Mechanisms of Motivation and Emotion

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FOUNDATIONS FOR UNDERSTANDING

The Nature and Value of Emotions Effects of Bodily Responses on Emotional Feelings Brain Mechanisms of Emotion The kaleidoscope that makes a day or a year of mental life has both fast-moving and slow-moving components. Sensations, perceptions, thoughts, and muscle movements flit through our consciousness and behavior at speeds measured in milliseconds. But slower changes, measurable in minutes or hours, modulate and help direct these rapid changes. The slower changes are referred to as *behavioral states*; they include variations in motivation, emotion, and level of arousal.

Are you hungry right now, or angry, or sleepy? Even as you attempt to study this chapter, your mental state affects your capacity to pay attention, and it may direct your attention to some things over others. If you are hungry, your thoughts of food may be capturing most of your attention. If you are sleepy, your reaction even to the most interesting ideas in the chapter may be "oh hmmmm ... zzzzzzz." Clearly, your mental state affects your momentary thoughts and actions. But what affects your mental state? What exactly makes you hungry, or angry, or sleepy? This is a fundamental question, one that links psychology tightly to the study of the brain and its interactions with the body's internal environment as well as the external environment. This chapter is about the physiological underpinnings of motivation and emotion. You will read first about general principles of motivation, from a physiological perspective, and then about hunger, sexual drive, sleep, dreams, and emotionality-in that order. Social and cultural influences on motivation and emotion, which are only touched on here, are discussed more fully in later chapters.

General Principles of Motivation

To *motivate*, in the most general sense of the term, is to set in motion. In psychology, the term *motivation* is often used to refer to the entire constellation of factors, some inside the organism and some outside, that cause an individual to behave in a particular way at a particular time. Motivation defined this way is a very broad concept, almost as broad as all of psychology. Every chapter in this book deals with one or another facet of motivation. Genes, learning, physiological variables, perceptual and thought processes, developmental variables, social experiences, and personality characteristics are all constructs that psychologists describe as contributors to motivation.

A more precise label for the specific topic of our present discussion is *motivational state* or *drive*. These terms are used interchangeably to denote an internal condition that orients an individual toward a specific category of goals and that can change over time in a reversible way. Different drives have different

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Motivation The predatory drive of this cat is activated by the goldfish-the incentive

goals. *Hunger* orients one toward food, *sex* toward sexual gratification, *curiosity* toward novel stimuli, and so on. For the most part, drives are thought of in psychology as hypothetical constructs. The psychologist does not observe a state of hunger, thirst, or curiosity inside the animal but infers the existence of that state from the animal's behavior. An animal is said to be hungry if it behaves in ways that bring it closer to food, to be sexually motivated if it behaves in ways that bring it into contact with a sexual partner, and to be curious if it seeks out and explores new environments. To say that the drive varies over time is to say that the animal will work harder, or accept more discomfort, to attain the goal at some times than at others. The assumption is that something inside the animal changes, causing it to behave differently, at different times in the same environment.

How do drives and incentives (a) complement one another and (b) influence one another in their contributions to motivation?

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Eut the inside interacts constantly with the outside. Motivated behavior is directed toward *incentives*, the sought-after objects or ends that exist in the external environment. Incentives are also called *reinforcers* (the term used in Chapter 4), *rewards*, or *goals*. The motivational state that leads you to stand in line at the cafeteria is presumably hunger, but the incentive for doing so is the hamburger you intend to purchase. Drives and incentives complement one another in the control of behavior; if one is weak, the other must be strong to motivate the goal-directed action. Thus, if you know that the cafeteria's hamburger tastes like cardboard (weak incentive), you are likely to wait in line for it only if your hunger drive is

strong; but if the cafeteria serves a really great hamburger (strong incentive), you are likely to wait even if your hunger drive is weak. Drives and incentives not only comple-

Drives and incentives not only complement each other but also influence each other's strength. A strong drive can enhance the attractiveness (incentive value) of a particular object: If you are very hungry, even a hamburger that tastes like cardboard might seem quite attractive. Conversely, a strong incentive can strengthen a drive: The savory aroma of a broiling hamburger wafting your way as you wait in line might increase your hunger drive, which might in turn induce you to eat something that previously wouldn't have interested you if, by the time you get to the grill, all the hamburgers are gone.



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Varieties of Drives

In general, drives motivate us toward goals that promote our survival and reproduction. Some drives promote survival by helping us maintain the internal bodily conditions that are essential for life.

Drives That Help Preserve Homeostasis

In a now classic book entitled *The Wisdom of the Body* (1932/1963), the physiologist > Walter B. Cannon described simply and elegantly the requirements of the tissues of the human body. For life to be sustained, certain substances and characteristics within the body must be kept within a restricted range, going neither above nor below certain levels. These include body temperature, oxygen, minerals, water, and energy-producing food molecules. Physiological processes, such as digestion and respiration, must continually work toward achieving what Cannon termed *homeostasis*, the constancy of internal conditions that the body must actively maintain. Most relevant for psychology, Cannon pointed out that maintenance of homeostasis involves the organism's outward behavior as well as its internal processes. To stay alive, individuals must find and consume foods, salts, and water and must maintain their body temperature through such means as finding shelter.

Cannon theorized that the basic physiological underpinning for some drives is a loss of homeostasis, which acts on the nervous system to induce behavior designed to correct the imbalance.

Following Cannon, psychologists and physiologists performed experiments showing that animals indeed do behave in accordance with the needs of their bodily tissues. For example, if the caloric (energy) content of its food is increased or decreased, an animal will compensate by eating less or more of it, keeping the daily intake of calories relatively constant. As another example, removal of the adrenal glands causes an animal to lose too much salt in its urine (because one of the adrenal hormones is essential for conserving salt). This loss of salt dramatically increases the animal's drive to seek out and eat extra salt, which keeps the animal alive as long as salt is available (Stricker, 1973).

The force of homeostasis in human behavior was dramatically and poignantly illustrated by the clinical case of a boy, referred to as D. W., who when 1 year old developed a great craving for salt (Wilkins & Richter, 1940). His favorite foods were salted crackers, pretzels, potato chips, olives, and pickles; he would also take salt directly from the shaker. When salt was denied him, he would cry until his parents gave in, and when he learned to speak, *salt* was one of his first and favorite words. D. W. survived until the age of 3½, when he was hospitalized for other symptoms and placed on a standard hospital idet. The hospital staff would not yield to his demands for salt, and he died within a few days. An autopsy revealed that his salt craving came from physiological need. His strong drive for salt and his ability to manipulate his parents into supplying it, even though they were unaware that he needed it, had kept D. W. alive for 2½ years—powerful evidence for "the wisdom of the body."

Limitations of Homeostasis: Regulatory and Non-Regulatory Drives

Homeostasis is a useful concept for understanding hunger, thirst, and the drives for >> salt, oxygen, and an appropriate body temperature, but not for understanding many other drives. Consider sex, for example. People are highly motivated to engage in sex, and sex serves an obvious evolutionary function, but there is no tissue need for it. No vital bodily substance is affected by engaging in sexual behavior; nobody can die from lack of sex (despite what an overly amorous someone may have

How is the concept of homeostasis related to that of drive? How is this relationship demonstrated in the case of a little boy who craved salt?



Ahh, homeostasis When the body's water level is reduced from prolonged athletic exertion, nothing is more satisfying than replenishing it. Thirst is a typical example of a regulatory drive.

> ______3 What is the distinction between regulatory and non-regulatory drives, and how can mammalian drives be classified into five categories based on function?

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told you). Psychologists find it useful to distinguish between two general classes of drives—regulatory and non-regulatory. A **regulatory drive** is one, like hunger, that helps preserve homeostasis, and a **non-regulatory drive** is one, like sex, that serves some other purpose.

A Functional Classification of Mammalian Drives

One way to think about the whole set of drives that we share with other mammals is to categorize them in accordance with their evolutionary functions—their roles in promoting survival and reproduction. In this regard, you may find it useful to distinguish among the following five categories of mammalian drives:

- Regulatory drives. As already noted, these are drives that promote survival by helping to maintain homeostatic conditions in the body. *Hunger* and *thirst* are prime examples.
- 2. Safety drives. These are drives that motivate an animal to avoid, escape, or fend off dangers such as precipices, predators, or enemies. The most obvious safety drive is *fear*, which motivates individuals to flee from danger. Another is *anger*, which is manifested when fight (or at least threat) rather than flight is needed to ensure one's safety. I will argue later in this chapter that *sleep* is also a safety drive. It evolved at least partly as a means of keeping animals tucked quietly away during that part of each 24-hour day when they would be most in danger if they were moving around.
- **3.** *Reproductive drives.* The most obvious of these are the sexual drive and the drive to care for young once they are born. When they are at a peak, these drives can be extraordinarily powerful. Animals (including people) will risk their lives to mate and to protect their offspring. As discussed in Chapter 3, sexual jealousy, including the anger associated with it, also serves the function of reproduction to the degree that it promotes the fidelity of one's sexual partner.
- 4. Social drives. Most mammals, and especially humans, require the cooperation of others to survive. The social drives include the drives for friendship and for acceptance and approval by the social groups of which one is a part. In humans, these drives can be as powerful as the regulatory, safety, and reproductive drives. People will risk their lives for friendship and for social approval. The social drives figure prominently in the discussions that run through Chapters 12, 13, and 14 of this book.
- **5.** *Educative drives.* These consist primarily of the drives to play and to explore (curiosity). As discussed in Chapter 4, the young of nearly all mammals practice life-sustaining skills through play, and mammals of all ages acquire useful information about their environment by exploring novel objects and territories. When other drives are not too pressing, the drives for play and exploration come to the foreground.

Human Drives That Seem Not to Promote Survival or Reproduction

What are two possible explanations of the universal human drives for art, music, and literature?

Not all of human motivation is easily understood in terms of survival and reproduction. For instance, humans everywhere like to produce and experience art, music, and literature (including oral stories and poetry). What motivates these activities? Do we have special, evolved aesthetic drives? If so, what adaptive functions prompted the natural selection of such drives?

At present, these questions are much debated and there is no firm answer. My own view is that the pursuits of art, music, and literature are natural extensions of our drives for play and exploration. These pursuits can exercise perceptual and motor skills, imagination, and creative thinking in ways that may be useful in future real-life situations and can also provide us with ideas for governing our own lives. Like other playful and exploratory activities, these pursuits help our minds to grow during periods when there are no more pressing survival needs that must be fulfilled.



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A somewhat different (but not incompatible) view, presented by Steven Pinker (1997), is that art, music, and literature appeal to us not because we have special drives for them but because they tap into many of our already existing drives and proclivities, which evolved for other purposes. For example, in describing the appeal of fiction, Pinker (1997, p. 539) writes: "When we are absorbed in a book or movie, we get to see breathtaking landscapes, hobnob with important people, fall in love with ravishing men and women, protect loved ones, attain impossible goals, and defeat wicked enemies." In this example, a book or movie appeals to our drives for sex, love, social esteem, parenting, achievement, and aggression. To suggest that art, music, and literature may be vicarious means of satisfying other drives rather than drives in and of them-

selves is not to diminish them. These pursuits enrich our lives immensely; they extend us beyond evolution's narrow dictates of mere survival and reproduction.

Of course, some things that people become motivated for are truly harmful. Drug addictions and compulsive gambling are artificial drives, created by human inventions, which can ruin people's lives. How these tap artificially into our natural drive mechanisms is a topic to which we shall return soon.

Early art We have no idea whether or not the person who produced this drawing—about 18,000 years ago in one of the Lascaux Caves, in France had a concept of or word for *art*. To the modern eye, however, this is a work of art. It may represent a universal human proclivity.

Reward Mechanisms of the Brain

As I noted earlier, motivated behavior involves the pursuit of *rewards* (also known as incentives, goals, or reinforcers). Let us now look more closely at the concept of reward and at research into how rewards act on the brain to promote and reinforce the behaviors that produced them.

Three Components of Reward: Liking, Wanting, and Reinforcement

Psychologically, the term *reward* has three interrelated but in some ways separable >>>meanings. A reward is something that we *like*, something that we *want*, and something that serves as a *reinforcer* in learning (Berridge & Robinson, 2003).

Liking refers to the subjective feeling of pleasure, or satisfaction, that occurs when one receives a reward. We know this feeling from our own experience. We experience pleasure from good food when we are hungry, from water when we are thirsty, from drifting off to sleep when we are tired, and from sexual activity when we are sexually motivated. We also experience pleasure from pay, praise, the company of good friends, play, music, discoveries made through exploration, and our own assessment of a job well done. Most of the things we seek in life bring us pleasure when we obtain them. We have no way to know for sure that other animals also experience pleasure from the rewards they receive, but their behavior certainly suggests that they do (see Figure 6.1).



> 5 What are three interrelated components of the concept of reward?

IFIGURE 6.1 Liking Many species of mammals show a similar facial reaction to good-tasting foods. The reaction includes a tongue protrusion, which looks as if the individual were lapping up the last bits of the food. This expression has been used as an objective index of "liking" in research involving food rewards in laboratory rats. (Berridge & Robinson, 2003.)



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Wanting refers to the desire to obtain a reward. This is the component of reward that links closely to the concept of motivation. Whereas pleasure occurs when a reward is received, wanting occurs before it is received. Wanting is typically measured by assessing the amount of effort an individual will exert, or the amount of pain the individual will bear, in order to obtain the reward. Usually objects that are wanted are also liked, but, as you will see later, it is possible to separate the two.

Reinforcement refers to the effects that rewards have in promoting learning. As discussed in Chapter 4 (in the section on operant conditioning), animals and people learn to attend to stimuli that signal the availability of rewards and, in the presence of those stimuli, to make responses that bring rewards. Somehow, through its effects on the brain, a reward helps to stamp in, or reinforce, the memory of stimuli and actions that occurred just before the reward was received. Such learning helps the individual to become more effective in finding and procuring the same type of reward in the future.

Studies of the brain have provided some clues to the mechanisms of each of these three components of reward.

The study of brain mechanisms of reward was initiated in the 1950s, when

James Olds and Peter Milner made a remarkable discovery. These researchers ob-

served, by accident at first, that when rats received electrical stimulation through thin wires implanted in certain brain areas, they behaved as if they were trying to get more of that stimulation. For example, if a rat happened to receive the stimulation while exploring a particular corner of the cage, the animal would return repeatedly to that corner. To see if such brain stimulation would serve as a reinforcer for learning, Olds and Milner tested rats in an apparatus in which they could electrically stimulate their own brains by pressing a lever (see Figure 6.2). With electrodes placed in certain brain areas, rats learned very quickly to press the lever and continued to press at high rates, sometimes for many hours without stopping (Olds

Subsequent research showed that rats and other animals will work hardest and longest to stimulate a tract in the brain called the *medial forebrain bundle*. The neurons of this tract that are most crucial for this rewarding effect have their cell bodies in nuclei in the midbrain and synaptic terminals in a large nucleus in the basal ganglia called the nucleus accumbens [uh-cum-bens] (see Figure 6.3). The nucleus accumbens itself has connections to large areas of the limbic system and the cerebral cortex, and it is now understood to be a crucial center for the behav-

When Olds and Milner inserted electrodes into the medial forebrain bundle,

they were tapping artificially into the brain's natural reward system. Subsequent research, involving the recording of activity in the brain, has shown that the me-

dial forebrain bundle and the nucleus accumbens become active in all sorts of situ-

ations in which an individual receives a reward, whether the reward is food, opportunity to copulate, novel objects to explore, or (in humans) a prize received

Identification of Reward Neurons in the Brain

ioral effects of rewards, in humans as well as in other mammals.

How did Olds and Milner identify reward pathways in the brain?

What is some evidence that the medial forebrain bundle and nucleus accumber are essential pathways for the effects of a wide variety of rewards?

& Milner, 1954).

FIGURE 6.2 Lever pressing for electrical stimulation to the brain Each time this rat presses the lever it receives a brief pulse of electrical current through an electrode implanted in its brain. Some of Olds' rats continued to press the lever for 24 hours without rest, as often as 5000 times per hour. (From Olds, 1956.)



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IFIGURE 6.3 A reward pathway in the brain The medial forebrain bundle is a neural tract consisting of neurons whose cell bodies are in nuclei in the midbrain and whose synaptic terminals are in the nucleus accumbens of the basal ganglia. Rats will quickly learn to press a lever to electrically stimulate the medial forebrain bundle or the nucleus accumbens.

for winning a game (Breiter & others, 2001; Damsma & others, 1992; Fiorello & others, 2003). Moreover, damage to either of these brain structures destroys all sorts of motivated behaviors. Without a functioning medial forebrain bundle or nucleus accumbens, animals will not work to obtain rewards and will die unless they are given food and water artificially through tubes into their stomachs (Grossman, 1979; Stricker, 1982).

Separating the "Liking" and "Wanting" Systems

Many of the neurons of the medial forebrain bundle that terminate in the nucleus >> accumbens release *dopamine* [dope-uh-mean] as their neurotransmitter. This release appears to be essential for the "wanting" component of reward, but not for the "liking" component. Animals that have been well trained to press a lever for some reward, such as food, show a release of dopamine into the nucleus accumbens just before they begin to press the lever, but not after they receive the reward (Phillips & others, 2003). This pattern is consistent with the idea that dopamine helps motivate the animal to work for the reward (promotes "wanting") but is not essential for the pleasure received from obtaining the reward.

More direct evidence that dopamine is involved in "wanting" but not "liking" comes from studies in which rats are treated with drugs that block the effect of dopamine in the nucleus accumbens. These animals continue to consume foods, copulate with sexual partners, and explore novel stimuli that are immediately present. They also continue to exhibit the facial "liking" expression (shown in Figure 6.1) when they taste a sugar solution. However, they do not continue to seek out or work for rewards that are not immediately present (Berridge & Robinson, 2003; Bevins, 2001; López & Ettenberg, 2002; Zhang & others, 2003). Their behavior suggests that they continue to enjoy the consumption of rewards, but are no longer concerned with (no longer behave as if they want) rewards that are absent. Conversely, drugs that increase the activity of dopamine in the nucleus accumbens increase the rate at which rats and other animals will work for food, but do not increase the facial "liking" response to sucrose or the animal's consumption of food that is immediately available (Berridge & Robinson, 2003).

What is some evidence that different neurotransmitters in the nucleus accumbens are involved in the "wanting" and "liking" components of reward?

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If dopamine is responsible for the "wanting" component of reward, what is responsible for the "liking" component? Some of the neurons of the medial forebrain bundle that terminate in the nucleus accumbens release not dopamine but a different transmitter, one that is in the endorphin [en-dorf-in] family. The term endorphin is short for endogenous morphine-like substance. (Endogenous means "created within the body.") Endorphins are chemicals created within the body that have effects similar to those of morphine and other opiate drugs such as opium and heroin. As discussed in Chapter 7, endorphins are best known for their role in inhibiting the sense of pain. Recent experiments suggest, however, that endorphins released into the nucleus accumbens are also crucial for the immediate pleasure experienced when rewards are received or consumed. Injections, into the nucleus accumbens, of drugs that increase the effectiveness of endorphins increase the facial "liking" reaction to sucrose (Berridge & Robinson, 2003) and also increase the amount of immediately present food that an animal will eat (Zhang & Kelley, 2000). In humans, drugs that decrease the effectiveness of endorphins have been reported to decrease people's perceived enjoyment of food and other rewards (Yeomans & Gray, 1996).

< The learning component of reward is closely related to the "wanting" compo-</p>

nent. Animals learn to associate certain cues with the availability of a reward, and

able. One line of evidence for this contention is the observation that dopamine release promotes long-term potentiation (LTP) of neural connections within the nucleus accumbens (Reynolds & others, 2001). As discussed in Chapter 5, LTP is believed to be part of the cellular basis for learning throughout the brain.

Role of Dopamine in Reinforcement for Learning

What evidence suggests that dopamine is crucial to the capacity of rewards to promote new learning?

the capacity of rewards to provlearning? those cues lead the animal to search for or work for the reward, which is the behavioral indicator of "wanting." The release of dopamine into the nucleus accumbens appears to be crucial not just for motivating animals to work for rewards, but also for their ability to learn to use cues to predict when and where rewards are avail-

> Other evidence comes from studies in which the amount of dopamine released into the nucleus accumbens is directly measured as animals anticipate and receive rewards (Schultz, 1998). If food is occasionally, at unpredictable times, presented to a hungry rat, a burst of dopamine is released into the nucleus accumbens each time food is presented. If the situation is then changed so that a signal light comes on 2 seconds prior to each presentation of food, the animal soon learns to anticipate food each time the light comes on. In this new situation, after several trials, no burst of dopamine release occurs when the food is presented, but a burst occurs, instead, when the light comes on. The animal continues to eat and apparently enjoy the food, but no dopamine release accompanies that behavior.

> This pattern of dopamine release is consistent with the idea that dopamine is involved in new learning (Schultz, 1998). When a reward is unexpected, dopamine release immediately after the reward helps to reinforce the remembered association between the reward and any stimulus or response that happened to precede it. When the cues and responses leading to a reward have already been well learned, however, there is no need for further reinforcement of that learning, and dopamine release in response to the reward ceases. Dopamine release now occurs in response to the signal preceding reward, because now the animal's interest lies in learning how to predict when the signal will appear or how to make it appear.

Drug Addiction: Hijacking the Brain's Reward System

10 How does an understanding of the brain's reward system help us to understand drug addiction and compulsive gambling? When Olds and Milner delivered electrical impulses into the medial forebrain bundle in rats, they were short-circuiting the rats' natural reward systems. When people take drugs such as cocaine, amphetamine, heroin, or opium they are doing essentially that same thing with their own brains. All these drugs, and other oftenabused drugs as well, exert their euphoric and habit-producing effects through action on the brain's reward pathways (Nestler & Malenka, 2004). In various ways, these drugs mimic or promote the effects of dopamine and endorphins in the nucleus accumbens.

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Rats fitted with tubes and mechanisms for pumping drugs into their bloodstreams will self-administer cocaine and other such drugs, and become addicted, but will stop self-administering the drugs if the nucleus accumbens is destroyed or chemically blocked (Wise, 1996). Rats will work as hard to administer tiny amounts of cocaine or amphetamine through a cannula (tiny tube) directly into the nucleus accumbens as they will to administer much larger amounts into the bloodstream (Hoebel & others, 1983; Wood & Emmett-Oglesby, 1989).

Our understanding of the brain's reward mechanisms gives us a clue as to why such drugs are addictive. Not only do they produce an immediate sense of euphoria, but, even more significant for the problem of addiction, they strongly activate the dopamine-receiving neurons in the nucleus accumbens that are responsible for promoting reward-based learning. Normal rewards, such as food, activate these neu-

rons only when the reward is unexpected; but cocaine and other abused drugs activate these neurons every time the drug is taken. The result may be a sort of super learning (Self, 2003). With each dose of the drug, the dopamine response acts to reinforce, once again, associations between any cues that are present in the environment and the feelings and behaviors of wanting and taking the drug. The result is the buildup of an extraordinarily strong craving and habit, which are triggered whenever cues that have been present during past drug taking are present.

It has often been observed that drug addicts gradually lose their "liking" of the drug (experience less pleasure) over time, even while their "wanting" of the drug increases (Kelley & Berridge, 2002). The loss in liking occurs, presumably, because of drug-induced changes in the brain that reduce the endorphin-mediated pleasure response. However, because the dopamine response is not reduced, the learned drug craving and habit continues to grow stronger with each dose (Kelley & Berridge, 2002; Nestler & Malenka, 2004). The craving itself, rather than any expected pleasure, becomes the main reason for taking the drug. Drug taking becomes a compulsion rather than something that one freely chooses to do for pleasure. (Another reason why drug addicts continue to take drugs, having to do with conditioned counteractive responses, is discussed in Chapter 4.)

A Brain-Based Theory of Compulsive Gambling

In North America somewhere between 1 and 2 percent of adults suffer from a compulsive, pathological drive to gamble (Grant & others, 2004), a drive that persists even though it may leave the person and his or her family in financial ruins. Compulsive gambling is in some ways similar to drug addiction (Holden, 2001).



Internet gambling Whether done on the Internet or at an actual casino, gambling can become compulsive. Because wins are essentially random and unpredictable, each win may result in a new burst of dopamine release, which helps to create the compulsion.



Wanting without liking After repeated use, a drug such as cocaine or heroine continues to promote dopamine release in the brain, and thus to reinforce "wanting," but the drug may no longer promote endorphin release, so the "liking" response no longer occurs.

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Gamblers claim to feel a euphoric high when they are playing their game and winning, and to experience withdrawal symptoms—such as sweating, restlessness, and sleeplessness—when they try to abstain. Every cue in the environment that has been previously associated with gambling elicits in them a strong, often irresistible urge to gamble. Our understanding of the brain's reward mechanisms gives us some clues about the origins of this compulsion.

Brain imaging studies (using the fMRI technique, discussed in Chapter 5) with healthy human subjects have revealed that games of chance, with monetary rewards, are powerful activators of the nucleus accumbens and other structures known to be part of the brain's reward system (Breiter & others, 2001; Knutsen & others, 2001). Because the payoff is never predictable, every instance of payoff results in a new burst of dopamine release in the nucleus accumbens, no matter how many times the person plays. Thus, gambling, like drug taking, overrides the brain's dopamine-conserving mechanism—the mechanism that shuts off the dopamine response when reward is predictable. Consciously, the person may know that the game pays off in a way that is unpredictable and uninfluenced by anything that he or she does, but the brain's primitive reward system nevertheless behaves as if it is constantly trying to learn how to predict and produce the reward. The repeated reinforcement, by dopamine, of associations between payoffs and the cues and behaviors that precede each payoff results in the buildup of an abnormally strong habit.

Drives as States of the Brain

The reward mechanisms just discussed are involved in all sorts of motivated behaviors. Their destruction produces an animal that fails to seek out or work for the objects and conditions that it needs for survival and reproduction. But what accounts for the differences among different drives? What makes an animal thirsty, or hungry, or sexually motivated, or desirous of sleep?

According to the *central-state theory of drives*, which is accepted by nearly all neuroscientists today, different drives correspond to neural activity in different sets of neurons in the brain. A set of neurons in which activity constitutes a drive is called a *central drive system*. Although the central drive systems for different drives must be at least partly different from one another, they may have overlapping components. For example, because hunger and sex are different drives, the neural circuits for them cannot be identical. However, those circuits may share components that produce behavioral effects that are common to both drives, such as increased alertness and general level of motor activity.

What characteristics must a set of neurons have to serve as a central drive system? First, it must receive and integrate the various signals that can raise or lower the drive state. For hunger, these signals include chemicals in the blood, the presence or absence of food in the stomach, and the sight and smell of food in the environment. Second, a central drive system must act on all the neural processes that would be involved in carrying out the motivated behavior. It must direct perceptual mechanisms toward stimuli related to the goal, cognitive mechanisms toward working out strategies to achieve the goal, and motor mechanisms toward producing the appropriate movements. Look back at Figure 5.21 (p. 157), which depicts a hierarchical model of the control of action, with mechanisms involved in motivation and planning at the top. The central drive systems are part of that top level of the hierarchy. To affect behavior (for example, to cause a hungry person to cross a room for some peanuts), they must influence the activity of motor systems at lower levels of the hierarchy.

Researchers have sound reasons to believe that the hypothalamus is the hub of many central drive systems. Anatomically, this brain structure is ideally located to play such a role (see Figure 6.4). It is centered at the base of the brain, just above the brainstem, and is strongly interconnected with higher areas of the brain. It also has direct connections to nerves that carry input from, and autonomic motor

11 — In theory, what characteristics must a set of neurons have to function as a central drive system? What characteristics of the hypothalamus seem to suit it to be a hub

of such systems?



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output to, the body's internal organs. It has many capillaries and is more sensitive to hormones and other substances carried by the blood than are other brain areas. Finally, through its connections to the pituitary gland, it controls the release of many hormones (as described in Chapter 5). Thus, the hypothalamus has all the inputs and outputs that central drive systems would be expected to have. And, as you will soon see, small disruptions in particular parts of the hypothalamus can have dramatic effects on an animal's drives.



IFIGURE 6.4 Location of the hypothalamus The hypothalamus is ideally situated to serve as a hub for central drive systems. It has strong connections to the brainstem below, the limbic system and cerebral cortex above, and the endocrine system (by way of its tie to the pituitary gland).

SECTION REVIEW

Drives are reversible internal conditions that orient individuals toward specific types of goals. Drives Reward Mechanisms in the Brain Drives (motivational states) complement and Reward mechanisms mediate three interrelated components of reward interact with incentives (the objects of wanting, liking, and reinforcement. drives) • The medial forebrain bundle and nucleus accumbens contain essential Regulatory drives (e.g., hunger) promote homeostasis, while nonregulatory drives pathways of the brain's reward system. Animals will work for electrical stimulation in these areas. (e.g., sex) serve other purposes. The release of dopamine into the nucleus accumbens is associated with wanting; the release of endorphins into this area is associated with liking. Mammalian drives can be classified by function into regulatory, safety, reproductive, The release of dopamine into the nucleus accumbens is also crucial to social, and educative categories. Humans reinforcement; it promotes learning about how to predict and obtain a given also exhibit drives toward art, music, and literature reward. Different drives involve different central drive • Addictive drugs and the random reward schedules used in gambling may systems. The hypothalamus is a hub of such override the mechanism that shuts down dopamine release, resulting in the overlearning of cues and habits. systems

Hunger: An Example of a Regulatory Drive

It is no accident that eating is one of life's great pleasures. Throughout our evolutionary history, the difficulty of finding adequate food was one of the major barriers, if not the major barrier, to survival. As a result, natural selection built into us (and other animals) powerful, robust hunger mechanisms that lead us to search for food, to eat when food is available, and to experience pleasure when we eat. Natural selection also built into us satiety mechanisms, which tend to keep us from overeating and becoming obese. But the satiety mechanisms are not as robust as the hunger mechanisms. In our evolutionary history, food scarcity was a much bigger problem than overabundance. Far more people died of starvation than of obesity (and this is still true in many parts of the world today). Things are different, however, for those of us who live in postindustrial parts of today's world. For us (and especially for those of us in North America), obesity has become a major health problem. In what follows, we will examine some of the mechanisms that control appetite and then look at the modern problem of obesity.

Neural and Hormonal Control of Appetite

The purpose of hunger and satiety is to regulate the amount of food materials in the body at an appropriate level for survival and well-being. Any regulatory system,

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12 What is meant by feedback control, and how does the arcuate nucleus of the hypothalamus serve as a control center for appetite?

whether it is part of a human-made machine or part of an animal, makes use of *feedback control*. The substance or quality being regulated feeds back upon the control ling device and inhibits the production of more of that substance or quality when an appropriate level is reached. A home thermostat, which controls the operation of the heating system, is a good example. The thermostat is sensitive to temperature. When the temperature is low, a switch closes in the thermostat, which turns on the furnace, which provides heat. When the temperature rises above the set level, the switch opens and the furnace turns off. Thus heat, produced by the furnace, the shack onto the thermostat to shut off the source of heat when enough of it is present. The mammalian brain regulates food intake in a manner that is a bit like the op-

eration of a home thermostat, but far more complicated. Sets of neurons in the brain's hypothalamus raise or lower the animal's drive to eat, and these neurons are themselves regulated by the body's deficit or surfeit of food materials. We might think of these neurons as the brain's "food-o-stat." When food materials are relatively low in the body, the food-o-stat cranks appetite up, which motivates the animal to consume more food. When food materials are plentiful in the body, various indicators of that plenitude feed back upon the food-o-stat and turn appetite off, or at least down a bit.

A Nucleus in the Hypothalamus Serves as an Appetite-Control Center

The neurons that comprise the food-o-stat exist in several closely interconnected portions of the hypothalamus, but are most concentrated in the *arcuite* [*arc*.yout] *nucleus*, which lies in the center of the lowest portion of the hypothalamus, very close to the pituitary gland (Korner & Leibel, 2003). This tiny brain area has been described as the "master center" of appetite control and weight regulation (Marx, 2003). It contains two classes of neurons that have opposite effects on appetite. One class, the appetite-stimulating neurons, connect to various parts of the brain and promote all the effects that are associated with increased hunger, including craving for food, increased attention to food-related cues, increased exploration in search of food, and heightened enjoyment of the taste of food. The other class, the appetite-suppressing neurons, have effects on various parts of the brain that are opposite to those of the appetite-stimulating neurons.

Both of these classes of arcuate neurons exert their effects on other brain areas through the release of slow-acting neurotransmitters. Unlike fast transmitters, slow transmitters (discussed in Chapter 5) have the capacity to alter neural activity for long periods of time—in this case for periods ranging from minutes to several hours. One of the neurotransmitters released by appetite-stimulating neurons is *neuropeptide Y*, which is the most potent appetite stimulator yet discovered. When injected into any of various regions in the hypothalamus, this chemical causes a previously sated animal to eat voraciously (Stanley & Gillard, 1994). The neurons of the arcuate nucleus are themselves acted upon by many different inputs that, in one way or another, reflect the need or lack of need for food.

Many Internal Signals Contribute to Short-Term Regulation of Appetite

Eating a large meal produces a number of physiological changes in the body. Among these are a slightly elevated body temperature (resulting from a heightened rate of metabolism), an increased blood level of glucose (a simple sugar molecule derived from the breakdown of carbohydrate foods), distention of the stomach and intestines (resulting from food inside those structures), and the release of certain hormones produced by endocrine cells in the stomach and intestines. There is evidence that all these changes can act either directly or indirectly on neurons in the arcuate nucleus and nearby areas of the hypothalamus to activate hungersuppressing neurons and inhibit hunger-stimulating neurons (Havel & others, 2000; Korner & Leibel, 2003). When all these effects are operating properly, the result is a decline in appetite for several hours following ingestion of a meal.



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"What if these guys in white coats who bring us food are, like, studying us and we're part of some kind of big experiment?"

One appetite-suppressing hormone that has received much recent attention is >>>> peptide $YY_{3:36}$ (abbreviated **PYY**), which is produced by special endocrine cells in the large intestine. Food entering the intestines after a meal stimulates secretion of PYY into the bloodstream. In humans, blood levels of the hormone begin to increase 15 minutes after a meal is eaten, peak at about 60 minutes, and remain elevated for as long as 6 hours after a large meal (Batterham & others, 2003). Research with rodents shows that one of the target tissues of PYY is the arcuate nucleus, where the hormone excites appetite-suppressing neurons and inhibits appetite-stimulating neurons (Marx, 2003).

In both rats and humans, injection of extra PYY into the bloodstream reduces total food consumed over the next several hours. In one double-blind experiment with humans, PYY injection reduced the amount of food eaten at a buffet luncheon, by both lean and obese human volunteers, by an average of about 30 percent and also reduced reported level of appetite in both groups (Batterham & others, 2003). The same researchers also found that lean subjects had higher baseline levels of naturally produced PYY than did obese subjects, and exhibited a much greater increase in PYY following a meal (see Figure 6.5). This result suggests that insufficient PYY production may be a contributing cause of obesity. As you can well imagine, pharmaceutical companies are currently investigating the possibility that PYY, or some modified form of it, might be developed and marketed as a weight-control drug.



I FIGURE 6.5 Blood levels of the PYY in obsee and lean human subjects Blood levels of the hormone PY were found to be lower in obses subjects than in lean subjects. After eating a meal, lean subjects showed a much larger increase in PYY than did the obses subjects. Researchers believe that this difference may be a cause of obesity, as PYY acts on the brain to suppress appetite. (From Batterham & others, 2003.)

Hat is the evidence that the hormone PYY helps reduce appetite after a meal and that underproduction of PYY may contribute to obesity?



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Leptin Contributes to the Long-Term Control of Appetite and Body Weight

How does the hormone leptin contribute to weight regulation, and why isn't leptin a good anti-obesity drug?



IFIGURE 6.6 Lacking leptin The mouse on the left, which lacks the gene needed to synthesize the weight-regulating hormone leptin, outweighs two genetically normal mice. Without leptin, the animal overeats and becomes obese.

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How do conditioned stimuli and the availability of many foods, with different flavors, contribute to appetite and obesity? In the short term, eating provides an immediate supply of building blocks (such as amino acids) and energy molecules (such as glucose) needed to grow, repair, and fuel the body. For the long term, eating more than is immediately needed adds to the amount of fat that is stored in special fat cells in various tissues of the body. Fat stores provide an extra source of energy that the body can call upon when food is not available in the environment. Too much fat, however, impedes movement and puts stress on all the body's organs. Not surprisingly, the hunger mechanism, when it is working optimally, is sensitive not just to the short-term indicators of a fact.

amount of food recently eaten but also to the amount of fat stored in the body.

Fat cells in mice, humans, and other mammals secrete a hormone, called *leptin*, at a rate that is directly proportional to the amount of fat that is in the cells (Woods & others, 2000). Leptin is taken up into the brain and acts on neurons in the arcuate nucleus and other parts of the hypothalamus to reduce appetite. Animals that lack either the gene needed to produce leptin or the gene needed to produce the receptor sites for leptin in the hypothalamus become extraordinarily obese (see Figure 6.6) (Friedman, 1997). Some people—though very few—lack the gene needed to produce leptin. Such individuals are extremely obese, but they reduce their eating, lose weight rapidly, and keep it off, when given daily injections of leptin (Farooqi & others, 2002).

When the hunger-suppressing effect of leptin was discovered, in the 1990s, there was much excitement about the possibility that injections of this hormone could help many people lose weight. Subsequent research, however, proved otherwise. The great majority of overweight people are not lacking in leptin. Their fat cells constantly produce very high levels of the hormone. Research shows that hunger is reduced by increased leptin up to a certain level, but most overweight people already have blood concentrations of leptin well above that level, and additional leptin has no effect (Marx, 2003). Other research suggests that many obese people feel chronically hungry not because they lack leptin but because their brains are relatively insensitive to the hormone (Friedman, 2003). A drug that helped restore leptin sensitivity might help them lose weight, but so far no such drug is in sight.

Roles of Sensory Stimuli in Control of Appetite

As you well know, hunger is provoked not just by events inside us but also by sensory stimuli in the environment. In the natural environment, food is not always available. That was especially true in the environment of our evolutionary ancestors. Evolution has led us and other animals to be opportunists; we are more likely to experience hunger when food is available than when it isn't.

Through classical conditioning (discussed in Chapter 4), any cues that have been previously associated with the opportunity to eat—such as the sight or smell of good food, the sound of a dinner bell, or the sight of a clock showing it is dinner time—can bring on a sudden surge of appetite. Such conditioning is reflected not just in reports of increased hunger but also in the occurrence of reflexive physiological responses, such as the secretion of saliva and digestive juices, that help to prepare the body for food and that add further to the sense of hunger (Woods & others, 2000).

The taste of food also plays a major role in appetite. People and laboratory animals who eat one type of food until they are satiated experience renewed appetite when a different food, with a different taste, is placed before them. This phenom-

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enon is referred to as sensory-specific satiety, and many experiments show that it is mediated primarily by the sense of taste (Raynor & Epstein, 2001). When people eat one food at a meal, their rating of the taste pleasantness of that food declines relative to their rating of the taste pleasantness of other foods. This effect begins immediately after eating the food and lasts typically for several hours. Experiments with animals show that the sight and smell of a new food can result in renewed activity in appetite-stimulating neurons in the hypothalamus after the animal has been sated on a different food (Rolls & others, 1986). Laboratory animals that can regularly choose from a variety of different-tasting foods eat more, and become fatter, relative to animals that have only one food choice, even if the nutritional content of the different foods is identical (Raynor & Epstein, 2001). People, too, eat more when there are more food choices (Raynor & Epstein, 2001).

Problems of Obesity

Human evolution occurred almost entirely in environments where food choices were far fewer, and far lower in fat and

sugar, than are the foods available to people in modern cultures. Natural selection built into us excellent mechanisms to defend against weight loss in times of scarcity but rather poor mechanisms to defend against obesity in times of plenty. Obesity is a cultural disease of our time and place.

To assess obesity, the World Health Organization uses a measure called the *body* mass index, or **BMI**, defined as body weight in kilograms divided by the square of the person's height in meters. A BMI of 25 or more is considered *overweight*, and one of 30 or more is considered *obese*. Thus a person who is 1.7 meters (5 feet 7 inches) tall and weighs 73 kilograms (161 pounds) is deemed overweight (BMI = $73/1.7^2 = 25.3$), and a person of that same height who weighs 88 kilograms (194 pounds) is considered obese (BMI = $88/1.7^2 = 30.4$).

By these criteria, according to a survey taken in 1999–2000, 65 percent of adults in the United States are overweight and 31 percent are obese (Hill & others, 2003). By comparison, in a similar survey just 10 years earlier, 56 percent of adults were overweight and 23 percent were obese (Hill & others, 2003). Obesity is also rising rapidly in many other parts of the world. As obesity rises, so does the rate of diseases that are secondary to it, including Type 2 diabetes, coronary heart disease, stroke, and certain types of cancers (Marx, 2003). In the United States, obesity is rapidly overtaking smoking as the major underlying cause of death (Marx, 2003). The immediate causes of this obesity epidemic are clear: People eat more and exercise less than they used to.

Effects of Genes and Culture on Body Weight

Within the United States or any other Western culture, the determination of who >>> does or does not become obese depends very much on genes and relatively little on the specific home environment (Barsh & others, 2000). The weights of adopted children correlate much more strongly with the weights of their biological parents than with those of their adoptive parents; identical twins usually have very similar weights even if raised in different homes; and pairs of biological siblings raised in different homes are, on average, nearly as similar to each other in weight as are pairs raised in the same home (Grilo & Pogue-Geile, 1991; Stunkard & others, 1986). This does not mean that body weight is little influenced by the environment. It simply means that the environmental conditions that promote obesity are fairly constant within Western cultures, so differences in weight have mostly to do with genetic differences in how individuals respond to those conditions.



Overriding the satiety mechanism People on a cruise typically gain weight, partly because of the large variety of food that is served. These people may become satiated with one type of food but still have an appetite for others.

> _______ 16 What is the evidence that within a culture, differences in body weight result mostly from differences in genes, but across cultures, environment plays a large role?

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PART 3 PHYSIOLOGICAL MECHANISMS OF BEHAVIOR

FIGURE 6.7 Effect of culture on body weight If the only Pima Indians you ever saw were those living in Mexico and following traditional Pima practices (like these photographed at a holy festival), you might conclude that they are genetically predisposed to be slender. Obesity is almost nonexistent among the Mexican Pima, However, their close relatives living across the border in the United States are often obese. That difference lies not in genes but in the choices of foods available.

17 On the basis of the reports of successful dieters and the advice of appetite researchers, what can people do to maintain a lower weight?



Across cultures, environmental differences can have a large effect on body weight. As one example, obesity is very common among Pima Indians living in Arizona but is essentially absent among their genetic relatives living in Mexico (see Figure 6.7). The Mexican Pimas subsist mainly on grains and vegetables; their culture does not include the high-calorie foods available to the American Pimas (Gibbs, 1996). The genes that promote obesity in our culture apparently do so by increasing the person's attraction to high-calorie foods, by decreasing one or another of the feedback effects that high food intake or fat level has on the hunger mechanisms in the hypothalamus, and by decreasing the body's ability to burn up excess calories quickly (Barsh & others, 2000; Friedman, 2003). Where high-calorie foods are unavailable, the same genes generally don't lead to obesity.

Problems of Dieting

Weight gained is often very difficult to lose. Decreased food intake not only activates the hunger mechanisms in the brain but can also produce a decline in basal metabolism (the rate at which calories are burned up while the individual is at rest), so food is converted more efficiently to fat (Keesey & Corbett, 1984; Leibel & others, 1995). In one extreme case, a woman managed to reduce her weight from 312 pounds to a still-obese but much healthier 192 pounds through diet. For at least 18 months after that she maintained her new weight, without losing any more, by eating a total of 1000 to 1200 calories a day-less than half of what most women would have to eat to maintain that weight (Rodin & others, 1989).

<< Despite the odds, some people do lose significant amounts of weight and avoid regaining it. A recent study of approximately 3,000 highly successful dieters, who had lost an average of over 60 pounds per person and had kept it off for an average of 5 years at the time of the study, revealed that they succeeded primarily by avoiding high-fat foods and by greatly increasing their exercise (Butler, 2004; Wing & Hill, 2004). Regular exercise not only burns up calories immediately but also builds muscle, which, even when resting, burns up calories at a higher rate than do other body tissues (Van Itallie & Kissileff, 1990).

Researchers who study appetite and metabolism often have some sensible advice for people who want to lose weight. Here is my summary of what I have gleaned from their work:

• Don't try to lose weight rapidly. Don't go on a diet that you can't stay on for the rest of your life. Weight rapidly lost through starvation dieting is rapidly regained when the diet ends.

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- Instead of reducing food intake to a level that leaves you hungry, shift the kind of food you eat, away from high-calorie fats and sweets and toward gut-filling but low-calorie vegetables, fruits, and complex carbohydrates (whole-grain breads and cereals). Contrary to the claims of diet books that promote high-fat, low-carbohydrate diets, people who keep weight off for long periods usually do so by sharply reducing their fat intake (Butler, 2004; Wing & Hill, 2004).
- Make the sensory-specific satiety effect work for you instead of against you. Provide yourself, over time, with a luscious variety of vegetables and fruits and very few choices of high-calorie meats and dairy products; and omit the pastries, soda pop, potato chips, and French fries entirely
- Eat each meal slowly, so as to enjoy it and to provide time for the food's satietyproducing effects to develop. If you still feel hungry after eating what you initially planned to eat, wait at least 15 minutes before deciding whether or not to eat more. By that time you may no longer feel hungry. It takes about 15 minutes for PYY and other satiety-producing consequences of a meal to begin to exert their effects on the brain's arcuate nucleus.
- · If you have a sedentary job or are a student, develop some pleasurable hobbies that involve muscle-building exercise for at least a few hours a week. (I for one can't stand jogging and get quickly bored with weight lifting, but I love bicycling, kayaking, and cross-country skiing.) And when you need to get from one place to another, use your muscles rather than an automobile or an elevator to convey you whenever you can.

Through such changes in habits, many people can lose a significant amount of weight and keep it off for a lifetime, without feeling deprived at all.

SECTION REVIEW

neurons

Hunger is a regulatory drive controlled by neural, hormonal, and sensory factors.

Control Mechanisms of Hunger

Obesity

- The arcuate nucleus of the hypothalamus is a feedback-based appetite control center, with both appetite-stimulating and appetite-suppressing
- Eating a large meal causes physiological changes, including the release of PYY, that influence the arcuate nucleus and nearby areas to reduce hunger.
- Leptin, a hormone produced by fat cells, helps to regulate body weight by acting on the hypothalamus to reduce appetite.
- Sensory stimuli also affect appetite, as illustrated by sensory-specific satiety and by the appetite-boosting power of learned cues that signal the availability of food.
- Within a culture, genetic differences are the primary determinant of who becomes obese, but across cultures, environmental differences play a substantial role.
- Decreasing food intake activates hunger mechanisms in the brain and can reduce basal metabolism, making weight loss harder
- Certain techniques help at least some people lose substantial amounts of weight and keep it off.

Sex: An Example of a Non-Regulatory Drive

Just as hunger is the most thoroughly studied regulatory drive, sex is the most thoroughly studied non-regulatory drive. As with hunger, most research on the physiological basis of the sex drive has been conducted with laboratory animals.

There are limits, of course, in the degree to which we can understand hunger, sex, or any drive in humans by studying it in other animals. Human culture, intellect, sensibility, and capacity for conscious self-control affect all human behavior in ways that cannot be studied in laboratory animals. People don't just eat; they dine,

18 Why is caution advised in extrapolating from laboratory animals to humans in the study of drives, especially the sex drive?

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which connotes all sorts of social, cognitive, and aesthetic influences. And people don't just copulate; they fall in love, compose romantic sonnets, gaze into each other's eyes over candlelit dinners, swear by the moon to be faithful, have affairs, suffer guilt, and engage in long, intimate discussions with their beloved. Keep in mind, as you read on, that our concern here is the basic physiological mechanisms that we humans share, more or less, with other mammals, not the whole range of issues concerning human sexuality. (Sex is discussed more from a social and cultural perspective in Chapter 12.)

Even when dealing with the copulatory act itself, humans differ quite sharply from rats and other laboratory animals. Among non-human mammals, including most other primates, copulation occurs in a stereotyped way, with one set of pos-

tures and movements for the female and a different set for the male (see Figure 6.8). Among humans, by contrast, the variety of ways to copulate is limited only by imagination. As you will discover when you read further, differences between humans and other species exist also in the hormonal regulation of the sexual drive, especially in females.

Hormonal Influences on Sexual Drive

Sex hormones affect sexual drive in humans and other mammals through their influences on the brain. Such influences are of two types: differentiating and activating. *Differentiating effects* occur before or (in some species) immediately after birth and cause the brain to develop in a male or female direction. They cause the *differences* between males and females in sexual drive and orientation. *Activating effects* occur later, around the time of puberty and after, when hormones work on the already-differentiated brain structures to prime, or *activate*, sexual drive. Neurons that are involved in sexual drive have special binding sites on them for sex hormones, and the hormones act there to enable the neurons to respond to sexually arousing stimuli in the environment. Here we shall look first at activating effects, and then at differentiating effects.

Hormones and the Onset of Sexual Drive Near Puberty

In both humans and other mammals, the production of sex hormones greatly increases at the onset of the developmental stage called *puberty*. In human males, increased *testosterone*, produced by the testes, stimulates such changes as beard growth and the male pattern of muscle development; in human females, increased estrogen, produced by the ovaries, stimulates such changes as breast growth. Less well known is the fact that, in humans as well as other animals, and in both sexes, the adrenal glands also produce sex hormones. In humans the primary adrenal sex hormone is *dehydroepiandrosterone*, mercifully abbreviated *DHEA*. This substance is commonly classed as a "male hormone"—that is, as an *androgen*—because it has effects in men similar to those of testosterone. However, it is produced as much in women as in men, and there is reason to believe that it is the primary hormone responsible for the onset of sexual feelings and attractions in young humans of both sexes (Herdt & McClintock, 2000; McClintock & Herdt, 1996).

Boys' and girls' adrenals begin to secrete DHEA at about 6 years of age, and the amount secreted rises continuously until the mid-teenage years, when it stabilizes at adult levels (see Figure 6.9). Research in both Western and non-Western cultures suggests that most people, of both sexes, recall their earliest clear feelings of sexual attraction to another person as occurring at about 10 years of age, well before the prominent physical changes brought on by testosterone or estrogen (Herdt & McClintock, 2000). If testosterone and estrogen brought on this developmental effect, then we would expect girls to experience sexual attraction at an earlier age than boys, since the rise in estrogen precedes the rise in testosterone by about 2 years, on average. But repeated studies find no difference between the sexes in the age of earliest sexual attraction, which is consistent with the idea that this feeling is brought on by DHEA, which increases at the same rate in the two sexes. DHEA,



IFIGURE 6.8 | Copulation in rats Rats, like most other non-human mammals, have a stereotyped (unvarying) pattern of copulation, with clearly different postures for the female and the male.

19 What roles are played by

What roles are played by testosterone, estrogen, and DHEA in the developmental changes around the time of puberty?

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I FIGURE 6.9 1 A sex hormone that increases at the same rate in girls and boys Shown here are the average blood levels of the adrenal androgen DHEA (dehydroepiandrosterone) in boys and girls as a function of age. This hormone is believed to activate the brain in a way that enables the young person, whether male or female and regardless of sexual orientation, to feel sexually attracted toward others, beginning at about age 10.

like other sex hormones, passes into the brain and alters activity in certain neural centers there, especially in the hypothalamus.

Activating Effects of Hormones on the Sex Drive in Male Mammals

In male mammals, including humans, the most crucial hormone for the mainte->> nance of the sexual drive after puberty is not DHEA but testosterone. Castration (removal of the testes, which produce the main supply of testosterone) causes a marked decline in the sex drive—not all at once, but gradually (Feder, 1984). It takes days to occur in rats, weeks in dogs, sometimes months in monkeys. But the injection of testosterone into the bloodstream of castrated animals fully restores their drive.

The sex drive can also be restored in castrated male animals by implanting a tiny crystal of testosterone into an area of the hypothalamus called the *medial pre-optic area* (see Figure 6.10 for its location) (Davidson, 1980). Neurons in the medial preoptic area contain many receptor sites for testosterone, and small lesions there abolish sexual behavior in male rats (Meisel & Sachs, 1994). Apparently, the medial preoptic area of the hypothalamus is a crucial part of the central drive system for sex in male rats other male animals that have been studied, and testosterone acts there in a rather prolonged way to enable neural activity to occur and sustain the drive.

Testosterone is also crucial for maintaining the sex drive in human males. Men castrated in an accident or for medical reasons almost always experience a decline (though often not a complete loss) in sex drive and behavior, and testosterone injections restore their drive, usually fully (Money & Ehrhardt, 1972). In other studies, testosterone injections administered to non-castrated men whose testes were producing abnormally low amounts of the hormone sharply increased their sexual behavior (Davidson & Myers, 1988; Davidson & others, 1979). This effect was more on drive than on sexual capability. Men with abnormally low levels of testosterone were usually capable of the mechanics of sexual behavior—including erection and ejaculation—but had little desire for it until injected with testosterone. The subjects in this research did not know when they were receiving testosterone and when they were not, so the results must have been due to the effects of the hormone and not to their expectations.

> _____20 What is some evidence that testosterone is needed to maintain the male's sex drive and that, at least in some species, it does so by direct action in the hypothalamus?



IFIGURE 6.10 Hypothalamic areas where hormones activate sexual behavior In rats, testosterone promotes male sexual behavior by activating neurons in the medial preoptic area of the hypothalamus, and estrogen promotes female sexual behavior by activating neurons in the ventromedial area of the hypothalamus.

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PART 3 PHYSIOLOGICAL MECHANISMS OF BEHAVIOR

Activating Effects of Hormones on the Sex Drive in Female Mammals

What evidence indicates that ovarian hormones act directly on the brain to activate the sexual drive in female rats? How do female non-human primates and humans differ from one another, and from rats, in regard to the role of hormones in regulating sexual drive? After puberty, a female's ovaries begin to secrete estrogen and progesterone in a cyclic pattern over time, producing the cycle of physiological changes referred to as the *menstrual cycle* in humans and the *estrous cycle* in other mammals. In both humans and non-humans, this cycle controls ovulation (the release of one or more eggs so that pregnancy can occur). In most mammals, it also tightly controls the sex drive—which ranges from very strong at the time of ovulation to nonexistent at other times. Removal of the ovaries completely abolishes sexual behavior in most non-human female mammals, and injection of hormones can fully restore it. For some species an injection of estrogen alone is most effective, and for others (including rats) a sequence of estrogen followed 2 or 3 days later by progesterone (another female hormone) is most effective.

At least in rats, the *ventromedial area* of the hypothalamus (see Figure 6.10) plays a role in sexual behavior in the female analogous to that of the preoptic area in the male. Insertion of small amounts of estrogen and progesterone directly into this area brings on sexual behavior in rats whose ovaries have been removed, and lesions in this area abolish sexual behavior in otherwise intact females (Mani & others, 1994; Pleim & Barfield, 1988; Schwartz-Giblin & others, 1989). Apparently the cyclic variation in ovarian hormones acts on the ventromedial area to cause the cyclic waxing and waning of sexual drive.

Female monkeys and apes depend less on ovarian hormones for sexual behavior than do most other female mammals. Most primate females can, and sometimes will, copulate with a sexually active male at any time in their hormone cycle, though they are more likely to seek out and initiate sexual contact with a male at the time in their cycle when they are ovulating and the level of ovarian hormones is high (Wallen, 1990). In at least some species of primates, including rhesus monkeys, adrenal androgens apparently help maintain sexual drive and behavior during times in the estrous cycle when ovarian hormones are low (Wallen, 1990).

Human females show still greater liberation of sexual behavior from cyclic hormonal control than do other primates. Women can experience a high or low sex drive at any time in their hormone cycle. In fact, debate exists as to whether women's sex drive is affected in a consistent way at all by the hormone cycle. Some researchers have found that, on average, women report a higher sexual drive (Adams & others, 1978), and manifest greater attraction to men with highly masculine features (Gangestad & others, 2004), around the time of ovulation than they do at other times in their hormonal cycle. But these effects are subtle compared to effects of the hormone cycle on sexual drive in the females of most other species. In women, ovarian hormones apparently play a relatively small role in regulation of the sex drive, and adrenal hormones play a larger role. The adrenals of adult women produce both DHEA and testosterone. In clinical studies, women whose ovaries have been removed generally do not report a decline in sexual drive, but women whose adrenals have been removed generally do report such a decline, and treatment with testosterone reliably increases the sex drive reported by women with low sex drive (Bancroft, 1978; Guay, 2001; Sherwin & Gelfand, 1987).

Brain-Differentiating Effects of the Early Presence or Absence of Testosterone

As noted in Chapter 3, the only initial difference between the two sexes, in all mammals, is that females have two X chromosomes and males have a small Y chromosome in place of the second X. A specific gene on the Y chromosome causes the growth of testes (the male gonads) from structures that would otherwise develop into ovaries (the female gonads) (Page & others, 1987). Before birth the testes begin to produce testosterone, which acts on the brain and other bodily structures to steer development in the male direction. The rudimentary genitals of the fetus develop into male structures (including the penis and scrotum) if testosterone is present,

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What are some effects of the presence or absence of testosterone before birth on development of the genitals, the brain, and behavior? CHAPTER 6
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and they develop into female structures (including the clitoris and vagina) if testosterone is absent. Early testosterone also promotes the development of brain pathways involved in the male sex drive and to inhibit the development of neural systems involved in the female sex drive (Gorski, 1996; Simerly, 2002). For an example of one such effect, see Figure 6.11.

In order to produce these brain-differentiating effects, testosterone must act within a critical period in the animal's development. In rats, this period runs from a few days before birth to a day or so after birth. In many other species, the critical period

ends before birth. The critical period for testosterone's effect on the brain is later than that for its effect on the genitals. Thus, manipulation of hormones at the appropriate time can produce animals that have the genitals of one sex but the brain structures and behavior of the other sex (Feder, 1984; Ward, 1992).

Perhaps you are wondering why a male hormone, not a female hormone, plays the key role in early sexual differentiation in mammals. The answer is that the female hormones (progesterone and estrogen) are produced by pregnant females at high levels and get into the tissues of all fetuses, of both sexes. If female hormones promoted growth of female structures during fetal development, all mammalian infants would be born looking like females. In birds and reptiles—which develop in eggs outside the mother's body—early sexual differentiation is determined by the presence or absence of estrogen, not testosterone (Adkins-Regan, 1981).

Brain Development and Sexual Orientation in Humans

It is quite possible that the brain mechanisms for male and female sexual motivation are less different from one another in humans than in most other mammalian species. As already noted, in people, unlike most other mammals, androgens may be the primary sex-drive hormones in both sexes. Moreover, men and women show similar patterns of physiological changes during sexual arousal and orgasm (Masters & others, 1992) and describe the subjective feelings they have during sex in similar ways (Vance & Wagner, 1976). But one general difference between men and women in sex drive is in the object of the drive: Most men are attracted to women, and most women are attracted to men. Might this difference result from a structural difference in the brain? If so, what creates that brain difference? The answers to these questions are not yet certain, but some interesting ideas about them have emerged from studies of people who are sexually attracted to members of their own sex rather than the opposite sex.

Using various criteria and survey methods, researchers estimate that 2 to 5 percent of men, and 1 to 2 percent of women, are exclusively homosexual in orientation (Rahman & Wilson, 2003). An apparently larger percentage, even harder to estimate precisely, are bisexual (attracted to members of both sexes), and bisexuality appears to be more common in women than in men (Rahman & Wilson, 2003).

Lack of Evidence for Social Learning Explanations of Sexual Orientation

In the past many psychologists argued that sexual orientation derives primarily from social learning, and a few still argue that today. But the existence of a signifcant homosexual population in every culture—including cultures where homosexuality is severely punished—presents a challenge to that hypothesis (LeVay & Hamer, 1994). If sexual orientation is learned through social experiences, then why would anyone developing in a society that glorifies heterosexuality and strongly discourages homosexuality grow up as a homosexual? Over the years psychologists have suggested many possible answers to that question aimed at preserving the learning hypothesis, but none has so far withstood the test of research.



in the rat's hypothalamus Shown here is a cross section of the medial preoptic area of the hypothalamus of a male rat (left) and a female rat (right). The dark spots represent cell bodies of neurons, and the dense cluster of them represents the sexual dimorphic nucleus (marked by red circles). The nucleus receives its name from the fact that it is clearly different in the two sexes: it is about 5 times larger, on average, in the male than in the female (Gorski & others, 1980). The difference is due entirely to the presence or absence of testosterone early in the rat's development. Early treatment with testosterone in females and early deprivation of testosterone in males can reverse this sex difference. The nucleus is believed to be part of the neural circuitry controlling male sexual drive and behavior (DeJonge & others, 1989)

>> 23 What evidence suggests that in humans

sexual orientation (a) is not a result of environmental variables that in the past were hypothesized as causal, (b) appears early in development, (c) correlates with certain anatomical brain differences, (d) is influenced by differences in genes, and (e) is influenced by the prenatal environment, including (in males) an effect on that environment induced by one's older brothers?

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Vinslow Townson / AP Photo

Gay marriage The idea that homosexual couples should be allowed to enjoy the benefits and take on the responsibilities of marriage is still politically controversial. The fact that the controversy is even occurring, however, illustrates that the culture has taken strides toward accepting homosexuality as a normal human condition.

Interviews and surveys of thousands of homosexuals and heterosexuals have found no consistent relationship between sexual orientation and the kinds of experiences in childhood or adolescence that have at times been theorized to cause homosexuality (Bell & others, 1981; Dawood & others, 2000; Rahman & Wilson, 2003). Such studies reveal no evidence that style of parenting, absence of the father or mother, early seduction or rape by someone of the same or opposite sex, or degree of opportunity for one or another type of sexual experience in adolescence contributes significantly to the development of sexual orientation. Such studies do show, however, that whatever its cause or causes, sexual orientation is usually a deeply ingrained and early-emerging aspect of one's being. Homosexuals and heterosexuals alike generally report that their sexual orientation was present, though not necessarily understood or accepted, in their childhood thoughts and fantasies, typically by the age of about 10 or 11 (Bell & others, 1981; Herdt & Boxer, 1993; Pattatucci & Hamer, 1995). The feeling of strong attraction to one sex or the other often exists for years before it is expressed. Most psychologists are now convinced that sexual orientation is not something that most people can choose or change through willpower or psychotherapy but, rather, is something that people discover about themselves.

Brain Correlates of Sexual Orientation

In humans, as in other mammals, there are certain measurable anatomical differences in the brains of males and females. Some brain areas are larger in men than in women, and the reverse is true for some other brain areas. Postmortem studies indicate that in a few of these brain areas, the male homosexual brain is more like the female heterosexual brain than like the male heterosexual brain (Rahman & Wilson, 2003). One such area is a nucleus in the anterior hypothalamus, which is larger in heterosexual men than it is in homosexual men or in heterosexual women and is believed to be the human homologue of the sexually dimorphic nucleus depicted in Figure 6.11 (LeVay, 1991; Rahman & Wilson, 2003). Comparable studies of the brains of homosexual women have not yet been done to see if their brains, at these locations, are like those of heterosexual men. Nobody knows for sure, but such brain differences may provide a biological foundation for differences in sexual orientation, and the brain differences themselves may result from early developmental influences of genes and the prenatal environment.

Effects of Genes and Prenatal Environment on Sexual Orientation

Genetic differences among individuals apparently play some role in determining sexual orientation but are not solely responsible. Several studies indicate that roughly 50 percent of the identical twin brothers and sisters of homosexual men and women are also homosexual, compared with about 15 percent of the same-sex non-identical twins or non-twin siblings of homosexual men and women (Rahman & Wilson, 2003).

Other studies, which are still quite controversial, suggest that sexual orientation might be affected by any of various factors—ranging from prenatal stress to certain medications taken during pregnancy—that alter the amount of testosterone or other androgen available to the fetus's brain during a critical period in development (Ellis & Ames, 1987; Ellis & others, 1988; Meyer-Bahlburg & others, 1995). Such work suggests that a high level of androgen action during the critical period may promote the development of brain mechanisms that predispose sexual attraction toward women and not toward men and that a relative lack of androgen during the same period may promote the opposite. Genes might influence sexual orienta-

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tion by influencing the degree to which androgen hormones can bind to the relevant brain areas during the critical developmental period.

The single most consistent, non-genetic influence on sexual orientation discovered to date is the *fraternal birth-order effect* on male homosexuality (Bogaet, 2003). The more older brothers a man has, the greater is the likelihood of his being homosexual. The effect is quite strong. One large study revealed that, on average, a couple's first son has a 2.0 percent chance of being homosexual and that this percentage increases to about 2.6 percent for a second son, 3.4 percent for a third son, 4.6 percent for a fourth son, and 6.0 percent for a fifth son (Blanchard, 2001). In contrast, the number of older sisters a man has plays no role in the likelihood of his being homosexual, and there is no birth-order effect on homosexuality in women.

The fraternal birth-order effect appears to have nothing to do with the amount or type of interaction that a boy has with his older siblings (Bogaert, 2003). The most prominent current hypothesis is that the effect results from a change in the prenatal environment that is induced by previously born sons (Blanchard, 2001; Rahman & Wilson,2003). Genes on the Y chromosome of a male fetus produce certain proteins that may help androgens to masculinize regions of the fetus's brain. These proteins are foreign to the mother's body, so if they get into her bloodstream they are likely to induce an immune reaction in her. With successive sons, the immune reaction could generate a sufficient supply of antibodies in the mother's bloodstream to inactivate one or more of those proteins within a developing fetus and thereby reduce the masculinizing effect of androgens in a portion of the fetus's brain. At present, such an explanation lies in the realm of reasonable speculation; there is no strong evidence supporting or refuting the hypothesis.

· Before birth, the presence or relative absence of testosterone pro-

• Before puberty, the adrenal hormone DHEA triggers the beginning of

brain differences that will affect the sex drive.

sexual feeling and attraction in girls and boys.

in females stimulates sexual maturation.

adrenal glands maintain the sex drive.

motes physical differentiation between males and females, including

At puberty, increased production of testosterone in males and estrogen

(except some primates). In human females, androgens produced by the

After puberty, testosterone maintains the sex drive in male mammals

(including men) while ovarian hormones do so in female mammals

SECTION REVIEW

Sex is a non-regulatory drive powerfully influenced by hormones.

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- Sexual Orientation
- Social learning explanations for homosexuality do not appear to be supported by the evidence.
- Sexual orientation may be related to prenatal brain development.
- Genes play a role in sexual orientation. Variation in the prenatal environment may also play a role.
- The more older brothers a man has, the greater the chance that he will be homosexual. This may derive from the mother's immune response to proteins produced by the Y chromosome in male fetuses.

The Sleep Drive

Sleepiness is clearly a drive. A sleepy person is motivated to go to sleep and will expend effort to reach a safe and comfortable place to do so. Achieving this goal and drifting off to sleep provides a sense of pleasure analogous to that which comes from eating when hungry or copulating when sexually motivated.

Sleepiness operates in some ways like a regulatory drive. As with hunger, thirst, or other regulatory drives, the longer one goes without satisfying the sleep drive, the stronger the drive becomes. But, unlike other regulatory drives, it is not clear what the sleep drive regulates, except sleep itself. Also, sleepiness is controlled not just by amount of sleep depivation but also by time of day. Regardless of how

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much sleep one has had recently, sleepiness tends to increase at night and decrease during daytime hours. Sleepiness is controlled not just by some consequence of sleep deprivation, but also by a biological clock that keeps time with the 24-hour day-night cycle.

The discussion in this section focuses on three questions: (1) What is sleep? (2) What are the functions of sleep? (3) What brain mechanisms control sleepiness and arousal?

Description of Sleep as a Physiological and Behavioral State

Sleep is a condition of relative unresponsiveness to the environment. Because people show little overt behavior and cannot answer questions when they are asleep, scientists who study this state must focus on physiological and subtle behavioral changes.

The most valuable index of sleep is based on the electroencephalogram (abbreviated EEG). As discussed in Chapter 5, the EEG is an amplified recording of the electrical activity of the brain that is picked up by electrodes pasted to the person's skull. The electrical signals can be stored and analyzed by a computer and can also be used to move pens up and down on a continuously moving roll of paper, producing records like those shown in Figure 6.12. The EEG recording is a gross index of the electrical activity of the brain, representing a sort of average of the activity of billions of neurons, with the greatest weight given to those lying closest to the recording site.

EEG Waves Accompanying Wakefulness and Stages of Sleep

24 How does a person's EEG change as the person goes from alert to relaxed to various sleep stages? When a person is relaxed but awake, with eyes closed, and not thinking of anything in particular, the EEG typically consists of large, regular waves called *alpha waves*, which occur at a frequency of about 8 to 13 cycles per second (see Figure

and sleep stages In general, as one goes from an alert to a relaxed state, and then to ever-deeper stages of sleep, the EEG waves become slower in frequency (fewer waves per second) and higher in amplitude (as shown by their greater vertical extent in the EEG record). The brief bursts of rapid waves called *sleep spindles* that appear in stage 2 are the most distinctive markers of the onset of sleep. Sleep stage 3 is not shown here; it is arbitrarily defined as the period when 10 to 50 percent of the EEG consists of delta waves. REM sleep, also not shown, is characterized by beta waves that look like those of the awake, attentive state. (From Snyder, F., & Scott, J., 1972)

FIGURE 6.12 EEG waves of waking



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6.12b). These relatively slow waves stem from a synchronized pulsing of neurons in the thalamus and cerebral cortex that occurs in the absence of focused mental activity or emotional excitement. When a person concentrates on an external stimulus or tries to solve a problem or becomes excited, the EEG pattern changes to low-amplitude, fast, irregular waves called beta waves (see Figure 6.12a). The low amplitude of these waves indicates that neurons are firing in an unsynchronized manner, such that their contributions to the EEG tend to cancel one another out. Whereas alpha waves are analogous to the large, regular waves that occur on a pond undisturbed by anything but a steady wind, beta waves are more akin to the effect of a million pebbles tossed suddenly onto the surface of the pond. The crests of the ripples created by some pebbles would cancel out the troughs created by others, resulting in a chaotic, high-frequency, lowamplitude pattern of ripples.

When a person falls asleep, the EEG goes through a fairly regular sequence of changes, which are used by researchers to divide sleep into four stages, illustrated in Figure 6.12c. *Stage 1* is a brief transition stage, when the person is first falling asleep, and *stages 2* through 4 are successively deeper stages of true sleep. As sleep deepens, an increased percent-

age of the EEG is devoted to slow, irregular, high-amplitude waves called *delta waves*. These waves, like others, are controlled by neurons in the thalamus that respond in an oscillating manner and synchronize the activity of billions of neurons in the cerebral cortex (Steriade & others, 1993). Corresponding to this EEG change, muscle tension, heart rate, and breathing rate decline, and the person becomes increasingly hard to awaken.

Cyclic Repetition of Sleep Stages Through the Night

Having reached stage 4, a person does not remain there for the rest of the night. Instead, after about 80 to 100 minutes of total sleep time, sleep rapidly lightens, returning through stages 3 and 2, and then a new, quite fascinating stage of sleep appears for a period of about 10 minutes or more. During this new stage the EEG is unsynchronized, looking much like the beta waves of alert wakefulness. On the basis of the EEG alone, one might think that the person had awakened, but direct observation shows that the person is sound asleep, and the record of muscle tension shows that the muscles are more relaxed than at any other sleep stage. Yet, consistent with the unsynchronized EEG, other indices of high arousal are apparent: Breathing and the heart rate become more rapid and less regular; penile erection occurs in males (even in infants and young boys); twitching movements occur in the small muscles of the fingers and face; and, most indicative of all, the eyes move rapidly back and forth and up and down under the closed eyelids. These eye movements, which can be recorded electrically along with the EEG, give this stage of sleep its name, rapid-eye-movement sleep, abbreviated REM sleep. As you may have guessed, it is during REM sleep that most dreams occur. REM sleep is also sometimes called emergent stage 1, because, even though it is different from the original stage 1, it marks the onset of a new sleep cycle. Stages 2, 3, and 4 are referred to collectively as non-REM sleep.

In a typical night's sleep, a person goes through four or five sleep cycles, each involving gradual descent into deeper stages of non-REM sleep, followed by a rapid lightening of non-REM sleep, followed by REM sleep (Hobson, 1995). Each complete cycle takes about 90 minutes. As depicted in Figure 6.13, the deepest non-REM sleep occurs in the first cycle or two. With each successive cycle, less time is spent in the deeper stages of non-REM sleep (stages 3 and 4), and more time is spent in light non-REM sleep (stage 2) and REM sleep.



Keeping track of sleep With electrodes attached to the man's scalp and other areas of his body, the researcher monitors EEG, eye movements, breathing rate, and other muscle movements to keep track of his stages of sleep and wakefulness

How do REM and non-REM sleep differ, and how do they cycle through the night?

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IFIGURE 6.13 The cycle of sleep stages through a night People usually go through four or five sleep cycles per night, each ending with a period of REM sleep. With successive cycles the depth of slow-wave sleep becomes less, and the amount of time spent in REM sleep increases. (From Snyder, F, & Scott, J., 1972.) The psychophysiclogy of sleep. In N. S. Greenfield & R. A. Sternbach (Eds.), *Handbock of psychophysiclogy*. Nev Yotk: Hol, Rinehart & Winston. Copright © 1972 by Holt, Rinehart & Winston, Inc., reproduced by permission of the publisher)



Dreams and Other Mental Activity During Sleep

26 What are some general characteristics of dreams that people describe when aroused from REM sleep, and how do these differ from the "sleep thought" that people describe when aroused from non-REM sleep? When people are awakened during REM sleep, they usually (in about 90 percent of the cases) report a mental experience that researchers call a *true dream* (Foulkes, 1985). Such a dream is experienced as if it were a real event rather than something merely imagined or thought about. The dreamer has the feeling of actually seeing or in other ways sensing various objects and people and of actually moving and behaving in the dream environment. Moreover, a true dream usually involves a progression of such experiences, woven into a somewhat coherent though often bizarre story. The more time the sleeper spends in REM sleep before awakening, the longer and more elaborate is the reported dream. Studies show that essentially everyone dreams several times a night. Those who believe that they rarely dream, or who can recall only fragments of dreams upon normal awakening in the morning, describe vivid, detailed dreams if awakened during REM periods.

Analyses of the contents of hundreds of reported dreams reveal a number of generalities about them (Domhoff, 2003). Most dreams are about people, objects, and activities that are well known and meaningful to the dreamer, but very few dreams are repetitions of events that actually happened in the dreamer's daytime experience. Most dreams involve emotions, especially negative emotions. Dreams involving fear, worry, or embarrassment are more common than joyous dreams. Among college students, dreams of being lost, of being late for an examination, and of being inappropriately dressed (or undressed) in public are common.

People who are awakened during non-REM sleep report some sort of mental activity just before awakening roughly half the time (Foulkes, 1985; Hobson, 1995). Such reports are usually not of true dreams but of *sleep thought*, which lacks the vivid sensory and motor hallucinations of true dreams and is more akin to daytime thinking. Often the subject of sleep thought is some problem that had been of concern during the day. For example, a student who had been cramming for a math exam might report working on a calculus problem while sleeping. A major difference between sleep thought and daytime thought is that the former is usually ineffective. Although the sleeper may feel that he or she is solving a calculus problem, questions upon awakening indicate that no real progress was made (Hobson, 1987).

During sleep a person is less responsive to events in the environment than when awake, but not completely unresponsive. The eyes are closed, but all of the other sensory channels remain open. In one study using fMRI, people's brains were observed to respond to various sounds when they were asleep in much the same way as they did when they were awake (Portas & others, 2000). Both when asleep and when awake, the sound of the person's own name had effects on emotional centers in the limbic system that did not occur in response to sounds that were less meaningful to the person. The fact that a parent can sleep through a thunderstorm but become aroused by the whimpering of his or her child in the next room is further evidence that sounds are sorted out to some degree by meaning in the sleeping person's brain.

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One characteristic of all mental activity during sleep is that it is quickly forgotten. Dreams, sleep thoughts, and sensory experiences during sleep are lost unless the person wakes up during them and thinks about them while awake. This is fortunate. Such forgetting prevents us from carrying around thousands of bizarre and confusing memories or of mixing up real events with dreamed ones.

Theories About the Functions of Sleep

Why must we sleep? Countless children have asked that question to protest being put to bed, and many scientists have asked it, too. Sleep must have come about in the course of evolution to serve some function or functions related to survival and reproduction; otherwise it would not be such a universal and compelling drive. Several theories have been proposed to explain the evolution of the sleep drive. The theories are not incompatible with one another, and they all appear to have some degree of validity.

The Preservation and Protection Theory

The preservation and protection theory of sleep derives primarily from comparison of sleep patterns across different species of animals. It posits that sleep came about in evolution to preserve energy and protect individuals during that portion of each 24-hour day when there is relatively little value and considerable danger in moving about. An animal needs only a certain number of hours per day to do the things that are necessary or useful for survival, and the rest of the time, according to this theory, it is better off asleep—quiet, hidden, and protected from predators and other possible dangers.

Support for this theory comes from evidence that variations in sleep time > among different species do not correspond with differences in physical exertion while awake but do correspond with feeding habits and ways of achieving safety (Allison & Cicchetti, 1976; Webb, 1982). At one extreme, large grazing animals such as bison and horses average only 2 or 3 hours of sleep per 24-hour day. Because of their large size and because they eat grass and other vegetation, which are extremely low in calories, they must spend most of their time eating, and therefore

What evidence supports the preservation and protection theory of sleep?

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Asleep and protected According to the preservation and protection theory, a major function of sleep is to keep animals quiet and hidden during that portion of the day or night when it would be most dangerous and least profitable for them to be moving about. they have little time to sleep. Moreover, because of their size and the fact that they cannot burrow or climb trees, such animals are not adept at finding safe nooks in which to sleep. Thus, they are safer awake.

Even among animals that are roughly the same size as each other, grazing animals sleep less than do meat-eaters. Sheep and goats, for example, sleep only 4 or 5 hours per 24 hours, while lions and tigers sleep 14 to 16 hours (Campbell & Tobler, 1984). Sheep and goats must spend more time eating than lions and tigers, and, because they are more preyed upon, the former are at much greater risk when asleep than are the latter. At the extreme in sleep time are opossums and bats, which average about 20 hours of sleep each 24-hour day. These two species need little time to obtain food (such as high-calorie insects or grubs), and they are adapted to hide in out-of-the-way places. According to the preservation and protection theory, they sleep so much because they have no need to be awake for long and are protected from predators while asleep.

In addition to explaining differences in total amount of sleep, the preservation and protection theory also explains differences in the time of day at which different species

sleep. Animals that rely heavily on vision generally forage during the day and sleep at night. Conversely, animals such as mice and rats that rely more on other senses, and are preyed upon by animals that use vision, generally sleep during the day and forage at night. The theory also offers an explanation for the fact that infants in most species of mammals sleep much more than adults. Infants who are being cared for by adults do not need to spend time foraging, and sleep protects them from wandering away into danger. Their sleep also gives their caregivers an opportunity to rest or attend to other needs.

It is interesting to speculate, in this vein, about the evolutionary conditions behind the &-hour nighttime sleep pattern that characterizes adult humans throughout the world. Humans are highly visual creatures who need light to find food and do other things necessary for survival. At night it may have been best for us, during most of our evolution, to be tucked away asleep in a cave or other hiding place, so as not to be tempted to walk about and risk falling over a cliff or being attacked by a nocturnal predator. Only during the past few centuries—an insignificant speck of evolutionary time—have lights and other contrivances of civilization made the night relatively safe for us. According to this line of thinking, our pattern of sleep might be in part a vestigial trait, a carryover from a period when the night was a time of great danger. To the degree that nighttime is still more dangerous than daytime, our pattern of sleep may continue to serve an adaptive function.

The Body-Restoration Theory

The *body-restoration theory* of sleep function is the theory that most people intuitively believe. It is the theory that your parents probably repeated to you as their reason for sending you to bed at a certain hour. According to this view, the body wears out during the day and sleep is necessary to put it back in shape.

Scientific support for this theory includes the observation that sleep *is* a time of rest and recuperation. The muscles are relaxed, metabolic rate is down, and growth hormone, which promotes body repair, is secreted at a much higher rate than during wakefulness (Douglas, 2002; Siegel, 2003). Also consistent with the restoration theory is the observation that prolonged, complete sleep deprivation in rats results in breakdown of various bodily tissues, leading, within about 3 weeks, to death (Everson, 1993; Everson & others, 1989).

The theory also offers an explanation for the general tendency of small mammals to sleep longer than large ones. Small mammals need to maintain a higher



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overall level of metabolism than do large mammals, because they lose body heat more rapidly, and higher metabolism leads to greater wear and tear on bodily tissues (Siegel, 2003). The theory does not, however, explain the large differences in sleep time between grazing animals and meat-eating animals that have similar body sizes and metabolic rates. It also does not explain the failure of researchers to find consistent positive correlations, either across species or within species, between the amount of time an animal sleeps and the average amount of energy it expends through vigorous activity during the day.

The fact that all vertebrate animals sleep at least an hour or two out of every 24 hours, regardless of the degree to which they are at risk while sleeping, suggests that some amount of sleep is needed for body repair. But the body-restoration theory does not provide a satisfactory explanation of the large differences among species in sleep time, and it offers no explanation for the fact that some animals sleep during the day while others sleep at night.

The Brain-Maintenance and Memory-Consolidation Theories of REM Sleep

If sleep in itself serves purposes of energy conservation, protection from danger, and bodily restoration, then what is the function of REM sleep? Why is restful non-REM sleep interrupted regularly by these periods of increased brain activity and energy expenditure? This question has generated much debate and research.

One long-standing theory is that REM sleep provides regular exercise to groups > of neurons in the brain (Hobson, 1988). Synapses can degenerate if they go too long without being active (Edelman, 1987), so neural activity during REM sleep may help preserve important circuits. One line of evidence for this theory is that the longer a person or animal sleeps, the greater is the proportion of sleep time spent in REM sleep. With longer sleep periods there may be more need to interrupt non-REM sleep. With longer ein fetuses and infants than in adults, regardless of species (see Figure 6.14). In fact, the peak of REM sleep in humans occurs in 30-day-old fetuses, which spend almost 24 hours a day in this state. Perhaps as their brains are developing in the relative isolation of the womb, they need to exercise sensory and motor pathways, and REM sleep is their means for doing that



29 What evidence supports the theories that REM sleep promotes the maintenance of brain circuits and the consolidation of newly learned skills?



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Sweet dreams? We have no idea whether human fetuses, such as this one of 7 months, experience dream sensations or not, but we do know that they spend most of their time in REM sleep.

30 How might dreams be explained as

inevitable consequences of the state of the brain during REM sleep? (Hobson, 1988). In the fetus, REM sleep is accompanied by body movements such as kicking and twisting, which are apparently triggered by the bursts of activity in motor areas of the brain, so muscles as well as brain circuits are exercised. By the time of birth a neural inhibitory system matures, which inactivates most motor neurons during REM sleep and thus prevents most movements that would otherwise occur. However, the motor neurons to the eyes and to various internal structures, such as the heart, remain uninhibited, so eye movements and increased heart rate persist as observable effects of the brain's activity.

Closely related to the theory that REM sleep helps to maintain existing brain circuits is the theory that such sleep plays a role in consolidating the effects of new learning. As discussed in Chapter 5, memory consolidation involves the selective strengthening of existing synapses and the manufacture of new ones. According to some sleep researchers, the physiological changes observed in the brain during REM sleep are ideal for promoting such growth (Hobson, 2002).

In many experiments, laboratory animals that were trained in a task and then tested several hours later performed better if they were permitted REM sleep during the period between training and testing than if they were deprived of such sleep (Smith, 1995; Maquet, 2001). Similar experiments with people have produced mixed results, depending apparently on the kind of task. There is little evidence that REM sleep improves subsequent recall for verbally learned factual information, but there is considerable evidence that it improves performance on perceptual-motor tasks—tasks that are more similar to those used with laboratory animals (Stickgold & others, 2001).

In one experiment, college students practiced the skill of locating, as quickly as possible, specific images hidden in visually textured backgrounds (Karni & others, 1994). After this training session, the students slept in the laboratory for about 8 hours. Some were deprived of REM sleep by being awakened whenever they entered that sleep stage; others were deprived of a comparable amount of non-REM sleep by being awakened regularly during non-REM sleep; and still others were allowed to sleep undisturbed through the night. The results were quite dramatic. Those who were deprived of non-REM sleep or were not sleep-deprived at all performed the perceptual task better right after the sleep period than they had just before the sleep period, but those deprived of REM sleep showed no such improvement. This effect occurred only for the newly learned task; lack of REM sleep had no effect on performance of a similar task that had been learned several days earlier. Subsequent experiments have shown that improved performance of such tasks occurs even after a 60- to 90-minute nap, as long as the nap includes at least some time spent in REM sleep (Mednick & others, 2003). Learned motor skills, such as tapping one's fingers in a particular sequence, comparable to playing a series of notes on a piano, also improve dramatically when a period of REM sleep follows initial practice (Walker & others, 2003).

Dreams Viewed as Side Effects of Brain Activity in REM Sleep

Nobody knows if dreams themselves have a function. One view prevalent among researchers today is that dreams don't serve any special purpose, but are side effects of physiological changes in REM sleep that do serve a purpose (Antrobus, 2000; Hobson, 2002, 2004). Neurons in visual and motor areas of the brain become active during REM sleep, and hallucinations of sights and movements may be an inevitable consequence of such activity. Neurons involved in memory retrieval and emotions also become active, and these may bring familiar images and feelings into the sleeping person's mind. In research done many years ago, electrical stimulation in portions of the cerebral cortex produced dreamlike hallucinations in people who were awake (Penfield & Perot, 1963). A similar phenomenon may well occur in REM sleep. In addition to producing hallucinations, the brain continues in REM sleep to engage in some degree of thought, just as it does in non-REM sleep. But now the thought becomes wrapped up in trying to make sense of the hallucinations. The result is the weaving of a story connecting CHAPTER 6 - MECHANISMS OF MOTIVATION AND EMOTION 209

one hallucination to the next—hence, the dream. Because of reduced mental capacity during sleep, the story is less logical than one the awake brain would develop, but it still contains some degree of logic.

Sometimes the side-effect theory just described is interpreted as an argument against the psychoanalytic view that dream analysis can be useful for understanding a person's mind. But that interpretation seems unjustified. Even if dreams are triggered by random events in the brain, the actual images, emotions, and story lines that constitute the dream are not random. They certainly contain elements based on the dreamer's experience, and because they occur at a time of reduced mental capacity, ideas or feelings that are normally suppressed by higher mental processes could emerge and perhaps be useful in psychoanalysis (Reiser, 1991). In fact, in one recent experiment, people were more likely to dream about a particular person if they were asked to suppress thoughts about that person just before going to sleep than if they were asked to think actively about that person just before going to sleep (Wegner & others, 2004). This finding is at least consistent with the idea that dream analysis might reveal ideas and concerns that a person is actively suppressing during the day.

Individual Variation in the Sleep Drive, and Effects of Failure to Satisfy That Drive

The sleep drive varies from person to person. Some need more sleep than the typical 8 hours a night to function well, and others need less (Douglas, 2002). At the extreme are rare people, referred to as *nonsomniacs*, who sleep far less than most of us and yet do not feel tired during the day. A study of nonsomniacs conducted many years ago by Ray Meddis (1977) suggests that such people are generally vigorous and healthy. One of Meddis's subjects was a 70-year-old nurse who reported that for most of her life she had slept about 50 minutes per night. She was very active during the day and usually spent the night in quiet activities, such as reading or painting. To verify her nonsomnia, Meddis observed her continuously for a prolonged period in the sleep lab. She slept not at all the first 3 days and nights in the lab, remaining cheerful and talkative throughout. Finally, on the fourth night, she slept a total of 99 minutes and awoke feeling fully rested.

The fact that nonsomnia is compatible with physical and psychological health adds to the evidence that only a relatively small amount of sleep is needed for body repair and growth of new synapses in the brain. Yet, most of us do need roughly 8 hours of sleep to function well. We need that sleep because we have a sleep drive that overwhelms our mind and makes us tired, miserable, and rela-

tively ineffective at mental tasks when we fail to meet it. The drive for that much sleep may have evolved for reasons other than body repair and brain growth, but that doesn't mean we can ignore it. It is important to distinguish nonsomnia, which is very rare, from *insomnia*, which is relatively common. An insomniac is someone who has a normal drive for sleep but who, for some reason (such as worry), has great difficulty sleeping at night. Unlike a nonsomniac, an insomniac feels tired all day as a result of not sleeping. And so do most people who voluntarily reduce their sleep.

Many laboratory studies have been conducted in which people with normal sleep drives have voluntarily stayed awake for periods of 3 or 4 days or even longer. After about 72 hours awake, some people begin to experience symptoms such as distorted perceptions and extreme irritability (Borbély, 1986). Sleepiness waxes and wanes during such studies, in accordance with the inner clock that controls it. How does insomnia differ from nonsomnia, and what negative consequences occur when people deliberately resist their sleep drive?

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Sleep deprivation can be dangerous The most reliable consequence of sleep deprivation is increased sleepiness, which, in a situation like this one, can be deadly.







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FIGURE 6.15 Decline and rise in vigilance during sleep deprivation In this experiment, university students were kept awake for 42 hours before the start of testing. Just before testing, some received a strong dose of caffeine, others received one of three doses of another stimulant drug. modafinil, and still others received a placebo. In the test of vigilance, conducted every hour, the subjects watched a time display on a computer screen and were instructed to press a button as quickly as possible each time it began to increment. The measure was response speed (the reciprocal of response time). As shown in the graph, in the placebo group vigilance declined steadily from mid-night to 8 a.m. and then began to increase. This is consistent with other evidence that the circadian rhythm of sleepiness and wake fulness continues even after long periods of sleep deprivation. There was no such decline in the groups receiving caffeine or in those receiving high doses of modafinil. (From Wesensten & others, 2002.)



People find it much harder to stay awake during the late night and early morning hours than they do during the rest of the 24-hour day, even after several days of sleep deprivation. In such experiments, scores on tests of vigilance, judgment, and creative thinking also wax and wane in a 24-hour cycle, keeping pace with sleepiness (Horne, 1979, 1988; Jennings & others, 2003). Scores on such tests decline when sleepiness rises, apparently because sleepy people have difficulty attending to the task and because their performance is often interrupted by brief moments of falling asleep, from which they arouse themselves. In general, stimulants such as caffeine, which counteract sleepiness, also remove the negative effects of sleep deprivation on the performance of such tasks. For an example of data showing the effects both of sleepiness and of stimulants on a test of vigilance, see Figure 6.15 (Wesensten & others, 2002).

In the real world outside of the laboratory, sleepiness, with its accompanying decline in attention and judgment, is dangerous. Many accidents in the workplace result from it, and sleepiness rivals drunkenness as a leading cause of traffic fatalities (Horne & Reyner, 1995, 2001).

Brain Mechanisms Controlling Sleep

In the early years of sleep research, some researchers believed that sleep is the natural state that the brain slips into when not aroused by external stimulation, so they saw no need to posit the existence of special sleep-inducing mechanisms. But such a view is inconsistent with the observation that sometimes sleepiness overwhelms us even when external stimulation is high, while at other times we can't sleep no matter how quiet, dark, and unstimulating the environment may be. We now know that sleepiness, like other drives, is actively promoted by neural mechanisms located in the hypothalamus and in brain areas closely connected to the hypothalamus.

Rhythm-Generating Neurons in the Hypothalamus Control the Daily Cycle of Sleepiness

In all animals, as I noted earlier, the sleep drive waxes and wanes in a cyclic manner over the 24-hour day. This cycle of sleepiness and wakefulness continues, in laboratory animals and human volunteers, even after many days in an artificial time-free environment—an environment where there are no regular changes in

32 What is some evidence that the sleep drive is affected by an internal clock, I

drive is affected by an internal clock, located in the hypothalamus, that can operate even without external time cues?

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lighting or other cues that could indicate the time of day. In such an environment, the cycle is typically a few minutes longer or shorter than 24 hours, and it varies from individual to individual, but it is remarkably constant within a given individual (Lavie, 2001; Takahashi & Zatz, 1982).

The technical term for any repetitive biological change that continues at close to a 24-hour cycle in the absence of external cues is *circadian* [sir-Kade-ee-un] *rhythm.* The term comes from the Latin words *circa*, meaning "about," and *dies*, meaning "day." The clock that controls the circadian rhythm of sleep in all mammals is located in a specific nucleus of the hypothalamus called the *suprachiasmatic* [soup-ra-kai-az-mat-ick] *nucleus*. If this nucleus is damaged, animals lose their regular sleep-wake rhythms and sleep at rather random times over the 24-hour day, and the same is true of human patients (Cohen & Albers, 1991). They also lose their daily rhythms of change in body temperature and secretion of certain hormones, such as cortisol, because the suprachiasmatic nucleus controls those rhythms, too. This nucleus contains rhythm-generating neurons, which gradually increase and decrease their rate of action potentials over a cycle of approximately 24 hours, even when surgically isolated from other parts of the brain (Cermakian & Boivin, 2003).

Input from the Eyes Synchronizes the Hypothalamic Clock to the Light-Dark Cycle

Under normal conditions, the circadian clock is reset each day by the waxing and >> waning of daylight, so rhythms occur in periods of exactly rather than approximately 24 hours. Experiments with animals show that the cycle can be lengthened or shortened, by as much as a couple of hours either way, by artificially changing the period of light and dark. And experiments with humans as well as other animals show that the cycle can be reset through exposure to bright fluorescent lights.

Charles Czeisler and his colleagues (1989) found that just a few hours of bright fluorescent lighting at night, coupled with avoidance of bright light during the daytime, can, within a few days, reverse a person's circadian clock so that he or she becomes sleepy during the day and alert at night. Very bright light is most effective at delaying the sleep phase of the cycle, but even levels comparable to those already occurring in well-lit offices, factories, and homes have a significant effect (Boivin & others, 1996). Such knowledge can be applied to help night workers adapt their bodily rhythms to their work hours. In one experiment, people simulating night work for a week were more alert at work, slept better during the day, and showed a more complete shift in their body temperature cycle if their work environment

was very brightly illuminated and their daytime sleep room was completely darkened than they did under more typical lighting conditions (Czeisler & others, 1990). The fact that light delays the onset of the sleep phase of the cycle may explain why most people living in modern societies, with electric lights on in the evening, have their peak periods of sleepiness in the wee hours of the morning rather than earlier at night (Lavie, 2001).

Brain researchers have found that changes in lighting influence the rhythm-generating neurons by way of a neural tract that runs from the retinas of the eyes to the suprachiasmatic nucleus. These neurons are different from those that are involved in vision, and they derive at least partly from light receptors in the retina that are different from the rods and cones (discussed in Chapter 7) that are involved in vision (Van Gelder, 2003). 33 What is some evidence that the internal clock is continuously reset by daily changes in light? Through what pathway does that resetting occur?

Resetting the circadian clock Steven Lockley and his colleagues have found that blue light works better than white light in resetting the circadian clock that times the onset of sleepiness. Timed exposures to such light can help shift workers and world travelers adapt their sleep schedules to the requirements of their work or travel.



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PART 3 PHYSIOLOGICAL MECHANISMS OF BEHAVIOR

Other Neurons in the Hypothalamus and Pons Promote Sleepiness or Wakefulness

How are sleepiness and arousal controlled by a sleep center in the preoptic area of the hypothalamus and by arousal centers in the posterior hypothalamus and pons? How are these centers influenced by the circadian clock? Researchers have identified a number of other nuclei in the brain that are crucially involved in the generation of sleepiness and wakefulness. One such nucleus is the ventrolateral preoptic nucleus, which lies in the hypothalamus in front of the suprachiasmatic nucleus (Saper & others, 2001). Electrical stimulation in this area can cause a previously alert animal to fall asleep, and lesions there can result in permanent sleeplessness, eventuating in death in rats. In humans, a viral infection that destroys neurons in this nucleus greatly reduces sleepiness and average daily sleep time (Saper & others, 2001). This nucleus receives neural connections from the suprachiasmatic nucleus, and it also receives input from other brain areas that are involved in the control of sleep and wakefulness. Neurons from this nucleus connect to many brain areas and release the inhibitory neurotransmitter GABA (Saper & others, 2001). The resulting dampening of neural activity throughout the brain is believed to bring on the state of sleepiness and, subsequently, sleep itself.

Other brain areas, including a nucleus in the posterior (rear) portion of the hypothalamus and in the pons, are crucially involved in arousing the brain and maintaining a state of alertness. These nuclei receive input from a variety of sources, including the suprachiasmatic nucleus, and axons from these neurons release excitatory neurotransmitter molecules onto post-synaptic neurons in many parts of the brain (Aston-Jones & others, 2001; Siegel, 2004). An individual's level of sleepiness or arousal at any given moment is believed to derive from a balance between the excitatory action coming from these neural centers and the inhibitory action coming from the ventrolateral preoptic nucleus (Saper & others, 2001).

SECTION REVIEW

There is a drive for sleep, a state involving relative unresponsiveness to the environment. **Basic Characteristics** Functions of Sleep **Brain Mechanisms** Researchers use EEG records to The preservation and protection theory asserts The suprachiasmatic nucleus in divide sleep into stages, with that sleep is needed to conserve energy and the hypothalamus acts as an increase safety. It is supported by cross-species comparisons of amount and timing of sleep. stage 4 being deepest. internal clock for sleepiness and wakefulness. A sleeper progresses through the sleep stages, from light to Light synchronizes the internal The body-restoration theory contends that the deep and rapidly back to light, in clock with the light-dark cycle. body needs sleep to recover from wear and approximately 90-minute cycles. tear. It is supported by sleep-deprivation studies A hypothalamic nucleus that REM (rapid-eye-movement) in animals receives input from the suprachiasmatic nucleus sleep occurs at the transition · REM sleep may function to maintain brain cirinduces sleep using an inhibitobetween one sleep cycle and cuits and consolidate new learning. the next ry neurotransmitter True dreams occur in REM Different people need different amounts of Other nuclei release excitatory sleep, and sleep thought occurs sleep. Sleep deprivation reduces performance. neurotransmitters to waken the especially on tests of vigilance. brain and keep it alert. in non-REM sleep.

Foundations for Understanding Emotions

Enough sleep; wake up and face the challenges of the day. Midterm exams are just around the corner, your family is after you to get your life in order, your lover has just left you for another, the surgeon says your nose and left ear will have to go, and a hungry tiger is crouched behind you. Are you awake? All these events have something in common: All (if you believe them) have the potential to elicit strong emotions.