Chapter 1

Biological rhythms and sleep

Unit 3

In this chapter, we will be looking at:

- biological rhythms
  - circadian, infradian and ultradian rhythms
  - the disruption of biological rhythms
- sleep states
  - the nature of sleep
  - explanations of sleep
  - lifespan changes in sleep
- disorders of sleep
  - insomnia
  - sleep walking
  - narcolepsy

Biological rhythms

A biological rhythm is a pattern of physiological processes, sometimes accompanied by psychological changes, which repeats itself on a regular basis over a specific period of time. In this section we will look at three kinds of biological rhythms: circadian, infradian and ultradian.

Description	Length of cycle
Circadian	Approximately 24 hours
Infradian	Longer than a day, for example monthly
Ultradian	More than once within a day

Circadian rhythms

This term comes from the Latin '*circa*', meaning 'about' and from '*dies*', meaning 'day'. There are many biological rhythms that vary regularly, following this pattern.

For example, heart rate, urine secretion, metabolic rate, respiration (breathing) and temperature are all highest at around 4 p.m. and lowest at around 4 a.m. Hormone levels also vary; prolactin, which stimulates milk production in females, rises in the middle of the night.

Circadian rhythms are maintained by external cues (Zeitgebers) such as light and dark

However, sleep is the most obvious example of a circadian rhythm, as we normally follow a regular 24-hour cycle of sleeping and waking. The regular pattern of sleep and waking is maintained by exogenous (external) factors, cues such as light and dark, or mealtimes; these are known as Zeitgebers (German for 'time givers'), and light appears to be the most important factor. The role of Zeitgebers was established in a classic study by the French explorer Michel Siffre (see Box 1.1).

Box 1.1 Siffre (1972)

Aim: To establish the role of light as a Zeitgeber in the cycle of sleep and waking.

Procedure: Siffre spent 7 months underground. He had adequate food and opportunities to take exercise and was always able to contact others by phone, but had no cues as to whether it was day or night. Changes in his pattern of sleep and waking were observed.

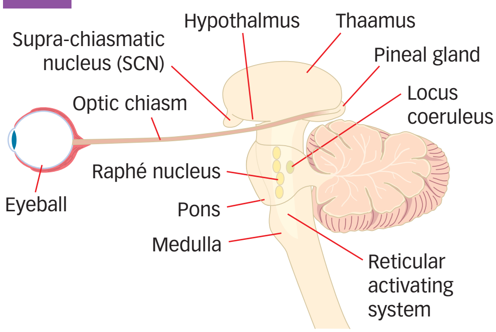
Results: After a time, he showed a 25-hour cycle of sleeping and waking.

Conclusion: The pattern of sleep and waking remains even when there are no external cues. However, the natural length of the cycle is 25 hours.

The results of this study suggest that as well as external cues, there must be endogenous (internal) factors, known as **pacemakers**, which maintain this rhythm when external Zeitgebers cannot be used.

The principal internal mechanism or **biological clock** that governs circadian rhythms appears to be the **supra-chiasmatic nuclei (SCN)** of the **hypothalamus** (Figure 1.1). SCN lesions have been shown to disrupt circadian rhythms (Ibuka and Kawamura 1975) and there is a correlation between cyclical changes in behaviour and the activity of neurons in that area of the brain (Rusak and Groos 1982). Other clocks, such as temperature changes, may regulate specific

Figure 1.1 *The brain physiology of arousal and sleep*



rhythms, but the SCN appears to have some controlling function. This makes sense, since the SCN receive nerve input directly from the retina of the eye, so can respond to the Zeitgebers of light and darkness. This information is passed to the **pineal gland**, which manufactures **melatonin**, a hormone that regulates many of the body's systems and is involved in bringing about sleep.

As well as this system of control, the SCN may also govern circadian rhythms by means of the secretion of **neuromodulators**, chemicals that affect the behaviour of neurotransmitters. Ralph et al. (1990) found that grafted SCN, even when they had not formed connections with other areas of the brain, were nonetheless able to establish circadian rhythms within a few days. Moreover, in a study of hamsters, Hard and Ralph (1998) found that if the animal receiving the graft had a slightly different cycle from the donor, the donor cycle was adopted.

However, a person can adapt their biological rhythms if necessary, for example if they work permanently at night, although the time it takes to do so varies from person to person and some people may never fully adapt.

Infradian rhythms

Infradian rhythms, where the cycle is longer than a day, include the menstrual cycle (28 days), testosterone secretion in males (21 days) and seasonal mating.

The most researched of these is the **menstrual cycle**. This relates to activity in the endocrine system that prepares the womb for the possibility of conception after egg cells are released. Several hormones are involved, coordinated by the **pituitary gland**. This gland may be influenced by levels of light and the secretion of melatonin, a view supported by research (see Box 1.2).

Box 1.2 Reinberg (1967)

Aim: To investigate the influence of light on biological rhythms.

Procedure: A young woman spent 3 months in a cave, with no external source of light, the only light available being a miner's lamp. The effects on the sleep/waking cycle and the infradian cycle of menstruation were noted.

Results: As in the Siffre study (Box 1.1), the woman's day lengthened, in this case to 24.6 hours. Her menstrual cycle shortened to 25.7 days. At the end of the study, it took almost a year for her menstrual cycle to return to normal.

Conclusion: The lack of light as a Zeitgeber resulted in changes both to the circadian rhythm of the sleep/waking cycle and to the infradian rhythm of menstruation, which was slow to adapt to the previous pattern even when light was restored.

The menstrual cycle may also be influenced by smell. It has been found that when a group of young women spend a lot of time together, their menstrual cycles tend to synchronise and so follow the same timing. Russell et al. (1980) showed that synchronisation could be produced simply by transferring samples of underarm sweat from one woman to another.

The phenomenon of **premenstrual syndrome (PMS)** is associated with hormonal changes during the menstrual cycle. This refers to the negative psychological effects experienced by up to 60% of women 4–5 days before a period. The symptoms may include irritability, depression, headaches, insomnia, lethargy and sometimes changes in appetite; both an increase and a decrease have been reported. Some researchers, for example Dalton (1964), have suggested that during this part of the menstrual cycle, women are more likely to have accidents, carry out crimes, commit suicide and to have reduced scores on IQ tests.

Clare (1985) points out that there is no detectable and consistent hormonal abnormality that differentiates those women who suffer from PMS from those who do not. Moreover there are often methodological problems with studies that have reported a link between PMS and aggression, accidents and so on. A small number of women do seem to be vulnerable to behavioural and emotional changes due to menstruation, but these have not been consistently linked with any particular phase of the menstrual cycle.

Ultradian rhythms

These rhythms are shorter than a day and include changes within a 24-hour period in heart rate and the secretion of hormones. Most of the research in this area has investigated the stages of sleep; this will be looked at in a later section.

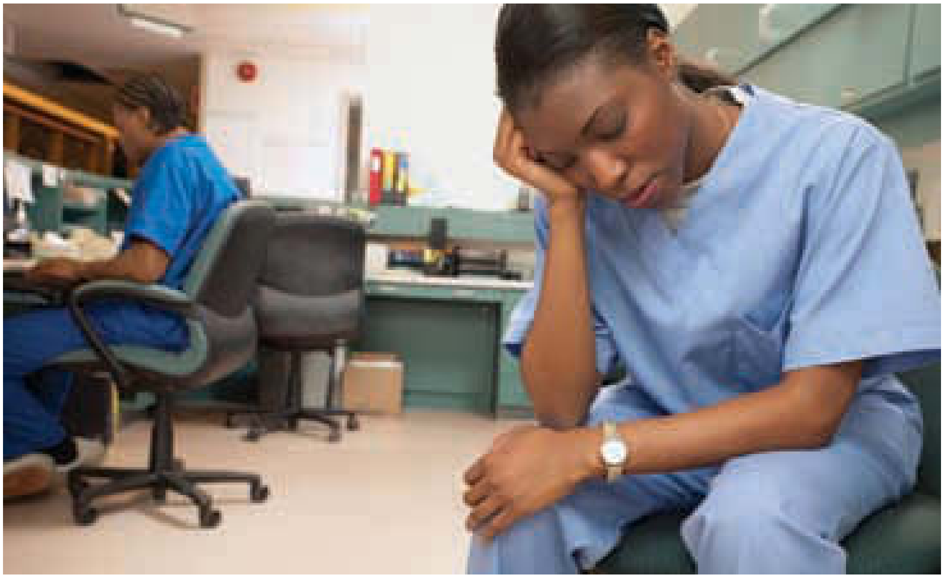
Summary

- Physiological and psychological processes generally show a rhythm or **cycle** that recurs over a period of time.
- **Circadian rhythms** are repeated approximately over 24 hours. The sleep/waking cycle is an example and is governed by external **Zeitgebers** and internal **pacemakers** involving the **SCN** of the **hypothalamus** and the **pineal gland**.
- **Infradian rhythms** recur over a period longer than a day. The **menstrual cycle** is an example.
- **Ultradian rhythms** recur over a period shorter than a day. The most researched example is the **stages of sleep**.

The disruption of biological rhythms

There has been a lot of research into what happens when biological rhythms are disrupted. This disruption can take two forms: **desynchronisation**, when different rhythms adapt at different rates, and **flattening**, when the amount of variation is decreased. Shift work and jetlag disrupt circadian and other biological rhythms; these are a source of stress as people have difficulty in making the rapid adjustments that are required, consequently there has been a considerable amount of research on both.

Shift work



Shift work can cause sleep disturbances that lead to accidents caused by sleepiness

In the short term, working rotating shifts can cause sleep disturbances, fatigue, stress, irritability, errors and accidents. For example, in a hospital-based survey, Gold et al. (1992) found that nurses who worked rotating shifts were twice as likely to fall asleep while driving to work as those who worked only day or evening shifts and were twice as likely to report an accident or error at work due to sleepiness. Costa (1999) has summarised the serious long-term effects of shift work (see Box 1.3).

Box 1.3 Costa (1999)

Research has shown that the long-term effects of shift working include:

- difficulties in social and family relationships
- the development of peptic ulcers
- chronic fatigue, anxiety and depression
- cardiovascular problems, for example hypertension and ischaemic heart disease
- consequences for women's health, for example pregnancy difficulties

The severity of the effects varies, depending on individual factors, such as age, personality and physiological characteristics; on the working situation, such as workload schedules; and on social conditions, such as the number and age of children, housing and commuting to work.

However, it has been estimated that around 20% of all workers have to leave shift work after a short time because of serious problems.

To some extent, the problems associated with shift work can be reduced by making changes in the workplace (see Box 1.4).

Box 1.4 Blakemore (1988)

Aim: To investigate possible improvements in health and productivity through changing the pattern of shift working.

Procedure: Workers in a chemical company in Utah were studied. The company operated a three-shift system, in which employees worked a day shift for a week, then a night shift and then an evening shift, before starting the cycle again. The effects of lengthening the period between shift changes and rotating the shifts in the opposite direction, i.e. clockwise, in line with the body's preference for a longer rather than a shorter day, were assessed.

Results: Both the health and the productivity of the workers improved.

Conclusion: It is possible to modify the effects of shift work.

Conversely, a shorter shift system can also be beneficial. Williamson and Sanderson (1986) found that bringing in a more rapidly rotating system, where workers never worked for more than three consecutive nights, led to improved health. Pisarski et al. (2008) also found that the negative effects on the health of shift workers could be reduced if they felt they had social support and some control at work.

Other ways of addressing these problems have focused on measures that may help to reset the body clock. Exposure to light can go some way towards helping shift workers overcome adjustment problems. Dawson and Campbell (1991) found that exposing workers to 4 hours of bright light on the first night of shift work could help people to adjust. There has also been a lot of interest in **chronobiotics**, substances that can readjust the timing of biological rhythms. Several studies, for example Touitou and Bogdan (2007), have found that the hormone **melatonin** can be used effectively in this way. However, while some modification is possible, there is as yet no way of eliminating completely the adjustment problems arising from shift work.

Jetlag

When we travel across different time zones, for example, if we fly to the USA, the Zeitgebers — the external cues that help to regulate circadian rhythms — give us information that conflicts with our internal biological clock, with the result that we feel tired when others are awake, get hungry at the wrong times and so on. It may take up to 10 days for our bodies to adjust. Some rhythms, such as temperature, adjust more quickly than others, such as ACTH production. There are also individual differences in how easily people adapt; some never do so completely.

guichaoua/Alamy

Those who frequently experience jetlag can suffer from long-term effects

The immediate effects of jetlag can include headache, sleepiness, irritability and difficulty in concentrating; these are usually overcome after a few days. However, for those who frequently experience jetlag, such as pilots and cabin crew, there may be long-term effects (see Box 1.5).

Box 1.5 Cho (2001)

Aim: To investigate the long-term effects of frequent jetlag.

Procedure: Participants were 20 healthy women who had worked for at least 5 years as flight attendants. Participants who were allowed only a few days rest between flights were compared with those allowed longer recovery periods.

Results: Those who had little recovery time between flights performed worse on memory tests. They showed slower reaction times and made more mistakes. Brain scans showed that they had significant shrinkage of the right temporal lobe of the brain, and this was correlated with high levels of the stress hormone cortisol, which affects the immune system.

Conclusion: Repeated jet lag, with insufficient recovery time between flights, can lead to reduced cognitive ability and brain damage.

Cho et al. (2000) also found that cabin crew had problems with working memory, which became apparent after several years of the disruption of circadian rhythms associated with their work.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It may take up to 10 days for our bodies to adjust from the effects of jetlag

One way of dealing with the immediate effects of jetlag is to try to stay awake longer. As shown earlier, in the absence of external cues (Zeitgebers), the body adjusts to a slightly longer cycle of around 25 hours, so extending the waking part of the cycle is more effective than trying to shorten it. This also explains why jetlag is less of a problem flying from east to west, and so lengthening the day, than from west to east.

As with shift work, **melatonin** can be effective in treating the immediate effects of jet lag (Herxheimer and Petrie 2002). However, it needs to be taken at the right time — at around the time of day that a traveller wants to fall asleep when he or she arrives at the destination — or it may make the problems worse. It may also interact badly with other medication, such as the blood-thinning drug Warfarin.

Summary

- Working **rotating shifts** disrupts circadian rhythms and so produces problems in the short term, but also has **long-term health implications**.
- Changing **working practices** can help to reduce the effects of shift working. **Exposure to light** and the use of **melatonin** have also been found to be effective, but cannot altogether overcome the problems.

- The adjustment of circadian rhythms when crossing time zones causes the short-term effects of **jetlag**.
- There are more serious **physiological effects** for people who experience this regularly without sufficient recovery periods.
- In the short term, jetlag can be reduced by **staying awake longer**. **Melatonin** can also be helpful.

Sleep states

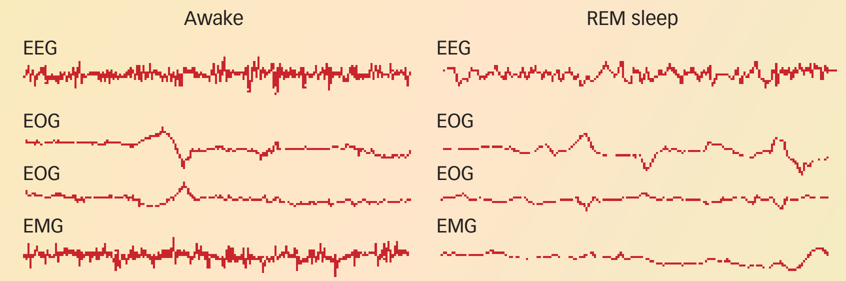
Physiological changes that occur during sleep have been well documented, and researchers have been interested in the mechanisms that bring about these changes. Another area of interest has been the question of why we need to sleep, and a number of explanations have been put forward. A further question is how sleep changes across the lifespan. We will be looking at all these aspects of sleep research in this section.

The nature of sleep

Stages of sleep

While the cycle of sleeping and waking has a circadian rhythm, there are also stages within sleep that are based on a 90-minute ultradian cycle. These have been recorded using different methods, shown in Figure 1.2.

Figure 1.2 *Figure 1.2 Methods of recording the stages of sleep*



EEG (electroencephalogram): measures electrical activity in different parts of the brain
EOG (electrooculogram): shows the electrical activity as a result of eye movements
EMG (electromyogram): records electrical activity from the chin muscles, and so gives information abut muscle tension

One particular kind of sleep that has been of interest to psychologists is **rapid eye movement (REM)** sleep. People experience several periods of this in the course of a night's sleep, during which the eyes make rapid movements that produce intense EOG activity. In an early study, Dement and Kleitman (1957) found that REM sleep shows a distinctive pattern of brain activity and that it is predominantly associated with dreaming. Following on from this, the different kinds of sleep we experience have been categorised (see Box 1.6).

Box 1.6 Rechtschaffen and Kales (1968): the stages of sleep

Stage 0: wakefulness, this is identified by the presence of low-amplitude, high-frequency beta waves in the EEG. As we relax, these are replaced by alpha waves, which are higher in amplitude but slower in frequency (8–12 cycles per second).

Stage 1: this is characterised by the appearance of theta waves in the EEG; these are slower (4–7 cycles per second) and more irregular. Breathing and heart rate slow down, body temperature falls and muscles relax. There may also be slow eye rolling shown on the EOG.

Stage 2: this usually occurs after about a minute and is characterised by brief bursts of high frequency EEG activity, known as sleep spindles, which last about 1 second. There are also K-complexes, which represent the brain's response to stimulation, either external, such as a noise in the room, or internal, such as a muscle movement. Very slow (1–3 cycles per second), high-amplitude delta waves start to appear. The EOG shows little activity and the EMG is reduced still further.

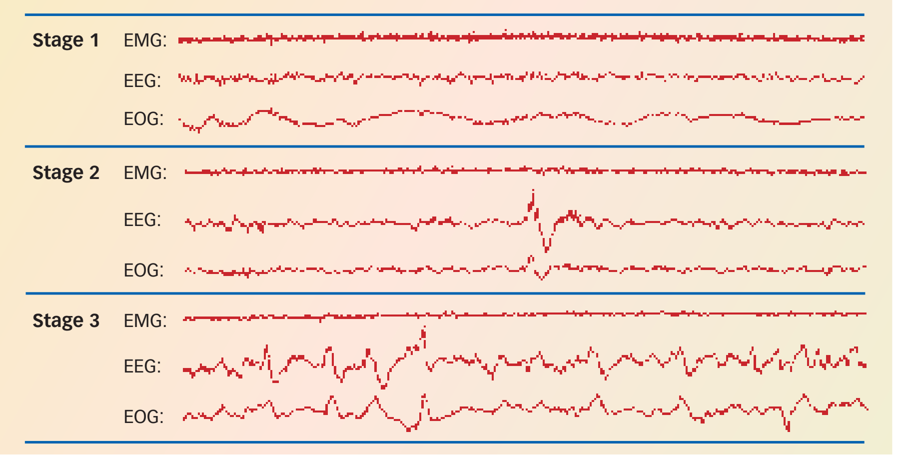
Stage 3: this occurs after about 20 more minutes. There is between 20% and 50% delta activity in the EEG.

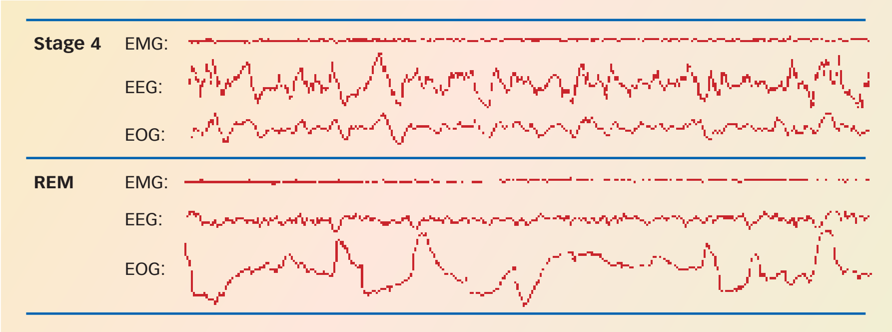
Stage 4: sleep follows shortly, when delta activity has increased to over 50% of the total and become even slower. Heart rate, blood pressure and body temperature are at their lowest and the muscles are relaxed. At this stage, people are difficult to wake and do not respond readily to external stimuli, so this is seen as a deeper form of sleep; it lasts for about 40 minutes.

Stages 3 and 4 are known as slow-wave sleep and stages 1–4 as non-REM (NREM) sleep.

Having descended what is known as the sleep staircase into deeper and deeper sleep, the sleeper then starts to climb back through stages 3 and 2; this is followed by REM sleep. The EEG pattern here resembles that in the waking, relaxed state with a high level of alpha activity. There are also PGO spikes, which are short bursts of high frequency, large amplitude activity from the pons, thalamus and visual cortex. Heart rate, blood pressure and breathing rate increase and become more irregular; rapid eye movements occur, where the eyes flick from side to side. The EMG record shows that in spite of these signs of activity, the muscles are in a state of virtual paralysis, apart from occasional twitches of the toes and fingers. This is when people are hardest to wake. It is also known as paradoxical sleep, since the brain is alert but the body is not. After about 10 minutes, we return to stage 2 and descend the sleep staircase again.

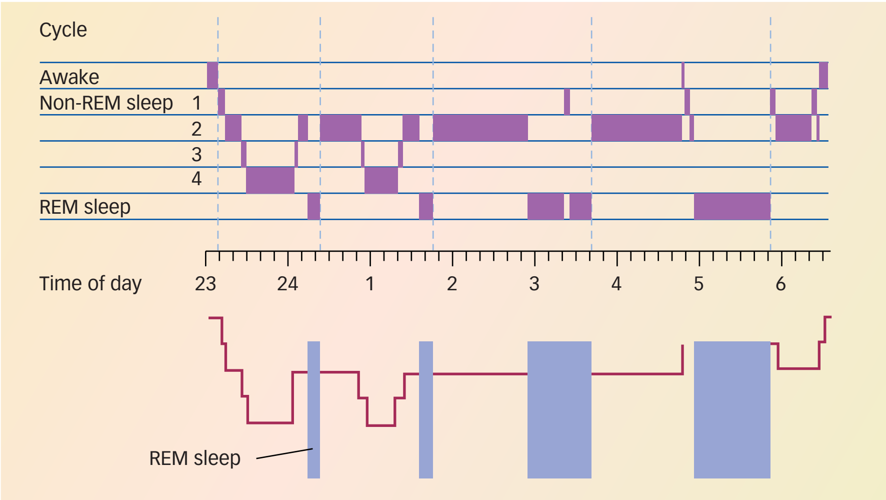
Figure 1.3 *EEG, EOG and EMG recordings in the various stages of sleep*





During the night, we usually experience around five sleep cycles, each of which lasts about 90 minutes. Stages 3 and 4 only occur in the first two cycles and the periods of REM get longer in the course of the night.

Figure 1.4 *Characteristic profile of a night's sleep.*



The exact pattern of sleep varies from person to person and can also vary in the same person on different nights. As we will see later, there are also changes in the pattern of sleep with age.

Summary

- The **stages of sleep** show a cycle of approximately **90 minutes**.
- Brain activity during sleep is recorded in several ways: **EEG**, **EOG** and **EMG**.
- The different stages show different patterns of **alpha**, **delta** and **theta** waves.
- **REM** sleep is the hardest kind of sleep to wake up from and is predominantly associated with **dreaming**. The other stages are **NREM** sleep.
- The stages of sleep show some variation between **individuals** and with **age**.

Mechanisms of sleep

Research into the mechanisms of sleep has looked at neurochemicals, circulating sleep-inducing chemicals and at neurological mechanisms involved in sleep, i.e. sleep centres and circuits.

We have already looked at the link with circadian rhythms; when it gets dark the eyes send messages to the SCN of the hypothalamus and from there to the pineal gland. The pineal gland starts to secrete melatonin, which makes us feel drowsy. Melatonin in turn influences neurons in the **raphé nuclei** in the brain stem, which produce the neurotransmitter **serotonin**. Serotonin in turn influences activity in the nearby **reticular activating system (RAS)** (see Figure 1.1).

The RAS is concerned with arousal. Early research has shown that low levels of activity in the RAS are associated with the onset of sleep. Moruzzi and Magoun (1949) and French (1957) found that a sleeping cat could be woken by stimulation of the RAS. More recent research seems to indicate that the RAS is involved with movement rather than arousal. In the context of sleep, serotonin is an inhibitory transmitter and lesions of the raphé nuclei, which are known to produce sleeplessness, will produce an almost complete loss of brain serotonin. Drugs that reduce levels of serotonin in the brain prevent sleep and those that increase levels of serotonin reverse this effect.

A different system appears to govern REM sleep. Jouvet (1967) found that destruction of the **locus coeruleus**, located in the pons, eliminates REM sleep. The locus coeruleus produces the neurotransmitters **noradrenaline** and **acetylcholine**. REM sleep occurs when there are increases in activity in acetylcholine systems and ends when there are increases in activity in noradrenaline systems. This can be linked to the finding that **monoamine oxidase inhibitors (MAOIs)** also eliminate REM sleep. These are drugs used to treat depression and increase levels of noradrenaline and serotonin.

Other research has focused on the idea that substances that promote sleep or wakefulness might be produced, accumulating in the blood during wakefulness and being destroyed during sleep. Support for this comes from Monnier and Hosli (1964), who were able to extract **delta-sleep-inducing-peptide (DSIP)** from rabbits, which induced sleep when injected into rats. Pappenheimer et al. (1975) obtained **Factor S** from the cerebrospinal fluid of sleep-deprived goats, which had the same effect; this has also been found in human urine (Garcia-Arraras and Pappenheimer 1983). However, the role of these chemicals is still unclear, as they have other effects as well, such as raising body temperature and stimulating the immune system.

There are further problems with this theory. Mukhametov (1984) has shown that in dolphins the two halves of the brain sleep separately, which

Summary

- The **hypothalamus**, **pineal gland** and **RAS** are involved in **NREM** sleep, which is influenced by the neurotransmitter **serotonin**.
- The **locus coeruleus** governs **REM** sleep. The neurotransmitters **acetylcholine** and **noradrenaline** are involved.
- Other chemicals may also be important in regulating sleep.

they would not be able to do if sleep was being produced by chemicals in a shared blood supply. Even more convincingly, conjoined twins, who also share the same blood supply, show different patterns of sleep.

Explanations of sleep

A number of theories have been put forward to explain why we sleep. Some of them are concerned with sleep in general, while others focus only on a particular kind of sleep. Evolutionary theories and restoration theories are important examples of the ideas that have been suggested.

Evolutionary explanations of sleep

Evolutionary theory aims to explain behaviour in terms of how it may be adaptive. In this context, **adaptive** refers to how a behaviour may promote survival long enough for an individual to breed and pass on their genes to the next generation. On the principle of **natural selection**, members of a species with characteristics that promote survival will be more successful in passing on their genes than individuals who do not share those characteristics.

Meddis (1975) suggested that sleep may be adaptive in terms of safety, drawing on the fact that some species sleep for longer than others and at different times. For example, predators such as lions, which are unlikely to be attacked by other animals and can quickly and easily meet their nutritional needs, sleep for long periods. In contrast, sheep, which have little defence against predators and need to spend a long time feeding to gain enough nutrition, only sleep for about 2 hours a day, in the form of brief naps. The amount of time spent asleep is therefore related to an animal's need for and method of obtaining food and its exposure to predators.

In a similar theory, in that it emphasises the adaptive nature of sleep, Webb (1982) has suggested that sleep is an instinctual behavioural response, related to the need to conserve energy by not expending energy when it is unnecessary or even counter-productive. It would be adaptive to keep out of danger when danger is most likely to occur and there is nothing to be gained by exposure.

This idea leads to the notion known as the **hibernation theory of sleep function** that sleep in humans has evolved because it promotes survival by preventing unnecessary energy expenditure, as we are much less likely to find food at night than during the day. It keeps us out of harm's way by immobilising us at night, when we are vulnerable.

Evolutionary theories are limited in that they attempt to explain differences in sleep patterns and durations, rather than the phenomenon of sleep itself. A major difficulty with this approach is that it has an explanation for completely opposite phenomena. For example, sleeping for longer or for shorter periods could both be seen as adaptive in a species in danger from predators. Sleeping for longer periods would conserve energy necessary to deal with predatory attacks and help to avoid attack. On the other hand, sleeping for shorter periods would mean alertness and so being better prepared to avoid predators.

As with most evolutionary theories, these ideas are impossible to test directly and are purely speculative. Evolutionary theory tends to be better at offering explanations of behaviour than in producing testable hypotheses.

Restoration theories of sleep

It seems reasonable, as evolutionary theories suggest, that safety and energy conservation could be two of the functions of sleep, but it is possible that it also serves other functions. Oswald (1966) has suggested that the purpose of sleep is to restore reserves of energy and repair the brain and body after the events of the day. This theory suggests that NREM sleep restores bodily processes, for example hormone levels, while REM sleep restores brain processes, for example by stimulating the synthesis of proteins.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Restoration theory of sleep explains why babies spend more time asleep

There are several lines of evidence to support Oswald's approach. First, it could explain why babies spend more time asleep than older people, since they would need more REM sleep to assist the development of the central nervous system. Second, people who have been given ECT, where an electric shock is given to the brain as a treatment for depression, show an increase in REM sleep for a period of 6–8 weeks afterwards (Grunhaus et al. 1994), which is about how long it would take to replace half the brain's protein. There is also evidence that we spend longer in NREM sleep after a physically demanding day (see Box 1.7).

Box 1.7 Shapiro (1981)

Aim: To investigate the effect of physical exercise on sleep.

Procedure: Participants were athletes, aged 18–26, who took part in an ultra-marathon, a 92-kilometre road race. Sleep recordings were made on the four nights following the race.

Results: The athletes showed a significant increase in sleep time — 1½ hours longer on the two nights immediately following the race — and specifically in deeper levels of NREM (slow-wave) sleep. Stage 4 sleep accounted for 45% of the total sleep time, where normally this is around 25%. The proportion of REM sleep decreased.

Conclusion: The extra time spent in slow-wave sleep allowed the body to recover from the exertion of the race.

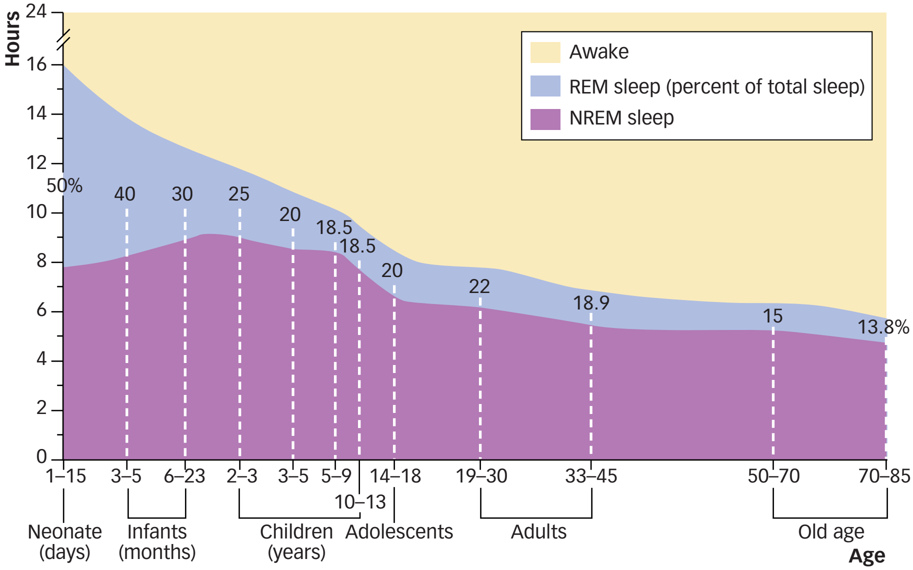
However, if restoration theories are correct, it might be expected that not taking any exercise should result in a shorter time spent asleep, but this does not appear to be the case. Ryback and Lewis (1971) found that healthy people who spent 6 weeks resting in bed showed no change to their sleep patterns.

A variation of restoration theory has suggested that sleep is not only a physiological restorative, for example allowing cell renewal to take place and neurotransmitter levels to be adjusted, but may also serve a similar psychological function. Kales et al. (1974) have shown that insomniacs have far more psychological problems than healthy people. This suggests that psychological problems could arise through a lack of the sleep, which would allow psychological restoration. However, this is correlational evidence, so could equally be interpreted in terms of insomniacs having difficulty sleeping because of their psychological problems.

Lifespan changes in sleep

There is variation across the lifespan in both the total amount of sleep a person experiences and in the proportion of time spent in the different stages of sleep.

Figure 1.5 *Changes in sleep across the lifespan*



Floyd et al. (2007) carried out a study of the amount of REM sleep across the lifespan. In a sample whose ages ranged from 18 to over 90, they found that REM sleep decreased by about 0.6% each decade. In the mid-70s, however, there was a small increase in REM sleep, while total sleep time decreased.

A more detailed analysis of a number of studies into changes in sleep across the lifespan has also been carried out (see Box 1.8).

Box 1.8 Obayon et al. (2004)

Aim: To carry out a meta-analysis of studies of the changes in sleep across the lifespan.

Procedure: An analysis was made of the findings of 65 studies of changes in sleep patterns across the lifespan, in which participants were aged from 5 to 102 years old.

Results: In children and adolescents, the total sleep time decreased significantly from typical levels only in studies carried out on school days. In adults, total sleep time and the percentages of slow wave and REM sleep all decreased significantly with age. Sleep latency (the period before falling asleep), the percentages of stage 1 and stage 2 sleep and waking up after falling asleep all increased significantly with age. However, after the age of 60, only sleep efficiency, i.e. the percentage of time between falling asleep and waking that is actually spent asleep, continued to decrease significantly.

Conclusion: There are a number of changes in the patterns of sleep across the lifespan.

Newborn infants sleep for around 16 hours a day, half of which is REM sleep. This drops to about 12 hours at 12 months old, a third of which is REM sleep. It has been suggested by Empson and Clarke (1970) that REM sleep is important for memory consolidation. This could account for the prominence of REM sleep in infants and young children, given the amount of new information they take in each day.

Obayon (2004) also noted that there are few studies that investigate sleep in school-aged children and adolescents, making it difficult to draw conclusions about the sleep of these age groups. In one study, Loessl et al. (2008) investigated the amount of sleep reported over a period of 2 weeks by school students aged 12–18. They found that the majority slept on average less than the 9 hours per night recommended for this age group, with even less sleep on school nights. However, few reported daytime sleepiness or impaired cognitive functioning, suggesting little effect of sleep deprivation on people of this age.

Morphy et al. (2007) found a significant correlation between age and insomnia, which we look at in the next section. Older people also appear to be more susceptible to the effects of sleep disturbance than younger people (see Box 1.9).

Box 1.9 Koller (1983)

Aim: To compare the health of workers working changing shifts and day workers.

Procedure: Workers in an oil refinery provided information about absence due to sickness, morbidity, severity of diseases and subjective complaints, which gave them an overall health score. The health scores of shift and day workers were compared.

Results: Older workers had lower health scores, but the pattern of deterioration differed for shift and day workers. In the shift workers, there was a steep decrease during the early years at work, a continued slight decrease in middle age, followed by a further sharp decrease. In day workers, there was little decrease up to middle age, followed by a sharper drop. The permanent shift workers showed an increase in absence through illness, in particular with gastro-intestinal problems and heart disease. They also reported more sleep disturbances.

Conclusion: Older workers are more vulnerable to the effects of sleep problems caused by the disruption of circadian rhythms involved in shift work.

Summary

- **Evolutionary theories** of sleep suggest that sleep is **adaptive**. Sleep patterns differ in different species and this difference is related to the need for obtaining food and avoiding predators. It also serves the function of conserving energy. While this kind of explanation seems feasible, these ideas **cannot be tested** directly.
- **Restoration theories** suggest that the purpose of sleep is to restore the brain and the body. There is evidence to support this view.
- Sleep may also be important in restoring **psychological functioning**.
- There are changes in both the amount of sleep and the proportion of REM sleep across the **lifespan**.

Disorders of sleep

Many of us have occasionally had a night where sleep was difficult, perhaps because we were worried about an exam the next day or have been disturbed by noises from the street outside. However, there are a number of sleep disorders that are not only distressing but also may threaten the individual's health. Some of these will be looked at in this section.

Insomnia

Insomnia can be defined as the chronic inability to get enough sleep due to difficulty in falling asleep, frequent waking during sleep and/or early morning waking. Kao et al. (2008) found that difficulty getting to sleep was the most commonly reported symptom, followed by early morning waking and difficulty staying asleep. Morphy et al. (2007) found that of over 2,000 English adults who took part in a questionnaire survey, 37% reported symptoms, though this may be something of an overestimate, since the data came only from those who chose to return the completed questionnaire and so may be biased. Several studies carried out in western Europe and North America have reported an incidence of around 20%. As well as being frustrating and unpleasant, insomnia creates a range of problems, such as daytime sleepiness and difficulties in concentration, memory and with personal relationships.

Insomnia can be divided into primary and secondary insomnia. **Primary insomnia** is psychophysiological in origin and linked to hyperarousal, though the precise mechanisms involved are not clear. **Secondary insomnia**, the more common type, is the result of psychiatric or organic illness, or the effect of prescription or illicit drugs, alcohol, or any combination of these factors.

Primary insomnia

Primary insomnia has been linked to **personality factors**. Wang et al. (2001) compared patients with chronic primary insomnia and healthy controls. The insomniacs scored higher on scales measuring neuroticism, anxiety and impulsivity.

They suggested that this could be the result of aberrant functioning of the hypothalamus, or a neurotransmitter imbalance. The results and conclusions are consistent with the findings of other studies in this area (see Box 1.10).

Box 1.10 Kales et al. (1983)

Aim: To assess personality characteristics of patients with chronic insomnia.

Procedure: The Minnesota Multiphasic Personality Inventory (MMPI) was used to draw up personality profiles. The results of 428 insomniacs and 100 healthy controls were compared.

Results: Unlike the controls, the insomniac profiles demonstrated neurotic depression, chronic anxiety, inhibition of emotions and an inability to express anger.

Conclusion: The results are consistent with the idea that handling stress and conflict through internalising emotions has physiological effects and is a major factor in insomnia.

There appears to be general agreement that a tendency towards the internalisation of emotions and the occurrence of stressful life events play a major role in the development of chronic insomnia.

It also seems that some insomniacs overestimate their sleep problems. Mercer et al. (2002) found that some greatly overestimate the time it takes them to fall asleep and underestimate how long they have slept. They may believe that they have spent much of the night awake, even though EEG recordings show that they have in fact been sleeping. Perlis et al. (2001) have suggested that this may arise as a result of a fault in the mechanisms that normally erase our memories during the transition from wakefulness to sleep, blurring the distinction between sleep and wakefulness and so creating a false perception of having been awake.

Secondary insomnia

Dejanovic et al. (2003) suggest that there are four different kinds of causes of secondary insomnia:

Causes of secondary insomnia

Environmental factors, including stress, emotional arousal, noise, shift work (and night work in general) and intercontinental flights from east to west or west to east. Narcotics, alcohol and drugs also come under this heading.

Organic problems, such as chronic pain.

Mental disorders, such as anxiety, depression and bipolar disorder.

Causes specific to sleep, of which perhaps the most important is a breathing disorder, obstructive sleep apnoea.

They suggest that primary insomnia should only be diagnosed when there is no other obvious cause.

Insomnia can be linked to **anxiety** disorders. In a study of medical students, Jiang et al. (2003) found that the life events experienced by insomniacs and controls were similar. The groups differed, however, in their tolerance of stressors. There is also a strong link between insomnia and clinical **depression**, of which early-morning waking is a typical symptom. In depressed patients, patterns of sleep nearly always change. There is typically an increase in REM sleep, REM sleep is entered more quickly than normal and there is a higher frequency of rapid eye movements.

Much of the research in this area is correlational, so it is not clear whether depression causes insomnia, or is a result of it. It is possible that depression can lead to insomnia and insomnia can lead to depression. There is evidence that both anxiety and depression can be a cause of insomnia (see Box 1.11).

Box 1.11 Jansson-Fröjmark and Lindblom (2008)

Aim: To investigate the relationships between depression, anxiety and insomnia.

Procedure: A sample of 3,000 participants completed a survey on anxiety, depression and insomnia and a follow-up survey a year later.

Results: There was a strong interrelationship between anxiety, depression and insomnia. Both anxiety and depression as measured on the first survey were predictive of the development of new cases of insomnia on the follow-up survey. Measures of insomnia on the first survey were also predictive of new cases of depression and anxiety in the follow-up survey.

Conclusion: There is a bi-directional relationship between anxiety, depression and insomnia; while insomnia can lead to anxiety and depression, these conditions can also lead to insomnia.

A major cause of insomnia is **obstructive sleep apnoea** (from the Greek, meaning 'without breath'). This occurs when the upper airway is temporarily blocked when a person is asleep, in spite of the respiratory muscles trying to inhale. The sleeper stops breathing for anything from a few seconds to more than a minute. Eventually a partial reawakening is triggered and the sleeper gasps in some air. This can happen over 100 times in one night. People with sleep apnoea rarely wake up during these episodes, but the effects the next day are sleepiness, irritability and other signs of sleep deprivation. The main symptom apart from the effects of sleep deprivation is snoring, which affects the vast majority of sufferers.

It is a serious condition, since it is associated with an increased probability of developing **hypertension** and **heart disorders**. It has also been associated with an increased risk of motor accidents, as a result of daytime sleepiness. Mulgrew et al. (2008) found that drivers with sleep apnoea were significantly more likely than controls to be involved in motor vehicle crashes and that the crashes they were involved in were more serious.

Sleep apnoea is more likely to affect older people, obese individuals and men rather than women. It is more likely to affect older people as the soft tissues in the upper airways become slacker as we get older. However, children can also suffer from sleep apnoea. In a large-scale study in Iceland, Martikainen et al. (1994) found that 3% of children aged from 6 months to 6 years had sleep apnoea.

Obesity is the most powerful predictor of sleep apnoea in adults, since fat deposits in the neck constrict the upper airways. Stradley and Crosby (1991) found that neck circumference, rather than general obesity, was a strong predictor of sleep apnoea. The relationship between obesity and sleep apnoea can also be a vicious cycle. Obesity can trigger sleep apnoea, but sleep apnoea is also associated with physiological abnormalities that promote weight gain. Added to this, sleep apnoea sufferers may snack on sugar-rich foods like chocolate and fizzy drinks to try to overcome their daytime sleepiness, so making things worse.

Adrian Muttitt/Alamy

Sleep apnoea can be treated with CPAP, which helps keep the airway open

There are also physiological characteristics of facial anatomy that influence whether or not a person will develop the disorder, suggesting a genetically-determined predisposition; those who have long faces, longer soft palates and narrower airways are more vulnerable. Dematteis et al. (2001) found that 11 of 14 members of one family suffered from sleep apnoea, linked to an abnormality of the pharynx.

Sleep apnoea can be treated successfully using **continuous positive airway pressure (CPAP)**. A machine is used, mainly by patients at home, which delivers a stream of compressed air via a hose to a nasal pillow, nose mask or full-face mask, keeping the airway open under air pressure so that unobstructed breathing becomes possible.

Summary

- **Primary insomnia** has been linked to **personality** characteristics, in particular the internalisation of emotions and to **hyperarousal**. Sufferers may also have **distorted beliefs** about how much they sleep.
- **Secondary insomnia** is more common. It is caused by **environmental factors**, **organic problems**, **mental disorders** or causes specific to sleep, especially **obstructive sleep apnoea**.
- **Depression** and **anxiety** are common in people suffering from insomnia; these conditions can cause insomnia, as well as being the result of it.
- **Sleep apnoea** is commonly a cause of insomnia. It is more common in older, male and obese people and may also be genetically predetermined. It can cause long-term **health problems**, but can be treated successfully.

Sleep walking

Sleep walking is known to specialists as **somnambulism** and is one of the **parasomnias**, a group of disorders that also includes nightmares and night terrors. Sleep walking takes place during NREM sleep — it would not be possible during REM sleep, as the muscles are then paralysed — and actions are carried out without the person having any conscious awareness of what they are doing.

People can carry out quite complex behaviours while sleep walking. Gunn and Gunn (2007) report people lining up all their shoes on the windowsill, rearranging furniture, or climbing out of a window in the middle of the night. Martin (2003) relates an anecdote of a lady waking up with the feeling that there was someone else in the room; she heard a thud and fainted with terror. When she awoke, she found that her butler, while sleep walking, had laid the table for 14 people on her bed.


Caro/Alamy

Somnambulists can carry out complex tasks when sleep walking

Sleep walking is relatively common in children and is usually benign and self-limiting. All that is usually necessary is to reassure the parents, make sure that steps are taken so that children cannot hurt themselves and wait for it to pass. It has been suggested that cognitive behavioural therapy may help childhood sufferers, but there has been only limited research into its effectiveness. Sleep walking reaches a peak in pre-adolescence and is relatively rare in adults. It is estimated to affect around 2% of the adult population worldwide. It is less common in older people and affects men and women equally.

Sleep walking is more likely to occur if the person is very tired, so **stress**, anxiety and any other factor that disturbs sleep may trigger sleep walking. Soldatos and Kales (1990) suggest that in adults it may be the result of underlying pathology, in particular stress or **depression**. It may also be the result of **trauma**. For example, Kurtz and Davidson (1973) report a case of somnambulism in an 11-year-old child who underwent a trauma following the injury of his father in an Israeli security operation. Ohayon (1999) identified several factors associated with sleep walking, which include: being between 15–24 years old; a subjective sense of choking or blocked breathing at night; sleep talking; and being involved in a road accident within the previous year. There is also some evidence that it may run in families (see Box 1.12).

Box 1.12 Hublin et al. (1997)

Aim: To investigate possible genetic effects in sleep walking.

Procedure: In a Finnish study, over 11,000 participants aged 33 to 60 years, including 1,045 monozygotic (MZ) and 1,899 dizygotic (DZ) twin pairs were studied. They were asked about the frequency of sleep walking, both in childhood and in adulthood.

Results: For sleep walking in childhood, there was a concordance rate of 0.55 for MZ and 0.35 for DZ twin pairs. For adults, the concordance rate was 0.32 for MZ and 0.06 for DZ twin pairs. Less than 1% of those who reported never having walked in their sleep in childhood did so as adults. Of those who reported walking in their sleep often or sometimes in childhood, 25% of men and 18% of women reported sleep walking as adults. In adult sleep walkers, 89% of men and 85% of women had a history of sleep walking in childhood. The genetic influence was calculated as 66% in men and 57% in women in childhood sleep walking and 80% in men and 36% in women in adult sleep walking.

Conclusion: The greater similarities between MZ than between DZ twins and the figures linking childhood and adult sleep walking suggest that there is a substantial genetic effect in sleep walking, both in adults and in children.

Most of the activities carried out during sleep walking are unproblematic when a person is awake, but when they occur during somnambulism, they could be potentially dangerous to the sleep walker or other people. Although the risk is slight, cases have been reported where sleepwalkers have hurt themselves or others (see Box 1.13).

Box 1.13 Broughton et al. (1994)

The case of Ken Parks took place in Canada in the 1980s. He was married with a young daughter and got on well with his in-laws. However, as a result of anxiety about gambling problems he only slept between 4–6 hours a night. To settle his debts, he embezzled money from work, lost his job and was charged with theft. He and his wife agreed that he should tell his in-laws everything.

The night before this was due to happen, he fell asleep in front of the television and the next thing he claimed to remember was looking at his mother-in law's face and seeing a knife in his hands and that he was bleeding. He drove straight to the police station, where he said: 'I think I have killed some people.' The police reconstructed what must have taken place. He had got up from the sofa, driven 14 miles to the house of his parents-in-law, strangled (but not killed) his father-in-law and stabbed his mother-in-law to death.

He was acquitted of the murder and attempted murder, as it was argued that he had been sleep walking during the entire episode and therefore had not acted voluntarily. This claim was strengthened by the fact that his story never varied and because he seemed genuinely upset by what had happened.

This kind of incident is thankfully rare but, rather more worryingly, increasing numbers of '**sleep driving**' cases are being reported, in which somnambulists get in their cars and drive, sometimes long distances, ignoring lanes, stoplights and stationary objects and after waking up have no memory of what they did.

Sleep walkers rarely seek medical help, but if the sleep walking is associated with anxiety, it can be treated with **anxiolytic drugs**, such as benzodiazepines (for example Diazepam). In a small-scale case study, Kennedy (2002) found that the relaxation achieved in **hypnosis** could be effective in reducing anxiety and through this the frequency of sleep walking. It has the advantage of being a relatively simple, non-invasive and inexpensive treatment. Sleep walking may also be associated with depression, in which case **SSRIs** such as Prozac can be effective. Some chronic sleep walkers suffer from **sleep-disordered breathing (SDB)**; Guilleminault et al. (2005) report that if this is treated, either by nasal continuous positive airway pressure (CPAP) or surgery, the sleep walking stops completely.

Summary

- Sleep walking is relatively common in **children**, but affects only around 2% of **adults**.
- It is associated with **stress**, **depression** and **trauma**. There may also be some **genetic** basis.
- On rare occasions, sleep walkers may harm others. **Sleep driving**, however, is being increasingly reported.
- Sufferers may be treated with **anxiolytics**, **hypnosis**, **SSRIs** or — in the case of breathing difficulties — with **CPAP**.

Narcolepsy

Narcolepsy affects about 0.05% of the population, around 1 in 2,000 people. In narcolepsy, problems in the brain mechanisms that control sleep and waking allow REM sleep to break through into waking consciousness. Symptoms include excessive daytime sleepiness and sufferers may experience **cataplexy**; they suddenly lose muscle tone so their arms and legs go limp, or they may collapse or fall as though they have suddenly fallen asleep. However, they remain conscious, so it is as if they are asleep and awake at the same time. Cataplectic attacks can last for a few seconds or for several minutes and may occur several times a day. The attacks appear to be triggered by emotions, such as fear or amusement, or by sexual arousal. In contrast to the normal sleep cycle, where REM sleep does not occur for an hour or so after falling asleep, narcoleptics experience REM sleep soon after falling asleep. This limits the amount of NREM sleep they get, which accounts for their daytime sleepiness. They also experience sleep paralysis, either at the start or at the end of a night's sleep, which may be accompanied by hallucinations.

However, the symptoms experienced by narcoleptics vary considerably, for example in the amount of sleepiness they experience. Some have disabling cataplexy, whereas others have no cataplexy at all, or only rare episodes. Usually patients with unexplained sleepiness, sleep-onset REM, sleep paralysis and hallucinations are diagnosed as narcoleptic even if they do not experience cataplexy, and the same disease process seems to be at work, whether or not patients experience cataplexy.

There is a strong genetic component in the development of narcolepsy. De Lecea et al. (1998) identified two peptides, synthesised only in the hypothalamus and which are now referred to as **hypocretins**, hypocretin-1 and hypocretin-2, sometimes also referred to as **orexins**. They are derived from a single gene and the disorder is the result of mutations in the genes synthesising these peptides or their receptors;

Parkes and Lock (1989) report that the genetic defect in narcolepsy has been located on the short arm of chromosome 6.

Thannickal et al. (2000) proposed that damage to the hypocretin system might be the cause of narcolepsy, i.e. that hypocretin-producing cells in the lateral hypothalamus are selectively destroyed in people who are genetically susceptible. This was supported in their study that examined the dead brains of narcoleptics and compared them with the brains of controls. They found a huge loss of hypocretin neurons in the brains of narcoleptics — on average a 93% reduction — compared with controls. This suggests that giving injections of hypocretin could help to alleviate the symptoms and John et al. (2000) found it indeed to be effective in reducing cataplexy in dogs, providing it was given in a carefully judged amount and in a specific area.

However, a genetic abnormality is not necessarily expressed, in other words carrying a gene for narcolepsy does not necessarily mean that a person will develop the disorder. The extent of genetic influence in the development of narcolepsy has been explored (see Box 1.14).

Box 1.14 Ohayon et al. (2005)

Aim: To establish the extent of genetic influence on the development of narcolepsy.

Procedure: A survey was carried out, using telephone interviews of 157 narcoleptics, 263 of their first-degree relatives and a matched group of controls. In addition 68 spouses of narcoleptics were also surveyed.

Results: Among the first-degree relatives of narcoleptics, 10.8% were also narcoleptic, with a much lower rate among controls. They were also more at risk than controls for other sleep disorders, such as sleep talking and sleep apnoea.

Conclusion: Genes are an important factor in narcolepsy.

The influence of environmental factors on the development of narcolepsy has also been investigated (see Box 1.15).

Box 1.15 Picchioni et al. (2007)

Aim: To assess the effects of environmental factors on the development of narcolepsy.

Procedure: Participants were 63 narcoleptics and 63 non-narcoleptic controls. They completed a questionnaire to assess the frequency and timing of stressors and infections.

Results: Several stressors, including a major change in sleeping habits, carried a significant risk. Among the infectious diseases that were investigated, only flu infections and unexplained fevers carried a significant risk. For both kinds of risk factors, exposure before puberty increased the risk of developing narcolepsy.

Conclusion: Environmental factors are important in the risk of developing narcolepsy.

It has also been suggested that a high body mass index (BMI) and a compromised immune system may play a part in the development of narcolepsy. However, it is not clear whether the associations that have been found between these factors and narcolepsy are a result of the disorder, rather than a cause.

Summary

- **Narcolepsy** is a relatively rare disorder as a result of problems with the mechanisms of sleep and waking. Some narcoleptics also experience **cataplexy**.
- There is a strong **genetic** component related to peptides called **hypocretins**.
- **Environmental factors** such as **stress** are also important.