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CHAPTER 10 MOTIVATION

Y ou're hurrying to get to an important job interview on time. You were running late this morning, so you skipped breakfast. Now you're starving. It seems as if every advertisement you see along your route features food – eggs, sandwiches, sweet and refreshing juices. Your stomach rumbles and you try to ignore it, but that is next to impossible. Every kilometer you go, you're that much hungrier. You nearly hit the car in front of you as you stare at a sign advertising pizza. In short, you have been overwhelmed by the motivational state known as hunger. A motivation is a condition that energizes behavior and gives it direction. It is experienced subjectively as a conscious desire – the desire for food, for drink, for sex. Most of us can choose whether or not to act on our desires. We can force ourselves to forgo what we desire, and we can make ourselves do what we would rather not do. Perhaps we can even deliberately choose not to think about the desires that we refuse to act on. But it is considerably more difficult – perhaps impossible – to control our motivations directly. When we are hungry, it is hard not to want food. When we are hot and thirsty, we cannot help wanting a cool breeze or a cold drink. Conscious choice appears to be the consequence, rather than the cause, of our motivational states. So what does control motivation, if not deliberate choice? The causes of motivation range from physiological events within the brain and body to our culture and social interactions with the other individuals who surround us. This chapter will discuss the control of basic motivations such as thirst, hunger, and sex. To a large extent these motivations arise from our biological heritage and reveal general principles about how motivation and reward work to give direction to behavior. For basic motivations like hunger, thirst, and sex, psychologists have traditionally distinguished between two types of theories of motivation. The difference concerns where the motivation comes from, what causes it, and how the motivation controls behavior. On the one hand are drive theories, which emphasize the role of internal factors in motivation. Some internal drives, such as those related to hunger or thirst, have been said to reflect basic physiological needs. For motivations like sex or aggression, drive factors seem less tied to absolute physiological needs. After all, does one ever need to aggressively attack another in the same way that one needs to eat or drink? Still, aggression and sex have been said to have drive aspects, both in the sense that internal factors such as hormonal state often appear important and in the sense that they may have evolved originally to fulfill basic ancestral needs. CHAPTER OUTLINE

On the other hand are incentive theories of motivation, which emphasize the motivational role of external events or objects of desire. Food, drink, sexual partners, targets of attack, relationships with others, esteem, money, and the rewards of success – all are incentives. Incentives are the objects of motivation. After all, our motivations don’t operate in a vacuum – when we want, we want something. The nature of that something pulls us in one direction or another. The goal might be tasty food, water to drink, a partner for interaction, expulsion of an intruder, or possession of a disputed resource. Many incentives also serve as rewards. They can produce pleasure and reinforce behavior that leads to them. Some incentives are primary reinforcers, meaning that they are able to act as rewards independently of prior learning. For example, a sweet taste or a sexual sensation may be pleasant the first time it is experienced. Other incentives are secondary reinforcers, meaning that they have gained their status as rewards at least partly through learning about their relationship to other events. For example, money or good grades can be effective incentives, based on our cultural experience with them and with the status and success they represent. For animals, a conditioned stimulus that has been paired with food can serve as an effective reward. In every case, learning is crucial to the formation of secondary reinforcers. Although less important, learning may even play a part in modulating the effectiveness of some primary reinforcers. For example, you may have been hungry when you were born – but you weren’t born with any idea of the foods that are now your favorites. Incentive theories of motivation focus especially on the relationship of learning and experience to the control of motivation. Drive and incentive theories provide different perspectives on the control of motivation. But the difference between the theoretical perspectives is primarily in their points of view, and there actually is no conflict between the two. It is widely acknowledged that both types of processes exist for almost every kind of motivation (Toates, 1986). But it is easier to focus on one type of control and thoroughly understand it before switching to the other. For this reason, we will consider drive processes in the first section and then turn to incentive processes in the second section. In the third section, we will integrate the two perspectives as we discuss eating and eating disorders, because both drive and incentive factors operate together in real life, and they often interact (see Figure 10.1). Consider again the example that opened this chapter. A drive factor (your hunger) enhanced the motivational effect of incentives (the advertisements depicting food). In fact, the taste of food becomes more pleasant to most people when they are hungry and less pleasant when they have eaten enough (Cabanac, 1979). Have you ever skipped lunch to better enjoy an evening feast? Or been scolded for snacking because it would ‘ruin your dinner’? Conversely, incentive factors can awaken drive states. Have you ever walked through the delicious aroma from a bakery or restaurant and suddenly realized that you were hungry? Yet even considering drives and incentive factors together leaves the story of motivation incomplete. Social and cultural factors also come into play. We introduce those in our discussion of eating and eating disorders and draw on them again in the fourth and final section on sexuality, a social motive considerably more complex than thirst or hunger. ª WOLFGANG AMRI j DREAMSTIME.COM The causes of motivation range from physiological events such as thirst to social aspirations and cultural influences such as those that create the desire to excel. FABRICE COFFRINI/AFP/GETTY IMAGES

DRIVES AND HOMEOSTASIS Our lives depend on keeping certain things the same. If the temperature of your brain changed by more than several degrees, you would quickly become unconscious. If the proportion of water in your body rose or fell by more than a few percent, your brain and body could not function and you would risk death. Humans and animals walk a tightrope of balance between physiological extremes. Like delicate and finely tuned machines, we cannot work unless our internal environment is in balance. But unlike most machines, we've been designed to maintain this balance ourselves. Even when the outside world changes, our internal states remain relatively stable. A great deal of basic motivation is directed toward helping to maintain our internal balance. To keep our internal world within the narrow limits of physiological survival, we have active control processes to maintain homeostasis, a constant internal state (homeo means 'equal', and stasis means 'static' or 'constant'). A homeostatic control process drives a system to actively work to maintain a constant state (that is, homeostasis). Homeostatic control processes can be psychological, physiological, or mechanical. A familiar example is the thermostat that runs your central heating boiler or air conditioner. Thermostats are designed to maintain temperature homeostasis. When you set your thermostat to a particular temperature, that temperature is the goal value or set point. A set point is the value that the homeostatic system tries to maintain. If the winter room temperature falls below the value you set, the thermostat is triggered: The discrepancy between its goal and the actual temperature causes it to activate the boiler. If the summer room temperature rises above the thermostat's set temperature for cooling, the thermostat activates the air conditioner. A thermostat coupled to both boiler and air conditioner can be used to keep your room at a stable temperature even as the seasons change. Many physiological processes work like thermostats: They activate motivations that help maintain homeostasis. Body temperature and homeostasis With a 10 °C drop in brain temperature, you'd lose consciousness. If your brain temperature rose more than 10 °C above normal, you would die. Even though you may have been in very hot or cold weather, your brain remained largely protected within a narrow range of several degrees centigrade. Homeostatic control systems, both physiological and psychological, are the reason for this constancy. Physiological responses such as sweating and shivering are part of the reason your brain temperature remains so constant. These physiological responses provide cooling in the form of evaporation and heating in the form of muscle activity. Psychological reactions also come into play as you begin to feel uncomfortably hot. You may find yourself wanting to shed clothing, have a cool drink, or find shade. But what turns on these physiological and psychological responses? When you are under the hot sun, your entire body becomes hot. Conversely, if you remain too long unprotected in the cold, your entire body becomes hypothermic (too cold). But only within your brain is the change of temperature actually detected. Neurons at several sites in the brain, especially within the preoptic (front) region of the hypothalamus at the base of the brain, are essentially neural thermostats (Satinoff, 1983). They begin to operate differently as their own temperature changes. These neurons serve as both the thermometer and the homeostatic set point within your body. When their temperatures diverge from their normal levels, their metabolism alters, Conscious desires e.g., 'I want that banana.' Behavioral attraction e.g., 'Reach for the banana.' Conscious pleasure e.g., 'This banana tastes great.' Drive signals e.g., Hunger pang Incentive motivation Physiological needs Learning e.g., 'Bananas have always satisfied my hunger.' External stimulus Figure 10.1 A Model of Basic Motives. An external stimulus, such as the sight of food, is compared to the memory of its past reward value. At the same time, physiological signals of hunger and satiety modulate the potential value at the moment. These two types of information are integrated to produce the final incentive motivation for the external stimulus, which is manifested in behavior and conscious experience.

(Adapted from Toates, 1986) DRIVES AND HOMEOSTASIS For more Cengage Learning textbooks, visit www.cengagebrain.co.uk

362 CHAPTER 10 MOTIVATION and this changes their activity or firing patterns. This triggers physiological reactions such as perspiration or shivering, which help correct your body temperature. In addition, it triggers your sensation of being too hot or too cold, which makes you want to seek shade or put on a coat, behavioral solutions to the same problems. When you are too hot, a cool breeze can feel good. Likewise, when you are too cold, a warm bath feels pleasant. But as your own internal temperature changes, your perception of these outside events also changes. Although ordinarily your entire body changes temperature by a degree or two when you are in situations that make you feel very hot or cold, it is only the slight change in your brain temperature that causes the change in the way you feel. The brain can be fooled into feeling hot or cold by merely changing the temperature of a relatively few neurons in the hypothalamus. For example, cooling of the hypothalamus alone (by painlessly pumping cold liquid through a small loop of tubing that has been surgically implanted into the hypothalamus) motivates a rat to press a bar to turn on a heat lamp that warms its skin – even though its overall body temperature has not been lowered (Satinoff, 1964). The hypothalamic neurons have detected a change in their own temperature away from the normal set point. Most of us have experienced a temporary change in set point. An illness can temporarily raise brain set points to several degrees above normal. Then the temperature they ‘seek’ becomes higher, and a fever results. Physiological reactions that elevate body temperature are activated. You shiver, and your body temperature begins to rise above normal. But in spite of the rise in temperature, you may still feel cold – even in a warm room – until your hypothalamic neurons rise all the way to their elevated set point. Thirst as a homeostatic process Satisfying thirst is an important homeostatic process. Thirst is the psychological manifestation of the need for water, which is essential for survival. What controls this process? After going without water or exercising intensively, the body begins to deplete two kinds of fluid reservoirs as water is gradually eliminated through perspiration, respiration, or urination. The first type of reservoir is made up of water contained within the cells. This water is mixed with the protein, fat, and carbohydrate molecules that form the structure and contents of the cell. The water inside your cells is your intracellular reservoir. The second type of reservoir is made up of water that is outside the cells. This water is contained in blood and other body fluids and is called the extracellular reservoir. Extracellular thirst results when our bodies lose water because we have gone without drinking or have exercised intensively. Water is extracted from the body by the kidneys in the form of urine, excreted by sweat glands in the skin, or breathed out of the lungs as vapor, and in For more Cengage Learning textbooks, visit www.cengagebrain.co.uk ª KATSYARYNA TSVIRKO j DREAMSTIME.COM Most bar owners know that salty foods trigger osmotic or intracellular thirst and thus induce customers to drink more. each case it comes most directly from the blood supply. The loss reduces the volume of extracellular fluid that remains; in turn, the loss of blood volume produces a reduction in blood pressure. You will not feel this slight change in blood pressure, but pressure receptors within your kidneys, heart, and major blood vessels detect it and activate sensory neurons that relay a signal to the brain. Neurons in the hypothalamus next send an impulse to the pituitary gland, causing it to release antidiuretic hormone (ADH) into the bloodstream. ADH causes the kidneys to retain water from the blood as they filter it. Rather than send this water on to become urine, the kidneys deliver it back to the blood. This happens whenever you go without drinking for more than several hours. For example, you may have noticed that your urine appears more concentrated in color at such times (for instance, when you wake up

after a night's sleep). In addition, the brain sends a neural signal to the kidneys that causes them to release the hormone renin. Renin interacts chemically with a substance in the blood to produce yet another hormone, angiotensin, which activates neurons deep within the brain, producing the desire to drink. You may recall that this entire chain of events is triggered by a drop in blood pressure caused by dehydration. Other events that cause dramatic loss of blood pressure

can also produce thirst. For example, soldiers wounded on the battlefield or injured people who have bled extensively may feel intense thirst. The cause of their craving is the activation of pressure receptors, which triggers the same chain of renin and angiotensin production, resulting in the experience of thirst (Fitzsimons, 1990). Intracellular thirst is caused by osmosis – the tendency of water to move from zones where it is plentiful to zones where it is relatively rare. It is primarily the concentration of 'salt' ions of sodium, chloride, and potassium that determine whether water is plentiful or rare. As the body loses water, these concentrations begin to rise in the bloodstream. In essence, the blood becomes saltier. The higher concentrations within the blood cause water to migrate from the relatively dilute insides of body cells – including neurons – toward the blood. In a process something like sucking up a puddle of water with a paper towel, water is pulled out of the neurons and other cells. Neurons within the hypothalamus become activated when higher salt concentrations in the blood pull water from them, causing them to become dehydrated. Their activation produces 'osmotic' or intracellular thirst, producing the desire to drink. Drinking replaces water in the blood, reducing the concentration of salt, which in turn allows water to return to neurons and other cells. That is why people become thirsty after eating salty food – even though they might not have lost water. INTERIM SUMMARY l Motivational states direct and energize behavior. They arise from two sources: internal drive factors and external incentive factors. l Drive factors tend to promote homeostasis: the preservation of a constant internal state. l Homeostasis involves (1) a set point, or goal value, for the ideal internal state, (2) a sensory signal that measures the actual internal state, (3) a comparison between the set point and the sensory signal, and (4) a response that brings the actual internal state closer to the set point goal. l Temperature regulation is an example of homeostasis. The regulated variable is the temperature of the blood, and sensors for this are located in various parts of the body, including the hypothalamus. Adjustments are either automatic physiological responses (e.g., shivering) or voluntary behavioral ones (e.g., putting on a sweater). l Thirst is another homeostatic motive that operates on two regulated variables, extracellular fluid and intracellular fluid. Loss of extracellular fluid is detected by bloodpressure sensors, neurons in major veins and organs that respond to a drop in pressure. Loss of intracellular fluid is detected by osmotic sensors, neurons in the hypothalamus that respond to dehydration. INCENTIVE MOTIVATION AND REWARD CRITICAL THINKING QUESTIONS 1 Homeostatic processes can produce both unconscious, automatic responses (e.g., shivering) and conscious, behavioral ones (e.g., getting under a blanket). Compare and contrast each form of motivation. Can you envision one form without the other? 2 Here we've discussed how two internal factors – extracellular thirst and intracellular thirst – motivate drinking. What other factors might motivate drinking? To what extent do you think social and cultural factors motivate drinking? INCENTIVE MOTIVATION AND REWARD Motivation typically directs behavior toward a particular incentive that produces pleasure or alleviates an unpleasant state: food, drink, sex, and so forth. In other words, incentive motivation – or wanting something – is typically associated with affect – or liking that same something. Precisely speaking, the term affect refers to the entire range of consciously experienced pleasure and displeasure. Yet in discussing motivation and reward, we typically emphasize the pleasure half of the continuum, the part that corresponds with liking. The sheer pervasiveness of affect in our experience of life has led some to suggest that pleasure has evolved to serve a basic psychological role (Cabanac, 1992). That role is to shape behavior by helping to define a psychological ‘common currency’ that reflects the value of each action we perform. Pleasure tends to be associated with stimuli that increase our ability to survive or our offspring’s ability to survive. These include tasty food, refreshing drink, and sexual reproduction. Painful or frustrating consequences are associated with events that threaten our survival: physical damage, illness, or loss of resources. The rewarding or affective consequences of an action, in other words, generally reflect whether that action is worth repeating. But to guide future actions, momentary pleasures and displeasures need to be learned, remembered, and attributed to relevant objects and events, imbuing those objects and events with incentive salience, meaning that these objects and events have become linked with anticipated affect, which grabs our attention and steers our seeking behavior. So, however closely interwoven incentive motivation and pleasurable rewards are in our conscious experiences of the world, this does not mean that ‘wanting’ and ‘liking’ are the same thing (Berridge, 2007). Indeed, as you will see in the Cutting Edge Research box and drug addiction section, they can diverge under special circumstances. One clear distinction concerns the timing

 CUTTING EDGE RESEARCH WANTING VERSUS LIKING If liking something in the past nearly always means that you will want it again in the future, how can researchers be sure that the brain’s dopamine system accounts for wanting, but not liking? This has been a tough distinction to make. Indeed, in much earlier work, the two concepts (wanting and liking) were fused together into the simpler notion of rewards. As early as the 1950s, researchers discovered that electrical stimulation of certain areas of the brain (namely, the hypothalamus) was a powerful reward, evidenced by the fact that animals and people would work in order to get it again (Olds, 1956). Animals and people clearly wanted and avidly sought out brain stimulation or other natural rewards – like food or sex – when paired with brain stimulation. This was taken as evidence that brain stimulation was itself a reward, both pleasurable (liked) and desired (wanted). The targeted brain sites were even dubbed ‘pleasure centers’ by many. And because dopamine neurons appeared to be crucial links within the so-called pleasure centers (Valenstein, 1976), for many decades – and continuing to this day – scientists have linked dopamine with pleasant affect (Isen, 2002). But the only way that scientists can distinguish ‘liking’ from ‘wanting’ is to use separate measures for each. Seeking out repeated experiences more closely coincides with the concept of ‘wanting’. How can we index ‘liking’ in a way that differentiates it from ‘wanting’? Recall that liking – or pleasurable affect – is experienced during consumption, not in anticipation of it. Facial and bodily movements often reveal the experience of pleasure. Consider the case of eating good food. We humans can generally tell when someone else – even an infant – likes the taste of something. When something tastes good, we tend to smile and lick our lips. Likewise, when something tastes bad, we tend to frown with our mouths open and our upper lips raised. It turns out that nonhuman primates and many other mammals, including rats, share some of these same facial expressions for good- and bad-tasting foods (Steiner, Glaser, Hawilo, & Berridge, 2001) (see Figure). If rewarding brain stimulation truly induces pleasure, then it should increase seeking as well as expressions of pleasure. Newly armed with separate measures of ‘wanting’ and ‘liking’, researchers tested this proposition. They found that electrically stimulating the brain’s dopamine system motivated animals to seek rewards like food, despite the fact that, as they become satiated, they show increasing distaste for the food through their facial expressions (Berridge & Valenstein, 1991). In another study, researchers studied a strand of mice with a genetic mutation that left them with very high For more Cengage Learning textbooks, visit www.cengagebrain.co.uk concentrations of dopamine in their brains. Compared to typical mice, these mutant mice not only ate and drank more, but also showed faster learning when rewarded with a Froot Loop, a piece of sweet breakfast cereal, suggesting that they attributed greater incentive salience ('wanting') to the cereal. Yet when researchers coded facial expressions when they ate the cereal, mutant mice and typical mice were no different, they liked it just the same (Pecina, Cagniard, Berridge, Aldridge, & Zhuang, 2003).The same pattern of results is found when drugs (like amphetamines) are injected into the brain's dopamine system (Wyvell & Berridge, 2000). Each of these experiments demonstrates 'wanting' without 'liking'. The opposite effect – 'liking' without 'wanting' – has also been demonstrated. Researchers injected a particular chemical neurotoxin into anesthetized rats, which selectively destroyed neurons in the brain's dopamine system but left all other neurons healthy and functioning. Once recovered from their operations, these rats were uninterested in food, water, or any other reward. They even voluntarily starved to death unless fed through a tube. Wanting was thus destroyed along with the dopamine neurons. But not so for liking. If sweet or bitter tastes were infused into the animals' mouths, they showed the same facial and bodily expressions as intact rats (Berridge & Robinson, 1998). So,paying closeattentiontolaboratoryrats' facialexpressions of pleasure and displeasure proved to be the key to unlocking the tight association between wanting and liking. From this new research, we can conclude that the brain's dopamine system accounts for 'wanting' a diverse array of natural and artificial incentives. Other brain systems – like the opiate system – have been found to underlie 'liking' (Berridge, 1999, 2003). PHOTOS COURTESY K. C. BERRIDGE. Displays of Liking in Humans and Animals These photos show human infant, primate, and rat affective displays to sweet and bitter tastes. Displays of 'liking' include tongue protrusions to a sweet taste (top photographs). Displays of 'disliking' include the gape (bottom photographs). (After Berridge, 1999, and Steiner et al., 2001)

of wanting and liking. Wanting is the anticipation of pleasure, as in the cravings that you experience when you think ahead to a delicious meal. Liking, by contrast, is the pleasure that you experience in the moment that you begin to eat that meal (Barbano & Cador, 2007). Liking something in the past usually contributes to wanting it in the future. This even occurs in short time spans, such as when a nibble of food whets your appetite for more. Through such processes, affective rewards (liking) can fuel incentive motivation (wanting). Wanting, in particular, appears to have evolved as a way for the brain to guide action in the future by keeping track of the good or bad consequences of past actions. If wanting is a kind of common currency for the value of diverse events, it makes sense that the brain should have a way of translating our different 'wants' into an equivalent 'monetary value'. There is indeed evidence that the brain may have a neural 'common currency' for reward. It is even possible that all rewards are desired precisely because they activate the same brain systems. This neural currency appears to be related to the level of activity within the brain's dopamine system (see Figure 10.2). The neurons of this system lie in the upper brain stem and send their axons through the nucleus accumbens and up to the prefrontal cortex. As their name implies, these neurons use the neurotransmitter dopamine to convey their message. The brain's dopamine system is activated by many kinds of natural rewards, or primary reinforcers, such as tasty food or drink or a desired sexual partner. The same Prefrontal cortex Nucleus accumbens VTA Figure 10.2 Dopamine Pathway Activated by Incentive Motivation. The brain's dopamine system is activated by rewarding stimuli and appears to underlie incentive motivation, or the feeling of 'wanting'. The neurotransmitter dopamine travels along the pathway from the ventral tegmental area (VTA) to the nucleus accumbens, and then on to the prefrontal cortex. (Adapted from NIDA, 2001) For more Cengage Learning textbooks, visit www.cengagebrain.co.uk INCENTIVE MOTIVATION AND REWARD neurons are also activated by many drugs that humans and animals find rewarding, such as cocaine, amphetamine, and heroin. The ability of virtually every reward, whether natural or artificial, to activate these neurons has led some psychologists to conclude that activity in this neural system constitutes the brain's common currency for tracking future rewards (Wise, 1982). Keep in mind, though, that the functioning of the brain's dopamine system is more closely linked to incentive motivation, or wanting. Rather than creating sensations of pleasure or liking per se, its activity appears to dispose individuals to want to repeat the event that caused the dopamine infusion, regardless of whether that event produces pleasure or displeasure. The Cutting Edge Research section describes the path-breaking studies that provide evidence for this distinction. Drug addiction and reward Addiction is a powerful motivation for some people. The craving for certain drugs, such as opiates (heroin or morphine), psychostimulants (amphetamine or cocaine) or synthetic street versions of these drugs, and certain other drugs (alcohol, nicotine), can become overwhelming (Leshner, 1997). Addicts may crave their drug so strongly that they will sacrifice job, family life and relationships, home, and even freedom to obtain it. Taking a drug once, or even once in a while, does not constitute addiction. Many Americans, for instance, have sampled at least one of the drugs just mentioned without becoming addicted. Even regular use (for example, regularly drinking wine with dinner) need not reflect addiction. Addiction occurs only when a pattern of compulsive and destructive drug-taking behavior has emerged; often the person compulsively craves the drug. Repeated drug use dramatically alters the incentive salience of the drug – creating pathological 'wanting'. What causes the transformation from trying out a drug, or engaging in social or recreational use, into addiction? Some drugs are especially powerful in their ability to produce addiction. Three major factors operate together to make psychoactive drugs more addictive than other incentives, although not all of these factors need be present for addiction to occur. The first is the ability of most addictive drugs to overactivate incentive systems in the brain. Because drugs act directly on brain neurons, they can produce levels of activity in the dopamine system that far surpass those produced by natural incentives. Euphoric drugs activate both pleasure (liking) and incentive (wanting) systems, perhaps because they activate both opiate and dopamine neural systems. Once experienced, the memory of such intense pleasure is a potent temptation to regain it again and again. But the memory of pleasure by itself would not be sufficient to produce addiction, at least for many people, without additional factors. The second factor is the ability

 of addictive drugs, if taken repeatedly, to produce unpleasant withdrawal syndromes. As a drug is taken again and again, the pleasure systems that it activates may become increasingly resistant to activation in an effort to regain their normal balanced state. This is, in part, the cause of tolerance, the need for a greater amount of a drug to achieve the same euphoria. In addition, after repeated exposure to the drug, the brain may activate processes that have consequences exactly opposite to those of the drug. These processes may help the brain remain in a balanced state when the drug is taken, but by themselves they are experienced as highly unpleasant. If the addict stops using the drug, the lack of activity in resistant pleasure systems and the activation of unpleasant drugopposite processes can produce withdrawal, an intensely aversive reaction to the cessation of drug use. This aversive state presents addicts with another motive to resume taking the drug, at least for as long as the withdrawal state lasts – typically several weeks. Finally, addictive drugs may produce permanent changes in brain incentive systems that cause cravings even after withdrawal is over. Repeated use of drugs like cocaine, heroin, or amphetamine, which activate the brain's dopamine systems, causes these neurons to become hyperactive or sensitized. Neural sensitization may be permanent, and it means that these dopamine neurons will be activated more highly by drugs and drug-related stimuli. Because the brain's dopamine system appears to mediate incentive motivation (wanting) more than pleasure (liking), its hyperactivation in addicts may cause exaggerated craving for the drug, even when drug experiences are no longer particularly positive (Robinson & Berridge, 2003). Neural sensitization lasts much longer than withdrawal. This may be why recovered addicts are in danger of relapse into drug use, even after they have completed detoxification programs. ª JOEL GORDON Addictive drugs can permanently change the brain's dopamine system, which creates a hyperactive craving for drugs. This is why objects and events associated with drug use continue to have strong incentive salience and produce cravings, even after recovering addicts have chosen to live drug-free. The combination of these factors sheds light on why psychoactive drugs, more than many other incentives, are able to produce addictions. These drugs directly activate brain pleasure and incentive systems to unmatched levels, produce withdrawal syndromes that drive a recovering addict back to the drug, and permanently hyperactivate the brain's dopamine system that causes drugs to be craved. This combination is hard to resist. People can also become pathologically dependent on things other than drugs – like gambling, food, shopping, work, even the Internet (Chou, Condron, & Belland, 2005). When engagement in such activity becomes compulsive and all-consuming, it is often called a behavioral addiction, even though it may not share all the same neurological properties of drug addictions. INTERIM SUMMARY l Incentive motivation (wanting something) is typically associated with pleasurable affect (liking that same something). Although some incentives – such as a sweet food when we are hungry – are powerful motivators by themselves, most incentives are established through learning. l The brain's dopamine system appears to underlie incentive motivation, or the experience of 'wanting'. Artificial activation of these neurons by drugs or electrical brain stimulation causes increased motivation for both natural and artificial incentives. l Drug addiction is a pattern of compulsive and destructive drug-taking behavior. Addictions to psychoactive drugs are difficult to overcome because of changes in drug tolerance, withdrawal symptoms, and neural sensitization. CRITICAL THINKING QUESTIONS 1 Research suggests that wanting and liking are separable psychological systems. Think of a time in your own life when you experienced wanting without liking (perhaps having to do with food). What caused the two to diverge? 2 Many addictive drugs change the brain's dopamine system, making these neurons hyperactive or sensitized. Because there is no known way to reverse these changes, it's reasonable to conclude that the brains of recovering addicts have been permanently altered. Knowing this, how can recovering addicts avoid relapse? How would you design an effective treatment program to prevent relapse?

HUNGER, EATING, AND EATING DISORDERS The control of hunger involves many of the same homeostatic concepts as thirst, but eating is much more complex than drinking. When we're thirsty, we generally need only water, and our thirst is directed toward anything that will provide it. But there are lots of different things to eat. We need to eat a number of different kinds of things (proteins, carbohydrates, fats, minerals) to be healthy. We need to select the proper balance of foods that contain these things. Evolution has given our brains ways of helping us select the foods we need (and avoid eating things that might poison us). Some of these ways involve the basic taste preferences we were born with. Others involve mechanisms for learning preferences for particular foods and aversions to others. Flavor is the most important factor in food preferences. Flavor contains both taste and odor components, but taste has been more important in human evolution.

Humans are born ‘programmed’ with likes and dislikes for particular tastes. Even infants respond to sweet tastes with lip-smacking movements and facial expressions indicative of pleasure (Steiner, 1979). They respond to bitter tastes by turning away and pulling their faces into expressions of disgust. Apes, monkeys, and a number of other species respond in the same way, as illustrated in the Cutting Edge Research section. Food manufacturers capitalize on our natural ‘sweet tooth’ to devise sweet foods that spur many people to overeat. Why do we find sweet foods and drinks so attractive? Evolutionary psychologists have suggested that it is because sweetness is an excellent ‘label’ that told our ancestors, foraging among unknown plants, that a ª VALERIA DINCU j DREAMSTIME.COM Evolutionary psychologists argue that sweet foods, like these pastries, are so compelling to us because ‘sweetness’ conveyed to our ancestors that a particular food was rich in sugar and calories. Of course, sugars were more rare in our ancestor’s environment than they are for us today. For more Cengage Learning textbooks, visit www.cengagebrain.co.uk HUNGER, EATING, AND EATING DISORDERS particular food or berry was rich in sugar, a class of digestible carbohydrate. Eating sweet foods is an excellent way to gain calories, and calories were not abundant in our evolutionary past. A similar labeling explanation has been advanced for our dislike of bitterness. The naturally bitter compounds that occur in certain plants can make those plants toxic to humans. Bitterness, in other words, is a label for a natural type of poison that occurs commonly. Ancestors who avoided bitter plants may have been more successful at avoiding such poisons (Rozin & Schulkin, 1990). A second way of developing food preferences is through an array of learning and social learning mechanisms. One of these is a preference based on the consequences of ingesting food with a particular taste. Experience with the nourishing consequences of a food leads to gradual liking for its taste through a process that is essentially a form of classical conditioning (Booth, 1991). Experience with other forms of taste-consequence pairings may also be the basis for developing preferences for tastes that are initially not pleasant, such as alcohol or coffee. In other words, the positive psychological or physical effects of alcohol or caffeinated coffee may cause us to develop preferences for these foods, even if we initially do not like their taste. The same kind of process can work in the opposite direction to produce strong dislike for a particular food. If your first sample of a tasty food or drink is followed sometime later by nausea or vomiting, you may find that the food is not tasty the next time you try it. The food hasn’t changed, but you have, because your new associative memories cause the food to subsequently be experienced as unwanted and unpleasant. This process is called conditioned aversion. Interactions between homeostasis and incentives Whatever particular foods we choose, it is clear that we must eat to maintain energy homeostasis. Body cells burn fuel to produce the energy required for the tasks they perform. Physical exercise causes muscle cells to burn extra fuel to meet the metabolic needs placed on them by energetic movement. By burning more fuel, they draw on stores of calories that have been deposited as body fat or other forms of ‘stored energy’. Even as you read this, the neurons of your brain are burning fuel to meet the metabolic needs created as they fire electrical impulses and make and release neurotransmitters. The main fuel used by these brain neurons is glucose, a simple sugar. Without fuel, neurons cannot work. Unfortunately, your brain doesn’t use more glucose when you ‘exercise it’ by thinking hard. Those neurons are always active and always consuming glucose, whether you are thinking hard or not. Concentrated thought or other psychological events may slightly alter the pattern of glucose use, but not the total amount.

368 CHAPTER 10 MOTIVATION Glucose is present in many fruits and other foods. It can also be manufactured by the liver out of other sugars or carbohydrates. Once you’ve eaten a meal, a great

deal of glucose will be absorbed into your bloodstream through the process of digestion. Even more will be created by your liver as it converts other forms of nutrients. In this way, a meal replenishes the fuel needed by your brain neurons and your body's other cells. Because our cells need fuel, we might expect hunger to be solely a homeostatic motivation controlled entirely by the need to keep sufficient sources of energy available. Indeed, homeostasis is the dominant principle operating in the control of hunger. Deficits in available fuels can trigger hunger, and surpluses can inhibit it. But even though homeostasis is crucial to understanding the control of hunger, incentive factors are equally important. That is, we want to eat perhaps as much as we need to eat (Lowe & Butryn, 2007). So we can't fully understand hunger unless we look at the interaction between homeostasis and incentives. The importance of interactions between homeostatic drive reduction and the taste and other incentive stimuli of food was made clear by a classic experiment by Miller and Kessen (1952). These investigators trained rats to run down a short path for a milk reward. In one case, the rats received milk as a reward in the ordinary way: They drank it. In the other case, the rats received exactly the same amount of milk, but in a more direct way: The milk was gently pumped into their stomachs through a tube passed into an artificial opening, or fistula, that had been implanted weeks before. Both of these rewards provided exactly the same number of calories. Both reduced the rats' fuel deficit to the same degree. But the rats learned to run for the milk reward much better when they were allowed to drink it. The milk was not a powerful motivator when it was pumped directly into the stomach, even though it reduced hunger just as well as when it went into the mouth. The rats needed to both taste the reward and have it reduce hunger. The importance of such interactions between oral incentives and drive reduction has been demonstrated in many ways since that original experiment (Toates, 1986). Food that bypasses the normal route of voluntary tasting and swallowing is not strongly motivating for either animals or humans. For example, people who are fed entirely by means of intravenous or intragastric infusions of nutrients often find these 'meals' unsatisfying. They may feel an intense desire to have some food that they can put into their mouths – even if they are required to spit it out again after chewing it. The strong desire for oral stimulation – above and beyond the satisfaction of caloric needs – is also reflected in our widespread use of artificial sweeteners, which provide flavor without calories. Food incentives, in the form of the pleasant sensory experience involved in eating palatable foods and drinks, thus are as crucial to appetite as caloric drive reduction. Learning is also an important part of the interaction between physiological hunger signals and the incentive stimuli of eating. Dramatic demonstrations can be seen in animals in which the act of eating is uncoupled from the ordinary caloric consequences by the implantation of a stomach fistula, which allows food to leave the stomach as well as to be put into it. If the fistula cap is removed, whatever is eaten will fall out rather than be digested. This is called sham feeding because the meal is a sham in the sense that it provides no calories. Sham-fed animals eat normal amounts and then stop. Why do they stop rather than continue eating? The answer becomes clear if one observes food intake during subsequent meals: The animals gradually increase the amount eaten as they learn that the meal conveys fewer calories than it once did (Van Vort & Smith, 1987). If the fistula cap is replaced so that everything is digested as it normally would be, the animals eat the 'too large' amount for their next few meals. Gradually, their meal size declines to normal levels as they learn that the food apparently is rich in calories once again. These observations have led to the hypothesis of conditioned satiety – that the fullness we feel after a meal is at least in part a product of learning (Booth, 1987). Humans also are capable of conditioned satiety. In one experiment, people were asked to eat several meals of a distinctive food that was rich in calories and of another food that was low in calories. Later, when the participants were again given the two foods, which were apparently the same as before but with the caloric content made equal, they found the food that had originally been higher in calories more satiating (Booth, 1991). Typically, ingesting sweets provides calories and therefore energy. But when this association is broken, as it is when foods are prepared with artificial sweeteners, like saccharin, our bodies compensate by gradually increasing caloric intake, leading to increases in body weight and even obesity – hardly the result those who eat ‘sugar-free’ seek. This ironic effect of consuming artificial sweeteners is thought to occur because their sham incentive value interferes with physiological homeostatic processes (Swithers & Davidson, 2008). A final form of interaction between food incentives and homeostatic drive is the phenomenon called alliesthesia (Cabanac, 1979), in which food (especially sweet food) tastes better when one is hungry. More generally, alliesthesia means that any external stimulus that corrects an internal trouble is experienced as pleasurable. For example, when people are asked to rate the palatability of sweet drinks either after a meal or after several hours without food, they give higher palatability ratings to the same drink when they are hungry than when they have recently eaten. Physiological hunger cues You may have noticed that when you are hungry your stomach sometimes growls. At such moments, the

stomach walls are engaged in muscular contractions, creating the burbling movements of its contents that you hear. Stomach contractions are most frequent when you are hungry and likely to feel that your stomach is empty. The association of these contractions with feelings of hunger led early investigators to hypothesize that pressure sensors in the stomach detect emptiness and trigger both contractions and the psychological experience of hunger. Later, psychologists and physiologists found that this coincidence is really just that – a coincidence. Stomach sensations from contractions are not the real cause of hunger. In fact, people who have had their stomachs surgically removed for medical reasons, so that food passes directly to the intestines, can still have strong feelings of hunger. The stomach does have receptors that are important to changes in hunger, but these receptors are primarily chemical in nature. They have more to do with feelings of satiety than with feelings of hunger. They are activated by sugars and other nutrients in stomach contents and send a neural signal to the brain. The physiological signal for hunger is more directly related to the real source of calories for neurons and other cells: levels of glucose and other nutrients in the body. The brain itself is its own sensor for deficiencies in available calories. You may remember that neurons in the brain use glucose as their principal source of energy. Neurons in particular parts of the brain, especially the brain stem and hypothalamus, are especially sensitive to glucose levels. When the level falls too low, the activity of these neurons is disrupted. This signals the rest of the brain, producing hunger. Such hunger can be produced artificially in laboratory animals even just after a meal. If chemicals that prevent neurons from burning glucose as a fuel are infused into an animal’s brain, the animal will suddenly seek out food. Its brain has been fooled into sensing a lack of glucose, even though glucose was actually present, because the neurons have been disrupted in the same way as they are when glucose is low. Peripheral signals To some degree, hunger is what we feel when we have no feeling of satiety. As long as caloric food is in our stomach or intestine, or calorie stores are high within our body, we feel relatively sated. When these decline, hunger ensues. The control of hunger is therefore the reverse of the control of satiety. Many physical systems contribute to the feeling of satiety after a meal. The first system is made up of the parts of the body that process food first: the stomach and intestine. Both the physical expansion of the stomach and the chemicals within the food activate receptors in the stomach’s walls. These receptors relay their signal to the brain through the vagus nerve, which carries signals from many other body organs as well. A second kind of satiety message comes from the duodenum, the part of the For more Cengage Learning textbooks, visit www.cengagebrain.co.uk HUNGER, EATING, AND EATING DISORDERS intestines that receives food directly from the stomach. This signal is sent to the brain as a chemical rather than through a nerve. When food reaches the duodenum, it causes it to release a hormone (cholecystokinin, or CCK) into the bloodstream. CCK helps promote physiological digestion, but it also has a psychological consequence. It travels through the blood until it reaches the brain, where it is detected by special receptors. This produces feelings of satiety. Hungry animals can be fooled into false satiety if microscopic amounts of CCK are infused into their brains shortly after they have begun a meal (Smith & Gibbs, 1994). Perhaps surprisingly, the brain's most sensitive signal of nutrient availability comes from neuronal receptors that are separate both from the brain and from food: neuronal receptors in the liver (Friedman, 1990). Receptors in the liver are highly sensitive to changes in blood nutrients after digestion. These signals are also sent to the brain through the vagus nerve. A hungry animal will stop eating almost immediately after even a tiny amount of nutrients are infused into the blood supply that goes directly to the liver. Why should the brain rely on nutrient signals from the liver rather than on its own detectors? The answer may be that the liver can more accurately measure the various types of nutrients used by the body. The brain detects chiefly glucose, but other forms of nutrients, such as complex carbohydrates, proteins, and fats, can be measured, stored, and sometimes converted into other nutrients by the liver. Its role as a general 'currency exchange' for various nutrients may allow the liver to make the best estimate of the total energy stores available to the body. Integration of hunger signals Signals for hunger and satiety are processed by the brain in two stages to produce the motivation to eat. First, signals from hunger receptors in the brain itself and satiety signals relayed from the stomach and liver are added together in the brain stem to detect the overall level of need (Grill & Kaplan, 1990). This 'integrated hunger assessment' is also connected in the brain stem to the sensory neural systems that process taste. Taste neurons in the brain stem may change their responsiveness during some forms of hunger and satiety (Scott & Mark, 1986), which may be part of the reason that food tastes more palatable when we are hungry. To become the conscious experience we know as hunger, and to stimulate the seeking of food, the hunger signal of the brain stem must be processed further in the forebrain. A key site for this processing is the hypothalamus (see Figure 10.3). Hunger is affected in two dramatically different ways by manipulations of two parts of the hypothalamus: the lateral hypothalamus (the parts on each side) and the ventromedial hypothalamus (the lower ['ventral'] and middle ['medial'] portion). Destruction of

370 CHAPTER 10 MOTIVATION Pituitary Hypothalamus Figure 10.3 The Hypothalamus and Pituitary. the lateral hypothalamus produces an apparent total lack of hunger, at least until the rest of the brain recovers and compensates (Teitelbaum & Epstein, 1962). This phenomenon is called the lateral hypothalamic syndrome. Animals that have had small lesions made in their lateral hypothalamus may simply ignore food. They may even reject it as though it tasted bad (for example, they may grimace and vigorously spit it out). Unless they are fed artificially, they will starve to death. Nearly the exact opposite pattern of behavior is seen with the ventromedial hypothalamic syndrome. Lesions of the ventromedial hypothalamus produce extreme appetites. Animals with such lesions eat voraciously and consume large quantities of food, especially if it is palatable. Not surprisingly, they gain weight until they become quite obese, up to double their normal body weight (King, 2006). Other manipulations of these brain sites also appear to change hunger. For example, electrical stimulation of the lateral hypothalamus produces overeating: the

exact opposite of a lesion of the lateral hypothalamus (and the same effect as a lesion of the ventromedial hypothalamus). An animal with a stimulating electrode in its lateral hypothalamus may begin to look for food and eat as soon as the stimulation begins – and to stop eating once it ends. Conversely, stimulation of the ventromedial hypothalamus will stop a hungry animal's ordinary eating. Neurochemical stimulation of the hypothalamus works in similar ways. For example, certain compounds such as neuropeptide Y, or opiate drugs such as morphine, can stimulate feeding when they are injected into the ventromedial hypothalamus. These drugs may temporarily stimulate hunger or make food taste better. Other drugs, such as amphetamines, can halt feeding when injected into parts of the lateral hypothalamus. For more Cengage Learning textbooks, visit www.cengagebrain.co.uk ª RICHARD HOWARD Damage to the ventromedial hypothalamus produces overeating and obesity. Many prescription diet drugs are chemically similar to amphetamines. Such drugs might inhibit appetite by acting on neurons in the hypothalamus. Around 1960, when the importance of the lateral hypothalamus and ventromedial hypothalamus to hunger were discovered, psychologists tended to view these sites simply as hunger or satiety centers. Since then, it has become clear that the concepts of 'hunger center' or 'satiety center' are too simplistic, for a number of reasons. One is that these sites are not the sole centers for hunger or satiety in the brain. They interact with many other brain systems to produce their effects. In fact, some of the same effects can be produced by manipulating related brain systems instead of the hypothalamus. For example, many of the effects of manipulating the lateral hypothalamus can be duplicated by manipulating the brain's dopamine system, which simply passes through the hypothalamus. Like lateral hypothalamic lesions, lesions in this dopamine-containing bundle of axons eliminate feeding. In fact, many early studies of lateral hypothalamic lesions actually destroyed both the dopamine systems and the neurons in the lateral hypothalamus itself. Conversely, the elicitation of feeding by electrical stimulation and by many drugs also depends partly on activation of the brain's dopamine system. Thus, rather than just one or two centers, many neuroanatomical and neurotransmitter systems are involved in appetite and satiety. One consequence of having many neural systems for appetite is that it is not possible to abolish eating by destroying just one site. Even in animals with lateral hypothalamic lesions, appetite will return eventually. If the rats are artificially fed for several weeks or months after the lesion, they will begin to eat again, but they will eat only enough to maintain their lower body weight. They seem to have reached homeostasis at a lower set point. In fact, rats can be 'protected' from the usual loss of eating that would follow a lateral hypothalamic lesion

420 Freely fed group Body weight (grams) 360 Starved group 300 –10 10 30 Before lesion After lesion Days Figure 10.4 Body Weight and the Lateral Hypothalamus. Before lesioning of the lateral hypothalamus, one group of rats was starved and another group was allowed to feed freely. After surgery, the starved animals increased their food intake and gained weight while the freely fed group lost weight. Both groups stabilized at the same weight level. (After Powley & Keesey, 1970) if they are put on a diet before the lesion that lowers their body weight (see Figure 10.4). This indicates that hypothalamic lesions don't actually destroy hunger. Instead, they may raise or lower the homeostatic set point for body weight that ordinarily controls hunger. Changing the set point is like resetting a thermostat: The system attempts to achieve the new body weight. The effect of ventromedial hypothalamic lesions also conforms to this idea. Animals with those lesions do not gain weight infinitely. Eventually they stop at a new, obese body weight. At that point, they eat only enough to maintain the new set point. But if they are put on a diet and drop below that 520 Body weight (grams) 400 280 Starved Force-fed Days 40 120 200 280 Figure 10.5 Effects of Forced

Feeding and Starvation on Rats with VMH Lesions. Following lesioning of the ventromedial hypothalamus, the rat overeats and gains weight until it stabilizes at a new, obese level. Forced feeding or starvation alters the weight level only temporarily; the rat returns to its stabilized level. (After Hoebel & Teitelbaum, 1966) For more Cengage Learning textbooks, visit www.cengagebrain.co.uk HUNGER, EATING, AND EATING DISORDERS set point, they will resume overeating in order to regain that body weight when they are finally given the opportunity (see Figure 10-5). Once they regain that level of obesity, they will halt once again. Obesity We have emphasized homeostatic processes in hunger, but eating behavior shows several departures from homeostasis. Some people's body weight is not as constant as the homeostatic viewpoint suggests. The most frequent deviation from homeostatic regulation of eating – at least for humans – is obesity. Obese is defined as being 30 percent or more in excess of one's appropriate body weight. Currently in the United States, obesity is considered an epidemic, with roughly 34 percent of U.S. adults meeting the criteria, a percentage that has nearly doubled in the past twenty years. Although the percentage of obese adults in Europe is generally far lower (18% across ten European countries; Peytremann-Bridevaux, Faeh, & Santos-Eggimann, 2007), the link between obesity and health problems makes it a pressing societal concern. The prevalence of obesity also varies among different groups. Physical obesity occurs about equally in both sexes, but the psychological perception of being overweight is more common among women. More than 50 percent of American women, compared with more than 35 percent of men, consider themselves overweight (Brownell & Rodin, 1994; Horm & Anderson, 1993). Obesity is a major health hazard. It contributes to a higher incidence of diabetes, high blood pressure, and heart disease. As if this were not bad enough, obesity can also be a social stigma, as obese people are often perceived as being indulgent and lacking in willpower (Crandall, 1994; Crocker, Cornwell, & Major, 1993). This allegation can be most unfair because, as we will see, in many cases obesity is due to genetic factors rather than overeating. Given the problems associated with obesity, it is not surprising that each year millions of people spend billions of dollars on diets and drugs to lose weight. Most researchers agree that obesity is a complex problem that can involve metabolic, nutritional, psychological, and sociological factors. Obesity probably is not a single disorder but a variety of disorders that all have fatness as their major symptom (Rodin, 1981). Asking how one becomes obese is like asking how one gets to Paris – there are many ways to do it, and which one you 'choose' depends on where you

372 CHAPTER 10 MOTIVATION are coming from (Offir, 1982). In what follows, we will divide the factors that lead to weight gain into two broad classes: (1) genetics and (2) calorie intake (overeating). Roughly speaking, people may become obese because they are genetically predisposed to metabolize nutrients into fat, even if they don't eat more than other people (metabolic reasons), or because they eat too much (for psychological or sociocultural reasons). Both factors may be involved in some cases of obesity, and in other cases genetics or overeating alone may be the culprit. Genetic factors It has long been known that obesity runs in families. In families in which neither parent is obese, only about 10 percent of the children are obese; if one parent is obese, about 40 percent of the children are also obese; and if both parents are obese, approximately 70 percent of the children are also obese (Gurney, 1936). These statistics suggest a biological basis of obesity, but other interpretations are possible – for example, perhaps the children are simply imitating their parents' eating habits. Recent findings, however, strongly support a genetic basis for obesity. Twin studies One way to get evidence about the role of genetics in obesity is to study identical twins. Because identical twins have the same genes, and

because genes supposedly play a role in weight gain, identical twins should be alike in their patterns of weight gain. In one experiment, 12 pairs of identical twins (all males) agreed to stay in a college dormitory for 100 days. The intent of the experiment was to get the twins to gain weight. Each man ate a diet that contained 1,000 extra calories per day. For men, 1,000 extra calories is the rough equivalent of eating four very large meals a day, instead of three regular meals. Also, the men's physical activity was restricted. They were not allowed to exercise and instead spent much of their time reading, playing sedentary games, and watching television. By the end of the 100 days, all of the men had gained weight, but the amount gained ranged from 9 to 30 pounds. However – and this is the key point – there was hardly any variation in the amount gained by the members of each pair of twins (the variation occurred between pairs of twins). In other words, identical twins gained almost identical amounts. Moreover, identical twins tended to gain weight in the same places. If one member of a pair of twins gained weight in his middle, so did the other; if one member of another pair of twins gained weight on his hips and thighs, so did the other (Bouchard, Lykken, McGue, Segal, & Tellegen, 1990). These results make it clear that both calorie intake and genetics contribute to weight gain. The fact that all the men in the study gained weight shows that increased calories translates into increased weight, which is hardly surprising. The fact that the amount of weight gained varied from one pair of twins to another but did not vary within a pair of twins suggests that genetic factors determine how much we gain when we increase our calorie intake. The results also make it clear why we should not assume that obese people necessarily eat more than nonobese people. Despite eating roughly the same amount (1,000 extra calories), the amount of weight gained by different pairs of twins varied. This difference seems to arise from how their bodies metabolized the extra calories. Some people's bodies tend to convert a larger proportion of calories into fat stores, and others are likely to burn off the same calories through different metabolic processes, regardless of how much is eaten (Ravussin et al., 1988). A critic might object to making too much of the study just described. Identical twins not only have identical genes but also grow up in very similar environments. Perhaps environmental factors were responsible for the identical twins being alike in weight gain. We need to study identical twins who have been reared apart and see how similar the members of a pair are in weight gain. This was done in a study conducted in Sweden (Stunkard et al., 1990). The researchers studied the weights of 93 pairs of identical twins reared apart, as well as that of 153 pairs of identical twins reared together. Members of a pair of twins reared apart were found to be remarkably similar in weight; indeed, they were as similar in weight as members of pairs of twins reared together. Clearly, genes are a major determinant of weight and weight gain. Fat cells Given that genes play a role in weight gain, we want to know some details of that role. In particular, what are the digestive and metabolic processes that are affected by genes and that mediate weight gain? One answer involves fat cells, where all body fat is stored. There are between 30 billion and 40 billion fat cells in the bodies of most normal adults, but the degree of excess weight carried by ordinary American adults varies by more than the 25 to 33 percent this figure would suggest. The additional variation comes from the size, rather than the mere number, of fat cells: The more calories one eats and fails to burn off, the larger existing fat cells become. In one study, obese participants were found to have three times as many fat cells as normal participants (Knittle & Hirsch, 1968). In other studies, researchers have shown that rats with double the usual number of fat cells tend to be twice as fat as control rats. And when researchers cut some of the fat cells out of young rats so that they had only half as many fat cells as their littermates, those rats grew up to be only half as fat as their littermates (Faust, 1984; Hirsch & Batchelor, 1976). Because there is a link between genes and the number of fat cells, and another link between the number of fat cells and obesity, through this chain, genes are connected to obesity.

Dieting and set points When people take diet drugs, a variety of things can happen. The drug might suppress appetite directly, which would reduce the feeling of hunger. Another drug might suppress the set point – the point at which body weight is set and that the body strives to maintain – rather than suppress appetite directly. For example, it has been suggested that some diet drugs have this effect (Stunkard, 1982), such as fenfluramine, no longer on the market due to its link to heart disease. Such an effect would be equivalent to direct appetite suppression as long as body weight was higher than the lowered set point. Once body weight fell to the lower level, appetite would return to just the degree needed to remain at that weight. When a person stopped taking the drug, the set point would return to its higher level, and the person would regain the weight that had been lost. Finally, some drugs, such as nicotine, may help people lose weight by elevating the metabolic rate of cells, causing them to burn more calories than they ordinarily would. One reason that the set point hypothesis has become popular among psychologists is the strong tendency for obese adults, both humans and animals, to return to their original body weight after ceasing dieting. In contrast to the young rats just described, even surgical removal of fat deposits by liposuction appears not to produce permanent weight loss when it is performed on adult rats: The adults regain the fat elsewhere. This also appears to be true of liposuction performed on obese human adults (Vogt & Belluscio, 1987). Some investigators have suggested that once adult levels of fat tissue have been reached, they are maintained at that level. The brain may detect changes in the level of body fat and influence hunger accordingly (Weigle, 1994). For example, an ‘obesity gene’ in mice is thought to control the ability of fat cells to produce a chemical ‘satiety signal’ (Zhang et al., 1994). Mice that lack this gene become obese. Ordinarily, the more body fat one has, the more a satiety signal is released into the blood. Whether human obesity involves a disruption in this satiety factor or gene is not yet known. But the possibility that the level of fat stores is kept constant may help explain why some obese people find it difficult not to regain weight that they lost through dieting. In sum, there are various routes by which genes can be responsible for excessive weight gain, including having many and large fat cells, having a high set point, and having a low metabolic rate. Overeating Although physiological factors such as fat regulation and metabolic rate are important determinants of body weight, there is no question that overeating can also cause obesity. The psychological factors that contribute to overeating include the breakdown of conscious restraints and emotional arousal.   Breakdown of conscious restraints Some people stay obese by going on eating binges after dieting. An obese man may break a two-day diet and then overeat so much that he eventually consumes more calories than he would have, had he not dieted at all. Because the diet was a conscious restraint, the breakdown of control is a factor in increased calorie intake. To gain a more detailed understanding of the role of conscious restraints, researchers have developed a questionnaire that asks about diet, weight history, and concern with eating (for example, How often do you diet? Do you eat sensibly in front of others, yet overeat when alone?). The results show that almost everyone – whether thin, average, or overweight – can be classified into one of two categories: people who consciously restrain their eating and people who do not. In addition, regardless of their actual weight, the eating behavior of restrained eaters is closer to that of obese individuals than to that of unrestrained eaters (Herman & Polivy, 1980; Ruderman, 1986). A laboratory study shows what happens when restraints are dropped. Restrained and unrestrained eaters (both of normal weight) were required to drink either two milkshakes, one milkshake, or none; they then sampled several flavors of ice cream and were encouraged to eat as much as they wanted (Herman & Mack, 1975). The more milkshakes the unrestrained eaters were required to drink, the less ice cream they consumed later. In contrast, the restrained eaters who had been preloaded with two milkshakes ate more ice cream than did those who drank one milkshake or none. Thus, individuals who are trying to restrain their eating by ignoring their ordinary impulse to eat more may also come to ignore the feelings of satiety that would ordinarily halt their desire to eat. Ironically, then, this is why conscious efforts to diet often backfire. Emotional arousal Overweight individuals often report that they tend to eat more when they are tense or anxious, and experimental results support these reports. Obese participants eat more in a high-anxiety situation than they do in a low-anxiety situation, but normal-weight participants eat more in situations of low anxiety (McKenna, 1972). Other research indicates that any kind of emotional arousal seems to increase food intake in some obese people. In one study, overweight and normal-weight participants saw a different film in each of four sessions. Three of the films aroused various emotions: one was distressing, one amusing, and one sexually arousing. The fourth film was a boring travelogue. After viewing each of the films, the participants were asked to taste and evaluate different kinds of crackers. The obese participants ate significantly more crackers after viewing any of the arousing films than they did after seeing the travelogue. Normal-weight individuals ate the same amount of crackers regardless of which film they had seen (White, 1977).

 The ability of emotional stress to elicit eating has been observed in other animals, too. This may mean that stress can activate basic brain systems that, under some conditions, result in overeating (Rowland & Antelman, 1976). Dieting and weight control Although genetic factors may limit the amount of weight we can comfortably lose, overweight people can still lose weight by following a weight-control program. For a program to be successful, though, it must involve something other than just extreme dieting. Limitations of dieting Unfortunately, most dieters are not successful, and those who succeed in shedding pounds often gain weight again after ceasing dieting. This state of affairs seems to be partly due to two deep-seated reactions to a temporary deprivation of food (which is what a diet is). The first reaction, as we’ve seen, is that deprivation per se can lead to subsequent overeating. In some experiments, rats were first deprived of food for four days, then allowed to feed until they regained their normal weights, and finally allowed to eat as much food as they wanted. These rats ate more than control rats with no history of deprivation. Thus, prior deprivation leads to subsequent overeating, even after the weight lost as a result of the deprivation has been regained (Coscina & Dixon, 1983). The second reaction of interest is that deprivation decreases metabolic rate, and as you may recall, the lower one’s metabolic rate, the fewer calories expended, and the higher one’s weight. Consequently, the calorie reduction during dieting is partly offset by the lowered metabolic rate, making it difficult for dieters to meet their goals. The reduced metabolic rate caused by dieting may also explain why many people find it harder and harder to lose weight with each successive diet: The body responds to each bout of dieting with a reduction in metabolic rate (Brownell, 1988). Both reactions to dieting – binge eating and lowered metabolic rate – are understandable in evolutionary terms. Until very recently in human history, whenever people experienced deprivation it was because of a scarcity of food in the environment. One adaptive response to such scarcity is to overeat and store in our bodies as much food as possible whenever it is available. Natural selection may have favored the ability to overeat following deprivation, which explains the overeating reaction. A second adaptive response to a scarcity of food in the environment is for organisms to decrease the rate at which they expend their limited calories, so natural selection may have favored the ability to lower one's metabolic rate during deprivation. This explains the second reaction of interest. Over the millennia, these two reactions have served our species well in times of famine, but once famine is not a concern – as in most economically developed For more Cengage Learning textbooks, visit www.cengagebrain.co.uk countries today – they prevent obese dieters from losing weight permanently (Polivy & Herman, 1985). Weight control programs To lose weight and keep it off, it seems that overweight individuals need to establish a new set of permanent eating habits (as opposed to temporary dieting) and engage in a program of exercise. Some support for this conclusion is provided by the following study, which compared various methods for treating obesity (Craighead, Stunkard, & O'Brien, 1981; Wadden et al., 1997). For six months, obese individuals followed one of three treatment regimens: (1) behavior modification of eating and exercise habits, (2) drug therapy using an appetite suppressant, and (3) a combination of behavior modification and drug therapy. Participants in all three treatment groups were given information about exercise and extensive nutritional counseling, including a diet of no more than 1,200 calories per day. Participants in the behavior modification groups were taught to become aware of situations that prompted them to overeat, to change the conditions associated with their overeating, to reward themselves for appropriate eating behavior, and to develop a suitable exercise regimen. In addition to the three treatment groups, there were two control groups: One consisted of participants waiting to take part in the study, and the other of participants who saw a physician for traditional treatment of weight problems. Table 10.1 presents the results of the study. The participants in all three treatment groups lost more weight Table 10.1 Weight loss following different treatments Weight loss in pounds at the end of six months of treatment and on a follow-up one year later. Participants in the two control groups were not available for the one-year follow-up. (L. W. Craighead, A. J. Stankard, & R. M. O'Brien (1981) 'Behavior Therapy and Pharmcotherapy for Obesity', in Archives of General Psychiatry, 38:763–768. Copyright © 1981 by the American Medical Association.) Weight loss after treatment Weight loss one year later Treatment groups Behavior modification only 24.0 19.8 Drug therapy only 31.9 13.8 Combined treatment 33.7 10.1 Control groups Waiting list 2.9 (gain) – Physician office visits 13.2 –

than the participants in the two control groups, with the group combining behavior modification and drug therapy losing the most weight and the behaviormodification-only group losing the least. However, during the year after treatment, a striking reversal developed. The behavior-modification-only group regained far less weight than the two other treatment groups; these participants maintained an average weight loss of nearly 20 pounds by the end of the year, whereas the weight losses for the drug-therapy-only group and the combinedtreatment group regained roughly two-thirds of the weight they had initially lost. What caused this reversal? An increased sense of selfefficacy or self-control may have been a factor. Participants who received the behavior-modification-only treatment could attribute their weight loss to their own efforts, thereby strengthening their resolve to continue controlling their weight after the treatment ended. Participants who received an appetite suppressant, on the other hand, probably attributed their weight loss to the medication and did not develop a sense of self-control. Another possible factor stems from the fact that the medication had decreased the participants' feelings of hunger, or temporarily lowered their set point, and consequently participants in the drug-therapy-only group and the combined-treatment group may not have been sufficiently prepared to cope with the increase in hunger they felt when the medication was stopped. Anorexia and bulimia Although obesity is the most common eating problem, the opposite problem has also surfaced in the form of anorexia nervosa and bulimia. Both of these disorders involve a pathological desire not to gain weight and disproportionately strike women. Anorexia nervosa is an eating disorder characterized by extreme, self-imposed weight loss – at least 15 percent of the individual's minimum normal weight. Some anorexics in fact weigh less than 50 percent of their normal weight. Despite the extreme loss of weight and the resulting problems, the typical anorexic denies that there is a problem and refuses to gain weight. In fact, anorexics frequently think that they look too fat. For females to be diagnosed as anorexic, in addition to the weight loss, they must also have stopped menstruating. The weight loss can lead to a number of dangerous side effects, including emaciation, susceptibility to infection, and other symptoms of undernourishment. These side effects can lead to death. Anorexia is relatively rare. Its prevalence across Western Europe and the United States is about 0.3 percent. However, this represents more than a doubling since the 1950s, although the frequency seems to have stabilized since the 1970s (Hoek & van Hoeken, 2003). Anorexia is 20 times more likely to occur in women than For more Cengage Learning textbooks, visit www.cengagebrain.co.uk HUNGER, EATING, AND EATING DISORDERS in men, and the majority of anorexics are young women between their teens and their thirties. Typically, anorexics are entirely focused on food, carefully calculating the amount of calories in anything they might consume. Sometimes this concern reaches the point of obsession, as when one anorexic commented to her therapist, 'Of course I had breakfast; I ate my Cheerio [a single small piece of breakfast cereal]', or when another said, 'I won't lick a postage stamp – one never knows about calories' (Bruch, 1973). The obsession with food and possible weight gains leads some anorexics to become compulsive exercisers as well, sometimes exercising vigorously several hours a day (Logue, 1991). Bulimia is an eating disorder characterized by recurrent episodes of binge eating (rapid consumption of a large amount of food in a discrete period of time), followed by attempts to purge the excess by means of vomiting or laxatives. The binges can be frequent and extreme. A survey of bulimic women found that most women binged at least once per day (usually in the evening) and that an average binge involved consuming some 4,800 calories (often sweet or salty carbohydrate foods). However, because of the purges that follow the binges, a bulimic person's weight may stay relatively normal, which allows bulimics to keep their eating disorder hidden. But this behavior can have a high physiological cost. Vomiting and use of laxatives can disrupt the balance of potassium in the body, which can result in problems like dehydration, cardiac arrhythmias, and urinary infections. Like anorexia, bulimia primarily afflicts young women. But bulimia is somewhat more frequent than anorexia, with an estimated 1.1 percent meriting a full diagnosis in Western Europe and the United States, and up to 5.4 percent showing at least some symptoms (Hoek & van Hoeken, 2003). Researchers have suggested a variety of causes for anorexia and bulimia, including social, biological, and personality or family factors. It is probably necessary for several of these factors to occur together for any individual to develop an eating disorder. Sociocultural causes Many psychologists have proposed that social and cultural factors play major roles in anorexia and bulimia. In particular, they point to Western society's emphasis on thinness in women. This emphasis has increased markedly in the past 40 years, which fits with the observation that the incidence of eating disorders has also increased during that period. An indication of this trend is the change in what people regard as a 'perfect' woman's figure. The photos place Jayne Mansfield, who was widely thought to have an ideal figure in the 1950s, next to a photo of actress Nicole Kidman, who reflects today's ideal. Kidman is clearly much thinner than Mansfield, especially in the hips and thighs, the region of the body with which most women experience deep dissatisfaction.

But how exactly do media images of the ‘ideal’ female body sink in and account for high rates of disordered eating? Insight into this process is offered by objectification theory, a sociocultural account of how being raised in a culture that sexually objectifies the female body (both within the visual mass media and within actual interpersonal encounters) fundamentally alters girls’ and women’s self-views and well-being (Fredrickson & Roberts, 1997). Sexual objectification occurs any time a person is treated first and foremost as a body valued for its sexual use to (or consumption by) others. Sexual objectification is a dehumanizing form of interpersonal regard. It reduces the targeted person’s full humanity to the status of an object for the observer’s benefit. The first psychological consequence of repeated exposure to cultural practices of sexually objectifying female bodies, the theory holds, is that girls and women learn to internalize an objectifying observer’s perspective on their own body. This preoccupation with physical appearance is termed self-objectification (see the Concept Review table). In brief, self-objectification means that a person thinks about and values her own body more from a third-person perspective, focusing on observable body attributes (‘How do I look?’), rather than from a first-person perspective, focusing on privileged, or unobservable body attributes (‘How do I feel?’). Self-objectification has been shown to be both a relatively stable trait – with some girls and women self-objectifying more than others – and a temporary state – with some situations pulling for self-objectification more than others (Breines, Crocker & Garcia, 2008). Self-objectification has been shown to affect women of various ethnic backgrounds (Hebl, King, & Lin, 2004) as well as gay men (Martins, Tiggemann & Kirkbride, 2007). Objectification theory claims that self-objectification causes a range of psychological and emotional reactions. First and foremost, self-objectification leads to a form of self-consciousness characterized by vigilant monitoring of the body’s outward appearance. This preoccupation with appearance has been shown to disrupt a person’s stream of consciousness and thereby limit the mental resources that she can devote to other activities (Quinn, Kallen, Twenge & Fredrickson, 2006). It also creates a predictable set of emotional reactions, including increased shame and anxiety and diminished positive emotions and sexual pleasure. Over time, these emotional reactions can accumulate and compound, which explains why certain health and mental health problems disproportionately afflict girls and women. Chief among these problems are various forms of disordered eating, which include anorexia and bulimia, as well as restrained eating (dieting) more generally. But the theory doesn’t stop there. It accounts for gender differences in depression and sexual dysfunction as well (see the Concept Review Table). Jayne Mansfield (left) represented the perfect female figure for the 1950s, whereas Nicole Kidman (right) represents the perfect female figure today.

CONCEPT REVIEW TABLE Objectification theory Objectification theory outlines the causes, features, and consequences of self-objectification, defined as a preoccupation with appearance characterized by vigilant appearance monitoring. (After Fredrickson et al., 1998) Theory Outline Theory Elements Causes Cultural practices of sexual objectification in media messages in interpersonal encounters Features Internalized observer’s perspective on self Vigilant appearance monitoring Disrupted mental resources Consequences Psychological experiences increased shame increased anxiety decreased positive emotions insensitivity to bodily cues Health and mental health risks disordered eating depression sexual dysfunction Risks for these three problems – disordered eating, depression, and sexual dysfunction – not only coincide with gender but also coincide with age. Intriguingly, the risks change in step with observable life-course changes in the female body: They first emerge for girls in early adolescence and lessen for women in late middle age. Objectification theory notes that women are most targeted for sexual objectification during their years of reproductive potential and uses this fact to explain these changing risk patterns over the life course. Initial survey studies with college women showed that self-objectification, feelings of shame about one's body, and disordered eating were all associated with one another (Noll & Fredrickson, 1998). But those are simply correlations. How do we know that self-objectification is a cause of disordered eating, and not just a consequence or a symptom? A series of clever laboratory experiments provided the necessary evidence. In these studies, participants – male and female college students – believed they were partaking in a study on consumer decisions. Under this guise, they sampled various products and rated how those products made them feel. When it came time to try on and evaluate a garment (in a private 'dressing room'), participants were randomly assigned to try on either a bulky sweater or a swimsuit (each was available in a range of sizes). For both men and women, trying on the swimsuit produced a self-conscious state of self-objectification. But that's where any similarity between men and women ended. For more Cengage Learning textbooks, visit www.cengagebrain.co.uk HUNGER, EATING, AND EATING DISORDERS Later came a difficult math test (presented as another study altogether). Men performed equally well on the math test regardless of what they were wearing. Women, by contrast, performed worse on the test when wearing less, consistent with the claim that self-objectification causes a disruption of mental resources. Still later came a taste test. After redressing in their own clothes, participants were asked to taste and evaluate a candy bar. Regardless of what they wore – swimsuit or sweater – most men ate the entire candy bar. The pattern of eating evident among the women, by contrast, was greatly affected by wearing the swimsuit. As the theoretical model reviewed in Concept Review Table predicts, women who wore the swimsuit experienced selfobjectification as well as shame about their current body. The emotional reaction of shame in turn predicted restrained eating, perhaps as a way to correct the shameful mismatch between their own body and the ultra-thin cultural ideals (Fredrickson, Roberts, Noll, Quinn, & Twenge, 1998). These results provide causal evidence in support of objectification theory, which aims to detail the psychological and emotional processes through which exposure of objectifying messages can 'get under the skin' and produce disordered eating. Biological causes Clearly, though, not everyone who is exposed to cultural messages of sexual objectification develops an eating disorder. Certain biological vulnerabilities may increase the tendency to develop eating disorders. One hypothesis is that anorexia is caused by malfunctions of the hypothalamus, the part of the brain that helps regulate eating. Anorexic individuals show lowered functioning of the hypothalamus and abnormalities in several of the neurochemicals that are important to the functioning of the hypothalamus (Fava, Copeland, Schweiger, & Herzog, 1989). With regard to bulimia, there may be a deficiency in the neurotransmitter serotonin, which plays a role in both mood regulation and appetite (Mitchell & deZwann, 1993) or in executive functioning, which affects decision making and impulse control (Brand, Franke-Sievart, Jacoby, Markowitsch, & Tuschen-Caffier, 2007). Familial causes Personality and family factors may also play a role in anorexia and bulimia. Many young women with eating disorders come from families that demand 'perfection' and extreme self-control but do not allow expressions of warmth or conflict (Bruch, 1973; Minuchin, Rosman, & Baker, 1978). Some young women may seek to gain some control over, and expressions of concern from, their parents by controlling their eating habits, eventually developing anorexia. Others may turn to binge eating when they feel emotionally upset or are painfully aware of their low self-esteem (Polivy & Herman, 1993). Therapies designed to help people with eating disorders regain healthy eating habits and deal with the emotional issues they face have proven useful (Agras, 1993; Fairburn & Hay, 1992). Drugs that regulate serotonin levels can also be helpful, particularly for people with bulimia (Mitchell & deZwann, 1993). Anorexia and bulimia are serious disorders, however, and people who have them often continue to have significant problems for several years. INTERIM SUMMARY l Humans have both innate and learned taste preferences and aversions that guide choice of foods. Homeostatic hunger signals, which arise when the body is low in calorie-containing fuels such as glucose, produce appetite partly by causing the individual to perceive food incentives as more attractive and pleasant. l Hunger is largely controlled by homeostatic deficit and satiety signals. Certain neurons in the brain, especially in the brain stem and hypothalamus, detect shortages in glucose and trigger hunger. Other nutrient detectors, especially in the liver, detect increasing energy stores and trigger satiety. A satiety signal, in the form of the hormone cholecystokinin, is also released from the intestines to help stop hunger and eating. l Two regions of the brain are critical to hunger: the lateral hypothalamus and the ventromedial hypothalamus. Destruction of the lateral hypothalamus leads to undereating; destruction of the ventromedial hypothalamus leads to overeating. l People become obese primarily because (1) they are genetically predisposed to be overweight or (2) they overeat (for psychological reasons). The influence of genes is mediated by their effect on fat cells, metabolic rate, and set points. As for overeating and obesity, obese people tend to overeat when they break a diet, eat more when emotionally aroused, and are more responsive to external hunger cues than normal-weight individuals. l In treating obesity, extreme diets appear ineffective because deprivation leads to subsequent overeating and to a lowered metabolic rate. What seems to work best is to establish a new set of permanent eating habits and engage in a program of exercise. l Anorexia nervosa is characterized by extreme, selfimposed weight loss. Bulimia is characterized by recurrent episodes of binge eating, followed by attempts to purge the excess by means of vomiting and laxatives. Possible causes of these eating disorders include personality factors such as low self-esteem, social factors such as a cultural emphasis on thinness and pervasive cultural messages that objectify the female body, and biological factors such as low serotonin levels. CRITICAL THINKING QUESTIONS 1 A potent negative emotion, such as feeling ashamed of one’s body, can contribute to both overeating and obesity, as well as to undereating and various eating disorders. Why is this so? Describe the pathways to each deviation from normal eating. What do you think determines which pathway is followed? 2 The text describes a number of problems associated with dieting, or restrained eating. Why does dieting continue to be very popular? What sociocultural factors come into play? GENDER AND SEXUALITY Like thirst and hunger, sexual desire is a powerful motivation. There are, however, some important differences. Sex is a social motive – it typically involves another person – whereas the survival motives concern only the individual. In addition, sex does not involve an internal deficit that needs to be regulated and remedied for the organism to survive. Consequently, social motives do not lend themselves to a homeostatic analysis. With regard to sex, two critical distinctions should be kept in mind. The first stems from the fact that, although we begin to mature sexually at puberty, the basis for our sexual identity is established in the womb. We therefore distinguish between adult sexuality (that is, beginning with changes at puberty) and early sexual development. The second distinction is between the biological and environmental determinants of sexual behaviors and feelings. For many aspects of sexual development and adult sexuality, a fundamental question is the extent to which the behavior or feeling in question is a product of biology (particularly hormones), environment and learning (early experiences and cultural norms), or interactions between biological and environmental factors. Early sexual development To have gratifying social and sexual experiences as adults, most individuals need to develop an appropriate gender identity, in which males come to think of themselves as males and females as females. This development is quite complex and actually begins before birth. For the first couple of months after conception, only the chromosomes of a human embryo indicate whether it will develop into a boy or a girl. Up to this stage, both sexes are identical in appearance and have tissues that will eventually develop into either testes or ovaries, as well as a genital tubercle that will become either a penis or a clitoris. But between two and three months after conception, a primitive sex

 If the embryonic sex glands produce enough androgen, the fetus will develop male genitals. Shown here is a male fetus 4 months after conception. gland, or gonad, develops into testes if the embryo is genetically male or into ovaries if the embryo is genetically female (see Chapter 2). Once testes or ovaries develop, they produce the sex hormones, which then control the development of the internal reproductive structures and the external genitals. The sex hormones are even more important for prenatal development than they will be for the expression of adult sexuality. The critical hormone in genital development is androgen. If the embryonic sex glands produce enough androgen, the newborn will have male genitals; if there is insufficient androgen, the newborn will have female genitals even if it is genetically male. Conversely, if androgens are added artificially, the newborn will have male genitals even if it is genetically female. In other words, the presence or absence of a male (Y) chromosome normally influences sexual development simply by determining whether the embryo will secrete androgens. The anatomical development of the female embryo does not require female hormones, only the absence of male hormones. In short, nature will produce a female unless androgen intervenes. The influence of androgen, called androgenization, extends far beyond anatomy. After it has molded the genitals, androgen begins to operate on the brain cells. Studies with rats provide evidence that prenatal androgen changes the volume and detailed structure of cells in the   fetus’s hypothalamus, an organ that regulates motivation in humans as well as in rats (Money, 1987). These effects of androgen essentially masculinize the brain and may be responsible for some masculine traits and behaviors that appear months or years later, such as higher levels of aggressiveness. In a series of experiments, pregnant monkeys were injected with androgen, and their female offspring were observed in detail. These offspring showed some anatomical changes (penises instead of clitorises) and also acted differently from normal females. They were more aggressive in play, more masculine in sexual play, and less intimidated by approaching peers (Goy, 1968; Phoenix, Goy, & Resko, 1968). These findings indicate that some gender-typical behaviors (such as greater aggression in males) are partly hormonally determined in nonhuman animals. Early hormonal abnormalities can also have the opposite consequence. They can ‘feminize’ the later sexual behavior of males. A striking example is ‘maternal stress’: a change in the sexual behavior of male rats whose mothers experienced high emotional stress during pregnancy (Ward, 1992). High levels of stress in a pregnant mother rat trigger hormonal events that result in a decrease in the amount of androgens produced by the male embryo’s testes. That, in turn, results in a reduction of androgen reaching the developing brain. The hypothalamus and other brain regions appear to develop differently in such embryos. When these male rats become adults, they show less male sexual behavior and may even show female patterns of copulation movement if they are mounted by another male. It is not known whether similar effects on brain development or behavior occur in humans. Although some believe that these experiments may provide insights into the basis of human heterosexual versus homosexual orientation, there are differences between the results of these animal experiments and human behavior. For example, male rats born to maternally stressed mothers tend to show less sexual behavior of any kind than ordinary male rats, but this is not true of gay men compared with heterosexual men. Nevertheless, these examples illustrate the importance of early hormonal environment for the later sexual behavior of nonhuman animals, and they raise the possibility that prenatal hormones may be important for human sexual motivation as well. Hormones versus environment In humans, much of what is known about the effects of prenatal hormones and early environment has been uncovered by studies of individuals who, for various reasons, were exposed to the prenatal hormones that would ordinarily be experienced by one sex but then were raised in a social role that would ordinarily typify the other sex. In most such cases, the assigned label and the sex role in which the individual is raised have a much

380 CHAPTER 10 MOTIVATION greater influence on gender identity than the individual’s genes and hormones. For example, many thousands of women born during the 1950s and 1960s were exposed to an anti-miscarriage drug, diethylstilbestrol, that had unexpected hormonelike effects on brain development. Ordinarily, the testosterone (the major androgen) secreted by a male embryo’s testes is converted in the brain into a substance similar to diethylstilbestrol. Pregnant women who took the drug therefore unknowingly exposed their fetus to a chemical environment similar to that experienced by the developing brain of a normal male. For male fetuses, this would have little consequence: Their brains were already exposed to male patterns of chemical stimulation. But the female fetuses were exposed to a male-like chemical stimulation for the period when their mothers took the drug. For the overwhelming majority of these daughters, the prenatal exposure had no detectable effect. Most girls who were exposed prenatally to diethylstilbestrol went on to grow up like other girls and to become indistinguishable from women with normal prenatal experience. Social environment, in other words, appears to have had a much greater influence on the sexual and gender development of these women than prenatal hormones. But this is not to say that prenatal chemical environment had absolutely no effect. Researchers have detected several subtle differences that characterize at least some of the women exposed to diethylstilbestrol. For example, a slightly higher proportion of these women appear to be homosexual or bisexual than would ordinarily be expected. Sexual orientation is not identical to gender identity, but in this case a slight effect of prenatal hormones on both may be reflected. (Sexual orientation is discussed in detail later in this section.) Similarly, these women show slightly lower ratings on some measures of ‘maternal interest’, such as finding infants attractive, even though they are not different from other women by most other measures of parental, sexual, or social behavior and attitudes (Ehrhardt et al., 1989). Such studies suggest that although prenatal hormonal events may have some subtle consequences for later sexual and social development, their effect is much weaker in humans than in nonhuman animals. For humans, social and cultural factors appear to be dominant (Money, 1980). There are, however, some studies that point to the opposite conclusion. The most famous of these occurred several years ago in remote villages of the Dominican Republic. It involved 18 XY individuals (genetic males) who, owing to a condition known as androgen insensitivity, were born with internal reproductive organs that were clearly male but with external genitals that were closer to those of females, including a clitoris-like sex organ. In androgen insensitivity, the gonads develop as normal testes and begin to secrete testosterone and other androgens. However, the receptor systems that would be activated by androgens are missing from at least some of For more Cengage Learning textbooks, visit www.cengagebrain.co.uk the body tissues that would ordinarily be masculinized by the hormones. Even though androgens are

secreted and are present in the bloodstream of such a boy, they do not produce the male pattern of genital and physical development. All 18 of the infants studied had been raised as girls, which was at odds with both their genes and their prenatal hormonal environment. When they reached puberty, the surge of male hormones produced the usual bodily changes and turned their clitoris-like sex organs into penis-like organs. The vast majority of these malesreared-as-females rapidly turned into males. They seemed to have little difficulty adjusting to a male gender identity. They went off to work as miners and woodsmen, and some found female sexual partners. In this case, biology triumphed over environment (Imperato-McGinley, Peterson, Gautier, & Sturla, 1979). There is controversy, however, about these Dominican boys who appeared to be girls. They do not seem to have been raised as ordinary girls (which is not surprising, in that they had ambiguous genitals). Rather, they seemed to have been treated as half-girl, half-boy, which could have made their subsequent transition to males easier (Money, 1987). A study in the United Kingdom compared 22 XY individuals with androgen insensitivity, all reared as girls and identifying as women in adulthood, to typical XX females. No differences were observed in life outcome measures, including quality of life, gender identity, sexual orientation, gender-typical behavior, marital status, and personality traits. This evidence underscores the importance of androgenization, suggesting that two X chromosomes and ovaries are not required for typical feminine development (Hines, Ahmed & Hughes, 2003; see also Mazur, 2005). In other cases, the results of conflict between prenatal hormones and social rearing are less clear. In the most dramatic example, identical twin boys had a completely normal prenatal environment. But at the age of eight months, one of the boys had his penis completely severed in what was supposed to be a routine circumcision. Ten months later, the parents authorized surgery to turn their child into a little girl – the testes were removed and a vagina was given preliminary shape. The child was then given female sex hormones and raised as a girl. Within a few years, the child seemed to have assumed a female gender identity: She preferred more feminine clothes, toys, and activities than her twin brother did. Because she appeared to be a normal girl in many ways, most investigators concluded that this was a case in which social environment had won out. However, studies of the child at the time she reached puberty revealed that the outcome was more complex (Diamond, 1982). As a teenager, she was unhappy and appeared to be confused about her sexuality, even though she had not been told about her original sex or the sexchange operation she had undergone. In interviews, she refused to draw a picture of a woman and instead would

draw only a man. Aspects of her body language, such as her walking gait and patterns of posture and movement, were masculine in appearance. Socially, she had considerably more than the usual degree of difficulty in forming relationships with her peers. A recent follow-up on this individual found that he eventually rejected the female gender identity and has successfully lived as a male since then (Diamond & Sigmundson, 1997). In the long run, the attempt to control his gender identity through socialization and to raise him as a 'normal girl' was unsuccessful. It is difficult to know the precise source of the difficulty he experienced in emotional and social adjustment at puberty. Explanations include the possibility that his early brain development as a male placed constraints on his later ability to adapt to a female gender identity. What can we conclude about gender identity? Clearly, prenatal hormones and environment are both major determinants of gender identity and typically work in harmony. When they clash, as they do in some individuals, most experts believe that environment will dominate. But this is a controversial area, and expert opinion may change as additional data are gathered. Adult sexuality Changes in body hormone systems occur at puberty, which usually begins between the ages of 11 and 14 (see Figure 10.6).

The hypothalamus begins to secrete chemicals called gonadotropin releasing factors; these stimulate the pituitary gland, which lies immediately below the hypothalamus. The pituitary secretes sex hormones, called gonadotropins, into the bloodstream. These circulate through the body and reach the gonads – ovaries in females and testes in males – which generate egg or sperm cells. Gonadotropins activate the gonads, causing them to secrete additional sex hormones into the bloodstream. In women, the hypothalamus releases its gonadotropin-releasing factors on a monthly cycle, rising and falling approximately every 28 days. This stimulates the pituitary to secrete two gonadotropins: follicle-stimulating hormone (FSH) and luteinizing hormone (LH), also on a monthly cycle. These hormones activate the ovaries. Follicle-stimulating hormone stimulates the ovaries to generate follicles, clusters of cells in the ovaries that allow fertile eggs to develop. Once a follicle is generated, it begins to secrete the female hormone, estrogen. Estrogen is released into the bloodstream to affect the body’s sexual development and, in many species of animals, to activate sexual motivation in the brain. The second gonadotropin, luteinizing hormone, is released from the pituitary slightly later than follicle-stimulating hormone. Luteinizing hormone causes ovulation, the release of a mature fertile egg cell from the follicle. When the follicle releases its egg, it also secretes a second female hormone, progesterone, which prepares the uterus for implantation   Brain hypothalamus Gonadotropin releasing factor Pituitary Men Women Gonadotropins ICSH FSH & LH TESTES Sperm maturation OVARIES Follicle maturation and ovulation Estrogen & Progesterone Androgens (Testosterone) Figure 10.6 The Hormonal System Involved in Sex. By way of hormones, the hypothalamus directs the pituitary, which in turn directs the gonads to secrete the sex hormones. of a fertilized egg and, in some species of animals, also activates sexual motivation in the brain. In men, the hypothalamus secretes gonadotropinreleasing factor in a constant fashion rather than in a monthly cycle. This causes the male pituitary to constantly release its gonadotropin, called interstitial cell stimulating hormone (ICSH), into the bloodstream. ICSH causes male testes to produce mature sperm cells and dramatically boost secretion of androgens, especially testosterone. Testosterone and other androgens stimulate the development of male physical characteristics and, in most species of animals, act on the brain to activate sexual desire. Effects of hormones on desire and arousal In many species, sexual arousal is closely tied to variations in hormonal levels. In humans, however, hormones play less of a role. One way to assess the contribution of hormones to sexual arousal is to study the effects of removing the gonads, a procedure called gonadectomy. (In males, removal of the testes is called castration.) In experiments with animals such as rats and guinea pigs, castration results in rapid decline and eventual disappearance of sexual activity. For humans, of course, there are no controlled experiments. Psychologists rely instead on observations of males with serious illnesses (such as cancer of the testes) who have undergone chemical castration (use of synthetic hormones to suppress or block

 the use of androgen). These studies typically show that some men lose interest in sex but others continue to lead a normal sex life (Money, Weideking, Walker, & Gain, 1976; Walker, 1978). Apparently androgen contributes to sexual desire only in some cases. Another way to measure the contribution of hormones to sexual desire and arousal in men is to look for a relationship between hormonal fluctuation and sexual interest. For example, is a man more likely to feel aroused when his testosterone level is high? It turns out that testosterone level may have no effect on copulatory function – as indicated by the ability to have an erection – but does increase desire, as indicated by sexual fantasies (Davidson, 1989). The major determinants of sexual desire in men, however, seem to be emotional factors. For males as well as females, the most common cause of low desire in couples seeking sex therapy is marital conflict (Goleman, 1988). Sexual desire is even less dependent on hormones in women. This contrasts with nonprimate species, in which female sexual behavior is highly dependent on sexual hormones. In all other animals, removal of the ovaries results in cessation of sexual activity. Such a female ceases to be receptive to the male and usually resists sexual advances. The major exception is the human female. Following menopause (when the ovaries have ceased to function), most women do not experience diminished sexual desire. In fact, some women show increased interest in sex after menopause, possibly because they are no longer concerned about becoming pregnant. There is evidence to indicate that women’s sexual desire is facilitated by trace amounts of sex hormones in the bloodstream (Sherwin, 1988) and that the types of men that women find attractive vary with normal monthly hormone fluctuation (Gangstad, Garver-Apgar, Simpson & Cousins, 2007). However, the level required is so low that it may be exceeded in most women and hence not play a significant role in changes in overall desire. Studies of the relationship between hormonal fluctuation and sexual arousal in premenopausal females lead to a similar conclusion: Normal changes in hormones control arousal in other animals but not in humans. In female mammals, hormones fluctuate cyclically, with accompanying changes in fertility. During the first part of the mammalian cycle (while the egg is being prepared for fertilization), the ovaries secrete estrogen, which prepares the uterus for implantation and also tends to arouse sexual interest. After ovulation occurs, both progesterone and estrogen are secreted. This fertility or estrous cycle is accompanied by a variation in sexual motivation in most mammalian species. Most female animals are receptive to sexual advances by a male only during the period of ovulation, when the estrogen level is at its highest; during this time, the female is said to be ‘in heat’. Among primates, however, sexual activity is less strongly influenced by the fertility cycle. Monkey, ape, and chimpanzee females copulate during all phases of the cycle, although ovulation is still the period of most intense sexual activity. In the human female, sexual desire and arousal seem to be affected much more by social and emotional factors. In sum, the degree of hormonal control over sexual behavior is lower in humans than in other animals. Still, even for humans there may be some hormonal control, as witnessed by the relationship between testosterone levels and sexual desire in men. Neural control In one sense, the primary sex organ is the brain. The brain is where sexual desire originates and where sexual behavior is controlled. In humans, the sexual function of the brain extends to the control of sexual thoughts, images, and fantasies. Within the brain, sexual hormones can influence neural function in adult individuals. Next, we discuss how sexual hormones also influence the physical growth and connection patterns of neurons in early life for all mammalian species, including humans, and in adults for at least some species (Breedlove, 1994). The nervous system is affected by sexual hormones at many levels. At the level of the spinal cord, neural circuits control the movements of copulation. In males, these include erection of the penis, pelvic movements, and ejaculation. All of these actions can be elicited in a reflex fashion in men whose spinal cords have been severed by injury and who have no conscious body sensations. Similarly, clinical studies of women with spinal injury indicate that vaginal secretions in response to genital stimulation and pelvic movements may be controlled by neural reflex circuits within the spinal cord (Offir, 1982). Higher levels of the brain, especially the hypothalamus, contain the neural systems that are important to ª DAVID C. FRITZ/ANIMALS, ANIMALS Sexual play among snow monkeys. Normal heterosexual behavior in primates depends not only on hormones and the development of specific sexual responses but also on an affectional bond with a member of the other sex.

more complex aspects of sexual behavior. For example, sexual pursuit and copulation can be elicited in both males and females of many animal species by electrical stimulation of hypothalamic regions. Even in humans, stimulation of brain regions near the hypothalamus has been reported to induce intense sexual feelings and desire (Heath, 1972). Conversely, lesions of the hypothalamus can eliminate sexual behavior in many species, including humans. Early experiences The environment also influences adult sexuality. Early experience is a major determinant of the sexual behavior of many mammals and can affect specific sexual responses. For instance, in their play, young monkeys exhibit many of the postures required later for copulation. When wrestling with their peers, infant male monkeys display hindquarter grasping and thrusting responses that are components of adult sexual behavior. Infant female monkeys retreat when threatened by an aggressive male infant and stand steadfastly in a posture similar to the stance required to support the weight of the male during copulation. These presexual responses appear as early as 60 days of age and become more frequent and refined as the monkey matures. Their early appearance suggests that they are innate responses to specific stimuli, and the modification and refinement of these responses through experience indicate that learning plays a role in the development of the adult sexual pattern. Experience also affects the interpersonal aspect of sex. Monkeys raised in partial isolation (in separate wire cages, where they can see other monkeys but cannot have contact with them) are usually unable to copulate at maturity. The male monkeys are able to perform the mechanics of sex: They masturbate to ejaculation at about the same frequency as normal monkeys. But when confronted with a sexually receptive female, they do not seem to know how to assume the correct posture for copulation. They are aroused, but they aimlessly grope the female or their own bodies. Their problem is not just a deficiency of specific responses. These monkeys have social or affectional problems. Even in nonsexual situations, they are unable to relate to other monkeys, exhibiting either fear and flight or extreme aggression. Apparently, normal heterosexual behavior in primates depends not only on hormones and the development of specific sexual responses but also on an affectional bond with a member of the other sex. This bond is an outgrowth of earlier interactions with the mother and peers, through which the young monkey learns to trust, to expose its delicate parts without fear of harm, to accept and enjoy physical contact with others, and to be motivated to seek the company of others (Harlow, 1971). Although we must be cautious about generalizing these findings to human sexual development, clinical observations   of human infants suggest certain parallels. Human infants develop their first feelings of trust and affection through a warm and loving relationship with their primary caretaker (see Chapter 3). This basic trust is a prerequisite for satisfactory interactions with peers. And affectionate relationships with other youngsters of both sexes lay the groundwork for the intimacy required for sexual relationships among adults. Cultural influences Culture also influences the expression of sexual desire. Unlike that of other primates, human sexual behavior is strongly determined by culture. For example, every society places some restrictions on sexual behavior. Incest (sexual relations within the family) is prohibited in almost all cultures. Other aspects of sexual behavior – sexual activity among children, homosexuality, masturbation, and premarital sex – are permitted in varying degrees by different societies. Among preliterate cultures, acceptable sexual activity varies widely. Some very permissive societies encourage autoerotic activities and sex play among children of both sexes and allow them to observe adult sexual activity. The Chewa of Africa, for example, believe that if children are not allowed to exercise themselves sexually, they will be unable to produce offspring later. The Sambia of New Guinea have institutionalized bisexuality: From prepuberty until marriage, a boy lives with other males and engages in homosexual practices (Herdt, 1984). In contrast, very restrictive societies try to control preadolescent sexual behavior and prevent children from learning about sex. The Cuna of South America believe that children should be totally ignorant about sex until they are married; they do not even permit their children to watch animals give birth. Although the most obvious way to study cultural differences is to investigate practices in different countries, one can also look at culture changes that occur within a country. One such change occurred in the United States and other Western countries between the 1940s and the 1970s. In the 1940s and 1950s, the United States and most other Western countries would have been classified as sexually restrictive. Traditionally, the existence of prepubertal sexuality had been ignored or denied. Marital sex was considered the only legitimate sexual outlet, and other forms of sexual expression (homosexual activities, premarital and extramarital sex) were generally condemned and often prohibited by law. Of course, many members of these societies engaged in such activities, but often with feelings of shame. Over the years, sexual activities became less restricted. Premarital intercourse became more acceptable and more frequent. Among American university-educated individuals interviewed in the 1940s, 27 percent of the women and 49 percent of the men had engaged in premarital sex by age 21 (Kinsey, Pomeroy, & Martin, 1948; Kinsey,

 Males Females 80 Percent who have had coitus 40 0 1940 1950 1960 1970 Year of study Figure 10.7 Reported Incidence of Premarital Coitus. Each data point represents findings from a study of the incidence of premarital sex among college men and women. Note the marked upward trend starting in the 1960s. (J. R. Hopkins (1977) ‘Sexual Behavior in Adolescence’, in Journal of Social Issues, Vol. 33(2):67–85. Adapted with permission of the Society for the Psychological Study of Social Issues.) Pomeroy, Martin, & Gebhard, 1953). In contrast, several surveys of American university students conducted in the 1970s reported percentages ranging from 40 percent to over 80 percent for both males and females (Hunt, 1974; Tavris & Sadd, 1977). Over the past several decades, there has been a gradual trend toward initiating sex at an earlier age. Roughly 50 percent of both men and women report having had sexual intercourse by age 16 or 17 (Laumann, Gagnon, Michael, & Michaels, 1994). Figure 10.7 gives the reported incidence of premarital intercourse in studies conducted over a 35-year span. Note that the change in sexual behavior was greater among women than among men and that the biggest changes occurred in the late 1960s. These changes led many observers of the social scene in the 1970s to conclude that a ‘sexual revolution’ had occurred. Today it seems that the sexual revolution has been stymied by the fear of sexually transmitted diseases, particularly AIDS. Moreover, the ‘revolution’ may have involved behavior more than feelings. In interviews with young couples in the U.S. in the 1970s, only 20 percent thought that sex between casual acquaintances was completely acceptable (Peplau, Rubin, & Hill, 1977). In a similar vein, although women are becoming more like men with regard to sexual behavior, they continue to differ from men in certain attitudes toward sex before marriage. The majority of women who engage in premarital sex do so with only one or two partners with whom they are emotionally involved. Men, in contrast, are more likely to seek sex with multiple partners (Laumann et al., 1994). However, within a given five-year period, the majority of both men and women are likely to have no more than one sexual partner (Laumann et al., 1994). Sex differences Studies of heterosexuals have shown that young men and women differ in their attitudes about sex; women are more likely than men to view sex as part of a loving relationship. Related to this, differences between women and men have been reported in the nature of the type of event that is most likely to elicit sexual jealousy: emotional infidelity or sexual infidelity. Whether measured by self-reports or by autonomic reactions such as heart rate, women react more strongly to the prospect of emotional infidelity (the prospect of their partner's forming a romantic relationship with someone else), regardless of whether the infidelity involves an actual sexual act. By contrast, men react more strongly to the prospect of sexual infidelity, regardless of whether their partner's sexual liaison involves an emotional commitment (Buss, Larsen, Western, & Semmelroth, 1992). Men and women are also sexually responsive to different sorts of stimuli, regardless of their sexual orientation. Heterosexual and homosexual men and women were shown a range of sexual films in a private, laboratory setting, while their genital responses were recorded continuously using psychophysiological sensors. The films depicted men and women engaging in same-sex intercourse, solitary masturbation, or nude exercise, or human heterosexual intercourse or animal copulation. Across all participants, genital responses were weakest to nude exercise and strongest to intercourse. Men's responses, however, depended primarily on the sex of the actors, with heterosexual men responding most to female actors and homosexual men responding most to male actors. By contrast, women's responses depended primarily on the level of sexual activity depicted, and not on the sex of the actors involved (Chivers, Seto, & Blanchard, 2007). Differences between the sexes apply to behavior as well as to attitudes. Women who engage in premarital sex are likely to have fewer sexual partners than men. Differences between male and female patterns of sexual behavior persist regardless of sexual orientation. For example, lesbian couples are likely to have sex less frequently than heterosexual couples, and gay male couples have sex more often than heterosexual couples. Such differences can be viewed as reflecting a continuum that extends from female-typical characteristics to male-typical characteristics (Buss, 1994a). Sexual orientation An individual's sexual orientation is the degree to which he or she is sexually attracted to persons of the other sex and/or to persons of the same sex. Like Alfred Kinsey, the

pioneering sex researcher of the 1940s, most behavioral scientists conceptualize sexual orientation as a continuum, ranging from exclusive heterosexuality to exclusive homosexuality. For example, on Kinsey's own 7-point scale, individuals who are attracted exclusively to persons of the other sex and who engage in sexual behavior only with such persons are at the heterosexual end of the scale (category 0); those who are attracted exclusively to persons of the same sex and who engage in sexual behavior only with such persons are at the homosexual end of the continuum (category 6). Individuals in categories 2 through 4 are usually defined as bisexual. This oversimplifies the situation, however, because sexual orientation comprises several distinct components, including erotic attraction or sexual desire, sexual behavior, romantic attraction, and self-identification as a heterosexual, homosexual, or bisexual person. It is not uncommon for an individual to be at different points on the scale for different components. For example, many people who are sexually attracted to persons of the same sex have never participated in any homosexual behaviors, and many who have had frequent homosexual encounters do not identify themselves as homosexual or bisexual persons. To further complicate matters, a subset of people show a complete lack of sexual attraction, termed asexuality, estimated at 1 percent of the population in a United Kingdom sample (Bogaert, 2004, 2006). Frequency of different sexual orientations In a survey of sexuality in the United States, 10.1 percent of adult men and 8.6 percent of adult women in a national random sample reported at least one of the following: (1) They were currently attracted 'mostly' or 'only' to persons of their own sex, (2) they found having sex with someone of the same sex 'somewhat' or 'very' appealing, or (3) they had engaged in sexual behavior with a person of the same sex since age 18 (Laumann et al., 1994). These percentages are similar to the percentage of people who are left-handed (about 8%). In terms of self-identification, 2.8 percent of the men and 1.4 percent of the women identified themselves as homosexual (or gay or lesbian) or bisexual – similar to the percentage of people in the U.S. who identify themselves as Jewish (2% to 3%). As the authors of the survey acknowledge, these percentages must be regarded as underestimates because many people are reluctant to report desires or behaviors that are still considered by some to be immoral or pathological. The problem was particularly acute in this survey because the interviews were conducted in the respondents' own homes, and other family members, including children, were also in the home at the time, although not necessarily in the room, during more than 20 percent of the interviews. And although actual homosexual behavior is somewhat atypical, the potential For more Cengage Learning textbooks, visit www.cengagebrain.co.uk GENDER AND SEXUALITY for homosexual responses – given the right person and the right situation – is rather common, estimated at 33 percent for men and 65 percent for women (Santtila, Sandnabba, Harlaar, Varjonen, Alanko, & von der Pahlen, 2008). Causes of sexual orientation The common question 'What causes homosexuality?' is scientifically misconceived because it implicitly assumes either that heterosexuality needs no explanation or that its causes are self-evident. Those who have thought about it at all are likely to conclude that because only heterosexual behavior results in reproduction, it must be the 'natural' outcome of evolution, so only deviations from heterosexuality (such as homosexuality) pose a scientific puzzle. Freud did not agree: '[heterosexuality] is also a problem that needs elucidation and is not a selfevident fact based upon an attraction that is ultimately of a chemical nature' (1905/1962, pp. 11–12). It is because we agree with Freud that we have called this section of the chapter 'sexual orientation' and not 'homosexuality'. At issue once again is the nature–nurture question, which we introduced in Chapter 1 and discussed in the chapter on development (Chapter 3) and will discuss again in the chapter on individual differences (Chapter 12): To what extent is an adult's sexual orientation determined by earlier life experiences or to innate biological influences, such as genes or prenatal hormones? The best data on earlier life experiences comes from an intensive, large-scale interview study of approximately 1,000 homosexual and 500 heterosexual men and women living in the San Francisco Bay area (Bell, Weinberg, & Hammersmith, 1981a). The study uncovered one – and only one – major factor that predicted a homosexual orientation in adulthood for both men and women: childhood gender nonconformity. As shown in Table 10.2, when asked what play activities they had or had not enjoyed as children, gay men and lesbians were significantly more likely than heterosexual men and women to report that they had not enjoyed activities typical of their sex and significantly more likely to report that they had enjoyed activities typical of the other sex. Gay men and lesbians were also more likely than their heterosexual counterparts to report that they had not been masculine (for men) or feminine (for women) as children. In addition to this gender nonconformity, gay men and lesbians were more likely to report having had more friends of the other sex. Studies like this rely on retrospections about one's childhood, which makes memory bias a legitimate threat to validity. The same findings about childhood gender nonconformity emerge, however, even with less biased research methods, for instance the study of childhood home videos (Rieger, Linsenmeier, Gygax & Bailey, 2008).

Two features of the data in Table 10.2 are worth noting. First, the findings are quite strong and similar for men and women: 63 percent of both gay men and lesbians had not enjoyed childhood activities typical of their sex, compared with only 10 to 15 percent of their heterosexual counterparts. Second, it is clear that women are more likely than men to have enjoyed activities typical of the other sex during childhood and to have had more childhood friends of the other sex. In fact, a majority of both the lesbians and the heterosexual women in this study were 'tomboys' –

that is, enjoyed boys’ activities as children. It is the nonenjoyment of sex-typical activities that appears to be the best predictor of an adult homosexual orientation for both men and women. The overall finding that childhood gender nonconformity predicts an adult homosexual outcome has now been confirmed in several other studies (Bailey & Zucker, 1995; Rieger et al., 2008), including several that followed gendernonconforming boys into adolescence and adulthood and assessed their sexual orientations (Green, 1987a, b; Zucker, 1990). In addition to the gender nonconformity finding, the San Francisco study also yielded many negative findings that were important because they disconfirmed common theories about the antecedents of a homosexual orientation. For example: l A person’s identification with the other-sex parent while growing up appears to have no significant impact on whether he or she turns out to be homosexual or heterosexual. This fails to confirm Freud’s psychoanalytic theory (discussed in Chapter 13), as well as other theories based on the dynamics of the person’s childhood family. l Gay men and lesbians were no more likely than their heterosexual counterparts to report having their first sexual encounter with a person of the same sex. Moreover, they neither lacked heterosexual experiences during their childhood and adolescent years nor found such experiences unpleasant. l A person’s sexual orientation is usually determined by adolescence, even though he or she might not yet have become sexually active. Gay men and lesbians typically experienced same-sex attractions about three years before they had engaged in any ‘advanced’ sexual activity with persons of the same sex. These last two sets of findings indicate that, in general, homosexual feelings, not homosexual behaviors, are the crucial antecedents of an adult homosexual orientation. They thus disconfirm any simple behavioral learning theory of sexual orientation, including the popular, laypersons’ version, which asserts that an individual can become gay by being ‘seduced’ by a person of the same sex or by having an admired, openly gay teacher, parent, or clergyperson. Cross-cultural data are also consistent with this conclusion. For example, in the Sambian culture of New Guinea, cited earlier, all boys engage in exclusively homosexual behaviors from prepuberty through late adolescence. At that point, virtually all of them marry and become exclusively heterosexual (Herdt, 1984). Finally, it is clear from all the studies that one’s sexual orientation is not something that one simply chooses. Gay men and lesbians do not choose to have erotic feelings toward persons of the same sex any more than heterosexual persons choose to have erotic feelings toward persons of the other sex. As the accompanying essays in the Seeing Both Sides section illustrate, behavioral scientists do disagree over the nature–nurture question – whether the major determinants of sexual orientation are rooted in biology or experience – but the public often misconstrues the question to be whether sexual orientation is determined by variables beyond the Table 10.2 Gender nonconformity in childhood In a large-scale interview study, gay men and lesbians were more likely than heterosexual men and women to report that they were gender nonconforming during childhood. (A. P. Bell, M. A. Weinberg, & S. K. Hammerstein, Sexual Preference: Its Development in Men & Women.) Men Women Gender nonconforming preferences and behaviors Gay Heterosexual Lesbian Heterosexual Had not enjoyed sex-typical activities 63% 10% 63% 15% Had enjoyed sex-atypical activities 48% 11% 81% 61% Atypically sex-typed (masculinity/femininity) 56% 8% 80% 24% Most childhood friends were other sex 42% 13% 60% 40%

individual’s control or is freely chosen. That is not the same question. Because most of the major theories of homosexuality based on childhood or adolescent experiences have not been supported by the evidence, many scientists now believe that the origins of both childhood gender nonconformity and adult homosexual orientation may lie in an individual’s biology, possibly in the genes or prenatal hormones. In Seeing Both Sides, two contrasting views of the current biological evidence are presented. One view clearly puts ‘nature’ over ‘nurture’, emphasizing the roles of genes and hormones in causing both childhood gender nonconformity and adult homosexual orientation. The other view, by contrast, takes a ‘nature and nurture’ approach (see Chapter 1). It presents a new theory called exotic-becomes-erotic, which posits a critical, albeit more limited role for biology in determining sexual orientation. The influence of nature comes first. The theory suggests that genes and hormones cause differences in childhood temperament and personality traits, which for some children can produce a dislike of sex-typical activities and a preference for sex-atypical activities. Next comes the influence of nurture. The theory asserts that engaging in sex-atypical activities places children in the company of the other sex (rather than the same sex), who come to be seen as similar to the self. Later, when adult sexuality is awakened, the theory contends that people find those who are viewed as different from the self (exotic) to be most sexually attractive (erotic), who turn out to be members of their same sex. The more general point within combined nature and nurture approaches is that just because a behavior might be advantageous from the standpoint of reproduction, it does not follow that evolution has ‘hardwired’ it into the species (nature only). A similar case for the combined action of nature and nurture can be made for the notion of imprinting, which is the early rapid learning that allows a newborn (or newly hatched) animal to develop an attachment to its mother (see Chapter 7). Within the first hours of life, infants of many species are ‘programmed’ to learn an emotional attachment to the closest social figure. Most often this is the mother, but if the first moving object seen is a human or a mobile toy, the imprinting process can produce attachment beyond species boundaries. Imprinting turns out to have consequences for later sexual behavior as well, because mate choices follow maternal imprinting. The imprinting instinct is genetic (nature), but as long as the environment (nurture) supports or promotes reproductively advantageous behavior often enough, attachment and reproductive behaviors need not necessarily get fully programmed into the genes. And just as ducklings encounter mother ducks most of the time, so, too, human societies see to it that men and women see each other as dissimilar often enough to ensure that the species will not perish from the earth. GENDER AND SEXUALITY INTERIM SUMMARY l Prenatal hormones contribute to sexual development. If the embryonic sex glands produce enough androgen hormones, the embryo will have a male pattern of genital and brain development. If androgens are low or missing, the embryo will have a female pattern of genital and brain development. l For nonhuman animals, prenatal hormones appear to be powerful determinants of adult sexual behavior. For humans, prenatal hormones appear to be less important than postnatalsocialgenderrolesindeterminingadultsexualbehavior. l The female hormones (estrogen and progesterone) and male hormones (androgens) are responsible for the changes in the body that occur at puberty, but in contrast to other animals, they play a limited role in human sexual arousal. In primates and humans, early social experiences with parents and peers have a large influence on adult sexuality, and for humans, cultural norms are also influential. l Recent studies have bolstered the claim that biological, genetic, hormonal, or neural factors may partly determine whether an individual will be heterosexual or homosexual, but the evidence is not conclusive. It is also unknown whether biological factors may influence sexual orientation directly or whether they instead contribute to other traits, such as gender conformity, that indirectly influence the development of sexual orientation. CRITICAL THINKING QUESTIONS 1 How does sexual identity differ from sexual orientation? 2 Why do you think many people believe that sexual desire and activity in humans is strongly influenced by hormones when the evidence suggests that it is not?

Throughout this chapter, we have seen that psychological and biological causes are so closely intertwined in the control of many motivations that they merge into one stream of events. Not only can biological causes control psychological motivations like hunger and thirst, but psychological processes and experiences control motivation and may feed back to control physiological responses. For example, repeated use of an addictive drug may permanently change particular brain systems. More commonly, the particular foods and drinks we desire are established as objects of choice largely by learning, and even the degree of satiety produced by a stomach full of food is influenced by previous experience. Our social attachments are determined largely by the consequences of earlier social interactions with particular individuals. When it comes to many motivational processes, biology and psychology are not separate domains but, rather, two aspects of control that continually interact to direct motivational processes.

 SEEING BOTH SIDES IS SEXUAL ORIENTATION INNATE OR SOCIALLY DETERMINED? Male sexual orientation is innate. Female sexual orientation is not well understood J. Michael Bailey, Northwestern University The central question regarding sexual orientation has been 'nature or nurture?' In recent years, however, researchers have taken a step back to ask: 'What is sexual orientation?' The answer differs for men and women, in ways that suggests that the nature–nurture question may also differ for them. For men, one plausible candidate for the meaning of sexual orientation is sexual arousal pattern. In the laboratory (and in life), heterosexual men are sexually aroused by erotic stimuli depicting attractive women but not attractive men, and homosexual men show the reverse pattern. Sexual arousal both motivates men to seek partners of their preferred type and enables them to have sex with them (Bailey, in press). Women show a very different pattern. On average, heterosexual women are as aroused to female sexual stimuli as they are to male stimuli, and lesbians show only a modest physiological preference for female stimuli (Chivers, Rieger, Latty, & Bailey, 2004; Chivers, Seto, & Blanchard, 2007). Evidently, sexual arousal patterns are not important influences on female sexuality. Women's sexuality appears to be more socially influenced than men's (Baumeister, 2000; Diamond, 2008). For example, women's sexual desire may be more 'fluid' than men's, less rigidly directed toward persons of a particular sex and more changeable over time, depending on relational factors such as romantic attachment (Diamond, 2008). Some have even questioned whether women have a sexual orientation directing their sexual choices (Bailey, in press). What causes some people to lust after men and others, women? To answer this question, ideally one would randomly assign individuals to have male or female biology (nature) and to have male or female social environment (nurture). Of course this would be unethical, but there are some rare circumstances that approximate the ideal experiment. For example, there have been two cases of normal infant boys whose penises were destroyed by surgical accidents and who were subsequently reared as girls (Bradley, Oliver, Chernick, & Zucker, 1998; Diamond & Sigmundson, 1997). One case retained her female identity but was primarily attracted to other women (Bradley et al., 1998). The other case both renounced her female sex, declaring herself a male, and married a woman (Colapinto, 2000; Diamond & Sigmundson, 1997). Another relevant condition is cloacal exstrophy, a congenital birth defect that causes both abdominal malformations and (in males) a poorly formed penis. For a time, male infants born with cloacal exstrophy were surgically and socially reassigned as girls. In a follow-up study of individuals born with cloacal exstrophy, all three adolescents born male but reared as females said they were attracted to females (Reiner & Gearhart, 2004). Thus, in all five relevant cases, sexual orientation was consistent with prenatal biology (nature) rather than postnatal rearing (nurture). It may seem as if five cases is too small a number to allow general conclusions, but the chances against all five of them turning out this way, if nurture were as important as nature, are astronomical. If one cannot make a male attracted to other males by cutting off his penis and rearing him as a girl, how likely is any hypothesis stressing social factors? It is important to note, however, that these individuals were all biologically male at birth. Individuals with female prenatal biology might be more sexually flexible. Studying people in other cultures can be illuminating both regarding cross-cultural similarities and differences. Similarities among cultures are consistent with the importance of nature, differences with the importance of nurture. All known cultures have homosexual individuals, but the expression of homosexuality shows some striking differences between cultures (Greenberg, 1990). In the contemporary West, including North American and Europe, the predominant expression is egalitarian homosexuality, in which two men with sexual preference for other men have sexual and romantic relationships with each other. This contrasts with cultures having a tradition of transgender male homosexuality. In these cultures, males attracted to other males take on a quasi-female 'third gender' identity, often having female names and female-like appearances. Examples include the hijras of India (Nanda, 1990), the travestis of Latin America (Kulick, 1998), and the fa'afafine of Samoa (Vasey and Bartlett, 2007). Individuals in these third-gender categories do not have sex with each other, but, rather, have sex with heterosexually identfied men, who presumably are attracted to their femininity. These individuals are similar to homosexual males in the West because both are sexually attracted to unambiguously male bodies, and both tend to be much more feminine than heterosexual men, starting in childhood (Bailey & Zucker, 1995; Vasey & Bartlett, 2007). Besides the obvious differences between the two types of homosexuality – self-presentation and typical sex partner – there is also a difference in societal attitudes towards them, at least in Samoa. The fa'afafine tend to be accepted by their families from childhood on, and are generally seen as valuable members of the community (Vasey & Bartlett, 2007).

Sexual identity could be socially determined Daryl J. Bem, Cornell University Dr. Bailey and I agree on the evidence showing a link or correlation between biological variables and sexual orientation. But, as Dr. Bailey points out, this does not preclude the influence of environmental factors. This leaves open the possibility that social variables are also involved in the development of sexual orientation. Accordingly, I have proposed a theory that attempts to combine biological and social variables into an overall account of sexual orientation development: the Exotic-Becomes-Erotic (EBE) theory (Bem, 1996). The path proposed by this theory is illustrated in the figure below. A) Biological variables B) Childhood temperaments C) Sex typical/atypical activity preferences (gender conformity/nonconformity) D) Feeling different from other/same sex peers ('exotic') E) Physiological arousal to other/same sex peers F) Erotic attraction to other/same sex persons   A ? B The theory proposes, first, that genetic, hormonal, and possibly other biological factors do not directly influence adult sexual orientation itself but, rather, influence a child's temperament and personality traits. Many personality traits have strong genetic or heritable components, including such childhood temperaments as aggression and activity level. B ? C Temperaments such as these predispose a child to enjoy some activities more than others: A more aggressive or active child will enjoy rough-and-tumble play (boy-typical activities); another will prefer to socialize quietly (girl-typical activities). Thus, depending on the sex of the child, he or she will be genetically predisposed to be gender conforming or gender nonconforming. As shown in Table 10.2, children also tend to have friends who share their activity preferences; for example, the child – male or female – who shuns competitive team sports will avoid playing with boys and seek out girls as playmates. C ? D Accordingly, gender conforming children will feel more different from children of the other sex; gender nonconforming children will feel more different from children of the same sex – that is, to see them as relatively more 'exotic' than children of the other sex. D ? E This feeling of being different creates heightened arousal. For the male-typical child, it may be felt as antipathy or contempt in the presence of girls ('girls are yucky'); for the female-typical child, it may be felt as timidity or apprehension in the presence of boys. For most children, however, this arousal will probably not be consciously felt. E ? F This arousal is transformed in later years into sexual arousal or erotic attraction: Exotic becomes erotic. Evidence for this last step comes, in part, from studies in which heterosexual male participants who had been physiologically (but nonsexually) aroused were found to be more sexually attracted to a woman than were men who had not been physiologically aroused. In other words, general physiological arousal can be experienced, interpreted, and transformed into actual sexual arousal. Indirect evidence exists for the theory's claim that childhood gender nonconformity intervenes between biological variables and sexual orientation. For example, studies of twins found that pairs of identical twins were more similar than pairs of fraternal twins on childhood gender nonconformity (Bailey & Pillard, 1995; Martin, Boomsma & Machen 1997). Similarly, DNA studies found that pairs of gay brothers who share the same piece of the X chromosome are also more alike on gender nonconformity than are gay brothers who do not share it (Hamer et al., 1993; Hu et al. 1995). More direct evidence comes from analysis of data from a large study of Australian twins which shows that childhood gender nonconformity is, in fact, the intervening link between the genes and sexual orientation (Bem, 2000). In short, the studies showing a link between

 biological variables and an adult homosexual orientation are consistent with EBE theory's assertion that the biology leads first to gender-nonconforming interests and preferences in childhood and, only subsequently, to the adult homosexual orientation. As the discussion in the text notes, the basic question is not 'What causes homosexuality?' but 'What causes sexual orientation?' One virtue of EBE theory is that it attempts to address that question because it applies to both heterosexuality and homosexuality. Because most societies emphasize the differences between males and females, most boys and girls will grow up feeling different from their other-sex peers and, hence, will come to be erotically attracted to them later in life. According to CHAPTER SUMMARY Motivational states direct and activate behavior. They arise from two sources: internal drive factors and external incentive factors. Drive factors tend to promote homeostasis: the preservation of a constant internal state. Homeostasis involves several components: a goal value or set point for the ideal internal state, a sensory signal that measures the actual internal state, a comparison between the goal value and the sensory signal, and finally, a response that brings the actual internal state closer to the goal value. Regulation of temperature is an example of homeostasis. The regulated variable is the temperature of the blood, and sensors for this are located in various parts of the body, including the hypothalamus. Adjustments are either automatic physiological responses (for example, shivering) or voluntary behavioral ones (such as putting on a sweater). Thirst is another homeostatic motive. There are two regulated variables, intracellular fluid and extracellular fluid. Loss of intracellular fluid is detected by osmotic sensors, neurons in the hypothalamus that respond to dehydration. Loss of extracellular fluid is detected by blood-pressure sensors, neurons in major veins and organs that respond to a drop in pressure. Intracellular and extracellular signals act together to produce thirst. EBE theory, this is why heterosexuality is the most common orientation across time and culture. Nevertheless, these studies do not prove that EBE theory is correct, and only further research can help us decide. The more important point I wish to emphasize here is that just because some human behavior is correlated with biological factors, it does not follow that these factors directly cause the behavior. For example, it is known that divorce is approximately as heritable as sexual orientation, and yet there are no biological scientists out searching for a 'divorce' gene. Instead they have sensibly assumed – and shown – that the link between the genes and divorce is mediated by intervening personality factors (Jockin, McGue, & Lykken, 1996). Incentive factors are goals in the outside world, such as food, water, sexual partners, and drugs. Incentives are the target of motivated behavior and are typically rewarding. Although some incentives – such as a sweet food when we are hungry – are powerful motivators by themselves, most incentives are established through learning. Many types of natural rewards may activate the brain's dopamine system. Activity in these neurons may constitute the neural basis for all incentives or 'wants'. Artificial activation of these neurons by drugs or electrical brain stimulation causes increased motivation for both natural and artificial incentives. Changes in this system, produced by repeatedly taking drugs that activate it, may partly cause the compulsive craving of addiction. Hunger has evolved to allow us to select an array of nutrients. Humans have innate taste preferences, such as for sweetness, and innate aversions, such as for bitterness, that guide our choice of foods. In addition, we may develop a wide variety of learned preferences and aversions. Homeostatic hunger signals, which arise when the body is low in caloriecontaining fuels such as glucose, produce appetite partly by causing the individual to perceive food incentives as more attractive and pleasant.

8 Hunger is largely controlled by homeostatic deficit and satiety signals. Certain neurons in the brain, especially in the brain stem and hypothalamus, detect shortages in glucose and trigger hunger. Other nutrient detectors, especially in the liver, detect increasing energy stores and trigger satiety. A satiety signal, in the form of the hormone cholecystokinin, is released from the intestines to help stop hunger and eating. Two regions of the brain are critical to hunger: the lateral hypothalamus and the ventromedial hypothalamus. Destruction of the lateral hypothalamus leads to undereating; destruction of the ventromedial hypothalamus leads to overeating. Although these regions were originally thought to be centers for hunger and satiety, hunger is not permanently destroyed by any lesion. Another interpretation of these effects is that the two regions of the hypothalamus exert reciprocal effects on the homeostatic set point for body weight. Damage to the lateral hypothalamus may lower the set point, and damage to the ventromedial hypothalamus may raise the set point. Diet drugs that alter appetite may work partly by affecting neurons in these regions of the hypothalamus. People become obese primarily because: (1) they are genetically predisposed to be overweight or (2) they overeat (for psychological reasons). The influence of genes is mediated by their effect on fat cells, metabolic rate, and set points. As for overeating and obesity, obese people tend to overeat when they break a diet, eat more when emotionally aroused, and are more responsive to external hunger cues than normal-weight individuals. In treating obesity, extreme diets appear ineffective because the deprivation leads to subsequent overeating and to a lowered metabolic rate. What seems to work best is to establish a new set of permanent eating habits and engage in a program of exercise. Anorexia nervosa is characterized by extreme, self-imposed weight loss. Bulimia is characterized by recurrent episodes of binge eating, followed by attempts to purge the excess by means of vomiting and laxatives. Possible causes of these eating disorders include personality factors such as low

www.cengagebrain.co.uk CHAPTER SUMMARY self-esteem, social factors such as a cultural emphasis on thinness and pervasive cultural messages that objectify the female body, and biological factors such as low serotonin levels. Prenatal hormones contribute to sexual development. If the embryonic sex glands produce enough androgen hormones, the embryo will have a male pattern of genital and brain development. If androgens are low or missing, the embryo will have a female pattern of genital and brain development. For nonhuman animals, prenatal hormones appear to be powerful determinants of adult sexual behavior. For humans, prenatal hormones appear to be much less important, although they may still play a role in later sexual behavior. In cases in which the hormonal exposure of the embryo is typical of one sex but the social role and gender after birth is more typical of the other sex (due to hormone imbalance, prenatal drugs, or a postnatal accident), the individual's development seems to correspond most closely to the postnatal social gender. The female hormones (estrogen and progesterone) and male hormones (androgens) are responsible for the changes in the body that occur at puberty, but they play a limited role in human sexual arousal. In contrast, in other animals there is substantial hormonal control over sex. Early social experiences with parents and peers have a large influence on adult sexuality in primates and humans. For humans, other environmental determinants of adult sexuality include cultural norms. Although Western society has become increasingly flexible regarding female and male sex roles, men and women may still differ in their attitudes toward sex and relationships. Recent studies have bolstered the claim that biological, genetic, hormonal, or neural factors may partly determine whether an individual will be heterosexual or homosexual, but the evidence is not conclusive. It is also unknown whether biological factors may influence sexual orientation directly or whether they instead contribute to other traits, such as gender conformity, that indirectly influence the development of sexual orientation.

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