, self-compassion relates to peoples' intentions to improve themselves or their situations following these difficulties (Breines & Chen, 2012; Leary et al., 2007).

Although self-compassion is associated with greater extroversion, agreeableness, conscientiousness, and most commonly with lower neuroticism (Arslan, 2016; Neff et al., 2007; Pfattheicher et al., 2017), research suggests that it can uniquely predict outcomes such as life satisfaction (Neff et al., 2018), psychological health (Neff et al., 2007), and constructive problem solving (Arslan, 2016) beyond these personality traits. Compared with neuroticism, self-compassion tends to focus more specifically on strategy use: Neuroticism captures individual differences in emotional reactivity more broadly, whereas self-compassion represents explicit "strategies for dealing with negative emotions and experiences" (Pfattheicher et al., 2017, p. 167)—such as showing oneself kindness and engaging in mindfulness. In our studies, we focused on testing how people's self-compassion relates to their specific coping strategies and self-improvement intentions.

**Self-Compassion and Coping.** Self-compassion tends to be positively related to adaptive coping strategies, such as acceptance (e.g., Sirois et al., 2015) and positive reframing (e.g., Neff et al., 2005; Sirois et al., 2015), although it is associated with fewer maladaptive avoidant coping strategies, such as denial, distraction, and behavioral disengagement (Neff et al., 2005; Sirois et al., 2015).

**Self-Compassion and Self-Improvement Intentions.** Self-compassion also plays an important role in peoples' intentions to improve themselves or the situation (i.e., their "self-improvement intentions") following emotional challenges. For example, students encouraged to reflect upon a setback with self-compassion (as opposed to self-enhancement) reported greater intentions to make amends after a wrongdoing (Breines & Chen, 2012), expressed greater willingness to accept personal responsibility for their role in a negative event (Leary et al., 2007), and elected to study longer after a difficult test (Breines & Chen, 2012).

These studies suggest that practicing self-compassion is associated with more adaptive coping and greater self-improvement intentions in response to emotional challenges. Consistent with these findings, we theorized that

self-compassion would be associated with these adaptive outcomes because it comprises mindfulness, self-kindness, and common humanity components. During emotionally challenging situations, when people are more mindfully aware of their thoughts and emotions (i.e., mindfulness), they would be more likely to select appropriate strategies to help themselves cope adaptively (as opposed to becoming overwhelmed by difficult emotions); when people view their circumstances from a place of acceptance, as opposed to judgment (i.e., self-kindness), they could be more willing to acknowledge areas for improvement, rather than resort to less helpful ways of coping, such as avoidance; and when people acknowledge that everyone makes mistakes and encounters emotional difficulties (i.e., common humanity), they may feel more empowered to face challenges head-on, rather than dwelling in self-pity. Therefore, by enabling people to mindfully perceive life's difficulties in a nonjudgmental, supportive manner, self-compassion could promote adaptive responses to emotional challenging situations.

### Barriers to Being Self-Compassionate

Despite the numerous benefits of self-compassion, many people face significant barriers to treating themselves self-compassionately. Some people, for instance, have fears about self-compassion, and therefore refrain from practicing self-compassion (Gilbert et al., 2014). Other people experience negative physiological responses to self-compassion-building exercises (Duarte et al., 2015; Rockliff et al., 2008). Another major barrier is peoples' "negative self-compassion beliefs"—or beliefs that self-compassion leads to less motivation, more self-indulgence, and less self-responsibility. Self-compassion researchers consider these beliefs to pose a major barrier toward practicing self-compassion (e.g., Germer & Neff, 2019; Gilbert et al., 2011; Neff & Germer, 2018).

For example, self-compassion interventions sometimes include, along with self-compassion skills training, educational material explaining how negative beliefs about self-compassion can, in fact, be misconceptions (e.g., Germer & Neff, 2019; Neff & Germer, 2018). In addition, one study found that participants who did not treat themselves with self-compassion were more likely to associate self-compassion with negative attributes, such as laziness and self-indulgence (Robinson et al., 2016). These studies suggest that negative self-compassion beliefs prevent people from treating themselves with compassion. However, to our knowledge, no empirical research has yet specifically isolated and tested the effects of such beliefs on peoples' practice of self-compassion and its downstream effects—the aim of our present article.

### Lay Theories

Lay theories are peoples' fundamental beliefs about themselves and the world (Dweck et al., 1995; Molden & Dweck,

2006). These beliefs about intelligence, empathy, personality, emotions, or other attributes predict whether people perceive and respond to challenges in adaptive or maladaptive ways (De Castella et al., 2018; Hong et al., 1999; Schumann et al., 2014; Yeager et al., 2013). For example, students who believed intelligence is malleable responded to difficulty and failure with greater mastery-oriented behaviors and persistence, compared with those who believed intelligence is fixed (Dweck, 2000; Hong et al., 1999; Mueller & Dweck, 1998). In studies of empathy, when participants were led to believe empathy can be developed (as opposed to a quality that's fixed), they were more likely to expend effort to empathize with others in challenging contexts (Schumann et al., 2014). To our knowledge, no existing studies have extended this framework to understanding—and changing—peoples' lay theories of self-compassion.

### Negative Beliefs about Self-Compassion

Likewise, people have beliefs about self-compassion—that self-compassion might lower motivation, lead to self-indulgence, or decrease a sense of responsibility (Germer & Neff, 2019; Neff & Germer, 2018). These negative self-compassion beliefs can be self-fulfilling because people who hold them may be more likely to avoid practicing self-compassion in emotionally challenging situations, which may undermine adaptive responding. In contrast, people who hold these negative beliefs to a lesser extent (or who have been primed in a way that reduces such beliefs) may be more inclined to practice self-compassion in difficult times, which in turn, should relate to more adaptive coping and greater self-improvement intentions. Therefore, we predicted and tested in our studies an indirect effect of negative self-compassion beliefs on adaptive coping and self-improvement intentions through peoples' reported practice of self-compassion.

### Overview

In three studies, we tested the hypothesis that participants' negative self-compassion beliefs would be associated with their practice of self-compassion, and in turn, their coping strategies and self-improvement intentions in response to emotionally difficult situations. In Study 1, we presented participants with three emotionally challenging scenarios and measured their negative self-compassion beliefs, as well as their reported intentions to practice self-compassion, coping strategies, and self-improvement intentions in response to these scenarios. In Study 2, we replicated our results in a real-world setting with Americans who felt disappointed by the 2016 U.S. Presidential election. In Study 3, we experimentally induced either positive or negative beliefs about self-compassion and examined their effects on participants' intentions to practice self-compassion in response to emotionally challenging scenarios 5 to 7 days afterward.

## Study 1: Negative Self-Compassion Beliefs Predict Responses to Challenging Events

In this study, we investigated whether peoples' self-compassion beliefs relate to how self-compassionately they respond in moments of adversity. We first presented participants with three hypothetical scenarios depicting emotionally challenging situations. We were interested in measuring participants' intentions to respond self-compassionately in these situations, utilize various adaptive or maladaptive coping strategies, and their self-improvement intentions. Then, we assessed participants' negative self-compassion beliefs. We tested the predictions that (a) the more people hold negative self-compassion beliefs, the less likely they would be to respond self-compassionately; (b) the greater their intentions to respond self-compassionately, the more likely they would report using adaptive coping strategies, the higher their self-improvement intentions, and the less likely they would report using maladaptive coping strategies; and (c) there would be an indirect effect of negative self-compassion beliefs on coping and self-improvement intentions through peoples' intentions to practice self-compassion in these situations.

### Participants

We conducted a power analysis for a bivariate correlation in G-POWER to determine a sufficient sample size using an alpha of .05, power of .80, and a medium effect size ( $r = .35$ ; Cohen, 1992). We aimed to detect a medium effect size, given the range of effect sizes found in past studies ( $rs = .17\text{--}.56$ ; Leary et al., 2007; Neff et al., 2005; Sirois et al., 2015) and financial cost considerations. Based on these assumptions, the desired sample size was 61. Therefore, we recruited 65 adults from Amazon Mechanical Turk for the study, an online crowdsourcing marketplace where people can be recruited virtually to complete surveys for pay (for more information about Mechanical Turk samples, see Paolacci & Chandler, 2014). Six people did not answer any questions after the consent form (which we had not anticipated), leaving a final sample of 59 participants (25 women, 34 men;  $M_{age} = 35.9$  years,  $SD_{age} = 11.2$  years; 68% White). We did not preregister this study or other studies within this article because we conducted them before preregistration became common practice.

### Procedure

We asked participants to imagine themselves in three emotionally challenging scenarios, each designed to elicit negative emotions, and to rate how they would respond to each of them separately (Leary et al., 2007). These scenarios asked participants to imagine (a) not studying sufficiently for an important test, resulting in them failing the test; (b) forgetting to call a grandparent back, only to learn afterward that

the grandparent had unexpectedly passed away; and (c) forgetting their lines in the middle of a performance, causing the production to abruptly halt (see Appendix A in the Supplemental Material). After each scenario, participants reported their intentions to respond self-compassionately, their intentions to use various kinds of coping strategies, and their self-improvement intentions. We additionally assessed social desirability in responding (to test how participants' responses to our negative self-compassion beliefs items might be related to concerns about social desirability), and participants' individual differences in self-compassion (to provide initial validation for their reported intentions to respond self-compassionately).

## Measures

**Emotional responses.** To check that our scenarios elicited negative emotions as intended, participants reported the extent to which they would feel 16 negative emotions (e.g., sad, angry, irritated) if the situation had *just* occurred, indicating responses on a scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). Averaging across the three scenarios, participants' ratings on emotion items were highly intercorrelated ( $rs > .51$ ,  $ps < .001$ ), and internal consistency of the scale as a whole was high ( $\alpha = .95$ ). Thus, we averaged items to create a composite "negative emotion" score.

**Responding self-compassionately to emotionally challenging scenarios.** To assess intentions to respond self-compassionately, participants completed an adapted version of the Self-Compassion Scale—Short Form (Raes et al., 2011), indicating responses on a scale ranging from 1 (*almost never*) to 5 (*almost always*). We adapted items that were originally worded to measure individual differences in self-compassion ("I try to be understanding and patient toward those aspects of my personality I don't like") to assess intentions to respond self-compassionately to a specific event ("I would try be understanding and patient toward those aspects of my personality I don't like"). Three subscales addressed the positive aspects of self-compassion: self-kindness, common humanity, and mindfulness. Three subscales addressed the negative counterparts of these aspects: self-judgment, isolation, and overidentification. Internal consistency of the scale was high across scenarios ( $\alpha = .89$ ), and participants' intentions to respond self-compassionately were highly correlated across all scenarios ( $rs > .62$ ,  $ps < .001$ ). Because we were interested in capturing how people would generally respond to these emotionally challenging events, we calculated a composite self-compassion score for each participant by averaging their intentions to respond self-compassionately across the three scenarios.

**Coping responses.** After reading each scenario, participants completed an adapted version of the Brief COPE (Carver,

1997) to rate how likely they would be to use various coping strategies in response to the event if it had *just* occurred, on a scale ranging from 1 (*I wouldn't do this at all*) to 4 (*I would do this a lot*). We adapted items that were originally worded to measure how people have generally coped in the past (e.g., "I've been getting help and advice from other people") to measure how participants predicted they would cope with the hypothetical events (e.g., "I would get help and advice from other people").

We grouped participants' ratings on these coping questions into three subscales: (a) emotion-focused coping (venting, positive reframing, humor, acceptance, and emotional support scales;  $\alpha = .87$  averaged across scenarios), (b) avoidant coping (self-distraction, denial, behavioral disengagement, self-blame, and substance use scales;  $\alpha = .71$  averaged across scenarios), and (c) problem-focused coping (active coping, planning, instrumental support, and religion scales;  $\alpha = .89$  averaged across scenarios). These categorizations are consistent with past research (Schnidler et al., 2007), and with theoretical and empirical distinctions between various types of coping strategies (Carver & Scheier, 1994; Folkman & Lazarus, 1985; Holahan & Moos, 1987; McWilliams et al., 2003; Zeidner, 1995).<sup>1</sup>

**Self-improvement intentions.** We were interested not only in how participants coped with the events but also in their inclination to improve themselves and the situation afterward. To assess self-improvement intentions, we created three face-valid items to measure the three core beliefs that people could have about self-compassion—namely, that it negatively affects motivation ("I would be motivated to fix the situation"), self-discipline ("I would try to become more self-disciplined"), and self-responsibility ("I would take personal responsibility for the situation"). Such single-item measures can be valid when they are straightforward to understand (Abdel-Khalek, 2006; Cheung & Lucas, 2014; Konrath et al., 2018). Participants indicated responses on a scale ranging from 1 (*extremely unlikely*) to 5 (*extremely likely*). We averaged items to create a "self-improvement intention" score and averaged scores across scenarios ( $\alpha = .85$ ).

**Negative self-compassion beliefs.** Drawing from prior work (Gilbert et al., 2011; Neff & Germer, 2013), we operationalized the three key negative self-compassion beliefs: the belief that self-compassion leads to (a) complacency (e.g., "I will become complacent if I accept my imperfections completely"), (b) self-indulgence (e.g., "If I'm kind toward my flaws, I won't have the discipline needed to succeed"), and (c) less self-responsibility (e.g., "I'll take less responsibility for my shortcomings if I don't constantly criticize myself"). Participants answered 10 items about their negative self-compassion beliefs (see Appendix B in the Supplemental Material for the item wording) using a 1 (*strongly disagree*) to 5 (*strongly agree*) scale ( $\alpha = .94$ ). High scores reflect the endorsement of more negative self-compassion beliefs. For

further details about item generation and selection, and the psychometric properties of our negative self-compassion beliefs measure, please refer to Appendices C and D in the Supplemental Material. Supplemental Table S1 presents means and standard deviations for each negative self-compassion beliefs item.

**Social desirability.** To assess whether desires to respond in a socially desirable way may have influenced participants' responses about their negative self-compassion beliefs, participants completed the 13-item Marlowe–Crowne Social Desirability Scale—Short Form C (M-C Form C; Crowne & Marlowe, 1960). Participants indicated either true or false in response to statements describing culturally approved, yet highly improbable, behaviors (e.g., "No matter who I'm talking to, I'm always a good listener"); scale internal consistency:  $\alpha = .85$ .

**Individual differences in self-compassion.** To validate our adapted self-compassionate responses measure, participants completed the 26-item Self-Compassion Scale (SCS; Neff, 2003), by indicating their responses on a scale ranging from 1 (*almost never*) to 5 (*almost always*). Internal consistency of the scale was high ( $\alpha = .94$ ).

## Results

We first report the results from our checks that (a) the three scenarios elicited negative emotions, (b) negative self-compassion beliefs were uncorrelated with social desirability, and (c) our adapted self-compassion measure was positively correlated with the original SCS (Neff, 2003). Afterward, we present the results of our hypothesis testing. Supplemental Table S2 presents correlations among all study measures.

**Scenarios elicited negative emotions.** Each of the three hypothetical scenarios were generally effective at eliciting negative emotions (Scenario 1:  $M = 3.49$ ,  $SD = 0.83$ ; Scenario 2:  $M = 3.15$ ,  $SD = 0.90$ ; Scenario 3:  $M = 3.64$ ,  $SD = 0.77$ ). Because each scenario elicited the same pattern of self-compassion and coping responses, we averaged across all scenarios in subsequent analyses. A one-sample  $t$  test indicated that participants' mean score on the negative emotion items, averaged across scenarios ( $M = 3.43$ ,  $SD = 0.70$ ), significantly differed from the midpoint of the scale,  $t(58) = 4.70$ ,  $p < .001$ , 95% confidence interval (CI) = [0.25, 0.61].

**Self-reported beliefs were not driven by social desirability concerns.** Participants' reported negative self-compassion beliefs were not significantly correlated with social desirability concerns ( $r = -.22$ ,  $p = .10$ ), suggesting that social desirability concerns were unlikely to have driven participants' reported self-compassion beliefs.

**Validation of the self-compassionate responses measure.** Our adapted self-compassionate responses measure was highly

correlated with the original self-compassion measure ( $r = .92$ ,  $p < .001$ ), providing preliminary evidence of its construct validity. Because we were primarily interested in how participants reacted to the scenarios, and given that results using both our adapted measure and the original self-compassion measure were the same, we focused on analyzing participants' reported intentions to practice self-compassion in context (instead of their individual differences in self-compassion) in this and following studies. Next, we present the results of our hypothesis testing.

**Negative self-compassion beliefs predicted reported practice of self-compassion.** Consistent with our hypothesis, negative self-compassion beliefs were negatively associated with intentions to respond self-compassionately ( $r = -.46$ ,  $p < .001$ )—the more strongly participants endorsed negative self-compassion beliefs, the lower their intentions to respond to the scenarios with self-compassion,  $B = -0.31$ , 95% CI = [-0.47, -0.15],  $SE = 0.08$ ,  $t(57) = -3.90$ ,  $p < .001$ . Notably, the magnitude of the correlation between negative self-compassion beliefs and intentions to respond self-compassionately was not so high as to suggest they are the same construct.

**Self-compassion predicted self-reported adaptive coping and self-improvement intentions.** Self-compassion predicted the adaptive coping strategies and self-improvement intentions that participants reported they would employ in response to the hypothetical negative events (see Table 1). Participants' intentions to respond self-compassionately was associated with more emotion-focused coping, more problem-focused coping, less avoidant coping, and greater self-improvement intentions.

**Indirect effects.** To test our prediction that negative self-compassion beliefs would relate to participants' coping and self-improvement intentions through their intentions to practice self-compassion, we conducted an indirect effects analysis using PROCESS with a bootstrap of 10,000 resamples (Preacher & Hayes, 2004). Supporting our hypothesis, we found significant indirect effects of negative self-compassion beliefs on emotion-focused coping, problem-focused coping, and self-improvement intentions, mediated by self-compassion. The indirect effect was not significant for avoidant coping, which trended in the predicted direction, but did not reach statistical significance. Consistent with recommended best practices (Preacher & Hayes, 2008), we report the bootstrapped 95% CIs (instead of  $p$  values) of the indirect effect estimates across all studies. Table 2 summarizes indirect effect coefficients and Figure 1 illustrates the indirect effects for problem-focused coping as an example.

## Discussion

Participants who endorsed more negative self-compassion beliefs reported lower intentions to respond to emotionally challenging scenarios with self-compassion. The lower their

**Table 1.** Study 1 Regression Coefficients of the Relationships Between Participants' Intentions to Practice Self-Compassion and Their Negative Self-Compassion Beliefs, Their Responses to the Scenarios (Coping Strategies and Self-Improvement Intentions).

Responses to scenarios	Intentions to practice self-compassion						Negative self-compassion beliefs					
	95% CI			SE	t	p	95% CI			SE	t	p
	B	LL	UL				B	LL	UL			
Emotion focused	0.40	0.22	0.57	0.09	4.56	<.001	-0.12	-0.25	0.02	0.07	-1.76	.08
Problem focused	0.42	0.21	0.64	0.11	3.91	<.001	-0.13	-0.29	0.03	0.08	-1.57	.12
Avoidant focused	-0.18	-0.31	-0.06	0.06	-2.87	.006	0.10	0.02	0.19	0.04	2.39	.02
Self-improvement	0.43	0.22	0.63	0.10	4.12	<.001	-0.18	-0.33	-0.03	0.08	-2.44	.02

Note. N = 59. Higher scores indicate a greater endorsement of negative self-compassion beliefs. CI = confidence interval; LL = lower limit; UL = upper limit.

**Table 2.** Study 1 Regression Coefficients for Tests of the Indirect Effect of Negative Self-Compassion Beliefs (IV) on Responses to Scenarios (DV), Mediated by Self-Compassion (M).

Effects on scenario responses	B	95% CI			t	p
		LL	UL	SE		
<b>Emotion-focused coping (DV)</b>						
Total effect	-0.12	-0.25	0.02	0.07	-1.76	.08
M → DV (controlling for X)	0.40	0.20	0.60	0.10	4.07	<.001
Direct effect	0.01	-0.13	0.14	0.07	0.10	.92
Indirect effect	-0.12	-0.22	-0.05	0.05		
<b>Problem-focused coping (DV)</b>						
Total effect	-0.13	-0.29	0.03	0.08	-1.57	.12
M → DV (controlling for X)	0.43	0.18	0.67	0.12	3.48	.001
Direct effect	0.01	-0.16	0.17	0.08	0.07	.94
Indirect effect	-0.13	-0.25	-0.04	0.05		
<b>Avoidant coping (DV)</b>						
Total effect	0.10	0.02	0.19	0.04	2.39	.02
M → DV (controlling for X)	-0.14	-0.28	0.00	0.07	-1.98	.05
Direct effect	0.06	-0.04	0.16	0.05	1.26	.21
Indirect effect	0.04	-0.02	0.11	0.03		
<b>Self-improvement intentions (DV)</b>						
Total effect	-0.18	-0.33	-0.03	0.08	-2.44	.02
M → DV (controlling for X)	0.38	0.15	0.61	0.12	3.27	.002
Direct effect	-0.07	-0.22	0.09	0.08	-0.84	.41
Indirect effect	-0.12	-0.26	-0.02	0.06		

Note. N = 59. Direct effect: X → DV (controlling for M). IV = independent variable; DV = dependent variable; M = intentions to respond with self-compassion; CI = confidence interval; LL = lower limit; UL = upper limit; X = negative beliefs about self-compassion (higher scores indicate greater endorsement of negative self-compassion beliefs).

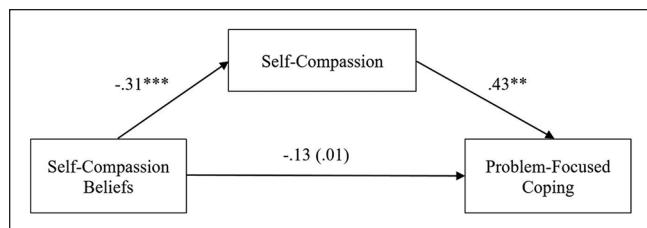
intentions to respond self-compassionately, the less likely participants were to report using adaptive coping strategies and the lower their self-improvement intentions. These results support the idea that negative self-compassion beliefs can pose barriers toward practicing self-compassion in times of adversity (Neff, 2003a, 2003b; Neff & Dahm, 2015), and that holding negative beliefs is associated with how adaptively people respond to challenging times.

Although we found support for both of our primary hypotheses, how participants reacted to our hypothetical scenarios may not generalize to how they respond to real-world

events. Study 2 addressed this by examining how people responded to one important event that affected the lives of many Americans—the 2016 U.S. presidential election.

## Study 2: Negative Self-Compassion Beliefs Predict Responses to a Real-World Emotionally Challenging Event

In this study, we examined the role of peoples' beliefs about self-compassion in response to a political event of great importance to the American people: Donald Trump's



**Figure 1.** Mediation of the effect of self-compassion beliefs on problem-focused coping by self-compassion in Study 1.

Note. Unstandardized regression coefficients are presented. The unstandardized regression coefficient between self-compassion beliefs and problem-focused coping, when controlling for people's intentions to respond with self-compassion, is in parentheses. Mediation by self-compassion for the outcomes of emotion-focused coping, avoidant coping, and self-improvement intentions can be similarly represented.  
 $^{**}p < .01$ .  $^{***}p < .001$ .

inauguration as the President of the United States. Many Americans experienced emotional reactions following the election. For example, in a November 2016 survey of 1,254 U.S. residents, 53% reported feeling uneasy, 41% sad, 41% scared, and 31% angry in response to the election of Trump (Pew Research Center, 2016). Many Americans continued to find the political climate stressful following President Trump's inauguration: In a January 2017 survey of 1,019 U.S. residents, 57% reported that the current political climate was a very or somewhat significant source of stress and 49% reported that the election outcome was a very or somewhat significant source of stress (American Psychological Association, 2016). Given the negative emotional response many Americans had in response to the 2016 U.S. presidential election, we found this a suitable context to study how people reacted to a real-world emotionally challenging event.

In Study 2, we tested the same hypotheses among individuals who experienced the inauguration as a negative event, specifically that (a) the more strongly they endorsed negative self-compassion beliefs, the less they would report practicing self-compassion in response to the inauguration; (b) the less they reported practicing self-compassion, the less they would report using adaptive coping strategies, the more they would report using maladaptive coping strategies, and the lower their self-improvement intentions in response to the event; and (c) there would be an indirect effect of negative self-compassion beliefs on these coping and self-improvement intentions, mediated by self-compassion.

## Participants

In this field study, we planned as a conservative estimate to detect a smaller effect size than in Study 1. We conducted a power analysis in G-POWER to determine a sufficient sample size for a bivariate correlation using an alpha of .05, power of .80, and a small–medium effect size ( $r = .20$ ; Cohen, 1992). Based on these assumptions, the desired sample size was 193.

**Screening survey.** We first screened for people who had experienced the political changes in America as an emotionally negative event, for recruitment in our survey. Forty-four days after the inauguration of Trump, we invited 529 people to complete a screening survey to ensure that we would have enough participants who met our criteria. We told participants the survey was part of a study about goal achievement. This survey included our main measures assessing peoples' political beliefs (i.e., how disappointed they felt with the unfolding policies, their voting decision, and their political views), along with filler questions about their self-reported goal achievement.

**Main survey.** Aiming for a final sample of 193, we sent out 285 study invitations 50 days after the inauguration. We sent invitations to all people who had reported feeling extremely dissatisfied ( $n = 201$ ), dissatisfied ( $n = 75$ ), or somewhat dissatisfied ( $n = 33$ ) with the unfolding policies. On Amazon Mechanical Turk, 213 participants completed the study between 50 and 53 days after Trump's inauguration (i.e., a response rate of 75%). We decided a priori to exclude participants who failed an attention check question because inattention can affect data quality and statistical power (e.g., Maniaci & Rogge, 2014). Hence, we excluded 23 participants, leaving a final sample of 186 participants (98 men, 85 women, two nonbinary;  $M_{age} = 36.6$  years,  $SD_{age} = 11.2$  years; 75% White). Forty-five participants reported not voting, nine reported voting for Donald Trump (the Republican candidate), 108 for Hillary Clinton (the Democratic candidate), eight for Gary Johnson (the Libertarian candidate), seven for Jill Stein (the Green Party candidate), four for another third-party candidate, and five did not disclose their voting decision. Fifty-three participants identified as "very liberal," 78 as "liberal," 35 as "moderate," 18 as "conservative," and two as "very conservative."

## Procedure

In our main survey, participants reported their opinions on the Trump Administration's policies, as well as their reported practice of self-compassion, emotional responses, coping strategies, and self-improvement intentions in response to American politics. Afterward, participants reported their negative self-compassion beliefs and their individual differences in self-compassion.

**Policy opinions.** To make opinions on political events salient during survey completion, participants first indicated the extent to which they agreed with 12 of the Trump Administration's unfolding policies on a scale of 1 (*strongly disagree*) to 7 (*strongly agree*). Participants had the option of selecting *unsure/don't know*. We included policies ranging from more controversial (e.g., repealing and replacing the Affordable Care Act) to less controversial (e.g., withdrawing from the Trans-Pacific Partnership).

**Emotional responses.** We measured participants' negative emotions, as a check that they indeed felt negatively toward American politics following Trump's inauguration. Participants indicated the degree to which they felt nine emotions (e.g., sad, angry, embarrassed) over the past 2 weeks about American politics on a scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). These negative emotion items were interspersed among filler questions about experiences of positive emotions. We again averaged the negative emotion items to create a composite negative emotion scale ( $\alpha = .89$ ).

**Coping responses.** Participants reported how they coped with American politics over the past 3 days by completing an adapted version of the Brief COPE (Carver, 1997), described in Study 1. To keep the survey length reasonable, we excluded subscales that had not correlated with self-compassion intentions in Study 1 (humor, self-distraction, denial, and substance abuse), with the exception of the venting subscale, which has previously been associated with individual differences in self-compassion (Neff et al., 2005). Again, we grouped coping strategies into three types: emotion-focused coping ( $\alpha = .76$ ), avoidant coping ( $\alpha = .75$ ), and problem-focused coping ( $\alpha = .84$ ).

**Reported practice of self-compassion.** Participants reported how self-compassionately they responded over the past 3 days, similar to the adapted measure described in Study 1 (adapted from Raes et al., 2011). However, rather than frame items in the hypothetical as we did with the adapted items in Study 1 (e.g., "I would be intolerant and impatient toward those aspects of my personality I don't like"), we framed these items to measure how in-the-moment responses (e.g., "I was intolerant and impatient toward those aspects of my personality I didn't like";  $\alpha = .77$ ).

**Self-improvement intentions.** We assessed components of participants' self-improvement intentions, namely, their motivation ("I feel motivated to help promote a positive political climate" and "I will engage actively in politics"), self-discipline ("I am trying to become more self-disciplined"), and self-responsibility ("I am taking personal responsibility for America's current political climate" and "I will invest time and energy into promoting a positive political climate") on a scale ranging from 1 (Strongly disagree) to 7 (Strongly agree). We again averaged items to create a "self-improvement intentions" scale ( $\alpha = .89$ ).

**Negative self-compassion beliefs.** Participants reported their negative self-compassion beliefs using the same 10 items described in Study 1 ( $\alpha = .94$ ). For additional psychometric properties of the items, see Appendices C and D in the Supplemental Material.

**Individual differences in self-compassion.** Participants completed the 26-item SCS to validate our adapted measure. Internal reliability of the scale was high ( $\alpha = .95$ ).

## Results

Supplemental Table S3 presents correlations among all study measures.

**Political event elicited negative emotions among sample.** As expected, participants rated that the political event generally elicited negative emotions ( $M = 3.18$ ,  $SD = 0.93$ ). A one-sample  $t$  test indicated that mean scores on the negative emotion items significantly differed from the midpoint of the scale, 95% CI of the mean difference from midpoint = [0.05, 0.32],  $t(185) = 2.69$ ,  $p = .008$ .

**Negative self-compassion beliefs predicted the reported practice of self-compassion.** Negative self-compassion beliefs were moderately negatively correlated with reported practice of self-compassion ( $r = -.37$ ,  $p < .001$ ). Consistent with Study 1, the more strongly participants endorsed negative self-compassion beliefs, the less they reported practicing self-compassion in response to the negative political event,  $B = -0.23$ ,  $[-0.31, -0.14]$ ,  $SE = 0.04$ ,  $t(184) = -5.44$ ,  $p < .001$ .

**Self-compassion predicted self-reported adaptive coping and self-improvement intentions.** Participants with greater self-compassionate responding reported using more adaptive strategies (emotion-focused coping, problem-focused coping), and fewer maladaptive coping strategies (avoidant coping). Participants with greater self-compassionate responding also reported greater self-improvement intentions (see Table 3).

**Indirect effects.** Next, we tested the predicted indirect effect of negative self-compassion beliefs on coping strategies and self-improvement intentions, through self-compassion. As hypothesized, all indirect effects were significant (see Table 4). The effect of participants' self-compassion beliefs on emotion-focused coping, problem-focused coping, avoidant coping, and self-improvement intentions were significantly mediated by their reported practice of self-compassion. Figure 2 represents the mediation results for problem-focused coping as an example.

## Discussion

Replicating Study 1's findings in a real-world political context, we found in Study 2 that the stronger participants' negative self-compassion beliefs, the less self-compassion they reported when coping with disappointment following the American political election. In turn, the less people reported practicing self-compassion, the fewer adaptive coping strategies and more maladaptive coping strategies they reported using, and the lower their self-improvement intentions following the election.

These correlational findings lent support for our theorized model but are insufficient to infer causality. Therefore, our next study experimentally manipulated participants' beliefs about self-compassion and measured how likely they

**Table 3.** Study 2 Regression Coefficients of the Relationship Between Participants' Reported Practice of Self-Compassion and Their Negative Self-Compassion Beliefs, and Their Responses to Political Dissatisfaction (Including Coping, and Self-Improvement Intentions).

Responses to political event	Self-compassion						Negative self-compassion beliefs					
	95% CI						95% CI					
	B	LL	UL	SE	t	p	B	LL	UL	SE	t	p
Emotion focused	0.22	.09	0.35	0.07	3.33	.001	0.03	-0.05	0.12	0.04	0.83	.41
Problem focused	0.16	0.01	0.31	0.08	2.16	.03	0.09	0.00	0.18	0.05	1.90	.06
Avoidant focused	-0.57	-0.71	-0.43	0.07	-7.92	<.001	0.28	0.19	0.37	0.05	6.10	<.001
Self-improvement	0.38	0.06	0.71	0.16	2.33	.02	0.09	-0.11	0.28	0.10	0.85	.40

Note. N = 186. Higher scores indicate a greater endorsement of negative self-compassion beliefs. CI = confidence interval; LL = lower limit; UL = upper limit.

**Table 4.** Study 2 Regression Coefficients for Tests of the Indirect Effect of Negative Self-Compassion Beliefs (IV) on Responses to Political Disappointment (DV), Mediated by Self-Compassion (M).

Effects on responses to political event	B	95% CI				
		LL	UL	SE	t	p
<b>Emotion-focused coping (DV)</b>						
Total effect	.03	-.05	.12	.04	0.83	.41
M → DV (controlling for X)	.28	.14	.42	.07	3.96	<.001
Direct effect	.10	.01	.18	.04	2.27	.02
Indirect effect	-.06	-.11	-.03	.02		
<b>Problem-focused coping</b>						
Total effect	.09	.00	.17	.05	1.90	.06
M → DV (controlling for X)	.25	.09	.40	.08	3.14	.002
Direct effect	.14	.05	.24	.05	2.97	.003
Indirect effect	-.06	-.11	-.02	.02		
<b>Avoidant coping</b>						
Total effect	.28	.19	.37	.05	6.10	<.001
M → DV (controlling for X)	-.46	-.61	-.31	.07	-6.17	<.001
Direct effect	.18	.09	.27	.05	3.90	<.001
Indirect effect	.10	.06	.16	.03		
<b>Self-improvement intentions</b>						
Total effect	.08	-.11	.28	.10	0.85	.40
M → DV (controlling for X)	.51	.16	.85	.18	2.88	.005
Direct effect	.20	-.01	.41	.11	1.87	.06
Indirect effect	-.11	-.20	-.03	.04		

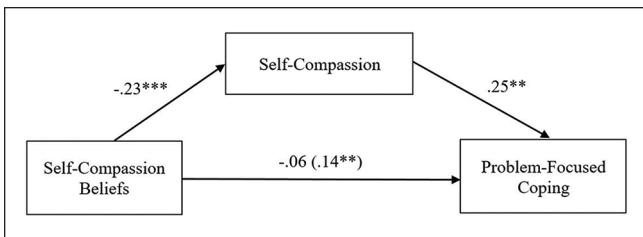
Note. N = 186. Direct effect: X → DV (controlling for M). IV = independent variable; DV = dependent variable; M = self-compassion; CI = confidence interval; LL = lower limit; UL = upper limit; X = negative beliefs about self-compassion (higher scores indicate a greater endorsement of negative self-compassion beliefs).

were to report responding self-compassionately to emotionally adverse events.

### Study 3: Causal Effects of Beliefs About Self-Compassion

Study 3 employed a two-wave design to investigate whether experimentally manipulating beliefs about self-compassion would causally affect participants' intentions to practice self-compassion in response to emotional challenges, which in turn, would be related to their coping strategies and

self-improvement intentions. At Time 1, we manipulated participants' beliefs about self-compassion; at Time 2, 5 to 7 days later, participants indicated how they would respond to three emotionally challenging scenarios (that were the same as those used in Study 1; Appendix A in the Supplemental Material). We hypothesized that promoting positive, as opposed to negative, self-compassion beliefs, would increase participants' intentions to practice self-compassion in response to emotionally challenging scenarios presented 5 to 7 days later. We again expected that intentions to be self-compassionate would relate to participants' use of more adaptive



**Figure 2.** Mediation of the effect of self-compassion beliefs on problem-focused coping by self-compassion in Study 2.

Note. Unstandardized regression coefficients are presented. The unstandardized regression coefficient between self-compassion beliefs and problem-focused coping, when controlling for reported practice of self-compassion, is in parentheses. Mediation by reported practice of self-compassion for the outcomes of emotion-focused coping, avoidant coping, and self-improvement intentions can be similarly represented.

\*\* $p < .01$ . \*\*\* $p < .001$ .

coping strategies (emotion-focused coping and problem-focused coping), fewer maladaptive coping strategies (avoidant coping), and greater self-improvement intentions. Finally, we tested the expected indirect effects described in earlier studies. Our random assignment controlled for individual differences in self-compassion and other factors that may explain the relationship between negative self-compassion beliefs and participants' reported intentions to practice self-compassion in the previous correlational studies.

## Participants

We expected a medium to large effect size of our experimental manipulation, because past lay theories research using scientific articles or videos to experimentally induce different beliefs have generally found medium to large differences between conditions (e.g., Aronson et al., 2001; Hong et al., 1999; Schuman et al., 2014). Power analysis using G-POWER with an alpha of .05 and specified power of .80 showed that a sample size of 90 would enable us to detect a predicted medium to large effect size of our manipulation ( $d = .60$ ; Cohen, 1992).

Aiming for a final sample of 90, and taking attrition between Parts 1 and 2 of the study (corresponding to Time 1 and Time 2, respectively) into account, we recruited 121 participants (50 women, 71 men;  $M_{age} = 32.9$  years,  $SD_{age} = 9.9$  years, 65% White) from Amazon Mechanical Turk for Part 1. Eighty-eight of these participants (73% of the original sample) also completed Part 2 of the study. Their demographic information did not significantly differ from those who completed only Part 1 (33 women, 55 men;  $M_{age} = 34.0$  years,  $SD_{age} = 10.5$  years, 73% White).

## Time 1 Premanipulation Measures

We invited people to participate in a study on "goal achievement." To support this cover story, we included filler questions

about goal achievement. Before receiving the manipulation, participants completed the 26-item SCS (Neff, 2003;  $\alpha = .95$ ), to assess whether our random assignment to condition worked.

## Self-Compassion Beliefs Manipulation

We randomly assigned participants to read one of two fictitious *Psychology Today* articles—a method researchers have successfully used to change peoples' beliefs (Chiu et al., 1997; Schumann et al., 2014).

Both articles detailed ways in which people could respond to challenges so as to better achieve their goals. In the "positive self-compassion beliefs" condition, the article explained how self-compassion facilitates personal growth and goal achievement, for example,

When we relate to ourselves in a self-compassionate way, it's safe for us to acknowledge our shortcomings and face the truth about ourselves . . . [Next time I make a mistake, I'll] remember that being understanding of my slip-ups . . . pays dividends. (See Appendix E in the Supplemental Material)

In the "negative self-compassion beliefs" condition, the article explained how self-compassion hinders personal growth and goal achievement, for example,

When we relate to ourselves in a self-compassionate way, it's difficult for us to acknowledge our shortcomings and face the truth about ourselves . . . [Next time I make a mistake, I'll] remember that being critical of my slip-ups . . . pays dividends. (See Appendix F in the Supplemental Material)

As a manipulation check, participants reported their negative self-compassion beliefs using the same items as in earlier studies ( $\alpha = .93$ ).

## Time 2 Postmanipulation Measures

Five days later, participants received a link to a survey where they responded to the three emotionally challenging scenarios used in Study 1 by reporting their emotions, intentions to practice self-compassion, intended coping strategies, and self-improvement intentions. Participants had up to 2 days to complete this survey.

**Emotional responses.** Participants indicated the degree to which they would feel nine negative emotions (e.g., sad, anxious, angry) if the scenario had just occurred to them, on a scale ranging from 1 (*very slightly or not at all*) to 5 (*extremely*). We averaged them into a composite score of negative emotions ( $\alpha = .94$ ).

**Responding self-compassionately to emotionally challenging scenarios.** We used our same self-compassion measure described in Study 1 ( $\alpha$  averaged across scenarios = .91).

**Coping responses.** We used the same coping measures described in Study 2 (as for each of the three coping types, averaged across scenarios,  $\geq .66$ ).

**Self-improvement intentions.** Participants completed the same items described in Study 1 ( $\alpha = .85$ ).

## Results

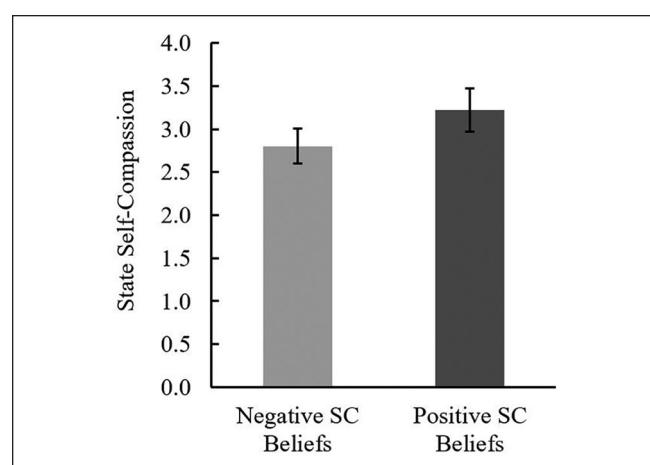
Supplemental Table S4 presents correlations among all study measures.

**Negative emotions check.** On average, participants rated that each of the three scenarios presented elicited negative emotions (Scenario 1:  $M = 3.71$ ,  $SD = 1.04$ ; Scenario 2:  $M = 3.59$ ,  $SD = 0.89$ ; Scenario 3:  $M = 3.56$ ,  $SD = 0.95$ ). We again averaged negative emotions across scenarios. A one-sample  $t$  test indicated that mean scores on the negative emotion items, averaged across scenarios ( $M = 3.62$ ,  $SD = 0.79$ ), were significantly different than the scale's midpoint,  $CI = [0.45, 0.79]$ ,  $t(87) = 7.31$ ,  $p < .001$ .

**Time 1 premanipulation measures.** Results showed that our randomization was effective: Participants' scores on the pre-manipulation survey measures, including their individual differences in self-compassion and demographics, did not differ by conditions (all  $ps > .40$ ).

**Manipulation checks.** Our articles successfully influenced participants' Time 1 beliefs about self-compassion: Participants randomly assigned to the negative self-compassion beliefs condition endorsed negative self-compassion beliefs more ( $M = 3.35$ ,  $SD = 0.84$ ;  $N = 61$ ) than those assigned to the positive self-compassion beliefs condition,  $M = 2.61$ ,  $SD = 0.99$ ;  $N = 60$ ; 95% CI of the mean difference = [41, 1.07],  $t(119) = 4.45$ ,  $p < .001$ ,  $d = 0.81$ .

**Promoting positive self-compassion beliefs increased self-compassionate responding.** Importantly, our findings showed that promoting positive self-compassion beliefs significantly increased participants' intentions to practice self-compassion in response to the emotionally challenging scenarios, 5 to 7 days later (see Figure 3). Participants in the positive self-compassion beliefs condition reported that they would respond to the scenarios with greater self-compassion ( $M = 3.22$ ,  $SD = 0.81$ ;  $N = 39$ ) than those in the negative self-compassion beliefs condition,  $M = 2.80$ ,  $SD = 0.73$ ;  $N = 49$ ; 95% CI of the mean difference = [-0.75, -0.10],  $t(86) = -2.57$ ,  $p = .01$ ,  $d = 0.54$ . The manipulation influenced participants' intentions to respond self-compassionately to the scenarios,  $B = 0.42$ ,  $[0.10, 0.75]$ ,  $SE = 0.16$ ,  $t(86) = 2.57$ ,  $p = .01$ , even when controlling for their individual differences in self-compassion ( $B = 0.31$ ,  $[0.08, 0.54]$ ,  $SE = 0.12$ ,  $t(85) = 2.66$ ,  $p = .01$ ).



**Figure 3.** Reported intentions to practice self-compassion by condition.

Note. Error bars depict  $\pm 1$  SE. SC = self-compassion.

**Self-compassion predicted self-reported coping and self-improvement intentions.** As hypothesized, self-compassion was associated with participants' coping strategies and self-improvement intentions in response to the scenarios. Participants with greater self-compassionate responding at Time 2 reported using more emotion-focused coping ( $B = 0.12$ ,  $[0.01, 0.24]$ ,  $SE = 0.06$ ,  $t(86) = 2.17$ ,  $p = .03$ ), more problem-focused coping ( $B = 0.17$ ,  $[0.03, 0.30]$ ,  $SE = 0.07$ ,  $t(86) = 2.45$ ,  $p = .02$ ), and less avoidant coping ( $B = -0.45$ ,  $[-0.55, -0.34]$ ,  $SE = 0.05$ ,  $t(86) = -8.46$ ,  $p < .001$ ). Reported intentions to practice self-compassion were also related to greater self-improvement intentions, but this relationship did not reach statistical significance ( $B = 0.15$ ,  $[-0.02, 0.33]$ ,  $SE = 0.09$ ,  $t(86) = 1.79$ ,  $p = .08$ ).

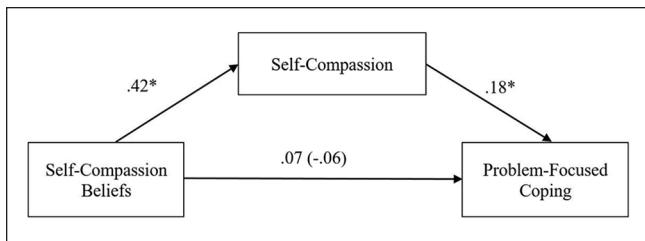
**Indirect effects.** We tested the hypothesized indirect effects (regression coefficients summarized in Table 5). As aforementioned, participants in the positive self-compassion beliefs condition reported greater intentions to respond to the scenarios with self-compassion than those in the negative self-compassion beliefs condition. Controlling for condition, greater reported practice of self-compassion predicted greater reported use of adaptive emotion-focused and problem-focused coping strategies, and less use of maladaptive avoidant coping strategies. However, contrary to our hypothesis, self-compassion did not significantly predict greater self-improvement intentions when controlling for condition.

Results demonstrated significant indirect effects of condition on emotion-focused coping, problem-focused coping, and avoidant coping through self-compassion as a mediator. The indirect effect on self-improvement intentions trended in the predicted direction but was not statistically significant. Figure 4 illustrates mediation results for problem-focused coping, as an example.

**Table 5.** Study 3 Regression Coefficients for Tests of the Indirect Effect of Self-Compassion Beliefs Condition (IV) on Scenario Responses (DV), Mediated by Self-Compassion (M).

Effects on scenario responses	<i>B</i>	95% CI		<i>t</i>	<i>p</i>
		LL	UL		
<b>Emotion-focused coping (DV)</b>					
Total effect	-0.01	-0.20	0.17	0.09	-0.15 .88
M → DV (controlling for X)	0.13	0.02	0.25	0.06	2.28 .02
Direct effect	-0.07	-0.25	0.11	0.09	-0.76 .45
Indirect effect	0.06	0.002	0.15	0.04	
<b>Avoidant coping (DV)</b>					
Total effect	-0.29	-0.50	-0.07	0.11	-2.65 .01
M → DV (controlling for X)	-0.43	-0.54	-0.32	0.05	-7.85 <.001
Direct effect	-0.11	-0.28	0.07	0.09	-1.23 .22
Indirect effect	-0.18	-0.32	-0.04	0.07	
<b>Problem-focused coping (DV)</b>					
Total effect	0.01	-0.21	0.23	0.11	0.10 .92
M → DV (controlling for X)	0.18	0.04	0.32	0.07	2.51 .01
Direct effect	-0.06	-0.29	0.16	0.11	-0.57 .57
Indirect effect	0.07	0.000	0.20	0.06	
<b>Self-improvement intentions (DV)</b>					
Total effect	0.13	-0.14	0.40	0.14	0.93 .35
M → DV (controlling for X)	0.14	-0.04	0.32	0.09	1.59 .12
Direct effect	0.07	-0.21	0.35	0.14	0.48 .63
Indirect effect	0.06	-0.02	0.18	0.05	

Note. *N* = 88. Direct effect: X → DV (controlling for M). IV = independent variable; DV = dependent variable; M = intentions to respond with self-compassion; CI = confidence interval; LL = lower limit; UL = upper limit; X = beliefs about self-compassion condition (0 = “negative self-compassion beliefs” condition, I = “positive self-compassion beliefs” condition).



**Figure 4.** Mediation of the effect of self-compassion beliefs manipulation (0 = “negative self-compassion beliefs” condition, I = “positive self-compassion beliefs” condition) on problem-focused coping by self-compassion in Study 3.

Note. Unstandardized regression coefficients are presented. The unstandardized regression coefficient between self-compassion beliefs and problem-focused coping, when controlling for people’s intentions to respond with self-compassion, is in parentheses. Mediation by self-compassion for the outcomes of emotion-focused coping, avoidant coping and self-improvement intentions can be similarly represented.

\**p* < .05.

## Discussion

Our Study 3 results found evidence supporting a causal relationship between peoples’ beliefs about self-compassion and their intentions to respond self-compassionately. In turn, the higher people’s intentions to practice self-compassion, the greater their reported use of emotion-focused coping

strategies, and the lower their reported use of maladaptive coping strategies. As before, we found indirect effects of peoples’ self-compassion beliefs on these coping patterns, mediated by their self-compassion. Unlike our previous correlational studies, there was a weak relation between reported intentions to practice self-compassion and self-improvement intentions here; hence, there was only a small and nonsignificant indirect effect for this outcome. Future studies should replicate this effect with larger samples and stronger belief manipulations. Overall, this study provides empirical evidence that (a) it is possible to change peoples’ beliefs about self-compassion with a brief belief induction article, with effects lasting over a period of 5 to 7 days and (b) changing peoples’ beliefs about self-compassion can potentially improve how they respond to emotional challenges.

## General Discussion

Self-compassion carries a range of psychological, physical, and interpersonal benefits (e.g., Hall et al., 2013; MacBeth & Gumley, 2012; Yarnell & Neff, 2013), yet many people do not treat themselves self-compassionately (Neff, 2003b). Our studies empirically examined the implications of an important barrier to developing self-compassion: peoples’ negative self-compassion beliefs. Across three studies, we found that people with stronger negative self-compassion

beliefs reported practicing less self-compassion in response to a real-world event and hypothetical scenarios. In turn, the less people reported practicing self-compassion, the less they reported using adaptive coping strategies, the more they reported using maladaptive coping strategies, and the lower their self-improvement intentions. Exposing people to a brief online message can change their beliefs about self-compassion, causally influencing their intentions to practice self-compassion—and reap its benefits—5 to 7 days later.

### Implications

Prior research has suggested that negative beliefs and emotions about self-compassion can be self-fulfilling, by reducing peoples' likelihood of practicing self-compassion (Gilbert et al., 2011; Robinson et al., 2016). Therefore, earlier self-compassion interventions have often included, along with skills training, educational content explaining how negative self-compassion beliefs are often misconceptions (e.g., Germer & Neff, 2019; Neff & Germer, 2018). However, it had yet to be empirically established whether these beliefs in and of themselves causally affect peoples' self-compassionate responding. Our work extends prior research by specifically isolating these negative self-compassion beliefs—and demonstrating that these beliefs, on their own, are associated with peoples' intentions to respond self-compassionately to hypothetical scenarios (Studies 1 and 3) and also their actual practice of self-compassion during real-world emotional challenges (Study 2). Moreover, the good news, as our results show, is that changing peoples' beliefs about self-compassion increases their intentions to practice self-compassion during difficult times, and in turn that self-compassion is associated with better coping and (to a smaller extent) greater self-improvement intentions. Therefore, our work contributes by showing that providing psychoeducation around negative self-compassion beliefs is itself an important, active treatment component in self-compassion interventions.

Our findings also suggest practical implications for intervening on self-compassion. Typically, self-compassion interventions take place in-person, over multiple sessions, and are delivered by highly specialized practitioners (e.g., Germer & Neff, 2013; Gilbert, 2010; Kirby et al., 2017). Although generally effective, these interventions are costly and dependent on specialized providers, of which there is often a shortage. Our results demonstrate that a brief online message can increase reported intentions to practice self-compassion—highlighting the promise of briefer, technology-delivered interventions (e.g., Mantelou & Karakasidou, 2017; Mitchell et al., 2018; Smeets et al., 2014). Such approaches are scalable, with the potential to reach a diverse sample of people who might otherwise not have access to specialized services. Yet, there is not enough research on the efficacy of brief, scalable online interventions to improve self-compassionate responding. Our work contributes practically by demonstrating one possible method (a brief online article educating people about self-compassion) that effectively raised self-compassion in a randomized, controlled experiment.

In addition, our research contributes to the rich literature on lay theories by extending it, for the first time, to the domain of self-compassion. We introduce beliefs about self-compassion as important lay theories that affect how people cope with adversity. To our knowledge, these studies are the first to demonstrate the causal effects of peoples' lay theories about self-compassion on their reported practice of self-compassion and its outcomes. This extends lay theory research, which has primarily focused on motivation and performance-related outcomes, such as persistence, and cognitive performance, and academic grades (Blackwell et al., 2007; Dweck & Yeager, 2019; Hong et al., 1999), and to a lesser extent, emotion regulation (e.g., Romero et al., 2014; Schumann et al., 2014; Tamir et al., 2007). Our studies underscore that people also hold beliefs about self-compassion, and that changing these beliefs fundamentally influences how they approach negative life events.

### Limitations and Future Directions

Our sample sizes, particularly in Studies 1 and 3, were modest. To supplement this, we conducted an internal meta-analysis of our results across our studies, which showed that our hypothesized effects were robust (see Appendix G in the Supplemental Material).

The main goal of our studies was to explicate how negative self-compassion beliefs affect the practice of self-compassion, as well as coping and self-improvement outcomes. Our negative self-compassion beliefs measure was an operationalization of this key construct, which was inspired by prior theories about self-compassion beliefs (e.g., Neff & Germer, 2013). Importantly, this operationalization tracked the psychological process we theorized. Future work can build upon our studies to refine and replicate these promising albeit preliminary psychometric properties of the negative self-compassion beliefs measure (see Appendices C and D in the Supplemental Material), and to compare the predictive value of this measure against potentially related constructs, such as fears of self-compassion (Gilbert et al., 2011). It is plausible that the fears of SCS, which emphasizes *affective* responses to self-compassion (including feelings of sadness, loss, emptiness, and fear; Gilbert et al., 2011), would be related to, yet theoretically and empirically distinguishable from, a measure of peoples' cognitive lay beliefs about the effectiveness of self-compassion.

Because we were interested in assessing (and changing) peoples' immediate reactions to hypothetical and real-life events, it was necessary to adapt the well-established Self-Compassion Scale—Short Form (Raes et al., 2011) into a measure that would capture peoples' practice of self-compassion in response to a specific situation. Supporting its construct validity, our self-compassion measure correlated highly with this individual difference measure (Study 1  $r = .92$ , Study 2  $r = .75$ , Study 3  $r = .72$ ). Hence, our self-compassion measure appeared to be appropriate for measuring self-compassionate responding in a specific context. Future

research can build upon our current measure to further develop and validate it, such as by testing its discriminant validity with related constructs, including neuroticism (Pfattheicher et al., 2017), or adding reverse-scored items.

To begin examining the effects of negative self-compassion beliefs, we primarily utilized self-report measures—a method consistent with prior literature that typically employs self-report methods to assess self-compassion, coping, and self-improvement intentions (Breines & Chen, 2012; Leary et al., 2007; Neff et al., 2005; Sirois et al., 2015; Zhang & Chen, 2016). Future studies could utilize behavioral outcome measures of self-compassion where appropriate. Relatedly, Studies 1 and 3 relied on hypothetical scenarios, which we complemented with a real-world Study 2 of the U.S. Presidential elections. For external validity, new studies could extend our work to a wider array of naturalistic settings.

Our findings provide a start to showing that it is possible to change peoples' beliefs about self-compassion and that these effects can last a week. There is a need for more longitudinal studies to track how long these effects persist, and further intervention research to continue refining the current manipulations into interventions that can create long-lasting change in peoples' beliefs about self-compassion.

## Conclusion

Self-compassion plays a powerful role in shaping how adaptively people respond to life's difficulties, however peoples' negative beliefs about self-compassion can pose obstacles to practicing it. Our research suggests that psychoeducation to correct these beliefs can be an important, active component of self-compassion interventions (Germer & Neff, 2019; Neff & Germer, 2018). These findings provide a stepping stone to what we hope will be more research on self-compassion beliefs and their implications. Such research could enable researchers and practitioners to more effectively promote self-compassion, especially among people who might otherwise face psychological barriers toward practicing it.

## Authors' Note

All data and materials have been made publicly available via the Open Science Framework and can be accessed at [https://osf.io/bxt4p/?view\\_only=f48d5de91a9346a286bc559ba4a07570](https://osf.io/bxt4p/?view_only=f48d5de91a9346a286bc559ba4a07570).

## Acknowledgments

The authors thank Mary Rutherford-Chwyl and Edward Chwyl for their input on initial survey items, and Shannon Smith for her editorial assistance.

## Author Contributions

C.C. designed the studies with guidance from P.C. and J.Z. C.C. collected the data. C.C. analyzed the data in consultation with P.C. C.C. and P.C. wrote the article, and J.Z. provided critical feedback.

## Declaration of Conflicting Interests

The author(s) declared no potential conflicts of interest with respect to the research, authorship, and/or publication of this article.

## Funding

The author(s) received no financial support for the research, authorship, and/or publication of this article.

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## Supplemental Material

Supplemental material is available online with this article.

## Note

1. Although one self-blame item in the Brief COPE avoidant coping subscale ("I've been criticizing myself") has theoretical overlap with the self-criticism dimension of self-compassion, this does not affect our interpretations of the relation between self-compassion and coping. First, removing this item did not change results—there was still a significant negative association between avoidant coping and self-compassion across studies (Study 1:  $B = -0.15, p = .03$ ; Study 2:  $B = -0.49, p < .001$ ; Study 3:  $B = -0.37, p < .001$ ). Second, self-compassion was associated with other forms of coping that did not have conceptual overlap with self-compassion.

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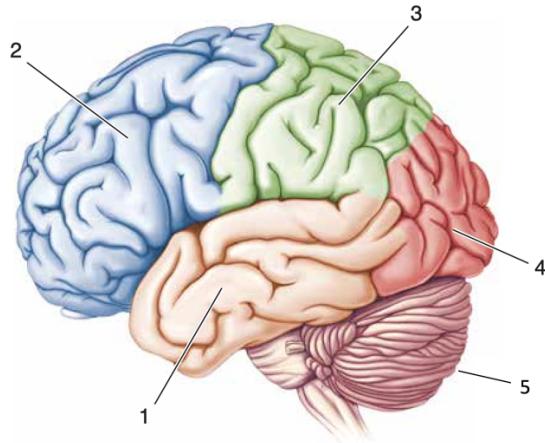
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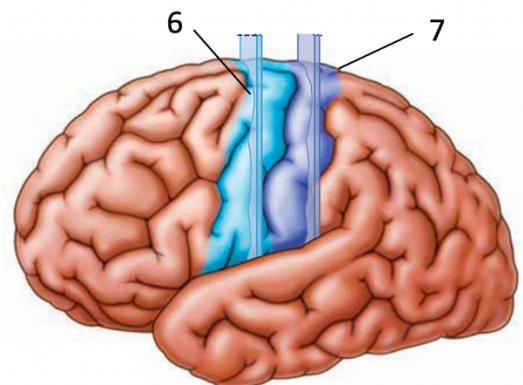
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## Quiz 2. Neuroanatomy

1. Fill in the appropriate names in the spaces provided.



1. \_\_\_\_\_  
2. \_\_\_\_\_  
3. \_\_\_\_\_  
4. \_\_\_\_\_  
5. \_\_\_\_\_



6. \_\_\_\_\_  
7. \_\_\_\_\_

2. Match each of the following brain structures with its role or function. (You will need to remember these terms and facts to understand later discussions of learning, memory, emotions, mental illness, anxiety, and other aspects of mind and behavior.)

- |       |                    |  |
|-------|--------------------|--|
| _____ | a. Corpus callosum | 1. primary structure for memory                      |
| _____ | b. Thalamus        | 2. important for emotions                            |
| _____ | c. Brainstem       | 3. secretes hormone that control pituitary function. |
| _____ | d. Amygdala        | 4. important for vision                              |
| _____ | e. Cerebellum      | 5. Involved in reward                                |
| _____ | f. Hypothalamus    | 6. involved in movement                              |
| _____ | g. Occipital lobe  | 7. Important for the basic functions of survival     |

- 
- 
- h. Basal ganglia
  - i. Hippocampus
  - 8. Projects sensory information to the cortex
  - 9. It connects the left and right hemispheres

3. Use one or two sentences to answer the following questions.

(1) What is cerebellum? What is the function of cerebellum?

(2) Where is Broca's area? What is Broca's area important for?

(3) What is phenomenology? Is it scientific?

# 3

# Biology and Behavior

## Ask & Answer

**3.1** How Does the Nervous System Operate? 76

**3.2** What Are the Basic Brain Structures and Their Functions? 89

**3.3** How Does the Brain Communicate with the Body? 104

**3.4** How Does the Brain Change? 109

**3.5** What Is the Genetic Basis of Psychological Science? 115

**IN 2012, JACK OSBOURNE, THE SON OF OZZY AND SHARON OSBOURNE,** was 26 years old (**FIGURE 3.1**). Two weeks after the birth of his daughter Pearl, he noticed a disturbing problem with his vision. He told *People* magazine (July 9, 2012) about an experience he had at a gas station: “I was talking to the attendant, and all of a sudden a black dot appeared in my vision . . . I was like, ‘That’s weird.’ The next day I woke up and the dot had turned into a cigar shape.” Eventually his vision deteriorated to the point that Jack could barely see out of his right eye. After a series of tests, physicians determined that Jack was in the early stages of multiple sclerosis (MS). On his mother’s television talk show *The Talk* (June 21, 2012), Osbourne said: “I guess I’ve been having symptoms for the last three or four years, but I didn’t realize it. . . . I had problems with my bladder, problems with my stomach, and then, about two years ago, my legs went numb for two months, and I just thought I had pinched a nerve.”

Multiple sclerosis is a disorder of the nervous system that is typically diagnosed between the ages of 20 and 40. It affects the brain and spinal cord, so that movements become jerky and victims lose the ability to coordinate their actions. Movement, coordination, vision, and cognition gradually deteriorate until they become severely impaired. MS affects about 2.5 million people throughout the world. The exact cause has not been identified, but research indicates that genetics and environment are important contributing factors. Although MS is incurable, symptoms are now manageable in some forms of the disease.



**FIGURE 3.1**  
**Multiple Sclerosis as a Disorder of the Nervous System**

Jack Osbourne is one of millions of people with multiple sclerosis. This disease damages nerve cells in the brain.

### neurons

The basic units of the nervous system; cells that receive, integrate, and transmit information in the nervous system. They operate through electrical impulses, communicate with other neurons through chemical signals, and form neural networks.

### central nervous system (CNS)

The brain and the spinal cord.

## Learning Objectives

- Distinguish between the two basic divisions of the nervous system.
- Distinguish between the functions of distinct types of neurons.
- Describe the structure of the neuron.
- Describe the electrical and chemical changes that occur when neurons communicate.
- Identify the major neurotransmitters and their primary functions.

Looking closely at multiple sclerosis helps us understand how the nervous system is critical in our ability to think and behave normally. In MS, damage to nerve cells limits their ability to send signals to other nerve cells and to receive signals from other nerve cells. To picture how a nerve cell communicates, imagine the plastic around a wire such as the cord for a lamp. Like the lamp cord, one part of the nerve cell is covered. The cord is covered not by plastic but by a fatty layer, which helps the cell to transmit signals to other nerve cells and other parts of the body. In MS, the fatty layer deteriorates, short-circuiting normal communication between nerve cells. And normal communication between nerve cells makes all thought, feeling, and behavior possible.

So, to know what makes us who we are, we need to understand how the nervous system works. We need to understand physiological processes and the genetic underpinnings of those processes. We also need to understand how aspects of our biology interact with our environments: How does nurture influence nature, and how does nature influence nurture?

As technology has advanced over the past three decades, researchers have learned a great deal about the biological basis of brain activity. Brain imaging techniques have shed light on the functions of different brain regions. Genetic analysis has revealed how certain disorders are passed from one generation to the next, made it possible to predict who will develop specific disorders, and helped identify the functions of specific genes related to psychological processes. You are about to learn how psychological activity is related to several aspects of biology, including genes, the endocrine system, and the nervous system.

## 3.1 How Does the Nervous System Operate?

The nervous system is responsible for everything people think, feel, or do. Essentially, each of us *is* a nervous system. The basic units of this system are the nerve cells, called **neurons** (**FIGURE 3.2**). These cells receive, integrate, and transmit information in the nervous system. Complex networks of neurons sending and receiving signals are the functional basis of all psychological activity. Although the actions of single neurons are simple to describe, human complexity results from billions of neurons. Each neuron makes contact with tens of thousands of other neurons. Neurons do not communicate randomly or arbitrarily, however. They communicate selectively with other neurons to form circuits, or *neural networks*. These networks develop through maturation and experience and repeated firing. In other words, permanent alliances form among groups of neurons.

### The Nervous System Has Two Basic Divisions

Neural networks are linked, and together they form the nervous system. The entire nervous system is divided into two basic units: the central nervous system and the

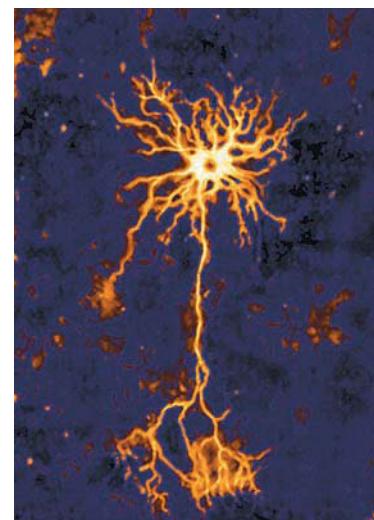
peripheral nervous system. The **central nervous system (CNS)** consists of the brain and the spinal cord, both of which contain massive numbers of neurons (**FIGURE 3.3**). The **peripheral nervous system (PNS)** consists of all the other nerve cells in the rest of the body. The CNS and PNS are anatomically separate, but their functions are highly interdependent. The PNS sends a variety of information to the CNS. The CNS organizes and evaluates that information and then directs the PNS to perform specific behaviors or make bodily adjustments.

As discussed more fully later in this chapter, the PNS includes the somatic and autonomic nervous systems. The somatic component of the PNS is involved in voluntary behavior, such as when you reach for an object to see how it feels. The autonomic component of the PNS is responsible for the less voluntary actions of your body, such as controlling heart rate and other bodily functions.

## Neurons Are Specialized for Communication

Neurons are specialized for communication. That is, unlike other cells in the body, nerve cells are excitable: They are powered by electrical impulses and communicate with other nerve cells through chemical signals. During the reception phase, neurons take in the chemical signals from neighboring neurons. During integration, incoming signals are assessed. During transmission, they pass their own signals to yet other receiving neurons.

**TYPES OF NEURONS** The three basic types of neurons are sensory neurons, motor neurons, and interneurons (**FIGURE 3.4**). **Sensory neurons** detect information from the physical world and pass that information along to the brain, usually through the spinal cord. To get a sense of how fast that process can work, think of the last time you touched something hot or accidentally pricked yourself with a sharp object, such as a tack. Those signals triggered your body's nearly instantaneous response and sensory



**FIGURE 3.2**  
**Human Neuron**

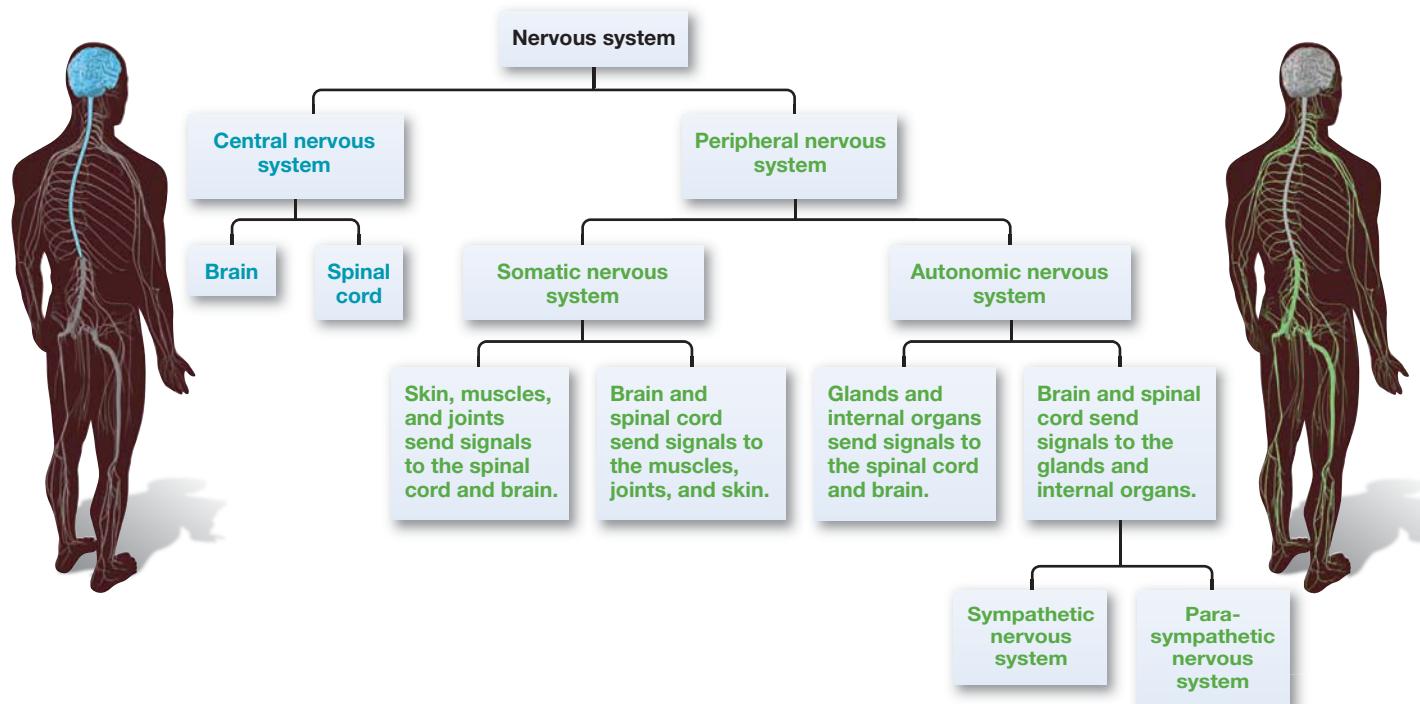
Neurons like this one are the basic units of the human nervous system.

### peripheral nervous system (PNS)

All nerve cells in the body that are not part of the central nervous system. The peripheral nervous system includes the somatic and autonomic nervous systems.

#### sensory neurons

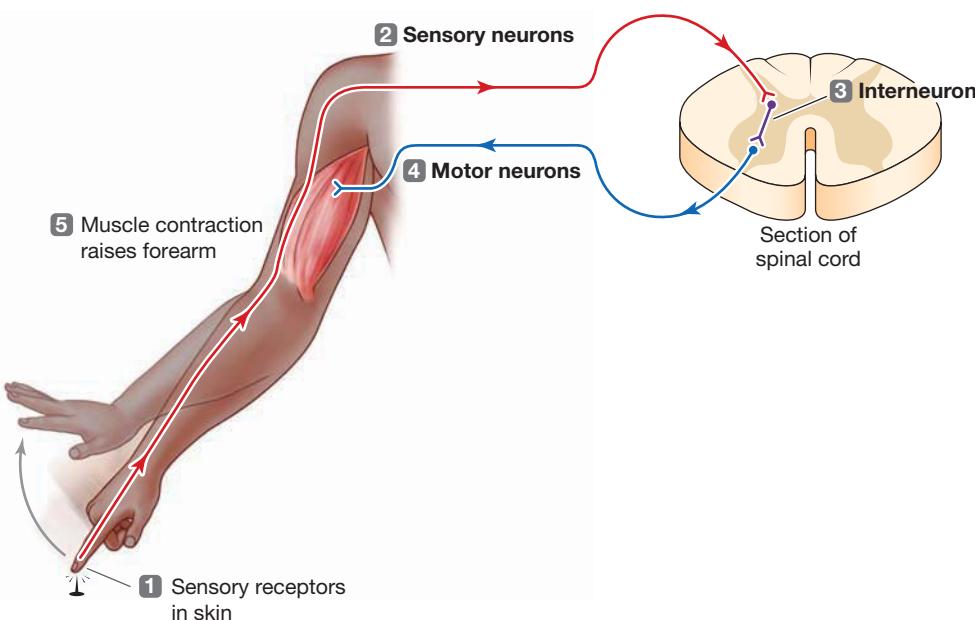
One of the three types of neurons; these neurons detect information from the physical world and pass that information to the brain.



**FIGURE 3.3** The Basic Divisions of the Nervous System

**FIGURE 3.4****The Three Types of Neurons**

**(Red line)** Receptors send signals to the brain for processing. Those signals travel through sensory neurons and the spinal cord. **(Blue line)** To produce a response, a signal is sent from the brain to the body through the spinal cord and motor neurons.



experience of the impact. The sensory nerves that provide information from the skin and muscles are called *somatosensory nerves*. (This term comes from the Greek for “body sense.” It means sensations experienced from within the body.)

**Motor neurons** direct muscles to contract or relax, thereby producing movement. **Interneurons** communicate within local or short-distance circuits. That is, interneurons integrate neural activity within a single area rather than transmitting information to other brain structures or to the body organs.

Sensory and motor neurons work together to control movement. For instance, if you are using a pen to take notes as you read these words, you are contracting and relaxing your hand muscles and finger muscles to adjust your fingers’ pressure on the pen. When you want to use the pen, your brain sends a message via motor neurons to your finger muscles so they move in specific ways. Receptors in both your skin and your muscles send back messages through sensory neurons to help determine how much pressure is needed to hold the pen. This symphony of neural communication for a task as simple as using a pen is remarkable, yet most of us employ motor control so easily that we rarely think about it. In fact, our *reflexes*, automatic motor responses, occur before we even think about those responses. For each reflex action, a handful of neurons simply convert sensation into action.

**motor neurons**

One of the three types of neurons; these neurons direct muscles to contract or relax, thereby producing movement.

**interneurons**

One of the three types of neurons; these neurons communicate within local or short-distance circuits.

**dendrites**

Branchlike extensions of the neuron that detect information from other neurons.

**cell body**

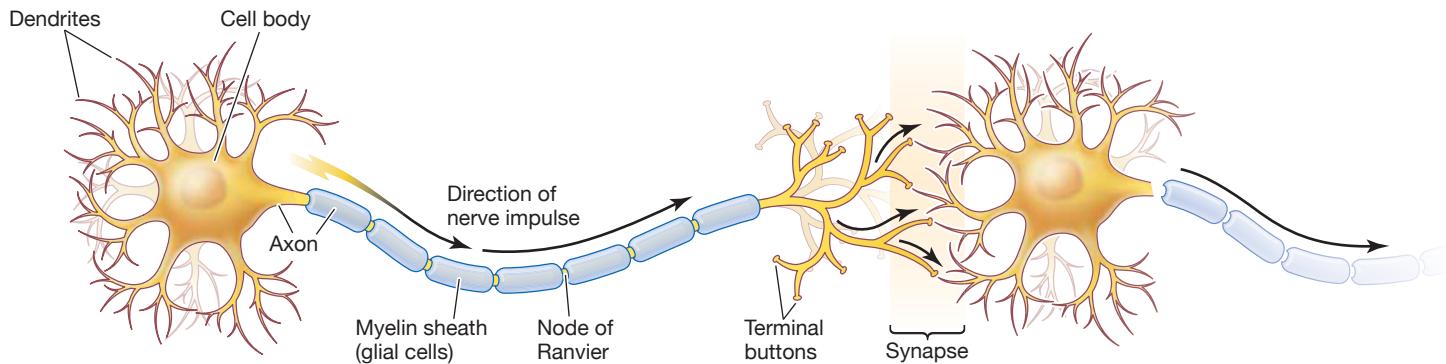
The site in the neuron where information from thousands of other neurons is collected and integrated.

**axon**

A long narrow outgrowth of a neuron by which information is transmitted to other neurons.

**NEURON STRUCTURE** In addition to performing different functions, neurons have a wide assortment of shapes and sizes. A typical neuron has four structural regions that participate in communication functions: the dendrites, the cell body, the axon, and the terminal buttons (**FIGURE 3.5**). The **dendrites** are short, branchlike appendages that detect chemical signals from neighboring neurons. In the **cell body**, also known as the *soma* (Greek for “body”), the information received via the dendrites from thousands of other neurons is collected and integrated.

Once the incoming information from many other neurons has been integrated in the cell body, electrical impulses are transmitted along a long, narrow outgrowth known as the **axon**. Axons vary tremendously in length, from a few millimeters to more than a meter. The longest axons stretch from the spinal cord to the big toe. You have heard the term *nerve*, as in Jack Osbourne’s reference to a “pinched nerve.” In



**FIGURE 3.5**  
**Neuron Structure**

Messages are received by the dendrites, processed in the cell body, transmitted along the axon, and sent to other neurons via chemical substances released from the terminal buttons across the synapse. (The myelin sheath, glial cells, and the nodes of Ranvier are discussed on pp. 81-82.)

in this context, a nerve is a bundle of axons that carry information between the brain and other specific locations in the body. At the end of the axon are knoblike structures called **terminal buttons**.

The site where chemical communication occurs between neurons is called the **synapse**. Neurons communicate by sending chemicals into the synapse, a tiny gap between the axon of the “sending” neuron and the dendrites of the “receiving” neurons. Chemicals leave one neuron, cross the synapse, and pass signals along to other neurons’ dendrites.

The neuron is covered with a *membrane*, a fatty barrier that does not dissolve in the watery environment inside and outside the neuron. The membrane is semipermeable. In other words, some substances move in or out of the membrane, and some do not. Located on the membrane are *ion channels*. These specialized pores allow *ions* to pass in and out of the cell when the neuron transmits signals down the axon. Ions are molecules, some charged negatively and some charged positively. By controlling the movement of ions, the membrane plays an important role in communication between neurons: It regulates the concentration of electrically charged molecules that are the basis of the neuron’s electrical activity.

## The Resting Membrane Potential Is Negatively Charged

When a neuron is resting, not active, the electric charge inside and outside the membrane is different. This difference is the **resting membrane potential**. The difference in the electrical charge occurs because the ratio of negative to positive ions is greater inside the neuron than outside it. Therefore, the electrical charge inside the neuron is slightly more negative than the electrical charge outside—typically  $-70$  millivolts (about  $\frac{1}{20}$  the charge of a AA battery). When a neuron has more negative ions inside than outside, the neuron is described as being *polarized*. The polarized state of the resting neuron creates the electrical energy necessary to power the firing of the neuron.

**THE ROLES OF SODIUM AND POTASSIUM IONS** Two types of ions that contribute to a neuron’s resting membrane potential are *sodium ions* and *potassium ions*. Although other ions are involved in neural activity, sodium and potassium are most important for this discussion.

### terminal buttons

At the ends of axons, small nodules that release chemical signals from the neuron into the synapse.

### synapse

The gap between the axon of a “sending” neuron and the dendrites of a “receiving” neuron; the site at which chemical communication occurs between neurons.

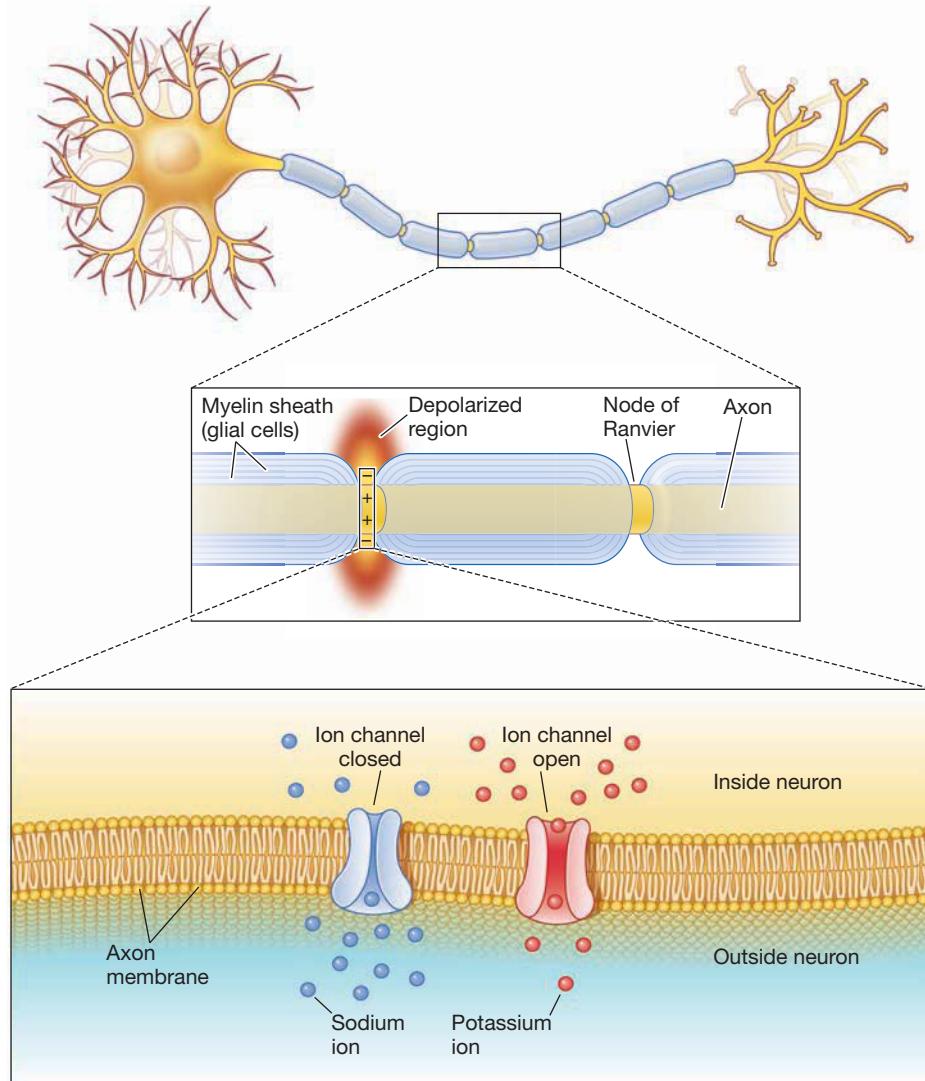
### resting membrane potential

The electrical charge of a neuron when it is not active.

**FIGURE 3.6**

### Resting Membrane Potential

A neuron at rest is polarized: It has a more negative electrical charge inside than outside. The passage of negative and positive ions inside and outside the membrane is regulated by ion channels, such as those located at the nodes of Ranvier.



Ions pass through the neuron membrane at the ion channels (**FIGURE 3.6**). Each channel matches a specific type of ion: Sodium channels allow sodium ions but not potassium ions to pass through the membrane, and potassium channels allow passage of potassium ions but not sodium ions. The flow of ions through each channel is controlled by a gating mechanism. When a gate is open, ions flow in and out of the cell membrane. A closed gate prevents their passage. Ion flow is also affected by the cell membrane's selective permeability. That is, much like a bouncer at an exclusive nightclub, the membrane allows some types of ions to cross more easily than others. Partially as a result of this selective permeability of the cell membrane, more potassium than sodium is inside the neuron.

Another mechanism in the membrane that contributes to polarization is the *sodium-potassium pump*. This pump increases potassium and decreases sodium inside the neuron, activity that helps maintain the resting membrane potential.

## Action Potentials Cause Neural Communication

Neural communication depends on a neuron's ability to respond to incoming stimulation. The neuron responds by changing electrically and then passing along signals to

other neurons. An **action potential**, also called *neural firing*, is the electrical signal that passes along the axon. This signal causes the terminal buttons to release chemicals that transmit signals to other neurons. The following sections examine some factors that contribute to the firing of an action potential.

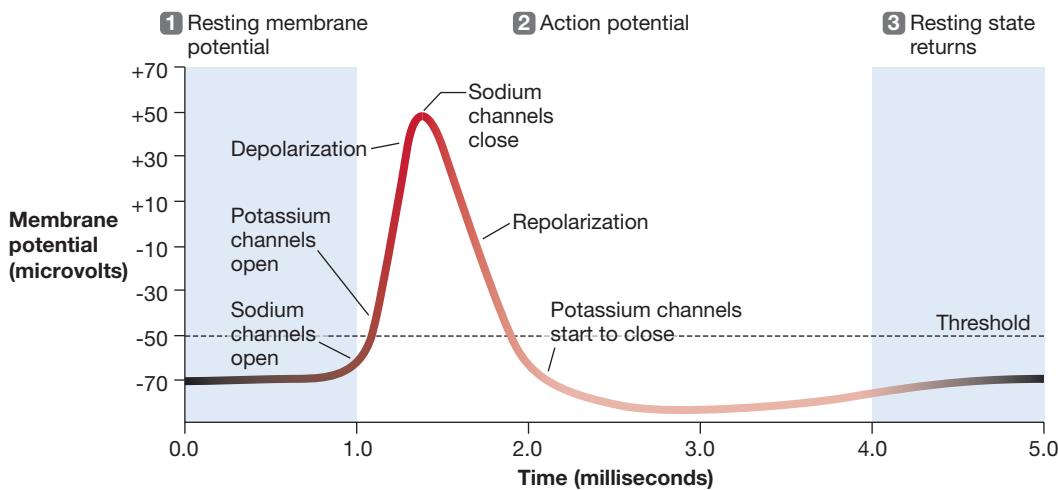
#### action potential

The electrical signal that passes along the axon and subsequently causes the release of chemicals from the terminal buttons.

**CHANGES IN ELECTRICAL POTENTIAL LEAD TO ACTION** A neuron receives chemical signals from nearby neurons through its dendrites. By affecting polarization, these chemical signals tell the neuron whether to fire. The signals arrive at the dendrites by the thousands and are of two types: excitatory and inhibitory. *Excitatory signals* depolarize the cell membrane (i.e., decrease polarization by decreasing the negative charge inside the cell). Through depolarization, these signals increase the likelihood that the neuron will fire. *Inhibitory signals* hyperpolarize the cell (i.e., increase polarization by increasing the negative charge inside the cell). Through hyperpolarization, these signals decrease the likelihood that the neuron will fire. Excitatory and inhibitory signals received by the dendrites are combined within the neuron. If the total amount of excitatory input surpasses the neuron's firing threshold (-55 millivolts), an action potential is generated.

When a neuron fires, the sodium gates in the cell membrane open. The open gates allow sodium ions to rush into the neuron. This influx of sodium causes the inside of the neuron to become slightly more positively charged than the outside. A fraction of a second later, potassium channels open to allow the potassium ions inside the cell membrane to rush out. This change from a negative charge to a positive one inside the neuron is the basis of the action potential. As the sodium ion channels close, the sodium ions stop entering the cell. Similarly, as the potassium ion channels close, potassium ions stop exiting the cell. Thus, during this process, the electrical charge inside the cell starts out slightly negative in its initial resting state. As the cell fires and allows more positive ions inside, the charge becomes positive. Through natural restoration, including the activity of the sodium-potassium pump, the charge then returns to its slightly negative resting state (**FIGURE 3.7**).

**ACTION POTENTIALS SPREAD ALONG THE AXON** When the neuron fires, the cell membrane's depolarization moves along the axon like a wave. Sodium ions rush through their channels, causing adjacent sodium channels to open. Thus, like toppling dominoes, sodium ion channels open in a series. The action potential always moves down the axon away from the cell body to the terminal buttons. These



**FIGURE 3.7**  
**Action Potential**

The electrical charge inside the neuron starts out slightly negative (resting membrane potential, -70 millivolts). As the neuron fires, it allows more positive ions inside the cell (depolarization). It then returns to its slightly negative resting state.

electrical signals travel quickly down most axons because of the fatty **myelin sheath** that encases and insulates many axons like the plastic tubing around wires in an electrical cord (see Figure 3.5).

The myelin sheath is made up of *glial cells*, commonly called *glia* (Greek for “glue”). The sheath grows along an axon in short segments. Between these segments are small gaps of exposed axon called the **nodes of Ranvier** (after the researcher who first described them). Because of the insulation provided by the myelin sheath, the action potential skips quickly along the axon. It pauses briefly to be recharged at each node along the axon. The entire process takes about  $\frac{1}{1,000}$  of a second, permitting the fast and frequent adjustments required for coordinating motor activity. For those axons without myelin, sodium channels along each part of the membrane must open. Action potentials are still generated, but the speed of conduction is decreased greatly.

Recall from the chapter opener that Jack Osbourne’s vision was affected because multiple sclerosis destroys the myelin sheath. Sensory and motor neurons must maintain their myelin to generate fast signals over long distances. Think of how fast you are able to remove your hand from a hot surface to avoid being burned. That speed of movement is the result of myelin, which allows you to feel the heat and reflexively remove your hand. Sensory and motor axons that have no insulation cannot transmit their action potentials as quickly or efficiently. The loss of myelin means that visual information is disrupted and motor actions become jerky and uncoordinated.

**ALL-OR-NONE PRINCIPLE** Any one signal received by the neuron has little influence on whether the neuron fires. Normally, a neuron is barraged by thousands of excitatory and inhibitory signals, and its firing is determined by the number and frequency of those signals. If the sum of excitatory and inhibitory signals leads to a positive change in voltage that exceeds the neuron’s firing threshold, an action potential is generated.

A neuron either fires or it does not. It works like a light switch that is either on or off, not like a dimmer switch. The **all-or-none principle** dictates that a neuron fires with the same potency each time. In other words, it does not fire in a way that can be described as weak or strong. What is affected by the strength of the stimulation is how often the neuron fires: The stronger the stimulation, the more frequently it fires action potentials.

For the sake of comparison, suppose you are playing a video game in which you fire missiles by pressing a button. Every time you press the button, a missile is launched at the same velocity as the previous one. It makes no difference how hard you press the button. If you keep your finger on the button, additional missiles fire in rapid succession. Likewise, if a neuron in the visual system, for example, receives information that a light is bright, it might respond by firing more rapidly and more often than when it receives information that the light is dim. Regardless of whether the light is bright or dim, the strength of the firing will be the same every time.

#### myelin sheath

A fatty material, made up of glial cells, that insulates some axons to allow for faster movement of electrical impulses along the axon.

#### nodes of Ranvier

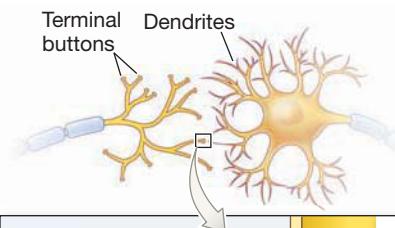
Small gaps of exposed axon, between the segments of myelin sheath, where action potentials take place.

#### all-or-none principle

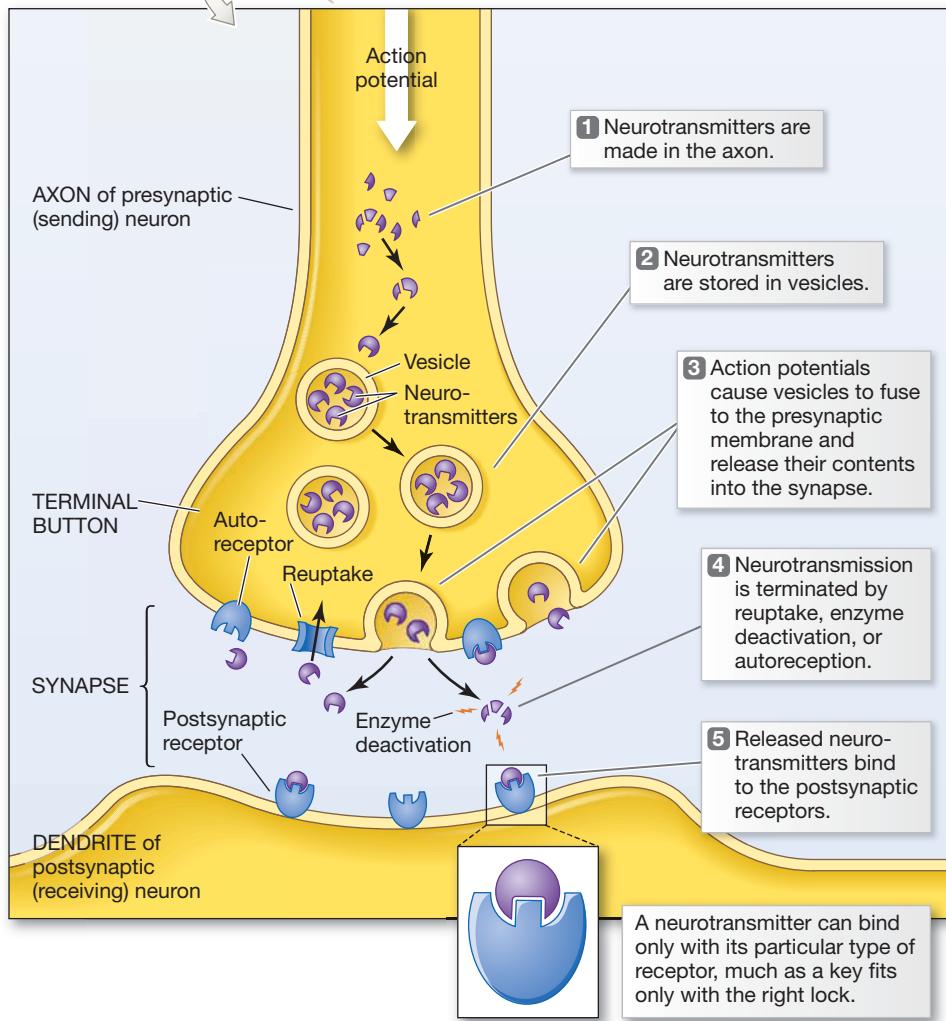
The principle that when a neuron fires, it fires with the same potency each time; a neuron either fires or not—it cannot partially fire, although the frequency of firing can vary.

## Neurotransmitters Bind to Receptors Across the Synapse

As noted earlier, neurons do not touch one another. They are separated by a small space known as the synapse, the site of chemical communication between neurons. Action potentials cause neurons to release chemicals from their terminal buttons. These chemicals travel across the synapse and are received by other neurons’ dendrites. The neuron that sends the signal is called the *presynaptic neuron*, and the one that receives the signal is called the *postsynaptic neuron*.



**FIGURE 3.8 How Neurotransmitters Work**



How do these chemical signals work (**FIGURE 3.8**)? Inside each terminal button are **neurotransmitters**, chemicals that are made in the axon and stored in vesicles (small, fluid-filled sacs). When released by the vesicles, the neurotransmitters convey signals across the synapse to postsynaptic cells.

After an action potential travels to the terminal button, it causes the vesicles to attach to the presynaptic membrane and release their neurotransmitters into the synapse. These neurotransmitters then travel across the synapse and attach themselves, or *bind*, to receptors on the postsynaptic neuron. **Receptors** are specialized protein molecules located on the postsynaptic membrane that specifically respond to the chemical structure of the neurotransmitter available in the synapse. The binding of a neurotransmitter with a receptor can cause ion channels to open or close more tightly, producing an excitatory or an inhibitory signal in the postsynaptic neuron. As mentioned previously, an excitatory signal encourages the neuron to fire. An inhibitory signal discourages it from firing.

#### neurotransmitters

Chemical substances that transmit signals from one neuron to another.

#### receptors

In neurons, specialized protein molecules on the postsynaptic membrane; neurotransmitters bind to these molecules after passing across the synapse.

**NEUROTRANSMITTERS BIND WITH SPECIFIC RECEPTORS** More than 60 chemicals convey information in the nervous system. Different neurotransmitters influence emotion, thought, or behavior. In much the same way as a lock opens only with the correct key, each receptor can be influenced by only one type of neurotransmitter.

Once a neurotransmitter is released into the synapse, it continues to bind with receptors and continues to exert an inhibitory or excitatory effect. It also blocks new signals until its influence is terminated. The three major events that terminate the neurotransmitter's influence in the synapse are *reuptake*, *enzyme deactivation*, and *autoreception*. **Reuptake** occurs when the neurotransmitter is taken back into the presynaptic terminal buttons. An action potential prompts terminal buttons to release the neurotransmitter into the synapse and then take it back for recycling. The cycle of reuptake and release repeats continuously. **Enzyme deactivation** occurs when an enzyme destroys the neurotransmitter in the synapse. Different enzymes break down different neurotransmitters. Neurotransmitters can also bind with receptors on the presynaptic neuron. These *autoreceptors* monitor how much neurotransmitter has been released into the synapse. When an excess is detected, the autoreceptors signal the presynaptic neuron to stop releasing the neurotransmitter.

All neurotransmitters have excitatory or inhibitory effects on action potentials. They do so by affecting the polarization of the postsynaptic cells. The effects are a function of the receptors that the neurotransmitters bind to. Recall the lock and key idea, in which a specific neurotransmitter binds only with certain receptors. The receptor always has a specific response, either excitatory or inhibitory. The same neurotransmitter can send excitatory or inhibitory postsynaptic signals, depending on the particular receptor's properties. In other words, the effects of a neurotransmitter are not a property of the chemical. Instead, the effects are a function of the receptor to which the neurotransmitter binds. Any neurotransmitter can be excitatory or inhibitory. Alternatively, it can produce radically different effects, depending on the properties of the receptor and on the receptor's location in the brain.

## Neurotransmitters Influence Mental Activity and Behavior

Much of what we know about neurotransmitters has been learned through the systematic study of how drugs and toxins affect emotion, thought, and behavior. Drugs and toxins can alter a neurotransmitter's action in many ways. For example, they can alter how a neurotransmitter is synthesized. They can raise or lower the amount of a neurotransmitter released from the terminal buttons. Or, by blocking reuptake, they can change the way a neurotransmitter is deactivated in the synapse and therefore affect the concentration of the neurotransmitter.

Drugs and toxins that enhance the actions of neurotransmitters are known as *agonists*. Drugs and toxins that inhibit these actions are known as *antagonists*. Drugs and toxins can also mimic neurotransmitters and bind with their receptors as if they were the real thing (**FIGURE 3.9**). Addictive drugs such as heroin, for example, have their effects because they are chemically similar to naturally occurring neurotransmitters. The receptors cannot differentiate between the ingested drug and the real neurotransmitter released from a presynaptic neuron. That is, although a neurotransmitter fits a receptor the way a key fits a lock, the receptor/lock cannot tell a real neurotransmitter/key from a forgery—either will open it.

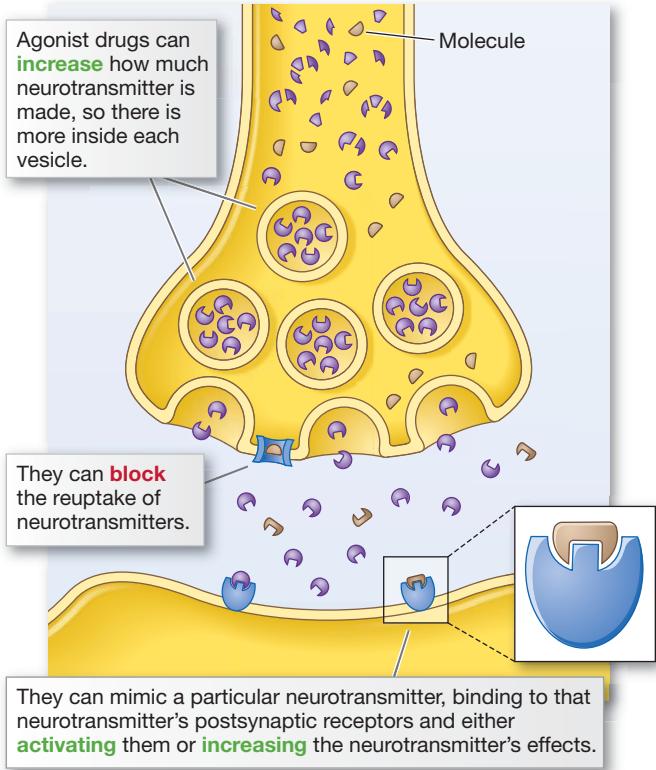
### reuptake

The process whereby a neurotransmitter is taken back into the presynaptic terminal buttons, thereby stopping its activity.

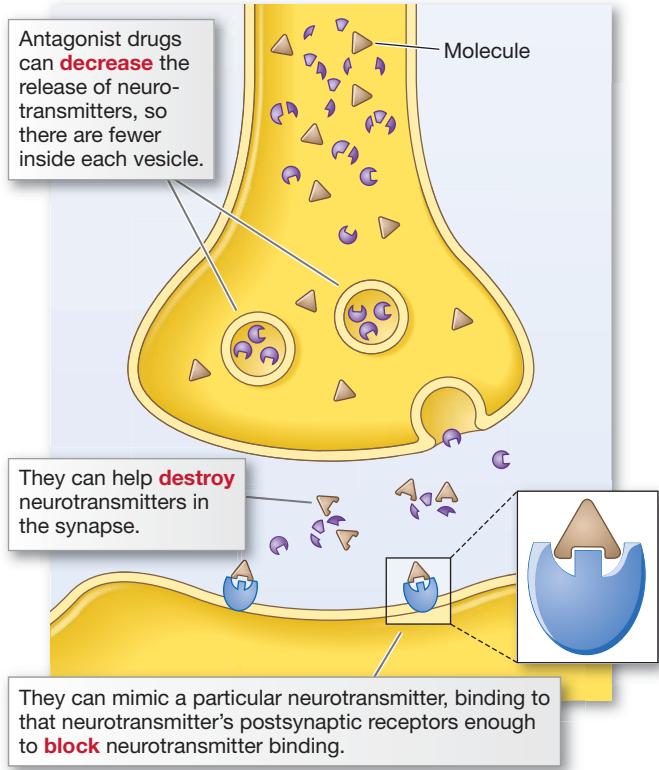
### acetylcholine (ACh)

The neurotransmitter responsible for motor control at the junction between nerves and muscles; it is also involved in mental processes such as learning, memory, sleeping, and dreaming.

## Agonists



## Antagonists



**FIGURE 3.9** How Drugs Work

Researchers often inject agonists or antagonists into animals to assess how neurotransmitters affect behavior. The goal is to develop drug treatments for many psychological and medical disorders. For instance, researchers can test the hypothesis that a certain neurotransmitter in a specific brain region leads to increased eating. Injecting an agonist into that brain region should increase eating. Injecting an antagonist should decrease eating.

**TYPES OF NEUROTRANSMITTERS** There are many kinds of neurotransmitters. Eight of them are particularly important in understanding how we think, feel, and behave (**TABLE 3.1**).

The neurotransmitter **acetylcholine (ACh)** is responsible for motor control at the junctions between nerves and muscles. After moving across synapses, ACh (pronounced A-C-H) binds with receptors on muscle cells, making the muscles contract or relax. For instance, ACh excites skeletal muscles and inhibits heart muscle. As is true of all neurotransmitters, whether ACh's effects will be excitatory or inhibitory depends on the receptors.

Botulism, a form of food poisoning, is caused by Botulinum toxin. This neurotoxin inhibits the release of ACh. The resulting paralysis of muscles leads to difficulty in chewing, difficulty in breathing, and often death. Because of its ability to paralyze muscles, very small doses of Botulinum toxin are used for cosmetic surgery. Physicians inject the toxin, popularly known as Botox, into the eyebrow region, paralyzing muscles that produce certain wrinkles (**FIGURE 3.10**). Because the effects wear off over time, a new dose of Botox needs to be injected every two to four months. If too much Botox is



**FIGURE 3.10**  
**Acetylcholine and Botox**

Acetylcholine (ACh) is responsible for motor control between nerves and muscles. Botox inhibits the release of ACh, paralyzing muscles. Here, a woman receives a Botox injection to remove wrinkles in her forehead.

**TABLE 3.1** Common Neurotransmitters and Their Major Functions

NEUROTRANSMITTER	PSYCHOLOGICAL FUNCTIONS
<b>Acetylcholine</b>	Motor control over muscles Learning, memory, sleeping, and dreaming
<b>Epinephrine</b>	Energy
<b>Norepinephrine</b>	Arousal, vigilance, and attention
<b>Serotonin</b>	Emotional states and impulsiveness Dreaming
<b>Dopamine</b>	Reward and motivation Motor control over voluntary movement
<b>GABA (gamma-aminobutyric acid)</b>	Inhibition of action potentials Anxiety reduction
<b>Glutamate</b>	Enhancement of action potentials Learning and memory
<b>Endorphins</b>	Pain reduction Reward

**epinephrine**

A monoamine neurotransmitter responsible for bursts of energy after an event that is exciting or threatening.

**norepinephrine**

A monoamine neurotransmitter involved in states of arousal and attention.

**serotonin**

A monoamine neurotransmitter important for a wide range of psychological activity, including emotional states, impulse control, and dreaming.

**dopamine**

A monoamine neurotransmitter involved in motivation, reward, and motor control over voluntary movement.

**GABA**

Gamma-aminobutyric acid; the primary inhibitory transmitter in the nervous system.

**glutamate**

The primary excitatory transmitter in the nervous system.

injected, however, the result can be an expressionless face, because Botox paralyzes the facial muscles used to express emotions, as in smiling and frowning.

Acetylcholine is also involved in complex mental processes such as learning, memory, sleeping, and dreaming. Because ACh affects memory and attention, drugs that are ACh antagonists can cause temporary amnesia. In a similar way, Alzheimer's disease, a condition characterized primarily by severe memory deficits, is associated with diminished ACh functioning (Geula & Mesulam, 1994). Drugs that are ACh agonists may enhance memory and decrease other symptoms, but so far drug treatments for Alzheimer's have experienced only marginal success.

Four transmitters (epinephrine, norepinephrine, serotonin, and dopamine) are grouped together because each has the same basic molecular structure. Together they are called *monoamines*. Their major functions are to regulate arousal, regulate feelings, and motivate behavior.

The neurotransmitter **epinephrine** was initially called *adrenaline*. This name is the basis for the phrase *adrenaline rush*, a burst of energy caused by the release of epinephrine that binds to receptors throughout the body. This energy boost is part of a system that prepares the body for dealing with threats from an environment (the fight-or-flight response, discussed in Chapter 11, "Health and Well-Being"). **Norepinephrine** is involved in states of arousal and alertness. It is especially important for vigilance, a heightened sensitivity to what is going on around you. Norepinephrine appears useful for fine-tuning the clarity of attention.

**Serotonin** is involved in a wide range of psychological activities. It is especially important for emotional states, impulse control, and dreaming. Low levels of serotonin are associated with sad and anxious moods, food cravings, and aggressive behavior. Some drugs block serotonin reuptake and thus leave more serotonin at the synapse to bind with the postsynaptic neurons. These drugs are used to treat a wide array of mental and behavioral disorders, including depression, obsessive-compulsive disorders, eating disorders, and obesity (Tollefson, 1995). One class of drugs that specifically target serotonin is prescribed widely to treat depression. These drugs,

which include Prozac, are referred to as *selective serotonin reuptake inhibitors*, or *SSRIs*.

**Dopamine** serves many significant brain functions, especially motivation and reward. Many theorists believe dopamine communicates which activities may be rewarding. For example, eating when hungry, drinking when thirsty, and having sex when aroused activate dopamine receptors and therefore are experienced as pleasurable. When we see food, dopamine activity motivates us to want to eat it. Dopamine activation is also involved in motor control and planning. It helps guide behavior toward things—objects and experiences—that will lead to additional reward.

A lack of dopamine may be involved in problems with movement, and dopamine depletion is implicated in Parkinson's disease. Parkinson's is a degenerative and fatal neurological disorder marked by muscular rigidity, tremors, and difficulty initiating voluntary action. It affects about 1 in every 200 older adults and occurs in all known cultures. The actor Michael J. Fox is one of the many famous people who have developed this disease (**FIGURE 3.11**). Most people with Parkinson's do not experience symptoms until after age 50, but as Fox's case makes clear, the disease can occur earlier in life.

With Parkinson's disease, the dopamine-producing neurons slowly die off. In the later stages of the disorder, people suffer from cognitive and mood disturbances. Injections of one of the chief building blocks of dopamine, *L-DOPA*, help the surviving neurons produce more dopamine. When used to treat Parkinson's disease, L-DOPA often produces a remarkable, though temporary, recovery.

A promising development in Parkinson's research is *deep brain stimulation*. This procedure involves surgically implanting electrodes deep within the brain and then using mild electrical stimulation in the regions affected by the disorder, much the way a pacemaker stimulates the heart. Deep brain stimulation of motor regions of the brains of Parkinson's patients reverses many of the movement problems associated with the disease (DeLong & Wichmann, 2008). Researchers have reported successful results from this treatment, lasting as long as eleven years (Rizzzone et al., 2014). Although DBS helps with the motor symptoms of Parkinson's, other symptoms of the disease progressively become worse over time.

**GABA** (gamma-aminobutyric acid) is the primary inhibitory neurotransmitter in the nervous system. It is more widely distributed throughout the brain than most other neurotransmitters. Without the inhibitory effect of GABA, synaptic excitation might get out of control and spread through the brain chaotically. Epileptic seizures may be caused by low levels of GABA (Upton, 1994). Drugs that are GABA agonists are widely used to treat anxiety disorders. For instance, benzodiazepines, which include drugs such as Valium and Xanax, help people relax. Ethyl alcohol—the type people drink—also facilitates GABA transmission, which is why alcohol is typically experienced as relaxing.

In contrast, **glutamate** is the primary excitatory transmitter in the nervous system and is involved in fast-acting neural transmission throughout the brain. Glutamate receptors aid learning and memory by strengthening synaptic connections. Excessive glutamate release can lead to overexcitement of the brain, which can produce seizures as well as destruction of neurons. Overexcitement caused by excess glutamate is



**FIGURE 3.11**  
**A Public Figure with Parkinson's**  
Michael J. Fox was diagnosed with Parkinson's disease in 1991 and disclosed his condition to the public in 1998. He has since created the Michael J. Fox Foundation, which advocates for research toward finding a cure for Parkinson's.

## endorphins

Neurotransmitters involved in natural pain reduction and reward.



**FIGURE 3.12**  
**Exercise and Endorphins**

Endorphins are involved in both pain reduction and reward, and scientists think that endorphin production can be stimulated by strenuous exercise. An endurance event, such as a marathon or a speed skating competition, will yield an enormous endorphin rush. Here, the final leg runner in the Saudi men's 4 × 400 relay team, Yousef Ahmed Masrahi, celebrates after finishing first in the men's relay final at the 16th Asian Games in Guangzhou on November 26, 2010.

linked to many diseases and types of brain damage. For example, much of the damage inflicted to the brain following a stroke or trauma to the brain is caused by the excessive release of glutamate that naturally occurs following brain injury (Choi & Rothman, 1990; Dhawan et al., 2011).

**Endorphins** are involved in both natural pain reduction and reward (**FIGURE 3.12**).

In the early 1970s, researchers established that opiate drugs such as heroin and morphine bind to receptors in the brain, and this finding led to the discovery of naturally occurring substances in the body that bind to those sites (Pert & Snyder, 1973). Called *endorphins* (short for *endogenous morphine*), these substances are part of the body's natural defense against pain.

Pain is useful because it signals to animals, human and nonhuman, that they are hurt or in danger and therefore should try to escape or withdraw. Pain can also interfere with adaptive functioning, however. If pain prevents animals from engaging in behaviors such as eating, competing, and mating, the animals fail to pass along their genes. Endorphins' painkilling, or analgesic, effects help animals perform these behaviors even when they are in pain. In humans, the administration of drugs, such as morphine, that bind with endorphin receptors reduces the subjective experience of pain. Apparently, morphine alters the way pain is experienced rather than blocking the nerves that transmit pain signals: People still feel pain, but they report detachment and do not care about the pain (Foley, 1993).

## Summing Up

### How Does the Nervous System Operate?

- Neurons are the nervous system's basic units. Their task is to receive, process, and pass information to other neurons.
- The nervous system is divided into two basic units: The central nervous system consists of the brain and spinal cord. The peripheral nervous system consists of all nerve cells beyond the brain and spinal cord.
- A neuron receives information at the dendrites and processes that information in its cell body. If the information is excitatory, the neuron will generate an action potential, or "fire." Firing sends a signal down the axon to release neurotransmitters into the synapse.
- Many neurons are insulated by a myelin sheath, which surrounds the axon and allows the action potential to travel rapidly.
- When a neuron is in a resting state, it is negatively charged. Whether a neuron fires depends on the combination of excitatory and inhibitory signals it receives. Receiving excitatory signals encourages the neuron to fire. Receiving inhibitory signals discourages it from firing.
- The intensity of the excitatory signal affects the frequency of neural firing but not its strength. Neurons fire on an all-or-none basis.
- After firing, ion channels close and the neuron returns to its negative resting state. Action potentials are terminated by the removal of neurotransmitters from the synapse. This removal occurs through reuptake, enzyme deactivation, or the actions of autoreceptors.
- Substances that enhance the actions of neurotransmitters are agonists. Substances that inhibit the actions of neurotransmitters are antagonists.
- Eight neurotransmitters are especially important for psychological research: Acetylcholine is involved in motor control and mental processes, such as memory. Epinephrine and norepinephrine are associated with energy, arousal, and attention. Serotonin is important for emotional states, impulse control, and dreaming. Dopamine is involved in reward, motivation, and motor control. GABA and glutamate are related to general inhibition and excitation. Endorphins are important in pain reduction.

## Measuring Up

1. Neurons communicate by electrochemical signal. Imagine that a neurotransmitter binds to a postsynaptic receptor. What would happen afterward? Put the following steps in the correct order so they describe this process.

- An action potential is generated down the axon.
- Neurotransmitters are released into the synapse.
- Through reuptake, the neurotransmitter returns to the presynaptic neuron.
- Sodium channels open.

2. Match each major neurotransmitter with its major functions.

Neurotransmitter	Major functions
a. norepinephrine	1. emotional states, impulse control, dreaming
b. glutamate	2. reward, motivation, voluntary muscle control
c. acetylcholine	3. generates excitatory action potentials, facilitates learning and memory
d. serotonin	4. arousal, vigilance, attention
e. endorphins	5. motor control, learning, memory, sleeping, dreaming
f. dopamine	6. reward, pain reduction
g. GABA	7. energy
h. epinephrine	8. inhibits action potentials, reduces anxiety

(2) a; 4; b; 3; c; 5; d; 1; e; 6; f; 2; g; 8; h; 7.

ANSWERS: (1) d, a, b, c.

## 3.2 What Are the Basic Brain Structures and Their Functions?

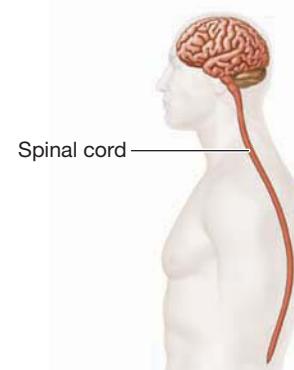
The brain is best viewed as a collection of interacting neural circuits. These circuits have accumulated and developed throughout human evolution. As our ancestors adapted to their environments, the brain has evolved specialized mechanisms to regulate breathing, food intake, body fluids, and sexual and social behavior, as well as sensory systems to aid in navigation and assist in recognizing friends and foes. Everything we are and do is orchestrated by the brain and, for more rudimentary actions, by the spinal cord (**FIGURE 3.13**). Early in life, overabundant connections form among the brain's neurons. Subsequently, life experiences help "prune" some of these connections to strengthen the rest, much as pruning weak or nonproductive branches will strengthen a fruit tree.

The brain's basic structures and their functions enable people to accomplish feats such as seeing, hearing, remembering, and interacting with others. Understanding these relationships also helps us understand psychological disorders.

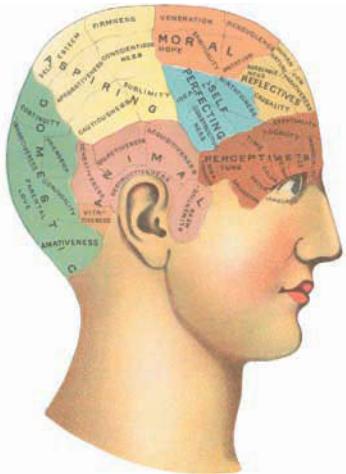
**EARLY RESEARCHERS DEBATED THE RELATIONSHIP BETWEEN STRUCTURE AND FUNCTION** By the beginning of the nineteenth century, anatomists understood the brain's basic structure reasonably well. But debates raged over how the brain produced mental activity. Did different parts do different things? Or were all areas of the brain equally important in cognitive activities such as problem solving and memory?

### Learning Objective

- Identify the basic structures of the brain and their primary functions.



**FIGURE 3.13**  
**The Brain and the Spinal Cord**  
This drawing illustrates the brain's exterior and its connection with the spinal cord. The view is from the left side of the body.



**FIGURE 3.14**  
**Phrenology**

In a phrenological map, each region of the skull is associated with a feature. Each association is meant to reflect a process occurring in the brain under the skull.

#### Broca's area

A small portion of the left frontal region of the brain, crucial for the production of language.

In the early nineteenth century, the neuroanatomist Franz Gall and his assistant, the physician Johann Spurzheim, hypothesized about the effects of mental activity on brain anatomy. Gall and Spurzheim proposed that if a person used a particular mental function more than other mental functions, the part of the brain where the emphasized function was performed would grow. This growth would produce a bump in the overlying skull. By carefully feeling the skull, one could describe the personality of the individual. This practice came to be known as *phrenology* (FIGURE 3.14).

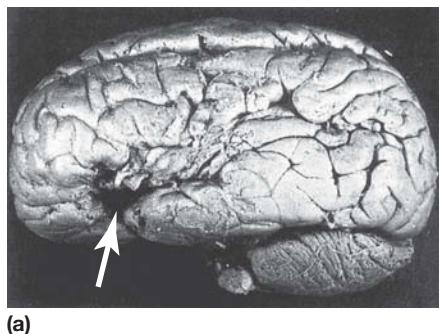
Gall was a physician, not a scientist. He noted correlations, but he did not practice the scientific method and sought only to confirm, not disprove, his ideas. In any case, at the time, the technology was not available to test this theory scientifically. Phrenology soon fell into the hands of frauds and quacks, but it helped spread the seemingly scientific principle that brain functions were localized.

The first strong scientific evidence that brain regions perform specialized functions came from the work of the nineteenth-century physician and anatomist Paul Broca (Finger, 1994). One of Broca's patients had lost the ability to say anything other than the word *tan*, though he could still understand language. After the patient died, in 1861, Broca performed an autopsy. When he examined the patient's brain, Broca found a large area of damage in a section of the front left side. This observation led him to conclude that this particular region was important for speech. Broca's theory has survived the test of time. This left frontal region, crucial for the production of language, became known as **Broca's area** (FIGURE 3.15).

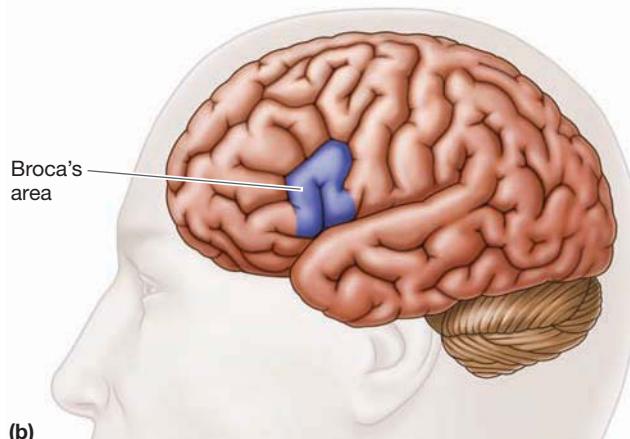
For most of human history, theorists and researchers have not had methods for studying ongoing mental activity in the working brain. In the 1980s, the invention of brain imaging methods changed that situation swiftly and dramatically. As discussed in the following section, the new imaging techniques have advanced our understanding of the human brain the way the development of telescopes advanced our understanding of astronomy—and the brain's structures and functions may be as complex as distant galaxies.

## Scientists Can Now Watch the Working Brain

Psychologists collect data about the ways people's bodies respond to particular tasks or events. For instance, when people are frightened, their muscles become tense and



(a)



(b)

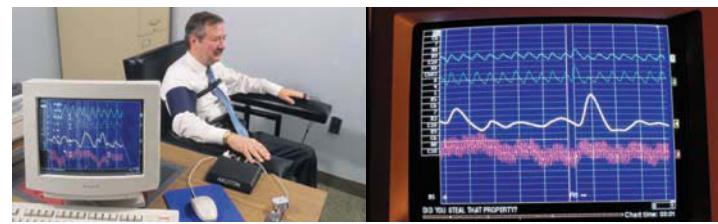
**FIGURE 3.15** **Broca's Area**

(a) Paul Broca studied a patient's brain and identified the damaged area as crucial for speech production.

(b) This illustration shows the location of Broca's area.

their hearts beat faster. Other bodily systems influenced by mental states include blood pressure, blood temperature, perspiration rate, breathing rate, and pupil size. Measurements of these systems are examples of *psychophysiological assessment*. In this type of testing, researchers examine how bodily functions (physiology) change in association with behaviors or mental states (psychology).

Police investigators often use *polygraphs*, popularly known as “lie detectors,” to assess some bodily states (FIGURE 3.16). The assumption behind these devices is that people who are lying experience more arousal and therefore are more likely to show physical signs of stress. This method is not precise, however, and so lie detectors do not accurately measure whether someone is lying. (The limitations of lie detectors are discussed further in the “What to Believe? Using Psychological Reasoning” feature in Chapter 10.)

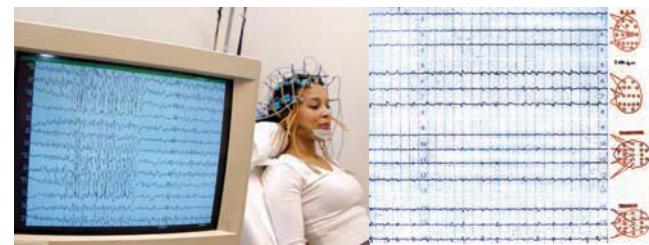


**FIGURE 3.16**

### Polygraph

A polygraph (lie detector) measures changes in bodily functions (e.g., heart rate, perspiration rate, blood pressure) related to behaviors or mental states. These changes are *not* reliable measures of lying.

**ELECTROPHYSIOLOGY** *Electrophysiology* is a data collection method that measures electrical activity in the brain. Small electrodes on the scalp act like small microphones that pick up the brain’s electrical activity instead of sounds. The device that measures brain activity is an **electroencephalograph** (EEG; FIGURE 3.17). This measurement is useful because different behavioral states produce different and predictable EEG patterns. As a measure of specific cognitive states, however, the EEG is limited. Because the recordings (*electroencephalograms*) reflect all brain activity, they are too “noisy” or imprecise to isolate specific responses to particular stimuli. A more powerful way of examining how brain activity changes in response to a specific stimulus involves conducting many trials with a single individual and averaging across the trials. Because this method enables researchers to observe patterns associated with specific events, it is called *event-related potential* (ERP).



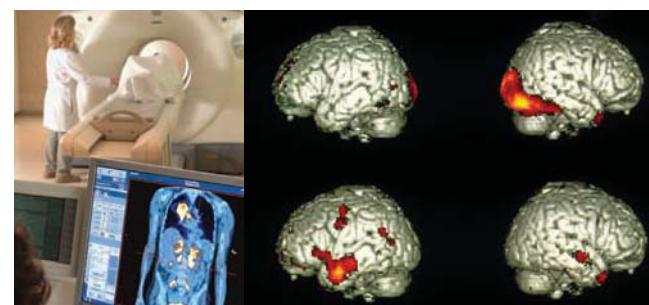
**FIGURE 3.17**

### Electroencephalograph (EEG)

An electroencephalograph (EEG) measures the brain’s electrical activity.

**BRAIN IMAGING** The brain’s electrical activity is associated with changes in the flow of blood carrying oxygen and nutrients to the active brain regions. *Brain imaging* methods measure changes in the rate, or speed, of the flow of blood to different regions of the brain. By keeping track of these changes, researchers can monitor which brain areas are active when people perform particular tasks or experience particular events. Imaging is a powerful tool for uncovering where different systems reside in the brain and how different brain areas interact to process information.

- **Positron emission tomography (PET)** After the injection of a relatively harmless radioactive substance into the bloodstream, a **positron emission tomography (PET)** scan enables researchers to find the most active brain areas (FIGURE 3.18). The increased blood flow carrying the radioactive material leads these regions to emit more radiation. One downside of PET is the need to inject a radioactive substance into the body. For safety reasons, researchers limit the use of this technology.
- **Magnetic resonance imaging (MRI)** With **magnetic resonance imaging (MRI)**, a powerful magnetic field is used to momentarily disrupt the brain’s



**FIGURE 3.18**

### Positron Emission Tomography

Positron emission tomography (PET) scans the brain’s metabolic activity.



**FIGURE 3.19**  
**Magnetic Resonance Imaging**

Magnetic resonance imaging (MRI) produces a high-resolution image of the brain.

#### magnetic resonance imaging (MRI)

A method of brain imaging that uses a powerful magnetic field to produce high-quality images of the brain.

#### functional magnetic resonance imaging (fMRI)

An imaging technique used to examine changes in the activity of the working human brain by measuring changes in the blood's oxygen levels.

#### transcranial magnetic stimulation (TMS)

The use of strong magnets to briefly interrupt normal brain activity as a way to study brain regions.



**FIGURE 3.20 Functional Magnetic Resonance Imaging**

Functional magnetic resonance imaging (fMRI) maps mental activity by assessing the blood's oxygen level in the brain.

magnetic forces (**FIGURE 3.19**). During this process, energy is released from brain tissue in a form that can be measured by detectors surrounding the head. Because different types of brain tissue release energy differently, the researchers can produce a high-resolution image of the brain. (The amount of energy released is very small, so having an MRI is not dangerous. Nor is there any danger in being exposed to the magnetic field at the levels used in research.) MRI is extremely valuable for providing information about the structure of the brain. For instance, it can be used to

determine the location of brain damage or of a brain tumor.

- **Functional magnetic resonance imaging (fMRI)** Functional magnetic resonance imaging (fMRI) makes use of the brain's blood flow to map the working brain (**FIGURE 3.20**). Whereas PET measures blood flow directly by tracking a radioactive substance, fMRI measures blood flow indirectly by assessing changes in the blood's oxygen level. As with all brain imaging methods, the participant performs a task that differs from the first one in only one way and that reflects the particular mental function of interest. The researchers then compare images to examine differences in blood flow and therefore brain activity.

**TRANSCRANIAL MAGNETIC STIMULATION** One limitation of brain imaging is that the findings are necessarily correlational. We know that certain brain regions are active while a task is performed. We do not know whether each brain region is necessary for that particular task. To see whether a brain region is important for a task, researchers ideally want to compare performances when that area is working effectively and when it is not. **Transcranial magnetic stimulation (TMS)** uses a very fast but powerful magnetic field to disrupt brain activity momentarily in a specific brain region (**FIGURE 3.21**). This technique has its limitations, particularly that it can be used only for short durations to examine brain areas close to the scalp. When used along with imaging, however, it is a powerful method for examining which brain regions are necessary for specific psychological functions.

The following sections discuss specific brain areas. While these areas do not work in isolation, each one is linked with particular mental processes and particular behaviors.

## The Brain Stem Houses the Basic Programs of Survival

The spinal cord is a rope of neural tissue. As shown in Figure 3.13, the cord runs inside the hollows of the vertebrae from just above the pelvis up into the base of the skull. One of its functions is the coordination

of reflexes, such as the reflexive movement of your leg when a doctor taps your knee or the reflexive movement of your arm when you jerk your hand away from a flame. The cord's most important function is to carry sensory information up to the brain and carry motor signals from the brain to the body parts below to initiate action.

In cross section, the spinal cord is seen to be composed of two distinct tissue types: the *gray matter*, which is dominated by neurons' cell bodies, and the *white matter*, which consists mostly of axons and the fatty myelin sheaths that surround them. Gray matter and white matter are clearly distinguishable throughout the brain as well. In the brain, gray matter consists mostly of neuron bodies that have nonmyelinated axons and communicate only with nearby neurons. White matter consists mostly of myelinated axons that travel between brain regions.

In the base of the skull, the spinal cord thickens and becomes more complex as it transforms into the **brain stem** (FIGURE 3.22). The brain stem consists of the *medulla oblongata*, the *pons*, and the *midbrain*. It houses the nerves that control the most basic functions of survival, such as heart rate, breathing, swallowing, vomiting, urination, and orgasm. A significant blow to this region can cause death. As a continuous extension of the spinal cord, the brain stem also performs functions for the head similar to those that the spinal cord performs for the rest of the body. Many reflexes emerge from here, analogous to the spinal reflexes; gagging is one example.

The brain stem also contains a network of neurons, known collectively as the *reticular formation*. The reticular formation projects up into the *cerebral cortex* (outer portion of the brain—discussed shortly) and affects general alertness. It is also involved in inducing and terminating the different stages of sleep (as discussed in Chapter 4, “Consciousness”).

## The Cerebellum Is Essential for Movement

The **cerebellum** (Latin, “little brain”) is a large protuberance connected to the back of the brain stem (FIGURE 3.23). Its size and convoluted surface make it look like an extra brain. The cerebellum is extremely important for proper motor function, and damage to its different parts produces very different effects. For example, damage to the little nodes at the very bottom causes head tilt, balance problems, and a loss of smooth compensation of eye position for head movement.

Try turning your head while looking at this book. Notice that your eyes remain focused on the material. Your eyes would not be able to do that if an injury affected the bottom of your cerebellum. Damage to the ridge that runs up the back of the cerebellum would affect your walking. Damage to the bulging lobes on either side would cause a loss of limb coordination, so you would not be able to perform tasks such as reaching smoothly to pick up a pen.

The cerebellum’s most obvious role is in motor learning and motor memory. It seems to be “trained” by the rest of the nervous system and operates independently and unconsciously. For example, the cerebellum allows you to ride a bicycle effortlessly while planning your next meal. In fact, the cerebellum may be involved in cognitive processes such as making plans, remembering events, using language, and experiencing emotion.



**FIGURE 3.21**  
**Transcranial Magnetic Stimulation**

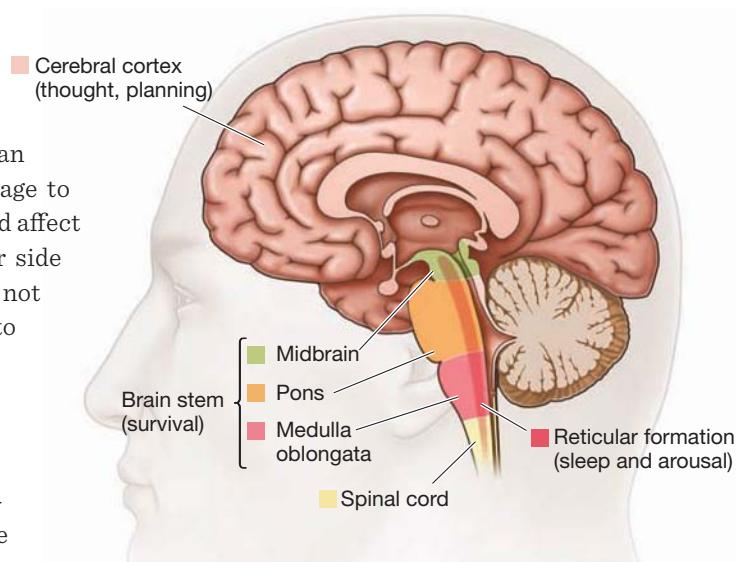
Transcranial magnetic stimulation (TMS) momentarily disrupts brain activity in a specific brain region.

### brain stem

An extension of the spinal cord; it houses structures that control functions associated with survival, such as heart rate, breathing, swallowing, vomiting, urination, and orgasm.

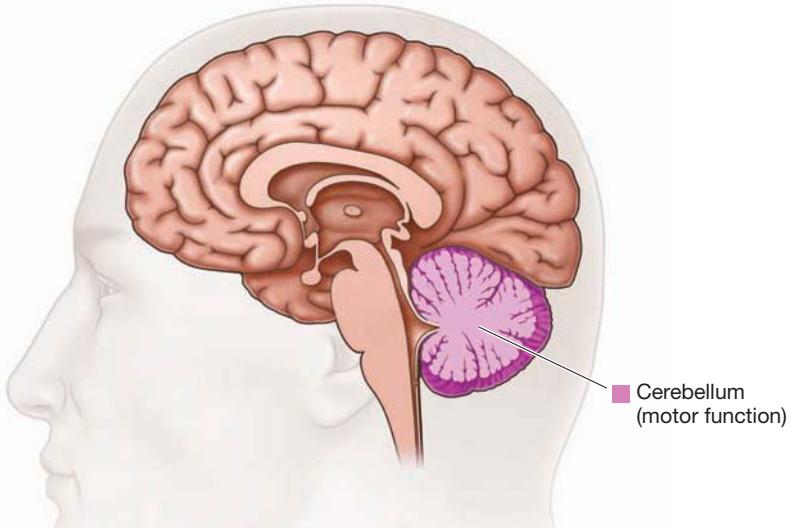
### cerebellum

A large, convoluted protuberance at the back of the brain stem; it is essential for coordinated movement and balance.



**FIGURE 3.22** **The Brain Stem**

This drawing shows the brain stem and its parts, in relation to the cerebral cortex.



**FIGURE 3.23 The Cerebellum**

The cerebellum is located at the back of the brain: It is below the cerebral cortex and behind the brain stem.

#### thalamus

The gateway to the brain; it receives almost all incoming sensory information before that information reaches the cortex.

#### hypothalamus

A brain structure that is involved in the regulation of bodily functions, including body temperature, body rhythms, blood pressure, and blood glucose levels; it also influences our basic motivated behaviors.

#### hippocampus

A brain structure that is associated with the formation of memories.

#### amygdala

A brain structure that serves a vital role in learning to associate things with emotional responses and in processing emotional information.

#### basal ganglia

A system of subcortical structures that are important for the planning and production of movement.

## Subcortical Structures Control Emotions and Appetitive Behaviors

Above the brain stem and cerebellum is the *forebrain*, which consists of the two cerebral hemispheres (left and right; **FIGURE 3.24**). From the outside, the most noticeable feature of the forebrain is the cerebral cortex. Below the cerebral cortex are the *subcortical* regions, which are so named because they lie under the cortex. Subcortical structures that are important for understanding psychological functions include the hypothalamus, the thalamus, the hippocampus, the amygdala, and the basal ganglia. Some of these structures belong to the *limbic system*.

*Limbic* is the Latin word for “border,” and this system serves as the border between the evolutionarily older parts of the brain (the brain stem and the cerebellum) and the evolutionarily newer part (the cerebral cortex). The brain structures in the limbic system are especially important for controlling appetitive behaviors (such as eating and drinking) and emotions (as discussed in Chapter 10, “Emotion and Motivation”).

**THALAMUS** The **thalamus** is the gateway to the cortex: It receives almost all incoming sensory information, organizes it, and relays it to the cortex. The only exception to this rule is the sense of smell. The oldest and most fundamental sense, smell has a direct route to the cortex. During sleep, the thalamus partially shuts the gate on incoming sensations while the brain rests. (The thalamus is discussed further in Chapter 5, “Sensation and Perception.”)

**HYPOTHALAMUS** The **hypothalamus** is the brain’s master regulatory structure. It is indispensable to the organism’s survival. Located just below the thalamus, it receives input from almost everywhere in the body and brain, and it projects its influence to almost everywhere in the body and brain. It affects the functions of many internal organs, regulating body temperature, body rhythms, blood pressure, and blood glucose levels. It is also involved in many motivated behaviors, including thirst, hunger, aggression, and lust.

**HIPPOCAMPUS AND AMYGDALA** The **hippocampus** takes its name from the Greek for “sea horse,” because of its sea horse shape. This structure plays an important role in the formation of new memories. It seems to do this important work by creating new interconnections within the cerebral cortex with each new experience.

The hippocampus may be involved in how we remember the arrangements of places and objects in space, such as how streets are laid out in a city or how furniture is positioned in a room. An interesting study to support this theory focused on London taxi drivers. Maguire and colleagues (2003) found that one region of the hippocampus was much larger in taxi drivers’ brains than in most other London

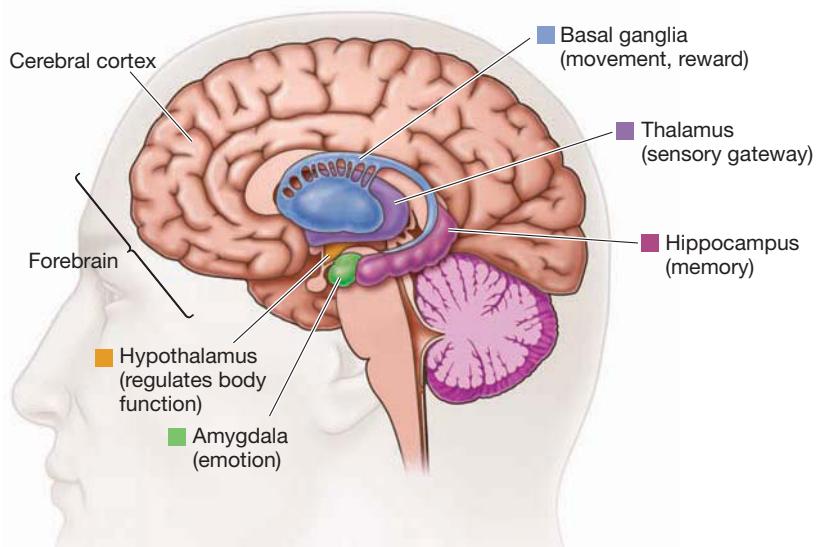
drivers' brains. Moreover, the volume of gray matter in the hippocampal region was highly correlated with the number of years of experience as a taxi driver. Is a person with a large hippocampus more likely to drive a taxi? Or does the hippocampus grow as the result of navigational experience? Recall from Chapter 2 that correlation does not prove causation. The Maguire study did not conclude that the hippocampus changes with experience. However, there is evidence that the hippocampus is important for navigating in our environments (Nadel et al., 2013).

The **amygdala** takes its name from the Latin for “almond,” because it has an almond shape. This structure is located immediately in front of the hippocampus. The amygdala is involved in learning about biologically relevant stimuli, such as those important for survival (Whalen et al., 2013). It plays a special role in responding to stimuli that elicit fear. The emotional processing of frightening stimuli in the amygdala is a hardwired circuit that has developed over the course of evolution to protect animals from danger. The amygdala is also involved in evaluating a facial expression's emotional significance (Adolphs et al., 2005). It appears to be part of a system that automatically directs visual attention to the eyes when evaluating facial expressions (Kennedy & Adolphs, 2010). Imaging studies have found that the amygdala activation is especially strong in response to a fearful face (Whalen et al., 1998).

The amygdala also intensifies the function of memory during times of emotional arousal. For example, a frightening experience can be seared into your memory for life, although (as discussed further in Chapter 7, “Memory”) your memory of the event may not be completely accurate. Research also shows that emotional arousal can influence what people attend to in their environments (Schmitz, De Rosa, & Anderson, 2009).

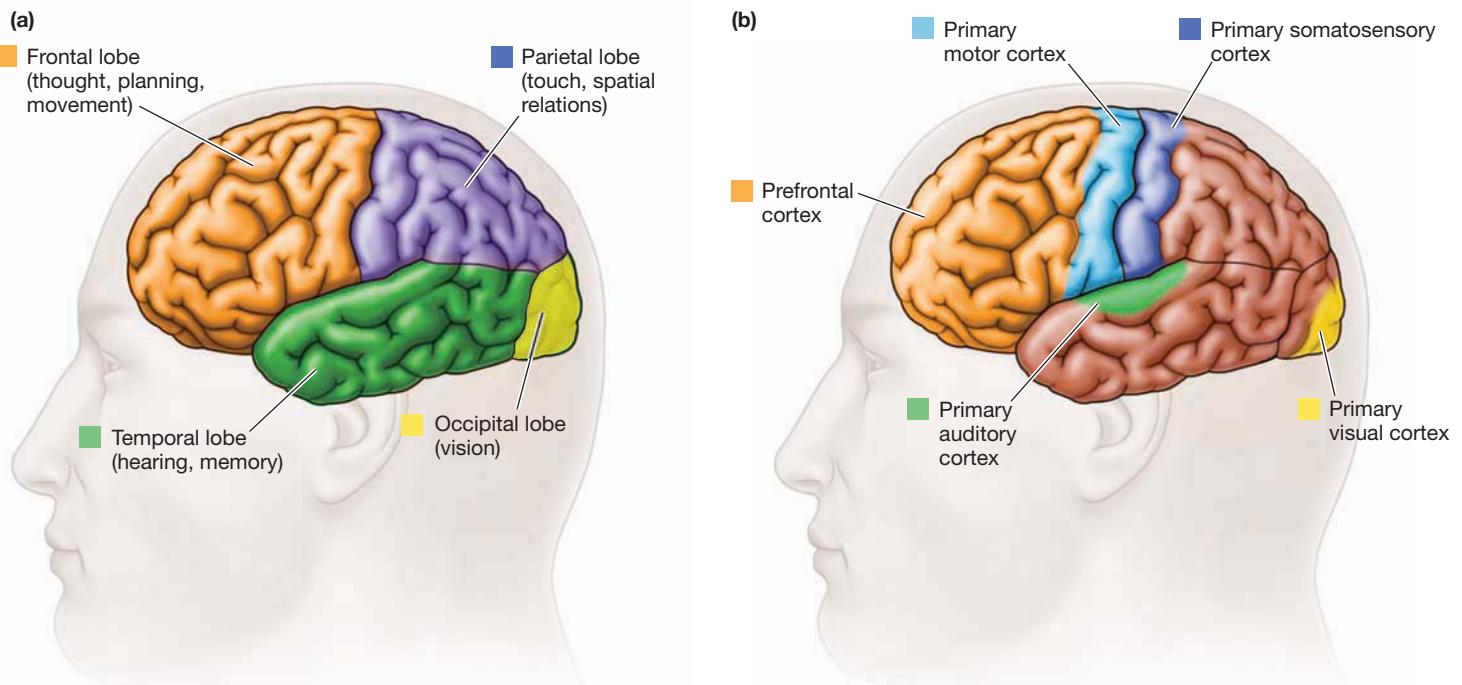
**THE BASAL GANGLIA** The **basal ganglia** are a system of subcortical structures crucial for planning and producing movement. These structures receive input from the entire cerebral cortex. They send that input to the motor centers of the brain stem. Via the thalamus, they also send the input back to the motor planning area of the cerebral cortex. Damage to the basal ganglia can produce symptoms that range from the tremors and rigidity of Parkinson's disease to the involuntary writhing movements of Huntington's disease. In addition, damage to the basal ganglia can impair the learning of movements and habits, such as automatically looking for cars before you cross the street.

One structure in the basal ganglia, the *nucleus accumbens*, is important for experiencing reward and motivating behavior. As discussed in Chapter 6, nearly every pleasurable experience—from eating food you like to looking at a person you find attractive— involves dopamine activity in the nucleus accumbens and makes you want the thing or person you are experiencing. The more desirable objects are, the more they activate basic reward circuitry in our brains.



**FIGURE 3.24 The Forebrain and the Subcortical Regions**

The subcortical regions are below the forebrain. They are responsible for many aspects of emotion and motivation.



**FIGURE 3.25 The Cerebral Cortex**

(a) This diagram identifies the lobes of the cerebral cortex.

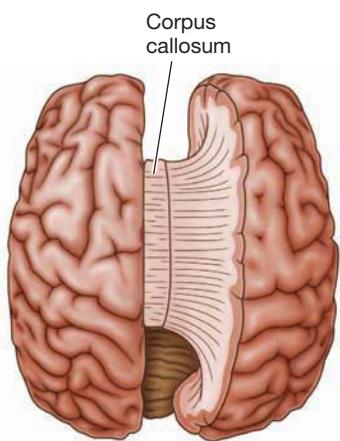
(b) The colored areas mark important regions within those lobes.

#### cerebral cortex

The outer layer of brain tissue, which forms the convoluted surface of the brain; the site of all thoughts, perceptions, and complex behaviors.

#### corpus callosum

A massive bridge of millions of axons that connects the hemispheres and allows information to flow between them.



**FIGURE 3.26**

#### The Corpus Callosum

In this top view of the brain, the right cerebral hemisphere has been pulled away to expose the corpus callosum. This fibrous structure connects the two hemispheres of the cerebral cortex.

## The Cerebral Cortex Underlies Complex Mental Activity

The **cerebral cortex** is the outer layer of the cerebral hemispheres and gives the brain its distinctive wrinkled appearance. (*Cortex* is Latin for “bark”—the kind on trees. The cerebral cortex does not feel like bark, however. It has the consistency of a soft-boiled egg.) Each hemisphere has its own cortex. In humans, the cortex is relatively enormous—the size of a large sheet of newspaper—and folded in against itself many times so as to fit within the skull. It is the site of all thoughts, detailed perceptions, and complex behaviors. It enables us to comprehend ourselves, other people, and the outside world. By extending our inner selves into the world, it is also the source of culture and communication. Each cerebral hemisphere has four “lobes”: the occipital, parietal, temporal, and frontal lobes (FIGURE 3.25). The **corpus callosum**, a massive bridge of millions of axons, connects the hemispheres and allows information to flow between them (FIGURE 3.26).

The **occipital lobes** are at the back portion of the head. Devoted almost exclusively to vision, they include many visual areas. By far, the largest of these areas is the **primary visual cortex**, the major destination for visual information. Visual information is typically organized for the cerebral cortex in a way that preserves spatial relationships. That is, the image relayed from the eye is “projected” more or less faithfully onto the primary visual cortex. As a result, two objects near one another in a visual image will activate neurons near one another in the primary visual cortex. Surrounding the primary visual cortex is a patchwork of secondary visual areas that process various attributes of the visual image, such as its colors, forms, and motions.

The **parietal lobes** are devoted partially to touch. Their labor is divided between the cerebral hemispheres. The left hemisphere receives touch information from

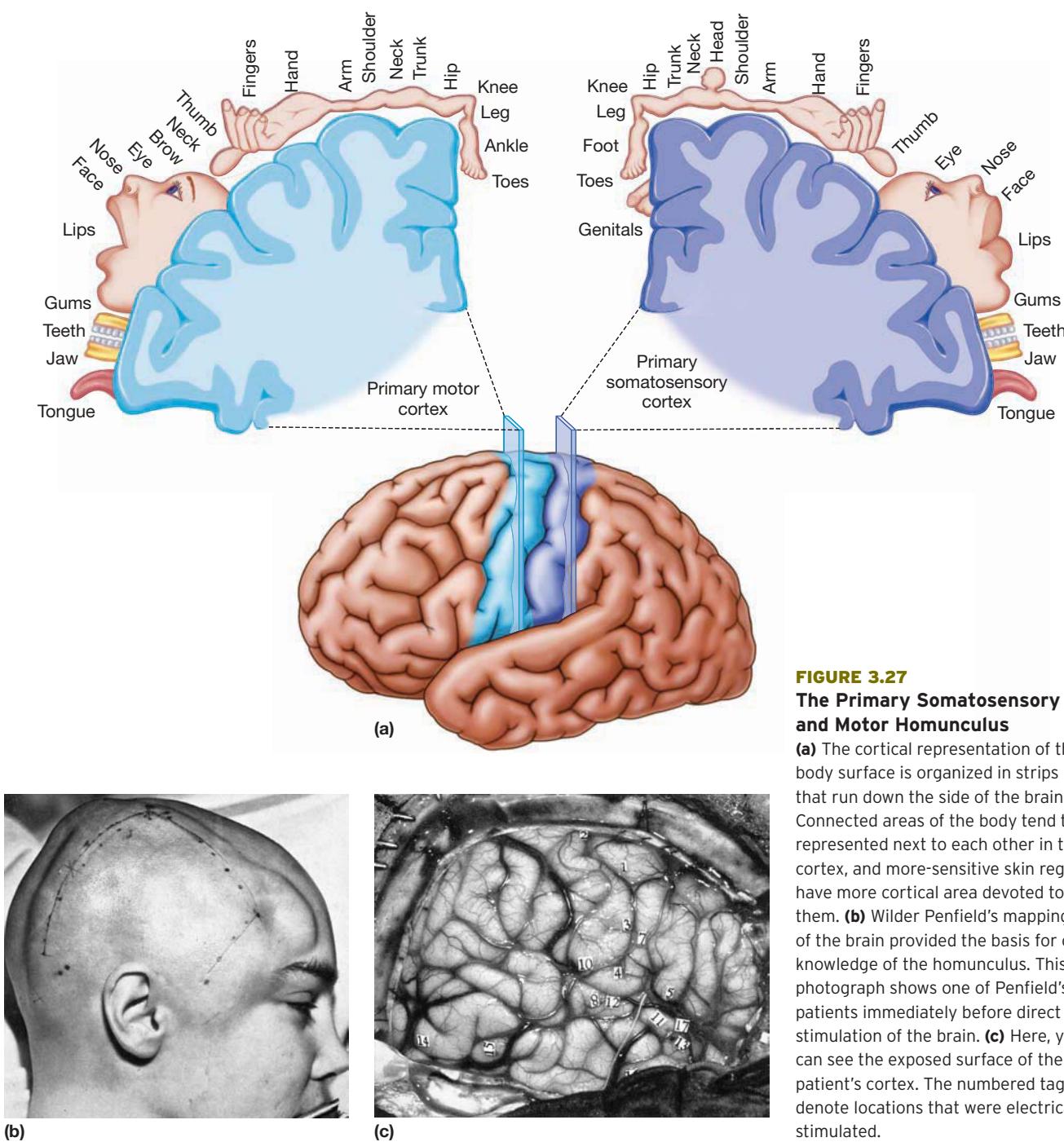
the right side of the body, and the right hemisphere receives touch information from the left side of the body. In each parietal lobe, this information is directed to the *primary somatosensory cortex*, a strip in the front part of the lobe, running from the top of the brain down the sides. The primary somatosensory cortex groups nearby sensations: For example, sensations on the fingers are near sensations on the palm. The result, covering the primary somatosensory area, is a distorted representation of the entire body: the *somatosensory homunculus* (the latter term is Greek for “little man”). The homunculus is distorted because more cortical area is devoted to the body’s more sensitive areas, such as the face and the fingers (**FIGURE 3.27A**).

### occipital lobes

Regions of the cerebral cortex—at the back of the brain—important for vision.

### parietal lobes

Regions of the cerebral cortex—in front of the occipital lobes and behind the frontal lobes—important for the sense of touch and for attention to the environment.



**FIGURE 3.27**

### The Primary Somatosensory and Motor Homunculus

**(a)** The cortical representation of the body surface is organized in strips that run down the side of the brain. Connected areas of the body tend to be represented next to each other in the cortex, and more-sensitive skin regions have more cortical area devoted to them. **(b)** Wilder Penfield's mappings of the brain provided the basis for our knowledge of the homunculus. This photograph shows one of Penfield's patients immediately before direct stimulation of the brain. **(c)** Here, you can see the exposed surface of the patient's cortex. The numbered tags denote locations that were electrically stimulated.



**FIGURE 3.28**  
**Hemineglect**

This drawing, made by a hemineglect patient, omits much of the flower's left side.

#### temporal lobes

Regions of the cerebral cortex—below the parietal lobes and in front of the occipital lobes—important for processing auditory information, for memory, and for object and face perception.

#### frontal lobes

Regions of the cerebral cortex—at the front of the brain—important for movement and higher-level psychological processes associated with the prefrontal cortex.

#### prefrontal cortex

The frontmost portion of the frontal lobes, especially prominent in humans; important for attention, working memory, decision making, appropriate social behavior, and personality.

This homunculus is based on brain mappings by the pioneering neurological researcher Wilder Penfield. Penfield created these mappings as he examined patients who were to undergo surgery for epilepsy (**FIGURE 3.27B**). The idea behind this work was to perform the surgery without damaging brain areas vital for functions such as speech. After a local anesthetic was applied to the scalp and while the patient was awake, Penfield would electrically stimulate regions of the brain and ask the patient to report what he or she was experiencing (**FIGURE 3.27C**). Penfield's studies provided important evidence about the amount of brain tissue devoted to each sensory experience.

The parietal lobe is also involved in attention. A stroke or other damage to the right parietal region can result in the neurological disorder *hemineglect*. Patients with this syndrome fail to notice anything on their left side even though their eyes work perfectly well. Looking in a mirror, they will shave or put makeup on only the right side of their face. If two objects are held up before them, they will see only the one on the right. Asked to draw a simple object, they will draw only its right half (**FIGURE 3.28**).

The **temporal lobes** hold the *primary auditory cortex*, the brain region responsible for hearing. Also within the temporal lobes are specialized visual areas (for recognizing detailed objects such as faces), plus the hippocampus and the amygdala (both critical for memory, as discussed earlier). At the intersection of the temporal and occipital lobes is the *fusiform face area*. Its name comes from the fact that this area is much more active when people look at faces than when they look at other things. In contrast, other regions of the temporal lobe are more activated by objects, such as houses or cars, than by faces. Damage to the fusiform face area can cause specific impairments in recognizing people but not in recognizing objects.

The **frontal lobes** are essential for planning and movement. The rearmost portion of the frontal lobes is the *primary motor cortex*. The primary motor cortex includes neurons that project directly to the spinal cord to move the body's muscles. Its responsibilities are divided down the middle of the body, like those of the sensory areas: For example, the left hemisphere controls the right arm, whereas the right hemisphere controls the left arm. The rest of the frontal lobes consists of the **prefrontal cortex**, which occupies about 30 percent of the brain in humans. Scientists have long thought that what makes humans unique in the animal kingdom is our extraordinarily large prefrontal cortex. However, there is evidence that what separates humans from other animals is not how much of the brain the prefrontal cortex occupies but rather the complexity and organization of prefrontal circuits—the way different regions within the prefrontal cortex are connected (Bush & Allman, 2004; Schoenemann, Sheehan, & Glotzer, 2005).

Parts of the prefrontal cortex are responsible for directing and maintaining attention, keeping ideas in mind while distractions bombard people from the outside world, and developing and acting on plans. The entire prefrontal cortex is indispensable for rational activity. It is also especially important for many aspects of human social life, such as understanding what other people are thinking, behaving according to cultural norms, and contemplating one's own existence. It provides both the sense of self and the capacity to empathize with others or feel guilty about harming them.

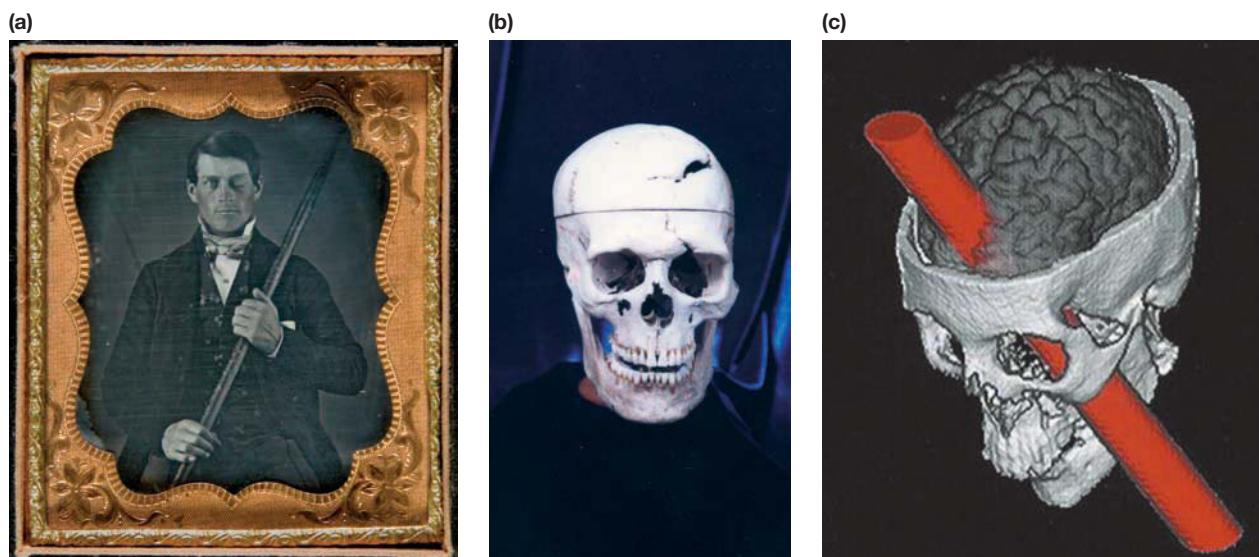
**THE PREFRONTAL CORTEX IN CLOSE-UP** Psychologists have learned a great deal of what they know about the functioning of different brain regions through the careful study of people whose brains have been damaged by disease or injury. Perhaps the most famous historical example of brain damage is the case of Phineas Gage. Gage's case provided the basis for the first modern theories of the prefrontal cortex's role in both personality and self-control.

In 1848, Gage was a 25-year-old foreman on the construction of Vermont's Rutland and Burlington Railroad. One day, he dropped a tool called a tamping iron, which was over a yard long and an inch in diameter. The iron rod hit a rock, igniting some blasting powder. The resulting explosion drove the rod into his cheek, through his frontal lobes, and clear out through the top of his head (**FIGURE 3.29**). Gage was still conscious as he was hurried back to town on a cart, and he was able to walk, with assistance, upstairs to his hotel bed. He wryly remarked to the awaiting physician, "Doctor, here is business enough for you." He said he expected to return to work in a few days. In fact, Gage lapsed into unconsciousness and remained unconscious for two weeks. Afterward, his condition steadily improved. Physically, he recovered remarkably well.

Unfortunately, Gage's accident led to major personality changes. Whereas the old Gage had been regarded by his employers as "the most efficient and capable" of workers, the new Gage was not. As one of his doctors later wrote, "The equilibrium or balance, so to speak, between his intellectual faculties and animal propensities seems to have been destroyed. He is fitful, irreverent, indulging at times in the grossest profanity . . . impatient of restraint or advice when it conflicts with his desires. . . . A child in his intellectual capacity and manifestations, he has the animal passions of a strong man" (Harlow, 1868, p. 340). In summary, Gage was "no longer Gage."

Unable to get his foreman's job back, Gage exhibited himself in various New England towns and at the New York Museum (owned by the circus showman P. T. Barnum). He worked at the stables of the Hanover Inn at Dartmouth College. In Chile, he drove coaches and tended horses. After a decade, his health began to decline, and in 1860 he started having epileptic seizures and died within a few months. Gage's recovery was initially used to argue that the entire brain works uniformly and that the healthy parts of Gage's brain had taken over the work of the damaged parts. However, the medical community eventually recognized that Gage's psychological impairments had been severe and that some areas of the brain in fact have specific functions.

Reconstruction of Gage's injury through examination of his skull has made it clear that the prefrontal cortex was the area most damaged by the tamping rod (Damasio, Grabowski, Frank, Galaburda, & Damasio, 1994). Recent studies of patients



**FIGURE 3.29 Phineas Gage**

Analysis of Gage's damaged skull provided the basis for the first modern theories about the role of the prefrontal cortex in both personality and self-control. (a) This photo shows Gage holding the rod that passed through his skull. (b) Here, you can see the hole in the top of Gage's skull. (c) This computer-generated image reconstructs the rod's probable path through the skull.



**FIGURE 3.30**

### Lobotomy

This photo shows Dr. Walter Freeman performing a lobotomy in 1949. Freeman is inserting an ice pick-like instrument under the upper eyelid of his patient to cut the nerve connections in the front part of the brain.

with injuries to this brain region reveal that it is particularly concerned with social phenomena, such as following social norms, understanding what other people are thinking, and feeling emotionally connected to others. People with damage to this region do not typically have problems with memory or general knowledge, but they often have profound disturbances in their ability to get along with others.

In the late 1930s, António Egas Moniz developed the *lobotomy*, a form of brain surgery that deliberately damaged the prefrontal cortex (**FIGURE 3.30**). Why would a surgeon want to perform this procedure? At the beginning of the 20th century, there was a significant increase in the number of patients living in mental institutions, and psychiatrists sought a physical means of treating these patients. The lobotomy generally left patients lethargic, emotionally flat, and therefore much easier to manage. It also left them disconnected from their social surroundings. Most lobotomies were performed in the late 1940s and early 1950s. In 1949, Egas Moniz received the Nobel Prize for developing the procedure, which was phased out with the arrival of drugs to treat psychological disorders.

## Splitting the Brain Splits the Mind

Studying people who have undergone brain surgery has given researchers a better understanding of the conscious mind. For example, on rare occasions when epilepsy does not respond to modern medications, surgeons may remove the part of the brain in which the seizures begin. Another strategy, pioneered in the 1940s and sometimes still practiced when other interventions have failed, is to cut connections within the brain to try to isolate the site where the seizures begin. After the procedure, a seizure that begins at that site is less likely to spread throughout the cortex.

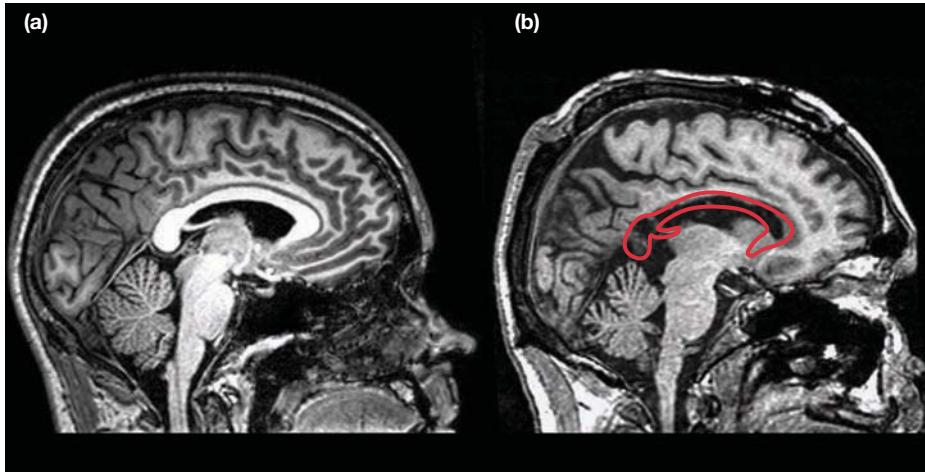
The major connection between the hemispheres that may readily be cut without damaging the gray matter is the corpus callosum (see Figure 3.26). When this massive fiber bundle is severed, the brain's halves are almost completely isolated from each other. The resulting condition is called **split brain**. This surgical procedure has provided many important insights into the basic organization and specialized functions of each brain hemisphere (**FIGURE 3.31**).

What is it like to have your brain split in half? Perhaps the most obvious thing about split-brain patients after their operations is how normal they are. Unlike patients after other types of brain surgery, split-brain patients have no immediately apparent problems. In fact, some early investigations suggested the surgery had not affected the patients in any discernible way. They could walk normally, talk normally, think clearly, and interact socially. In the 1960s, this book's coauthor Michael Gazzaniga, working with the Nobel laureate Roger Sperry, conducted a series of tests on split-brain patients. The results were stunning: Just as the brain had been split in two, so had the mind!

The hemispheres normally work together. Images from the visual field's left side (left half of what you are looking at) go to the right hemisphere. Images from the visual field's right side go to the left hemisphere (**FIGURE 3.32**). The left hemisphere also controls the right hand, and the right hemisphere controls the left hand. In a healthy person, the corpus callosum allows the hemispheres to communicate so that the right brain knows what the left is doing. By contrast, in split-brain patients, the hemispheres are separated, so this communication cannot take place—the hemispheres function as completely independent entities. This division allows researchers to independently examine the function of each hemisphere without the influence of the other. The researchers can provide information to, and receive information from, a single hemisphere at a time.

### split brain

A condition that occurs when the corpus callosum is surgically cut and the two hemispheres of the brain do not receive information directly from each other.



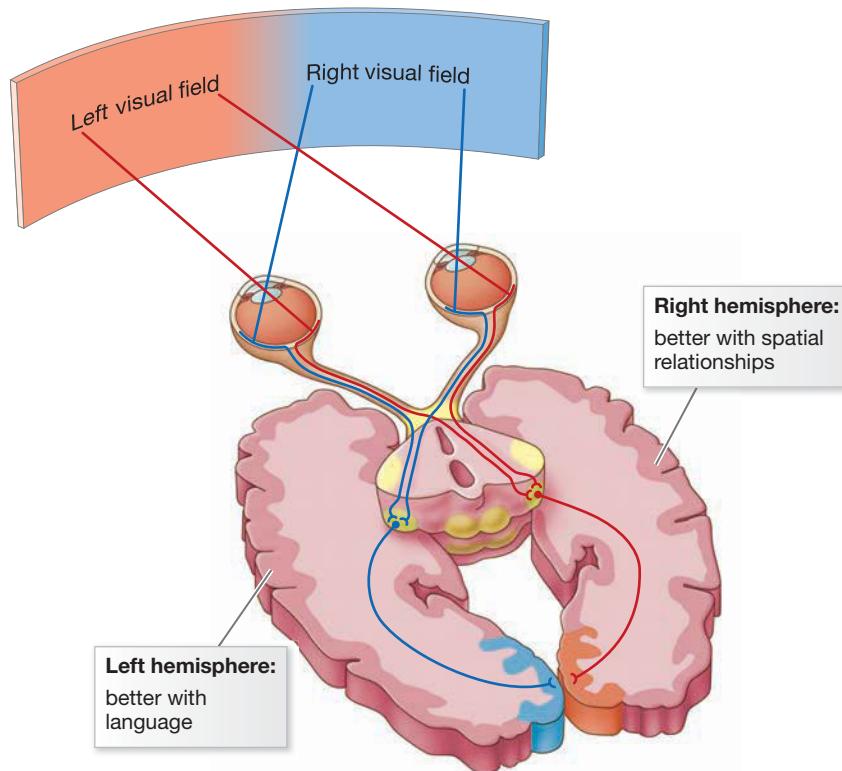
**FIGURE 3.31**

### Split Brain

(a) This image shows the brain of a normal person whose corpus callosum is intact. (b) This image shows the brain of a patient whose corpus callosum has been cut (area indicated by the red outline). With the corpus callosum severed, the two hemispheres of the brain are almost completely separated.

Psychologists have long known that in most people the left hemisphere is dominant for language. If a split-brain patient sees two pictures flashed on a screen briefly and simultaneously—one to the visual field's right side and one to the left side—the patient will report that only the picture on the right was shown. Why is this? The left hemisphere (or “left brain”), with its control over speech, sees only the picture on the right side. It is the only picture a person with a split brain can talk about.

In many split-brain patients, the right hemisphere has no discernable language capacity. The mute right hemisphere (or “right brain”), having seen the picture on the left, is unable to articulate a response. However, the right brain can act on its perception: If the picture on the left was of a spoon, the right hemisphere can easily pick out an actual spoon from a selection of objects. It uses the left hand, which is controlled by the right hemisphere. Still, the left hemisphere does not know what the right one saw.



**FIGURE 3.32**

### Visual Input

Images from the left side go to the brain's right hemisphere. Images from the right side go to the left hemisphere.

# What to Believe? Using Psychological Reasoning

## Failing to Notice Source Credibility: Are There “Left Brain” and “Right Brain” Types of People?

Many psychologists are leery about dealing with the popular press. They want psychological studies to become known by the public, but they do not want the findings to be garbled by the media. Seeing their research twisted in the press can be maddening in part because it overshadows the very findings the scientists have so proudly obtained. One of the authors of this textbook knows about such problems from personal experience.

As noted in the text, Michael Gazzaniga and Roger Sperry conducted research on the activity of the two hemispheres after the corpus callosum was severed. When the hemispheres have been surgically disconnected and are separately examined, each hemisphere displays different abilities. This discovery provided a wealth of data, but the media has gone far beyond Gazzaniga and Sperry's early findings.

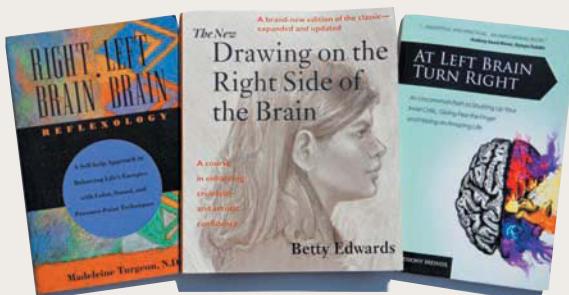
You have probably heard the idea that some people are “left brain” logical types and others are “right brain” artistic types. According to this popular notion, people differ to the extent that their right or left hemispheres dominate their thinking styles. Left-brain thinkers are said to be more analyti-

### The evidence is overwhelming: People are not either left-brain or right-brain dominant.

cal, rational, and objective. Right-brain thinkers are said to be more creative and to view the world more holistically and subjectively. Moreover, a dominant left brain supposedly suppresses right-brain creativity, so people could become more creative and passionate if their right hemisphere were released.

This false idea has permeated society (**FIGURE 3.33**). Multiple tests are available, particularly on the Internet, to determine whether you are left- or right-brain dominant. Countless pop psychology books give advice on living better by emphasizing your particular brain style or drawing on the other style. Teachers have been heavily influenced by the idea (Alferink & Farmer-Dougan, 2010). They have been urged to develop different classroom plans

for left-brain thinkers than for right-brain thinkers, and they have been encouraged to liberate the “more creative” right brain. According to one recent study, nearly 90 percent of teachers in the U.K. and the Netherlands believe in the idea of left-brain versus right-brain thinking (Dekker et al., 2012).



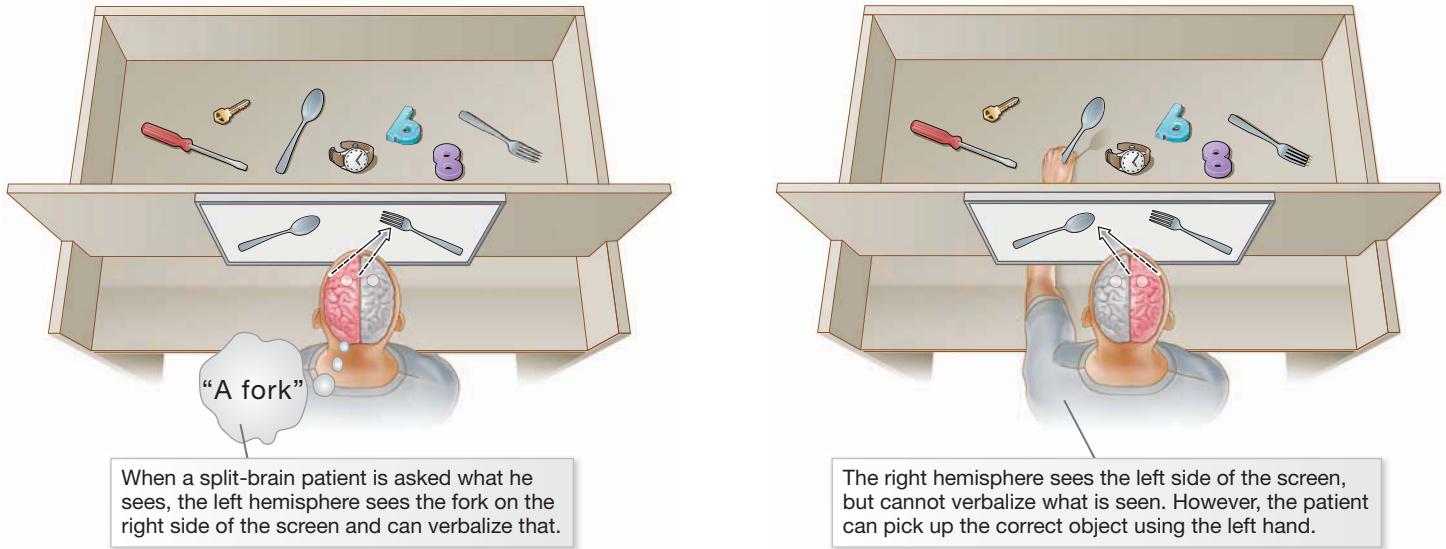
**FIGURE 3.33** Left Brain Versus Right Brain

The media has helped promote the false ideas that individuals are dominant on one side of the brain or the other and that such different styles are important for classroom learning.

As noted in Chapter 1, the media loves a good story. To make scientific studies attention grabbing, journalists often oversimplify research findings and apply them in ways that go far beyond what can be concluded from the evidence. In this case, the evidence is overwhelming: People are not either left-brain or right-brain dominant (Hines, 1987).

The hemispheres are specialized for certain functions, such as language or spatial relationships. However, each hemisphere is capable of carrying out most cognitive processes, though sometimes in different ways. Most cognitive processes involve the coordinated efforts of both hemispheres. A recent study that examined brain activity in over 1,000 individuals ages 7 to 29 found no differences between people in the extent to which their right or left hemisphere was active (Nielsen et al., 2013). In contrast to the theory that a liberated right brain leads to better learning, some evidence suggests that people who perform best at math are those whose two hemispheres work most closely together (Prescott et al., 2010).

Of course, whenever you read media stories about psychological findings, you need to think about the source of your information. If you are really interested in the finding, consider looking up the original article to see if the journalist represented that article accurately. This advice is especially important if you plan to use the information in your life. Findings from psychological science often have practical implications for daily living, but the value of research can be spoiled if the media outlet spreading the information gets it wrong.



**FIGURE 3.34 Split-Brain Experiment: The Left Hemisphere Versus the Right Hemisphere**

Splitting the brain, then, produces two half brains. Each half has its own perceptions, thoughts, and consciousness (**FIGURE 3.34**).

Normally, the competencies of each hemisphere complement each other. The left brain is generally hopeless at spatial relationships, whereas the right hemisphere is much more proficient. In one experiment (Bogen & Gazzaniga, 1965), a split-brain participant is given a pile of blocks and a drawing of a simple arrangement in which to put them. For example, the participant is asked to produce a square. When using the left hand, controlled by the right hemisphere, the participant arranges the blocks effortlessly. However, when using the right hand, controlled by the left brain, the participant produces only an incompetent, meandering attempt. During this dismal performance, the right brain presumably grows frustrated, because it makes the left hand try to slip in and help! You will learn more about split-brain patients in Chapter 4, “Consciousness.”

## Summing Up

### What Are the Basic Brain Structures and Their Functions?

- Early researchers debated the relationship between human brain structures and brain functions. New imaging techniques have advanced our understanding of the brain.
- The spinal cord carries sensory information from the body to the brain and motor information from the brain to the body. It also produces reflexes.
- The brain stem serves survival functions, such as breathing, swallowing, and urination.
- At the back of the brain stem is the cerebellum. This structure is associated with coordinated movement, balance, and motor learning.
- Beneath the cerebral cortex are a number of structures that serve unique functions: The hypothalamus regulates bodily functions. The thalamus serves as a way station through which sensory information travels to the cortex. The hippocampus is involved in memory formation. The amygdala influences emotional states. The structures of the basal ganglia are involved in the planning and production of movement as well as in reward.
- The cerebral cortex is the outer surface of the brain and is divided into lobes. The occipital lobes are associated with vision. The parietal lobes are associated with touch and attention. The temporal lobes are associated with hearing, memory, facial perception, and object

perception. The frontal lobes, which contain the prefrontal cortex, are associated with movement, higher-level psychological processes, and personality.

- When the two hemispheres of the brain are surgically split, the left hemisphere displays different abilities than the right hemisphere.

## Measuring Up

1. Match each of the following brain structures with its role or function. (You will need to remember these terms and facts to understand later discussions of learning, memory, emotions, mental illness, anxiety, and other aspects of mind and behavior.)

Brain structure	Role/function
a. brain stem	1. primary structure for memory
b. cerebellum	2. sensory relay station
c. basal ganglia	3. important for emotions
d. hypothalamus	4. divided into four lobes
e. thalamus	5. regulates vital functions such as body temperature
f. hippocampus	6. involved in reward
g. amygdala	7. regulates breathing and swallowing
h. cerebral cortex	8. "little brain," involved in movement

2. Match each lobe of the brain with its function.

Lobe	Function
a. frontal	1. hearing
b. occipital	2. thought
c. parietal	3. touch
d. temporal	4. vision

(2) a. 2; b. 4; c. 3; d. 1.

ANSWERS: (1) a. 7; b. 8; c. 6; d. 5; e. 2; f. 4; g. 3; h. 4.

## Learning Objectives

- Differentiate between the subdivisions of the nervous system.
- Identify the primary structures of the endocrine system.
- Explain how the nervous system and the endocrine system communicate to control thought, feeling, and behavior.

## 3.3 How Does the Brain Communicate with the Body?

Recall that the peripheral nervous system (PNS) transmits a variety of information to the central nervous system (CNS). It also responds to messages from the CNS to perform specific behaviors or make bodily adjustments. In the production of psychological activity, however, both of these systems interact with a different mode of communication within the body, the endocrine system.

### The Peripheral Nervous System Includes the Somatic and Autonomic Systems

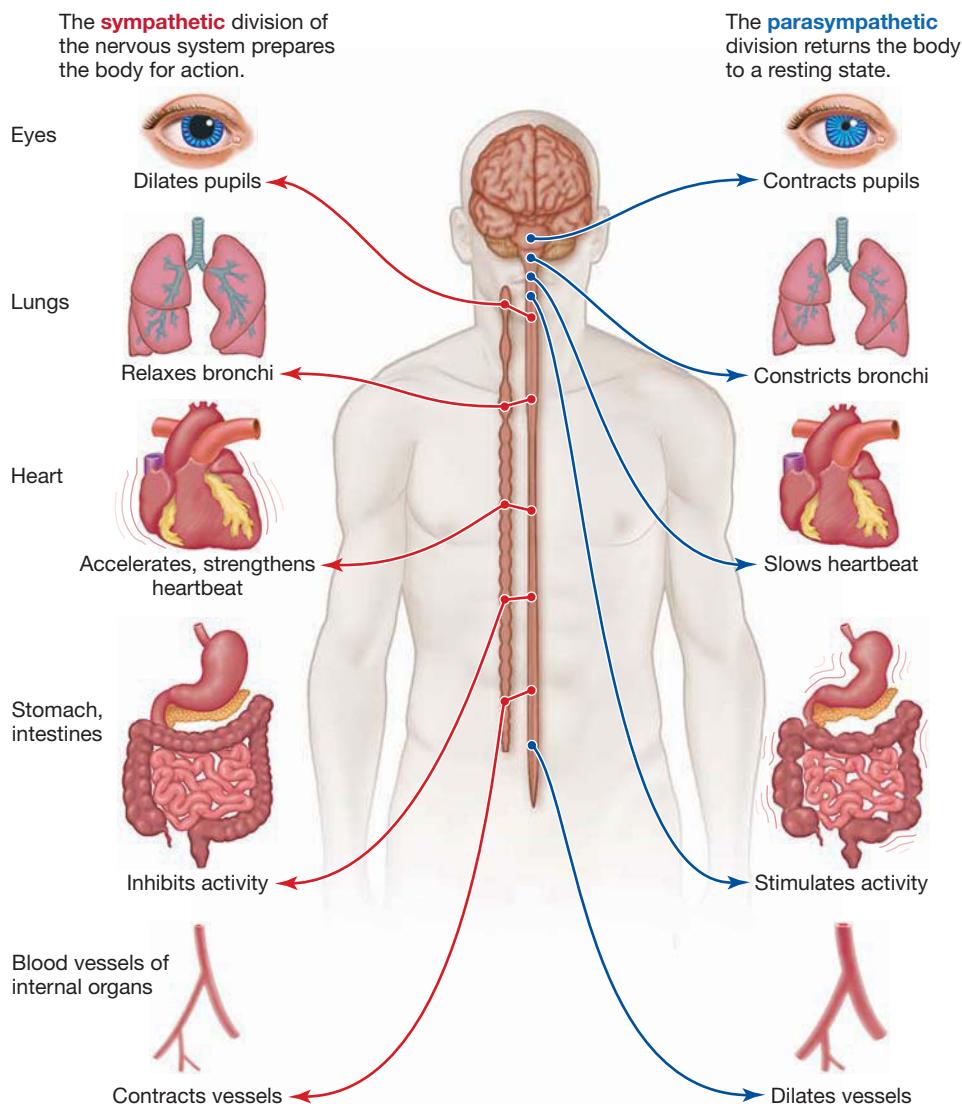
Recall that the PNS has two primary components: the somatic nervous system and the autonomic nervous system (see Figure 3.3). The **somatic nervous system (SNS)** transmits sensory signals to the CNS via nerves. Specialized receptors in the skin, muscles, and joints send sensory information to the spinal cord, which relays it to the brain. In addition, the CNS sends signals through the SNS to muscles, joints, and skin to initiate, modulate, or inhibit movement.

The second major component of the PNS, the **autonomic nervous system (ANS)**, regulates the body's internal environment by stimulating glands (such as sweat glands) and by maintaining internal organs (such as the heart). Nerves in the ANS also carry somatosensory signals from the glands and internal organs to the CNS.

These signals provide information about, for example, the fullness of your stomach or how anxious you feel.

**SYMPATHETIC AND PARASYMPATHETIC DIVISIONS** Two types of signals, sympathetic and parasympathetic, travel from the central nervous system to organs and glands, controlling their activity (**FIGURE 3.35**). To understand these signals, imagine you hear a fire alarm. In the second after you hear the alarm, signals go out to parts of your body telling them to prepare for action. As a result, blood flows to skeletal muscles; epinephrine is released, increasing your heart rate and blood sugar; your lungs take in more oxygen; your digestive system suspends activity as a way of conserving energy; your pupils dilate to maximize visual sensitivity; and you perspire to keep cool.

These preparatory actions are prompted by the autonomic nervous system's **sympathetic division**. Should there be a fire, you will be physically prepared to flee. If the alarm turns out to be false, your heart will return to its normal steady beat, your breathing will slow, you will resume digesting food, and you will stop perspiring. This return to a normal state will be prompted by the ANS's **parasympathetic division**. Most of your internal organs are controlled by inputs from sympathetic and parasympathetic systems. The more aroused you are, the greater the sympathetic system's dominance.



#### somatic nervous system (SNS)

A component of the peripheral nervous system; it transmits sensory signals and motor signals between the central nervous system and the skin, muscles, and joints.

#### autonomic nervous system (ANS)

A component of the peripheral nervous system; it transmits sensory signals and motor signals between the central nervous system and the body's glands and internal organs.

##### sympathetic division

A division of the autonomic nervous system; it prepares the body for action.

##### parasympathetic division

A division of the autonomic nervous system; it returns the body to its resting state.

**FIGURE 3.35**  
**The Sympathetic and Parasympathetic Divisions of the Autonomic Nervous System**

### endocrine system

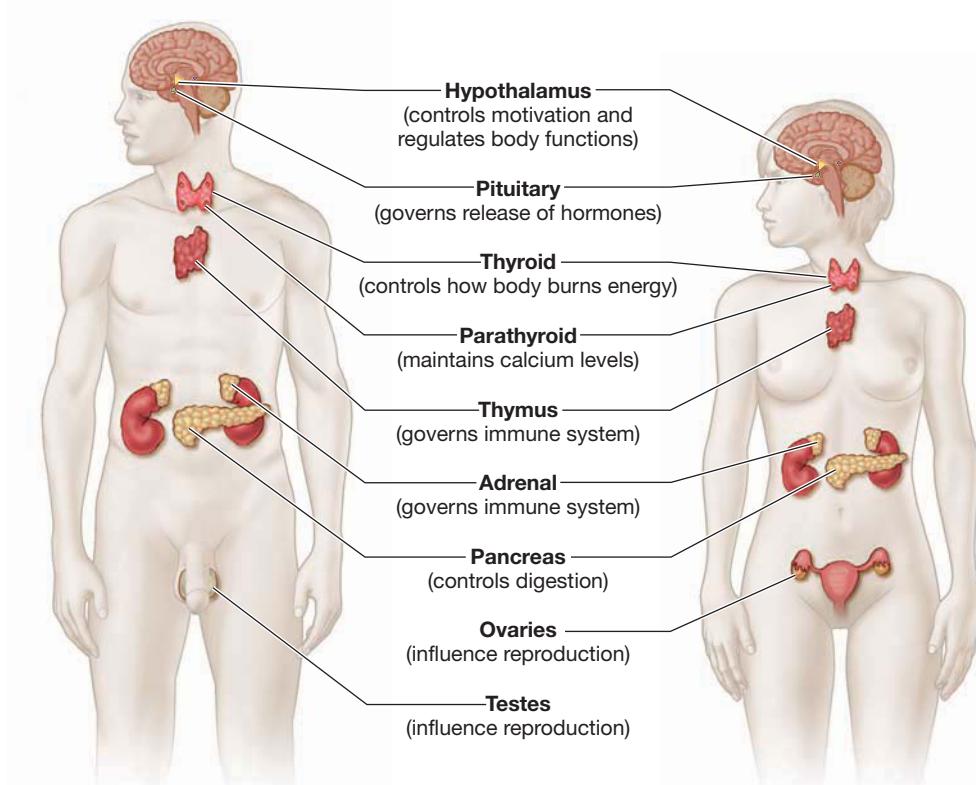
A communication system that uses hormones to influence thoughts, behaviors, and actions.

It does not take a fire alarm to activate your sympathetic nervous system. For example, when you meet someone you find attractive, your heart beats quickly, you perspire, you might start breathing heavily, and your pupils widen. Such signs of sexual arousal provide nonverbal cues during social interaction. These signs occur because sexual arousal has activated the ANS's sympathetic division. The SNS is also activated by psychological states such as anxiety or unhappiness. Some people worry a great deal or do not cope well with stress. Their bodies are in a constant state of arousal. Important research in the 1930s and 1940s by Hans Selye demonstrated that chronic activation of the SNS is associated with medical problems that include heart disease and asthma. Selye's work is discussed further in Chapter 11, "Health and Well-Being."

## The Endocrine System Communicates Through Hormones

Like the nervous system, the **endocrine system** is a communication network that influences thoughts, behaviors, and actions. Both systems work together to regulate psychological activity. For instance, from the nervous system the brain receives information about potential threats to the organism. The brain communicates with the endocrine system to prepare the organism to deal with those threats. (The threats could involve physical injury or be psychological, such as nervousness at having to talk in front of a group.) The main differences between the two systems are in their mode and speed of communication: Whereas the nervous system is fast and uses electrochemical signals, the endocrine system is slower and uses *hormones*.

**Hormones** are chemical substances released into the bloodstream by the ductless *endocrine glands*, such as the pancreas, thyroid, and testes or ovaries (FIGURE 3.36). Once released, hormones travel through the bloodstream until they reach their target tissues, where they bind to receptor sites and influence the tissues. Because they travel through the bloodstream, hormones can take from seconds to hours to exert



**FIGURE 3.36**  
The Hypothalamus and the Major Endocrine Glands

their effects. Once hormones are in the bloodstream, their effects can last for a long time and affect multiple targets.

**HORMONES' EFFECTS ON SEXUAL BEHAVIOR** An example of hormonal influence is in sexual behavior. The main endocrine glands influencing sexual behavior are the **gonads**: the testes in males and the ovaries in females. Although many people talk about “male” and “female” hormones, the two major gonadal hormones are identical in males and females. What differs is the quantity: *Androgens* such as testosterone are more prevalent in males, whereas *estrogens* such as estradiol and progesterone are more prevalent in females. Gonadal hormones influence the development of secondary sex characteristics (e.g., breast development in females, growth of facial hair in males). Gonadal hormones also influence adult sexual behavior.

For males, successful sexual behavior depends on having at least a minimum amount of testosterone. Prior to puberty, surgical removal of the testes, or *castration*, diminishes the capacity for developing an erection and lowers sexual interest. Yet a man castrated after puberty will be able to perform sexually if he receives an injection of testosterone. Testosterone injections do not increase sexual behavior in healthy men, however, and this finding suggests that a healthy man needs only a minimum amount of testosterone to perform sexually (Sherwin, 1988).

In females, the influence of gonadal hormones is much more complex. Many nonhuman female animals experience a finite period, *estrus*, when the female is sexually receptive and fertile. During estrus, the female displays behaviors designed to attract the male. Surgical removal of the ovaries terminates estrus: No longer receptive, the female ends her sexual behavior. However, injections of estrogen reinstate estrus. Women’s sexual behavior may have more to do with androgens than estrogens (Morris, Udry, Khan-Dawood, & Dawood, 1987). According to pioneering work by Barbara Sherwin (1994, 2008), women with higher blood levels of testosterone report greater interest in sex, and testosterone injections increase women’s sexual interest after surgical removal of the uterus.

Women’s sexual activity is not particularly linked to the menstrual cycle (Breedlove, Rosenzweig, & Watson, 2007). However, when they are ovulating, heterosexual women find men who look and act masculine more attractive (Gangestad, Simpson, Cousins, Garver-Apgar, & Christensen, 2004), and they show greater activity in brain regions associated with reward while viewing attractive male faces (Rupp et al., 2009). In addition, women report having lower self-esteem when ovulating, and their greater motivation to find a mate during that time may increase their efforts to appear attractive (Hill & Durante, 2009). Indeed, one study found that when their fertility was highest, women showed up for a laboratory study wearing more-revealing clothing than they normally wore (Durante, Li, & Haselton, 2008). Multiple recent studies are now providing evidence that using hormonal contraceptives might significantly alter both female and male mate choice by removing the hormone-related mid-cycle change in preferences (for a review: Alvergne & Lummaa [2010]).

## Actions of the Nervous System and Endocrine System Are Coordinated

All the communication systems described in this chapter link neurochemical and physiological processes to behaviors, thoughts, and feelings. These systems are fully



"You've been charged with driving under the influence of testosterone."

### **hormones**

Chemical substances, released from endocrine glands, that travel through the bloodstream to targeted tissues; the tissues are subsequently influenced by the hormones.

### **gonads**

The main endocrine glands involved in sexual behavior: in males, the testes; in females, the ovaries.

### **pituitary gland**

A gland located at the base of the hypothalamus; it sends hormonal signals to other endocrine glands, controlling their release of hormones.

integrated and interact to facilitate survival. They use information from the organism's environment to direct adaptive behavioral responses. Ultimately, the endocrine system is under the central nervous system's control. The brain interprets external and internal stimuli, then sends signals to the endocrine system. The endocrine system responds by initiating various effects on the body and on behavior.

The endocrine system is primarily controlled by the hypothalamus (for the location of this structure, see Figure 3.36; for a more detailed look, see Figure 3.24) via signals to the **pituitary gland**, which is located at the base of the hypothalamus. Neural activation causes the hypothalamus to secrete a particular one of its many *releasing factors*. The particular releasing factor causes the pituitary to release a hormone specific to that factor, and the hormone then travels through the bloodstream to endocrine sites throughout the body. Once the hormone reaches the target sites, it touches off the release of other hormones, which subsequently affect bodily reactions or behavior. The pituitary is often referred to as the "master gland" of the body: By releasing hormones into the bloodstream, it controls all other glands and governs major processes such as development, ovulation, and lactation. This integration can be finely tuned.

Consider physical growth. *Growth hormone (GH)*, a hormone released from the pituitary gland, prompts bone, cartilage, and muscle tissue to grow or helps them regenerate after injury. Since the 1930s, many people have administered or self-administered GH to increase body size and strength. Many athletes have sought a competitive advantage by using GH. For example, in early 2013, the legendary cyclist Lance Armstrong admitted to using GH and other hormones, including testosterone, to gain a competitive advantage. In an interview with Oprah Winfrey, Armstrong claimed that because doping was so pervasive in the sport, it was impossible for any cyclist to win a major championship without doping (**FIGURE 3.37**).

The releasing factor for GH stimulates the eating of protein by making it especially enjoyable (Dickson & Vaccarino, 1994). The area of the hypothalamus that stimulates release of GH is also involved in sleep/wake cycles. Thus, the bursts of GH, the need for protein, and the consumption of protein are controlled by the body's internal clock. All these connections illustrate how the CNS, the PNS, and the endocrine system work together to ensure the organism's survival: These systems prompt the behaviors that provide the body with the substances it needs when it needs them.



**FIGURE 3.37**

### **Growth Hormone and Cycling**

In January 2013, Lance Armstrong appeared on *The Oprah Winfrey Show* to admit using doping techniques to enhance his cycling performance.

## **Summing Up**

### **How Does the Brain Communicate with the Body?**

- The central nervous system—the brain and spinal cord—attends to the body and its environment, initiates actions, and directs the peripheral nervous system and endocrine system to respond appropriately.
- The peripheral nervous system is made up of the somatic nervous system and autonomic nervous system. The autonomic nervous system controls sympathetic and parasympathetic activity.
- The endocrine system consists of a number of endocrine glands, such as the pituitary and the adrenal glands. The central nervous system, peripheral nervous system, and endocrine system use chemicals to transmit their signals. Transmission in the nervous system occurs across synapses, whereas transmission in the endocrine system uses hormones that travel through the bloodstream.
- Gonadal hormones (estrogen, progesterone, and testosterone) are important in the development of secondary sex characteristics and in sexual behavior.

- The hypothalamus controls the endocrine system by directing the pituitary to release hormones that affect other endocrine glands.
- The various communication systems are integrated and promote behavior that is adaptive to the organism's environment.

## Measuring Up

- Complete each statement by choosing one of the following terms: peripheral nervous system (PNS); somatic nervous system; autonomic nervous system (ANS); sympathetic division; parasympathetic division.
  - You are studying quietly in the library when a friend jumps out from behind a partition and scares you, making your heart race. Your \_\_\_\_\_ has been affected.
  - When you calm down and return to your former (not scared) state, your \_\_\_\_\_ is affected.
  - The \_\_\_\_\_ controls movement by carrying signals from the central nervous system to the muscles.
  - The \_\_\_\_\_ has two primary components: the somatic nervous system and the autonomic nervous system.
  - The \_\_\_\_\_ consists of two main divisions that regulate the body's internal environment.
- Which of the following statements are true? Choose as many as apply.
  - All (normal) people of both sexes secrete testosterone and estrogen.
  - Men have gonads, and women have ovaries.
  - The endocrine system acts more slowly than the nervous system.
  - Hormones are secreted from several places in the body.
  - The pituitary gland is called the master gland.
  - The central nervous system and the peripheral nervous system work together, whereas the endocrine system works independently.
  - Women's sexual responsiveness is related more to androgens (such as testosterone) than to estrogen.

(2) Choices a, c, d, e, and g are true.

**ANSWERS:** (1) a. sympathetic division; b. parasympathetic division; c. somatic nervous system (SNS); d. peripheral nervous system (PNS); e. autonomic nervous system (ANS).

## 3.4 How Does the Brain Change?

When Michelle Mack was a youngster, her parents realized that she was different from other children because even simple tasks could give her problems. They could not explain these differences. When Mack was 27 years old, they learned that she was missing the left hemisphere of her brain (**FIGURE 3.38**). Doctors suspected that Mack's condition was the result of a stroke she had experienced in the womb.

Without a left hemisphere, Mack should have shown severe deficits in skills processed in that half of the brain. For example, the left hemisphere controls language, and it controls motor actions for the right side of the body. Losing a hemisphere as an adult would result in devastating loss of function. But Mack's speech is only minimally affected, and she can move the right side of her body with some difficulty. Mack is able to lead a surprisingly independent life. She graduated from high school, has a job, pays her bills, and does chores. Where did her capabilities come from? Somehow, her right hemisphere developed language processing capabilities as well as functions that ordinarily occur across both hemispheres.

Michelle Mack's case shows that nurture can influence nature. Over time, Mack interacted with the world. Her experiences enabled her brain to reorganize itself. Her right hemisphere took over processing for the missing left hemisphere.

## Learning Objectives

- Explain how environmental factors, including experiences, influence brain organization.
- Describe sex differences in brain structure and function.

(a)



(b)



**FIGURE 3.38**  
**Michelle Mack and a Case of Extreme Plasticity**

(a) While in her mother's womb, Michelle Mack suffered a stroke that obliterated her left hemisphere (shown here, in a scan taken when she was an adult, as the black areas on the right). (b) Over time, Mack's right hemisphere took over the duties of the left hemisphere—language production and moving the right side of the body—to a surprising extent. Mack's case shows the plasticity of the brain.

### plasticity

A property of the brain that allows it to change as a result of experience or injury.

In fact, despite the great precision and the specificity of its connections, the brain is extremely adaptable. Over the course of development, throughout a constant stream of experience, and after injury, the brain continually changes. This property is known as **plasticity**.

## Experience Fine-Tunes Neural Connections

Connections form between brain structures when growing axons are directed by certain chemicals that tell them where to go and where not to go. The major connections are established by chemical messengers, but the detailed connections are governed by experience. If a cat's eyes are sewn shut at birth, depriving the animal of visual input, the visual cortex fails to develop properly. If the sutures are removed weeks later, the cat is permanently blind, even though its eyes function normally. Adult cats that are similarly deprived do not lose their sight (Wiesel & Hubel, 1963). Evidently, ongoing activity in the visual pathways is necessary in order to refine the visual cortex enough for it to be useful. In general, such plasticity has *critical periods*. During these times, particular experiences must occur for development to proceed normally.

To study the effects of experience on development, researchers reared rats in a number of different laboratory environments. For instance, one group was raised in deprived circumstances compared to that of normal laboratory rats, with minimal comfort and no opportunities for social interaction. Another group was raised in an enriched environment, with many companions, interesting things to look at, puzzles, obstacles, toys, running wheels, and even balance beams. The “luxury” items might simply have approximated rat life in the wild, but they enabled the luxury group to develop bigger, heavier brains than the deprived group (Rosenzweig, Bennett, & Diamond, 1972). Thus, it appears that experience is important for normal development and maybe even more so for superior development. Nowadays, as a result of these findings, most laboratory animals are kept in environments that provide enrichment (Simpson & Kelley, 2011). Some evidence suggests that the opportunity for physical exercise might have the most beneficial effects on brain development and learning (Mustroph et al., 2012).

## Females' and Males' Brains Are Mostly Similar but May Have Revealing Differences

Everything a person experiences alters his or her brain, and females and males differ in their life experiences. They also differ in their hormonal makeups. The differences between females' and males' brains reveal the intertwined influences of biology and environment. In general, males have larger brains than females, but for both sexes the sizes of brain structures are highly variable (Giedd et al., 1997). In any case, larger brains are not necessarily better, because longer distances between brain regions can translate into slower communication. Both in the womb and after birth, hormonal differences between the sexes affect brain development (Lombardo et al., 2012). As a result, hormonal difference might influence the way males and females differ on some cognitive tasks, such as the ease with which they mentally rotate objects or recall parts of a story (Kimura, 1999). But the different ways that men and women are treated in society may also contribute to these differences on cognitive tasks (Miller & Halpern, 2014).

There is evidence that men and women may perform the same task by using different parts of the brain. For example, Richard Haier and colleagues (2005) found that females and males may solve some complex problems, such as items on IQ tests, differently. Females show greater use of language-related brain regions, whereas males show greater use of spatial-related brain regions, even when participants are matched for intelligence.

As discussed earlier in the chapter, to some extent the brain's two hemispheres are lateralized: Each hemisphere is dominant for different cognitive functions. A considerable body of evidence indicates that females' brains are more bilaterally organized for language. In other words, the brain areas important in processing language are more likely to be found in both halves of females' brains. In males' brains the equivalent language areas are more likely to be in only one hemisphere, usually the left (Phillips et al., 2001; **FIGURE 3.39**).

One source of data that supports this distinction between male and female brains is people's experiences following strokes. Even when patients are matched on the location and severity of the brain damage caused by a stroke, women are less impaired in language use than men are (Jiang, Sheikh, & Bullock, 2006). A possible reason for women having better outcomes is that, because language is represented in both halves of their brain, damage to half of the brain will have less effect on a woman's ability to process language than it would if most of the language areas were in the damaged half of the brain.

A related hypothesis, in accord with the idea that women's brains are more bilaterally organized, is that the halves of women's brains are connected by more neural fibers than men's are. Remember that the corpus callosum connects the brain's two halves (see Figure 3.20). Some researchers have found that a portion of the corpus callosum is larger in women (Gur & Gur, 2004). More recently, researchers have shown that many of the connections in the typical female brain run from side to side across the hemispheres, whereas in the typical male brain they run from back to front within each hemisphere (Ingalhalikar et al., 2014).

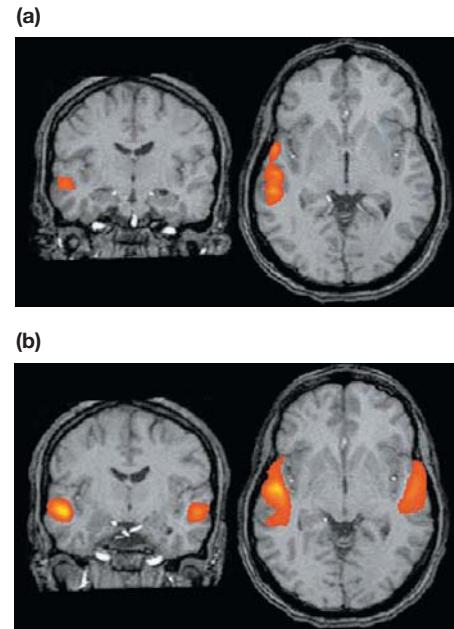
While differences between females' brains and males' brains may be revealing, in fact their brains are mostly similar. Ultimately, the interplay of biological and environmental effects on the brain is reflected in both the differences and the similarities between females' and males' brains.

## The Brain Rewires Itself Throughout Life

Brain plasticity decreases with age. Even into very old age, however, the brain can grow new connections among neurons and even grow new neurons. The rewiring and growth within the brain represents the biological basis of learning.

**CHANGE IN THE STRENGTH OF CONNECTIONS UNDERLIES LEARNING** In every moment of life, we gain memories: experiences and knowledge that are acquired instantaneously and may be recalled later, as well as habits that form gradually. All these memories are reflected in the brain's physical changes.

Psychologists widely accept that changes in the brain are most likely not in its larger wiring or general arrangement. The changes are mainly in the strength of existing connections. One possibility is that when two neurons fire simultaneously, the synaptic connection between them strengthens. The strengthened synaptic connection makes these neurons more likely to fire together in the future. Conversely, *not* firing simultaneously tends to weaken the connection between two neurons. This theory can be summarized by the catchphrase *fire together, wire together*. First proposed in the 1940s, by the renowned psychologist Donald Hebb (1949), this idea is consistent with much experimental evidence and many theoretical models. It accounts for two phenomena: the "burning in" of an experience (a pattern of neural firing becomes more likely to recur, and its recurrence leads the mind to recall an event) and the ingraining of habits (repeating a behavior makes the person tend to perform that behavior automatically). More rarely, entirely new connections grow between neurons. This new growth is a major factor in recovery from brain injury.

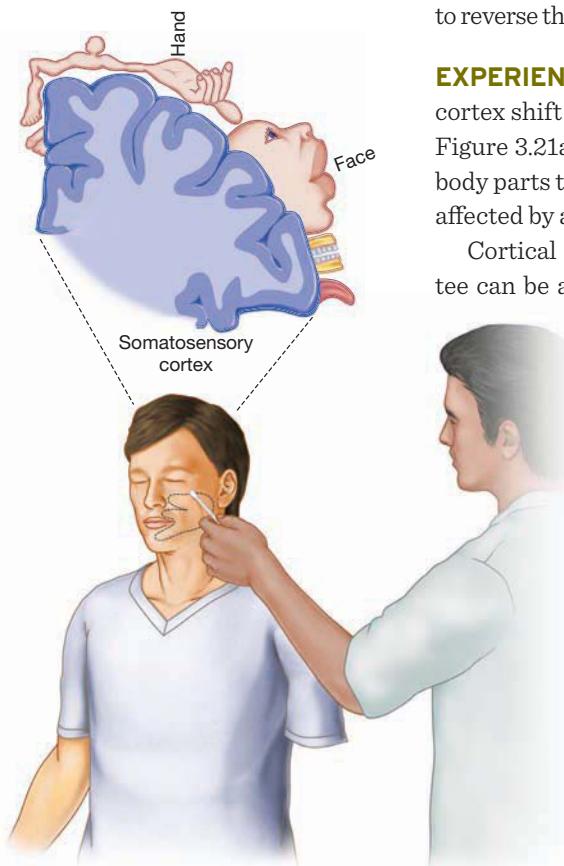


**FIGURE 3.39**  
**Males' Brains Versus Females' Brains**

A considerable body of evidence indicates that female brains are more bilaterally organized for language than males' brains. For example, researchers studied men and women listening to someone reading aloud. As these fMRI images show, (a) the men listened with one side of their brains, whereas (b) the women tended to listen with both sides.

Until about 20 years ago, scientists believed that adult brains produced no new brain cells. There is now evidence that new neurons are produced in some brain regions (Eriksson et al., 1998). The production of new neurons is called *neurogenesis*. A fair amount of neurogenesis apparently occurs in the hippocampus. Recall from earlier in this chapter that the hippocampus is involved in the storage of new memories. These memories are eventually transferred to the cortex as the hippocampus is continuously overwritten. Perhaps, without disrupting memory, neurons in the hippocampus can be lost and replaced.

Elizabeth Gould and her colleagues have demonstrated that environmental conditions can play an important role in neurogenesis. For example, they have found that for rats, shrews, and marmosets, stressful experiences—such as being confronted by strange males in their home cages—interfere with neurogenesis during development and adulthood (Gould & Tanapat, 1999). When animals are housed together, they typically form dominance hierarchies that reflect social status. Dominant animals, those who possess the highest social status, show greater increases in new neurons than subordinate animals do (Kozorovitskiy & Gould, 2004). Thus, social environment can strongly affect brain plasticity, a dynamic process we are only beginning to understand. Neurogenesis may underlie neural plasticity. If so, further research might enable us, through neurogenesis, to reverse the brain's natural loss of neurons, thereby slowing age-based mental decline.



**FIGURE 3.40**  
**Cortical Remapping Following Amputation**

The participant felt a cotton swab touching his cheek as if it were on his missing hand.

**EXPERIENCE CHANGES THE BRAIN** The functions of portions of the cerebral cortex shift in response to their activity. Recall the somatosensory homunculus (see Figure 3.21a). As that representation makes clear, more cortical tissue is devoted to body parts that receive more sensation or are used more. Again, wiring in the brain is affected by amount of use.

Cortical reorganization can also have bizarre results. For example, an amputee can be afflicted with a *phantom limb*, the intense sensation that the amputated body part still exists. Some phantom limbs are experienced as moving normally, such as being used to gesture in conversation, whereas some are frozen in position. Moreover, a phantom limb is often accompanied by pain sensations, which may result from the misgrowth of the severed pain nerves at the stump. The cortex misinterprets the pain as coming from the place where those nerves originally came from. This phenomenon suggests that the brain has not reorganized in response to the injury and that the missing limb's cortical representation remains intact.

The neurologist V. S. Ramachandran has discovered that an amputee who has lost a hand may, when his or her eyes are closed, perceive a touch on the cheek as if it were on the missing hand (Ramachandran & Hirstein, 1998). Apparently, on the somatosensory homunculus the hand is represented next to the face. The unused part of the amputee's cortex (the part that would have responded to the now-missing limb) assumes to some degree the function of the closest group, representing the face. Somehow, the rest of the brain has not kept pace with the somatosensory area enough to figure out these neurons' new job, so the neurons formerly activated by a touch on the hand are activated by a touch on the amputee's face. The brain still codes the input as coming from the hand, and thus the amputee experiences a "phantom hand" (**FIGURE 3.40**).

## The Brain Can Recover from Injury

Just as the brain reorganizes in response to amount of use, it also reorganizes in response to brain damage. Following an injury in the cortex, the surrounding gray

matter assumes the function of the damaged area, like a local business scrambling to pick up the customers of a newly closed competitor. This remapping seems to begin immediately, and it continues for years. Such plasticity involves all levels of the central nervous system, from the cortex down to the spinal cord.

Reorganization is much more prevalent in children than in adults, in accord with the sensitive periods of normal development. Young children afflicted with severe and uncontrollable epilepsy that has paralyzed one or more limbs sometimes undergo a *radical hemispherectomy*. This surgical procedure removes an entire cerebral hemisphere. Just as in the case of Michelle Mack, the remaining hemisphere eventually takes on most of the lost hemisphere's functions. The children regain almost complete use of their limbs. However, adults cannot undergo radical hemispherectomy. If the procedure were performed on adults, the lack of neural reorganization in their brains would lead to permanent paralysis and loss of function.

## Summing Up

### How Does the Brain Change?

- During development and across the life span, the circuitry of the brain is constantly reworked in response to experience.
- Males' brains and females' brains are mainly similar, but their differences may be revealing. Males' brains are larger, which does not necessarily mean better. Females' brains are organized more bilaterally for language. Men and women may perform the same cognitive task by using different parts of the brain.
- An understanding of the brain's organization and plasticity has enabled researchers to better understand conditions such as phantom limb syndrome. Neurogenesis, the creation of new neurons, may underlie neural plasticity.
- The brain can reorganize after a brain injury. However, children's brains demonstrate much greater reorganization after brain injury than adults' brains.

## Measuring Up

1. Which of the following statements are examples of how environment can affect brain development or function? Place an X next to each example.
  - \_\_\_\_\_ a. The hippocampus may be larger than average in experienced taxi drivers.
  - \_\_\_\_\_ b. Laboratory rats raised in enriched environments developed heavier brains than laboratory rats raised in standard environments.
  - \_\_\_\_\_ c. Neurogenesis is more likely in socially dominant animals than in subordinate ones.
  - \_\_\_\_\_ d. Lack of visual stimulation from birth can result in a lack of development of the visual cortex, even if the eyes function normally.
2. Indicate whether the following statements, about the ways in which females' and males' brains differ, are true or false.
  - \_\_\_\_\_ a. Males' brains generally are larger than females' brains.
  - \_\_\_\_\_ b. Males' brains are more likely to be bilaterally organized.
  - \_\_\_\_\_ c. Researchers have found that sex differences in the brain are caused entirely by hormonal differences.
  - \_\_\_\_\_ d. Sex differences in the brain indicate that males and females have essentially different abilities.
  - \_\_\_\_\_ e. Females tend to use language-related areas for problem-solving, whereas males tend to use spatial-related areas.

(2) a. true; b. false; c. false; d. true; e. false.

brain development or function.

**ANSWERS:** (1) Choices a, b, c, and d are all examples of how environment can affect

# Using Psychology in Your Life



## Will My Learning Disability Prevent Me from Succeeding in College?

Have you been diagnosed with a learning disability? Do you suspect you might have one?

According to the National Center for Learning Disabilities (2009), a learning disability is a “neurological disorder that affects the brain’s ability to receive, process, store, and respond to information.” One of the most common learning disabilities is dyslexia, which involves difficulties in acquiring and processing language, leading to problems with reading, spelling, or writing (**FIGURE 3.41**). Someone who has difficulty spelling or writing might, alternatively, have the learning disability dysgraphia, a disorder of written expression.

Learning disabilities may become apparent in childhood or later in life. Individuals might excel academically in high school, but the new academic and organizational challenges of college might help reveal a learning disability.

If you have a learning disability or suspect you have one, the earlier you seek help, the sooner you will have access to the resources available on your campus that will help you learn. Contact the disability support office or someone at Student Affairs, and they will be able to direct you. If your learning disability is verified, disability support office staff will work with you to determine the types of accommodations necessary to enable you to get the most of your academic experience.

Given your particular strengths and weaknesses in processing information, some types of accommodations will be helpful, whereas others will not. Disability support office staff will let your professors know you are entitled to a specific type of accommodation, but they will not tell your professors about the nature of your learning disability. For example, a disability support office staff member might send a note to your professors that reads “[Your name will go here], a student in your introductory psychology course, has provided evidence of a condition that requires academic accommoda-



**FIGURE 3.41**

### An Inspiring Example

The celebrity chef Jamie Oliver suffers from dyslexia. His disability has hardly kept him from achieving his career goals. Here, in June 2010, Oliver is announcing Home Cooking Skills, a new and inspirational program he has co-created to teach basic cooking skills to young people in England.

tion. As a result, please provide [him or her] with time and a half on exams and on in-class writing assignments.”

Of course, you can also speak directly with individual professors about your learning disability and the kinds of resources likely to help you. Linda Tessler, a psychologist who works with persons with learning disabilities, writes:

It must be clear that you are not asking for standards to be lowered. You are using tools to help you perform. To pass, you must perform the task that your classmates perform. You may, however, need to get there in a different way. Dyslexic students have to read the textbook just as nondyslexic students do. They may just do it differently through the use of books on tape. (Tessler, 1997, p. 2)

Will a learning disability prevent you from succeeding in college? Not if you can help it, and you can help it by advocating for yourself. Line up the resources you need to ensure that you are able to reap the rewards of college.

## 3.5 What Is the Genetic Basis of Psychological Science?

Jack Osbourne is experiencing the symptoms of MS because the neurons in his brain are becoming demyelinated. The affected neurons cannot carry the electrical signals that tell his muscles what to do. But why does he have this disease? Is it a genetic condition he inherited from his parents? Could environmental influences such as childhood nutrition be involved? Some researchers believe that people inherit a predisposition to respond to particular—as yet unknown—environmental triggers that produce MS. Whatever the cause of Jack Osbourne’s disorder, how he copes with the condition will depend partly on his psychological makeup.

So far, this chapter has presented the basic biological processes underlying psychological functions. The following section considers how genes and environment affect psychological functions. From the moment of conception, we receive the genes we will possess for the remainder of our lives, but to what extent do those genes determine our thoughts and behaviors? How do environmental influences, such as the families and cultures in which we are raised, alter how our brains develop and change?

Until the last few years, genetic research focused almost entirely on whether people possessed certain types of genes, such as genes for psychological disorders or for particular levels of intelligence. Although it is important to discover the effects of individual genes, this approach misses the critical role of environmental factors in shaping who we are. While the term *genetics* is typically used to describe how characteristics such as height, hair color, and eye color are passed along to offspring through inheritance, it also refers to the processes involved in turning genes “on” and “off.” Research has shown that environmental factors can affect **gene expression**. This term refers to whether a particular gene is turned on or off. Environmental factors may also influence how a gene, once turned on, influences our thoughts, feelings, and behavior.

Genetic predispositions are often important in determining the environments people select for themselves. So, once again, biology and environment mutually influence each other. All the while, biology and environment—in other words, one’s genes and every experience one ever has—fluence the development of one’s brain.

### All of Human Development Has a Genetic Basis

Within nearly every cell in the body is the genome for making the entire organism. The *genome* is the master blueprint that provides detailed instructions for everything from how to grow a gallbladder to where the nose gets placed on a face. Whether a cell becomes part of a gallbladder or a nose is determined by which genes are turned on or off within that cell, and these actions are in turn determined by cues from both inside and outside the cell. The genome provides the options, and the environment determines which option is taken (Marcus, 2004).

Within each cell are **chromosomes**. These structures are made of *deoxyribonucleic acid (DNA)*, a substance that consists of two intertwined strands of molecules in a double helix shape. Segments of those strands are called **genes** (**FIGURE 3.42**).

In a typical human, nearly every cell contains 23 pairs of chromosomes. One member of each pair comes from the mother, the other from the father. In other words, each parent contributes half of a person’s DNA, half of his or her genes.

Each gene—a particular sequence of molecules along a DNA strand—specifies an exact instruction to manufacture a distinct *polypeptide*. One or more polypeptides

### Learning Objectives

- Explain how genes are transmitted from parents to offspring.
- Discuss the goals and methods of behavioral genetics.
- Explain how environmental factors, including experience, influence genetic expression.

#### gene expression

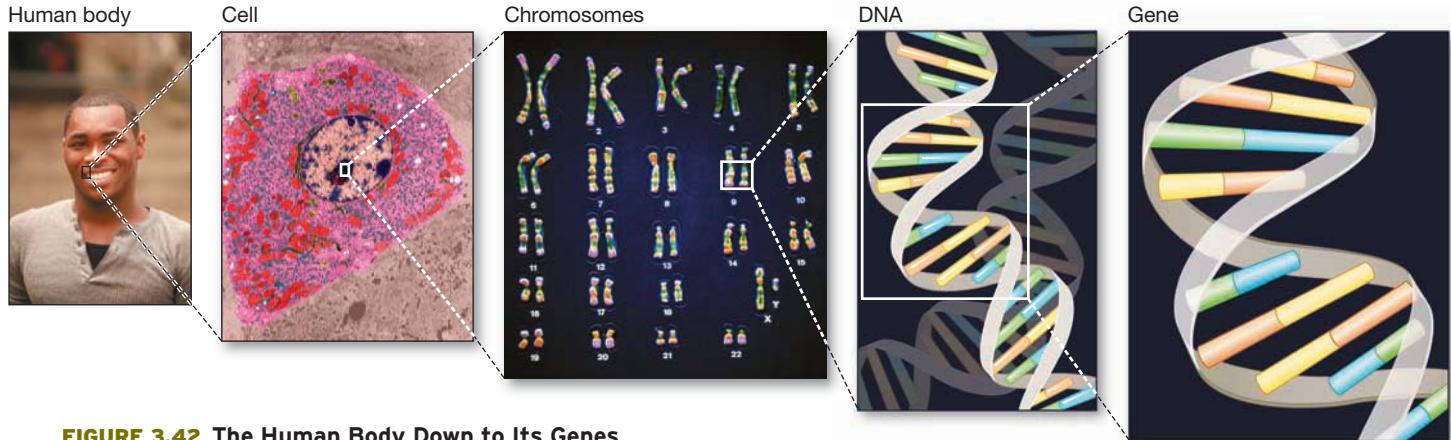
Whether a particular gene is turned on or off.

#### chromosomes

Structures within the cell body that are made up of DNA, segments of which comprise individual genes.

#### genes

The units of heredity that help determine the characteristics of an organism.



**FIGURE 3.42** The Human Body Down to Its Genes

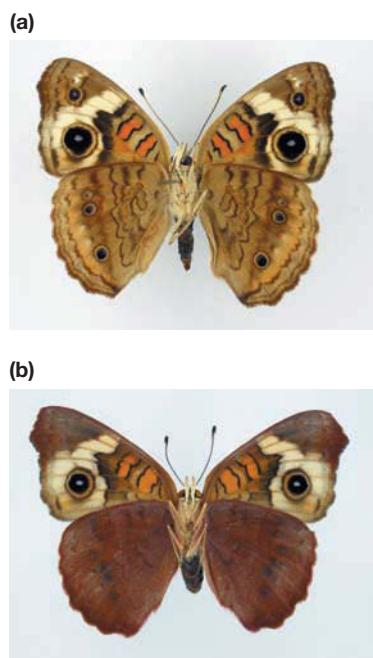
Each cell in the human body includes pairs of chromosomes, which consist of DNA strands. DNA has a double helix shape, and segments of it consist of individual genes.

make up a protein. Proteins are the basic chemicals that make up the structure of cells and direct their activities. There are thousands of different types of proteins, and each type carries out a specific task. The environment determines which proteins are produced and when they are produced.

For example, a certain species of butterfly becomes colorful or drab, depending on the season in which it is born (Brakefield & French, 1999). The environment causes a gene to be expressed during the butterfly's development that is sensitive to temperature or day length (**FIGURE 3.43**). Similarly, although each cell in the human body contains the same DNA, cells become specialized, depending on which of their genes are expressed. As noted earlier, gene expression determines the body's basic physical makeup, but it also determines specific developments throughout life. It is involved in all psychological activity. Gene expression allows us to sense, to learn, to fall in love, and so on.

In February 2001, two groups of scientists published separate articles that detailed the results of the first phase of the *Human Genome Project*, an international research effort. This achievement represents the coordinated work of hundreds of scientists around the world to map the entire structure of human genetic material. The first step of the Human Genome Project was to map the entire sequence of DNA. In other words, the researchers set out to identify the precise order of molecules that make up each of the thousands of genes on each of the 23 pairs of human chromosomes (**FIGURE 3.44**).

One of the most striking findings from the Human Genome Project is that people have fewer than 30,000 genes. That number means humans have only about twice as many genes as a fly (13,000) or a worm (18,000), not much more than the number in some plants (26,000), and fewer than the number estimated to be in an ear of corn (50,000). Why are we so complex if we have so few genes? The number of genes might be less important than subtleties in how those genes are expressed and regulated (Baltimore, 2001).



**FIGURE 3.43**  
Gene Expression and Environment

The North American buckeye butterfly has seasonal forms that differ in the color patterns on their wings. (a) Generations that develop to adulthood in the summer—when temperatures are higher—take the “linea” form, with pale beige wings. (b) Generations that develop to adult in the autumn—when the days are shorter—take the “rosa” form, with dark reddish-brown wings.

## Heredity Involves Passing Along Genes Through Reproduction

The first clues to the mechanisms responsible for heredity were discovered by the monk Gregor Mendel around 1866. The monastery where Mendel lived had a long history of studying plants. For studying inheritance, Mendel developed an



**FIGURE 3.44**  
**Human Genome Project**

A map of human genes is presented by J. Craig Venter, president of the research company Celera Genomics, at a news conference in Washington on February 12, 2001. This map is one part of the international effort by hundreds of scientists to map the entire structure of human genetic material.

**dominant gene**

A gene that is expressed in the offspring whenever it is present.

**recessive gene**

A gene that is expressed only when it is matched with a similar gene from the other parent.

**genotype**

The genetic constitution of an organism, determined at the moment of conception.

**phenotype**

Observable physical characteristics, which result from both genetic and environmental influences.

(a)



(b)



**FIGURE 3.45**  
**Parent Plants Display Genetic Differences**

Mendel studied pea plants. To observe the effects of cross-breeding, he started with (a) pea plants with purple flowers, and (b) pea plants with white flowers.

experimental technique, *selective breeding*, that strictly controlled which plants bred with which other plants.

In one simple study, Mendel selected pea plants that had either only purple flowers or only white flowers (**FIGURE 3.45**). He then cross-pollinated the two types to see which color of flowers the plants would produce. Mendel found that the first generation of pea offspring tended to be completely white or completely purple. If he had stopped there, he would never have discovered the basis of heredity. However, he then allowed each plant to self-pollinate into a second generation. This second generation revealed a different pattern: Of the hundreds of pea plants, about 75 percent had purple flowers and 25 percent had white flowers. This 3:1 ratio repeated itself in additional studies. It also held true for other characteristics, such as pod shape.

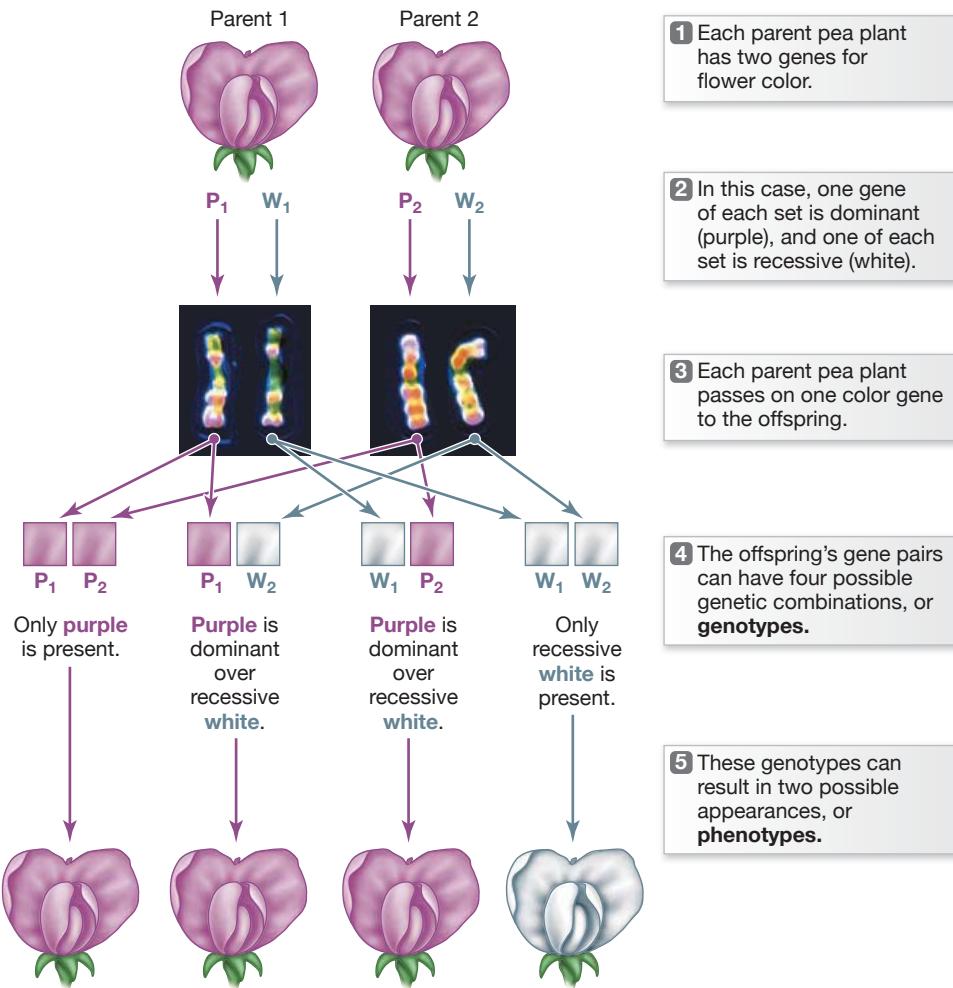
From this pattern, Mendel deduced that the plants contained separate units, now referred to as genes, that existed in different versions (e.g., white and purple). In determining an offspring's features, some of these versions would be dominant and others would be recessive. We now know that a **dominant gene** from either parent is expressed (becomes apparent or physically visible) whenever it is present. A **recessive gene** is expressed only when it is matched with a similar gene from the other parent. In pea plants, white flowers are recessive, so white flowers occur only when the gene for purple flowers is not present. All "white genes" and no purple ones was one of the four possible combinations of white and purple genes in Mendel's experiments.

**GENOTYPE AND PHENOTYPE** The existence of dominant and recessive genes means that not all genes are expressed. The **genotype** is an organism's genetic makeup. That genetic constitution is determined at the moment of conception and never changes. The **phenotype** is that organism's observable physical characteristics and is always changing.

Genetics, or nature, is one of the two influences on phenotype. So, for instance, in Mendel's experiments, two plants with purple flowers had the same phenotype but might have differed in genotype. Either plant might have had two (dominant) genes for purple. Alternatively, either plant might have had one (dominant) purple gene and

**FIGURE 3.46**  
**Genotypes and Phenotypes**

Mendel's experiments with cross-breeding pea plants resulted in purple flowers 75 percent of the time and white flowers 25 percent of the time.



one (recessive) white gene (FIGURE 3.46). Environment, or nurture, is the second influence on phenotype. For instance, humans inherit their height and skin color. But good nutrition leads to increased size, and sunlight can change skin color.

**POLYGENIC EFFECTS** Mendel's flower experiments dealt with single-gene characteristics. Such traits appear to be determined by one gene each. When a population displays a range of variability for a certain characteristic, such as height or intelligence, the characteristic is *polygenic*. In other words, the trait is influenced by many genes (as well as by environment).

Consider human skin color. There are not just three or four separate skin colors. There is a spectrum of colors. The huge range of skin tones among Americans alone shows that human skin color is not inherited the same way as flower color was in Mendel's research. The rich variety of skin colors (phenotype) is not the end product of a single dominant/recessive gene pairing (genotype). Instead, the variety shows the effects of multiple genes.

## Genotypic Variation Is Created by Sexual Reproduction

Although they have the same parents, siblings may differ from each other in many ways, such as eye color, height, and personality. These differences occur because each

person has a specific combination of genes. Most cells in the human body contain 23 pairs of chromosomes. These pairs include the sex chromosomes, which are denoted X and Y because of their shapes. Females have two X chromosomes. Males have one X chromosome and one Y (**FIGURE 3.47**).

After one sperm and one egg combine during fertilization, the resulting fertilized cell, known as a *zygote*, contains 23 pairs of chromosomes. Half of each pair of chromosomes comes from the mother, and the other half comes from the father. From any two parents, 8 million different combinations of the 23 chromosomes are possible. The net outcome is that a unique genotype is created at conception, accounting for the *genetic variation* of the human species.

The zygote grows through *cell division*. This process has two stages: First the chromosomes duplicate. Then the cell divides into two new cells with an identical chromosome structure. Cell division is the basis of the life cycle and is responsible for growth and development.

### GENETIC MUTATIONS: ADVANTAGEOUS, DISADVANTAGEOUS, OR BOTH?

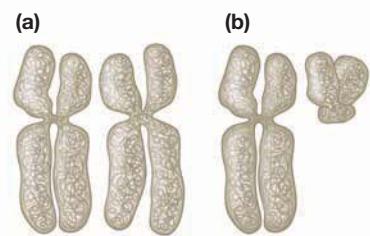
Errors sometimes occur during cell division, leading to *mutations*, or alterations in the DNA. Most mutations are benign and have little influence on the organism. Occasionally, a genetic mutation produces a selective advantage or disadvantage in terms of survival or reproduction. That is, mutations can be adaptive or maladaptive. The evolutionary significance of such a change in adaptiveness is complex. If a mutation produces an ability or behavior that proves advantageous to the organism, that mutation may spread through the population. The mutation may spread because those who carry the gene are more likely to survive and reproduce.

Consider *industrial melanism*. This phenomenon accounts for the fact that in areas of the world with heavy soot or smog, moths and butterflies tend to be darker in color. What has created this dark coloration? Before industrialization, landscapes (trees, buildings, etc.) were lighter in color. Predators were more likely to spot darker insects against pale backgrounds, so any mutation that led to darker coloring in insects was disadvantageous and was eliminated quickly through natural selection. But with industrialization, pollution darkened the landscapes. Darker coloring in insects therefore became advantageous and more adaptive because the darker insects were harder to see against the darker backgrounds (**FIGURE 3.48**).

What about genetic mutations that are disadvantageous adaptively, such as by leading to disease? Genes that lead to diseases that do not develop until well beyond reproductive age do not have a reproductive disadvantage and are not removed from the population. The dominance or recessiveness of a gene also helps determine if it remains in the gene pool.

For instance, *sickle-cell disease* is a genetic disorder that alters the bloodstream's processing of oxygen. It can lead to pain, organ and bone damage, and anemia. The disease occurs mainly in African Americans: Approximately 8 percent of African Americans are estimated to have the (recessive) gene for it (Centers for Disease Control and Prevention, 2011b). Because the sickle-cell gene is recessive, only those African Americans who receive it from both parents will develop the disease. Those who receive a recessive gene from only one parent have what is called *sickle-cell trait*. They may exhibit symptoms under certain conditions (such as during exercise), but they will have a generally healthy phenotype in spite of a genotype that includes the trait (**FIGURE 3.49**).

Recessive genes do not interfere with most people's health. For this reason, the recessive genes for diseases such as sickle-cell anemia can survive in the gene pool. This particular gene also has some benefit in that it increases resistance to malaria, a parasitic disease prevalent in certain parts of Africa. People with only one sickle-cell



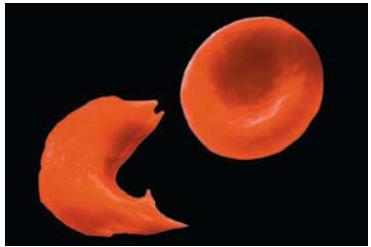
**FIGURE 3.47**  
**Sex Chromosomes**

(a) In females, the 23rd pair of chromosomes consists of two X chromosomes. (b) In males, the 23rd pair consists of one X and one Y chromosome. The Y chromosome is much smaller than the X chromosome.



**FIGURE 3.48**  
**Industrial Melanism**

These moths illustrate industrial melanism. As shown here, it is easier to spot light-colored insects against dark backgrounds. Because predators have an easier time catching insects they can spot, darker moths and darker butterflies are better able to survive in more-polluted areas. As a result, the lighter moths and lighter butterflies in those areas tend to die off, leaving more of the moths and butterflies with the selective advantage of darkness.



**FIGURE 3.49**  
**Sickle-Cell Disease**

Sickle-cell disease occurs when people receive recessive genes for the trait from both parents. It causes red blood cells to assume the distinctive "sickle" shape seen here in the left cell. Sickle-cell disease is most common among African Americans.

gene enjoy this resistance without suffering from sickle-cell disease. In contrast to recessive gene disorders like this one, most dominant gene disorders are lethal for most of their carriers and therefore do not last in the gene pool.

## Genes Affect Behavior

What determines the kind of person you are? What factors make you more or less bold, intelligent, or able to read a map? Your abilities and your psychological traits are influenced by the interaction of your genes and the environment in which you were raised or to which you are now exposed. The study of how genes and environment interact to influence psychological activity is known as *behavioral genetics*. Behavioral genetics has provided important information about the extent to which biology influences mind, brain, and behavior.

Any research suggesting that abilities to perform certain behaviors are biologically based is controversial. Most people do not want to be told that what they can achieve is limited or promoted by something beyond their control, such as their genes. It is easy to accept that genes control physical characteristics such as sex, race, eye color, and predisposition to diseases such as cancer and alcoholism. But can genes determine whether people will get divorced, how happy they are, or what careers they choose?

Increasingly, science indicates that genes lay the groundwork for many human traits. From this perspective, people are born essentially like undeveloped photographs: The image is already captured, but the way it eventually appears can vary based on the development process. Psychologists study the ways in which characteristics are influenced by nature, nurture, and their combination. In other words, who we are is determined by how our genes are expressed in distinct environments.

**BEHAVIORAL GENETICS METHODS** Most of us, at one time or another, have marveled at how different siblings can be, even those raised around the same time and in the same household. The differences are to be expected, because most siblings do not share identical genes or identical life experiences. Within the household and outside it, environments differ subtly and not so subtly. Siblings have different birth orders. Their mother may have consumed different foods and other substances during her pregnancies. They may have different friends and teachers. Their parents may treat them differently. Their parents are at different points in their own lives.

It is difficult to know what causes the similarities and differences between siblings, who always share some genes and often share much of their environments. Therefore, behavioral geneticists use two methods to assess the degree to which traits are inherited: twin studies and adoption studies.

*Twin studies* compare similarities between different types of twins to determine the genetic basis of specific traits. **Monozygotic twins**, or *identical twins*, result from one zygote (fertilized egg) dividing in two. Each new zygote, and therefore each twin, has the same chromosomes and the same genes on each chromosome (**FIGURE 3.50A**). However, monozygotic twins' DNA might not be as identical as long thought, due to subtle differences in how the mother's and father's genes are combined (Bruder et al., 2008). **Dizygotic twins**, sometimes called *fraternal* or *nonidentical twins*, result when two separately fertilized eggs develop in the mother's womb simultaneously. The resulting twins are no more similar genetically than any other pair of siblings (**FIGURE 3.50B**). To the extent that monozygotic twins are more similar than dizygotic twins, the increased similarity is considered most likely due to genetic influence.

*Adoption studies* compare the similarities between biological relatives and adoptive relatives. Nonbiological adopted siblings may share similar home environments,

### monozygotic twins

Also called *identical twins*; twin siblings that result from one zygote splitting in two and therefore share the same genes.

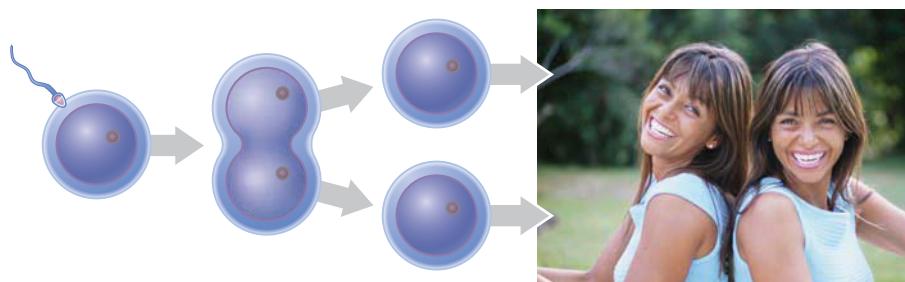
### dizygotic twins

Also called *fraternal twins*; twin siblings that result from two separately fertilized eggs and therefore are no more similar genetically than nontwin siblings.

### (a) Monozygotic (identical) twins

One sperm fertilizes one egg...

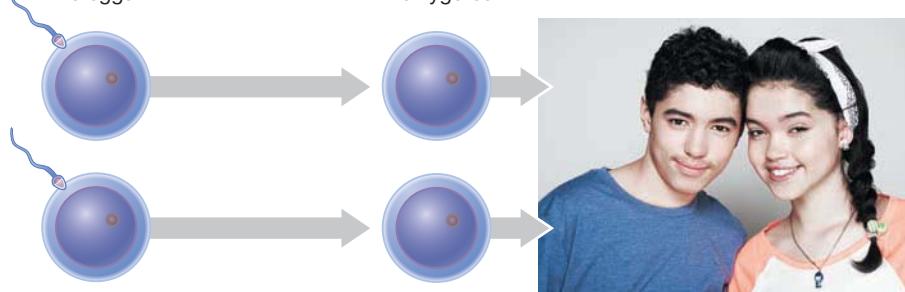
and the zygote splits in two.



### (b) Dizygotic (fraternal) twins

Two sperm fertilize two eggs...

which become two zygotes.



but they will have different genes. Therefore, the assumption is that similarities among nonbiological adopted siblings have more to do with environment than with genes.

How much influence would you say your home life has had on you? It turns out that growing up in the same home has relatively little influence on many traits, including personality traits. Indeed, after genetic similarity is controlled for, even biological siblings raised in the same home are no more similar than two strangers plucked at random off the street. (This point is examined in greater detail in Chapter 9, "Human Development," and Chapter 13, "Personality.")

One way to conduct a behavioral genetic study is to compare monozygotic twins who have been *raised together* with ones who were *raised apart*. Thomas Bouchard and his colleagues at the University of Minnesota identified more than 100 pairs of identical and nonidentical twins, some raised together and some raised apart (1990). The researchers examined a variety of these twins' characteristics, including intelligence, personality, well-being, achievement, alienation, and aggression. The general finding from the Minnesota Twin Project was that identical twins, whether they were raised together or not, were likely to be similar (**FIGURE 3.51**).

Some critics have argued that most of the adopted twins in the Minnesota study were raised in relatively similar environments. This similarity came about, in part, because adoption agencies try to match the child to the adoptive home. However, this argument does not explain the identical twins Oskar Stohr and Jack Yufe, who were born in Trinidad in 1933 (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). Oskar was raised Catholic in Germany and eventually joined the Nazi Party. Jack was raised Jewish in Trinidad and lived for a while in Israel. Few twins have more-different backgrounds. Yet when they met, at an interview for the study, they were wearing similar clothes, exhibited similar mannerisms, and shared odd habits, such as flushing the toilet before using it, dipping toast in coffee, storing rubber bands on their wrists, and enjoying startling people by sneezing loudly in elevators.

## FIGURE 3.50

### The Two Kinds of Twins

**(a)** Monozygotic (identical) twins result when one fertilized egg splits in two. **(b)** Dizygotic (fraternal) twins result when two separate eggs are fertilized at the same time.



### FIGURE 3.51

#### Identical Twins Raised Apart Are Also Similar

Identical twins Gerald Levey and Mark Newman, participants in Dr. Bouchard's study, were separated at birth. Reunited at age 31, they discovered they were both firefighters and had similar personality traits.

Some critics feel that nothing more than coincidence is at work in these case studies. They argue that if a researcher randomly selected any two people of the same age, many surprising similarities would exist in those people and their lives, just by coincidence, even if the people and their lives differed in most other ways. But twins and other relatives share similarities beyond coincidental attributes and behavior quirks. For instance, intelligence and personality traits such as shyness tend to run in families because of strong genetic components.

Moreover, there is some evidence that twins raised apart may be more similar than twins raised together. This phenomenon might occur if parents encouraged individuality in twins raised together by emphasizing different strengths and interests as a way of helping each twin develop as an individual. In effect, the parents would actively create a different environment for each twin.

**UNDERSTANDING HERITABILITY** *Heredity* is the transmission of characteristics from parents to offspring by means of genes. A term that is often confused with *heredity* but means something different is **heritability**. This term refers to the proportion of the variation in some specific trait in a population, not in an individual, that is due to genetics. That is, the trait cannot be due to environment or random chance.

Consider a specific trait, height, in a particular population, American women. The heritability for a trait depends on the *variation*: the measure of the overall difference among a group of people for that particular trait. To know the heritability of height, we need to know how much individual American women vary in that trait. Once we know the typical amount of variation within the population, we can see whether people who are related—sisters or a mother and daughter—show less variation than women chosen at random.

Say that within the population of American women, height has a heritability of .60. This figure means that 60 percent of the variation in height among American women is genetic. It does not mean that any individual necessarily gets 60 percent of her height from genetics and 40 percent from environment. Heritability estimates aid in identifying the causes of differences between individuals in a population.

For researchers to perform a heritability analysis, there must be variation in the population. For instance, almost everyone has two legs. There is very little variability in the population. More people lose legs through accidents than are born without them. Thus, the heritability value for having two legs is nearly zero, despite the obvious fact that the human genome includes instructions for growing two legs. The key lesson here is: Estimates of heritability are concerned only with the extent that people differ in terms of their genetic makeup within the group. So, the next time you hear that some trait or other is heritable, you need to appreciate that this refers to the distribution of that trait within a group, not to particular persons in that group.

## Social and Environmental Contexts Influence Genetic Expression

In a longitudinal study of criminality, Avshalom Caspi and his colleagues (2002) followed a group of more than 1,000 New Zealanders from their births in 1972–73 until adulthood. The group was made up of all the babies that were born in the town of Dunedin over the course of a year. Every few years, the researchers collected enormous amounts of information about the participants and their lives. When the participants were 26 years old, the investigators examined which factors predicted who became a violent criminal.

### heritability

A statistical estimate of the extent to which variation in a trait within a population is due to genetics.

Prior research had demonstrated that children who are mistreated by their parents are more likely to become violent offenders. But not all mistreated children become violent, and these researchers wanted to know why not. They hypothesized that the enzyme monoamine oxidase (MAO) is important in determining susceptibility to the effects of mistreatment, because low levels of MAO have been implicated in aggressive behaviors (this connection is discussed further in Chapter 12, “Social Psychology”).

The gene that controls MAO is called MAOA and comes in two forms. One form of the MAOA gene leads to higher levels of MAO, and the other form leads to lower levels. Caspi and colleagues found that boys with the low-MAOA gene appeared to be especially susceptible to the effects of early-childhood mistreatment. Those boys were also much more likely to be convicted of a violent crime than those with the high-MAOA gene. Only 1 in 8 boys was mistreated *and* had the low-MAOA gene. That minority, however, were responsible for nearly half of the violent crimes committed by the group (see “Scientific Thinking: Caspi’s Study of the Influence of Environment and Genes,” on p. 124).

The New Zealand study is a good example of how nature and nurture together affect behavior—in this case, unfortunately, violent behavior. Nature and nurture are inextricably entwined.

**EPIGENETICS** An exciting new field of genetic study is *epigenetics* (Berger, Kouzarides, Shiekhattar, & Shilatifard, 2009; Holliday, 1987). This term literally means “on top of genetics.” Here, environment is seen as layered over genetics. Epigenetics researchers are looking at the processes by which the environment affects genetic expression. They have found that various environmental exposures do not alter DNA, but they *do* alter DNA expression. That alteration makes it more or less likely that a gene will be expressed. For example, living under stress or consuming a poor diet makes some genes more active and some less active.

According to recent research, these changes in how DNA is expressed can be passed along to future generations (Daxinger & Whitelaw, 2012). The process is somewhat like giving a child a broken toy and saying, “Here is the toy, but it doesn’t work very well.” The child may then give that toy to his or her own child. The biological mechanisms are too complex to consider here. A simple way to think about epigenetic processes is that a parent’s experiences create tags on DNA that tell it when to express, and these tags are passed along with the DNA. They may then be passed along to future generations.

The potential implications of epigenetics for understanding health problems and health benefits are enormous. It is possible that smoking cigarettes or drinking alcohol, like chronic stress or bad nutrition, can create epigenetic tags (Pembrey et al., 2006). Further research will reveal how individuals’ life circumstances might change how their genes operate and how such processes may affect future generations (Grossniklaus, Kelly, Ferguson-Smith, Pembrey, & Lindquist, 2013).

## Genetic Expression Can Be Modified

Researchers can employ various gene manipulation techniques to enhance or reduce the expression of a particular gene or even to insert a gene from one animal species into the embryo of another. The researchers can then compare the genetically modified animal with an unmodified one to test theories about the affected gene’s function (**FIGURE 3.52**). Such techniques have dramatically increased our understanding of how gene expression influences thought, feeling, and behavior.

For instance, some of the transgenic mice discussed in Chapter 2 are called *knockouts*. Within these research mice, particular genes have been “knocked out,” or rendered inactive by being removed from the genome or disrupted within the genome. If a gene is important for a specific function, knocking out that gene should interfere



**FIGURE 3.52**  
**Genetic Modifications**

The two white mice and three brown mice in this photo are genetically normal. The sixth mouse is hairless because it has been genetically modified. Specifically, this mouse has received two *nu* genes, which cause the “nude” mutation. These genes also affect the immune system, so the mouse is a good laboratory subject for studies related to immune function.

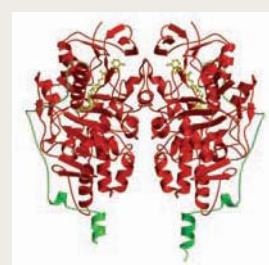
# Scientific Thinking

## Caspi's Study of the Influence of Environment and Genes

**HYPOTHESIS:** The MAOA gene regulates enzyme monoamine oxidase (MAO) and may be important in determining susceptibility to the effects of maltreatment, because low levels of MAO have been implicated in aggressive behaviors.

### RESEARCH METHOD:

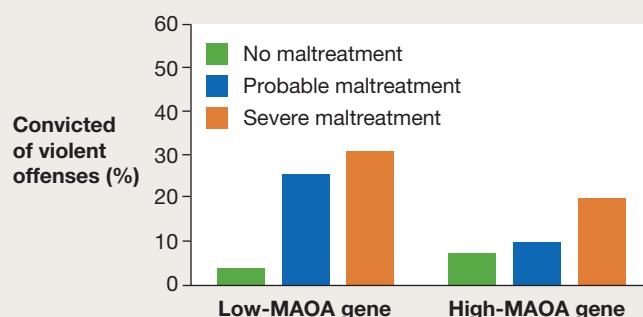
- 1 A group of more than 1,000 New Zealanders were followed from birth to adulthood.
- 2 Researchers measured which children were mistreated by their parents (**nurture**).
- 3 Researchers measured the presence of the MAOA gene, which comes in two forms. One form leads to higher levels of MAO, and the other form leads to lower levels (**nature**).



- 4 Researchers measured the tendency toward criminal behavior.



**RESULTS:** Those who had the MAOA gene for low MAO activity were much more likely than others to have been convicted of violent crimes if they had been maltreated as children. The effects of maltreatment had less influence on those with the high-MAOA gene.



**CONCLUSION:** Nature and nurture can work together to affect human behavior.

**SOURCE:** Caspi, A., McClay, J., Moffit, T. E., Mill, J., Martin, J., Craig, I. W., et al. (2002). Role of genotype in the cycle of violence in maltreated children. *Science*, 29, 851–854.

with the function. This experimental technique has revolutionized genetics, and in recognition the 2007 Nobel Prize was awarded to the three scientists who developed it: Mario Capecchi, Oliver Smithies, and Sir Martin Evans.

One remarkable finding from genetic manipulation is that changing even a single gene can dramatically change behavior. Through various gene manipulations, researchers have created anxious mice, hyperactive mice, mice that cannot learn or remember, mice that groom themselves to the point of baldness, mice that fail to take care of their offspring, and even mice that progressively increase alcohol intake when stressed (Marcus, 2004; Ridley, 2003).

In one study, a gene from the highly social prairie vole was inserted into the developing embryos of normally antisocial mice. The resulting transgenic mice exhibited social behavior more typical of prairie voles than of mice (Insel & Young, 2001). Another study found that knocking out specific genes led mice to forget other mice they had previously encountered. These “knockouts” also failed to investigate new mice placed in their cages, though normal mice would do so readily. In essence, knocking out one gene led to multiple impairments in social recognition (Choleris et al., 2003).

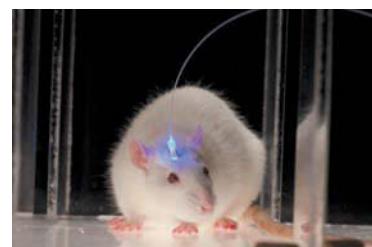
These findings do not indicate that mice have a specific gene for being social. It indicates that—in mice and in humans—changing one gene’s expression leads to the expression or nonexpression of a series of other genes. This effect ultimately influences even complex behaviors. In other words, genes seldom work in isolation to influence mind and behavior. Rather, complex interaction among thousands of genes gives rise to the complexity of human experience.

**OPTOGENETICS** One problem with most studies of brain function is that they use correlational methods. Recall from Chapter 2 that correlational techniques do not allow us to show causality. For example, fMRI studies show which areas of the brain are most active while a person performs a task. These findings do not mean there is a causal relationship between the brain activity and the task.

To address this limitation, scientists have recently pioneered *optogenetics*. This research technique provides precise control over when a neuron fires. That control enables researchers to better understand the causal relationship between neural firing and behavior. Optogenetics combines the use of light (optics) with gene alterations (Boyden et al., 2005; **FIGURE 3.53**). The genes are altered to change a particular subpopulation of neurons in the brain. Specifically, the membrane ion channels are changed within the neurons (recall that ion channels allow ions to enter the neuron and trigger action potentials). The changes to the membrane ion channels make these specific neurons sensitive to different types of light (e.g., red, green, blue). By inserting fiber optics into that region of the brain and shining a particular type of light, researchers are able to trigger action potentials in the neurons of interest (Williams & Deisseroth, 2013). Using similar techniques, researchers can modify neurons so that firing is inhibited when light is presented (Berndt, Lee, Ramakrishnan, & Deisseroth, 2014).

These techniques allow researchers to show that activating or deactivating specific neurons causes changes in brain activity or behavior. For instance, turning on one set of neurons led animals to act more anxiously (Tye et al., 2011). Turning off another set of neurons reduced cocaine use in animals addicted to that drug (Stefanik et al., 2013).

However, shining a light in a particular brain region will not be used to change human behavior. Rather, the technique allows researchers to better understand the causal relationships between brain activity and behavior. The development of optogenetics is an excellent example of how cutting-edge methods allow researchers to ask increasingly direct questions about biology and behavior.



**FIGURE 3.53**  
**Optogenetics**

This mouse is showing optogenetic display.

## Summing Up

### What Is the Genetic Basis of Psychological Science?

- Human behavior is influenced by genes.
- People inherit physical attributes and personality traits from their parents.
- The Human Genome Project has mapped the basic sequence of DNA, providing information that will help scientists increase their understanding of individual differences in people's characteristics and develop treatments for genetically based disorders.
- In addition to studying the heritability of traits, researchers study how and when genes are expressed. Genetic expression is affected by environment, including experience.
- Epigenetics is the study of how environment can alter genetic expression.
- Scientific techniques, including the study of transgenic mice and optogenetics, help scientists learn more about how genes and brain activity control behavior.

## Measuring Up

### 1. What is the difference between genotype and phenotype?

- a. Genotype refers to an organism's genetic makeup. Phenotype refers to observable characteristics that result from genetic and environmental influences.
- b. Genotype refers to monozygotic twins' (nearly) identical genetic makeup. Phenotype refers to dizygotic twins' genetic makeup.
- c. Genotypes can be modified by experiences. Phenotypes can be modified only if the underlying genes are knocked out.
- d. Genotypes direct the experiences organisms seek for themselves. Phenotypes cannot affect environmental events.

### 2. What is the relation between optogenetics and gene manipulation studies?

- a. Gene manipulations and optogenetics alter membrane ion channels so that different neurotransmitters bind with receptors.
- b. Gene manipulations and optogenetics alter membrane ion channels so that optogenetics can trigger neural firing.
- c. Gene manipulations lead to epigenetic changes, but optogenetics does not.
- d. Optogenetic tags are passed to future generations, but gene manipulations are not.

(2) b. Gene manipulations alter membrane ion channels so that optogenetics can trigger neural firing.

ANSWERS: (1) a. Genotype refers to an organism's genetic makeup. Phenotype refers to observable

# Your Chapter Review

## Chapter Summary

### 3.1 How Does the Nervous System Operate?

- **The Nervous System Has Two Basic Divisions:** Nerve cells, or neurons, are the basic units of the human nervous system. Neurons are linked as neural networks, and neural networks are linked together. The entire nervous system is divided into two basic units: the central nervous system (the brain and the spinal cord) and the peripheral nervous system (all the other nerve cells in the rest of the body).
- **Neurons Are Specialized for Communication:** Neurons receive and send electrical and chemical messages. All neurons have the same basic structure, but neurons vary by function and by location in the nervous system.
- **The Resting Membrane Potential Is Negatively Charged:** A neuron at rest is polarized. That is, it has a greater negative electrical charge inside than outside. The passage of negative and positive ions inside and outside the membrane is regulated by ion channels, such as those located at the nodes of Ranvier.
- **Action Potentials Cause Neural Communication:** Changes in a neuron's electrical charge are the basis of an action potential, or neural firing. Firing is the means of communication within networks of neurons.
- **Neurotransmitters Bind to Receptors Across the Synapse:** Neurons do not touch. They release chemicals (neurotransmitters) into the synapse, a small gap between the neurons. Neurotransmitters bind with the receptors of postsynaptic neurons, thus changing the charge in those neurons. Neurotransmitters' effects are halted by reuptake of the neurotransmitters into the presynaptic neurons, by enzyme deactivation, or by autoreception.
- **Neurotransmitters Influence Mental Activity and Behavior:** Neurotransmitters have been identified that influence aspects of the mind and behavior in humans. For example, neurotransmitters influence emotions, motor skills, sleep, dreaming, learning and memory, arousal, pain control, and pain perception. Drugs and toxins can enhance or inhibit the activity of neurotransmitters by affecting their synthesis, their release, and the termination of their action in the synapse.

### 3.2 What Are the Basic Brain Structures and Their Functions?

- **Scientists Can Now Watch the Working Brain:** Electrophysiology (often using an electroencephalograph, or EEG) measures the brain's electrical activity. Brain imaging is done using positron emission tomography (PET), magnetic resonance imaging (MRI), and functional magnetic resonance imaging (fMRI). Transcranial

magnetic stimulation (TMS) disrupts normal brain activity, allowing researchers to infer the brain processing involved in particular thoughts, feelings, and behaviors.

- **The Brain Stem Houses the Basic Programs of Survival:** The top of the spinal cord forms the brain stem, which is involved in basic functions such as breathing and swallowing. The brain stem includes the medulla, which controls heart rate, breathing, and other autonomic functions. The brain stem also includes the pons and the reticular formation, a network of neurons that influences general alertness and sleep.
- **The Cerebellum Is Essential for Movement:** The cerebellum ("little brain") is the bulging structure connected to the back of the brain stem. This structure is essential for movement and controls balance.
- **Subcortical Structures Control Emotions and Appetitive Behaviors:** The subcortical structures play a key part in psychological processes because they control vital functions (the hypothalamus), relay of sensory information (the thalamus), memories (the hippocampus), emotions (the amygdala), and the planning and production of movement (the basal ganglia).
- **The Cerebral Cortex Underlies Complex Mental Activity:** The lobes of the cortex play specific roles in vision (occipital), touch (parietal), hearing and speech comprehension (temporal), and movement, rational activity, social behavior, and personality (frontal).
- **Splitting the Brain Splits the Mind:** The hemispheres can be split from each other to reveal their primary functions.

### 3.3 How Does the Brain Communicate with the Body?

- **The Peripheral Nervous System Includes the Somatic and Autonomic Systems:** The somatic system transmits sensory signals and motor signals between the central nervous system and the skin, muscles, and joints. The autonomic system regulates the body's internal environment through the sympathetic division, which responds to alarm, and the parasympathetic division, which returns the body to its resting state.
- **The Endocrine System Communicates Through Hormones:** Endocrine glands produce and release chemical substances. These substances travel to body tissues through the bloodstream and influence a variety of processes, including the stress response and sexual behavior.
- **Actions of the Nervous System and Endocrine System Are Coordinated:** The endocrine system is largely controlled through the actions of the hypothalamus and the pituitary gland. The hypothalamus controls the release of hormones from the pituitary gland. The pituitary controls the release of hormones from other endocrine glands in the body.

### 3.4 How Does the Brain Change?

- **Experience Fine-Tunes Neural Connections:** Chemical signals influence cell growth and cell function. Experiences, particularly during critical periods, influence cell development and neural connections.
- **Females' and Males' Brains Are Mostly Similar but May Have Revealing Differences:** Females' and males' brains are more similar than different. They are different, however: Males' brains are larger than females' (on average), though larger does not necessarily mean better. Females' brains are organized more bilaterally for language. Men and women may perform the same cognitive task by using different parts of the brain.
- **The Brain Rewires Itself Throughout Life:** Although brain plasticity decreases with age, the brain retains the ability to rewire itself throughout life. This ability is the biological basis of learning. Anomalies in sensation and in perception, such as phantom limb syndrome, are attributed to the cross-wiring of connections in the brain.
- **The Brain Can Recover from Injury:** The brain can reorganize its functions in response to brain damage. However, this capacity decreases with age.

### 3.5 What Is the Genetic Basis of Psychological Science?

- **All of Human Development Has a Genetic Basis:** Human behavior is influenced by genes. Through genes, people inherit both physical attributes and personality traits from their parents.

Chromosomes are made of genes, and the Human Genome Project has mapped the genes that make up humans' 23 chromosomal pairs.

- **Heredity Involves Passing Along Genes Through Reproduction:** Genes may be dominant or recessive. An organism's genetic constitution is referred to as its genotype. The organism's observable characteristics are referred to as its phenotype. Many characteristics are polygenic.
- **Genotypic Variation Is Created by Sexual Reproduction:** An offspring receives half of its chromosomes from its mother and half of its chromosomes from its father. Because so many combinations of the 23 pairs of chromosomes are possible, there is tremendous genetic variation in the human species. Mutations resulting from errors in cell division also give rise to genetic variation.
- **Genes Affect Behavior:** Behavioral geneticists examine how genes and environment interact to influence psychological activity and behavior. Twin studies and research on adoptees provide insight into heritability.
- **Social and Environmental Contexts Influence Genetic Expression:** Genes and environmental contexts interact in ways that influence observable characteristics. Epigenetics studies how genes may change due to experience.
- **Genetic Expression Can Be Modified:** Genetic manipulation has been achieved in mammals such as mice. Animal studies using gene knockouts, which allow genes to be turned on and off, are valuable tools for understanding genetic influences on behavior and on health. In optogenetics, researchers modify genes to trigger action potentials in neurons.

## Key Terms

acetylcholine (ACh), p. 85	frontal lobes, p. 98	parasympathetic division, p. 105
action potential, p. 81	functional magnetic resonance imaging (fMRI), p. 92	parietal lobes, p. 96
all-or-none principle, p. 82	GABA, p. 87	peripheral nervous system (PNS), p. 77
amygdala, p. 95	gene expression, p. 115	phenotype, p. 117
autonomic nervous system (ANS), p. 104	genes, p. 115	pituitary gland, p. 108
axon, p. 78	genotype, p. 117	plasticity, p. 110
basal ganglia, p. 95	glutamate, p. 87	positron emission tomography (PET), p. 91
brain stem, p. 93	gonads, p. 107	prefrontal cortex, p. 98
Broca's area, p. 90	heritability, p. 122	receptors, p. 83
cell body, p. 78	hippocampus, p. 94	recessive gene, p. 117
central nervous system (CNS), p. 77	hormones, p. 106	resting membrane potential, p. 79
cerebellum, p. 93	hypothalamus, p. 94	reuptake, p. 84
cerebral cortex, p. 96	interneurons, p. 78	sensory neurons, p. 77
chromosomes, p. 115	magnetic resonance imaging (MRI), p. 91	serotonin, p. 86
corpus callosum, p. 96	monozygotic twins, p. 120	somatic nervous system (SNS), p. 104
dendrites, p. 78	motor neurons, p. 78	split brain, p. 100
dizygotic twins, p. 120	myelin sheath, p. 82	sympathetic division, p. 105
dominant gene, p. 117	neurons, p. 76	synapse, p. 79
dopamine, p. 87	neurotransmitters, p. 83	temporal lobes, p. 98
electroencephalograph (EEG), p. 91	nodes of Ranvier, p. 82	terminal buttons, p. 79
endocrine system, p. 106	norepinephrine, p. 86	thalamus, p. 94
endorphins, p. 88	occipital lobes, p. 96	transcranial magnetic stimulation (TMS), p. 92
epinephrine, p. 86		

# Practice Test

1. Which label accurately describes neurons that detect information from the physical world and pass that information along to the brain?
  - a. motor neuron
  - b. sensory neuron
  - c. interneuron
  - d. glia
2. Parkinson's disease is associated with the loss of neurons that produce which of the following neurotransmitters?
  - a. acetylccholine
  - b. norepinephrine
  - c. dopamine
  - d. serotonin
3. Drugs can produce the following actions on neurotransmitter activity. Label each example as either an agonist or antagonist effect.
  - a. mimic the neurotransmitter and activate the postsynaptic receptor
  - b. block the reuptake of neurotransmitter
  - c. decrease neurotransmitter release
  - d. clear neurotransmitter from the synapse
4. Which of the following statements about behavioral genetics is false?
  - a. Heritability refers to traits passed from parent to offspring.
  - b. Similarities among nonbiological adopted siblings are inferred to reflect environmental influences.
  - c. Identical twins raised apart are often more similar than identical twins raised together.
  - d. Greater similarities between monozygotic twins compared to dizygotic twins are inferred to reflect genetic influences.
5. In what order are incoming signals processed by a neuron? Place a 1, 2, 3, or 4 in front of each of the following parts of a neuron.  
 soma  
 terminal buttons  
 dendrites  
 axon
6. Which statement about the resting membrane potential is false?
  - a. The inside of the neuron is negatively charged relative to the outside.
  - b. The cell membrane allows more sodium than potassium ions to cross easily.
  - c. Action of the sodium-potassium pump results in more potassium inside the neuron.
  - d. The polarization of charge creates the electrical energy that powers the action potential.
7. Which of the following techniques can provide information about whether a particular brain region is necessary for a task?
  - a. electroencephalograph (EEG)
  - b. functional magnetic resonance imaging (fMRI)
  - c. positron emission tomography (PET)
  - d. transcranial magnetic stimulation (TMS)
8. Which statement about split-brain patients is true?
  - a. They have had surgery to therapeutically remove one hemisphere of the brain.
  - b. The left hemisphere can perceive stimuli, but the right hemisphere cannot.
  - c. The left hemisphere can verbally report its perception. The right hemisphere cannot articulate what it saw but can act on its perception.
  - d. The left hemisphere is analytical, and the right hemisphere is creative.

The answer key for the Practice Tests can be found at the back of the book.

# The Role of Neuroscience Within Psychology: A Call for Inclusiveness Over Exclusiveness

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In the present article, we appraise the increasingly prominent role of neuroscience within psychology and offer cautions and recommendations regarding the future of psychology as a field. We contend that the conflict between eliminative reductionism (the belief that the neural level of analysis will eventually render the psychological level of analysis superfluous) and emergent properties (the assumption that higher-order mental functions are not directly reducible to neural processes) is critical if we are to identify the optimal role for neuroscience within psychology. We argue for an interdisciplinary future for psychology in which the considerable strengths of neuroscience complement and extend the strengths of other sub-fields of psychology. For this goal to be achieved, a balance must be struck between an increasing focus on neuroscience and the continued importance of other areas of psychology. We discuss the implications of the growing prominence of neuroscience for the broader profession of psychology, especially with respect to funding agency priorities, hiring practices in psychology departments, methodological rigor, and the training of future generations of students. We conclude with recommendations for advancing psychology as both a social science and a natural science.

**Keywords:** neuroscience, emergent properties, funding agency priorities, hiring practices, training of students

Psychology has long been a hybrid discipline, bordering on traditional social sciences such as sociology and anthropology (Hedges, 1987), on the one hand, and on biological disciplines such as neuroscience and genetics, on the other (Cacioppo, Berntson, Sheridan, & McClintock, 2000). As a consequence, it is not surprising that psychology has often been beset by intradisciplinary tensions arising from its

differing and at times clashing constituencies. Reflecting these tensions, psychology has frequently suffered from an identity crisis (Henriques, 2004): Is our field primarily a social science, a natural science, or both? In recent years, this vexing question has assumed increasing urgency with the heightened emphasis on neuroscience within psychology (Miller, 2010). Yet, the broader implications of this emphasis for the future of psychology as a discipline have received relatively little attention. Our aim in this article is to begin to address this issue, which is critical for the future of our field.

From a social science perspective, psychologists have traditionally pursued such questions as the motivations underlying decisions and behavior, the correlates and causes of personality and intelligence, the nature of intergroup processes, the experiences of racial, ethnic, and cultural groups, and ways of helping employees succeed at work. Psychology has consistently been shaped by its neighboring science of biology, but our field's biological side has been acquiring increasing predominance over the past two decades. This recent shift has given several commentators, even some of those who are sympathetic to biological approaches to psy-

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We thank Milan Jelenić for the thoughtful conversations that helped to shape this article and Lauren Reinke for her help with coding job advertisements.

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chology, considerable pause (Berenbaum, 2013; Kagan, 2013; Miller, 2010; Satel & Lilienfeld, 2013).

A number of excellent analyses have explored the conceptual hazards of adopting an overly exclusively biological approach to psychology (e.g., Kagan, 2013; Miller, 2010), and we do not intend to duplicate these efforts here. Instead, our overarching goals in this article are more applied and pragmatic compared with previous analyses. Specifically, we review ways in which funding agency priorities may be, at least to some extent, driving the acceleration of neuroscience research, and we raise a number of issues that should be considered when interpreting the relationship between neural processes and psychological or behavioral phenomena. We maintain that the trend toward a growing focus on neuroscience within psychology carries important, and often unarticulated, metatheoretical assumptions regarding the levels of analysis at which psychological phenomena are best conceived and approached. These assumptions, in turn, carry practical implications of considerable importance for the short- and long-term future of our field. We explore these implications across three major domains—hiring practices, methodological rigor, and the training and education of future generations of students. Finally, **and just as importantly, we explore the optimal role of neuroscience within a complete and cohesive science of psychology.** We argue that an excessively narrow emphasis on neuroscience may be problematic not only for the more traditionally “psychological” dimensions of our discipline (e.g., social, personality, cross-cultural psychology), but also for neuroscience itself. More precisely, we maintain that for neuroscientific approaches to psychology to realize their full scientific potential, they will need to make con-

certed efforts to establish fruitful linkages across multiple levels of analysis, including the more traditionally psychological levels, as well as to benefit from methodological advances in other domains of psychology. Hence, we contend that neuroscientific approaches to psychology must be certain not to sever their “vital blood supply” from other fields of psychology.

### The Recent Expansion of Neuroscience Within Psychology: Promise and Perils

The recent changes in emphasis on neuroscience within psychology are perhaps best understood within a historical context. Throughout most of the 20th century, much of psychology unjustly neglected the neural level of analysis. The study of psychopathology is a case in point. For example, until the late 1960s, models of the etiology of schizophrenia were dominated by psychogenic explanations (e.g., the schizophrenogenic mother, family interaction patterns, life stressors) that largely ignored the biological level of explanation (see Dolnick, 1998). This nearly exclusive “sociotropic” focus (Meehl, 1972) had baleful consequences; among other things, it contributed to sending researchers down blind alleys and blaming generations of parents for a disorder for which they were not causally responsible.

In recent years, much of psychology’s focus has shifted sharply toward the “biotropic” (Meehl, 1972) end of the spectrum (see Deacon, 2013). To some extent, this change appears to have been a healthy self-correction from the radical environmentalism of the early and mid-20th century (Pinker, 2003), which routinely disregarded or deemphasized biological correlates and influences. Nevertheless, the swing of the pendulum toward the “biotropic” end of the spectrum has provided ample cause for concern.

### The Dangers of Fervent Monism

As Kendler (2014) observed, there are hazards to “fervent monism” of any kind in psychology and psychiatry. By fervent monism, he meant the propensity to rely too heavily on only one explanatory level in Comte’s (1830-1842; see also Cole, 1983) famed hierarchy of the sciences. Foremost among these dangers is an undue neglect of explanatory levels that may be crucial for solving the riddle of highly multifaceted psychological problems such as psychopathology, aggression, divorce, intergroup conflict, and prejudice. All of these problems can probably be understood fully and remediated only by an examination of multiple levels of analysis, including the neuroscientific, the motivational, the social, and the cultural.

The most recent past president of the Association for Psychological Science, Nancy Eisenberg (2014), recently expressed similar cautions regarding fervent monism. While applauding the remarkable advances in genetics, neurosci-

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ence, and autonomic psychophysiology within psychology, Eisenberg commented with some alarm on the “increasing tendency to assume that studying genetic/neural/physiological processes is more important than research on behavior and psychological processes *per se* because biological findings will eventually explain most of human psychological functioning” (p. 1). She further voiced concerns that this propensity is increasingly evident in “the funding priorities at some of the National Institutes of Health . . . it can also be seen in the hiring patterns of many psychology departments that place a priority on hiring people who study biological processes or aspects of cognition that can be tied to neuroscience” (p. 1).

As one informal, but perhaps telling, manifestation of [N. Eisenberg's \(2014\)](#) concerns, a number of psychology departments have recently modified their names to underscore a focus on neuroscience ([Beins, 2012](#)). Such names include “Department of Psychological and Brain Sciences” (e.g., University of California, Santa Barbara, Johns Hopkins University, Indiana University, Dartmouth University, Boston University, University of Louisville, University of Massachusetts–Amherst) and “Department of Psychology and Neuroscience” (e.g., Duke University, Baylor University, University of Colorado at Boulder). On the one hand, these name changes apply to only a minority of psychology departments, so they hardly represent a groundswell. On the other hand, most of these name changes have occurred in the past 5 to 10 years, raising the possibility that more are on the way. [Lilienfeld \(2012a\)](#) noted the logical confusion exemplified by these new department names. Specifically, these names imply that neuroscience stands apart from psychology, as opposed to being one valuable approach to

psychology, among many. To press the matter a bit further, imagine individuals' reactions to a department called the “Department of Psychology and Intergroup Processes” or the “Department of Psychology and Personality Research.” Many people would be understandably puzzled by these names given that intergroup processes and personality research are merely two approaches to psychology, not disciplines that stand *sui generis* from psychology.

In many ways, the situation in contemporary psychology parallels that of our sister field of psychiatry, in which the substantial majority of research now focuses on biological correlates and putative etiological factors ([Stone, Whitham, & Ghaemi, 2012](#)). In the mid-1980s, psychiatrist [Leon Eisenberg \(1986\)](#) lamented that his field had gone from being largely “brainless” to being largely “mindless.” By mindless, he meant a substantial neglect of the role of psychosocial factors and patients’ subjective perceptions of their experiences (see also [L. Eisenberg, 2000](#)). We are concerned that psychology may be following in psychiatry’s footsteps (see also [Satel & Lilienfeld, 2013](#)). To invoke an example that bears on both fields, an analysis of over 9,000 abstracts from international conferences on schizophrenia indicated that 75% of the presentations focused on biological correlates and influences, with only 5% focusing on psychosocial factors ([Calton, Cheetham, D'Silva, & Glazebrook, 2009](#)). However, research strongly suggests that life stressors play a key role in the maintenance and perhaps onset of schizophrenia, especially in the presence of a genetic predisposition ([Jones & Fernyhough, 2007](#)).

Some of the change afoot in psychology almost surely reflects a shift in scientific priorities and interests, as has happened several times in the history of our field. For example, the appeal of psychoanalysis, at least in the United States, began to fade with the increasing preeminence of radical behaviorism, and the appeal of radical behaviorist approaches in turn diminished markedly during the cognitive revolution of the 1970s ([Anderson, 2004](#); but see [O'Donohue, Ferguson, & Naugle, 2003](#), for a dissenting view on whether this “revolution” was a valid scientific revolution). In the last 20 years or so, we have seen psychology increasingly embrace the brain as the most important level of analysis for understanding psychological phenomena such as emotions, thoughts, mood and anxiety disorders, addictions, and social problems ([Miller, 2010](#)). Is this increasing emphasis on neuroscience, which we term the *neuroscience movement*, a scientific epoch that will come and go, much like the psychoanalytic and radical behaviorist movements of the early- and mid-20th century? Or has the neuroscience movement instead laid down roots in a way that psychoanalysis and radical behaviorism arguably did not? It is too early to tell, but there are growing indications that the effects of this movement on psychology are likely to be enduring.



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### The Advent of Neuroimaging Technologies

Although neuroscience research within psychology has been thriving for many decades, the introduction of functional magnetic resonance imaging (fMRI) and other exciting brain-imaging tools has breathed new life into many important psychological questions, and accelerated the progress of neuroscience by providing scientists with increasingly direct access to ongoing neural events (Diener, 2010). The advent of these new neuroimaging tools has allowed scientists to further peer into the brain and has helped in identifying the neurobiological correlates of behavioral, social, emotional, and developmental processes. To be fair, many neuroscience-related fields and techniques do not rely on traditional brain imaging, such as single-neuron recording (Moxon, Leiser, Gerhardt, Barbee, & Chardin, 2004), event-related potentials (Liu, Goldberg, Gao, & Hong, 2010), optogenetics (Deisseroth, 2011), and gene-based analyses (Krumm, O’Roak, Shendure, & Eichler, 2014). The fields of behavioral (Nelson & Mizumori, 2013), cognitive (Gazzaniga, Ivry, & Mangun, 2008), and affective (Armony & Vuilleumier, 2013) neuroscience have evolved largely from an interest in identifying the neurobiological circuitry associated with human experience and behavior. In many respects, these endeavors have met with impressive success. For example, we now have a clearer sense of the damage that drugs and alcohol often inflict on the brain (Goldstein & Volkow, 2011; Zeigler et al., 2005); we have gained a better understanding of how cultural contexts and mores help to “soft-wire” the brain to react to environmental stimuli (Park & Huang, 2010); and we know much about the brain regions that are activated when individuals are asked

to perform self-evaluations (Pfeifer, Lieberman, & Dapretto, 2007) and express admiration and virtue (Immordino-Yang, McColl, Damasio, & Damasio, 2009). Extremely promising and rapidly growing subfields of psychology, such as social neuroscience (Cacioppo et al., 2002) and cultural neuroscience (Kitayama & Tompson, 2010), have emerged in recent decades from the simultaneous consideration of neurobiological and experiential phenomena.

### Shifts in Funding Agency Research Priorities

The neuroscience movement may be motivated by financial as well as scientific shifts. It has become increasing apparent that federal funding agencies, such as the National Institutes of Health (NIH), have been turning their focus increasingly to neuroscience research (Dorsey et al., 2006; Gewin, 2013). The four NIH institutes whose missions are most closely related to psychological research—the National Institute on Child Health and Human Development, the National Institute on Mental Health (NIMH), the National Institute on Drug Abuse (NIDA), and the National Institute on Alcohol Abuse and Alcoholism—have experienced directorship changes in the last decade that have shifted their priorities considerably toward neuroscience and other biological fields, and away from the social sciences. For example, of the four NIDA divisions, only one (epidemiology, services, and prevention) adopts a social-science focus; and of the four NIMH divisions, only one (services and intervention research) adopts a social-science focus. The remaining divisions of both institutes are focused primarily on biological approaches, most notably those in neuroscience, genetics, and microbiology. Although biological research is clearly important, care must also be taken to ensure that social-science research continues to be supported at the funding agency level.

One might contend that NIMH, in particular, has recently embraced an ethos that runs the risk of fervent monism. For example, the draft of NIMH’s newest “Strategic Plan” ([http://www.nimh.nih.gov/about/strategic-planning-reports/Strategic\\_Plan\\_2015\\_public\\_comment\\_148461.pdf](http://www.nimh.nih.gov/about/strategic-planning-reports/Strategic_Plan_2015_public_comment_148461.pdf)) asserts that NIMH “encourages basic scientists to identify molecular or neural mechanisms of specific domains of mental function” (p. 9). Among the strategic objectives of the NIMH plan are “defining the *biological basis* of complex behaviors” (p. 15), “describing the molecules, cells, and neural circuits associated with complex behaviors” (p. 17), and “mapping the connectomes for mental illness” (p. 18). The ostensible implication here is that the psychological level of analysis will be less fruitful than the biological level for breakthroughs in our understanding of psychopathology.

Other recent NIMH initiatives have also fueled concerns regarding a disproportionate emphasis on the biological level of explanation. Several years ago, NIMH proposed the



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Research Domain and Criteria (RDoC) as a long-term alternative to the current diagnostic manual, the *Diagnostic and Statistical Manual of Mental Disorders*, currently in its fifth edition (Insel et al., 2010; Sanislow et al., 2010). The goal of RDoC, which we believe to be potentially valuable (Lilienfeld, 2014), is to identify markers of brain circuits linked to psychobiological systems (e.g., reward systems, negative emotion systems, attentional systems) that are implicated in mental disorders. Many aspects of RDoC are clearly worthwhile, including loosening the longstanding hegemony of the *Diagnostic and Statistical Manual of Mental Disorders* over descriptive psychiatry and clinical psychology (Lilienfeld, 2014). Furthermore, in principle, RDoC and NIMH's broader initiatives are potentially consistent with a consideration of multiple levels of analysis (Sanislow et al., 2010). Indeed, the RDoC matrix acknowledges levels other than the biological and leaves room for developmental influences on behavior (Cuthbert, 2014). Although RDoC conceptualizes mental disorders as conditions marked by dysfunction in brain circuitry, it is agnostic regarding the etiology of these conditions, as well as regarding the optimal level of analysis at which to assess them.

At the same time, there are risks that RDoC, unless carefully executed, may inadvertently neglect alternative levels of analysis. Five of the eight targeted levels of analysis in the RDoC matrix for research—genes, molecules, cells, circuits, and physiology—focus largely or entirely on the biological rungs of the hierarchy, whereas only two—behavior and self-reports—focus explicitly on the psychological rungs of the hierarchy (see <http://www.nimh.nih.gov/research-priorities/rdoc/research-domain-criteria-matrix.shtml>).

Moreover, several leading figures in the psychiatric community have described RDoC in large measure as a search for “biomarkers” (see Lilienfeld, 2014, for a discussion), suggesting that there may be a widespread perception that RDoC is privileging the search for indicators at the biological level of analysis.

The new “Strategic Plan for the National Institute on Drug Abuse” adopts a similar approach. For example, one of NIDA’s goals is to “improve our understanding of brain circuits related to drug abuse and addiction at the cellular, circuit, and connectome levels” (National Institute on Drug Abuse, 2015). The implication is that addiction is a disease of the brain, and that treating this diseased neurobiology is the key to ameliorating drug and alcohol addiction (Satel & Lilienfeld, 2013). A number of critics, however, have identified problems with this brain-disease model of addiction (e.g., Hall, Carter, & Forlini, 2015). Although NIDA’s focus on neuroscience is somewhat less prominent than NIMH’s, the message appears to be that the NIH is focusing more and more on neuroscience and other biological levels of analysis.

These existing NIH initiatives are augmented by President Obama’s new Brain Research Through Advancing Innovative Neurotechnologies (BRAIN) initiative (<http://www.whitehouse.gov/share/brain-initiative>), which directs research almost exclusively to developing more sophisticated technological tools to unlock mysteries at the biological levels of analysis, particularly at the neuronal level (Insel, Landis, & Collins, & The NIH BRAIN Initiative, 2013). The Obama BRAIN initiative complements the Human Brain initiative, funded by the European Commission. These two ambitious funding efforts may be well worth pursuing, although a growing cadre of scholars has expressed concerns that they may yield less long-term fruit than hoped. As cognitive scientist Gary Marcus (2014) pointed out, we will ultimately need more than advanced technologies. For genuine breakthroughs to occur, we need better theories of brain functioning and, especially, better conceptual models of how the neural and psychological levels of analysis intersect. Marcus noted that “What we are really looking for is a *bridge*, some way of connecting two separate scientific languages—those of neuroscience and psychology” (p. A17; emphasis in original). In the lingo of philosophy of science, substantial breakthroughs in many psychological domains will probably necessitate the development of “bridge laws” (De Jong, 2002; see also Lilienfeld, 2007) to connect adjacent levels of analysis (see Akiskal & McKinney, 1973, for a landmark early effort in this regard in the domain of clinical depression). For such bridge laws to be discovered, investments must be made not only in neuroscience per se but also in the crucial interfaces between neuroscience and more traditional psychological disciplines, such as social, affective, and personality psychology. These efforts, in turn, will require bidirectional

cross-talk and collaboration between scholars in both neuroscience and other domains of psychology.

### The Place of Neuroscience Within the Broader Discipline of Psychology: Conceptual Issues

Needless to say, the proper role of neuroscience within psychology is exceedingly complex and controversial. A delineation of several crucial conceptual issues, some of which involve metatheoretical assumptions that have often received insufficient attention within psychology, is therefore needed to frame discussions regarding the implications for neuroscience for the future of our field. We begin with reductionism, which is an essential component of the brain-behavior relationship.

#### Reductionism: Two Flavors

The majority of neuroscience research rests on the bedrock assumption of reductionism—the belief that all behavioral, experiential, cognitive, and emotional processes are rooted in neurobiology (Satel & Lilienfeld, 2013). Nevertheless, the relation of neuroscience to other fields of psychology hinges crucially on *which kind* of reductionism is assumed. At least two overarching types of reductionism can be distinguished: *constitutive* and *eliminative* (also termed *greedy reductionism* by Dennett, 1993; see also Fodor, 1968; Lilienfeld, 2007). These two forms of reductionism hold dramatically different implications for the role of neuroscience within psychology (Lilienfeld, 2007).

Constitutive reductionism accepts the largely uncontroversial position, which we endorse, that the brain enables the mind, and that the mind is not a spooky, immaterial essence separate from the brain. Hence, constitutive reductionists grant that the mind is what the brain (and the rest of the central nervous system) does. This view rejects substance dualism, the position that the mind and brain are materially different, but leaves open the possibility of property dualism, the position that mind and brain have different properties that coexist at different levels of analysis. In contrast, eliminative reductionism goes considerably further to contend that the brain-based level of analysis will ultimately “explain away” and subsume all of psychology. Hence, eliminative reductionists reject both substance and property dualism. From their perspective, the psychological level of analysis is, in principle, superfluous to understanding behavior, adding little or nothing to the physiological level, which will eventually “gobble up” the psychological level. In other words, the psychological level of analysis will eventually be shown to be unnecessary, and is already well on the way to being eroded by new discoveries in neuroscience.

**Levels of analysis in psychology.** We have referred on several occasions to the concept of different levels of anal-

ysis within psychology, but we have not yet formally explicated this concept. The levels of analysis concept is essential for an adequate understanding of eliminative reductionism (Kandler, 2005; Lilienfeld, 2007; but see Miller, 2010, for a critique of the levels of analysis view). From the levels of analysis perspective, we can conceptualize psychological phenomena from the vantage point of differing rungs on a hierarchy of explanation, ranging from the molecular and physiological levels of explanation at the lower rungs, to the cognitive, emotional, motivational, and personological levels of the middle rungs, to the social, cultural, and historical levels at the highest rungs. The traditionally “psychological levels” occupy the middle (especially) and higher rungs in the hierarchy of explanation. Furthermore, this perspective posits that all levels are needed for a complete understanding of human psychological phenomena, with differing research problems best lending themselves to different levels. For example, investigators striving to understand the causes of Alzheimer’s disease will justifiably focus their efforts on the lower levels of the hierarchy; in contrast, those striving to understand the causes of marital conflict will justifiably focus their efforts at the middle and perhaps higher levels. Although there is clear evidence that psychotherapeutic interventions change brain function (which is to be fully expected from a constitutive reductionist view) and in some cases change brain structure (e.g., Matto et al., 2014), a counselor attempting to analyze a married couple’s emotional conflicts would be unlikely to subject them to functional brain imaging. Rather, the counselor would be more likely to focus on the motives, conflicts, interests, and other key influences at the psychological level of analysis, that can serve as targets for intervention (Satel & Lilienfeld, 2013). Indeed, one might contend that a focus on the neural level of analysis in this case would be ill-advised, as it could distract the counselor from other, potentially more profitable, points of intervention.

**Emergent properties.** When understood from the levels of analysis perspective, eliminative reductionists (e.g., Searle, 2002) believe that only the lowermost rungs of explanation will ultimately be necessary to understand human behavior. Skeptics of eliminative reductionism, ourselves included, posit the existence of emergent properties (O’Connor, 1994; but see Searle, 2002, for a critique of emergent properties). Emergent properties are higher-order functions that arise from exceedingly complex interactions among lower-order properties; hence, they cannot merely be reduced to lower-order properties. For example, the phenomena of traffic and the schooling of fish are frequently cited emergent phenomena that may not be fully reducible to the lower-order behavior of the individual units (cars and fish, respectively) involved. Similarly, many psychological phenomena, such as motives, emotions, and personality traits, may be higher-order, emergent properties that are not fully reducible to the level of neural elements (Kagan,

2013). In the example of the married couple in the counselor's office, the fact that these conflicts can be mapped or imaged in the couple's brains does not obviate the need for the counselor to examine their thoughts, feelings, motives, and other higher-level psychological properties. This position is not new. Indeed, a little over a century ago, pioneering psychiatrist Adolph Meyer (1912) took issue with the "dogma of the neurologizing tautology . . . that only those facts are scientific which can be reduced to terms of nerve cells" (p. 321).

As a social science, psychology encompasses the study of such phenomena as intergroup conflicts and wars (Moshman, 2011; Tajfel & Turner, 1986), the development of personal meaning and identity (Erikson, 1950; Frankl, 1959; Steger, Kashdan, & Oishi, 2008; Vignoles, Schwartz, & Luyckx, 2011), spirituality and religiosity (Paloutzian & Park, 2008), international migration and acculturation (Sam & Berry, 2010), and sexual orientation (Savin-Williams & Ream, 2007), all of which may be emergent properties that are not readily decomposable into lower-order neural elements. Although neuroscience methods will almost certainly contribute to our understanding of these phenomena (and have already done so in some cases), an integrative science of psychology will ostensibly require expertise in and attention to emergent properties as well as to the neural processes that facilitate them.

To the extent to which the emergent properties view has merit, social-science constructs such as self-esteem, agency, well-being, and identity should be studied alongside (and in some cases, apart from) neural processes. Supporting the concept of emergent properties, Swann, Chang-Schneider, and Larsen McClarty (2007) reviewed evidence that people's views of themselves are important, and neurological evidence suggests that low self-esteem and life satisfaction predict poor cortisol regulation and hippocampal decline in adulthood (Pruessner et al., 2005). The finding that self-evaluations at the conscious level can predict biological processes at the cellular level raises questions regarding the assumption that social-science processes are expendable and that neurobiology is necessarily a first cause. As a further example, Antoni et al. (2009) found that, among women in breast cancer treatment, a cognitive-behavioral intervention to reduce anxiety was efficacious in maintaining immunologic functioning during treatment, and that reductions in anxiety mediated the effects of the intervention on immunologic outcomes. This finding illustrates that social-science processes matter and can be important markers of health.

Among humans, brain activity is clearly necessary to support agentic functioning and other aspects of consciousness—that is, that constitutive reductionism is a fundamental truism. For example, someone with an impaired prefrontal cortex cannot make reasoned decisions, and someone with hippocampal damage will experience difficulties with

episodic memory. Moreover, the emergent properties hypothesis does not preclude brain activity or impairment from directly influencing conscious experience, as in the effects of oxytocin injections on feelings of closeness and attachment (e.g., Kosfeld, Heinrichs, Zak, Fischbacher, & Fehr, 2005). The effects of psychotropic medication on anxiety and depression are also examples of how chemical changes in the brain can directly affect mood (e.g., Katzman, 2004). Nevertheless, as Beauregard (2012) inferred from his experiments, individuals can often distinguish effects that were caused by a drug or other external influence from those that the person believes to have caused on her or his own. Similarly, Baumeister (2008) found that volitional actions, such as solving puzzles, are more mentally tiring compared with "automatic" actions that are undertaken without conscious effort. To the extent to which active, volitional, deliberate actions reflect agency, some support may be provided for an emergent properties perspective (but see Pronin, Wegner, McCarthy, & Rodriguez, 2006, for an alternative view).

Psychosocial adjustment indices such as self-esteem, life satisfaction, and well-being are premised on the presence of a self that is reflexively evaluated. All of these constructs refer to introspectively posed questions such as "How am I doing in my life?" (Ryan & Deci, 2001, 2006). Such questions may reflect events that cannot be understood exclusively at the neural level of analysis. As a result, the "me" asking these questions may not be reducible entirely to neurobiological processes. Indeed, social science adjustment indices such as self-esteem are important largely to the extent that subjective experience can be separated from neurobiology. For example, self-esteem is often invoked as a mechanism through which people view their lives as having meaning and view themselves as important (see Pyszczynski, Greenberg, Solomon, Arndt, & Schimel, 2004; Zeigler-Hill, 2013). As another example, someone with low self-esteem would typically be referred to counseling or "talk therapy" only if improving self-esteem is considered a worthy outcome in its own right. Conversely, if the goal is to alter the brain chemistry associated with low self-esteem, then psychotropic medication will typically be the initial treatment of choice (Deacon, 2013; Insel, 2009). Of course, interventions, such as psychotherapy, delivered to affect emergent-property outcomes can also exert important effects on neurobiological outcomes (Linden, 2006)—suggesting that the links between neural and experiential processes are often bidirectional.

## Logical Leaps in Interpreting Neural-Psychological Connections

To conceptualize the optimal role of neuroscience within psychology, careful thinking and dialogue among researchers possessing differing types of expertise will be necessary.

Nevertheless, one unappreciated hazard of eliminative reductionism may be the embrace of certain logical leaps, which often reflect a propensity to draw inferences that extend beyond one's data and the methods used to collect those data (see Satel & Lilienfeld, 2013; and Shermer, 2008, for discussions). We discuss some of these logical leaps here.

Especially with regard to neuroimaging, several logical leaps that are especially salient: (a) inferring causation from correlational evidence (Miller, 2010); (b) neuroessentialism, the assumption that neural processes are all that are needed to understand thoughts, feelings, and behavior (Haslam, 2011); (c) neurorealism, the assumption that brain imaging is an inherently more dependable and genuine source of data than are mental states (Racine, Bar-Ilan, & Illes, 2005); and (d) neuroredundancy, the failure of brain imaging data to provide information that could instead be gleaned by extant information, such as by simply asking people about their subjective experiences (Satel & Lilienfeld, 2013). Another logical challenge to the interpretation of neuroimaging data is reverse inference, the conclusion that activation in a brain area reflects an underlying psychological state (Poldrack, 2006), such as the assumption that amygdala activation in a given individual necessarily reflects fear. Although reverse inferences drawn from brain imaging data are sometimes accurate and are not logical errors per se (Ariely & Berns, 2010), they are inevitably fallible conclusions that should be invoked with caution and corroborated by means of triangulation with other data. All of these inferential leaps, some of which can contribute to error, can lead individuals to place more weight on neuroscientific data than is objectively warranted. Given that brain imaging techniques are in their relative infancy, it is entirely possible that stronger conclusions will be able to be drawn as these technologies continue to evolve. At the present time, however, more caution may be warranted when interpreting brain imaging data.

As we mentioned at the outset of this article, although neuroimaging is among the fastest accelerating and most widely known neuroscience methods, a range of other methods are also used by neuroscience researchers to understand the links between neural and behavioral or experiential phenomena. Many of these other methods focus on molecular and cellular levels of analysis (see Krumm et al., 2014; Soltesz & Staley, 2008, for examples), although many researchers who conduct work using these methods are keenly interested in spanning multiple levels of analysis, including more traditional "psychological" levels that encompass emotion and overt behavior (Gross, 2013). These approaches generally do not involve the logical leaps that we have enumerated vis-à-vis neuroimaging research. As a consequence, the reader should not interpret the caveats regarding neuroimaging that we have described as applying in equal force to all neuroscience-related methods.

**Neuroseduction.** Fueling the overarching concern regarding logical leaps inferred from neuroimaging studies are findings that many individuals may be susceptible to being persuaded by dubious conclusions when they are accompanied by neuroscience explanations, a phenomenon sometimes termed *neuroseduction*. For example, in a now-famous study, Weisberg, Keil, Goodstein, Rawson, and Gray (2008) found that merely inserting the words "brain scans show" can lead undergraduates (but not neuroscience researchers) to accept logically flawed explanations derived from neuroimaging studies (but see Farah & Hook, 2013, for a critique of "neuroseduction" research). In particular, nonexperts were more likely to endorse tautological (circular) explanations, such as the hypothesis that the curse of knowledge occurs "because" of frontal lobe circuitry implicated in knowledge of oneself, when these explanations contained a brief mention of neuroimaging data. In a recent study of college students, Fernandez-Duque, Evans, Christian, and Hedges (2015) reported that including superfluous neuroscience information in the descriptions of studies similarly rendered participants more likely to accept tautological explanations as logical.

As a consequence, neuroscience researchers must be especially careful to draw only those conclusions that their data can support. Overstating causal conclusions that can be drawn from neuroscience research, as in other domains of psychological research, can readily foster misunderstandings among members of the lay public (Satel & Lilienfeld, 2013). For instance, numerous studies refer to the "effects" of the neuropeptide oxytocin on human social behavior (e.g., Heinrichs & Domes, 2008; McDonald & McDonald, 2010), including parenting and interpersonal closeness, even though much of the evidence for this assertion derives from correlational data. Similarly, some authors have argued or implied that immaturity in the prefrontal cortex *causes* adolescent risk taking (Cauffman & Steinberg, 2000), or that dysfunction in the mirror neuron system is causally implicated in autism spectrum disorder (see Hickok, 2014, for a critique). Most recently, neurosurgeon Itzhak Fried has claimed that genocidal behavior is rooted largely in an interrelated set of neurobiological dysfunctions that he labels "Syndrome E" (Abbott, 2015). In other cases, the media have presented brain imaging data as implying a causal link between the size of, or activity in, certain brain structures and psychopathology. For example, a press release for a study in the prestigious British medical journal *Lancet*, which reported differences in the volume of certain brain areas in individuals with and without attention-deficit/hyperactivity disorder, proclaimed that "Brain-imaging study sheds more light on underlying cause of attention-deficit hyperactivity syndrome" (EurekAlert, 2003). Nevertheless, it is unclear whether these brain differences contribute to attention-deficit/hyperac-

tivity disorder, result from it, or are reflections of unidentified third variables.

**Mental disorders as brain diseases.** Another important domain in which logical slippage has at times been evident is the framing of mental disorders. As many authors have observed (e.g., Deacon, 2013; Gustavsson et al., 2011), mental disorders are increasingly being conceptualized—in both psychology and psychiatry—as “brain diseases.” For example, Thomas Insel (Insel et al., 2013; see also Insel & Cuthbert, 2015), current director of NIMH, has consistently argued that “We need to think of these [mental disorders] as brain disorders” (National Institute on Mental Health, 2013).

Although we certainly applaud the increasing incorporation of the biological level of analysis and of biological indicators in conceptualizations of psychopathology, conceptualizing mental disorders as brain diseases is both logically confused and confusing. From the standpoint of constitutive reductionism, mental disorders are necessarily brain diseases at *some level*, because all psychological phenomena, normal and abnormal, are necessarily mediated by the brain and the remainder of the central nervous system. In this respect, the assertion that psychological disorders are brain diseases is not testable; rather, this assertion is a scientifically uninteresting truism. Although mental disorders are of course brain diseases at some level of analysis, mental disorders can just as validly be regarded as psychological diseases, as the psychological level of functioning is by definition impaired in mental disorder (Wakefield, 1992). Hence, the decision to prioritize the neural level of analysis above the psychological level when conceptualizing psychopathology (e.g., Insel & Cuthbert, 2015) appears difficult to justify on logical grounds. Moreover, regarding psychological disorders exclusively as brain diseases risks confusing biological *mediation* with biological *etiology*. The fact that all mental disorders are *enabled* by brain functioning does not necessarily imply that they are *caused* by abnormalities in brain hardware (see Harre, 2002, for a discussion of the distinction between enabling and causation in neuroscience). Instead, at least some mental disorders may be largely or entirely psychosocial in etiology, although these psychosocial influences necessarily operate by exerting proximal effects on brain functioning. Further, from a clinical perspective, there is evidence that conceptualizing mental disorders as brain diseases decreases clinicians’ empathy toward patients (Lebowitz & Ahn, 2014), suggesting that the “brain disease” model can have adverse impacts on clinical practice.

**Neuroscience and psychological intervention.** A final domain of potential concern that we address is the at times premature insertion of neuroscience into clinical and educational practice, especially psychotherapy. We view

the movement to develop “brain-based psychotherapies” with ambivalence. On the one hand, we concur with authors (e.g., Barlow, 2014) who have contended that neuroscience may ultimately inform the design of effective psychotherapies. For example, the basic neuroscience of fear conditioning and extinction may assist in crafting more effective interventions for anxiety disorders (Milad, Rosenbaum, & Simon, 2014). In addition, there is preliminary but encouraging evidence that functional neuroimaging data may predict differential response to treatment (see also Craske, 2014), such as response to cognitive–behavioral therapy versus selective serotonin reuptake inhibitors in patients with major depression (McGrath et al., 2013).

On the other hand, it is presently unclear whether neuroscience offers treatment developers or practitioners much tangible guidance above and beyond what is afforded by an understanding of overt behavior, affect, and cognition alone (Andreas, 2013), suggesting that the recent trend toward brain-based therapies often runs afoul of the neuroredundancy problem (Satel & Lilienfeld, 2013). An illustration of this difficulty can be found in an article (Cappas, Andres-Hyman, & Davidson, 2005; see also Cozzolino, 2002) outlining seven principles of psychotherapy derived from basic research on neuroscience. Most or all of these principles, such as “experience transforms the brain” (Cappas et al., 2005, p. 375), “cognitive and emotional processes work in partnership” (p. 377), and “bonding and attachment provide the foundation for change” (p. 379), may possess substantial validity, but it is not clear whether any of them provide fresh insights that have not been evident to psychologists for decades. For example, the proposition that “experience transforms the brain” is unquestionably true, but it appears to do little more than recast the truism that “Experience can change behavior” in neural language. Nor does it point to novel approaches to clinical intervention.

Nevertheless, this limitation has not prevented enthusiastic advocates from developing and promoting brain-based therapies and other interventions, such as Brainspotting, which supposedly draw on basic knowledge about the brain to inform clinical interventions. For example, Brainspotting claims to use the visual field to turn the “scanner” [in the visual system] back on itself and to guide the brain to find internal information. “By keeping the gaze focused on a specific external spot, we maintain the brain’s focus on the specific internal spot where the trauma is stored, in order to promote the deep processing that leads to the trauma’s release and resolution” (Grand, 2013, p. 4). Nevertheless, like most brain-based treatments, Brainspotting has never been subjected to controlled experimental tests. Arguably, the deeper problem with importing neuroscience into the psychotherapy do-

main is that we do not presently know enough about the linkages between brain and behavior to meaningfully bridge the relevant levels of analysis in a way that could inform the design or implementation of psychological treatments.

Many of the same considerations, we might add, also apply to the premature application of neuroscience concepts to early education in the guise of “brain-based learning techniques.” To take merely one example among many (Goswami, 2004), the popular educational technique Brain Gym, now used in over 80 countries, purports—among other things—to boost blood flow to the brain by massaging specific bodily regions (“brain buttons”), thereby ostensibly enhancing child learning. Nevertheless, this technique has not been subjected to controlled experimental trials, nor is it supported by biological plausibility (see Howard-Jones, 2014, for a critique).

### Effects of the Neuroscience Movement on Psychological Research and Hiring Practices

Given the direction that NIMH and other federal funding agencies have taken in directing more of their funding toward neuroscience, it is important to ascertain the potential effects that these funding priorities have had on psychological researchers and on hiring practices within psychology departments. To examine these effects systematically, we collected data from two sources—an online survey of researchers in three fields of psychology and statistics on psychology job postings over a 3-year period.

### Pressures on Researchers to Include Neuroscience Measures

During the fall of 2014, we conducted a brief online survey of members of three medium-to-large psychological organizations (one developmental, one social/personality, and one clinical). We did not recruit from neuroscience organizations because we were interested in the extent to which individuals from other fields of psychology would report being pressured by funding agencies to engage in neuroscience research. Messages were posted to the listservs for these three organizations. The survey was posted online for 5 weeks, and a total of 204 U.S.-based respondents provided data. Of the 204, 26 participants were excluded because they were not employed in academic institutions. The remaining 178 participants ranged in rank as follows: graduate student (15.7%), postdoctoral associate (8.8%), adjunct instructor (3.4%), assistant professor (17.6%), associate professor (10.3%), full professor (24.5%), and other professional (6.4%). Although, not surprisingly, the majority of participants were from developmental (30.9%), social/personality (14%), and clinical

(32.6%) disciplines, the sample also included participants from other disciplines including cognitive (4.5%), counseling (1.7%), health (1.7%), industrial/organizational (1.1%), and quantitative psychology (1.1%).

The survey asked a number of questions related to the state of psychology as a field, but here we report data on respondents who had submitted at least one federal grant application. Specifically, of those individuals who had submitted at least one grant proposal ( $n = 133$ ; 69.1% of the total sample), 16.4% indicated that they had been directly asked by a grant review committee to include neuroscientific measures in at least one proposal. In addition, 42.0% of the participants stated they have “sometimes” (15.0%), “often” (13.2%), or “always or almost always” (13.8%) felt pressured to include neuroscientific measures in their research and grant applications. To the extent to which these results are generalizable, many non-neuroscientists who are engaging in neuroscience-related research may be doing so, at least in part, due to pressure from funding agencies. One limitation of our survey data is that we cannot ascertain how often the recommendations of grant review committees to include neuroscience measures were scientifically legitimate. Nevertheless, our findings raise the distinct possibility that perceived funding agency priorities and directives toward neuroscience research may be driving the design of psychological research, perhaps in many cases unduly so.

### Analysis of Psychology Department Hiring Trends

Using past issues of the *American Psychological Association Monitor*, we compiled a list of all tenure-track or tenured faculty positions advertised in psychology departments in “very research-intensive universities” (see <http://classifications.carnegiefoundation.org/>) during calendar years 2011, 2012, and 2013, the 3 full years in which advertisements from back issues of the *Monitor* were available during the summer of 2014 (when the coding was conducted). We selected “very research intensive universities” because these are the institutions most likely to require or strongly encourage faculty to submit grant applications. Duplicate advertisements (i.e., instances in which the same advertisement was posted during multiple months) were analyzed only for the first month in which they appeared. Lecturer, postdoctoral, and research associate positions were excluded from our analysis.

The third author and a trained research assistant coded each advertisement in terms of the extent to which the position was targeted toward each of several subfields of psychology (clinical, cognitive, developmental, social/personality, neuroscience, counseling, and quantitative). For each advertisement, coders indicated, on a 4-point scale, whether each subfield was not mentioned (0), listed as one of several potential target areas (1), preferred (2), or re-

quired (3). A reliability check between the two coders, estimated using the 2013 advertisements, indicated agreement rates ranging from 87% to 100% in terms of the extent to which each subdiscipline was not referenced, mentioned, preferred, or required for each position.

We report results in terms of advertisements in which each subfield was required, encouraged, and/or mentioned (see Table 1). Neuroscience was among the most prevalent subfields in all 3 years, especially in 2013, although clinical was also prominent. The representation of neuroscience within psychology job advertisements increased from 40% in 2011 and 33% in 2012 to 50% in 2013, whereas the representation of clinical remained fairly steady across years (between 33% and 40%). Cognitive and neuroscience fluctuated together across the 3 years, with no greater than 3 percentage points separating the two categories in any of the 3 years. This trend may be reflective of a hiring trend involving new variants of cognitive neuroscience, such as computational cognitive neuroscience (Gros, 2009). Social/personality also increased from 24% in 2011 to 29% in 2012 to 48% in 2013, perhaps at least in part reflecting the increasing prominence of social neuroscience (e.g., Cacioppo & Decety, 2011; Ochsner & Gross, 2008). Within positions referencing, preferring, or requiring neuroscience expertise, neuroimaging was at least mentioned in 22% of advertisements in 2011, 50% of advertisements in 2012, and 44% of advertisements in 2013.

These statistics suggest that faculty hiring in neuroscience occupies a prominent role in psychology departments, with positions including a neuroscience emphasis accounting for one third to half of faculty lines in recent years (and with neuroimaging positions accounting for up to half of faculty lines in the neuroscience category). Whether this trend is healthy or unhealthy for our field is a legitimate topic of debate. To the degree to which neuroscience research is interdisciplinary, this trend may be viewed to some extent as expanding or supplementing, rather than replacing, other areas of expertise. In contrast, decreasing the emphasis on hiring faculty in nonneuroscience fields of psychology may in some cases be problematic, especially if neuroscientists in certain psychology departments do not have a sufficient number of potential collaborators in other domains (e.g.,

social psychology, personality, developmental psychology, cross-cultural psychology) to forge meaningful connections in their research across diverse levels of analysis.

Along with research programs undertaken by individual researchers, hiring priorities and decisions hold substantial implications regarding which subfields of psychology will be allowed to flourish. In turn, new faculty hires will be training future generations of students. Moreover, the breadth versus narrowness of candidates who are hired will shape the breadth versus narrowness to which future generations of students will be exposed and with which they are trained. If the percentage of tenure-track psychology faculty lines geared toward neuroscientists continues to increase, the risk is that we may witness a gradual atrophy of other subfields as individuals working in these subfields age, retire, and are not replaced with new hires working in the same or similar areas. Hence, these statistics merit further thoughtful investigation and discussion by psychology faculty members and administrators.

## Methodological Rigor and Implications for Methodological Training

The neuroscience movement may also hold implications for the conduct and methodological rigor of research in psychology. One might legitimately contend that our unmatched capacity to design, conduct, analyze, and interpret behavioral research is what makes psychology unique as a discipline (Lilienfeld, 2012b). Heretical as it may appear to ask, might the heightened focus on neuroscience diminish our field's distinctive contribution in this regard?

## Statistical Power and Replicability in Neuroscience Studies

Although statistical power has been a concern in many areas of psychology for several decades (Cohen, 1962; Rosnow & Rosenthal, 1989), recent concerns regarding the replicability of findings in medicine, psychology, and other fields (Ioannidis, 2005; Pashler & Wagenmakers, 2012) have sensitized psychological researchers to the need to ensure that studies are adequately powered. It is commonly understood that underpowered studies may be less likely than adequately powered studies to detect genuine effects. What appears to be less well known is that, when positive results do emerge from underpowered studies, they are more likely than results from adequately powered studies to be statistical flukes, a phenomenon known as the "winner's curse" (Button et al., 2013). For example, Forstmeier and Schielzeth (2011) found that, when using generalized linear modeling techniques with small sample sizes ( $N \leq 50$ ), Type I error rates may be as high as 60% if interaction terms are included in the model. Those authors found that this Type I error inflation is most likely due to overestimation of

**Table 1**  
*Percent of Advertisements in Which Subfields Were Required, Encouraged, and/or Mentioned*

Subfield	2011 Advertisements	2012 Advertisements	2013 Advertisements
Neuroscience	39.7%	33.3%	50.0%
Social/Personality	24.1%	29.2%	47.8%
Developmental	27.6%	19.8%	41.3%
Cognitive	41.4%	30.2%	50.0%
Clinical	39.7%	33.3%	37.0%
Quantitative	22.4%	20.8%	17.4%

effect sizes. By definition, findings based on Type I errors should not be replicable.

These considerations are relevant to ongoing debates concerning the role of neuroscience in psychology, because many human neuroscience studies, especially those in the field of neuroimaging, are severely underpowered. Button et al. (2013) found that the average post hoc statistical power of neuroimaging studies was approximately 8% (one tenth of the standard 80% that is considered desirable as a lower bound for statistical power; Cohen, 1988), and the average post hoc statistical power for human neuroscience studies in general was approximately 18%. Along with low sample sizes, high levels of measurement error are another potential challenge for the statistical power of neuroimaging studies. In a review of imaging studies, Bennett and Miller (2010) reported that the test-retest reliabilities of fMRI measures were generally modest, with intraclass correlations averaging .50. These reliabilities would not typically be deemed acceptable for standard psychological measures such as questionnaires or interviews. Given NIMH's ambitious agenda, and given its current director's expressed stance that "we can understand mental disorders as developmental brain disorders . . . [where] recent research hints at the heterogeneity of mental disorders at the level of genes and brain circuits" (Insel, 2009, p. 130), it is critical that research conducted to implement this agenda and test these propositions be adequately powered and replicable.

Exacerbating concerns regarding the winner's curse in neuroscience is a review (Jennings & Van Horn, 2012) suggesting that the neuroimaging literature is characterized by positive publication bias, such that studies that do not support linkages between brain activation patterns and either behavior or subjective experience are considerably less likely than other studies to be accepted for publication. If so, the linkages between neural correlates and psychological phenomena—and the contention that neural processes underlie psychological processes—may be weaker in magnitude than often assumed. In fairness, however, the problem of positive publication bias within psychology is hardly unique to neuroscience (Ferguson & Brannick, 2012).

Further, if researchers are to be pressured by grant review committees to engage in neuroscience research, and if psychology departments are increasingly directing their tenure-track faculty lines toward neuroscientists, then the level of methodological rigor of research in this area, especially the statistical power of empirical studies, must increase. One means of accomplishing this goal is to strongly encourage collaboration across neuroimaging laboratories that use a common methodological protocol (see Poline et al., 2012). This approach is not a panacea, in part because variations across fMRI laboratories account for about 8% of the variance in blood oxygen-level dependent signal results (Costafreda et al., 2007). Another investigation found that the median intraclass cor-

relation of fMRI findings across imaging centers that used the same hardware configurations was only .22 (Friedman et al., 2008). These formidable methodological challenges notwithstanding, data sharing procedures across laboratories will be necessary steps toward boosting statistical power and thereby diminishing the risk of both false negative findings and the winner's curse.

## Training of Students

The breadth of psychology as a field has traditionally been an advantage for students, in that there are many potential areas and subfields from which to choose. Psychology is applicable to the school system, the legal system, intergroup processes, the workplace, the family, inpatient and outpatient psychological clinics, medical settings, the armed forces, and many other arenas. A wide variety of disciplinary and theoretical approaches can be drawn upon to address problems in these and other settings, providing a rich array of opportunities for students to receive training and seek employment. For example, students seeking to study the workplace can adopt a social, clinical, developmental, or legal psychology perspective and can choose from among a diverse array of theoretical and metatheoretical orientations.

The increasing biologization and medicalization of our field poses a challenge in this regard. One often-overlooked implication of the recent neuroscience movement within psychology concerns the training of students, particularly graduate students. As we noted earlier, the trend toward hiring more faculty members with neuroscience interests almost certainly decreases the number of faculty lines available in other areas. If taken to an extreme, this movement has the potential to constrict the range of areas in which students receive training. If sufficient numbers of faculty members are not hired in areas related to potential emergent-property phenomena, such as adolescent peer affiliations and the intergroup processes responsible for political conflict, there is a risk that graduate students would not be exposed to the sufficient breadth of training that they need to understand, appreciate, and master the broader subject matter of psychology. As a consequence, opportunities to train students for careers in these areas—either within or outside academia—may erode along with them. If emergent-property areas are deemphasized, students may lose opportunities to be trained in methodological approaches (e.g., psychometrics) and conceptual foundations (e.g., the phenomenology of emotion) that lie at a largely psychological level of analysis.

An interrelated concern relates to graduate training in methodology and statistics. As observed earlier, this concern has taken on renewed importance with the recognition that many findings in psychology, including neuroscience, may be less replicable than previously assumed (Pashler &

Wagenmakers, 2012). This may even be true in domains often assumed to be highly replicable, such as structural brain imaging research (Boekel et al., 2013). Indeed, given the well-documented problems with replicability in biologically and medically based research (Dwan et al., 2008; Ioannidis, 2008), and given that the NIH is now emphasizing the replicability of scientific research (Collins & Tabak, 2014), it is essential that students in all fields of psychological science be trained in the most rigorous scientific and statistical methods.

We therefore sought to obtain a sense of the extent of methodological and statistical training that neuroscience graduate students are receiving in psychology departments, relative to the extent of such training obtained by students in other subfields of psychology. We identified the top 50 universities as ranked by *U.S. News and World Report* where a neuroscience doctoral program was housed within a psychology department, and in which a social/personality psychology doctoral program (where social/personality psychology is known for rigorous methodological training; Smith & Harris, 2006) was also available. We contacted the program directors for these doctoral programs and asked for the number of methodological and statistics courses required for each program. Of the 50 programs that we contacted, 32 (64%) responded and indicated that they had both neuroscience and social/personality doctoral programs housed within their psychology department, and of these 32, 23 provided information on both neuroscience and social/personality doctoral programs. Within these 23 programs, on average, neuroscience students were required to take 2.22 ( $SD = 1.13$ ) methodological and statistical courses, whereas social/personality students were required to take 3.22 ( $SD = 1.54$ ) methodological and statistical courses. The difference between the number of required methodology/statistics courses across the two types of programs was statistically significant,  $t(22) = 2.90$ ,  $p < .01$ , Cohen's  $d = .74$ , reflecting a large effect size. These numbers suggest that, at least relative to their counterparts in social/personality psychology, neuroscience students in psychology departments may not be receiving potentially crucial methodological and statistical training. Moreover, these findings corroborate and update those of a previous survey of psychology graduate programs, which similarly suggested that neuroscience graduate students in psychology departments received significantly less exposure to statistics and methodology compared with students in other areas of psychology (Aiken, West, & Millsap, 2008). Of course, given that neuroscience research is often conducted—and neuroscience courses taught—in multiple departments (e.g., psychology, biology, medicine, engineering, economics), some universities may provide statistical or other methodological training in nonpsychology departments (and such training would not have been counted in either our or Aiken et al.'s reviews). Students may also take methodological and statis-

tical courses beyond what is required by their respective programs. Nonetheless, at the very least, these findings raise the question of whether neuroscience graduate students in psychology departments will enter research positions with sufficient statistical and methodological knowledge to benefit from and collaborate effectively with colleagues in other subfields of psychology.

## Quo Vadis? The Role of Neuroscience Within a Full-Fledged Discipline of Psychology

With these considerations in mind, how should our field move forward, and what role should neuroscience play within a complete discipline of psychology? The myriad challenges we have outlined will require psychology to give further thought to its boundaries. Clearly, neuroscience is an essential component of psychology because it allows us to understand the workings of the brain, the effects of brain damage on psychological and neurological functioning, and the brain regions and processes associated with specific mental activities. Such information is helpful, and at times critical, for such clinical disciplines as neurology and neurosurgery, whose missions focus on restoring brain functioning in impaired or disabled individuals. Information gleaned from neuroscience studies is also enormously useful for clinical neuropsychology, which focuses largely on identifying functional deficits tied to damage in specific brain regions. We are also certain that neuroscience will become increasingly important for an adequate understanding of psychological and social problems, especially in conjunction with higher levels of analysis. Our question, again, is, "What else is important in psychology?" Although sociologists often study social problems such as unemployment, nonmarital childbearing, and immigration, psychology is needed to study how these problems affect the lives of individuals and the ways in which they can be ameliorated through interventions. A combination of neuroscience and social science approaches will be necessary for addressing these issues.

One step that would be helpful in this regard is to expand the nomological network surrounding the term *health*. Probably fueled in part by the appointments of physicians and biologists as directors of the majority of NIH institutes, "health" is now defined in largely biological terms. The U.S. government's Healthy People 2020 initiative (Healthy People, 2012) includes such biological indicators as arthritis, blood disorders, cancer, diabetes, and HIV, as well as such health-related behaviors as substance use and nutrition. For the first time, psychological well-being, defined in social-science terms, is listed as an objective of Healthy People 2020. This is certainly grounds for optimism, provided that funding agencies are willing to support research with well-being as a primary outcome. Further, "health" could be expanded to include a supportive work or school

environment, a coherent sense of identity that can support effective decision-making regarding career and relationships, and successful postmigration adaptation among immigrants. As we have discussed throughout this article, psychology is both a social science and a natural science, and both of these aspects of our discipline must be nurtured and allowed to flourish. Indeed, collaborations between social-science and biologically based psychologists will be needed to address key health challenges of the 21st century, especially when health is conceptualized in both social-science and biological terms.

### Implications for the Training and Education of Students

Psychology is both a basic science and an applied, service-oriented field, and this dual mission requires a focus on complementary natural-science and social-science topics and methods. As observed earlier, as a field, we need to ensure that graduate students in biological areas of psychology receive adequate training in statistics and methodology to allow them to profit from collaborations with researchers in other domains. In addition, these students, as well as students in other subfields of psychology, need to be equipped not merely with the substantive and technical skills of their specialty, but also with critical thinking skills that will allow them to avoid the inferential leaps about which we have cautioned, such as neuroessentialism, neurorrealism, and uncorroborated reverse inference (Racine et al., 2005). In this way, they should hopefully be less susceptible to widespread and tempting misinterpretations of data derived from neuroimaging methods and other paradigms.

To allow them to bridge across multiple levels of analysis when they become full-fledged researchers, graduate students across all domains of psychology also need to be provided with as broad a knowledge base as possible (Cacioppo, 2013; Lilienfeld & O'Donohue, 2007). They cannot reasonably be expected to become experts in all psychology subfields, of course, but they need sufficient exposure to—and appreciation of the scientific value of—diverse levels of analysis to profit from collaborations with scholars who approach psychology from diverse explanatory perspectives. The recently articulated Delaware Project model of clinical science training (Shoham et al., 2014) may serve as a useful blueprint not only for clinical psychology, but also for many other psychological domains. Among other goals, this project emphasizes “collaborative fluency” (p. 12) and exposure to cutting-edge developments across multiple domains of psychology, including neuroscience, the basic sciences of learning, affect, and cognition, social psychology, and the like.

### Conclusion: The Future of Psychology

We have begun with a noncontroversial proposition: Brain science *is* an integral component of psychology. The argument we have advanced is simply that social-science concerns are essential as well, and that psychology must maintain a focus on such concerns as it continues to expand into natural science domains. If traditional psychology is to increase its areas of convergence with the natural sciences, a goal we applaud, it must also retain at least one foot in social science so that a wider, integrative scientific perspective can be formulated to include both social and biological components. Conversely, if neuroscience is to thrive and flourish, it must collaborate effectively with other domains of psychology to establish bridge laws and forge meaningful linkages across different levels of analysis (Marcus, 2014). In doing so, it must be careful not to accord short shrift to one or more of these levels.

Clearly, neuroscience has brought an enormous wealth of data and knowledge to the field of psychology. It has helped psychologists to begin to demystify the brain, to learn how the brain develops and adapts, and to better identify the links between brain functions and psychological processes. Understandably, psychologists have become fascinated by neuroscience and the scientific potential that it brings. Our mission has been to acknowledge these important advances while raising key caveats regarding how subfields of psychology that lie higher in the explanatory hierarchy must also be encouraged to flourish. We have also argued that neuroscience must be humble in its aspirations, and not advance claims or make promises that go well beyond the extant data, such as assertions that an understanding of the brain is leading to a revolution in psychotherapy (e.g., Cozzolino, 2002). Just as important, we have aimed to advance the complementary argument that neuroscientific approaches to psychology are unlikely to flourish without an adequate consideration of higher levels of analysis.

In the terminology of Knudsen (2003), the field of psychology must be careful to avoid falling into a “unification trap” (see also Henriques, 2014). Such a trap occurs when one dominant paradigm, in this case neuroscience, becomes so powerful that it begins to discourage intellectual growth in other paradigms, resulting in the fervent monism that troubled Kendler (2014; see also N. Eisenberg, 2014). As Knudsen observed, unification traps can become reinforced by the short-term success of a single methodological or conceptual approach at Kuhnian puzzle-solving (see Kuhn, 1970), which can in turn lead to premature enthusiasm regarding this approach’s scientific potential. Although striving toward unification and consilience is a laudable scientific goal (Wilson, 1998), this goal cannot be accomplished without a full appreciation of multiple levels of analysis.

The optimal contribution of neuroscience is, in our view, to *add to* and *complement*, rather than to *replace*, social-science literatures. The growing priorities placed on the hiring of neuroscientists in psychology departments may be counterproductive in this regard, especially if these trends continue to accelerate. If we are seeking to harness the interdisciplinary potential of both natural science and social science approaches to psychology (Magnusson, 2012), our hiring priorities and funding initiatives need to reflect this goal.

As observed earlier (see also Miller, 2010; Satel & Lilienfeld, 2013), the causal links between psychological and biological processes are almost certainly bidirectional in most cases. An eliminative reductionist perspective, in which behaviors, thoughts, feelings, and other experiences can be completely explained by biological processes at the cellular and molecular levels, may be difficult to square with much current scholarship in neuroscience and in the broader field of psychology. Nevertheless, given the dependence of researchers, departments, and universities on federal grant funding, priorities emphasized by funding agencies and by their review committees may “force the hands” of researchers, departments, and universities to prioritize neuroscience at the expense of other approaches. In our view, this state of affairs would be counterproductive. The bidirectional links between neural and behavioral processes can be studied adequately only if such research is robustly and consistently supported at the funding agency level.

Moreover, if some psychological questions are most effectively addressed at the social-science level, such as the unemployment crisis among young adults and the best means of ameliorating marital conflict, social-science research must continue to be among the major priorities at funding agencies. A Department of Psychological Science can focus primarily on social science research and still be true to its name. With the benefit of hindsight, we can understand why Watson (1928) and other early methodological behaviorists argued for delimiting psychology to the science of observable behavior. Their position was largely in response to the predominance of both psychoanalytic approaches on unconscious conflicts and to the research of introspectionists, such as Titchener and his colleagues (who purported to use the self-examination of mental contents to derive a model of the structure of consciousness; Green, 2010), which they viewed with some justification as undermining psychology’s aspiration to scientific rigor. At the same time, we can acknowledge that research on personality traits and motives, values, self-perceptions, and agency can be scientific. As a field, psychology should aim to be inclusive, rather than exclusive, in terms of what is included under its auspices (Pervin, 2012). If our field is to be inclusive, funding agency priorities, hiring practices in psychology departments, and the training of graduate students must reflect this goal (Cacioppo, 2013).

Finally, psychologists must continue to focus on the areas in which our expertise is sorely needed. These domains range from understanding the roots of, and solutions for, intergroup conflict to improving relationship stability and career trajectories to inhibiting the progression of diabetes, cancer, HIV, and other chronic and infectious diseases. Both social-science and biologically based psychological expertise will be needed to accomplish these goals, and each type of expertise will be more or less important for specific questions. Some social problems will require social-science expertise; some physiological problems will require natural-science expertise, and most problems will surely require both types of expertise. We must therefore ensure that both types of expertise are promoted, retained, and nurtured. It is our hope that this article will play at least a modest role in launching an important and long overdue conversation about the future of psychology and our shared investment in that future.

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Received February 20, 2014

Revision received May 18, 2015

Accepted June 17, 2015 ■



# Neural correlates of interspecies perspective taking in the post-mortem Atlantic Salmon: An argument for multiple comparisons correction

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## INTRODUCTION

With the extreme dimensionality of functional neuroimaging data comes extreme risk for false positives. Across the 130,000 voxels in a typical fMRI volume the probability of a false positive is almost certain. Correction for multiple comparisons should be completed with these datasets, but is often ignored by investigators. To illustrate the magnitude of the problem we carried out a real experiment that demonstrates the danger of not correcting for chance properly.

## METHODS

**Subject.** One mature Atlantic Salmon (*Salmo salar*) participated in the fMRI study. The salmon was approximately 18 inches long, weighed 3.8 lbs, and was not alive at the time of scanning.

**Task.** The task administered to the salmon involved completing an open-ended mentalizing task. The salmon was shown a series of photographs depicting human individuals in social situations with a specified emotional valence. The salmon was asked to determine what emotion the individual in the photo must have been experiencing.

**Design.** Stimuli were presented in a block design with each photo presented for 10 seconds followed by 12 seconds of rest. A total of 15 photos were displayed. Total scan time was 5.5 minutes.

**Preprocessing.** Image processing was completed using SPM2. Preprocessing steps for the functional imaging data included a 6-parameter rigid-body affine realignment of the fMRI timeseries, coregistration of the data to a  $T_1$ -weighted anatomical image, and 8 mm full-width at half-maximum (FWHM) Gaussian smoothing.

**Analysis.** Voxelwise statistics on the salmon data were calculated through an ordinary least-squares estimation of the general linear model (GLM). Predictors of the hemodynamic response were modeled by a boxcar function convolved with a canonical hemodynamic response. A temporal high pass filter of 128 seconds was included to account for low frequency drift. No autocorrelation correction was applied.

**Voxel Selection.** Two methods were used for the correction of multiple comparisons in the fMRI results. The first method controlled the overall false discovery rate (FDR) and was based on a method defined by Benjamini and Hochberg (1995). The second method controlled the overall familywise error rate (FWER) through the use of Gaussian random field theory. This was done using algorithms originally devised by Friston et al. (1994).

## DISCUSSION

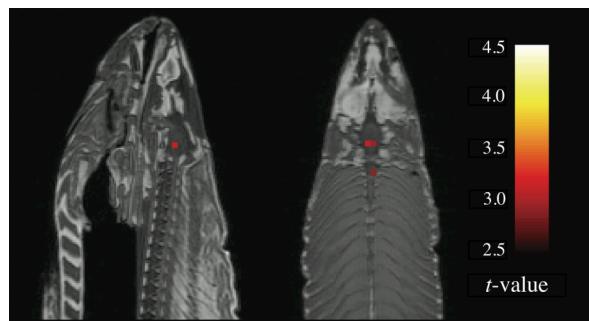
Can we conclude from this data that the salmon is engaging in the perspective-taking task? Certainly not. What we can determine is that random noise in the EPI timeseries may yield spurious results if multiple comparisons are not controlled for. Adaptive methods for controlling the FDR and FWER are excellent options and are widely available in all major fMRI analysis packages. We argue that relying on standard statistical thresholds ( $p < 0.001$ ) and low minimum cluster sizes ( $k > 8$ ) is an ineffective control for multiple comparisons. We further argue that the vast majority of fMRI studies should be utilizing multiple comparisons correction as standard practice in the computation of their statistics.

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## GLM RESULTS

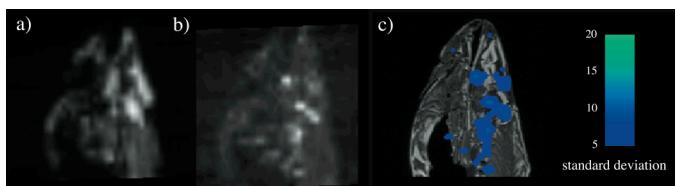


A  $t$ -contrast was used to test for regions with significant BOLD signal change during the photo condition compared to rest. The parameters for this comparison were  $t(131) > 3.15$ ,  $p(\text{uncorrected}) < 0.001$ , 3 voxel extent threshold.

Several active voxels were discovered in a cluster located within the salmon's brain cavity (Figure 1, see above). The size of this cluster was  $81 \text{ mm}^3$  with a cluster-level significance of  $p = 0.001$ . Due to the coarse resolution of the echo-planar image acquisition and the relatively small size of the salmon brain further discrimination between brain regions could not be completed. Out of a search volume of 8064 voxels a total of 16 voxels were significant.

Identical  $t$ -contrasts controlling the false discovery rate (FDR) and familywise error rate (FWER) were completed. These contrasts indicated no active voxels, even at relaxed statistical thresholds ( $p = 0.25$ ).

## VOXELWISE VARIABILITY



To examine the spatial configuration of false positives we completed a variability analysis of the fMRI timeseries. On a voxel-by-voxel basis we calculated the standard deviation of signal values across all 140 volumes.

We observed clustering of highly variable voxels into groups near areas of high voxel signal intensity. Figure 2a shows the mean EPI image for all 140 image volumes. Figure 2b shows the standard deviation values of each voxel. Figure 2c shows thresholded standard deviation values overlaid onto a high-resolution  $T_1$ -weighted image.

To investigate this effect in greater detail we conducted a Pearson correlation to examine the relationship between the signal in a voxel and its variability. There was a significant positive correlation between the mean voxel value and its variability over time ( $r = 0.54$ ,  $p < 0.001$ ). A scatterplot of mean voxel signal intensity against voxel standard deviation is presented to the right.

