Most of us have a fondness for eating and sex—the two highly esteemed motivated behaviors discussed in Chapter 12 and 13. But the amount of time devoted to these behaviors by even the most amorous gourmets pales in comparison to the amount of time spent sleeping. Most of us will sleep for well over 175,000 hours in our lifetimes. This extraordinary commitment of time implies that sleep fulfills a critical biological function. But what is it? And what about dreaming: Why do we spend so much time dreaming? And why do we tend to get sleepy at about the same time every day? Answers to these questions await you in this chapter.

Almost every time I lecture about sleep, somebody asks “How much sleep do we need?” Each time, I provide the same unsatisfying answer: I explain that there are two fundamentally different answers to this question, but neither has emerged a clear winner.

One answer stresses the presumed health-promoting and recuperative powers of sleep and suggests that people need as much sleep as they can comfortably get—the usual prescription being at least 8 hours per night. The other answer is that many of us sleep more than we need to and are consequently sleeping part of our life away. Just think how your life could change if you slept 5 hours per night instead of 8. You would have an extra 21 waking hours each week, a mind-boggling 10,952 hours each decade.

As I prepared to write this chapter, I began to think of the personal implications of the idea that we get more sleep than we need. That is when I decided to do something a bit unconventional. I am going to participate in a sleep-reduction experiment—by trying to get no more than 5 hours of sleep per night—11:00 P.M. to 4:00 A.M.—until this chapter is written. As I begin, I am excited by the prospect of having more time to write, but a little worried that this extra time might cost me too dearly.

It is now the next day—4:50 Saturday morning to be exact—and I am just sitting down to write. There was a party last night, and I didn’t make it to bed by 11:00; but considering that I slept for only 3 hours and 35 minutes, I feel quite good. I wonder what I will feel like later in the day. In any case, I will report my experiences to you at the end of the chapter.

The following case study challenges several common beliefs about sleep. Ponder its implications before proceeding to the body of the chapter.

**The Case of the Woman Who Wouldn’t Sleep**

Miss M . . . is a busy lady who finds her ration of twenty-three hours of wakefulness still insufficient for her needs. Even though she is now retired she is still busy in the community, helping sick friends whenever requested. She is an active painter and . . . writer. Although she becomes tired physically, when she needs to sit down to rest her legs, she does not ever report feeling sleepy. During the night she sits on her bed . . . reading, writing, crocheting or painting. At about 2:00 A.M. she falls asleep without any preceding drowsiness often while still holding a book in her hands. When she wakes about an hour later, she feels as wide awake as ever . . .

We invited her along to the laboratory. She came willingly but on the first evening we hit our first snag. She announced that she did not sleep at all if she had interesting things to do, and by her reckoning a visit to a university sleep laboratory counted as very interesting. Moreover, for the first time in years, she had someone to talk to for the whole of the night. So we talked.

In the morning we broke into shifts so that some could sleep while at least one person stayed with her and entertained her during the next day. The second night was a repeat performance of the first night . . .

In the end we prevailed upon her to allow us to apply EEG electrodes and to leave her sitting comfortably on the bed in the bedroom. She had promised that she would co-operate by not resisting sleep although she entertained her during the next day. The second night was a repeat performance of the first night . . .

The only substantial difference between her sleep and what we might have expected . . . was that it was of short duration . . . [After 99 minutes], she had no further interest in sleep and asked to . . . join our company again.


### 14.1 Stages of Sleep

Many changes occur in the body during sleep. This section introduces you to the major ones.

**Three Standard Psychophysiological Measures of Sleep**

There are major changes in the human EEG during the course of a night’s sleep. Although the EEG waves that accompany sleep are generally high-voltage and slow, there are periods throughout the night that are dominated by low-voltage, fast waves similar to those in nonsleeping individuals. In the 1950s, it was discovered that rapid eye movements (REMs) occur under the closed eyelids of sleepers during these periods of low-voltage, fast EEG activity. And in 1962,
Berger and Oswald discovered that there is also a loss of electromyographic activity in the neck muscles during these same sleep periods. Subsequently, the electroencephalogram (EEG), the electrooculogram (EOG), and the neck electromyogram (EMG) became the three standard psychophysiological bases for defining stages of sleep.

Figure 14.1 depicts a volunteer participating in a sleep experiment. A participant’s first night of sleep in a laboratory is often fitful. That’s why the usual practice is to have each participant sleep several nights in the laboratory before commencing a sleep study. The disturbance of sleep observed during the first night in a sleep laboratory is called the first-night phenomenon. It is well known to graders of introductory psychology examinations because of the creative definitions of it that are offered by students who forget that it is a sleep-related, rather than a sex-related, phenomenon.

Four Stages of Sleep EEG

There are four stages of sleep EEG: stage 1, stage 2, stage 3, and stage 4. Examples of these are presented in Figure 14.2.

After the eyes are shut and a person prepares to go to sleep, alpha waves—waxing and waning bursts of 8- to 12-Hz EEG waves—begin to punctuate the low-voltage, high-frequency waves of alert wakefulness. Then, as the person falls asleep, there is a sudden transition to a period of stage 1 sleep EEG. The stage 1 sleep EEG is a low-voltage, high-frequency signal that is similar to, but slower than, that of alert wakefulness.

There is a gradual increase in EEG voltage and a decrease in EEG frequency as the person progresses from stage 1 sleep through stages 2, 3, and 4. Accordingly, the stage 2 sleep EEG has a slightly
higher amplitude and a lower frequency than the stage 1 EEG; in addition, it is punctuated by two characteristic wave forms: K complexes and sleep spindles. Each K complex is a single large negative wave (upward deflection) followed immediately by a single large positive wave (downward deflection)—see Cash and colleagues (2009). Each sleep spindle is a 1- to 2-second waxing and waning burst of 12- to 14-Hz waves. The stage 3 sleep EEG is defined by the occasional presence of delta waves—the largest and slowest EEG waves, with a frequency of 1 to 2 Hz—whereas the stage 4 sleep EEG is defined by a predominance of delta waves.

Once sleepers reach stage 4 EEG sleep, they stay there for a time, and then they retreat back through the stages of sleep to stage 1. However, when they return to stage 1, things are not at all the same as they were the first time through. The first period of stage 1 EEG during a night’s sleep (initial stage 1 EEG) is not marked by any striking electromyographic or electrooculographic changes, whereas subsequent periods of stage 1 sleep EEG (emergent stage 1 EEG) are accompanied by REMs and by a loss of tone in the muscles of the body core.

After the first cycle of sleep EEG—from initial stage 1 to stage 4 and back to emergent stage 1—the rest of the night is spent going back and forth through the stages. Figure 14.3 illustrates the EEG cycles of a typical night’s sleep and the close relation between emergent stage 1 sleep, REMs, and the loss of tone in core muscles. Notice that each cycle tends to be about 90 minutes long and that, as the night progresses, more and more time is spent in emergent stage 1 sleep, and less and less time is spent in the other stages, particularly stage 4. Notice also that there are brief periods during the night when the person is awake, although he or she usually does not remember these periods of wakefulness in the morning.

Let’s pause here to get some sleep-stage terms straight. The sleep associated with emergent stage 1 EEG is usually called REM sleep (pronounced “rehm”), after the associated rapid eye movements; whereas all other stages of sleep together are called NREM sleep (non-REM sleep). Stages 3 and 4 together are referred to as slow-wave sleep (SWS), after the delta waves that characterize them.

REMs, loss of core-muscle tone, and a low-amplitude, high-frequency EEG are not the only physiological correlates of REM sleep. Cerebral activity (e.g., oxygen consumption, blood flow, and neural firing) increases to waking levels in many brain structures, and there is a general increase in the variability of autonomic nervous system activity (e.g., in blood pressure, pulse, and respiration). Also, the muscles of the extremities occasionally twitch, and there is often some degree of penile erection in males.

**REM Sleep and Dreaming**

Nathaniel Kleitman’s laboratory was an exciting place in 1953. REM sleep had just been discovered, and Kleitman and his students were driven by the fascinating implication of the discovery. With the exception of the loss of tone in the core muscles, all of the other measures suggested that REM sleep episodes were emotion-charged. Could REM sleep be the physiological correlate of dreaming? Could it provide researchers with a window into the subjective inner world of dreams? The researchers began by waking a few sleepers in the middle of REM episodes:

The vivid recall that could be elicited in the middle of the night when a subject was awakened while his eyes were moving rapidly was nothing short of miraculous. It seemed to open . . . an exciting new world to the subjects whose only previous dream memories had been the vague morning-after recall. Now, instead of perhaps some fleeting glimpse into the dream world each night, the subjects could be tuned into the middle of as many as ten or twelve dreams every night. (From Some Must Watch While Some Must Sleep by William C. Dement, Portable Stanford Books, Stanford Alumni Association, Stanford University, 1978, p. 37. Used by permission of William C. Dement.)

Strong support for the theory that REM sleep is the physiological correlate of dreaming came from the observation that 80% of awakenings from REM sleep but only 7% of awakenings from NREM (non-REM) sleep led to dream recall. The dreams recalled from NREM sleep tended to
be isolated experiences (e.g., “I was falling”), while those associated with REM sleep tended to take the form of stories, or narratives. The phenomenon of dreaming, which for centuries had been the subject of wild speculation, was finally rendered accessible to scientific investigation.

**Testing Common Beliefs about Dreaming**

The high correlation between REM sleep and dream recall provided an opportunity to test some common beliefs about dreaming. The following five beliefs were among the first to be addressed:

- Many people believe that external stimuli can become incorporated into their dreams. Dement and Wolpert (1958) sprayed water on sleeping volunteers after they had been in REM sleep for a few minutes, and then awakened them a few seconds later. In 14 of 33 cases, the water was incorporated into the dream report. The following narrative was reported by one participant who had been dreaming that he was acting in a play:

  I was walking behind the leading lady when she suddenly collapsed and water was dripping on her. I ran over to her and water was dripping on my back and head. The roof was leaking....I looked up and there was a hole in the roof. I dragged her over to the side of the stage and began pulling the curtains. Then I woke up. (p. 550)

- Some people believe that dreams last only an instant, but research suggests that dreams run on “real time.” In one study (Dement & Kleitman, 1957), volunteers were awakened 5 or 15 minutes after the beginning of a REM episode and asked to decide on the basis of the duration of the events in their dreams whether they had been dreaming for 5 or 15 minutes. They were correct in 92 of 111 cases.

- Some people claim that they do not dream. However, these people have just as much REM sleep as normal dreamers. Moreover, they report dreams if they are awakened during REM episodes (Goodenough et al., 1959), although they do so less frequently than do normal dreamers.

- Penile erections are commonly assumed to be indicative of dreams with sexual content. However, erections are no more complete during dreams with frank sexual content than during those without it (Karacan et al., 1966). Even babies have REM-related penile erections.

- Many people believe that sleepwalking (somnambulism) and sleepwalking (somnambulism) occur only during dreaming. This is not so (see Dyken, Yamada, & Lin-Dyken, 2001). Sleepwalking has no special association with REM sleep—it can occur during any stage but often occurs during a transition to wakefulness. Sleepwalking usually occurs during stage 3 or 4 sleep, and it never occurs during dreaming, when core muscles tend to be totally relaxed (Usui et al., 2007). There is no proven treatment for sleepwalking (Harris & Grunstein, 2008).

**Interpretation of Dreams**

Sigmund Freud believed that dreams are triggered by unacceptable repressed wishes, often of a sexual nature. He argued that because dreams represent unacceptable wishes, the dreams we experience (our manifest dreams) are merely disguised versions of our real dreams (our latent dreams): He hypothesized an unconscious censor that disguises and subtracts information from our real dreams so that we can endure them. Freud thus concluded that one of the keys to understanding people and dealing with their psychological problems is to expose the meaning of their latent dreams through the interpretation of their manifest dreams.

There is no convincing evidence for the Freudian theory of dreams; indeed, the brain science of the 1890s, which served as its foundation, is now obsolete. Yet many people accept the notion that dreams bubble up from a troubled subconscious and that they represent repressed thoughts and wishes.

The modern alternative to the Freudian theory of dreams is Hobson’s (1989) activation-synthesis theory (see Eiser, 2005). It is based on the observation that, during REM sleep, many brain-stem circuits become active and bombard the cerebral cortex with neural signals. The essence of the activation-synthesis theory is that the information supplied to the cortex during REM sleep is largely random and that the resulting dream is the cortex’s effort to make sense of these random signals.

**Why Do We Sleep, and Why Do We Sleep When We Do?**

Now that you have been introduced to the properties of sleep and its various stages, the focus of this chapter shifts to a consideration of two fundamental questions about sleep: Why do we sleep? And why do we sleep when we do?

Two kinds of theories for sleep have been proposed: recuperation theories and adaptation theories. The differences between these two theoretical approaches are revealed by the answers they offer to the two fundamental questions about sleep.

The essence of recuperation theories of sleep is that being awake disrupts the homeostasis (internal physiological stability) of the body in some way and sleep is required to restore it. Various recuperation theories differ in terms of the particular physiological disruption they...
proposes as the trigger for sleep—for example, it is commonly believed that the function of sleep is to restore energy levels. However, regardless of the particular function postulated by restoration theories of sleep, they all imply that sleepiness is triggered by a deviation from homeostasis caused by wakefulness and that sleep is terminated by a return to homeostasis.

The essence of adaptation theories of sleep is that sleep is not a reaction to the disruptive effects of being awake but the result of an internal 24-hour timing mechanism—that is, we humans are programmed to sleep at night regardless of what happens to us during the day. According to these theories, we have evolved to sleep at night because sleep protects us from accident and predation during the night. (Remember that humans evolved long before the advent of artificial lighting.)

Adaptation theories of sleep focus more on when we sleep than on the function of sleep. Some of these theories even propose that sleep plays no role in the efficient physiological functioning of the body. According to these theories, early humans had enough time to get their eating, drinking, and reproducing out of the way during the daytime, and their strong motivation to sleep at night evolved to conserve their energy resources and to make them less susceptible to mishap (e.g., predation) in the dark (Rattenborg, Martinez-Gonzales, & Lesku, 2009; Siegel, 2009). Adaptation theories suggest that sleep is like reproductive behavior in the sense that we are highly motivated to engage in it, but we don’t need it to stay healthy.

Comparative Analysis of Sleep

Sleep has been studied in only a small number of species, but the evidence so far suggests that most mammals and birds sleep. Furthermore, the sleep of mammals and birds, like ours, is characterized by high-amplitude, low-frequency EEG waves punctuated by periods of low-amplitude, high-frequency waves (see Siegel, 2008). The evidence for sleep in amphibians, reptiles, fish, and insects is less clear: Some display periods of inactivity and unresponsiveness, but the relation of these periods to mammalian sleep has not been established (see Siegel, 2008; Zimmerman et al., 2008). Table 14.1 gives the average number of hours per day that various mammalian species spend sleeping.

For example, some marine mammals, such as dolphins, sleep with only half of their brain at a time so that the other half can control resurfacing for air (see Rattenborg, Amlaner, & Lima, 2000). It is against the logic of natural selection for some animals to risk predation while sleeping and for others to have evolved complex mechanisms to permit them to sleep, unless sleep itself serves some critical function.

Second, the fact that most mammals and birds sleep suggests that the primary function of sleep is not some special, higher-order human function. For example, suggestions that sleep helps humans reprogram our complex brains or that it permits some kind of emotional release to maintain our mental health are improbable in view of the comparative evidence.

Third, the large between-species differences in sleep time suggest that although sleep may be essential for survival, it is not necessarily needed in large quantities (refer to Table 14.1). Horses and many other animals get by quite nicely on 2 or 3 hours of sleep per day. Moreover, it is important to realize that the sleep patterns of mammals and birds in their natural environments can vary substantially from their patterns in captivity, which is where they are typically studied (see Horne, 2009). For example, some animals that sleep a great deal in captivity sleep little in the wild when food is in short supply or during periods of migration (Siegel, 2008).

<table>
<thead>
<tr>
<th>Mammalian Species</th>
<th>Hours of Sleep per Day</th>
</tr>
</thead>
<tbody>
<tr>
<td>Giant sloth</td>
<td>20</td>
</tr>
<tr>
<td>Opossum, brown bat</td>
<td>19</td>
</tr>
<tr>
<td>Giant armadillo</td>
<td>18</td>
</tr>
<tr>
<td>Owl monkey, nine-banded armadillo</td>
<td>17</td>
</tr>
<tr>
<td>Arctic ground squirrel</td>
<td>16</td>
</tr>
<tr>
<td>Tree shrew</td>
<td>15</td>
</tr>
<tr>
<td>Cat, golden hamster</td>
<td>14</td>
</tr>
<tr>
<td>Mouse, rat, gray wolf, ground squirrel</td>
<td>13</td>
</tr>
<tr>
<td>Arctic fox, chinchilla, gorilla, raccoon</td>
<td>12</td>
</tr>
<tr>
<td>Mountain beaver</td>
<td>11</td>
</tr>
<tr>
<td>Jaguar, vervet monkey, hedgehog</td>
<td>10</td>
</tr>
<tr>
<td>Rhesus monkey, chimpanzee, baboon, red fox</td>
<td>9</td>
</tr>
<tr>
<td>Human, rabbit, guinea pig, pig</td>
<td>8</td>
</tr>
<tr>
<td>Gray seal, gray hyrax, Brazilian tapir</td>
<td>6</td>
</tr>
<tr>
<td>Tree hyrax, rock hyrax</td>
<td>5</td>
</tr>
<tr>
<td>Cow, goat, elephant, donkey, sheep</td>
<td>3</td>
</tr>
<tr>
<td>Roe deer, horse</td>
<td>2</td>
</tr>
</tbody>
</table>
Fourth, many studies have tried to identify some characteristic that identifies various species as long sleepers or short sleepers. Why do cats tend to sleep about 14 hours a day and horses only about 2? Under the influence of recuperation theories, researchers have focused on energy-related factors in their efforts. However, there is no strong relationship between a species’ sleep time and its level of activity, its body size, or its body temperature (see Siegel, 2005). The fact that giant sloths sleep 20 hours per day is a strong argument against the theory that sleep is a compensatory reaction to energy expenditure—similarly, energy expenditure has been shown to have little effect on subsequent sleep in humans (Driver & Taylor, 2000; Youngstedt & Kline, 2006). In contrast, adaptation theories correctly predict that the daily sleep time of each species is related to how vulnerable it is while it is asleep and how much time it must spend each day to feed itself and to take care of its other survival requirements. For example, zebras must graze almost continuously to get enough to eat and are extremely vulnerable to predatory attack when they are asleep—and they sleep only about 2 hours per day. In contrast, African lions often sleep more or less continuously for 2 or 3 days after they have gorged themselves on a kill. Figure 14.4 says it all.

**Figure 14.4** After gorging themselves on a kill, African lions often sleep almost continuously for 2 or 3 days. And where do they sleep? Anywhere they want!

Interpretation of the Effects of Sleep Deprivation: The Stress Problem

I am sure that you have experienced the negative effects of sleep loss. When you sleep substantially less than you are used to, the next day you feel out of sorts and unable to function as well as you usually do. Although such experiences of sleep deprivation are compelling, you need to be cautious in interpreting them. In Western cultures, most people who sleep little or irregularly do so because they are under extreme stress (e.g., from illness, excessive work, shift work, drugs, or examinations), which could have adverse effects independent of any sleep loss. Even when sleep deprivation studies are conducted on healthy volunteers in controlled laboratory environments, stress can be a contributing factor because many of the volunteers will find the sleep-deprivation procedure itself stressful. Because it is difficult to separate the effects of sleep loss from the effects of stressful conditions that may have induced the loss, results of sleep-deprivation studies must be interpreted with particular caution.

Unfortunately, many studies of sleep deprivation, particularly those that are discussed in the popular media, do not control for stress. For example, almost weekly I read an article in my local newspaper decrying the effects of sleep loss in the general population. It will point out that many people who are pressured by the demands of their work schedule sleep little and experience a variety of health and accident problems. There is a place for this kind of research because it identifies a problem that requires public attention; however, because the low levels of sleep are hopelessly confounded with high levels of stress, many sleep-deprivation studies tell us little about the functions of sleep and how much we need.

Predictions of Recuperation Theories about Sleep Deprivation

Because recuperation theories of sleep are based on the premise that sleep is a response to the accumulation of some debilitating effect of wakefulness, they make the following three predictions about sleep deprivation:

- Long periods of wakefulness will produce physiological and behavioral disturbances.
- These disturbances will grow steadily worse as the sleep deprivation continues.
- After a period of deprivation has ended, much of the missed sleep will be regained.

Have these predictions been confirmed?
Two Classic Sleep-Deprivation Case Studies

Let’s look at two widely cited sleep-deprivation case studies. First is the study of a group of sleep-deprived students, described by Kleitman (1963); second is the case of Randy Gardner, described by Dement (1978).

The Case of the Sleep-Deprived Students

While there were differences in the many subjective experiences of the sleep-evading persons, there were several features common to most. During the first night the subject did not feel very tired or sleepy. He could read or study or do laboratory work, without much attention from the watcher, but usually felt an attack of drowsiness between 3 A.M. and 6 A.M. Next morning the subject felt well, except for a slight malaise which always appeared on sitting down and resting for any length of time. However, if he occupied himself with his ordinary daily tasks, he was likely to forget having spent a sleepless night. During the second night reading or study was next to impossible because sitting quietly was conducive to even greater sleepiness. As during the first night, there came a 2–3-hour period in the early hours of the morning when the desire for sleep was almost overpowering. Later in the morning the sleepiness diminished once more, and the subject could perform routine laboratory work, as usual. It was not safe for him to sit down, however, without danger of falling asleep, particularly if he attended lectures.

The third night resembled the second, and the fourth day was like the third. At the end of that time the individual was as sleepy as he was likely to be. Those who continued to stay awake experienced the wavelike increase and decrease in sleepiness with the greatest drowsiness at about the same time every night. (Kleitman, 1963, pp. 220–221)

The Case of Randy Gardner

As part of a 1965 science fair project, Randy Gardner and two classmates, who were entrusted with keeping him awake, planned to break the then world record of 260 hours of consecutive wakefulness. Dement read about the project in the newspaper and, seeing an opportunity to collect some important data, joined the team, much to the comfort of Randy’s worried parents. Randy proved to be a friendly and cooperative subject, although he did complain vigorously when his team would not permit him to close his eyes for more than a few seconds at a time. However, in no sense could Randy’s behavior be considered abnormal or disturbed. Near the end of his vigil, Randy held a press conference attended by reporters and television crews from all over the United States, and he conducted himself impeccably. When asked how he had managed to stay awake for 11 days, he replied politely, “It’s just mind over matter.” Randy went to sleep exactly 264 hours and 12 minutes after his alarm clock had awakened him 11 days before. And how long did he sleep? Only 14 hours the first night, and thereafter he returned to his usual 8-hour schedule. Although it may seem amazing that Randy did not have to sleep longer to “catch up” on his lost sleep, the lack of substantial recovery sleep is typical of such cases.

Experimental Studies of Sleep Deprivation in Humans

Since the first studies of sleep deprivation by Dement and Kleitman in the mid-20th century, there have been hundreds of studies assessing the effects on humans of sleep-deprivation schedules ranging from a slightly reduced amount of sleep during one night to total sleep deprivation for several nights (see Durmer & Dinges, 2005). The studies have assessed the effects of these schedules on many different measures of sleepiness, mood, cognition, motor performance, physiological function, and even molecular function (see Cirelli, 2006).

Even moderate amounts of sleep deprivation—for example, sleeping 3 or 4 hours less than normal for one night—have been found to have three consistent effects. First, sleep-deprived individuals display an increase in sleepiness: They report being more sleepy, and they fall asleep more quickly if given the opportunity. Second, sleep-deprived individuals display negative affect on various written tests of mood. And third, they perform poorly on tests of vigilance, such as watching a computer screen and responding when a moving light flickers.

The effects of sleep deprivation on complex cognitive functions have been less consistent (see Drummond et al., 2004). Consequently, researchers have preferred to assess performance on the simple, dull, monotonous tasks most sensitive to the effects of sleep deprivation (see Harrison & Horne, 2000). Nevertheless, a growing number of studies have been able to demonstrate disruption of the performance of complex cognitive tasks by sleep deprivation (Blagrove, Alexander, & Horne, 2006; Durmer & Dinges, 2005; Killgore, Balkin, & Wesensten, 2006; Nilsson et al., 2005) although a substantial amount of sleep deprivation (e.g., 24 hours) has often been required to produce consistent disruption (e.g., Killgore, Balkin, & Wesensten, 2006; Strangman et al., 2005).

The disruptive impact of sleep deprivation on cognitive function has been clarified by the discovery that only some cognitive functions are susceptible. Many early studies of...
the effect of sleep deprivation on cognitive function used tests of logical deduction or critical thinking, and performance on these has proved to have been largely immune to the disruptive effects of sleep loss. In contrast, performance on tests of executive function (cognitive abilities that appear to depend on the prefrontal cortex) has proven much more susceptible (see Nilsson et al., 2005). Executive function includes innovative thinking, lateral thinking, insightful thinking, and assimilating new information to update plans and strategies.

The adverse effects of sleep deprivation on physical performance have been surprisingly inconsistent considering the general belief that a good night’s sleep is essential for optimal motor performance. Only a few measures tend to be affected, even after lengthy periods of deprivation (see Van Helder & Radomski, 1989).

Sleep deprivation has been found to have a variety of physiological consequences such as reduced body temperature, increases in blood pressure, decreases in some aspects of immune function, hormonal changes, and metabolic changes (e.g., Dinges et al., 1994; Kato et al., 2000; Knutson et al., 2007; Ogawa et al., 2003). The problem is that there is little evidence that these changes have any consequences for health or performance. For example, the fact that a decline in immune function was discovered in sleep-deprived volunteers does not necessarily mean that they would be more susceptible to infection—the immune system is extremely complicated and a decline in one aspect can be compensated for by other changes. This is why I want to single out a study by Cohen and colleagues (2009) for commendation: Rather than studying immune function, these researchers focused directly on susceptibility to infection and illness. They exposed 153 healthy volunteers to a cold virus. Those who reported sleeping less than 8 hours a night were not less likely to become infected, but they were more likely to develop cold symptoms. Although this is only a correlational study (see Chapter 1) and thus cannot directly implicate sleep duration as the causal factor, experimental studies of sleep and infectious disease need to follow this example and directly measure susceptibility to infection and illness.

After 2 or 3 days of continuous sleep deprivation, most study participants experience microsleeps, unless they are in a laboratory environment where the microsleeps can be interrupted as soon as they begin. Microsleeps are brief periods of sleep, typically about 2 or 3 seconds long, during which the eyelids droop and the subjects become less responsive to external stimuli, even though they remain sitting or standing. Microsleeps disrupt performance on tests of vigilance, but such performance deficits also occur in sleep-deprived individuals who are not experiencing microsleeps (Ferrara, De Gennaro, & Bertini, 1999).

It is useful to compare the effects of sleep deprivation with those of deprivation of the motivated behaviors discussed in Chapters 12 and 13. If people were deprived of the opportunity to eat or engage in sexual activity, the effects would be severe and unavoidable: In the first case, starvation and death would ensue; in the second, there would be a total loss of reproductive capacity. Despite our powerful drive to sleep, the effects of sleep deprivation tend to be subtle, selective, and variable. This is puzzling. Another puzzling thing is that performance deficits observed after extended periods of sleep deprivation disappear so readily—for example, in one study, 4 hours of sleep eliminated the performance deficits produced by 64 hours of sleep deprivation (Rosa, Bonnett, & Warm, 2007).

**Sleep-Deprivation Studies with Laboratory Animals**

The carousel apparatus (see Figure 14.5) has been used to deprive rats of sleep. Two rats, an experimental rat and its yoked control, are placed in separate chambers of the apparatus. Each time the EEG activity of the experimental rat indicates that it is sleeping, the disk, which serves as the floor of half of both chambers, starts to slowly rotate. As a result, if the sleeping experimental rat does not awaken immediately, it gets shoved off the disk into a shallow pool of water. The yoked control is exposed to exactly the same pattern of disk rotations; but if it is not sleeping, it can easily avoid getting dunked by walking in the direction opposite to the direction of disk rotation. The experimental rats typically died after about 12 days.
while the yoked controls stayed reasonably healthy (see Rechtschaffen & Bergmann, 1995).

The fact that humans and rats have been sleep-deprived by other means for similar periods of time without dire consequences argues for caution in interpreting the results of the carousel sleep-deprivation experiments (see Rial et al., 2007; Siegel, 2009). It may be that repeatedly being awakened by this apparatus kills the experimental rats not because it keeps them from sleeping but because it is stressful. This interpretation is consistent with the pathological problems in the experimental rats that were revealed by postmortem examination: swollen adrenal glands, gastric ulcers, and internal bleeding.

You have already encountered many examples in this book of the value of the comparative approach. However, sleep deprivation may be one phenomenon that cannot be productively studied in nonhumans because of the unavoidable confounding effects of extreme stress (see Benington & Heller, 1999; D’Almeida et al., 1997; Horne, 2000).

**REM-Sleep Deprivation**

Because of its association with dreaming, REM sleep has been the subject of intensive investigation. In an effort to reveal the particular functions of REM sleep, sleep researchers have specifically deprived sleeping volunteers of REM sleep by waking them up each time a bout of REM sleep begins.

REM-sleep deprivation has been shown to have two consistent effects (see Figure 14.6). First, following REM-sleep deprivation, participants display a REM rebound; that is, they have more than their usual amount of REM sleep for the first two or three nights (Brunner et al., 1990). Second, with each successive night of deprivation, there is a greater tendency for participants to initiate REM sequences. Thus, as REM-sleep deprivation proceeds, participants have to be awakened more and more frequently to keep them from accumulating significant amounts of REM sleep. For example, during the first night of REM-sleep deprivation in one experiment (Webb & Agnew, 1967), the participants had to be awakened 17 times to keep them from having extended periods of REM sleep; but during the seventh night of deprivation, they had to be awakened 67 times.

The compensatory increase in REM sleep following a period of REM-sleep deprivation suggests that the amount of REM sleep is regulated separately from the amount of slow-wave sleep and that REM sleep serves a special function. This finding, coupled with the array of interesting physiological and psychological events that define REM sleep, has led to much speculation about its function.

Considerable attention has focused on the potential role of REM sleep in strengthening explicit memory (see Chapter 11). Many reviewers of the literature on this topic have treated the positive effect of REM sleep on the storage of existing memories as well established, and researchers have moved on to study the memory-promoting effects of other stages of sleep (e.g., Deak & Stickgold, 2010; Rasch & Born, 2008; Stickgold & Walker, 2007) and the physiological mechanisms of these memory-promoting effects (e.g., Rasch et al., 2007). However, two eminent sleep researchers,
Robert Vertes and Jerome Seigel (2005), have argued that the evidence that REM sleep strengthens memory is unconvincing (see Vertes, 2004). They point out, for example, that numerous studies failing to support a mnemonic (pertaining to memory) function of REM sleep have been ignored. They also question why the many patients who have taken antidepressant drugs that block REM sleep experience no obvious memory problems, even if they have taken the drugs for months or even years. In one study, pharmacologically blocking REM sleep in human volunteers did not disrupt consolidation of verbal memories, and it actually improved the consolidation of the memory of motor tasks (Rasch et al., 2008).

The default theory of REM sleep is a different approach (Horne, 2000). According to this theory, it is difficult to stay continuously in NREM sleep, so the brain periodically switches to one of two other states. If there is any immediate bodily need to take care of (e.g., eating or drinking), the brain switches to wakefulness; if there are no immediate needs, it switches to the default state—REM sleep. According to the default theory, REM sleep and wakefulness are similar states, but REM sleep is more adaptive when there are no immediate bodily needs. Indirect support for this theory comes from the many similarities between REM sleep and wakefulness.

A study by Nykamp and colleagues (1998) supported the default theory of REM sleep. The researchers awakened young adults every time they entered REM sleep, but instead of letting them go back to sleep immediately, substituted a 15-minute period of wakefulness for each lost REM period. Under these conditions, the participants, unlike the controls, were not tired the next day, despite getting only 5 hours of sleep, and they displayed no REM rebound. In other words, there seemed to be no need for REM sleep if periods of wakefulness were substituted for it. This finding has been replicated in rats (Oniani & Lortkipanidze, 2003), and it is consistent with the finding that as antidepressants reduce REM sleep, the number of nighttime awakenings increases (see Horne, 2000).

**Sleep Deprivation Increases the Efficiency of Sleep**

One of the most important findings of human sleep-deprivation research is that individuals who are deprived of sleep become more efficient sleepers (see Elmenhorst et al., 2008). In particular, their sleep has a higher proportion of slow-wave sleep (stages 3 and 4), which seems to serve the main restorative function. Because this is such an important finding, let’s look at six major pieces of evidence that support it:

- Although people regain only a small proportion of their total lost sleep after a period of sleep deprivation, they regain most of their lost stage 4 sleep (e.g.,

  - After sleep deprivation, the slow-wave EEG of humans is characterized by an even higher proportion than usual of slow waves (Aeschbach et al., 1996; Borbély, 1981; Borbély et al., 1981).
  - People who sleep 6 hours or less per night normally get as much slow-wave sleep as people who sleep 8 hours or more (e.g., Jones & Oswald, 1966; Webb & Agnew, 1970).
  - If individuals take a nap in the morning after a full night’s sleep, their naptime EEG shows few slow waves, and the nap does not reduce the duration of the following night’s sleep (e.g., Åkerstedt & Gillberg, 1981; Hume & Mills, 1977; Karacan et al., 1970).
  - People who gradually reduce their usual sleep time get less stage 1 and stage 2 sleep, but the duration of their slow-wave sleep remains about the same as before (Mullaney et al., 1977; Webb & Agnew, 1975).
  - Repeatedly waking individuals during REM sleep produces little increase in the sleepiness they experience the next day, whereas repeatedly waking individuals during slow-wave sleep has major effects (Nykamp et al., 1998).

The fact that sleep becomes more efficient in people who sleep less means that conventional sleep-deprivation studies are virtually useless for discovering how much sleep people need. Certainly, our bodies respond negatively when we get less sleep than we are used to getting. However, the negative consequences of sleep loss in inefficient sleepers does not indicate whether the lost sleep was really needed. The true need for sleep can be assessed only by experiments in which sleep is regularly reduced for many weeks, to give the participants the opportunity to adapt to getting less sleep by maximizing their sleep efficiency. Only when people are sleeping at their maximum efficiency is it possible to determine how much sleep they really need. Such sleep-reduction studies are discussed later in the chapter, but please pause here to think about this point—it is extremely important, and it is totally consistent with the growing appreciation of the plasticity and adaptiveness of the adult mammalian brain.

This is an appropriate time, here at the end of the section on sleep deprivation, for me to file a brief progress report. It has now been 2 weeks since I began my 5-hours-per-night sleep schedule. Generally, things are going well. My progress on this chapter has been faster than usual. I am not having any difficulty getting up on time or getting my work done, but I am finding that it takes a major effort to stay awake in the evening. If I try to read or watch a bit of television after 10:30, I experience microsleeps. My so-called friends delight in making sure that my transgressions are quickly interrupted.
rats, sleep for much of the day and stay awake at night. Kreitzman, 2004; circadian
rhythms with a variety of
back again once every 24 hours, and most surface-dwelling
The world in which we live cycles from light to dark and
nocturnal animals
much of the night; in contrast,
sleep–wake cycle. Humans take advantage of the light of day
to take care of their biological needs, and then they sleep for
much of the night; in contrast, nocturnal animals, such as
rats, sleep for much of the day and stay awake at night.

Although the sleep–wake cycle is the most obvious circadian rhythm, it is difficult to find a physiological, biochemical, or behavioral process in animals that does not display some measure of circadian rhythmicity (Gillette & Sejnowski, 2005). Each day, our bodies adjust themselves in a variety of ways to meet the demands of the two environments in which we live: light and dark.

Our circadian cycles are kept on their once-every-24-hours schedule by temporal cues in the environment. The most important of these cues for the regulation of mammalian circadian rhythms is the daily cycle of light and dark. Environmental cues, such as the light–dark cycle, that can entrain (control the timing of) circadian rhythms are called zeitgebers (pronounced “ZITE-gay-bers”), a German word that means “time givers.” In controlled laboratory environments, it is possible to lengthen or shorten circadian cycles somewhat by adjusting the duration of the light–dark cycle; for example, when exposed to alternating 11.5-hour periods of light and 11.5-hour periods of dark, subjects’ circadian cycles begin to conform to a 23-hour day. In a world without 24-hour cycles of light and dark, other zeitgebers can entrain circadian cycles. For example, the circadian sleep–wake cycles of hamsters living in continuous darkness or in continuous light can be entrained by regular daily bouts of social interaction, hoarding, eating, or exercise (see Mistlberger et al., 1996; Sinclair & Mistlberger, 1997). Hamsters display particularly clear circadian cycles and thus are frequent subjects of research on circadian rhythms.

Free-Running Circadian Sleep–Wake Cycles
What happens to sleep–wake cycles and other circadian rhythms in an environment that is devoid of zeitgebers? Remarkably, under conditions in which there are absolutely no temporal cues, humans and other animals maintain all of their circadian rhythms. Circadian rhythms in constant environments are said to be free-running rhythms, and their duration is called the free-running period. Free-running periods vary in length from subject to subject, are of relatively constant duration within a given subject, and are usually longer than 24 hours—about 24.2 hours is typical in humans living under constant moderate illumination (see Czeizler et al., 1999). It seems that we all have an internal biological clock that habitually runs a little slow unless it is entrained by time-related cues in the environment.

A typical free-running circadian sleep–wake cycle is illustrated in Figure 14.7. Notice its regularity. Without any external cues, this man fell asleep at intervals of approximately 25.3 hours for an entire month. The regularity of free-running sleep–wake cycles despite variations in physical and mental activity provides support for the dominance of circadian factors over recuperative factors in the regulation of sleep.

Free-running circadian cycles do not have to be learned. Even rats that are born and raised in an unchanging

Scan Your Brain

Before continuing with this chapter, scan your brain by completing the following exercise to make sure you understand the fundamentals of sleep. The correct answers appear at the end of the exercise. Before proceeding, review material related to your errors and omissions.

1. The three standard psychophysiological measures of sleep are the EEG, the EMG, and the _____.
2. Stage 4 sleep EEG is characterized by a predominance of _____ waves.
3. _____ stage 1 EEG is accompanied by neither REM nor loss of core-muscle tone.
4. Dreaming occurs predominantly during _____ sleep.
5. The modern alternative to Freud’s theory of dreaming is Hobson’s _____ theory.
6. There are two fundamentally different kinds of theories of sleep: recuperation theories and ________ theories.
7. The effects of sleep deprivation are often difficult to study because they are often confounded by ______.
8. Convincing evidence that REM-sleep deprivation does not produce severe memory problems comes from the study of patients taking certain _____ drugs.
9. After a lengthy period of sleep deprivation (e.g., several days), a person’s first night of sleep is only slightly longer than usual, but it contains a much higher proportion of _____ waves.
10. _____ sleep in particular, rather than sleep in general, appears to play the major restorative role.

14.4 Circadian Sleep Cycles
The world in which we live cycles from light to dark and back again once every 24 hours, and most surface-dwelling species have adapted to this regular change in their environment with a variety of circadian rhythms (see Foster & Kreitzman, 2004; circadian means “lasting about a day”). For example, most species display a regular circadian sleep–wake cycle. Humans take advantage of the light of day to take care of their biological needs, and then they sleep for much of the night; in contrast, nocturnal animals, such as rats, sleep for much of the day and stay awake at night.
laboratory environment (in continuous light or in continuous darkness) display regular free-running sleep–wake cycles that are slightly longer than 24 hours (Richter, 1971).

Many animals display a circadian cycle of body temperature that is related to their circadian sleep–wake cycle: They tend to sleep during the falling phase of their circadian body temperature cycle and awaken during its rising phase. However, when subjects are housed in constant laboratory environments, their sleep–wake and body temperature cycles sometimes break away from one another. This phenomenon is called internal desynchronization (see De La Iglesia, Cambras, & Díez-Noguera, 2008). For example, in one human volunteer, the free-running periods of both the sleep–wake and body temperature cycles were initially 25.7 hours; then, for some unknown reason, there was an increase in the free-running period of the sleep–wake cycle to 33.4 hours and a decrease in the free-running period of the body temperature cycle to 25.1 hours. The potential for the simultaneous existence of two different free-running periods suggests that there is more than one circadian timing mechanism, and that sleep is not causally related to the decreases in body temperature that are normally associated with it.

There is another point about free-running circadian sleep–wake cycles that is incompatible with recuperation theories of sleep. On occasions when subjects stay awake longer than usual, the following sleep time is shorter rather than longer (Wever, 1979). Humans and other animals are programmed to have sleep–wake cycles of approximately 24 hours; hence, the more wakefulness there is during a cycle, the less time there is for sleep.

**Jet Lag and Shift Work**

People in modern industrialized societies are faced with two different disruptions of circadian rhythmicity: jet lag and shift work. **Jet lag** occurs when the zeitgebers that control the phases of various circadian rhythms are accelerated during east-bound flights (phase advances) or decelerated during west-bound flights (phase delays). In **shift work**, the zeitgebers stay the same, but workers are forced to adjust their natural sleep–wake cycles in order to meet the demands of changing work schedules. Both of these disruptions produce sleep disturbances, fatigue, general malaise, and deficits on tests of physical and cognitive function. The disturbances can last for many days; for example, it typically takes about 10 days to completely adjust to the phase advance of 10.5 hours that one experiences on a Tokyo-to-Boston flight.

What can be done to reduce the disruptive effects of jet lag and shift work? Two behavioral approaches have been proposed for the reduction of jet lag. One is gradually shifting one’s sleep–wake cycle in the days prior to the flight. The other is administering treatments after the flight that promote the required shift in the circadian rhythm. For example, exposure to intense light early in the morning following an east-bound flight accelerates adaptation to the phase advance. Similarly, the results of a study of hamsters (Mrosovsky & Salmon, 1987) suggest that a good workout early in the morning of the first day after an east-bound flight might accelerate adaptation to the phase advance; hamsters that engaged in one 3-hour bout of wheel running 7 hours before their usual period of activity adapted quickly to an 8-hour advance in their light–dark cycle (see Figure 14.8 on page 368).

Companies that employ shift workers have had success in improving the productivity and job satisfaction of those workers by scheduling phase delays rather than phase advances; whenever possible, shift workers are transferred from their current schedule to one that begins later in the day (see Driscoll, Grunstein, & Rogers, 2007). It is much more difficult to go to sleep 4 hours earlier and
get up 4 hours earlier (a phase advance) than it is to go to sleep 4 hours later and get up 4 hours later (a phase delay). This is also why east-bound flights tend to be more problematic for travelers than west-bound flights.

A Circadian Clock in the Suprachiasmatic Nuclei

The fact that circadian sleep–wake cycles persist in the absence of temporal cues from the environment indicates that the physiological systems that regulate sleep are controlled by an internal timing mechanism—the circadian clock.

The first breakthrough in the search for the circadian clock was Richter’s 1967 discovery that large medial hypothalamic lesions disrupt circadian cycles of eating, drinking, and activity in rats. Next, specific lesions of the suprachiasmatic nuclei (SCN) of the medial hypothalamus were shown to disrupt various circadian cycles, including sleep–wake cycles. Although SCN lesions do not greatly affect the amount of time mammals spend sleeping, they do abolish the circadian periodicity of sleep cycles. Further support for the conclusion that the suprachiasmatic nuclei contain a circadian timing mechanism comes from the observation that the nuclei display circadian cycles of electrical, metabolic, and biochemical activity that can be entrained by the light–dark cycle (see Mistlberger, 2005; Saper et al., 2005).

If there was any lingering doubt about the location of the circadian clock, it was eliminated by the brilliant experiment of Ralph and his colleagues (1990). They removed the SCN from the fetuses of a strain of mutant hamsters that had an abnormally short (20-hour) free-running sleep–wake cycle. Then, they transplanted the SCN into normal adult hamsters whose free-running sleep–wake cycles of 25 hours had been abolished by SCN lesions. These transplants restored free-running sleep–wake cycles in the recipients; but, remarkably, the cycles were about 20 hours long rather than the original 25 hours. Transplants in the other direction—that is, from normal hamster fetuses to SCN-lesioned adult mutants—had the complementary effect: They restored free-running sleep–wake cycles that were about 25 hours long rather than the original 20 hours.

Although the suprachiasmatic nuclei are unquestionably the major circadian clocks in mammals, they are not
the only ones (e.g., Tosini et al., 2008). Three lines of experiments, largely conducted in the 1980s and 1990s, pointed to the existence of other circadian timing mechanisms:

- Under certain conditions, bilateral SCN lesions have been shown to leave some circadian rhythms unaffected while abolishing others.
- Bilateral SCN lesions do not eliminate the ability of all environmental stimuli to entrain circadian rhythms; for example, SCN lesions can block entrainment by light but not by food or water availability.
- Just like suprachiasmatic neurons, cells from other parts of the body display free-running circadian cycles of activity when maintained in tissue culture.

Neural Mechanisms of Entrainment

How does the 24-hour light–dark cycle entrain the sleep–wake cycle and other circadian rhythms? To answer this question, researchers began at the obvious starting point: the eyes (see Morin & Allen, 2006). They tried to identify and track the specific neurons that left the eyes and carried the information about light and dark that entrained the biological clock. Cutting the optic nerves before they reached the optic chiasm eliminated the ability of the light–dark cycle to entrain circadian rhythms; however, when the optic tracts were cut at the point where they left the optic chiasm, the ability of the light–dark cycle to entrain circadian rhythms was unaffected. As Figure 14.9 illustrates, these two findings suggested that the sensory tracts that mediate the entrainment of circadian rhythms by light-dark cycles branch off from the optic nerve in the vicinity of the optic chiasm. This finding led to the discovery of the retinohypothalamic tracts, which leave the optic chiasm and project to the adjacent suprachiasmatic nuclei.

Surprisingly, although the retinohypothalamic tracts mediate the ability of light to entrain photoreceptors, neither rods nor cones are necessary for the entrainment. The mystery photoreceptors have proven to be neurons, a rare type of retinal ganglion cells with distinctive functional properties (see Berson, 2003; Hattar et al., 2002). During the course of evolution, these photoreceptors have sacrificed the ability to respond quickly and briefly to rapid changes of light in favor of the ability to respond consistently to slowly changing levels of background illumination. Their photopigment is melanopsin (Hankins, Peirson, & Foster, 2007; Panda et al., 2005).

Genetics of Circadian Rhythms

An important breakthrough in the study of circadian rhythms came in 1988 when routine screening of a shipment of hamsters revealed that some of them had abnormally short 20-hour free-running circadian rhythms. Subsequent breeding experiments showed that the abnormality was the result of a genetic mutation, and the gene that was mutated was named tau (Ralph & Menaker, 1988).

Although tau was the first mammalian circadian gene to be identified, it was not the first to have its molecular structure characterized. This honor went to clock, a mammalian circadian gene discovered in mice. The structure
of the clock gene was characterized in 1997, and that of the tau gene was characterized in 2000 (Lowrey et al., 2000). The molecular structures of several other mammalian circadian genes have now been specified (see Morse & Sassone-Corsi, 2002).

The identification of circadian genes has led to three important discoveries:

- The same or similar circadian genes have been found in many species of different evolutionary ages (e.g., bacteria, flies, fish, frogs, mice, and humans), indicating that circadian genes evolved early in evolutionary history and have been conserved in various descendant species (see Cirelli, 2009).

- Once the circadian genes were discovered, the fundamental molecular mechanism of circadian rhythms was quickly clarified. The key mechanism seems to be gene expression, that is, the transcription of proteins by the circadian genes displays a circadian cycle (see Dunlap, 2006; Hardin, 2006; Meyer, Saez, & Young, 2006).

- The identification of circadian genes provided a more direct method of exploring the circadian timing capacities of parts of the body other than the SCN. Molecular circadian timing mechanisms similar to those in the SCN exist in most cells of the body (see Green & Menaker, 2003; Hastings, Reddy, & Maywood, 2003; Yamaguchi et al., 2003). Although most cells contain circadian timing mechanisms, these cellular clocks are normally entrained by neural and hormonal signals from the SCN.

The Case of Constantin von Economo, the Insightful Neurologist

During World War I, the world was swept by a serious viral infection of the brain: encephalitis lethargica. Many of its victims slept almost continuously. Baron Constantin von Economo discovered that the brains of deceased victims who had problems with excessive sleep all had damage in the posterior hypothalamus and adjacent parts of the midbrain. He then turned his attention to the brains of a small group of victims of encephalitis lethargica who had had the opposite sleep-related problem: In contrast to most victims, they had difficulty sleeping. He found that the brains of the deceased victims in this minority always had damage in the anterior hypothalamus and adjacent parts of the basal forebrain. On the basis of these clinical observations, von Economo concluded that the posterior hypothalamus promotes wakefulness, whereas the anterior hypothalamus promotes sleep.

Since von Economo’s discovery of the involvement of the posterior hypothalamus and the anterior hypothalamus in human wakefulness and sleep, respectively, that involvement has been confirmed by lesion and recording studies in experimental animals (see Szymusiak, Gvilia, & McGinty, 2007; Szymusiak & McGinty, 2008). The locations of the posterior and anterior hypothalamus are shown in Figure 14.10.

Reticular Formation and Sleep

Another area involved in sleep was discovered through the comparison of the effects of two different brain-stem transections in cats. First, in 1936, Bremer severed the brain stems of cats between their inferior colliculi and superior colliculi in order to disconnect their forebrains from ascending sensory input (see Figure 14.11). This surgical preparation is called a cerveau isolé preparation (pronounced “ser-VOE ees-o-LAY”—literally, “isolated forebrain”).

Bremer found that the cortical EEG of the isolated cat forebrains was indicative of almost continuous slow-wave sleep. Only when strong visual or olfactory stimuli were presented (the cerveau isolé has intact visual and olfactory input) could the continuous high-amplitude, slow-wave activity be changed to a desynchronized EEG—a low-amplitude, high-frequency EEG. However, this arousing effect barely outlasted the stimuli.

Next, for comparison purposes, Bremer (1937) transected (cut through) the brain stems of a different group of cats. These transections were located in the caudal brain stem, and thus, they disconnected the brain from the rest of the nervous system (see Figure 14.11). This experimental...
disrupted normal sleep–wake cycles of cortical EEG only when they severed the reticular formation core of the brain stem; when the partial transections were restricted to more lateral areas, which contain the ascending sensory tracts, they had little effect on the cortical EEG (Lindsey, Bowden, & Magoun, 1949). Second, it was shown that electrical stimulation of the reticular formation of sleeping cats awakened them and produced a lengthy period of EEG desynchronization (Moruzzi & Magoun, 1949).

In 1949, Moruzzi and Magoun considered these four findings together: (1) the effects on cortical EEG of the cervae isolé preparation, (2) the effects on cortical EEG of the encéphale isolé preparation, (3) the effects of reticular formation lesions, and (4) the effects on disrupted normal sleep–wake cycles of cortical EEG only when they severed the reticular formation core of the brain stem; when the partial transections were restricted to more lateral areas, which contain the ascending sensory tracts, they had little effect on the cortical EEG (Lindsey, Bowden, & Magoun, 1949). Second, it was shown that electrical stimulation of the reticular formation of sleeping cats awakened them and produced a lengthy period of EEG desynchronization (Moruzzi & Magoun, 1949).

In 1949, Moruzzi and Magoun considered these four findings together: (1) the effects on cortical EEG of the cervae isolé preparation, (2) the effects on cortical EEG of the encéphale isolé preparation, (3) the effects of reticular formation lesions, and (4) the effects on
sleep of stimulation of the reticular formation. From these four key findings, Moruzzi and Magoun proposed that low levels of activity in the reticular formation produce sleep and that high levels produce wakefulness (see McCarley, 2007). Indeed, this theory is so widely accepted that the reticular formation is commonly referred to as the reticular activating system, even though maintaining wakefulness is only one of the functions of the many nuclei that it comprises.

**Reticular REM-Sleep Nuclei**

The fourth area of the brain that is involved in sleep controls REM sleep and is included in the brain area I have just described—it is part of the caudal reticular formation. It makes sense that an area of the brain involved in maintaining wakefulness would also be involved in the production of REM sleep because of the similarities between the two states. Indeed, REM sleep is controlled by a variety of nuclei scattered throughout the caudal reticular formation. Each site is responsible for controlling one of the major indices of REM sleep (Datta & MacLean, 2007; Siegel, 1983; Vertes, 1983)—a site for the reduction of core-muscle tone, a site for EEG desynchronization, a site for rapid eye movements, and so on. The approximate location in the caudal brain stem of each of these REM-sleep nuclei is illustrated in Figure 14.12.

Please think for a moment about the broad implications of these various REM-sleep nuclei. In thinking about the brain mechanisms of behavior, many people assume that if there is one name for a behavior, there must be a single structure for it in the brain: In other words, they

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**FIGURE 14.12** A sagittal section of the brain stem of the cat illustrating the areas that control the various physiological indices of REM sleep. (Based on Vertes, 1983.)
assume that evolutionary pressures have acted to shape the human brain according to our current language and theories. Here we see the weakness of this assumption: The brain is organized along different principles, and REM sleep occurs only when a network of independent structures becomes active together. Relevant to this is the fact that the physiological changes that go together to define REM sleep sometimes break apart and go their separate ways—and the same is true of the changes that define slow-wave sleep. For example, during REM-sleep deprivation, penile erections, which normally occur during REM sleep, begin to occur during slow-wave sleep. And during total sleep deprivation, slow waves, which normally occur only during slow-wave sleep, begin to occur during wakefulness. This suggests that REM sleep, slow-wave sleep, and wakefulness are not each controlled by a single mechanism. Each state seems to result from the interaction of several mechanisms that are capable under certain conditions of operating independently of one another.

### 14.6 Drugs That Affect Sleep

Most drugs that influence sleep fall into two different classes: hypnotic and antihypnotic. **Hypnotic drugs** are drugs that increase sleep; **antihypnotic drugs** are drugs that reduce sleep. A third class of sleep-influencing drugs comprises those that influence its circadian rhythmicity; the main drug of this class is melatonin.

#### Hypnotic Drugs

The **benzodiazepines** (e.g., Valium and Librium) were developed and tested for the treatment of anxiety, yet they are the most commonly prescribed hypnotic medications. In the short term, they increase drowsiness, decrease the time it takes to fall asleep, reduce the number of awakenings during a night’s sleep, and increase total sleep time (Krystal, 2008). Thus, they can be effective in the treatment of occasional difficulties in sleeping.

Although benzodiazepines can be effective therapeutic hypnotic agents in the short term, their prescription for the treatment of chronic sleep difficulties, though common, is ill-advised (Riemann & Perlis, 2008). Five complications are associated with the chronic use of benzodiazepines as hypnotic agents:

- Tolerance develops to the hypnotic effects of benzodiazepines; thus, patients must take larger and larger doses to maintain the drugs’ efficacy and often become addicted.
- Cessation of benzodiazepine therapy after chronic use causes **insomnia** (sleeplessness), which can exacerbate the very problem that the benzodiazepines were intended to correct.
- Benzodiazepines distort the normal pattern of sleep; they increase the duration of stage 2 sleep, while actually decreasing the duration of stage 4 and of REM sleep.
- Benzodiazepines lead to next-day drowsiness (Ware, 2008) and increase the incidence of traffic accidents (Gustavsen et al., 2008).
- Most troubling is that chronic use of benzodiazepines has been shown to substantially reduce life expectancy (see Siegel, 2010).

Evidence that the raphé nuclei, which are serotonergic, play a role in sleep suggested that serotoninergic drugs might be effective hypnotics. Efforts to demonstrate the hypnotic effects of such drugs have focused on **5-hydroxytryptophan (5-HTP)**—the precursor of serotonin—because 5-HTP, but not serotonin, readily passes through the blood–brain barrier. Injections of 5-HTP do
reverse the insomnia produced in both cats and rats by the serotonin antagonist PCPA; however, they appear to be of no therapeutic benefit in the treatment of human insomnia (see Borbély, 1983).

**Antihypnotic Drugs**

The mechanisms of the following three classes of antihypnotic drugs are well understood: cocaine-derived stimulants, amphetamine-derived stimulants, and tricyclic antidepressants. The drugs in these three classes seem to promote wakefulness by boosting the activity of catecholamines (norepinephrine, epinephrine, and dopamine)—by increasing their release into synapses, by blocking their reuptake from synapses, or both. The antihypnotic mechanisms of two other stimulant drugs, codeine and modafinil, are less well understood.

The regular use of antihypnotic drugs is risky. Antihypnotics tend to produce a variety of adverse side effects, such as loss of appetite, anxiety, tremor, addiction, and disturbance of normal sleep patterns. Moreover, they may mask the pathology that is causing the excessive sleepiness.

**Melatonin**

Melatonin is a hormone that is synthesized from the neurotransmitter serotonin in the pineal gland (see Moore, 1996). The pineal gland is an inconspicuous gland that

[FIGURE 14.13 The location of the pineal gland, the source of melatonin.]

René Descartes, whose dualistic philosophy was discussed in Chapter 2, once believed to be the seat of the soul. The pineal gland is located on the midline of the brain just ventral to the rear portion of the corpus callosum (see Figure 14.13).

The pineal gland has important functions in birds, reptiles, amphibians, and fish (see Cassone, 1990). The pineal gland of these species has inherent timing properties and regulates circadian rhythms and seasonal changes in reproductive behavior through its release of melatonin. In humans and other mammals, however, the functions of the pineal gland and melatonin are not as apparent.

In humans and other mammals, circulating levels of melatonin display circadian rhythms under control of the suprachiasmatic nuclei (see Gillette & McArthur, 1996), with the highest levels being associated with darkness and sleep (see Foulkes et al., 1997). On the basis of this correlation, it has long been assumed that melatonin plays a role in promoting sleep or in regulating its timing in mammals.

In order to put the facts about melatonin in perspective, it is important to keep one significant point firmly in mind. In adult mammals, pinealectomy and the consequent elimination of melatonin appear to have little effect. The pineal gland plays a role in the development of mammalian sexual maturity, but its functions after puberty are not at all obvious.

Does *exogenous* (externally produced) melatonin improve sleep, as widely believed? The evidence is mixed
(see van den Heuvel et al., 2005). However, a meta-analysis (a combined analysis of results of more than one study) of 17 studies indicated that exogenous melatonin has a slight, but statistically significant, soporific (sleep-promoting) effect (Brzezinski et al., 2005).

In contrast to the controversy over the soporific effects of exogenous melatonin in mammals, there is good evidence that it can shift the timing of mammalian circadian cycles. Indeed, several researchers have argued that melatonin is better classified as a chronobiotic (a substance that adjusts the timing of internal biological rhythms) than as a soporific (see Scheer & Czeisler, 2005). Arendt and Skene (2005) have argued that administration of melatonin in the evening increases sleep by accelerating the start of the nocturnal phase of the circadian rhythm and that administration at dawn increases sleep by delaying the end of the nocturnal phase.

Exogenous melatonin has been shown to have a therapeutic potential in the treatment of two types of sleep problems (see Arendt & Skene, 2005). Melatonin before bedtime has been shown to improve the sleep of those insomniacs who are melatonin-deficient and of blind people who have sleep problems attributable to the lack of the synchronizing effects of the light–dark cycle. Melatonin’s effectiveness in the treatment of other sleep disorders remains controversial.

### Clinical Implications

**Insomnia**

Many cases of insomnia are iatrogenic (physician-created)—in large part because sleeping pills (i.e., benzodiazepines), which are usually prescribed by physicians, are a major cause of insomnia. At first, hypnotic drugs may be effective in increasing sleep, but soon the patient may become trapped in a rising spiral of drug use, as tolerance to the drug develops and progressively more of it is required to produce its original hypnotic effect. Soon, the patient cannot stop taking the drug without running the risk of experiencing withdrawal symptoms, which include insomnia. The case of Mr. B. illustrates this problem.

**Mr. B., the Case of Iatrogenic Insomnia**

Mr. B. was studying for a civil service exam, the outcome of which would affect his entire future. He was terribly worried about the test and found it difficult to get to sleep at night. Feeling that the sleep loss was affecting his ability to study, he consulted his physician. . . . His doctor prescribed a moderate dose of barbiturate at bedtime, and Mr. B. found that this medication was very effective . . . for the first several nights. After about a week, he began having trouble sleeping again and decided to take two sleeping pills each night. Twice more the cycle was repeated, until on the night before the exam he was taking four times as many pills as his doctor had prescribed. The next night, with the pressure off, Mr. B. took no medication. He had tremendous difficulty falling asleep, and when he did, his sleep was terribly disrupted. . . . Mr. B. then realized that he had a serious case of insomnia, and returned to his sleeping pill habit. By the time he consulted our clinic several years later, he was taking approximately 1,000 mg sodium amytal every night, and his sleep was more disturbed than ever. . . . Patients may go on for years and years—from one sleeping pill to another—never realizing that their troubles are caused by the pills.

(“Mr. B., the Case of Iatrogenic Insomnia,” from Some Must Watch While Some Must Sleep by William C. Dement, Portable Stanford Books, Stanford Alumni Association, Stanford University, 1978, p. 80. Used by permission of William C. Dement.)

In one study, insomniacs claimed to take an average of 1 hour to fall asleep and to sleep an average of only 4.5 hours per night; but when they were tested in a sleep laboratory, they were found to have an average sleep latency (time to fall asleep) of only 15 minutes and an average nightly sleep duration of 6.5 hours. It used to be common medical
Hypersomnia

Narcolepsy is the most widely studied disorder of hypersomnia. It occurs in about 1 out of 2,000 individuals (Ohayon, 2008) and has two prominent symptoms (see Nishino, 2007). First, narcoleptics experience severe daytime sleepiness and repeated, brief (10- to 15-minute) daytime sleep episodes. Narcoleptics typically sleep only about an hour per day more than average; it is the inappropriateness of their sleep episodes that most clearly defines their condition. Most of us occasionally fall asleep on the beach, in front of the television, or in that most soporific of all daytime sites—the large, stuffy, dimly lit lecture hall. But narcoleptics fall asleep in the middle of a conversation, while eating, while scuba diving, or even while making love.

The second prominent symptom of narcolepsy is cataplexy (Houghton, Scammell, & Thorpy, 2004). Cataplexy is characterized by recurring losses of muscle tone during wakefulness, often triggered by an emotional experience. In its mild form, it may simply force the patient to sit down for a few seconds until it passes. In its extreme form, the patient drops to the ground as if shot and remains there for a minute or two, fully conscious.

In addition to the two prominent symptoms of narcolepsy (daytime sleep attacks and cataplexy), narcoleptics often experience two other symptoms: sleep paralysis and hypnagogic hallucinations. Sleep paralysis is the inability to move (paralysis) just as one is falling asleep or waking up. Hypnagogic hallucinations are dreamlike experiences during wakefulness. Many healthy people occasionally experience sleep paralysis and hypnagogic hallucinations. Have you experienced them?

Three lines of evidence suggested to early researchers that narcolepsy results from an abnormality in the mechanisms that trigger REM sleep. First, unlike normal people, narcoleptics often go directly into REM sleep when they fall asleep. Second and third, as you have already learned, narcoleptics often experience dreamlike states and loss of muscle tone during wakefulness.

Some of the most exciting current research on the neural mechanisms of sleep in general and narcolepsy in particular began with the study of a strain of narcoleptic dogs. After 10 years of studying the genetics of these narcoleptic dogs, Lin and colleagues (1999) finally isolated the gene that causes the disorder. The gene encodes a receptor protein that binds to a neuropeptide called orexin (sometimes called hypocretin), which exists in two forms: orexin-A and orexin-B (see Sakurai, 2005). Although discovery of the orexin gene has drawn attention to genetic factors in narcolepsy, the concordance rate between identical twins is only about 25% (Raizen, Mason, & Pack, 2006).

Several studies have documented reduced levels of orexin in the cerebrospinal fluid of living narcoleptics and in the brains of deceased narcoleptics (see Nishino & Kanbayashi, 2005). Also, the number of orexin-releasing neurons has been found to be reduced in the brains of narcoleptics (e.g., Peyron et al., 2000; Thannickal et al., 2000).

Where is orexin synthesized in the brain? Orexin is synthesized by neurons in the region of the hypothalamus that has been linked to the promotion of wakefulness: the
posterior hypothalamus (mainly its lateral regions). The orexin-producing neurons project diffusely throughout the brain, but they show many connections with neurons of the other wakefulness-promoting area of the brain: the reticular formation. Currently, there is considerable interest in understanding the role of the orexin circuits in normal sleep–wake cycles (see Sakurai, 2007; Siegel, 2004).

Narcolepsy has traditionally been treated with stimulants (e.g., amphetamine, methylphenidate), but these have substantial addiction potential and produce many undesirable side effects. The an hypnotic stimulant modafinil has been shown to be effective in the treatment of narcolepsy, and antidepressants can be effective against cataplexy (Thorpy, 2007).

**REM-Sleep–Related Disorders**

Several sleep disorders are specific to REM sleep; these are classified as *REM-sleep–related disorders*. Even narcolepsy, which is usually classified as a hypersomnic disorder, can reasonably be considered to be a REM-sleep–related disorder—for reasons you have just encountered.

Occasionally, patients who have little or no REM sleep are discovered. Although this disorder is rare, it is important because of its theoretical implications. Lavie and others (1984) described a patient who had suffered a brain injury that presumably involved damage to the REM-sleep controllers in the caudal reticular formation. The most important finding of this case study was that the patient did not appear to be adversely affected by his lack of REM sleep. After receiving his injury, he completed high school, college, and law school and established a thriving law practice.

Some patients experience REM sleep without core-muscle atonia. It has been suggested that the function of REM-sleep atonia is to prevent the acting out of dreams. This theory receives support from case studies of people who suffer from this disorder—case studies such as the following one.

### The Case of the Sleeper Who Ran Over Tackle

I was a halfback playing football, and after the quarterback received the ball from the center he lateraled it sideways to me and I’m supposed to go around end and cut back over tackle and—this is very vivid—as I cut back over tackle there is this big 280-pound tackle waiting, so I, according to football rules, was to give him my shoulder and bounce him out of the way. ... [W]hen I came to I was standing in front of our dresser and I had [gotten up out of bed and run and] knocked lamps, mirrors and everything off the dresser, hit my head against the wall and my knee against the dresser. (Schenck et al., 1986, p. 294)

Presumably, REM sleep without atonia is caused by damage to the nucleus magnocellularis or to an interruption of its output. The *nucleus magnocellularis* is a structure of the caudal reticular formation that controls muscle relaxation during REM sleep. In normal dogs, it is active only during REM sleep; in narcoleptic dogs, it is also active during their cataleptic attacks.

**Evolutionary Perspective**

Numerous studies have compared short sleepers (those who sleep 6 hours or less per night) and long sleepers...
(those who sleep 8 hours or more per night). I focus here on the 2004 study of Fichten and colleagues because it is the most thorough. The study had three strong features:

- It included a large sample (239) of adult short sleepers and long sleepers.
- It compared short and long sleepers in terms of 48 different measures, including daytime sleepiness, daytime naps, regularity of sleep times, busyness, regularity of meal times, stress, anxiety, depression, life satisfaction, and worrying.
- Before the study began, the researchers carefully screened out volunteers who were ill or under various kinds of stress or pressure; thus, the study was conducted with a group of healthy volunteers who slept the amount that they felt was right for them.

The findings of Fichten and colleagues are nicely captured by the title of their paper, "Long sleepers sleep more and short sleepers sleep less." In other words, other than the differences in sleep time, there were no differences between the two groups on any of the other measures—no indication that the short sleepers were suffering in any way from their shorter sleep time. Fichten and colleagues report that these results are consistent with most previous comparisons of short and long sleepers (e.g., Monk et al., 2001), except for a few studies that did not screen out subjects who slept little because they were under pressure (e.g., from worry, illness, or a demanding work schedule). Those studies did report some negative characteristics in the short-sleep group, which likely reflected the stress experienced by some in that group.

Long-Term Reduction of Nightly Sleep

Are short sleepers able to live happy productive lives because they are genetically predisposed to be short sleepers, or is it possible for average people to adapt to a short sleep schedule? There have been only two published studies in which healthy volunteers have reduced their nightly sleep for several weeks or longer. In one (Webb & Agnew, 1974), a group of 16 volunteers slept for only 5.5 hours per night for 60 days, with only one detectable deficit on an extensive battery of mood, medical, and performance tests: a slight deficit on a test of auditory vigilance.

In the other systematic study of long-term nightly sleep reduction (Friedman et al., 1977; Mullaney et al., 1977), 8 volunteers reduced their nightly sleep by 30 minutes every 2 weeks until they reached 6.5 hours per night, then by 30 minutes every 3 weeks until they reached 5 hours, and then by 30 minutes every 4 weeks thereafter. After a participant indicated a lack of desire to reduce sleep further, the person spent 1 month sleeping the shortest duration of nightly sleep that had been achieved, then 2 months sleeping the shortest duration plus 30 minutes. Finally, each participant slept however long was preferred each night for 1 year. The minimum duration of nightly sleep achieved during this experiment was 5.5 hours for 2 participants, 5.0 hours for 4 participants, and an impressive 4.5 hours for 2 participants. In each participant, a reduction in sleep time was associated with an increase in sleep efficiency: a decrease in the amount of time it took to fall asleep after going to bed, a decrease in the number of nighttime awakenings, and an increase in the proportion of stage 4 sleep. After the participants had reduced their sleep to 6 hours per night, they began to experience daytime sleepiness, and this became a problem as sleep time was further reduced. Nevertheless, there were no deficits on any of the mood, medical, or performance tests administered throughout the experiment. The most encouraging result was that an unexpected follow-up 1 year later found that all participants were sleeping less than they had previously—between 7 and 18 hours less each week—with no excessive sleepiness.

Long-Term Sleep Reduction by Napping

Most mammals and human infants display polyphasic sleep cycles; that is, they regularly sleep more than once per day. In contrast, most adult humans display monophasic sleep cycles; that is, they sleep once per day. Nevertheless, most adult humans do display polyphasic cycles of sleepiness, with periods of sleepiness occurring in late afternoon and late morning (Stampi, 1992a). Have you ever experienced them?

Do adult humans need to sleep in one continuous period per day, or can they sleep effectively in several naps as human infants and other mammals do? Which of the two sleep patterns is more efficient? Research has shown that naps have recuperative powers out of proportion with their brevity (e.g., Milner & Cote, 2008; Smith et al., 2007), suggesting that polyphasic sleep might be particularly efficient.

Interest in the value of polyphasic sleep was stimulated by the legend that Leonardo da Vinci managed to generate a steady stream of artistic and engineering accomplishments during his life by napping for 15 minutes every 4 hours, thereby limiting his sleep to 1.5 hours per day. As unbelievable as this sleep schedule may seem, it has been replicated in several experiments (see Stampi, 1992b). Here are the main findings of these truly mind-boggling experiments: First, participants required a long time, several weeks, to adapt to a polyphasic sleep schedule. Second, once adapted to polyphasic sleep, participants were content and displayed no deficits on the performance tests they were given. Third, Leonardo’s 4-hour schedule works quite well, but in unstructured working situations (e.g., around-the-world solo sailboat races), individuals often vary the duration of the cycle without feeling negative consequences. Fourth, most people display a strong preference for particular sleep durations...
(e.g., 25 minutes) and refrain from sleeping too little, which leaves them unrefreshed, or too much, which leaves them groggy for several minutes when they awake—an effect called **sleep inertia** (e.g., Fushimi & Hayashi, 2008; Ikeda & Hayashi, 2008; Wertz et al., 2006). Fifth, when individuals first adopt a polyphasic sleep cycle, most of their sleep is slow-wave sleep, but eventually they return to a mix of REM and slow-wave sleep.

The following are the words of artist Giancarlo Sbragia, who adopted Leonardo’s purported sleep schedule:

> This schedule was difficult to follow at the beginning. . . . It took about 3 wk to get used to it. But I soon reached a point at which I felt a natural propensity for sleeping at this rate, and it turned out to be a thrilling and exciting experience.

> . . . How beautiful my life became: I discovered dawns, I discovered silence, and concentration. I had more time for studying and reading—far more than I did before. I had more time for myself, for painting, and for developing my career. (Sbragia, 1992, p. 181)

### Effects of Shorter Sleep Times on Health

For decades, it was believed that sleeping 8 hours or more per night is ideal for promoting optimal health and longevity. Then, a series of large-scale epidemiological studies conducted in both the United States and Japan challenged this belief (e.g., Ayas et al., 2003; Kripke et al., 2002; Patel et al., 2003; Tamakoshi & Ohno, 2004). These studies did not include participants who were a potential source of bias, for example, people who slept little because they were ill, depressed, or under stress. The studies started with a sample of healthy volunteers and followed their health for several years.

The results of these studies are remarkably uniform (Kripke, 2004). Figure 14.14 presents data from Tamakoshi and Ohno (2004), who followed 104,010 volunteers for 10 years. You will immediately see that sleeping 8 hours per night is not the healthy ideal that it has been assumed to be: The fewest deaths occurred among people sleeping between 5 and 7 hours per night, far fewer than among those who slept 8 hours. You should be aware that other studies that are not as careful in excluding volunteers who sleep little because of stress or ill health find more problems associated with short sleep (see Cappuccio et al., 2008), but any such finding is likely an artifact of pre-existing ill health or stress, which is more prevalent among short sleepers.

Because these epidemiological data are correlational, it is important not to interpret them causally (see Grandner & Drummond, 2007; Stamatakis & Punjabi, 2007; Youngstedt & Kripke, 2004). They do not prove that sleeping 8 or more hours a night causes health problems: Perhaps there is something about people who sleep 8 hours or more per night that leads them to die sooner than people who sleep less. Thus, these studies do not prove that reducing your sleep will cause you to live longer—although some experts are advocating sleep reduction as a means of improving health (e.g., Youngstedt & Kripke, 2004). These studies do, however, provide strong evidence that sleeping less than 8 hours is not the risk to life and health that it is often made out to be.

### Long-Term Sleep Reduction: A Personal Case Study

I began this chapter 4 weeks ago with both zeal and trepidation. I was fascinated by the idea that I could wring 2 or 3 extra hours of living out of each day by sleeping less, and I hoped that adhering to a sleep-reduction program while writing about
sleep would create an enthusiasm for the subject that would color my writing and be passed on to you. I began with a positive attitude because I was aware of the relevant evidence; still I was more than a little concerned about the negative effect that reducing my sleep by 3 hours per night might have on me and my writing.

The Case of the Author Who Reduced His Sleep

Rather than using the gradual stepwise reduction method of Friedman and his colleagues, I jumped directly into my 5-hours-per-night sleep schedule. This proved to be less difficult than you might think. I took advantage of a trip to the East Coast from my home on the West Coast to reset my circadian clock. While I was in the East, I got up at 7:00 A.M., which is 4:00 A.M. on the West Coast, and I just kept on the same schedule when I got home. I decided to add my extra waking hours to the beginning of my day rather than to the end so there would be no temptation for me to waste them—there are not too many distractions around this university at 5:00 A.M.

Figure 14.15 is a record of my sleep times for the 4-week period that it took me to write a first draft of this chapter. I didn’t quite meet my goal of sleeping less than 5 hours every night, but I didn’t miss by much: My overall mean was 5.05 hours per night. Notice that in the last week, there was a tendency for my circadian clock to run a bit slow; I began sleeping in until 4:30 A.M. and staying up until 11:30 P.M.

What were the positives and negatives of my experience? The main positive was the added time to do things: Having an extra 21 hours per week was wonderful. Furthermore, because my daily routine was out of sync with everybody else’s, I spent little time sitting in traffic. The only negative of the experience was sleepiness. It was no problem during the day, when I was active. However, staying awake during the last hour before I went to bed—an hour during which I usually engaged in sedentary activities, such as reading—was at times a problem. This is when I became personally familiar with the phenomenon of microsleeps, and it was then that I required some assistance in order to stay awake. Each night of sleep became a highly satisfying but all too brief experience.

I began this chapter with this question: How much sleep do we need? Then, I gave you my best professorial it-could-be-this, it-could-be-that answer. However, that was a month ago. Now, after experiencing sleep reduction firsthand and reviewing the evidence yet again, I am less inclined toward wishy-washiness on the topic of sleep. The fact that most committed subjects who are active during the day can reduce their sleep to about 5.5 hours per night without great difficulty or major adverse consequences suggested to me that the answer is 5.5 hours of sleep. But that was before I learned about polyphasic sleep schedules. Now, I must revise my estimate downward.

Conclusion

In this section, you have learned that many people sleep little with no apparent ill effects and that people who are average sleepers can reduce their sleep time substantially, again with no apparent ill effects. You also learned that the health of people who sleep between 5 and 7 hours a night does not suffer; indeed, epidemiological studies indicate that they are the most healthy and live the longest. Together, this evidence challenges the widely held belief that humans have a fundamental need for at least 8 hours of sleep per night.
Themes Revisited

The thinking creatively theme pervaded this chapter. The major purpose of the chapter was to encourage you to re-evaluate conventional ideas about sleep in the light of relevant evidence. Has this chapter changed your thinking about sleep? Writing it changed mine.

The evolutionary perspective theme also played a prominent role in this chapter. You learned how thinking about the adaptive function of sleep and comparing sleep in different species have led to interesting insights. Also, you saw how research into the physiology and genetics of sleep has been conducted on nonhuman species.

Think about It

1. Do you think your life could be improved by changing when or how long you sleep each day? In what ways? What negative effects do you think such changes might have on you?
2. Some people like to stay up late, some people like to get up early, others like to do both, and still others like to do neither. Design a sleep-reduction program that is tailored to your own preferences and lifestyle and that is consistent with the research literature on circadian cycles and sleep deprivation. The program should produce the greatest benefits for you with the least discomfort.
3. How has reading about sleep research changed your views about sleep? Give three specific examples.
4. Given the evidence that the long-term use of benzodiazepines actually contributes to the problems of insomnia, why are they so commonly prescribed for its treatment?
5. Your friend tells you that everybody needs 8 hours of sleep per night; she points out that every time she stays up late to study, she feels lousy the next day. What evidence would you provide to convince her that she does not need 8 hours of sleep per night?

Key Terms

14.1 Stages of Sleep

Electroencephalogram (EEG) (p. 357)
Electrooculogram (EOG) (p. 357)
Electromyogram (EMG) (p. 357)
Alpha waves (p. 357)
Delta waves (p. 358)
Initial stage 1 EEG (p. 358)
Emergent stage 1 EEG (p. 358)
REM sleep (p. 358)
Slow-wave sleep (SWS) (p. 358)
Activation-synthesis theory (p. 359)

14.2 Why Do We Sleep, and Why Do We Sleep When We Do?

Recuperation theories of sleep (p. 359)
Adaptation theories of sleep (p. 360)

14.3 Effects of Sleep Deprivation

Executive function (p. 363)
Microsleeps (p. 363)
Carousel apparatus (p. 363)

14.4 Circadian Sleep Cycles

Circadian rhythms (p. 366)
Zeitgebers (p. 366)
Free-running rhythms (p. 366)
Free-running period (p. 366)
Internal desynchronization (p. 367)
Jet lag (p. 367)
Circadian clock (p. 368)
Suprachiasmatic nuclei (SCN) (p. 368)
Melanopsin (p. 369)
Tau (p. 369)

14.5 Four Areas of the Brain Involved in Sleep

Cerveau isolé preparation (p. 370)
Desynchronized EEG (p. 370)
Encéphale isolé preparation (p. 371)
Reticular activating system (p. 372)

14.6 Drugs That Affect Sleep

Hypnotic drugs (p. 373)
Antihypnotic drugs (p. 373)
Melatonin (p. 373)
Benzodiazepines (p. 373)
5-Hydroxytryptophan (5-HTP) (p. 373)
Pineal gland (p. 374)
Chronobiotic (p. 375)

14.7 Sleep Disorders

Insomnia (p. 375)
Hypersomnia (p. 375)
Iatrogenic (p. 375)
Sleep apnea (p. 376)
Periodic limb movement disorder (p. 376)
Restless legs syndrome (p. 376)
Narcolepsy (p. 376)
Cataplexy (p. 376)
Sleep paralysis (p. 376)
Hypnagogic hallucinations (p. 376)
Orexin (p. 376)
Nucleus magnocellularis (p. 377)

14.8 Effects of Long-Term Sleep Reduction

Polyphasic sleep cycles (p. 378)
Monophasic sleep cycles (p. 378)
Sleep inertia (p. 379)
Test your comprehension of the chapter with this brief practice test. You can find the answers to these questions as well as more practice tests, activities, and other study resources at www.mypsychlab.com.

1. In which stage of sleep do delta waves predominate?
   a. initial stage 1  
   b. emergent stage 1  
   c. stage 2  
   d. stage 3  
   e. stage 4  

2. The results of many sleep-deprivation studies are difficult to interpret because of the confounding effects of
   a. sex.  
   b. dreaming.  
   c. shift work.  
   d. memory loss.  
   e. stress.  

3. The carousel apparatus has been used to
   a. entertain sleep-deprived volunteers.  
   b. synchronize zeitgebers.  
   c. synchronize circadian rhythms.  
   d. deprive rodents of sleep.  
   e. block microsleeps in sleep-deprived humans.  

4. Dreaming occurs during
   a. initial stage 1 sleep.  
   b. stage 2 sleep.  
   c. stage 3 sleep.  
   d. stage 4 sleep.  
   e. none of the above  

5. Circadian rhythms without zeitgebers are said to be
   a. entrained.  
   b. free-running.  
   c. desynchronized.  
   d. internal.  
   e. pathological.  