KEY KNOWLEDGE

- Changes to a person’s sleep–wake cycle and susceptibility to experiencing a circadian phase disorder, including sleep–wake shifts in adolescence, shift work and jet lag.
- The effects of partial sleep deprivation (inadequate sleep either in quantity or quality) on a person’s affective (amplified emotional responses) behavioural and cognitive functioning.
- The distinction between dyssomnias (including narcolepsy and sleep-onset insomnia) and parasomnias (including sleep apnoea and sleep walking) with reference to the effects on a person’s sleep–wake cycle.
- The interventions to treat sleep disorders including cognitive behavioural therapy (with reference to insomnia) and bright light therapy (with reference to circadian phase disorders).

<table>
<thead>
<tr>
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Some nights, we fall asleep easily and the night passes with little or no interruption to our sleep. When we awaken after a good night's sleep we feel terrific — refreshed, energised and ready to take on the world. Other nights, onset of sleep is slow, perhaps not until well into the early morning hours. Or we may fall asleep quite quickly but awaken too many times throughout the night. We usually don't feel so great after these types of 'bad' sleep experiences. Merely getting out of bed when the alarm goes off can be a huge effort. We may snap at the first person we see over something that is really quite trivial. At school or work we may lack motivation, find it hard to concentrate for too long and react more slowly than usual. However, we usually recover quite quickly from isolated 'bad' sleep experiences, especially if we follow it up a with 'good' night's sleep at the next available opportunity (Epstein & Mardon, 2007).

Some people don't sleep as much or as well as they would like to on a regular basis. They may have trouble with the timing of their sleep, falling asleep, staying asleep, waking up, staying awake and/or a problem with the quality of their sleep after they manage to fall asleep. Their problem with sleep quantity or quality occurs not because they choose to, but because their sleep–wake cycle is disturbed in some way.

The term sleep disturbance is used to refer to any sleep-related problem that disrupts an individual's normal sleep–wake cycle, including problems with sleep onset, waking from sleep and abnormal behaviour occurring during sleep. The disruption may be temporary, occasional or persistent. If a sleep disturbance regularly disrupts sleep, causing distress or impairment in important areas of everyday life during normal waking hours, then it is usually referred to as a sleep disorder. This means that sleep disorders generally are considered serious disturbances to the normal sleep–wake cycle (American Academy of Sleep Medicine [AASM], 2014a, APA, 2013).

Sleep disorders are often classified as either primary or secondary, depending on their root cause. This assists understanding of the symptoms and helps with the planning of treatment.

A primary sleep disorder is a sleep disorder that cannot be attributed to another condition or use of a substance, such as another sleep disorder, a mental disorder, a medical problem or the use of a legal or illegal drug. The sleep disorder is the main, or ‘primary’, cause of the sleep problem. It occurs in its own right and cannot be explained by another condition. For example, someone may experience regular awakenings throughout their major sleep episode because they have the primary sleep disorder called insomnia.

A secondary sleep disorder involves a prominent sleep problem that is a by-product of or results from another condition, or use of a substance. For example, someone may experience regular awakenings whenever they sleep because of their back pain, a bladder problem, a breathing irregularity, stress, an anxiety disorder or depression. In this case, the sleep problem is 'secondary' to something else — another underlying condition. It is believed to improve with treatment of the underlying condition so the target of treatment would be the underlying ‘secondary’ condition (AASM, 2001, 2014a).

Distinguishing between primary and secondary sleep disorders depends on whether the sleep problem is considered a sleep disorder or a symptom of another condition. Although primary sleep disorders do not arise from other conditions, they can often contribute or lead to secondary ones. For example, the ongoing experience of poor sleep quantity or quality due to insomnia or an irregular sleep–wake pattern, both of which are primary sleep disorders, may contribute to an anxiety disorder or depression.

Disturbed sleep, whether because of timing, quantity, quality or some other problem, can adversely impact on our health and wellbeing. Common effects are daytime sleepiness, tiredness, lack of energy, difficulty concentrating, slower reaction times and mood change. A range of other impairments to various aspects of our emotional, behavioural and cognitive functioning have also been identified. Some sleep disturbances or disorders are a risk factor for the presence or subsequent development of a serious physical or mental health problem. Some sleep disorders, such as those that are breathing-related or involve seizures, can even be life-threatening.

Sleep disturbances and disorders are very common, affecting virtually everyone at some point in their lives. However, they are largely under-reported, under-recognised, under-diagnosed, and often untreated. While ongoing sleep problems typically cause some degree of personal distress and interfere to some extent with an individual's behaviour and everyday functioning, virtually all of them can be successfully treated or managed.

In this chapter we examine the effects of a range of sleep disorders and possible treatments. We also re-visit sleep deprivation to examine the effects of inadequate sleep quantity or quality in a more substantial way than in chapter 8.
DYSSOMNIAS AND PARASOMNIAS

More than 80 different types of sleep disorders are described in the various classification systems that are used for diagnostic purposes by health professionals. The disorders are usually grouped in categories and sub-categories. Two traditionally used categories are dyssomnias and parasomnias.

Generally, dyssomnias involve problems with sleep–wake cycle processes, such as difficulty falling or staying asleep, inability to prevent sleep onset, or a disruption to the timing of the circadian sleep–wake cycle. By contrast, parasomnias involve inappropriate disruptions of sleep by some abnormal sleep-related event, such as sleep walking, teeth grinding and terrifying dreams. Unlike dyssomnias, parasomnias do not involve a dysfunction in any process or mechanism that generates or times sleep (Kennedy, 2002; Thorpy, 2012).

In some cases, symptoms of a dyssomnia (such as excessive daytime sleepiness) may result from awakenings caused by the disruptive behaviour of a parasomnia during sleep or by another dyssomnia. Similarly, someone who regularly experiences nightmares (a parasomnia) may also experience insomnia (a dyssomnia) because of an inability to sleep soundly.

Most individuals suffering from a parasomnia express concern about the unusual behaviour or experiences they have during sleep, rather than insomnia or excessive daytime sleepiness. By contrast, dyssomnias are not usually associated with abnormal behaviour or experiences and primarily produce a complaint of insomnia or excessive sleepiness.

All dyssomnias and parasomnias are primary sleep disorders. This means the specific disorders within each category are independent of any other condition or disorder and the main cause of the sleep disturbance.

**FIGURE 10.2** A dyssomnia involves a problem with a sleep–wake cycle process (such as difficulty falling or staying asleep), whereas a parasomnia involves disruption of sleep by an abnormal event (such as a frightening dream that awakens the sleeper).

**LEARNING ACTIVITY 10.1**

**Review questions**

1. Explain the meaning of sleep disturbance.
2. Under what conditions is a sleep disturbance likely to be considered a sleep disorder?
3. (a) Distinguish between a primary and secondary sleep disorder.
   (b) Explain how primary and secondary sleep disorders can contribute to each other’s symptoms.
4. (a) What are dyssomnias and parasomnias?
   (b) Give two examples of dyssomnias and parasomnias.
   (c) Distinguish between dyssomnias and parasomnias with reference to two key characteristics.

**LEARNING ACTIVITY 10.2**

**Reflection**

Consider the comment in the chapter introduction that sleep disorders are largely under-reported, under-recognised, under-diagnosed and often untreated. What do you think?
Dyssomnias

Dyssomnias are sleep disorders that produce difficulty initiating, maintaining and/or timing sleep. This results in a problem either falling asleep, staying asleep and/or excessive sleepiness. As a consequence, the person suffers from changes in the quantity (amount) or quality (restfulness) of their sleep, and/or a problem with when they fall asleep (AASM, 2014a).

Among the more common dyssomnias are different types of insomnia (when it is hard to fall asleep or stay asleep) and various circadian rhythm phase disorders such as the sleep–wake cycle shift that occurs in adolescence or a disturbance to the sleep–wake cycle that may be due to shift work or when we fly across time zones and experience jet lag (SHF, 2016e).

Although many dyssomnias can originate or develop from causes outside of the body, such as lifestyle factors or sleep environment conditions, they are primarily attributable to some kind of change to the mechanisms and processes that generate or time sleep, including naturally occurring changes and abnormalities (AASM, 2014a; Kennedy, 2002; Thorpy, 2012).

In this section we examine two examples of dyssomnias, with reference to their effects on a person’s sleep–wake cycle — narcolepsy and sleep-onset insomnia. These two disorders are opposites. Narcolepsy involves a struggle to stay awake, whereas sleep-onset insomnia involves a struggle to fall asleep.

Narcolepsy

Most people can be overcome by an urge to sleep at an odd or inconvenient time. For some people, this is common and disruptive. They may fall asleep in the daytime at unexpected times, against their will. This can happen even though they feel they are getting enough sleep at night. Their brain is unable to regulate their sleep–wake cycle normally.

Narcolepsy is a sleep disorder involving excessive sleepiness during normal waking hours. It may occur with other symptoms such as cataplexy, sleep paralysis and hallucinations. Symptoms usually develop over several months between the ages of 10 and 20 years, most commonly in adolescence, and last a lifetime.

A person with narcolepsy will usually feel extremely tired and sleepy throughout the day. This will happen even though they believe that they are getting enough sleep at night. The sleepiness can occur regardless of the time, what they are doing or where they are. They will want to go to sleep, rather than just feel tired or weary without a strong need for sleep, as tends to happen in people without narcolepsy (Bruck, 2006; NSF, 2016f).

Many people with narcolepsy find that they have great difficulty maintaining their concentration while at school or work. In some cases, they may also experience an urge to sleep that will be hard to control unless they have had a nap. This involuntary lapse into sleep is commonly called a sleep attack. A sleep attack might last for minutes or up to an hour or so, sometimes for no more than a few seconds at a time. There is a tendency to fall asleep directly or quickly into REM sleep within about 10–15 minutes. Sleep attacks usually occur suddenly, without warning. Typically, the individual sleeps for 10 to 20 minutes and awakens refreshed, but within the next two to three hours begins to feel sleepy again, and the pattern repeats itself. Sleepiness can often be tolerated for a while with much effort and attention focussed on staying awake. Eventually, however, it is impossible to resist the urge to sleep.

Sleep attacks can disrupt the sleep–wake cycle at any time, irrespective of the amount of sleep obtained the night before. They are more likely to occur in passive situations when inactive or situations in which tiredness is common, such as when travelling in public transport, attending a meeting that requires no active participation, listening to a long lecture, or when at the movies or theatre. However, sleep attacks may also occur in situations where sleep normally never occurs, including dangerous situations. For example, they may occur when eating a meal, engaged in a conversation, during an exam, while actively participating in a meeting, when standing or walking, or when riding a bike or driving a car (AASM, 2014a).

FIGURE 10.3 Narcolepsy is primarily characterised by excessive sleepiness during normal waking hours. A person may feel sleepy anywhere, at any time when awake. In some cases, they may have a sleep attack and fall directly into REM sleep regardless of what they are doing.

Video outlining narcolepsy symptoms and its neurological basis 5m
Key symptoms

Everyone with narcolepsy experiences excessive sleepiness when awake and it is usually the first symptom to appear and indicate presence of the disorder. There are also three other major symptoms — cataplexy, hallucinations during sleep onset or when awakening and brief episodes of total paralysis at the beginning or end of sleep. These symptoms are less common than excessive sleepiness and not necessarily experienced by all people with narcolepsy. Narcolepsy can be hard to diagnose when sleepiness is the only symptom because there are lots of reasons why people experience sleepiness.

Cataplexy is the sudden loss of muscle tone while conscious, resulting in weakening of muscles and loss of voluntary control of affected muscles. The duration of an episode of cataplexy (sometimes called a cataplectic attack) is usually short, ranging from a few seconds to several minutes, and recovery is immediate and complete. The loss of muscle tone varies in severity and can be localised or affect the entire body. For example, it can range from a barely perceptible slight slackening of the facial muscles, to buckling of the knees, weakness in the arms, or a total collapse of the body with a fall to the ground and an inability to move, speak or keep the eyes open. But even during the most severe episodes, the person remains conscious and fully aware of what is happening around them, memory is not impaired, and breathing is unaffected (AASM, 2014a).

Cataplexy can occur any time during the waking period and doesn’t always coincide with a sleep attack. It is often provoked by a strong emotion, such as excitement, surprise, sadness or anger. Laughter is a common trigger. For example, a funny joke that causes heart-felt laughter can initiate partial or total muscle weakness. People who have narcolepsy without cataplexy experience the persistent sleepiness but no emotionally triggered muscle weakness, and generally have less severe symptoms (Hines & Morton, 2015).

People with narcolepsy may also experience dream-like hallucinations just as they are falling asleep or waking up. These are often called hypnagogic hallucinations when they occur during sleep onset and hypnopompic hallucinations during awakening. Most often, the content is primarily visual, but any of the other senses can be involved. The hallucinations are unusually vivid, realistic and often very frightening, especially if accompanied by an episode of sleep paralysis. It can sometimes also take a while to distinguish the hallucination from reality after waking up (AASM, 2014a; APA, 2013).

Sleep paralysis is another symptom of narcolepsy. Sleep paralysis is the temporary inability to move and speak during sleep onset or when waking up. It may last for a few seconds to a minute or so and resembles a cataplectic episode that affects the entire body. Sleep paralysis is often reported to be a frightening experience, particularly when accompanied by a sensation of being unable to breathe. Episodes often occur with hypnagogic hallucinations, which intensifies the frightful nature of the experience. As with cataplexy, people remain conscious during sleep paralysis and quickly recover their full capacity to move and speak (AASM, 2014a).

Sleep paralysis can occur by itself, without narcolepsy. We all experience sleep paralysis during REM sleep, but this typically occurs within a sleep cycle. Because of their tendency to enter sleep directly through REM rather than NREM sleep, people with narcolepsy often experience sleep paralysis as they’re falling asleep. This also helps explain why they are more likely to experience hypnagogic hallucinations together with their sleep paralysis.

Narcolepsy is a rare sleep disorder which effects about 1 in 2000 people (less than 0.05% of the general population). Age of onset is most common during puberty and adolescence. There is no cure but symptoms can be treated and it is a manageable
condition. Most people with narcolepsy endure many years of daytime sleepiness before seeking treatment because sleepiness is not indicative of a disorder or disease to most people. Sometimes people confuse narcolepsy with a tropical disease called sleeping sickness which is caused by the bite of an infected tsetse fly, an insect native to Africa.

Effects on sleep–wake cycle

Narcolepsy significantly disrupts the sleep–wake cycle and its regulation. Generally, people with the disorder experience excessive sleepiness when they don’t want to, sleep more often than they would like to and when they don’t want to, and the transition from being awake to asleep is unstable. Furthermore, when sleep attacks occur, sleep often begins with REM sleep. This also means that fragments of REM sleep are occurring involuntarily throughout the individual’s normal waking hours.

With narcolepsy, the boundaries surrounding and between wakefulness and sleep are less distinct, and elements of sleep and wakefulness can mix together. As apparent in the symptoms of narcolepsy, REM sleep can become so poorly regulated that the paralysis or dreaming that normally occurs only in REM sleep can mix into wakefulness, causing cataplexy and hallucinations (Scammel, 2013).

Excessive sleepiness also tends to rapidly progress to an inability to maintain good sleep at night. In the same way that people with narcolepsy have difficulty maintaining wakefulness during the day, many have difficulty maintaining sleep at night. They do not typically sleep more or less than the average person, but they often experience fragmented night time sleep, waking up 4 or 5 times each night for 10–20 minutes, which, in turn, can worsen daytime sleepiness (APA, 2013; NSF, 2016f; Scammel, 2013).

**TABLE 10.1 Sleep–wake patterns of people with and without narcolepsy**

<table>
<thead>
<tr>
<th>People with narcolepsy</th>
<th>People without narcolepsy</th>
</tr>
</thead>
<tbody>
<tr>
<td>During the day</td>
<td></td>
</tr>
<tr>
<td>Feel drowsy and fall asleep easily</td>
<td>Feel alert, awake and don’t readily fall asleep</td>
</tr>
<tr>
<td>Often enter REM sleep and dream during naps</td>
<td>Generally do not enter REM sleep or dream during naps</td>
</tr>
<tr>
<td>Hallucinations on waking common</td>
<td>Hallucinations on waking uncommon</td>
</tr>
<tr>
<td>Sleep paralysis common</td>
<td>Sleep paralysis uncommon</td>
</tr>
<tr>
<td>May experience cataplexy</td>
<td>Never experience cataplexy</td>
</tr>
<tr>
<td>During the night</td>
<td></td>
</tr>
<tr>
<td>Spontaneously wake from sleep</td>
<td>Generally sleep well</td>
</tr>
<tr>
<td>Can enter REM sleep within 15 minutes</td>
<td>First enter NREM sleep, then take 60–90 minutes to enter REM sleep</td>
</tr>
</tbody>
</table>


**FIGURE 10.5** A person’s normal sleep–wake cycle is disrupted by narcolepsy.
BOX 10.1

**What causes narcolepsy?**

The actual cause of narcolepsy remains unclear. It is considered to be a neurological disorder, specifically a central nervous system disorder involving the brain’s inability to regulate sleep–wake cycles normally (NSF, 2016).

Researchers have found that most cases of narcolepsy with cataplexy (and in rare cases without cataplexy) are associated with low levels of hypocretins, which are neurotransmitters produced in the hypothalamus. Hypocretins help sustain alertness and prevent REM sleep from occurring at inappropriate times. In people who have narcolepsy with cataplexy, most of the hypocretin-producing neurons die off, possibly because the body’s immune system selectively attacks them.

The consequent lack of hypocretins results in lasting sleepiness and poor control of REM sleep. As apparent in the symptoms of narcolepsy, REM sleep can become so poorly regulated that the paralysis of dreaming that normally occurs only in REM sleep can mix into wakefulness, causing cataplexy and hallucinations.

Relatively little is known about the cause of narcolepsy without cataplexy. Genetics, age and the autoimmune system are also believed to play roles in the development of narcolepsy (Scammel, 2013).
BOX 10.2

Epworth Sleepiness Scale

The Epworth Sleepiness Scale (ESS) was first published in 1991 by Dr Murray Johns of the Sleep Disorders Unit at Melbourne’s Epworth Hospital. It was designed as a simple assessment of a person’s general level of daytime sleepiness. In 1997 it was slightly revised to add an extra sentence of instructions.

The ESS is widely used throughout the world for sleep research and diagnostic purposes. It is a subjective, self-administered questionnaire that lists eight situations or activities for which individuals rate their sleepiness on a 4-point scale (0–3). An overall score is then calculated to indicate a level of daytime sleepiness. When used for diagnostic purposes, it is one of a number of assessments that would be conducted by the sleep specialist.

THE EPWORTH SLEEPINESS SCALE

Name: ____________________________

Today’s date: ________________, Your age (years): ______________________

Your sex (male = M; female = F): __________________________

How likely are you to doze off or fall asleep in the following situations, in contrast to just feeling tired? This refers to your usual way of life in recent times. Even if you have not done some of these things recently try to work out how they would have affected you.

Use the following scale to choose the most appropriate number for each situation:

0 = would never doze
1 = slight chance of dozing
2 = moderate chance of dozing
3 = high chance of dozing

It is important that you answer each question as best you can.

<table>
<thead>
<tr>
<th>Situation</th>
<th>Chance of dozing</th>
</tr>
</thead>
<tbody>
<tr>
<td>Sitting and reading</td>
<td></td>
</tr>
<tr>
<td>Watching TV</td>
<td></td>
</tr>
<tr>
<td>Sitting, inactive in a public place (e.g. a theatre or a meeting)</td>
<td></td>
</tr>
<tr>
<td>As a passenger in a car for an hour without a break</td>
<td></td>
</tr>
<tr>
<td>Lying down to rest in the afternoon when circumstances permit</td>
<td></td>
</tr>
<tr>
<td>Sitting and talking to someone</td>
<td></td>
</tr>
<tr>
<td>Sitting quietly after a lunch without alcohol</td>
<td></td>
</tr>
<tr>
<td>In a car, while stopped for a few minutes in the traffic</td>
<td></td>
</tr>
</tbody>
</table>

Thank you for your cooperation

Scoring: The 8 scores are added to find a total.

Interpretation:

The higher the score, the higher the person’s level of daytime sleepiness. The following score ranges indicate different levels of sleepiness. The 0–10 range is widely considered to be the ‘normal’ range, but scores for other categories may vary. For example, some sleep specialists do not distinguish between mild or moderate sleepiness, while others may estimate severe sleepiness as a score of 16 or even lower.

- Normal range in healthy adults: 0–10
- Mild sleepiness: 11–14
- Moderate sleepiness: 15–17
- Severe sleepiness: 18 or higher


Sleep-onset insomnia

Everyone has an occasional night of bad sleep from which they usually recover within a day or so. There are some people, however, who struggle nightly with their sleep. They take a long time to fall asleep, they may awaken many times during the night, or their sleep may even end in the middle of the night well before they want it to. Some may fall asleep quickly and then have trouble falling asleep again. When it is only to wake up during the night, wide awake and alert, this is a sleep disorder — it may occur in the absence of any other disorder or condition. Insomnia may also be secondary to another sleep disorder; for example, it is commonly associated with sleep-related breathing disorders (e.g., obstructive sleep apnoea), movement disorders (e.g. restless legs or periodic limb movements during sleep) and circadian rhythm sleep disorders.

The difficulty initiating or maintaining sleep may also occur with a mental or physical health condition such as stress, anxiety, pain, an illness, disease or effects of a substance (Schutte-Rodin, et al., 2008).

As with other dyssomnias, insomnia is a primary sleep disorder — it may occur in the absence of any other disorder or condition. Insomnia may also be secondary to another sleep disorder; for example, it is commonly associated with sleep-related breathing disorders (e.g., obstructive sleep apnoea), movement disorders (e.g. restless legs or periodic limb movements during sleep) and circadian rhythm sleep disorders. The difficulty initiating or maintaining sleep may also occur with a mental or physical health condition such as stress, anxiety, pain, an illness, disease or effects of a substance (Schutte-Rodin, et al., 2008).

Generally, insomnia can be situational or recurrent (both of which may be referred to as acute insomnia). Situational insomnia lasts a few days or weeks and is often associated with life events that may include a sudden change to the
sleeper’s environment or sleep–wake cycle. It usually resolves once the initial causal event passes. Recurrent insomnia occurs irregularly and involves episodes of sleep difficulty interspersed with occasional nights of restful sleep. The insomnia bouts tend to be associated with the occurrence of stressful events (APA, 2013).

The term sleep-onset insomnia (also called initial insomnia) is used to refer specifically to the sleep disorder involving persistent difficulty falling asleep at the usual sleep time. It takes a long time to fall asleep, but the person can usually sleep through the night once sleep starts (or through the day if they are a shiftworker). Sleep-onset insomnia is distinguished from sleep maintenance insomnia which involves difficulty staying asleep (middle insomnia) and/or awakening prematurely from sleep with an inability to fall asleep again (late insomnia). Many people experience a combination of these types of insomnia, sometimes all three (APA, 2013).

Key symptoms
The experience of sleep-onset insomnia is a unique one for each individual with the disorder. There are, however, a number of key symptoms and criteria that are commonly used as part of the diagnostic process. Along with the pattern of sleep disturbance, these may include:

- regular failure to fall asleep within about 20–30 minutes after intending to go to sleep
- complaint of poor quality sleep that does not leave the individual feeling rested upon awakening (called nonrestorative sleep) or a consistently reduced amount of total sleep, either of which is associated with difficulty falling asleep
- the sleep difficulty occurs at least three nights a week
- the sleep difficulty is experienced for at least three months (but if less than three months may be described as recurrent or episodic sleep-onset insomnia)
- the sleep difficulty occurs despite adequate opportunity to sleep (which helps distinguish insomnia from insufficient sleep due to behaviour and lifestyle factors)
- the sleep difficulty does not occur in the course of another sleep disorder and is not due to another disorder or the effects of a substance
- difficulty falling asleep causes significant impairment in behaviour or important areas of everyday functioning, such as at school, work and in social or recreational situations (APA, 2013; Schutte-Rodin, et al., 2008).

Insomnia is the most prevalent of all sleep disorders and most frequently occurs together with another condition of some type, either another sleep disorder and/or a physical or mental health disorder. It is estimated that about 30% of adults have symptoms of insomnia at some time, and about 5–10% of adults have a persistent insomnia disorder. The onset of symptoms can occur at any time in life, although insomnia complaints are more prevalent among older adults. Like most other sleep disorders, the symptoms can be treated and it is a manageable condition (APA, 2013; NSF, 2016g).

Effects on sleep–wake cycle
As with narcolepsy and other sleep disorders, sleep-onset insomnia can significantly disrupt the sleep–wake cycle and its regulation. Generally, the individual experiences changes in the amount, restfulness and the timing of their sleep. Common complaints are sleep onset occurring much later than desired, sleep is nonrestorative (not restful) and/or total sleep time is less than desired (Schutte-Rodin, et al., 2008).

Many people who experience sleep-onset insomnia are often frustrated, anxious or stressed about not being able to fall asleep when they want to and therefore not get enough sleep to properly fulfil their daily commitments, which can make the problem worse. The preoccupation with sleep and distress due to the inability to sleep may lead to a vicious cycle — the more the person tries to sleep, the more frustration and anxiety build up and impair sleep onset. Consequently, excessive attention and efforts to sleep can override and inhibit the normal sleep-onset mechanisms (APA, 2013).

In some cases of persistent sleep-onset insomnia, when the person also starts to regularly experience difficulty waking up in the morning, the continually delayed sleep onset may disrupt the circadian sleep–wake cycle to the extent that a delayed sleep phase (‘timing’) disorder develops. When this occurs, the times when they naturally feel sleepy and awaken occur later, so their paired sleep–wake times are later than desired. This may partly solve their problems of sleep quantity and/or quality, but their sleep–wake cycle can be out-of-sync with time dependent requirements of the rest of society which may create new problems.
LEARNING ACTIVITY 10.3

Review questions

1. (a) What is narcolepsy?
   (b) Explain the meaning of each of the following in relation to narcolepsy. Ensure you refer to key distinguishing characteristics.
      (i) excessive sleepiness during NWC
      (ii) sleep attack
      (iii) cataplexy
      (iv) hallucinations
      (v) sleep paralysis
   (c) Which of (i)–(v) above is an essential symptom of narcolepsy?
   (d) Give three examples of how narcolepsy can effect a person’s circadian sleep–wake cycle.
   (e) Explain why narcolepsy is classified as a dyssomnia.
   (f) To what extent can many of the symptoms of narcolepsy be viewed as the intrusion of REM sleep period into a person’s waking life?

2. (a) What is insomnia?
   (b) Explain the meaning of sleep-onset insomnia with reference to three symptoms relating to the sleep–wake cycle.
   (c) Under what conditions would sleep-onset insomnia be considered acute or chronic?
   (d) Give three examples of how sleep-onset insomnia can effect a person’s circadian sleep–wake cycle.
   (e) Consider the two hypnograms in figure 10.8. Compare and contrast the sleep–wake cycles of the two individuals with reference to three distinguishing features, ensuring you refer to relevant data in the graphs.
   (f) Explain how someone can perpetuate their sleep-onset insomnia through their thoughts, feelings or behaviour.

LEARNING ACTIVITY 10.4

Reflection

Suppose you were diagnosed as having either narcolepsy or sleep-onset insomnia. Write a couple of paragraphs to give a snapshot of your experience during a break at school or work, ensuring you refer to several key symptoms.
**Parasomnias**

Parasomnias are sleep disorders characterised by the occurrence of inappropriate physiological and/or psychological activity during sleep or sleep-to-wake transitions (Kennedy, 2016b). This activity may occur in association with specific sleep states or stages (such as REM sleep or a specific NREM sleep stage) and/or during the transitional stages of sleep (such as wake-to-sleep and sleep-to-wake). Essentially, they may occur while falling asleep, sleeping, between sleep stages, or during arousal from sleep.

Unlike dyssomnias, parasomnias are not abnormalities of processes underlying the sleep-wake cycle, sleep states, or in the quantity or timing of sleep or wakefulness. They are specific events that occur predominantly during a sleep episode, such as abnormal sleep-related motor activity, behaviours, emotions, perceptions, dreaming and autonomic nervous system functioning. Many of the parasomnias result from inappropriate activation of the central nervous system (usually transmitted through the autonomic nervous system or skeletal muscles) during various states or stages of sleep. Some result from a failure of neural processes, as can occur when respiration is impaired in breathing-related sleep disorders (AASM, 2014a; Kennedy, 2002; Thorpy, 2012).

Table 10.2 is a classification system for sleep disorders that lists some of the parasomnias in categories based on whether they are primary (occurring in their own right) or secondary (occur because of another condition). Primary parasomnias are also classified in subcategories based on whether events occur during NREM sleep, REM sleep or either of these. Secondary sleep parasomnias are undesirable or troublesome motor, behavioural or physiological events that occur during sleep. Generally, the secondary sleep parasomnias result from the activity of one of the bodily systems and are classified according to the system with which they are associated (Kennedy, 2002).

Sleep apnoea and sleep walking and are among the more common and better-known parasomnias. In this section we examine these two disorders, with reference to their effects on a person’s sleep-wake cycle.

**FIGURE 10.9** Parasomnias are sleep disorders characterised by the occurrence of inappropriate physiological and/or psychological activity during sleep or sleep-to-wake transitions.
TABLE 10.2 Classification of sleep parasomnias

<table>
<thead>
<tr>
<th>PRIMARY SLEEP PARASOMNIAS</th>
<th></th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>A. NREM parasomnias</strong></td>
<td></td>
</tr>
<tr>
<td>1. Sleep starts (e.g. brief body jerks just after sleep onset)</td>
<td></td>
</tr>
<tr>
<td>2. Disorders of arousal (Sleep terrors and Sleep walking in the DSM-5)</td>
<td></td>
</tr>
<tr>
<td>3. Sleep drunkenness (e.g. prolonged transition from sleep to waking)</td>
<td></td>
</tr>
<tr>
<td><strong>B. REM parasomnias</strong></td>
<td></td>
</tr>
<tr>
<td>1. Dream anxiety attacks (Nightmare Disorder in the DSM-5)</td>
<td></td>
</tr>
<tr>
<td>2. Hypnopogenic hallucinations and/or sleep paralysis</td>
<td></td>
</tr>
<tr>
<td>3. REM sleep behaviour disorder (e.g. abnormal movements during REM sleep instead of the expected loss of muscle tone)</td>
<td></td>
</tr>
<tr>
<td><strong>C. Non-sleep state specific parasomnias</strong></td>
<td></td>
</tr>
<tr>
<td>1. Bruxism (e.g. repeated grinding or crunching of teeth during sleep)</td>
<td></td>
</tr>
<tr>
<td>2. Enuresis (e.g. involuntary urination during sleep after the age at which bladder control usually occurs)</td>
<td></td>
</tr>
<tr>
<td>3. Rhythmic movement disorder (e.g. body movements such as body rocking while on hands and knees or head banging on the pillow repeated regularly over an extended period)</td>
<td></td>
</tr>
<tr>
<td>4. Periodic movements of sleep (e.g. episodes of simple, repetitive, involuntary muscle movements during sleep such as the tightening or flexing of a leg muscle)</td>
<td></td>
</tr>
<tr>
<td>5. Sleep talking (also called somniloquy)</td>
<td></td>
</tr>
<tr>
<td><strong>SECONDARY SLEEP PARASOMNIAS</strong></td>
<td></td>
</tr>
<tr>
<td><strong>A. Central nervous system</strong></td>
<td></td>
</tr>
<tr>
<td>1. Seizures</td>
<td></td>
</tr>
<tr>
<td>2. Headaches</td>
<td></td>
</tr>
<tr>
<td><strong>B. Cardiopulmonary</strong></td>
<td></td>
</tr>
<tr>
<td>1. Sleep-related arrhythmias (e.g. heart beats irregularly, too fast, or too slow)</td>
<td></td>
</tr>
<tr>
<td>2. Nocturnal asthma (e.g. asthma symptoms disturb sleep)</td>
<td></td>
</tr>
<tr>
<td>3. Sleep apnoea (e.g. sleep-related breathing disorder)</td>
<td></td>
</tr>
<tr>
<td><strong>C. Gastrointestinal</strong></td>
<td></td>
</tr>
<tr>
<td>(e.g. acid reflux or symptoms like heartburn; painful contractions within oesophagus — the muscular tube connecting mouth and stomach)</td>
<td></td>
</tr>
</tbody>
</table>


Note: Other classification systems have more or less sleep disorders, sometimes with different names. They may also use more categories and classify disorders differently. For example, the Diagnostic and Statistical Manual of Mental Disorders (DSM-5) has a Parasomnias category but not Enuresis. In addition, sleep apnoea is categorised as a Breathing-Related Sleep Disorder, not a Parasomnia.

Sleep apnoea

Sleep apnoea is an involuntary cessation of breathing that occurs during as sleep. The term apnoea (sometimes spelled apnea) literally means ‘without breath’. The duration of the stoppage is usually short (about 10 seconds) but can last for a minute or longer. Eventually, the brain detects the lack of breathing or a drop in oxygen level and triggers an arousal from sleep so that breathing can be renewed. The arousal disrupts sleep and sleep quality, but restores muscle tone, thus opening the airway, and normal breathing resumes.

People with untreated sleep apnoea stop breathing repeatedly during their sleep, sometimes hundreds of times. An apnoea usually ends with a loud snore as the sleeper renews their breathing. This is sometimes accompanied by vocalisations that consist of gasps, choking sounds, moans, or mumblings. There may also be a body jerk or arm flinging, or the individual may even sit upright momentarily. Awakenings tend to be short and abrupt. Often, the sleeper will awaken long enough for their breathing pattern to become regular again before they go back to sleep. In most cases, the sleeper is unaware of these breath stoppages because they don’t always trigger an awakening involving full arousal (APA, 2013; AASM, 2014a).

This cycle of sleep apnoea — cessation of breathing, a brief awakening, renewal of breathing — is likely to repeat itself many times throughout a sleep episode. Furthermore, these multiple interruptions of the individual’s normal sleep–wake cycle can lead to insomnia and daytime sleepiness.

The severity of sleep apnoea varies considerably and generally depends on the number of apnoeas per hour of sleep, their duration and the extent to which they impair everyday functioning. Major symptoms include loud snoring; frequent awakenings; being out of breath, with a dry mouth or a headache; unrefreshing sleep regardless of duration; and daytime sleepiness, tiredness or fatigue. Given apnoea involves involuntary cessation of breathing, it is a potentially life-threatening sleep disorder.

The Apnoea-Hypopnoea Index (AHI) was created to quantify the severity of sleep apnoea. For this index, apnea refers to the total absence of airflow and hypopnea refers to a reduction in airflow. Generally, the AHI is calculated by dividing the number of apnoeas and hypnoeas with a duration of at least 10 seconds by the number of hours of sleep. The score indicates the number of breathing stoppages per hour, with less than five considered normal. Mild apnoea is 5–15 stoppages per hour, moderate is 15–30, and severe is greater than 30 (APA, 2013).
Three types of sleep apnoea are commonly described: obstructive, central and mixed. Of the three, obstructive sleep apnoea is the most common.

Obstructive sleep apnoea is caused by an upper airway obstruction during sleep. This usually occurs when the soft tissue in the back of the throat relaxes during sleep, thereby narrowing the airway. Similarly, if you sleep on your back (in a supine position), gravity can cause the tongue to fall back with the onset of muscle relaxation. This narrows the airway, which reduces the amount of air that can reach your lungs. A narrow floppy throat is also more likely to vibrate during sleep, which causes snoring. Large tonsils and adenoids (spongy tissue between the back of the nose and throat), being overweight or having a minor or major facial abnormality that may change the size of airways can also cause an obstruction and result in snoring.

Loud snores or brief gasps that alternate with episodes of silence that usually last 20 to 30 seconds form a characteristic snoring pattern associated with obstructive sleep apnoea. The snoring can also be very disruptive to the sleep of other people, especially bed partners. Obstructive sleep apnoeas are more prevalent in REM sleep, but may also occur during NREM sleep, particularly stages 1 and 2. They are rare during stages 3 and 4 (AASM, 2001; Mokhlesi, 2012).

Obstructive sleep apnoea involves a struggle to breathe against a blocked airway. With central sleep apnoea, the airway is not blocked but breathing stops because the brain fails to maintain breathing for reasons that remain unclear. There is no effort to breathe during a stoppage, the apnoeas occur more frequently, last longer and snoring is not as prominent as in obstructive sleep apnoea. Central apnoeas can occur at any time in a sleep episode but are most prevalent during NREM sleep, including sleep onset (AASM, 2001; Dempsey, et al., 2010; SHF, 2016f).

Mixed sleep apnoea, as the name implies, is a combination of obstructive and central sleep apnoea. The suspensions of breathing typically begin as central apnoeas but conclude as airway blockages (Lavie, 1996). With each apnoea, the brain arouses the sleeper so that breathing can resume. As a result, sleep is extremely fragmented and of poor quality.

Sleep apnoea is extremely common. It is estimated that about 5% of Australians suffer from this sleep disorder (especially if overweight), with around one in four men over the age of 30 years experiencing the disorder to some degree (Better Health Channel, 2016). However, there are many people with the disorder who have not been diagnosed or received treatment. For example, despite dozens or even hundreds of awakenings per night, most individuals with obstructive sleep apnoea have no recollection of the arousals. In fact, often the only evidence of this disorder is daytime fatigue and reports of loud snoring from bed partners.

As with other sleep disorders, there are various treatment options. Generally, treatment depends on the type of disorder, its symptoms and their severity for the individual involved.
Effects on sleep–wake cycle
Sleep apnoea severely disturbs sleep through multiple interruptions to the sleep process. With each apnoea, the individual must wake up enough to regain muscle control and renew their breathing. Each breathing cessation causes some degree of arousal, interfering with the natural progression of the sleep episode.

People can be partly or fully aroused many times during sleep, so sleep episodes tend to be very fragmented. For example, a person who stops breathing 100 times during sleep, often for 10 seconds or so, is aroused at least that many times in order to renew their breathing. This impacts on the total amount of actual sleep time as well as sleep quality.

Individuals with sleep apnoea typically complain about their sleep being unrestful (and therefore nonrestorative). The sleep fragmentation caused by apnoeas may continually interrupt sleep and also prevent and therefore reduce normal amounts of NREM stages 3 and 4 deep sleep. This partly accounts for individuals usually waking from a sleep episode feeling tired and not refreshed. Individuals undergoing treatment for apnoea often enter a prolonged period of REM sleep or NREM deep sleep as soon as the apnoeas disappear, indicating that the apnoeas have reduced access to these sleep states (Lavie, 1996).

Excessive sleepiness, loss of alertness for prolonged periods and fatigue during the normal waking period are extremely common among people with apnoea, as are other symptoms more directly associated with insomnia. For example, a common complaint of central apnoea is an inability to maintain sleep (AASM, 2014a; APA, 2013).

Sleep walking
Sleep walking, sometimes called somnambulism, involves getting up from bed and walking about or performing other behaviours while asleep. A sleep walking episode may involve activities that vary in type, degree of complexity and duration. The level of activity may be calm, moderate or vigorous. For example, a sleep walker may arise from bed calmly and quietly walk around for a couple of minutes then return to bed. By contrast, a sleep walker may bolt from the bed and move vigorously as if agitated or making a frantic attempt to escape from a fearful stimulus (AASM, 2014a; APA, 2013).

Most sleep walkers typically engage in activities that are routine and of low complexity. The episode usually ends spontaneously if the sleep walker is left alone. For example, they will return to bed, lie down and continue to sleep without awakening.

The sleep walker’s eyes are usually open but their eyes have a ‘blank stare’ or glassy ‘look right through you’ appearance. Movements often occur in a confused and clumsy manner. During a calm episode, however, the sleep walker tends to maintain coordination and is often able to successfully walk up or down stairs and navigate around large obstacles. When they arise from bed, they may immediately walk towards a stimulus such as a light or noise, or they may walk aimlessly around a room or from room to room. Children usually walk to their parents’ bedroom (Kennedy, 2002). Sleep walkers do not walk with their arms extended in front of them as is sometimes inaccurately depicted in movies.

Sometimes, the sleep walker may perform routine, well-learnt activities, such as opening doors, dressing, cleaning, eating or packing a bag to go to school or work. They may even leave the house. More complex behaviours such as driving a car are possible, but rare. Occasionally, the sleep walker may perform socially inappropriate behaviours, such as urinating in a bin or cupboard, especially if a child. Sleep talking may also occur during an episode. For example, they may say or mumble a few words. However, the sleep walker is typically unresponsive to any attempt to communicate with them (AASM, 2014a; APA, 2013; Mason & Pack, 2007; NSF, 2016b).

A major concern about sleep walking is the risk of self-injury. It can result in falls and injuries. In most cases sleep walkers do not suffer any harm, but occasionally they may injure themselves and have also been known to injure others (and in rare cases even commit murder). Painful injuries sustained during sleep walking are often not perceived until the individual awakens (APA, 2013; Kennedy, 2002; Mason & Pack, 2007).
Sleep walking episodes may occur up to 3 or 4 times a week. They generally last only a few minutes, rarely beyond 15 minutes (but have been known to last as long as one hour or so). More than one episode a night is rare. There is little awareness of what is going on during an episode. When an episode is over, most people are able to remember very little of what they did, if anything at all. The frequency of sleep walking may be underestimated because of episodes that are unremembered or unobserved by someone else (APA, 2013; Bruck, 2006; Kennedy, 2002; Mason & Pack, 2007).

Sleep walking usually occurs during the deep sleep of NREM stages 3 and 4 when we have no sleep paralysis and are therefore able to move around. This means that it is also more likely to take place during the first third of a sleep episode. It is most commonly first initiated at the end of the first or second episode of slow wave sleep. Sleep walking can also be initiated in the lighter stages of NREM sleep, and the sleep walker may be partially aroused during the episode (AASM, 2001; APA, 2013; NSF, 2016h).

Many people mistakenly believe that it is dangerous to waken sleep walkers. It can, however, be quite dangerous not to wake a sleep walker in case they harm themselves. While sleep walkers can be awakened, it is usually with great difficulty because of their deep sleep state. When awakened, they may not recognise family or friends and it can take up to 20 minutes to calm them. If the sleep walker wakes suddenly during an episode, they are often confused and disoriented (Bruck, 2006; NSF, 2016h).

Sleep walking is very common in childhood. It is estimated that between 10–30% of children have had at least one sleep walking episode, and that 2–3% walk often (APA, 2013). Sleep walking episodes can occur as soon as a child is able to walk, but sleep walking typically begins at about age seven, generally before age 10, and ends before age 15 or so (Kennedy, 2002). For some people, however, sleep walking may continue for most of their life. The longer a child keeps sleep walking into their adolescent years, the greater the chance that it will go on into early adulthood. It is estimated to occur among 8% of secondary school students and about 1–5% of adults (APA, 2013; Bruck, 2006; NSF, 2016h).

The cause of sleep walking remains unclear, especially the neurological basis of the disorder. One reason is that the objective study of sleep walking is often difficult because episodes rarely occur in sleep laboratories. It is an unusual disorder, especially as the brain is partially aroused as if in a waking state, which enables often complex behavioural activities, and partially in deep NREM sleep, with no conscious awareness of these actions (Lopez, et al., 2013).

Sleep walking may happen for no obvious reason. The more common triggers include stress, anxiety and the use of particular medications, especially those that reduce or suppress REM sleep. Several studies have demonstrated a significant family history of sleep walking. The chance of having the disorder can greatly increase if one or both parents had sleep walking episodes as a child or adult. In addition, medical conditions such as seizures and fever can increase the likelihood of sleep walking, and episodes have also been associated with other sleep disorders, particularly sleep apnoea and sleep terror disorder (see box 10.3).

Generally, sleep walking is not considered to be a serious sleep disorder. Although it can disrupt sleep if the sleep walker awakens and can be unsettling for parents of child sleep walkers, sleep walking is not associated with any significant physical or mental health condition or any long-term complications. Common advice is that action should be taken only if sleep walking happens too often, if there is a major risk of harm to the sleep walker or someone else, or if the sleep walking is adversely impacting on the individual's everyday functioning.

There is no specific treatment for sleep walking, but basic changes that improve sleep habits can
reduce or eliminate episodes. For example, simply getting the amount of sleep needed each night can prevent episodes for some people. Hypnosis has been found to be very effective, and the use of relaxation techniques and certain medications have also been found to be helpful in reducing the incidence of sleep walking in some people (APA, 2013; Kennedy, 2002; NSF, 2016h).

**Effects on sleep-wake cycle**

Sleep walking typically occurs in the first third of a sleep episode and is most commonly initiated during NREM stages 3 or 4 when the individual is in deep sleep. It is primarily distinguished by mobility and activity during sleep, which is abnormal for sleep. There is also considerable difficulty in arousing the individual during a sleep walking episode. However, this is not abnormal given it is also expected of the non-sleep walker in a deep sleep state.

If sleep walkers wake up suddenly, they are often confused and can take a while to get a sense of what’s happening and where they are, which is what usually occurs with anyone who is abruptly awaken from deep sleep. Sometimes, the sleep walker may not be able to quickly go back to sleep after a sudden awakening. This sleep disturbance means there is a loss of deep sleep and that the sleep episode will be fragmented (but not as fragmented as often occurs with sleep apnoea). Loss of deep sleep in particular is likely to result in a sleep episode that is not as restful as normal, which will probably make the individual more tired than usual during the day.

It is widely believed that the effects of sleep walking tend to be confined to the nighttime episodes, but some researchers have found that sleep walkers do experience daytime effects from the disorder. For example, Canadian psychologist Antonio Zadra and his colleagues (2013) have reported that nearly 45% of sleep walkers experience daytime sleepiness and tend to do so more often than non-sleep walkers. In addition, adolescent sleep walkers experienced daytime fatigue but tended to more easily mask their tiredness and sleepiness. However, if they were given the opportunity to have a nap, they went to sleep faster than non-sleep walkers. In addition, they performed worse on concentration tasks than non-sleep walkers.

A French study that compared over 140 adult sleep walkers with a control group of non-sleep walkers also found a significant link between sleep walking and daytime impairments. The sleep walkers used in the study had a median age of 30 years. All had been diagnosed with the disorder at the sleep disorder clinic where the researchers worked and were subsequently asked to participate in the study. Twenty-two per cent reported daily episodes of sleep walking and 43% had weekly episodes. Table 10.3 shows some of their results. On the basis of their results, the researchers concluded that there were significant associations between sleep walking and daytime sleepiness, fatigue and insomnia. However, the researchers noted that these sleep walkers had found their condition debilitating enough to seek professional assistance, so they may not be representative of sleep walkers in the general population (Lopez, at al., 2013).
#### TABLE 10.3  Daytime functioning and night time sleep problems in sleep walkers and controls

<table>
<thead>
<tr>
<th></th>
<th>Sleepwalkers (%) (n=100)</th>
<th>Controls, (%) (n=100)</th>
<th>P value</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Daytime sleepiness</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Epworth sleepiness scale score (median)</td>
<td>10.00</td>
<td>6.50</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Daytime fatigue</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Chalder Fatigue Scale score (median)</td>
<td>6.00</td>
<td>3.00</td>
<td></td>
</tr>
<tr>
<td><strong>Insomnia</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Total Insomnia Severity Index score (median)</td>
<td>14.00</td>
<td>4.00</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td><strong>Score on Insomnia Severity Index subcategories</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Difficulty initiating sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never/rarely</td>
<td>63.22</td>
<td>84.29</td>
<td>0.01</td>
</tr>
<tr>
<td>Moderate</td>
<td>21.84</td>
<td>12.86</td>
<td></td>
</tr>
<tr>
<td>Frequently/often</td>
<td>14.94</td>
<td>2.86</td>
<td></td>
</tr>
<tr>
<td>Difficulty maintaining sleep</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never/rarely</td>
<td>36.78</td>
<td>77.14</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Moderate</td>
<td>32.18</td>
<td>17.14</td>
<td></td>
</tr>
<tr>
<td>Frequently/often</td>
<td>31.03</td>
<td>5.71</td>
<td></td>
</tr>
<tr>
<td>Early morning awakenings</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never/rarely</td>
<td>51.72</td>
<td>74.29</td>
<td>0.005</td>
</tr>
<tr>
<td>Moderate</td>
<td>18.39</td>
<td>17.14</td>
<td></td>
</tr>
<tr>
<td>Frequently/often</td>
<td>29.89</td>
<td>8.57</td>
<td></td>
</tr>
<tr>
<td>Satisfaction</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Very satisfied/Satisfied</td>
<td>12.64</td>
<td>50.00</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Moderately satisfied</td>
<td>21.84</td>
<td>28.57</td>
<td></td>
</tr>
<tr>
<td>Dissatisfied/very dissatisfied</td>
<td>65.52</td>
<td>21.43</td>
<td></td>
</tr>
<tr>
<td>Interference</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Never/rarely</td>
<td>28.14</td>
<td>81.16</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Moderate</td>
<td>21.84</td>
<td>13.04</td>
<td></td>
</tr>
<tr>
<td>Frequently/often</td>
<td>54.02</td>
<td>5.80</td>
<td></td>
</tr>
<tr>
<td>Noticeability</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate/frequently/often versus never/rarely</td>
<td>32.18</td>
<td>7.14</td>
<td>&lt;0.0001</td>
</tr>
<tr>
<td>Distress</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Moderate/frequently/often versus never/rarely</td>
<td>77.01</td>
<td>7.14</td>
<td>&lt;0.0001</td>
</tr>
</tbody>
</table>


#### LEARNING ACTIVITY 10.5

**Reflection**

Some researchers have found that sleep walkers engage in many more violent behaviours during sleep walking episodes than is commonly reported. For example, the person attempting to awaken the sleep walker can be violently attacked. Rarely, homicide during an apparent sleep walking episode has also been reported (Lopez, R., et al., 2013). Comment on whether sleep walking should be a legitimate murder or manslaughter defence with reference to sleep walking as an altered state of consciousness.

**eBookplus**

Media reports on sleep walking homicide cases

Transcript from an Australian trial
**BOX 10.3**

**Nightmares and sleep terrors**

Sleep can be an awful time for some people who experience parasomnias. The symptoms of these disorders range from the mundane to the horrifying, and worrying about what might happen during a night of sleep can cause some people who experience parasomnias to want to avoid sleep (Epstein & Mardon, 2007).

Nightmares and sleep terrors are among the most easily identifiable parasomnias. Both involve disturbing dreams but they have a number of distinguishable features.

*Nightmares* (also called *dream anxiety attacks*) are frightening dreams that occur during REM sleep. These are typically experienced in the last third of a sleep episode. Dream content is usually recalled in vivid detail if the person wakes up, but sometimes at a later time.

A common theme is the dreamer’s experience of helplessness in undesirable circumstances. The dreams have visual images that are frightening enough or negative emotions that are strong enough to cause the dreamer to wake up scared or anxious. This feature differentiates a nightmare from a ‘bad’ dream that doesn’t cause awakening.

The nightmare is almost always a long, complicated dream that becomes increasingly frightening toward the end. Fear of death is often present but the element of fright or anxiety is an essential feature. A child will often dream of frightening imaginary creatures, such as monsters or ghosts. An adult may also dream about imaginary events that are threatening or harmful in some way, but this is less common when compared with childhood nightmares. The longer duration of nightmares is one of the many features that distinguish them from sleep terrors (AASM, 2014a; SHF, 2016g).

Nightmares are more likely to occur during times of stress, anxiety, fatigue or personal trauma. Illness, medications, illegal drugs or even watching a scary film can also set them off. They usually start at ages three to six years but can occur at any age. They are experienced more commonly by children than adults, and decrease in frequency with age. It is estimated that 10% to 50% of children have them, with the number of adults much less at about 2.5% to 10%. They are more common among women than men, but this may be due to their willingness to discuss or report them more than men do (SHF, 2016g).

Many nightmares occur for no particular reason, although being sleep deprived makes them more likely. They have also been associated with certain medications, alcohol and various illegal drugs. In some cases, recurring, intense nightmares can follow a traumatic experience (Bruck, 2006).

*Sleep terrors* (once called *night terrors*) are characterised by sudden awakening from NREM stage 3 or 4 sleep in a terrified state. These are typically experienced during the first third of a sleep episode, most often during the first sleep cycle. The person often sits upright in bed, is unresponsive to external stimuli, and is confused and disoriented. There is little or no recall of the dream on awakening (SHF, 2016h).

Sleep terrors tend to occur less frequently than nightmares and typically last only a few minutes, but are generally more distressing. There is usually much more fear or anxiety than with a nightmare. A person may awaken screaming or crying, sweating profusely, with wide eyes and dilated pupils and with a terrified expression on their face. They may speak incoherently and appear in a state of confusion or panic. Their breathing is usually rapid and their heart rate is often double or treble its normal rate. While in this distressed state, they are usually unresponsive and difficult to comfort. The terrified reactions may last for several minutes until they eventually relax or return to sleep.

By contrast, nightmares usually do not involve major motor activity. There is considerably less anxiety, vocalisation and autonomic nervous system activity during a nightmare than during a sleep terror (AASM, 2014a).

Sleep terrors are far less common than nightmares. It is estimated that they occur in 1–5% of children. In adults it is even less, about 1% to 2% (SHF, 2016h). Sleep terrors are much more common in pre-school children (three to five years), especially in boys. Children’s experiences of sleep terrors are usually temporary and they normally stop having them as they get older. One explanation for this is that the amount of NREM stages 3 and 4 sleep experienced is greater in childhood and diminishes with age. Triggers for sleep terror episodes include sleep deprivation, anxiety, a sudden noise, fever and depressant medications. People who have sleep terrors often sleep walk as well (AASM, 2014a; Hartmann et al., 1987).

**FIGURE 10.15** Nightmares are frightening dreams that typically occur during REM sleep and can be recalled on awakening. Sleep terrors are terrifying dreams that typically occur during NREM deep sleep and there is little or no recall of the dream on awakening. They are more distressing than nightmares.
LEARNING ACTIVITY 10.6

Review questions

1. (a) What is sleep apnoea?
   (b) List three common symptoms of sleep apnoea.
   (c) Describe two possible causes of sleep apnoea.
   (d) Give a possible reason for why obstructive sleep apnoea is more prevalent in REM sleep.
   (e) Give three examples of how sleep apnoea can affect a person’s circadian sleep–wake cycle.
   (f) Explain why sleep apnoea is classified as a parasomnia rather than a dyssomnia.

2. (a) What is sleep walking?
   (b) (i) When in a sleep episode is sleep walking most likely to occur?
   (ii) Why is sleep walking unlikely to occur during REM sleep?
   (c) List four distinguishing features of sleep walking.
   (d) Give three examples of how sleep walking can effect a person’s circadian sleep–wake cycle.
   (e) Explain why sleep walking is classified as a parasomnia rather than a dyssomnia.

3. Complete the table below to summarise key features of the sleep disorders examined in this section. Ensure you also complete details for the dyssomnia and parasomnia concepts.

<table>
<thead>
<tr>
<th>Sleep disorder</th>
<th>What is it?</th>
<th>Key symptoms/ features</th>
<th>When most likely to occur in a sleep episode</th>
<th>Effects on circadian sleep–wake cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Dyssomnia</td>
<td>Sleep-onset insomnia</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Parasomnia</td>
<td>Sleep apnoea</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>Sleep walking</td>
<td></td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

CIRCADIAN RHYTHM PHASE DISORDERS

Under normal conditions, our internally programmed circadian sleep–wake cycle and the sleep–wake schedule we maintain are closely aligned. This is essential to our ability to keep sleep and wakefulness in-sync with our environment and to undertake our daily activities as best we can. The importance of synchronisation becomes apparent when our sleep–wake cycle and sleep–wake schedule get out of phase, or ‘out of synch’.

Circadian rhythm phase disorders (also called circadian rhythm sleep–wake disorders) are a group of sleep disorders involving sleep disruption that is primarily due to a mismatch between an individual’s sleep–wake pattern and the pattern that is desired or required. The disruption may be caused by:

- a naturally occurring change or a malfunction of biological mechanisms or processes regulating the sleep–wake cycle, or
- a mismatch between an individual’s sleep–wake cycle and the sleep–wake schedule required by their school, work or social schedule, or
- a mismatch between an individual’s sleep–wake cycle and the day–night cycle of their physical environment (APA, 2013).

Circadian rhythm phase disorders essentially involve a problem with the timing of the sleep and wake states. The individual cannot sleep when sleep is desired, needed, or expected. As a result of sleep episodes occurring at the least preferable or inappropriate times, the corresponding wake periods may occur at undesired times. Therefore, the individual usually complains of insomnia and/or excessive sleepiness. For most circadian rhythm phase disorders, once sleep is initiated, the sleep episode will tend to have its natural duration, with NREM-REM sleep cycles occurring as they normally do (AASM, 2014a).

Circadian rhythm phase disorders are primarily attributable to mechanisms and processes that generate and time sleep, and produce difficulty initiating, maintaining and/or timing sleep, so they are commonly classified as dyssomnias.

In this section, we examine three circadian rhythm phase disorders: the sleep–wake cycle shift in adolescence and disturbance of the cycle through shift work or by air travel across time zones. All three can result in a mismatch between our circadian sleep–wake cycle and our desired or required sleep–wake schedule. When this happens, our sleep–wake cycle can also become misaligned with our daily activity pattern.
Sleep–wake cycle shift in adolescence

Numerous research studies have found that adolescents need about 9.25 hours sleep a night to function at their best when awake, yet many males and females between the ages of 13 and 19 years sleep considerably less than this every night. Studies of adolescent sleep patterns also indicate that this is a period of high sleep disturbance. The time around puberty is associated with the onset of a characteristic pattern of sleep problems. These are associated with the tendency to stay up longer in the evenings and include feeling sleepy at a much later time, insufficient night-time sleep on weekdays and considerable difficulty waking in the morning (Bruck, 2006; Carskadon, 2002; Short, et al., 2013).

Insufficient sleep can have significant effects on daytime alertness and normal daytime functioning. For adolescents at school, it can affect the ability to concentrate, think and learn. Daytime impairments can include excessive sleepiness; inattention and mentally ‘drifting off’ in class; problems with staying motivated to complete class work; lethargy; and difficulties with mood regulation and behaviour control (Blunden, 2013; Bruck, 2006).

Psychologists explain adolescent sleep patterns and problems in terms of biologically driven changes and psychological and social factors that interact to exert considerable pressure towards going to sleep at a later time than would naturally occur.

Biological influences on an adolescent’s sleep primarily involve the biological clock regulating the circadian sleep–wake cycle through melatonin secretions. During adolescence, there is a hormonally induced shift of the sleep–wake cycle forward by about 1 to 2 hours. More specifically, the timing of melatonin secretion that induces sleep onset peaks later in the 24-hour cycle and makes the adolescent sleepier 1 to 2 hours later. Their bodies are not ready to sleep when their real-world clock shows that it is time to sleep.

This change in the timing of the major sleep episode is known as a sleep–wake cycle shift and affects an adolescent’s ability to fall asleep at the earlier times they did as a child. So a ten year old may have been sleepy and ready for bed at 9 pm every night but at, say, 15, doesn’t feel at all sleepy at 9 pm. The naturally occurring delay in the timing of sleep onset also means that there is a biologically driven need to sleep one to two hours longer given the changed sleep onset timing. That’s not a problem if the time of getting up in the morning can be chosen. However, most adolescents do not have that luxury on most days of the week. School or work starts at a set time even if their biological clock makes them feels like it’s one or two hours too early. This means that early school (or work) starts don’t allow the adolescent to sleep in and get the additional sleep that would naturally occur (Blunden, 2013; Bruck, 2006).

In sum, the entire sleep–wake cycle is delayed by 1–2 hours in relation to the desired sleep and wake-up time. This type of sleep–wake cycle shift is called a delayed sleep phase disorder. Efforts to advance the timing of sleep onset, such as early bedtime, dimming the lights earlier, restricting the use of digital technologies when in bed to earlier times and relaxation techniques can be helpful at times, but generally result in little permanent success (AASM, 2014a; APA, 2013).

Some people are more affected by melatonin delaying their evening wave of sleepiness than others. Many cope with the change in their hours of sleep...
but some do not. A significant problem is that nightly sleep loss due to having to wake up earlier than the body wants to can accumulate as a sleep debt.

**Sleep debt** is sleep that is owed and needs to be made up because daily sleep requirements have not been met. It is sometimes described as the difference between the amount of sleep that is needed to function at an optimal level and the amount a person actually has. For example, a nightly sleep debt of 90 minutes between Monday and Friday would add up to a total sleep debt of 7.5 hours. On the weekends, adolescents will often sleep in to make up their sleep loss. However, this usually results in going to bed even later, which can temporarily shift the major sleep episode further forward so that by Monday morning, getting out of bed to go to school (or work) is harder than on any other day.

Sleep debt does not continue to build up until repaid. For example, if you slept for one hour less than you needed to for 28 days, this does not mean that you need to sleep for 28 extra hours to function at an optimal level again. Generally, after a period of sleep deprivation, only some of the sleep debt needs to be recovered. It the sleep–wake cycle is allowed to take its natural course without interruption from an external cue, we will tend to sleep longer on the first night and possibly the second, by which time the hours of sleep will have reduced back to the optimal amount required.

Psychological and social factors also influence an adolescent's sleep habits, often in ways that contribute to their sleep–wake cycle shift and associated sleep problems. Adolescents typically like to exert their growing need for independence, which can include making decisions about when to go to bed or sleep. Many usually decide to go to bed or sleep later, particularly as early sleep times are associated with childhood. Adolescents also experience increased demands on their time for socialising and increased academic or work demands compared to when they were children. Many have casual or part-time jobs. Adolescents who work long hours or who stay up late doing homework, studying, texting, catching up with others on Facebook, watching movies, playing with phone apps and listening to loud music are more likely to experience greater difficulty waking up in the morning than those who do not (Blunden, 2013).

Essentially, sleep seems to be a low priority for many adolescents. Research suggests that the ‘typical’ adolescent's natural time to fall asleep may be 11 pm or later (NSF, 2016i). Despite this, many stay awake long after their biological clock has promoted sleep onset. This typically results in erratic sleep habits that compound sleep problems, build up an excessive sleep debt and result in sleep deprivation to an extent that functioning during waking time is significantly impaired.

If puberty is considered as marking the onset of adolescence, with its associated delay in evening sleepiness, a relevant question that arises involves when adolescence ends. More specifically, when does the delay in sleepiness start to wear off and the adolescent feel like going to bed a bit earlier, more like the time of their parents? Research suggests that there is an abrupt change in the timing of sleep around the age of 20 years, suggesting that this may be a biological marker of the end of adolescence (Bruck, 2006).
BOX 10.4

Sleep tips for adolescents

• Make sleep a priority. Decide what you need to change to get enough sleep to stay healthy, happy and smart!
• Naps can help pick you up and make you work more efficiently, if you plan them right. Naps that are too long or too close to bedtime can interfere with your regular sleep.
• Make your room a sleep haven. Keep it cool, quiet and dark. If you need to, get eyeshades or blackout curtains. Let in bright light in the morning to signal your body to wake up.
• No pills, vitamins or drinks can replace good sleep. Consuming caffeine close to bedtime can impair sleep onset because it is a stimulant, so avoid coffee, tea, energy drinks, soft drinks and chocolate late in the day so you can get to sleep at night. Nicotine and alcohol will also interfere with your sleep.
• Establish a bed and wake-time and stick to it, coming as close as you can on the weekends. A consistent sleep schedule will help you feel less tired since it allows your body to get in sync with its natural patterns. You will find that it’s easier to fall asleep at bedtime with this type of routine.

• Don’t eat, drink, or exercise within a few hours of your bedtime. Don’t leave your homework for the last minute. Try to avoid the TV, computer and mobile phone in the hour before you go to bed. Stick to quiet, calm activities, and you’ll fall asleep much more easily!
• If you do the same things every night before you go to sleep, you teach your body the signals that it’s time for bed. Try taking a bath or shower (this will leave you extra time in the morning), or reading a book.
• Try keeping a diary or to-do lists. If you jot notes down before you go to sleep, you’ll be less likely to stay awake worrying or stressing.
• When you hear your friends talking about their all-nighters, tell them how good you feel after getting enough sleep.
• You can’t change your sleep–wake cycle shift, but activities at night that are calming can help counteract heightened alertness.

Source: adapted from National Sleep Foundation (2016). Teens and sleep (Sleep Topics). Retrieved from https://sleepfoundation.org/sleep-topics/teens-and-sleep/page/0/1

LEARNING ACTIVITY 10.7

Review questions

1. Explain the meaning of the term sleep–wake cycle shift.
2. Briefly describe the sleep–wake cycle shift that occurs during adolescence.
3. (a) Why is the sleep–wake cycle shift classified as a circadian phase disorder?
   (b) Explain why this type of shift could also be classified as a dyssomnia rather than a parasomnia.
4. Explain why there is a sleep–wake cycle shift during adolescence, ensuring you adopt a biopsychosocial perspective.
5. (a) What is sleep debt?
   (b) How is it calculated?
   (c) How can sleep debt adversely impact on the sleep–wake cycle?
6. What are two potential consequences of the adolescent sleep–wake shift on sleep patterns or activity, other than sleep debt?
7. To what extent may it be possible to readjust or compensate for the sleep–wake shift? Explain your answer.

LEARNING ACTIVITY 10.8

Reflection

Comment on whether there should be a later start to the school day to accommodate the sleep–wake cycle shift in adolescence and whether this would be an effective means of addressing sleep-related problems of adolescents at school.

Shift work

We live in a globalised world that operates 24 hours a day, 7 days a week (called ‘24/7’). Shift work is a type of work schedule designed to meet the demands of a 24/7 society. The practice typically divides the 24 hour day into shifts — set periods of time of about 8 hours or so during which employees perform their duties.

In Australia, three traditional shifts are the day, afternoon and night shifts. Day shifts typically start and end during the daytime, afternoon shifts start mid-afternoon and end in the evenings, and night shifts start late in the evening and end during the daytime. These may be on a fixed schedule and require employees to work the same shift on a regular, ongoing basis, or they may be on a rotating schedule and require employees to change shifts every so often to work a mix of day and/or afternoon and/or night shifts.
Numerous jobs within our society involve shift work. For example, shift work is common for police, paramedics and fire fighters; doctors and nurses; pilots and airline staff; customs and immigration officers; hospitality staff; transport drivers; security staff; mail sorters; miners and factory workers. Many of these jobs involve some degree of danger to the individual involved or carry significant responsibility for the safety and wellbeing of others. It is estimated that at least 1.5 million Australian employees (16%) follow a shift work schedule in their main job, with the most common type of shift being the rotating shift (45% of those who work shift work) (ABS, 2013).

Psychologists are particularly concerned about sleep disturbances associated with shift work that takes place outside the times of the normal ‘9 to 5’ work day, especially at night when the work is scheduled during the habitual hours of sleep. We are not nocturnal beings. Our body has a sleep–wake cycle that is biologically programmed to sleep best at night and to be awake and most alert during the day and early evening. Night shift work in particular disrupts this cycle and causes sleep-related problems.

People who work on permanent night shift tend to experience problems with sleep quantity and quality more than people who do not do shift work. They often complain of being tired, both on and off the job. It is often not easy to sleep enough or to sleep well during the day. Many sleep less when they go to bed in the morning after a night shift. The reduction in sleep amount may be between 1 to 4 hours less a day than someone who doesn’t work shifts. The sleep loss and circadian cycle disruption represent the main causes of sleepiness among shift workers. Many accumulate a sleep debt as they struggle to adjust to the disruption while juggling work and lifestyle demands (AASM, 2014a).

Night shift workers also have a greater tendency to sleep twice during the day — a major episode in the morning after work and then a nap of an hour or so before going to work. However, they often find it difficult to fall asleep and/or maintain sleep during the day despite attempts to optimise environmental conditions for sleep. For example, during the day, there is more light, the phone rings more frequently and visitors may arrive. All these can interfere with daytime sleeping, fragmenting the major sleep episode and thereby compromising the quality of the sleep episode. Difficulties with sleep onset or maintenance may lead to a difficulty in awakening. Overall, the major sleep episode of the night shift worker is reported by a significant number as unsatisfactory and unrefreshing. In addition, it is common for night shift workers to revert to daytime routines for a day or two during days off, which tends to make their circadian rhythm for the sleep–wake cycle unstable (AASM, 2014a).
Excessive sleepiness is often experienced during the night and may impair performance because of the reduced alertness. This has consequences for safety. For example, it is believed to contribute to the significant number of on-the-job accidents in the middle of the night or in the early hours of the morning, when employee performance also tends to be significantly lower. There is also a higher risk of accidents on the road, driving to and from work (AASM, 2014a; Dement & Vaughan, 1999; SHF, 2016i).

Work rosters with rotating shift work schedules are associated with a higher frequency of sleep disturbances than rosters with fixed schedules. In particular, the most difficult rotating schedules to adjust to are those that change too quickly from one shift type to another because of the lack of time for the sleep–wake cycle to adjust and align with the day-night cycle of the individual’s environment and other external sleep–wake cues.

Generally, if rotating shifts have to be used, the longer a person works on a particular shift, the more likely it is that their sleep–wake cycle will make at least some adjustments, and the better for the individual. A work roster for which the individual has longer periods on each shift before rotating to the next shift also tends to be better because it allows the individual to have a longer period off between one shift rotation and the next. This gives the body more time to reset its sleep–wake cycle to get in-sync with the external environment. A schedule with three-week shifts is generally considered preferable to one-week or three-day rotations.

We also tend to adapt more quickly when assigned to successively later shifts rather than to successively earlier shifts. It therefore tends to be best when the move from one shift to the next is a forward move so the new shift begins later in the day. For example, if a person has been working a day shift from 7 am to 3 pm, their next shift should be the afternoon shift, say from 3 pm until 11 pm, rather than moving backwards to an 11 pm to 7 am shift. Because our natural sleep–wake cycle is closer to 25 hours (see box 9.1), by moving forwards through the shift rotation, the cycle is disrupted less than if a worker moved backwards through a shift rotation. Thus, workers will tend to adapt better and experience less disruption to their physiological and psychological functioning with a forward move than a backward move (Czeisler, Moore-Ede & Coleman, 1982; SHF, 2016i).

Some shift workers complain of excessive sleepiness at work and impaired sleep at home on a persistent basis to the extent that they may be diagnosed as having shift work sleep disorder. The two primary symptoms of shift work disorder are insomnia when a person is trying to sleep, and excessive sleepiness when a person needs to be awake and alert. Complaints tend to be more common and severe in relation to night shift work and inappropriately scheduled rotating rosters that can include double shifts and quick shift changes (AASM, 2014a).

Relatively few people seem to fully adapt to the night shift even after many years of night shift work, in part because of resumption of full daytime activities and night-time sleep during weekends and vacations. For example, a person may work the night shift for five consecutive nights, followed by two days and two nights off. During this ‘weekend’, the person may revert to a typical night-time–sleep/daytime–awake schedule in order to spend time with family and friends. This causes their internal sleep–wake cycle to shift again, thus requiring another adjustment when the night shift work week begins. Without a constant sleep–wake schedule during the entire week, the body’s internal circadian rhythm may always remain out of sync with the external environment (Czeisler, 2007).
LEARNING ACTIVITY 10.9

Review questions
1 Explain how shift work can disrupt a person’s sleep–wake cycle and make them susceptible to a circadian phase disorder.
2 What are the two major symptoms of shift work disorder?
3 Give an example of a change to sleep timing, quantity and quality that may be caused by shift work, ensuring you explain each change.
4 In what ways can psychological and social factors contribute to the development or maintenance of a circadian phase disorder due to shift work?
5 (a) What two features of a ‘shift-friendly’ roster may minimise sleep–wake cycle disruption and shift work effects?
   (b) Explain the rationale underlying each desirable roster feature.
6 Explain whether it would be worthwhile to manipulate lighting conditions in a night shift workplace to minimise sleepiness.
7 Examine the hypnograms in figures 10.6 and 10.8. Draw hypnograms showing a possible sleep–wake cycle of a night shift worker and that of a day shift worker.

Jet lag
If you have travelled by aeroplane across two or more time zones in one trip, then there is a good chance that you have experienced jet lag, especially if you went in an easterly direction. Jet lag, also called time zone change syndrome, is a sleep disorder due to a disturbance to the circadian sleep–wake cycle caused by rapid travel across multiple time zones. Shifting to a new time zone in this way results in a mismatch between our internal circadian biological clock and the external environment — our biological clock is out of sync with the actual time in the time zone of the new environment. For example, our body feels that it is time to go to sleep when others are having breakfast or it is in the middle of the afternoon in the new time zone, and we feel wide awake when it is late at night and everyone else is in bed, fast asleep.

Because jet travel is quick, our sleep–wake cycle remains aligned to the environmental time cues of the home time because there has been insufficient time to adjust to the new time cues. Consequently, our natural sleep–wake cycle (along with other circadian rhythms) is out of sync and in conflict with the light–dark and other time cues of the external environment. The desynchronisation is temporary and our brain and body need to adjust to the new environmental conditions, including resetting of the sleep–wake cycle. In the interim, we experience the effects of jet lag during the adjustment process.

FIGURE 10.21 Long-distance aeroplane passengers who rapidly travel across multiple time zones experience jet lag because their circadian sleep–wake cycle remains aligned to the environmental time cues of their home time zone.
Jet lag effects include both physical and psychological symptoms that may leave us with sleep problems, feeling unwell and having more difficulty functioning than normal. We tend to experience varying degrees of difficulties in initiating or maintaining sleep (e.g. when trying to sleep at a time that is out-of-synch with our home time), excessive sleepiness (e.g. during the period when our biological clock is set for sleep), reduced daytime alertness, impaired concentration and cognitive performance, and digestive problems. A vague feeling of bodily discomfort or ‘not feeling right’, as may be experienced when starting to feel ill, is also common. Called ‘malaise’, this feeling is believed to primarily result from the loss of harmony among the various biological rhythms governed by the circadian system, some of which adjust to the new time zone more rapidly than others. Problems that can occur solely or largely as a result of cabin conditions in the aeroplane, such as a headache, a blocked nose, nausea and muscle cramps, are not considered jet lag symptoms (AASM, 2014a).

Research studies have found that jet lag tends to be associated with a greater number of arousals and a greater percentage of NREM stage 1 sleep during the first two to three sleep episodes after arrival compared to home-based sleep. Most often, the second half of the sleep episode is the more severely disrupted, regardless of the direction of the travel (AASM, 2014a).

The severity and duration of jet lag symptoms vary considerably, depending on the number of time zones crossed in one journey, the direction (east or west) of the travel, the timing of takeoff and arrival, sleep timing, duration and quality on the flight, and personal characteristics of the individual involved (e.g. older adults usually have more difficulty adjusting to time differences than younger adults and children). Generally, the further you travel and more time zones you cross in one journey, the longer it is likely to take to adjust. In addition, as shown in figure 10.22, most people find it less disruptive and symptoms do not last as long when they travel in a westerly direction (which lengthens their day), in contrast to travelling in an easterly direction (which shortens their day). Jet lag following north–south travel does not occur if there is no more than a 1- or 2-hour change in time zone (AASM, 2014a).

The sleep–wake cycle disturbances generally reduce after two to three days at the destination. Adaptation of the timing of biological functions other than sleep and waking may take up to eight or more days. Overall, adaptation is an entrainment process requiring our biological clock to be reset through the influence of environmental time cues in the new location. Our biological clock readjusts itself a little bit each day until it is aligned with the external environment. The environmental cues that influence the entrainment process include exposure to light in the first few days following travel, being active, and eating meals and sleeping at appropriate times in the new time zone.

For most people, jet lag symptoms are temporary and the overall experience of jet lag is an occasional minor inconvenience, especially when considered in relation to the excitement of the new destination. Symptoms can sometimes be severe and limit what we do, but there are usually very few symptoms by the third day after the flight. However, individuals who routinely travel back and forth across multiple time zones (e.g. flight crew, diplomats, business executives) may experience chronic, longer-term sleep disturbances, daytime performance impairments and other symptoms similar to those experienced by shift workers (AASM, 2014a).

**Travelling west is best**

Jet lag is less disruptive when we travel in a westerly direction. Travelling west is best because when we travel in a westerly direction we follow the apparent pathway of the sun. This results in less of a mismatch between our biological clock and the day-night cycle of the external environment. For example, London time is 10 hours behind Melbourne time. If you left Melbourne at 8 am to fly to London, therefore, despite the flying time being approximately 22 hours, you would arrive in London at 8 pm on the same day (London time).

While most people would experience jet lag after a flight of this nature, the effects are less in the case of westerly travel than on a return trip because the creation of a longer day is more in tune with the inclination of the body’s biological clock to extend the day. When flying east, we travel in the opposite direction to the sun's apparent movement, so the day becomes ‘shortened’. This runs counter to the natural tendency of our biological clock. The result is a greater mismatch between the internal and external rhythms and so the effects of jet lag are heightened.

On the return flight from London to Melbourne, travel is in an easterly direction. If your departure time from London is 8 am, the 22 hour flight and 10 hour time difference will mean that you arrive in Melbourne at 4 pm the next day (Melbourne time, which is 6 am London time). On your arrival in Melbourne your biological clock will be functioning on London time (i.e. 6 am) while the actual time is 4 pm, which results in a disruptive mismatch between internal and external rhythms.

Studies of jet lag have led researchers to identify two types of interruptions to circadian rhythms. Easterly travel, which shortens the sleep–wake cycle, is called *phase-advance* and this runs counter...
to the cycle’s natural tendency to drift towards 25 hours and lengthen the day. Travel in a westerly direction results in phase-delay when the day is lengthened in accordance with our body's natural tendency towards a 25-hour day. Phase-advance requires more adjustment by the traveller than phase-delay.

Until the sleep–wake biological clock is reset and in sync with the environment, the individual is likely to continue to feel the effects of jet lag. This may create problems if, for example, soon after arrival, an individual has to perform a task that requires concentration, such as attending an important meeting or playing in a golf or tennis tournament.

**Overcoming jet lag**

Resetting the biological clock for the sleep–wake cycle to the destination time as quickly as possible is the key to overcoming jet lag and minimising its effects. To begin the adjustment process to a ‘new’ time, travellers should start to change their eating, sleeping and other behaviour patterns to accord with the ‘destination time’ routines. While in transit, environmental cues can be manufactured to be in harmony with the destination time, minimising the effects of jet lag on arrival. For example, upon boarding the plane, changing watches to the destination time zone is a good starter. Depending on the destination time, turning on the light to simulate daytime or sitting in a window seat and opening the shades to take in more light may be appropriate. Trying to sleep in-sync with the likely sleep time at the destination may also help. Night-time could be simulated by wearing sunglasses or an eye mask. If on arrival it is daytime, spending time outside can also speed up the adjustment to the new time zone by helping the biological clock to reset.

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**LEARNING ACTIVITY 10.10**

**Review questions**

1. What is jet lag?
2. Explain why jet lag occurs with reference to the circadian sleep–wake cycle.
3. Explain whether jet lag is likely to occur under each of the following conditions:
   (a) one time zone (rather than multiple time zones)
   (b) slow travel (rather than rapid travel)
   (c) rapid travel in a north or south direction (rather than east or west).
4. Give three examples of circadian cycle disturbances associated with jet lag.
5. Why is travelling east worse for jet lag?
6. Give two examples of behaviours that can minimise jet lag on a Melbourne to London direct flight and explain why these could be effective.

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**FIGURE 10.22** Travelling west is best. Most people have a natural circadian day that is longer than 24 hours, so the effects of jet lag tend to be more noticeable and last longer after travelling east (which shortens the day) than after travelling west (which lengthens the day).

EFFECTS OF PARTIAL SLEEP DEPRIVATION

Research shows that inadequate sleep is a common problem in Australia. People in all age groups report not getting enough good quality sleep. The sleep experiences of adolescents and shift workers illustrate the nature and extent of this issue. Our ability to function and feel well while we are awake significantly depends on whether we are getting enough total sleep and enough of each type of sleep. It also depends on whether we are sleeping at a time when our body is prepared and ready to sleep.

There are no rules on how much sleep we need but it is clear that we each need a certain amount of total sleep at different ages or stages of development to help ensure that we function at our best and maintain good health. Our major sleep episode of the day needs to be of the correct quantity and quality and occur when we are ready for sleep. It is best that this episode occurs as one consolidated block. Fragmented sleep is not good quality sleep. We also need to have the correct amounts of the different sleep types to be considered normal sleep. This includes NREM and REM sleep. Normal sleep consists of the correct quantity and quality of both NREM and REM sleep. As humans we are capable of adapting in ways that enables us to survive on less sleep than we actually need. However, when we aren't getting enough sleep, it is important to consider how well our bodies and our minds are functioning and the impact on our quality of life (Blunden, 2008).

When we fail to get enough sleep, we experience sleep deprivation. Sleep deprivation is a general term used to describe a state caused by inadequate quantity or quality of sleep, either voluntarily or involuntarily. This means that sleep deprivation may occur because we choose to go without sleep, such as when we stay up all night with friends or to watch a sports match. It may also occur due to reasons outside our control, such as when we work a night shift roster, travel rapidly across multiple time zones or have a sleep disorder.

Sleep quantity refers to the amount of sleep. This can be measured objectively using time. Sleep quality refers to how well we feel we have slept. This primarily relies on subjective self-report measures. We tend to judge sleep quality on the basis of how rested or recovered we feel on waking and throughout the day, so psychologists often use the terms ‘restfulness’ and ‘restorative’ when describing sleep quality. Sleep quantity influences our perception of sleep quality. The number of interruptions or arousals (partial or full) during a sleep episode are also commonly considered when we judge sleep quality because these influence whether our sleep episode is consolidated or fragmented (Harvey, et al., 2008).

Researchers often distinguish between partial and total sleep deprivation. Partial sleep deprivation involves having less sleep (either quantity or quality) than what is normally required. This may occur periodically or persistently over the short-term or long-term. For example, someone may have too little sleep for one or more days, weeks, months and so on. Most sleep disorders are associated with partial sleep deprivation that occur routinely over a prolonged period. By contrast, total sleep deprivation involves not having any sleep at all over a short-term or long-term period. The person stays awake for one or more days or weeks. This usually takes place under extreme conditions, such as when people try to break records. The longest period of total sleep deprivation that is widely recognised is 18.7 days (see box 10.5).

Psychologists have researched both types of sleep deprivation, investigating the effects of partial and total deprivation across short and prolonged periods. In studying sleep deprivation, researchers investigate psychological and/or physiological effects. They may study inadequate sleep in relation to total sleep time, NREM sleep, REM sleep or some other feature of a sleep episode or the entire sleep–wake cycle. In many cases, they study sleep recovery patterns following sleep loss as this provides insights on sleep patterns, sleep functions and other aspects of sleep.

In this section we examine the effects of partial sleep deprivation on a person's affective, behavioural and cognitive functioning. These are often interrelated and overlap, so it is can be difficult to draw a neat line between sleep deprivation effects in relation to such broad categories of human functioning.

You have undoubtedly experienced partial sleep deprivation. It often results in a range of uncomfortable side effects. The severity and extent of the effects depend on a range of factors, including the amount of total sleep loss, the nature of the sleep loss, when sleep loss occurs, why it occurs, its frequency, the period of time over which the sleep deprivation occurs and the personal characteristics of the individual involved.

Generally, the effects of partial sleep deprivation tend to be minor and temporary when they occur occasionally or on a short-term basis. When the accrued sleep debt is repaid, the person will quickly recover from the sleep loss effects. But with successive nights of inadequate sleep, the sleep debt can accumulate and sleep deprivation effects can multiply. Although we do not need to fully compensate for lost hours of sleep to recover from sleep deprivation effects, there is considerable research evidence that long-term sleep deprivation places the individual at a greater risk for a range of diseases and health problems, including obesity, diabetes and various cardiovascular diseases. It is also associated with an increased risk of accident and injury in people of all age groups.

There are many reasons why a person may not get enough sleep or experience poor sleep quality at any given time. However, the most common causes of partial sleep deprivation (without the presence of a sleep disorder) are lifestyle factors, including school or work-related factors. Consequently, most people are affected.
Sleep-disrupting lifestyle factors, if not changed, can also lead to the development of a sleep disorder. For example, habitually staying up late or drinking caffeinated beverages before the major sleep episode can cause sleep-onset insomnia. Sleep deprivation may not only trigger a sleep disorder, it can also be the consequence of having a sleep disorder. For example, consider the loss of sleep quantity and quality associated with sleep apnoea.

**Figure 10.23** Many parents of young infants experience partial sleep deprivation involving loss of both sleep quantity and quality.

**Affective functioning**

Many people tend to be easily irritated or short-tempered after they awaken from poor sleep, which you may know through personal experience. The link between sleep deprivation and mood change has been long-established by psychological research. It has been observed repeatedly by researchers among all sorts of participants under numerous sleep deprivation conditions.

Psychologists have also investigated links between sleep deprivation and other aspects of affective (emotional) functioning. Many have found that sleep deprivation can interfere with emotion regulation and reactivity. In particular, there is a strong link between inadequate sleep and our ability to control our emotions, often resulting in amplified emotional responses. Our emotional reactions may be too quick and more intense or exaggerated, often out of all proportion to how we would ordinarily react when not sleep deprived.

Sleep loss seems to compromise our brain’s ability to process emotional information, make accurate emotional perceptions and then regulate how we respond emotionally. We can find it harder to accurately judge other people’s emotions and reactions, making us more prone to unwarranted emotional outbursts. For example, some studies have found a strong link between sleep deprivation and impaired facial recognition of emotions and between sleep deprivation and reduced emotional empathy. Both can impact on our ability to identify and appreciate the emotional state of others, which are important aspects of our emotional decision making and reactions in our everyday interactions with others (Guadagni, et al., 2014; van der Helm, Gujar & Walker, 2010).

When we haven’t slept well, our emotional response threshold can be lowered, increasing our emotional reactivity and making us more likely to overreact to relatively neutral events. Sleep loss can also have a detrimental effect on our ability to sort out the unimportant from the important, and this can lead to poor judgments in relation to our emotional responses. We may overreact emotionally to trivial matters when there is actually no need to react. We may feel provoked or emotionally explode when no provocation actually exists. We may find it harder to control impulses. For example, some studies have found that sleep loss is associated with becoming aggressive more quickly than usual and with the outward expression of aggressive impulses. We are more likely to quarrel with other people and get frustrated and overreact in traffic jams. Even a single night of inadequate sleep can have these effects (Goldstein & Walker, 2014; Guadagni, et al., 2014; Gujar, et al., 2011; Kamphuis, et al., 2012).

NREM and REM sleep seem to play different roles in emotion regulation. For example, research findings suggest that emotional reactivity is more likely to occur with REM sleep deprivation (Rosales-Lagarde, et al., 2012). However, the exact neural processes that account for the link between sleep and emotion regulation remain unclear. They share the complex set of brain structures called the limbic system (which includes the amygdala), so this area has been a target of research interest (Gruber, et al., 2014; Talbot, et al., 2010; Yoo, et al., 2007).

**Figure 10.24** Sleep loss is associated with becoming aggressive more quickly than usual and with the outward expression of aggressive impulses. We are more likely to quarrel with other people and get frustrated and overreact in traffic jams.
Sleep deprivation also directly influences many aspects of our behaviour. One of the immediate effects on our performance can be **sleep inertia** — the performance impairment that occurs immediately after awakening. This is a sleep-to-wake transition effect that can follow a poor night's sleep, especially if abruptly awoken during slow wave deep sleep or when sleep duration is insufficient (Bruck & Pisani, 1999).

With sleep inertia, the individual typically feels groggy, partly awake and disoriented as they transition toward full alertness. Sometimes described as a 'state of grogginess', sleep inertia is strongest at wake time, but dissipates, or decays, rapidly thereafter. It usually lasts for a few minutes but can last for much longer (Santhi, et al., 2013).

Sleep inertia can interfere with the ability to perform a wide range of behavioural and cognitive tasks, including the simplest of everyday actions. Overall, our reaction time tends to be slow and we tend to perform below our best until we reach full alertness and recover from the inertia effects. Motor and cognitive functions in particular are not at their full capacity during sleep inertia, so performing tasks that require full alertness but can compromise the safety of the individual involved and others need to be avoided. For example, road traffic and on-the-job accidents can occur during sleep inertia.

Awakening during the deep sleep of NREM stages 3 and 4 produces more sleep inertia than awakening in stage 1 or 2. Waking up during REM sleep produces sleep inertia more like awakening from deep sleep than light sleep stages. Sleep inertia may also be experienced after a short nap. In addition, it tends to last longer when a person has been sleep deprived, as compared to no deprivation (Bruck & Pisani, 1999; Santhi, et al., 2013).

The primary behavioural effect of sleep deprivation is **excessive sleepiness** during normal waking time. Excessive sleepiness most commonly occurs during the day, but it may be present at night in a person, such as the shift worker who has their major sleep episode during the day. As well as affecting our mood, emotions and emotional reactivity, excessive sleepiness involves difficulty in maintaining an alert awake state. Fatigue is a common symptom. There is a persistent feeling of tiredness and lack of energy. Like sleep inertia, fatigue contributes to drowsiness, difficulty maintaining concentration and reduced awareness on the environment. It reduces our efficiency and we tend to take longer to finish tasks, have slower than normal reaction times and make more mistakes. These can have significant negative effects on performance of our daily activities, especially those requiring vigilance or sustained attention. Slower reaction time in particular is a significant impairment when driving or doing other tasks that require a quick response. You don't need to fall asleep at the wheel to be a danger. And slower reactions can affect people in all types of situations.

Sometimes lack of sleep or excessive sleepiness may result in unintended, involuntary lapses into sleep called microsleeps. A **microsleep** is a brief period of sleep, lasting up to a few seconds. During a microsleep the person typically has a fixed gaze, a blank expression on their face and doesn't blink. They may remain sitting or standing and they become less responsive to external stimuli. After a microsleep, which may last between 1–10 seconds, the person may have no recollection of what happened during their microsleep. They won't remember going into the microsleep, but may be aware of a lapse in concentration when they wake up (Bruck, 2006).

Microsleeps can affect how you function. For example, if you're listening to the teacher explaining something in class, you might miss some of the information or feel like you don't understand the point on your return to normal waking consciousness. In reality, though, you may have slept through part of the lesson and not been aware of it.

Research studies have identified many other aspects of behaviour functioning associated with partial sleep deprivation. These include:

- impaired regulation or control of behaviour
  - e.g. behaviour problems at home; naughtiness and disruptive behaviour at school; risk-taking behaviour by adolescents
- higher teacher rated inattentiveness by students in class
- poorer teacher rated social functioning by school children
- school lateness and absenteeism
- lower participation rate in extracurricular activities at school
- higher injury rates and injury prone behaviours in preschool age and school age children
- reduced motor coordination, particularly eye–hand coordination
- reduced speed and accuracy.
Cognitive functioning

Research studies have long established that sleep deprivation may impair cognitive functioning. This has been found in relation to a wide range of abilities of varying complexity, many of which are also involved in our affective and behavioural functioning.

It is clear that even a relatively small amount of sleep deprivation can adversely affect attention. In particular, excessive sleepiness due to sleep deprivation tends to reduce alertness and our ability to stay focused on a task. With prolonged sleep deprivation, we tend to experience lapses in selective attention and reduced ability to divide our attention on tasks that require simultaneous attention to multiple sources of information. These skills are required for the performance of many everyday tasks such as driving a motor vehicle or cooking the family dinner, as well as numerous jobs in the workplace. Tasks often begin well, but performance tends to deteriorate as task duration increases (Goel, et al., 2009; Jackson, et al., 2011).

The greater the sleep deprivation, the more likely it is that attention will be impaired and that errors associated with loss of attention will increase. This is even more likely when a task lacks interest or complexity. For example, when sleep deprived research participants are required to complete simple, monotonous, repetitive tasks, such as identifying bleeps and flashing lights on a computer monitor, they will inevitably make a significantly higher number of errors than when they had not been deprived of sleep. In the real world, these types of errors can occur in visual tasks similar to those involved in reading x-rays, CAT scans, baggage screening and even air traffic control. Errors in these types of contexts can have devastating consequences.

As described in chapter 8, one full night's of sleep deprivation is similar to having a blood alcohol content of 0.05., which is one of the reasons sleep deprivation is an issue of public concern.

When sleep deprived, our ability to think clearly tends to reduce, especially for tasks that require more complex thought (such as when solving maths problems). We are also more likely to think in irrational ways, and have difficulty making decisions and solving problems that require creative thinking. There is a tendency to need more time to analyse situations and respond physically to events as they happen. We tend to lose situational awareness and it is easier to overlook important details. In children, sleep deprivation has been found to reduce verbal creativity and the ability to think abstractly. The ability to do tasks that need visual and spatial abilities (such as working with different patterns or maps) or work involving eye-hand coordination, such as drawing and writing, may also be affected. Adults experience similar impairments (Bruck, 2006; Goel, et al., 2009; Gruber, et al., 2014).

There is considerable research evidence that sleep deprivation may impair various learning and memory processes. Generally, sleep-deprived participants tend to perform worse on learning and memory tasks, compared with well-rested individuals, especially when sleep deprivation is prolonged. For example, reduced attention can adversely impact on acquisition of new information during learning. Similarly, processing information in short-term working memory can be significantly impaired, making it difficult to keep details in conscious awareness for use when required (Goel, et al., 2009; Gruber, et al., 2014).

The deterioration in cognitive functioning from prolonged partial sleep deprivation has further implications for shift workers in jobs with significant responsibility for the health and wellbeing of others. For example, medical staff in the emergency department of a hospital cannot afford to miss any changes in vital signs and must be able to think clearly and make decisions quickly if a patient's condition changes. Likewise, it is critical that an air-traffic controller, who must continually scan a monitor for small but significant changes in aircraft position, doesn't miss important information. In these types of situations, errors in judgment, as well as wrong decisions and lack of clear, logical thinking, may result in loss of human life.

**FIGURE 10.26** For many shift workers, including doctors in hospital emergency departments who have to grab opportunities to sleep whenever they can, prolonged sleep deprivation can impact on their ability to think clearly and make decisions.
**BOX 10.5**

**Total sleep deprivation**

Studies on the effects of prolonged total sleep deprivation in humans have tended to rely on convenience samples. Case studies of people who performed sleep deprivation stunts while monitored by psychologists or doctors are among the better-known. These have mainly involved individuals who have deprived themselves of sleep for 10 or more consecutive days. In all cases, there were no long-lasting effects, either psychologically or physiologically. Most observed and self-reported effects of prolonged total sleep deprivation were temporary and disappeared after the individual slept uninterrupted and their sleep–wake cycle returned to normal.

One of the best-known sleep deprivation stunts is that of 17-year-old American Randy Gardner. In 1964 Gardner stayed awake for a world record 264 consecutive hours (11 days and 11 nights) as part of a high school science project. Unlike the previous world record holder, Gardner did not use stimulants to help stay awake. There was a gradual onset of various impairments as the sleep loss period progressed. Overall, he became irritable and had difficulty concentrating, thinking clearly and remembering things. By the fourth day he was experiencing hallucinations and delusions. For example, he saw fog that wasn’t present, believed a street sign to be a person and imagined himself to be a famous football player. By the ninth day his thinking became fragmented, his speech was slurred and he often did not finish sentences. He was generally unsmiling and expressionless. His vision was blurred and his right eye was making involuntary sidewise movements, which caused him considerable bother.

Although Gardner experienced a range of debilitating effects in the sleep deprivation period, there were no significant lasting effects. For the first three days after the stunt, Gardner slept longer than his usual 8 hours (15 hours on the first night, 12 hours the second night and 10.5 hours the third night). His first night of sleep was predominantly slow wave and REM sleep. During the day, he continued his usual activities without difficulty. In all, it took Gardner about 3 days to resume his normal sleep–wake cycle. Follow-up tests 10 days after the stunt confirmed that Gardner had suffered no long-term harmful effects. However, it was apparent that he withstood prolonged total sleep deprivation better than others. This has been partly attributed to his younger age, not taking stimulants and the home setting in which the stunt was conducted (Dement, 1976).

In 1977, English woman Maureen Weston went without sleep for 18.7 days during a rocking chair marathon. This is recognised as the world record (Guiness) for the longest period without sleep. Weston is reported as having experienced hallucinations, paranoia, blurred vision, slurred speech and memory and concentration lapses, but no lasting effects. It is unclear whether she used stimulants.

Studies show that once a sleep-deprived person is able to catch up on a big chunk of the lost sleep and reset their sleep–wake cycle and other biological rhythms, the physiological and psychological effects tend to disappear.

Some psychologists explain the finding that there are usually few lasting effects of sleep deprivation as being due to the difficulty in ensuring that participants in sleep deprivation studies are, in fact, completely sleep-deprived. Total sleep deprivation is difficult to ensure because after a period of prolonged sleeplessness, people automatically drift into periods of microsleep over which they have no control.

**FIGURE 10.27** Randy Gardner (seated) with two classmates helping keep him awake for a medical check-up at a hospital during the sleep deprivation stunt.
**LEARNING ACTIVITY 10.11**

**Review questions**

1. Explain the meaning of the term sleep deprivation with reference to sleep quantity and quality.

2. (a) Explain the meaning of ‘sleep quantity and quality’ with reference to key characteristics.
   (b) How are sleep quantity and sleep quality commonly measured?
   (c) What is a possible objective measure of sleep quality?

3. How are partial and total sleep deprivation defined?

4. (a) What is sleep inertia and when is it more likely to occur?
   (b) Explain whether a person is asleep or awake when experiencing sleep inertia.

5. (a) What is a microsleep and when is it more likely to occur?
   (b) Explain whether a microsleep is a mini-version of a major sleep episode.

6. (a) Prepare a table or Venn diagram in which you summarise some of the possible effects of partial sleep deprivation using the headers affective, behavioural and cognitive.
   (b) Twins Sara and Adam partied for two consecutive nights on the weekend of their 18th birthday and had almost no sleep throughout this period. On the morning after the last party, both attended their casual jobs. Sara is employed as a lifeguard at the local pool. Her duties include closely monitoring activity in the pool to ensure swimmers’ safety, responding quickly to unsafe situations and dealing calmly with swimmers behaving in an unsafe manner. Adam works at a Tattslotto agency. His duties include scanning Tattslotto tickets, identifying when to pay out on winning tickets from beeps on the computer and explaining to customers the processes involved in filling out their tickets.
   (i) Describe the possible impact for Sara and Adam of their sleep deprivation on their respective abilities to do the tasks required in their casual jobs.
   (ii) When Sara and Adam had their first major sleep episode, what were the likely pattern and proportions of NREM and REM sleep?

7. (a) How quickly and how well do people usually recover from the effects of partial sleep deprivation?
   (b) What key factor(s) would influence(s) recovery from sleep deprivation?

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**LEARNING ACTIVITY 10.12**

**Reflection**

What do sleep deprivation effects suggest about the purpose and function of sleep?

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**LEARNING ACTIVITY 10.13**

**Experimental design on sleep deprivation**

Describe an experiment that could be conducted to compare the effects of partial sleep deprivation on performance of simple and complex tasks.

- Present your experimental design as a flowchart showing the key features: aim, ethical issues, research hypothesis, operationalised independent and dependent variables, sampling procedure, experimental groups, participant allocation procedure.
- If your hypothesis were supported, what results might be obtained, what conclusion(s) could be drawn and what generalisation(s) could be made? Ensure that your results are consistent with theoretical expectations.
- Explain your choice of experimental design and identify two potential extraneous or confounding variables that are taken account of in your design.

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**INTERVENTIONS TO TREAT SLEEP DISORDERS**

Most sleep disorders can be treated successfully and a number of effective interventions are available. Making some lifestyle changes can help relieve mild or occasional symptoms, but professional support may be required if symptoms persist and are causing unwanted distress or problems in everyday functioning. There are a range of treatment options, with the choice depending on the disorder, its symptoms and severity, the potential risks and benefits of treatment options, and the individual involved.

In this section, we examine the use of cognitive behavioural therapy to treat insomnia and the use of bright light therapy for circadian rhythm phase disorders.

**Cognitive behavioural therapy**

As the name suggests, cognitive behavioural therapy, commonly referred to as CBT, is a type of psychotherapy that combines cognitive and behavioural therapies to treat mental health problems and disorders. It has also been successfully applied to the treatment of a range of sleep disorders, especially those for which lifestyle factors and inappropriate sleep habits significantly contribute to the onset or experience of symptoms.

A core assumption of CBT is that the way people feel and behave is largely a product of the way they think. Unlike other types of ‘talking’ therapies, CBT does not involve talking freely about whatever comes to mind or dwelling on events in a person’s past to
gain an insight into their psychological state. It is not a ‘lie on the couch’ type of therapy.

CBT provides a structured program that tends to be relatively short-term and focused on the ‘here and now’ — how a person’s current thoughts, feelings and behaviours are presently affecting them. Although CBT recognises that events in a person’s past have shaped the way they currently think and behave, this is not the focus. CBT aims to find solutions on how to change a person’s current thoughts and behaviours so that they can function better now and in the future.

Many people with insomnia develop sleep-related thoughts and behaviours that have the unintended consequence of maintaining or worsening their sleep problems. These include, for example, misunderstandings about the causes of insomnia, faulty beliefs about sleep-promoting practices, amplifications of the consequences of insomnia or poor sleep and unrealistic sleep expectations. Often, their behaviours, such as spending a lot more time in bed, sleeping in and daytime napping, begin as strategies to combat the sleep problem by getting more sleep. Their inappropriate thoughts and behaviours can also lead to excessive worry or anxiety about not sleeping, apprehensions about daytime impairments, fear of sleeplessness, heightened physical arousal when trying to sleep, a mental hyperarousal state (‘a racing mind’), and constant clock watching when in bed, all of which are counterproductive, especially if they become conditioned with sleep time or the normal sleep environment (APA, 2013; Schutte-Rodin, et al., 2008).

According to cognitive behavioural therapy for insomnia, often called CBT-I, identifying and changing these negative thoughts and inappropriate behaviours about insomnia and sleep with more balanced and realistic ones is very important to alleviating symptoms or overcoming the disorder. Consequently, the goal of CBT-I is to help individuals with insomnia identify and replace thoughts and behaviours that cause or worsen their insomnia with thoughts and behaviours that minimise the likelihood of their insomnia and promote good sleep.

Overall, a CBT-I program consists of about 8–10 sessions. An important starting point in making effective changes through CBT is for the individual to understand the difference between thoughts, feelings and behaviour, their interrelationship and their relevance to sleep and insomnia. A CBT-I program may therefore include an introductory sleep education session during which the individual is assisted to understand the sleep–wake cycle, insomnia and factors that can cause and maintain insomnia. The individual may also be encouraged to complete a sleep diary as they learn and apply the various CBT strategies. This allows improvement to be monitored and assessed.

**Cognitive component**

The cognitive part of CBT-I assists the individual to recognise and change inappropriate or dysfunctional attitudes, beliefs and other thoughts about their sleep. This includes addressing anxiety or preoccupation with sleep difficulty and learning how to control or eliminate worries and negative thoughts that prevent sleep onset. After identifying faulty or dysfunctional thoughts, alternative interpretations of what is making the person anxious or causing concern about sleep may be offered so that they are able to think about their insomnia in a different way. For example, they may be encouraged to develop and maintain realistic expectations of their sleep, not blame insomnia for all their daytime impairments, not believe that losing a night’s sleep will bring awful consequences, not give too much importance to sleep and to develop some tolerance to the effects of lost sleep (APA, 2016b).

An example of how thoughts can influence sleep onset is as follows:

**Fact:** ‘I’m not feeling very sleepy right now.’

**Thoughts:** ‘It’s already 1.30 a.m. I’m never going to fall asleep. Everybody else is sleeping. I’m no good at sleeping. I don’t know how I’m going to cope at work.’

**Emotions:** Fear of sleeplessness and helplessness

**Consequence:** Inadequate sleep quantity and quality.

In this example, the individual has thought about the fact that they are not very sleepy right now in a faulty way — by generalising that they will never get any sleep at all. The reality is that people with insomnia do sleep for some time most nights, but
tend to underestimate their total sleep time afterward. Therefore, a thought such as ‘I’m never going to fall asleep’ is highly likely to be false. The individual places more emphasis on this thought than the fact itself. Their thinking also triggers inappropriate emotional reactions that fuel their faulty thoughts, further affecting their ability to sleep.

‘Through the cognitive component of CBT-I, the individual would be encouraged to consider an alternative way of thinking about their sleep situation; for example:

**Fact**: ‘I’m not feeling very sleepy right now.’

**Thoughts**: ‘I’m not sleepy now, but I usually get some sleep during the night. I will eventually feel sleepy. I always make it through the next day without any disaster’ or ‘It doesn’t matter whether or not I fall asleep. Rest is still good for me — it does not have to be sleep. I can function well with little sleep. I will relax and not worry about it. I will fall asleep when my body is ready.’

**Consequence**: Less anxiety, less aroused/more relaxed, positive thinking and therefore increased likelihood of sleep.

In appraising the situation more accurately, realistically and positively rather than negatively, the individual does not place undue pressure on themselves to fall asleep, is less psychologically and/physically aroused, and is less likely to behave in ways that impair sleep onset or return to sleep after a premature awakening (Mental Health Foundation, 2011).

**Stimulus control therapy**

Stimulus control therapy for insomnia was developed by American psychologist Richard Bootzin in 1972 to overcome the learned associations acquired by people with insomnia and to lead them to form new associations that suit sleep. It has since been modified by other psychologists or researchers but the basic approach is still the same.

According to Bootzin, people with insomnia often spend long periods of time in bed trying to fall asleep or get back to sleep. This can result in their bed and bedroom becoming associated with behaviours that are incompatible with sleep, such as watching television, texting, eating, reviewing the day’s events, planning, worrying, lying awake, and becoming anxious, stressed or frustrated from trying to fall asleep or get back to sleep. Their bed and bedroom may also become conditioned stimuli for anxiety, stress or frustration associated with being unable to fall asleep. The more time they spend in bed trying to sleep or engaging in activities that don’t suit sleep, the stronger the learned associations become, which perpetuates their difficulty in falling asleep (Bootzin & Epstein, 2011; Bootzin & Perlis, 2011).

![Figure 10.29](image)

**Figure 10.29** ‘I’m never going to fall asleep.’ The cognitive component of CBT-I directly targets attitudes, beliefs and thoughts that interfere with sleep onset.

**Behavioural component**

The behavioural part of CBT-I helps the individual develop good sleep habits and avoid behaviours that prevent them from sleeping well. Two of the most effective behavioural therapy techniques for insomnia are called stimulus control therapy and sleep hygiene education.

![Figure 10.30](image)

**Figure 10.30** ‘Clock-watching’ is one behaviour that can maintain sleep-onset insomnia and make it even harder to fall asleep.

**TABLE 10.1**

<table>
<thead>
<tr>
<th>Technique</th>
<th>Description</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Stimulus control therapy</strong></td>
<td>Helps individuals develop good sleep habits and avoid behaviours that prevent them from sleeping well.</td>
</tr>
<tr>
<td><strong>Sleep hygiene education</strong></td>
<td>Teaches individuals how to create a sleep-friendly environment and establish healthy sleep practices.</td>
</tr>
</tbody>
</table>
The aims of stimulus control therapy are to strengthen the bed and bedroom as cues ('stimuli') for sleep, to weaken them as cues for behaviours that are incompatible with sleep, and to establish a regular sleep-wake schedule that is consistent with the circadian sleep-wake cycle. It essentially involves bedroom behaviours and practices designed to re-establish an association between sleep and the bed and bedroom.

These are apparent in the stimulus control instructions given to people with insomnia in order to address the following points: when to go to sleep; what activities are permitted or disallowed when in bed; what to do if sleep is not attained within a reasonable period of time; when to rise in the morning; and daytime napping. It is also necessary to caution individuals not to ‘clock-watch’.

The stimulus control instructions and their underlying rationale are:

1. Determine an appropriate time to go to bed based on feelings of sleepiness rather than what time it is. Only lie down and go to sleep when you are sleepy. Bear in mind being sleepy is not the same thing as being tired. (This instruction helps prevent lying in bed engaging in negative sleep-related thoughts.)

2. Do not use your bed for anything else except sleep; that is, do not read, use a computer, send text messages, check email, talk on the phone, watch television, eat or worry in bed. (Reserving the bed for sleep helps to establish new sleep habits, whereas engaging in activities performed when awake result in arousal, making it difficult to sleep. It is also important for the bed and bedroom to be conditioned stimuli for sleep, not arousal or wakefulness.)

3. If you find yourself unable to fall asleep, get up, and go into another room and engage in a relaxing activity such as some light reading or using a relaxation technique until you do feel drowsy. Stay up as long as you wish and then return to the bedroom to sleep. (Getting out of bed if not sleepy strengthens the association between the bed and bedroom and falling asleep. Getting out of bed when unable to sleep also helps develop a perception of control over insomnia.)

4. If you still cannot fall asleep within 10 minutes, repeat step 3. Do this as often as is necessary throughout the night, but do not clock watch. Repeat this step as many times as you need to during the night. (Clock watching is an action that reinforces wakefulness.)

5. Set an alarm and get up at the same time every morning (even on weekends), irrespective of how much sleep you got during the night. (Irregular sleep-wake times disrupt the circadian sleep-wake cycle.)

6. Do not nap during the day. (Not napping minimises disruption to the sleep-wake cycle while helping ensure you fall asleep more easily because you are more tired. More easily falling asleep is also a reinforcer, which helps maintain compliance with the SCT instructions and further strengthens the association between the bed and bedroom and falling asleep.)
Stimulus control therapy and conditioning principles

Stimulus control therapy originated from an analysis of sleep using operational and classical conditioning principles.

From an operant conditioning perspective, sleep is viewed as an instrumental act (i.e., an operant) intended to produce reinforcement (i.e., sleep). Stimuli associated with sleep become discriminative stimuli (antecedents) for the occurrence of reinforcement. Difficulty in falling asleep, or in returning to sleep after awakening, may be due to inadequate stimulus control.

The operant conditioning goals of stimulus control are to strengthen sleep-compatible associations with the bed and bedroom environment and to remove sleep incompatible ones; the classical conditioning goals are to break the association between the bedroom and insomnia.

The stimulus control instructions decrease the bed and bedroom as cues for arousal and re-establish the bed and bedroom as strong cues for sleep. They additionally promote a more regular circadian sleep–wake cycle (Sharma & Andrade, 2012).

Sleep hygiene education

Sleep hygiene education is often used in conjunction with stimulus control therapy to assist the person with insomnia to change their sleep-related activities. Sleep hygiene education involves providing information about practices that tend to improve and maintain good sleep and full daytime alertness. The term sleep hygiene is often used interchangeably with sleep habits because it involves changing basic lifestyle habits that influence sleep onset, good quality sleep and alertness during the normal waking period (NSF, 2016).

Like all types of hygiene, the sleep hygiene practices used by an individual can be appropriate and support good sleep or inappropriate and inhibit good sleep. Inappropriate practices may include irregular sleep onset and wake times, stimulating and alerting activities before bedtime, and consuming stimulants too close to sleep time. These do not necessarily cause sleep disturbance in all people. For example, an irregular bedtime or wake time that produces insomnia in one person may not be important in another. However, there is considerable research evidence that certain practices tend to be highly effective in the treatment of insomnia and helping people with insomnia to establish and maintain a regular sleep–wake pattern seven days a week.

Good sleep hygiene practices include the following:

- Establish a regular relaxing sleep schedule and bedtime routine. Maintain a regular sleep–wake schedule, particularly a regular wake-up time in the morning. Waking up late during weekends or days when you are off school or work can also disrupt the sleep–wake cycle. It is not possible to do the same thing every day, but it should be most days. Try to avoid emotionally upsetting conversations and activities before trying to sleep. Don't dwell on, or bring worries, concerns or problems to bed.
- Associate your bed and bedroom with sleep. It's not a good idea to use your bed to watch TV, make phone calls, listen to the radio, read or study. Such activities can not only inhibit sleep onset, but weaken the sleep and bed/bedroom association.
- When you cannot sleep get up.
- Avoid napping during the normal waking period. It can disrupt the sleep–wake cycle, especially if they are longer than 20 to 30 minutes or occur close to the major sleep episode.
- Avoid stimulants such as caffeine, nicotine and alcohol too close to bedtime. While alcohol can speed up sleep onset, it can disrupt the second half of the sleep episode as the body begins to metabolise the alcohol, thereby causing arousal and making it harder to stay asleep. Other stimulants can delay sleep onset.
- Avoid activities that are stimulating in the hour before bed. This includes vigorous exercise, video games, television, movies and important discussions as they can be arousing and some inhibit melatonin (light emission).
- Exercise can promote good sleep. Exercise regularly for at least 20 minutes during the day, preferably more than four to five hours prior to bedtime if vigorous. A strenuous workout just before you go...
to bed will keep you awake for longer than normal because it creates arousal and should therefore be avoided. A relaxation technique such as progressive muscle relaxation or a relaxation exercise like yoga can be used before bed to help initiate sleep onset.

- **Food can be disruptive just before sleep.** Your digestive system also follows a biological rhythm. It is ready to digest food during the day, but not at night-time. Stay away from large meals close to bedtime. Although it is important to not be hungry at bedtime, having a full stomach makes it difficult to sleep. The evening meal should be at least 2 hours before bedtime. Some people find that having a small snack at bedtime helps them to sleep better. Avoid foods that require more digestion time, such as red meat, raw vegetables, spicy foods and most take-away foods. Easy-to-digest foods include fish, poultry, cooked vegetables, soup and yoghurt.

- **Ensure adequate exposure to natural light.** Exposure to natural light helps maintain a normal sleep–wake cycle. (Bonnet & Arand, 2016; NSF, 2016j; SHFj, 2016)

**Bright light therapy**

Interventions to treat circadian rhythm phase disorders aim to re-set the biological clock regulating a person’s sleep–wake cycle to align it with the sleep–wake schedule they desire or require. For example, the more ongoing sleep–wake cycle shift in adolescence or due to shift work can cause natural sleep time to overlap with waking time activity such as school or work, which can impact on alertness, attention, reaction time and various other aspects of functioning. Given that light exposure can cause our biological clock to advance or delay, thereby affecting the phase (‘timing’) of our sleep–wake cycle, light can be used to re-set the biological clock and gradually shift someone’s circadian sleep–wake cycle to a more appropriate or conventional schedule.

**Bright light therapy**, also called phototherapy, involves timed exposure of the eyes to intense but safe amounts of light. When used for circadian rhythm phase disorders, the aim is to shift an individual’s sleep–wake cycle to a desired schedule, typically the day–night cycle of their physical environment. The light may be sunlight or artificial. In many places, sunlight is not available at the right intensity at the required time for the right amount of time to be used for therapeutic purposes. Artificial light is therefore used as an alternative as it can affect the biological clock in the same way that sunlight does.

Various types of lamps, visors and other devices have been devised for use in bright light therapy. A light box is the most commonly used device. The box houses fluorescent tubes that produce light of variable intensity. As shown in figure 10.34, it sits on top of a table or desk and is portable. During a treatment session, which is usually self-administered at home, the individual has to keep within a certain distance of the box, usually about 30 centimetres from it. Generally, the light that is emitted is brighter than indoor light but not as bright as direct sunlight. There is no need to look directly into the light. Instead, the person may simply face in the direction of the box. It is therefore possible to do activities such as texting, reading, gaming or even eating during a light exposure session. The light will be reflected from surfaces and received by the eyes for transmission to the SCN, which will then influence melatonin secretion from the pineal gland.

Bright light therapy requires a number of sessions across a number of days until the body adjusts to the new times. Exposure sessions can last from 15 minutes to two hours, once or twice a day, depending on the disorder, the required phase shift, the light intensity used, the equipment and the individual. Generally, the three important variables are to use the light at the right time of day at the right intensity for the right amount of time. The timing of the light exposure in particular is critical. There is a peak or optimal time for light exposure and the closer to the time an individual is exposed to light, the more effective the treatment is likely to be. The peak time can be determined by core body temperature.
Circadian rhythm phase disorders for which bright light therapy may be used include the following.

**Delayed sleep phase disorder:** This causes people to feel sleepier much later at night than is desired and experience later sleep onset, as occurs with the sleep–wake cycle shift during adolescence. As a result, their waking time also shifts to later in the morning. This sleep pattern can interfere with their schedule of activities for the day. To correct delayed sleep phase, light exposure generally takes place during the early morning hours (e.g. between 6–8 am) to help advance the circadian rhythm to an earlier time (i.e. shift the phase forward) so that the person will be sleepier earlier and wake up earlier.

**Advanced sleep phase disorder:** This causes people to feel sleepier much earlier at night than is normal, resulting in symptoms of sleepiness much earlier than desired, an early sleep onset and an awakening that is earlier than desired. Circadian rhythms of older people tend to be phase advanced when compared with those of younger people, but is generally considered to be a relatively rare disorder. To correct it, light exposure takes place early at night/ in the evening to help delay the circadian rhythm to a later time (i.e. shift the phase backward) so that the person will be sleepier later and wake up later.

**Shift work:** This occurs due to a work schedule, such as night shift, that takes place during the time when the body wants to sleep. This means that the person has to try to sleep when their body expects to be awake. In general, using light treatment in the evening can help someone who regularly works nights. In such cases, it is also best to avoid daylight between the end of the shift and sleep time. Dark sunglasses or special goggles can be worn to help. Correcting a shift work sleep disorder is particularly difficult because the required work schedules, days off and social activities can alter exposure to light from day to day. The instability of the sleep–wake cycle due to frequent changes in the sleep times makes it harder to re-set the biological clock.

**Jet lag:** Jet lag causes sleep problems and other symptoms when people rapidly cross many time zones on a flight. The timing of light exposure depends on the direction of travel and the number of time zones crossed. Generally, when travelling east, the sleep–wake cycle should be advanced, so light therapy in the morning at the destination may help after easterly travel. For travelling west, the sleep–wake cycle should be delayed, so bright light in the evening may help reduce jet lag.

Bright light therapy has been found to be effective for treating the various phase disorders, at least partially for many people. Many people have reported sufficient improvement that enables them to function reasonably well on their new schedule. It does not seem to produce any major side effects when used within the proper limits for intensity and time. Minor side effects may include eye irritation, headache, nausea and dryness of skin (AASM, 2014b; Dodson & Zee, 2010).

![Figure 10.34](image1.png) An example of a light box designed for bright light therapy. A suitable device must be capable of producing light intensity of at least 2500 lux, with 10,000 lux generally considered ‘bright light’ for therapeutic purposes (lux is a unit of illumination intensity as perceived by the human eye). Indoor evening room light is usually less than 100 lux and a brightly lit office is typically less than 500 lux. Outdoor light is much brighter. For example, a cloudy grey winter day is around 4000 lux and a sunny day can be 50,000 to 100,000 lux or more (Westrin & Lam, 2007).

![Figure 10.35](image2.png) Normal circadian sleep–wake cycle with a waking time of 7 am. The timing of light exposure is crucial to bright light therapy. Optimal times for different circadian rhythm phase shifts can be determined by core body temperature (another circadian rhythm tied to the sleep–wake cycle).

The sleep–wake cycle shift is gradually changed. For example, bright light exposure might occur for 45 minutes each day for a week at times that are scheduled to get progressively earlier or later depending on the direction of the desired phase shift. Regular sleep patterns help to keep the biological clock set at the new time.
LEARNING ACTIVITY 10.14

Review questions

1. (a) What is cognitive behavioural therapy?
   (b) How would a CBT therapist most likely view the cause(s) of insomnia and factors maintaining its symptoms?
   (c) List three examples of dysfunctional cognitions and three examples of inappropriate behaviours that may contribute to the development or maintenance of insomnia.
   (d) What would a CBT therapist prioritise for change when developing a treatment plan for insomnia?
   (e) How might a CBT therapist achieve cognitive change in someone with insomnia?
   (f) Name, describe and give relevant examples of two behavioural techniques that may be used in CBT for insomnia.
   (g) (i) Using classical conditioning principles, explain how someone’s bed and bedroom may become conditioned stimuli for anxiety associated with being unable to fall asleep. You may use a labelled diagram to illustrate the three phase process.
   (ii) Explain how operant conditioning can maintain or strengthen sleep compatible associations with the bed and bedroom environment.
   (h) Why is CBT a suitable intervention for insomnia but not for circadian rhythm phase disorders?
   (i) Explain how sleep hygiene education could be used as part of a treatment plan for the sleep–wake cycle shift in adolescence, with reference to three practices of specific relevance to the disorder.

2. (a) What is bright light therapy?
   (b) What three features of timing are crucial to its effective use?
   (c) How does bright light therapy influence circadian phase change?
   (d) Complete the following table to summarise use of bright light therapy as a therapeutic intervention for various circadian phase disorders.
   (e) Under what circumstances would bright light therapy be a suitable intervention for insomnia?

<table>
<thead>
<tr>
<th>Circadian rhythm phase disorder</th>
<th>Example</th>
<th>Features of desynchronised phase shift</th>
<th>Timing of light exposure to re-shift sleep–wake cycle</th>
</tr>
</thead>
<tbody>
<tr>
<td>Delayed sleep phase disorder</td>
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<tr>
<td>Advanced sleep phase disorder</td>
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<td>Shift work disorder</td>
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<tr>
<td>Time zone change syndrome</td>
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</table>

LEARNING ACTIVITY 10.15

Reflection

To what extent does CBT for insomnia reflect the biopsychosocial model?
CHAPTER 10 REVIEW

CHAPTER SUMMARY

SLEEP DISTURBANCES

Dyssomnias and parasomnias

Dyssomnias

Sleep-onset insomnia

Key symptoms

Effects on sleep–wake cycle

Narcolepsy

Key symptoms

Effects on sleep–wake cycle

Sleep–wake cycle shift in adolescence

Circadian rhythm phase disorders

Shift work

Travelling west is best

Jet lag

Overcoming jet lag

Affective functioning

Behavioural functioning

Cognitive functioning

Effects of partial sleep deprivation

Cognitive behavioural therapy

Cognitive component

Stimulus control therapy

Sleep hygiene education

Interventions to treat sleep disorders

Behavioural component

Bright light therapy

Sleep walking

Effects on sleep–wake cycle

Sleep apnoea

Effects on sleep–wake cycle

Sleep–wake cycle shift in adolescence

Circadian rhythm phase disorders

Shift work

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Jet lag

Overcoming jet lag

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Cognitive component

Stimulus control therapy

Sleep hygiene education

Dyssomnias

Sleep-onset insomnia

Key symptoms

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Narcolepsy

Key symptoms

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Sleep hygiene education

Interventions to treat sleep disorders

Behavioural component

Bright light therapy

Sleep walking

Effects on sleep–wake cycle

Sleep apnoea

Effects on sleep–wake cycle

Cognitive component

Stimulus control therapy

Sleep hygiene education
### KEY TERMS

- bright light therapy p. 00
- circadian rhythm phase disorders p. 00
- cognitive behavioural therapy p. 00
- dyssomnias p. 00
- insomnia p. 00
- jet lag p. 00
- microsleep p. 00
- narcolepsy p. 00
- parasomnias p. 00
- partial sleep deprivation p. 00
- sleep apnoea p. 00
- sleep debt p. 00
- sleep deprivation p. 00
- sleep disorder p. 00
- sleep disturbance p. 00
- sleep hygiene education p. 00
- sleep inertia p. 00
- sleep walking p. 00
- sleep-onset insomnia p. 00
- sleep–wake cycle shift p. 00
- stimulus control therapy p. 00
- total sleep deprivation p. 00

### LEARNING CHECKLIST

Complete the self-assessment checklist below, using ticks and crosses to indicate your understanding of this chapter’s key knowledge (a) before and (b) after you attempt the chapter test. Use the ‘Comments’ column to add notes about your understanding.

<table>
<thead>
<tr>
<th>Key knowledge I need to know about</th>
<th>Self-assessment of key knowledge I understand before chapter test</th>
<th>Self-assessment of key knowledge I need to do more work on after chapter test</th>
<th>Comments</th>
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CHAPTER TEST

SECTION A — Multiple-choice questions

Choose the response that is correct or that best answers the question.
A correct answer scores 1, an incorrect answer scores 0.
Marks will not be deducted for incorrect answers.
No marks will be given if more than one answer is completed for any question.

Question 1
Sleep walking is most likely to take place within _____ hours of falling asleep.
A. 1–2
B. 3–4
C. 5–5
D. 7–8

Question 2
Regularly occurring irresistible urges to sleep during normal waking time are most likely to be associated with
A. narcolepsy.
B. sleep apnoea.
C. insomnia.
D. circadian rhythm phase disorders.

Question 3
Impairments of daily functioning associated with partial sleep deprivation are best explained in terms of
A. proportions of REM and NREM sleep.
B. external cues in the environment.
C. biologically induced hormones.
D. accrued sleep debt.

Question 4
The most common behavioural effect of sleep deprivation is
A. sleeplessness.
B. restlessness.
C. excessive sleepiness.
D. emotional reactivity.

Question 5
Which sleep disorder is characterised by a cycle of activity involving cessation of breathing, a brief awakening and renewal of breathing?
A. sleep walking
B. insomnia
C. sleep apnoea
D. narcolepsy

Question 6
Which of the following is best described as a circadian rhythm phase disorder?
A. jet lag
B. insomnia
C. excessive sleepiness
D. excessive sleeplessness

Question 7
Hypnagogic hallucinations are most likely to occur during
A. sleep onset.
B. NREM sleep.
C. REM sleep.
D. transitioning from NREM to REM sleep.

Question 8
Which of the following would be best classified as a sleep disorder involving arousal from NREM sleep?
A. jet lag
B. sleep walking
C. excessive sleeping
D. sleep-onset insomnia

Question 9
A sleep disorder that is associated with inappropriate activation of the central and/or autonomic nervous systems during sleep is best described as a
A. dyssomnia.
B. parasomnia.
C. sleep disturbance.
D. circadian rhythm phase disorder.

Question 10
Which of the following characteristics is more likely to be observed during the major sleep episode of someone with narcolepsy rather than someone without narcolepsy?
A. little or no REM sleep
B. sleep is entered through NREM stage 4
C. first REM sleep period about 10 minutes following sleep onset
D. first REM sleep period at about 60–90 minutes of sleep onset

Question 11
Which of the following statements about sleep deprivation is correct?
A. Sleep deprivation has no psychological effects.
B. Sleep deprivation has lasting physiological effects.
C. Sleep deprivation affects performance on cognitive tasks.
D. The effects of sleep deprivation disappear only after the individual has slept for the same amount of time they were sleep deprived.
Question 12
Which of the following sleep disorders is best described as an arousal disorder for which the border between sleep and wakefulness is continually blurred?
A. narcolepsy
B. sleep apnoea
C. jet lag
D. sleep–wake shift in adolescence

Question 13
Sleep inertia is most likely to occur during
A. sleep onset.
B. awakening.
C. REM sleep.
D. NREM sleep.

Question 14
Circadian rhythm phase disorders are best described as disturbances primarily involving
A. shift work.
B. difficulty initiating or maintaining sleep.
C. unwanted physical movements or actions during sleep.
D. a sleep pattern that is misaligned with lifestyle demands and social expectations.

Question 15
A core symptom of narcolepsy is
A. excessive sleep.
B. excessive sleepiness.
C. rapid onset of sleep.
D. inadequate sleep.

SECTION B — Short-answer questions
Answer all questions in the spaces provided. Write using blue or black pen.

Question 1 (3 marks)
(a) A sleep walker is likely to have a __________ arousal threshold. 1 mark
(b) Explain why it is unlikely that a sleep walker would be acting out a dream. 2 marks

Question 2 (2 marks)
Describe two distinguishing characteristics of a microsleep.

Question 3 (4 marks)
Explain the distinction between dyssomnias and parasomnias with reference to an example of each disorder category.

Question 4 (5 marks)
(a) What is jet lag? 1 mark

(b) AFL teams based on the west coast of Australia regularly travel by jet for up to five or more hours to compete with teams on the east coast of Australia. Explain whether these west coast teams are likely to be affected by jet lag on arrival at their destination. Ensure you refer to the conditions when jet lag is most likely to occur. 4 marks
Question 5 (4 marks)
Compare cataplexy associated with narcolepsy and sleep paralysis associated REM sleep, ensuring you refer to two similarities and two differences.

Question 6 (2 marks)
What is the difference between an advanced sleep phase disorder and a delayed sleep phase disorder in relation to sleep–wake times?

Question 7 (4 marks)
(a) What is the most common cause of sleep apnoea? 1 mark

(b) List three disturbances to an individual’s major sleep episode that are commonly associated with sleep apnoea. 3 marks

Question 8 (4 marks)
(a) Explain the meaning of sleep hygiene. 2 marks

(b) Describe a sleep hygiene practice that could contribute to the development of each of the following disorders.
   (i) sleep-onset insomnia 1 mark

   (ii) a circadian rhythm phase disorder 1 mark

Question 9 (4 marks)
(a) Define sleep deprivation as commonly used in the study of sleep. 1 mark

(b) Explain the meaning of amplified emotional responses in relation to sleep deprivation. 1 mark

(c) What are two factors that can influence the speed of recovery from sleep deprivation? 2 marks
**Question 10** (9 marks)
(a) Define sleep-onset insomnia with reference to three symptoms. 3 marks

(b) Explain how someone with sleep-onset insomnia could develop a circadian rhythm phase disorder and name the probable type of phase disorder. 2 marks.

(c) What would be the main aim of cognitive behavioural therapy for insomnia? 1 mark

(d) What would be three goals of stimulus control therapy for insomnia? 3 marks

**Question 11** (8 marks)
(a) Explain how shift work can disrupt a person’s sleep–wake cycle and make them susceptible to a circadian rhythm phase disorder. 3 marks

(b) Give an example of a change to sleep timing, quantity and quality that may be caused by shift work. 3 marks

(c) Give two examples of how a shift worker could readjust or compensate for a sleep–wake shift. 2 marks

**Question 12** (11 marks)
Noah is approaching adolescence and even keener to assert his individuality and independence. He goes to bed and sleep at different times during the week, and these times vary even more on weekends when he can sleep in as much as he wants to. Noah believes that it doesn’t matter if he stays up late to study or socialise on Friday or Saturday night because he can sleep in as late as he wants the following day.

(a) Explain why it is important for Noah to maintain a regular sleep–wake schedule even on weekends if he wants to limit or avoid a sleep–wake cycle shift. 3 marks
(b) What is bright light therapy? 1 mark

(c) What three features of timing are crucial to its effective use? 3 marks

(d) How could bright light therapy influence a re-shift of circadian phase change during adolescence? 4 marks

Question 13 (15 marks)

An experiment to investigate treatment of insomnia with melatonin used 12-year-old children as participants, all of whom had persistently experienced sleep-onset insomnia for more than 12 months. Each child was randomly allocated to one of two groups. The experimental group took a pill containing 5mg of melatonin each night throughout a four-week period. The control group was given a placebo throughout the same period of time. In this experiment, neither the research assistants who distributed the medication nor the participants were aware of the treatment allocation — that is, which participants received the melatonin pill and which participants received the placebo. The results showed that children treated with melatonin slept significantly better in relation to sleep quantity and quality, and had improved health during the period of treatment compared with children not treated with melatonin over the same period. The researchers concluded that children with insomnia may be helped by melatonin treatment, at least in the short term. The long-term effects of melatonin use for insomnia would be a target of future research.

(a) Suggest a research hypothesis for the experiment that would be supported by the results obtained. 2 marks

(b) Name the experimental design. 1 mark

(c) Identify the experimental and control conditions. 2 marks

(d) Identify the operationalised independent and dependent variables. 2 marks

(e) Why was random allocation used? 1 mark

(f) Why was a placebo used in the control condition? 1 mark
(g) Explain whether a single- or double-blind procedure was used and why it was used. 2 marks

(h) Explain two ethical issues that are of particular relevance to this experiment. 2 marks

(i) Explain two potential limitations of the experiment. 2 marks

Return to the checklist on page xxx and complete your self-assessment of areas of key knowledge where you need to do more work to improve your understanding.

**eBookplus**

The answers to the multiple-choice questions are in the answer section at the end of this book and in eBookPLUS.

The answers to the short-answer questions are in eBookPLUS.

Note that you can also complete Section A of the chapter test online through eBookPLUS and get automatic feedback. int-0000