Chapter 1
Biological rhythms and sleep

SPECIFICATION

Biological rhythms and sleep	
Biological rhythms	• Circadian, infradian, and ultradian rhythms, including the role of endogenous pacemakers and of exogenous zeitgebers. • Consequences of disrupting biological rhythms, for example, shift work, jet lag.
Sleep states	• The nature of sleep. • Functions of sleep, including evolutionary explanations and restoration theory. • Lifespan changes in sleep.
Disorders of sleep	• Explanations for insomnia, including primary and secondary insomnia. • Factors influencing insomnia, for example, apnoea, personality. • Explanations for other sleep disorders, including sleep walking and narcolepsy.

BIOLOGICAL RHYTHMS: THE CIRCADIAN RHYTHM

Biological rhythms are cyclical changes in the way biological systems behave. The most obvious rhythm is the sleep-wake cycle – people and many animals go to sleep when it is dark and wake up when it is light. There are other many other rhythms, such as the opening and closing of flowers with daylight or the seasonal patterns of activity in hibernating animals. What controls these rhythms?

- Internal biological 'clocks', called **endogenous pacemakers**.
- External cues from the environment, called **exogenous zeitgebers**. These include sunlight, food, noise, or social interaction.

'Endogenous' means 'inside', whereas 'exogenous' means 'outside'.

The word 'zeitgeber' is from the German meaning 'time-giver'.

CAVE MAN

Michel Siffre is a specialist in the study of the human internal clock. On several occasions he has spent long periods of time living underground in order to study his own biological rhythms. Underground, in a cave, he had no external cues to guide his rhythms – no daylight, no clocks, no radio. He simply woke, ate, and slept when he felt like it. The only thing influencing his behaviour was his internal 'clock' or 'free-running rhythm'. Before Siffre's experiment nothing was known about these endogenous biological rhythms.

In his first underground sojourn of 61 days in the southern Alps in 1962, he resurfaced on 17 September believing the date was 20 August. On the second occasion he spent six months in a Texan cave (Siffre, 1975). His natural circadian rhythm settled down to just over 24 hours but sometimes this would change dramatically to as much as 48 hours. On his final underground sojourn in 1999, he was 60 years old. He was interested in the effects of ageing on biological rhythms. He found that his internal clock ticked more slowly than when he was a young man. He also found that his sleep patterns had changed (see page 4).

CIRCADIAN RHYTHMS

Circadian rhythms are those rhythms that last about 24 hours; the word circadian comes from the Latin 'circa' (about) plus 'dies' (a day). The two best-known circadian rhythms are the sleep-wake cycle and the body temperature cycle.

The sleep-wake cycle

You might think that the reason you go to sleep and wake up at fairly regular times is because of changes in daylight – you feel sleepy when it gets dark and are roused by sunlight streaming through your curtains; or perhaps your sleep-wake cycle is governed by knowing what time of day it is. These are external cues. Psychologists have investigated what happens when a person is free of such external cues using temporal isolation studies, i.e. the biological rhythm is allowed to be 'free running' and unaffected by external cues. One of the most memorable studies was conducted by the French cave explorer, Michel Siffre (see left). His experiences showed that the free-running cycle settles down to a regular rhythm which is a little more than 24 hours. His case study was supported by other studies. For example, Aschoff and Wever (1976) placed participants in an underground World War II bunker in the absence of environmental and social time cues. They found that most people displayed circadian rhythms between 24 and 25 hours, though some rhythms were as long as 29 hours.

These studies show that circadian rhythms persist despite isolation from natural light, which demonstrates the existence of an endogenous 'clock'. However this research also shows that external cues are important because the clock was not perfectly accurate: it varied from day to day.

Folkard *et al.* (1985) conducted an experiment to see if external cues could be used to override the internal clock. A group of 12 people lived in a cave for three weeks, isolated from natural light and other time cues. These volunteers agreed to go to bed when the clock indicated 11.45 pm and to get up when it indicated 7.45 am. Initially the clock ran normally, but gradually they quickened the clock until it was indicating the passing of 24 hours when actually only 22 hours had passed. At the beginning, the volunteers' circadian cycle matched the clock, but, as it quickened, their rhythm no longer matched the clock and continued to follow a 24-hour cycle rather than the 22-hour cycle imposed by the experiment (except for one participant who did adapt to the 22-hour cycle). Overall, this suggests that the circadian rhythm can only be guided to a limited extent by external cues.

An interesting feature of these experiments is that, as soon as the experiments are over, the participants take only a few days to resynchronise their cycles to the available external time cues (such as clocks and daylight), showing the importance of such external cues.

Core body temperature

Core body temperature is one of the best indicators of the circadian rhythm. It is lowest at about 4.30 am (about 36°C) and highest at around 6.00 pm (about 38°C). There is a slight trough just after lunch which is not just due to the effects of having had lunch – the dip occurs even when people have not eaten. In many countries the practice of having an afternoon siesta is related to this dip in body temperature. The temperature dip is a bi-daily rhythm, which is an example of the **ultradian rhythms** we will look at on the next spread.

Hormones

Hormone production also follows a circadian rhythm. **Cortisol** is at its lowest around midnight and peaks around 6.00 am. Cortisol is a hormone produced when we are stressed but is also related to making us alert when we wake up, and can explain why, if we awaken at 4.00 am, it is hard to think clearly. It is because cortisol levels are not sufficiently high for alertness. **Melatonin** (which induces sleepiness) and **growth hormone** are two other hormones that have a clear circadian rhythm, both peaking at around midnight.

▶ The French physicist Jacques d'Ortous de Mairan observed in 1729 that mimosa plants opened their leaves in the morning and closed them at night even when placed inside a box that let in no light. This is evidence of a biological clock in a plant.

WWW **Try some activities at http://faculty.washington.edu/chudler/clock.html, or a Daily Rhythm Test at www.bbc.co.uk/science/humanbody/sleep/crt/**

COMMENTARY

The sleep-wake cycle

Research methodology – Early research studies suffered from an important flaw when estimating the 'free-running' cycle of the human circadian rhythm. In all studies participants were isolated from variables that might affect their circadian rhythms such as clocks, radios and daylight. However they were not isolated from artificial light because it was thought that dim light, in contrast to daylight, would not affect the circadian rhythm. Recent research suggests that this may not be true; for example, Czeisler *et al.* (1999) altered participants' circadian rhythms down to 22 hours and up to 28 hours just using dim lighting.

Individual differences – There are two important types of individual difference. One is the cycle length; research has found that circadian cycles in different people can vary from 13 to 65 hours (Czeisler *et al.*, 1999). The other type of individual difference relates to cycle onset – individuals appear to be innately different in terms of when their circadian rhythms reach their peak. For example, Duffy *et al.* (2000) found that morning people prefer to rise early and go to bed early (about 6.00 am and 10.00 pm), whereas evening people prefer to wake and go to bed later (10.00 am and 1.00 am).

Core body temperature

The circadian variation in core body temperature has been linked to cognitive abilities. For example, Folkard *et al.* (1977) looked at the learning ability of 12- and 13-year-old children who had stories read to them at either 9.00 am or 3.00 pm. After one week, the afternoon group (higher core body temperature) showed both superior recall and comprehension, retaining about 8% more meaningful material. This suggests that long-term recall is best when body temperature is highest. Gupta (1991) found that performance on IQ tests was best at 7.00 pm as compared with 9.00 am or 2.00 pm, a factor which might be an important consideration when taking examinations.

There is evidence that temperature changes do actually cause the changes in cognitive performance. Giesbrecht *et al.* (1993) lowered body temperature (by placing participants in cold water) and found that cognitive performance was worse on some tasks.

However other research has found that the link is spurious. For example, Hord and Thompson (1983) tested cognitive performance in a field rather than lab situation and didn't find any correlation between core temperature and cognitive performance. It may be that higher core body temperature leads to increased physiological arousal and this leads to improved cognitive performance (Wright *et al.*, 2002).

THE BIOLOGICAL APPROACH

The studies on this page are typical of the biological approach to understanding behaviour: they propose that human behaviour can be explained in terms of structures in the brain and in terms of hormonal activity. However, human behaviour is often more complex than this because people can override biologically determined behaviours by making choices about what they do.

On the other hand, sometimes it may not be possible to override biological factors and biological rhythms may be a case in point. A powerful example of this was the study of a young man who was blind from birth and had a circadian rhythm of 24.9 hours. He was exposed to various exogenous zeitgebers such as clocks and social cues yet found great difficulty reducing his internal pace. This made it very difficult for him to function and, as a result, he had to take stimulants in the mornings and sedatives at night in order to get his biological rhythm in time with the rest of the world (Miles *et al.*, 1977).

REAL-WORLD APPLICATIONS

Chronotherapeutics (also called chronopharmacology) is the study of how timing (chronos) affects drug treatments (therapy). Since the circadian rhythm affects digestion, heart rate, hormone secretions and other functions, this should be taken into account when taking drugs. For example, medications that act on certain hormones may have no effect if taken when target hormone levels are low but are fully effective if taken when levels are high. Another example is taking aspirin to treat heart attacks (which normally occur in the early morning). This is most effective at around 11.00 pm which allows the aspirin to peak in the blood stream (this takes 2–4 hours). A further application of this research is when deciding on the best time to study. You are most alert in the morning and early evening, so those are the best times to work.

CAN YOU…? No.1.1

…**1** Write a description of circadian rhythms (a) in about 200–300 words and (b) in about 100–150 words.

…**2** Present **eight** detailed criticisms of research into circadian rhythms, including **at least two** topics from the synoptic toolkit (see introduction). Each criticism should be about 50 words. Remember that criticisms can be positive as well as negative.

…**3** Use all this material to write a 600-word answer to the question: *'Describe and evaluate research on circadian rhythms, with reference to endogenous pacemakers and exogenous zeitgebers.'* (9 marks + 16 marks)

VALIDITY

The study by Michel Siffre might be described as a case study – it is the study of one individual and therefore has unique features. His body's behaviour may not be typical of all people and, in addition, living in a cave may have particular effects due to, for example, the fact that it is cold. However, subsequent studies above ground have confirmed the findings of research in cave environments.

Siffre's study was also an experiment – he controlled key variables (exogenous zeitgebers) to observe the effects on the sleep-wake cycle. The experimental approach is important because it allows us to demonstrate causal relationships.

INFRADIAN AND ULTRADIAN RHYTHMS

The circadian rhythm isn't the only biological rhythm – there are two other important rhythms. The ultradian rhythm spans less than a day, one example is the bi-daily temperature rhythm described on the previous spread. The infradian rhythm has a period of more than one day but less than one year, such as the menstrual cycle in women.

ULTRADIAN RHYTHMS

Sleep stages

In humans, daily cycles of wakefulness and sleep follow a circadian rhythm. However, within the sleep portion of this cycle another type of rhythm, an example of an ultradian rhythm exists. These are the five stages of sleep (outlined below). The first four stages are called **NREM sleep** (non-random eye movements) and the fifth stage is **REM sleep** (rapid eye movement), so called because of the accompanying movements of the eye beneath the closed eyelids. One sleep cycle goes through all five stages and lasts about 90 minutes.

Stages 1 and 2 are light sleep, characterised by a change in the electrical activity of the brain. The awake brain produces a typical pattern called a *beta wave*. As you become more relaxed, your brain waves become slower and more regular, and have a greater amplitude. This is called an *alpha wave*. As you go to sleep, the waves slow down further i.e. have a greater wave frequency. This is called a *theta wave*, which is accompanied by bursts of activity – increased wave frequency (*sleep spindles*) and increased wave amplitude (*K complexes*). Stages 3 and 4 are characterised by even slower *delta waves*. These stages are called **slow wave sleep (SWS)**. In this stage it is very hard to wake someone up, though a person is not unconscious and will be aroused by, for example, their baby crying. In deep sleep (SWS) most of the body's physiological 'repair work' is undertaken and important biochemical processes take place such as the production of **growth hormones**. In REM sleep there is fast, desynchronised EEG activity resembling the awake brain.

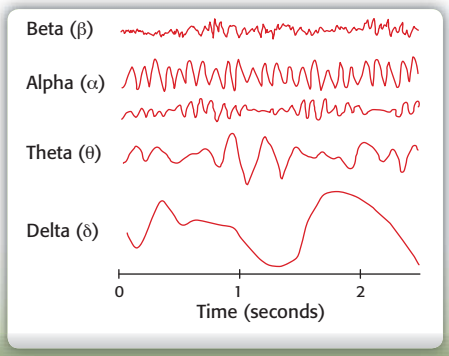
These cycles continue throughout the night with the SWS period getting shorter and REM periods getting slightly longer as the night progresses. Each sleep cycle is about 60 minutes in early infancy, increasing to 90 minutes during adolescence.

Basic rest-activity cycle

The 90-minute sleep cycle is itself located within a 24-hour circadian cycle, so it would make sense to find that this 90-minute 'clock' was also ticking throughout the day. It is called the **basic rest-activity cycle (BRAC)**. Friedman and Fisher (1967) observed eating and drinking behaviour in a group of psychiatric patients over periods of six hours. They detected a clear 90-minute cycle in eating and drinking behaviour.

NREM sleep	
Stages 1 and 2	Relaxed state, easily woken. Alpha and theta waves, heart rate slows, temperature drops.
Stages 3 and 4 SWS	Delta waves, metabolic rate slowest, growth hormone produced.
REM sleep	
REM	Called 'paradoxical sleep' because brain and eyes active but body paralysed.

▲ The 90-minute ultradian sleep rhythm – descending the 'sleep staircase'.



▶ The illustration shows EEG (electroencephalograph) recordings of characteristic brain waves. As a person goes to sleep their brain waves increase in amplitude (the height of the wave) and the wave frequency also increases (the distance between the crest of one wave and the next).

INFRADIAN RHYTHMS

Monthly cycles

The most obvious infradian rhythm is the human female menstrual cycle driven by fluctuating hormone levels. The function of the menstrual cycle is to regulate ovulation. The **pituitary gland** releases hormones (FSH – *follicle stimulating hormone*, and LH – *luteinising hormone*) which stimulate a follicle in one ovary to ripen an egg and also triggers the release of the female hormone **oestrogen**. Once the egg has ripened, the ruptured follicle starts to secrete **progesterone** which causes the lining of the womb to prepare for a pregnancy by increasing its blood supply. About two weeks after ovulation, if there is no pregnancy, progesterone is reduced and this causes the lining of the womb to be shed.

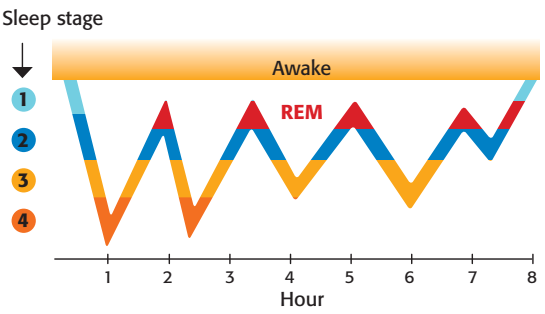
Less well known are the monthly rhythms affecting males. In one study 21 male participants had their body temperature and alertness levels measured over periods varying from 49 to 102 days. The study found some evidence in males for a periodic variation of both body temperature and subjective ratings of morning alertness, with a cycle length of approximately 20 days (Empson, 1977).

Seasonal affective disorder (SAD)

Infradian rhythms don't have to be monthly, they can also apply to behaviours that occur once a year. Some people suffer from a depressive condition called **seasonal affective disorder (SAD)**. They become depressed during the winter months and recover during the summer. We may all experience some lowering of mood when there is more darkness than daylight but SAD sufferers experience severe symptoms.

What causes this depression? Research studies have shown that the hormones **melatonin** and **serotonin** are secreted when it is dark (by the **pineal gland**); the more darkness, the more melatonin and serotonin. Research has also shown that these hormones may be a cause of chronic depression (see page 178).

▼ An illustration of the changing ultradian rhythm of sleep during the night.



'Chronobiology' is the study of biological rhythms.

A DETERMINIST APPROACH

On this spread we have described two psychological disorders which arise from infradian rhythms – PMS and SAD. PMS has been used as a legal defence – for example, in one case a Ms English drove her car into her married lover after an argument, killing him. She was charged with murder but ultimately placed on probation because it was argued in court that her actions were related to severe PMS (Johnson, 1987). Dr Katharina Dalton, a GP and pioneer researcher into PMS, has often acted as an expert witness and argued that severe PMS was akin to a mental disorder and therefore individuals should not be held responsible for their actions. This suggests that biological rhythms may be beyond our control.

On the other hand, there is evidence that we can 'will' our biological rhythms to change. One study found that people who were told to wake up at earlier times of the night than usual had higher levels of the stress hormone **ACTH** (which contributes to the waking-up process) than normal at the designated time and they woke up earlier (Born *et al.*, 1999).

COMMENTARY

Sleep stages

One issue with studies on REM sleep is the assumption that it is dreaming. Dement and Kleitman (1957) were the first to demonstrate this link. They woke participants up at the times when their brain waves were characteristic of REM sleep and found that participants were highly likely to report dreaming. However they also found that dreams were recorded outside REM sleep and that sleepers, when awoken in REM sleep, were not always dreaming. The importance of the REM/dream link is that it potentially provides a way to identify when someone is dreaming and therefore might provide theorists with a way to explain dreaming – for example Hobson and McCarley (1977) proposed that dreams are just are a psychological read-out of the random electrical signals typical of REM sleep. However such theories of dreaming are based on the erroneous assumption that REM activity = dreaming.

Basic rest-activity cycle

The basic rest-activity cycle shows that sleep stages are part of a continuum – a 90-minute cycle that occurs throughout the day within the circadian rhythm. The importance of this 90-minute rhythm is probably as a form of timing to ensure that the biological processes in the body work in unison, in the way that a conductor keeps an orchestra in time.

Menstruation

Exogenous cues – The menstruation cycle is normally governed by an endogenous system – the release of hormones under the control of the pituitary gland. However, it can be controlled by exogenous cues. Research has shown that when several women live together and do not take oral contraceptives, they tend to menstruate at the same time every month. In one study, daily samples of sweat were collected from one group of women and rubbed on to the upper lip of women in a second group. The groups were kept separate yet their menstrual cycles became synchronised with their individual odour donor (Russell *et al.*, 1980). This suggests that the synchronisation of menstrual cycles can be affected by **pheromones** – chemicals which are released in, for example, sweat. Pheromones act like hormones but are produced by someone else's body rather than your own.

Consequences of the menstrual cycle – Premenstrual syndrome (PMS) is a disorder that affects many women during the week before menstruation begins (when progesterone rises). Symptoms can include acne, anxiety, depression, food cravings, fatigue, headaches, insomnia, water retention, mood swings and aggression. For many years, PMS was dismissed as being only a psychological problem (it's all in your head), but we now know that it is a physiological problem (with psychological symptoms) caused mainly by hormonal changes related to the infradian rhythm. Dalton (1964) found that PMS was also associated with an increase in accidents, lower achievement academically, suicides and crime (see above).

Seasonal affective disorder

SAD has been explained in terms of being a natural outcome of infradian rhythms, but alternatively it could be the consequence of a disrupted *circadian* rhythm. In the UK, as the seasons change from summer to winter, the circadian rhythms may be thrown out of phase. People continue to get up at about the same time but often go to bed earlier because it is darker earlier. This means that the biological system gets the impression that time is shifting and the result is similar to jet lag, which is discussed later in this chapter.

REAL-WORLD APPLICATIONS

The understanding of the role of darkness in SAD has led to effective therapies, most notably the use of **phototherapy**, as illustrated below. This uses very strong lights in the evening and/or early morning to increase levels of melatonin and serotonin. The lights are between 6,000 and 10,000 Lux which is equivalent to full daylight; a 60 watt light bulb produces about 1000 Lux.

SAD sufferers have reported that daily use of such boxes is enough to relieve them of their feelings of lethargy, depression and other related symptoms. However there is some question about whether this may be due to a **placebo effect** (a belief that the therapy will work). One study found that a placebo condition (fake negative-ion generator) was less effective, but 32% of participants did improve with the placebo alone (Eastman *et al.*, 1998).

CAN YOU...? No.1.2

...1 Write a description, in about 100–150 words, of ultradian rhythms.

...2 Present **four** detailed criticisms of research into ultradian rhythms, including **at least one** topic from the synoptic toolkit (see introduction). Each criticism should be about 50 words.

...3 Write a description of infradian rhythms in about 150 words.

...4 Present **four** detailed criticisms of research into infradian rhythms, including at least one topic from the synoptic toolkit (see introduction). Each criticism should be about 50 words.

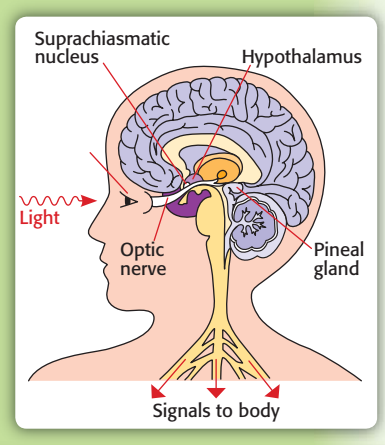
...5 Use all this material to write a 600-word answer to the question: '*Describe and evaluate research on ultradian and infradian rhythms*'. (9 marks + 16 marks)

...6 Are explanations of biological rhythms deterministic? To what extent is this a good or a bad thing?

ENDOGENOUS PACEMAKERS, EXOGENOUS ZEITGEBERS

The previous two spreads looked at different kinds of biological rhythms. Such rhythms have evolved for two reasons. First the world we live in has cyclic changes – day and night, summer and winter, and so on. Animals and plants need to be attuned to these changes and therefore they need to have internally-managed rhythms (endogenous pacemakers) that are similar to the likely cyclic changes in the environment. However these rhythms also need to be fine-tuned by external cues (exogenous zeitgebers) in order to remain in time with the external world which fluctuates in its rhythms (e.g. shorter days in winter).

The second reason is that biological organisms are complex systems where lots of different chemical processes are going on. To co-ordinate these processes you need something like the conductor of an orchestra to keep everything in time. This is another reason for having an internal clock, or endogenous pacemaker.



► Diagram of the brain showing the position of the suprachiasmatic nucleus and pineal gland.

ENDOGENOUS PACEMAKERS

The suprachiasmatic nucleus

In mammals, the main endogenous pacemaker is a tiny cluster of nerve cells called the **suprachiasmatic nucleus (SCN)**, which lies in the **hypothalamus**. It is located just above the place where the optic nerves from each eye cross over (called the optic chiasm – thus 'supra', which means 'above' the chiasm). The SCN obtains information about light from the eye via the optic nerve. This happens even when our eyes are shut, because light penetrates the eyelids. If our endogenous clock is running slow (e.g. the sun rises earlier than the day before), morning light automatically shifts the clock ahead, putting the rhythm in step with the world outside.

In fact each SCN is actually a pair of structures, one in each hemisphere of the brain, and each of these is divided into a ventral and dorsal SCN. The ventral SCN is relatively quickly reset by external cues, whereas the dorsal SCN is much less affected by light and therefore more resistant to being reset (Albus *et al.*, 2005).

The pineal gland and melatonin

The SCN sends signals to the **pineal gland**, directing it to increase production of the hormone **melatonin** at night. Melatonin induces sleep by inhibiting the brain mechanisms that promote wakefulness. In birds and reptiles the pineal gland lies just beneath the bone of the skull and is directly regulated by light; light inhibits the production of melatonin. In fact many lizards have a 'third eye' near the pineal gland which actually protrudes through a small opening in the skull and receives information about light.

EXOGENOUS ZEITGEBERS

The process of resetting the biological clock with exogenous zeitgebers is known as **entrainment**. The opposite of entrainment is 'free-running' – where the biological clock operates in the absence of any exogenous cues.

Light

Light is the dominant zeitgeber in humans. As we have seen, light can reset the body's main pacemaker, the SCN. It can also reset the other oscillators located throughout the body because the protein CRY (cryptochrome), which is part of the protein clock (see left), is light-sensitive. This may explain why Campbell and Murphy (1998) found that if you shine light on the back of participants' knees this shifted their circadian rhythms.

Social cues

Until fairly recently, biologists thought that social cues were the main zeitgebers for human circadian rhythms. We eat meals at socially determined mealtimes, and go to bed and wake up at times designated as appropriate for our age, and so on. Our daily rhythms appeared to be entrained by social convention, not internal biology. Today we know that light is the dominant zeitgeber, but in fact it is also now understood that all parts of the body produce their own oscillating rhythms and some of these are not primarily reset by light. For example, the zeitgeber for cells in the liver and heart is likely to be mealtimes because these cells are reset by eating (Davidson, 2006).

Temperature

Biological rhythms can also be entrained by temperature. For example, leaves on deciduous trees change colour and drop off because of changes in temperature as well as day length. Temperature is also a factor in the onset of hibernation. In the absence of light, temperature may be the dominant zeitgeber (López-Olmeda *et al.*, 2006).

THE TICKING OF THE BIOLOGICAL CLOCK

The basis of the circadian rhythm lies in interactions between certain proteins, creating the 'tick' of the biological clock; it is an ingenious negative feedback loop. Darlington *et al.* (1998) first identified such proteins in the fruit fly, *drospholia*.

- In the morning, two proteins, CLOCK and CYCLE (CLK-CYC) bind together.
- Once joined, CLK-CYC produce two other proteins, PERIOD and TIME (PER-TIM).
- PER-TIM has the effect of rendering the CLK-CYC proteins inactive, so that, as PER-TIM increases, CLK-CYC decreases and therefore PER-TIM starts to decrease too (negative feedback).

This loop takes about 24 hours and, hey presto, you have the biological clock! The actual proteins vary from animal to animal. In humans the main pairs are CLOCK-BMAL1 and PER-CRY (BMAL1 and CRY are also proteins).

This protein mechanism is present in the SCN (the central oscillator), and is also present in cells throughout the body (peripheral oscillators). The presence of peripheral oscillators explains why there are different rhythms for different functions such as hormone secretion, urine production, blood circulation and so on.

CUTTING-EDGE RESEARCH

New techniques have enabled researchers to study the behaviour of cells, molecules and genes, and produce an increasingly complex picture of how living organisms function. Chronobiologists now understand that endogenous rhythms are determined by a complex synchronised multioscillator system, but they are still trying to work out the distribution of these oscillators and the exogenous cues that affect them. There are enormous health, safety and economic benefits to figuring out how the circadian clock works because desynchronised body clocks reduce alertness and can lead to major accidents (see page 9).

COMMENTARY

Research evidence

Evidence for the role of endogenous and exogenous factors is apparent in many of the studies reviewed on the previous two spreads, and can be useful commentary on this topic.

The role of the SCN has been demonstrated in animal studies. Morgan (1995) bred 'mutant' hamsters so they had circadian rhythms of 20 hours instead of 24 hours, and then transplanted their SCNs into normal hamsters. The normal hamster then displayed the mutant rhythms.

Separate rhythms – Under normal conditions the central oscillator (the SCN) coordinates all other body rhythms, but in certain circumstances the body's separate oscillators will desynchronise. For example, a young woman, Kate Aldcroft, spent time in a cave. After 25 days her temperature rhythm was a 24-hour one, yet her sleep rhythm was on a 30-hour cycle (Folkard, 1996). Such desynchronisation leads to symptoms similar to **jet lag** – which is essentially a state of desynchronised biological rhythms (see next spread).

The power of artificial lighting

Light is the dominant zeitgeber, but the question is whether any lighting level will act as a zeitgeber. In the early studies of biological rhythms (e.g. Siffre and Aschoff and Wever), participants were exposed to artificial lighting but it was assumed this would not be bright enough to entrain rhythms. On the other hand Campbell and Murphy (see left) shifted circadian rhythms just by shining a light on the back of someone's knees. Recent research has shown that, in general, artificial lighting does have an effect. For example, Boivin *et al.* (1996) found that circadian rhythms can be entrained by ordinary dim lighting, though bright lighting was more effective (this study is described on the next spread).

If dim lighting does reset the biological clock, then the fact that we live in an artificially lit world may have some negative consequences. For example, Stevens (2006) suggests that exposure to artificial lighting disrupts circadian rhythms and thus disrupts melatonin production and this might ultimately explain why women in industrialised (and well-lit) societies are more likely to develop breast cancer.

When the biological system fails

The downside of a biologically-determined system is that, when it fails, it may cause a multitude of problems. One example of this is when there are mutations in the genes which contribute to the ticking of the biological clock. Familial advanced sleep-phase syndrome (FASPS) has been linked to an inherited defect in one of the PER genes (Chicurel, 2001). This syndrome typically causes sleep onset at around 7.00 pm, and spontaneous awakening at around 2.00 am in affected family members who therefore have great difficulty leading a normal life. There are many other sleep phase disorders; in fact some research suggests that brain changes during adolescence lead to a form of **delayed sleep phase disorder** which would explain why some adolescents have rather unusual sleep patterns (see page 10).

The blended system

It sounds as if we are talking about two systems – one endogenous and the other exogenous – but such neat divisions do not really exist. Apart from total isolation experiments, the running of the biological clock is a combined endogenous-exogenous exercise.

A CHIPMUNK'S TALE OF SURVIVAL

It is claimed that biological rhythms have adaptive value (an evolutionary approach to understanding behaviour). One adaptive advantage would be that the circadian clock enables tight temporal scheduling of physiological and behavioural programmes (Anton *et al.*, 2005). The other adaptive advantage would be that biological rhythms allow an animal (or plant) to anticipate daily environmental events, such as the patterns of light and dark. Patricia DeCoursey investigated this by functionally removing the SCN in 30 chipmunks (a lesion was made so that the SCN was no longer connected). The chipmunks were returned to their natural habitat and observed alongside two other groups of chipmunks – 24 surgical controls and 20 intact controls. After 80 days, significantly more of the SCN-lesioned chipmunks had been killed by weasels. This was presumably because these chipmunks remained awake in their burrows and the weasels could hear the noise and were able to locate the chipmunks (DeCoursey *et al.*, 2000).

NON-HUMAN ANIMAL RESEARCH

A lot of data reported on this spread is derived from experiments with animals. There are two issues to consider. First, the issue of harm to animals involved in the research (such as the chipmunks), and second, the issue of generalisability to humans. In terms of the first issue, if we accept that such research does have important applications to human behaviour, then the harm to animals may be considered acceptable as long as the key principles of non-human animal research are adhered to (see page 285). As regards the second issue, we do know that the systems differ from one animal to the next (for example reptilian biological rhythms have direct input to the pineal gland). Therefore it is important to check any animal findings against research with humans.

CAN YOU...? No.1.3

...1 Write a description, in about 100–150 words, of endogenous pacemakers, including research evidence.

...2 Write a description, in about 100–150 words, of exogenous zeitgebers, including research evidence.

...3 Present **eight** detailed criticisms of the methods used in researching endogenous pacemakers and/or exogenous zeitgebers, including **at least two** topics from the synoptic toolkit (see introduction). Each criticism should be about 50 words. Ensure that any synoptic points are fully contextualised.

...4 Present **two** comments about the advantages and/or disadvantages of biological rhythms.

...5 Use all this material to write a 600-word answer to the question: '*Discuss the role of endogenous pacemakers and exogenous zeitgebers in biological rhythms*'. (9 marks + 16 marks)

CONSEQUENCES OF DISRUPTING BIOLOGICAL RHYTHMS

Biological rhythms are driven by endogenous pacemakers (oscillators). As we have seen, some of these oscillators are easily reset by exogenous zeitgebers (such as daylight, mealtimes and so on) whereas other oscillators are more resistant. The result is desynchronisation. The two most common examples of the disruption of biological rhythms and the resultant desynchronisation are shift work and aeroplane travel – resulting in **shift lag** and **jet lag**.

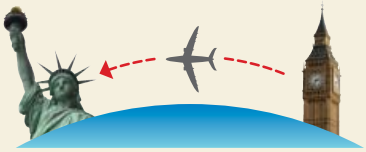
When you have a temperature it makes you feel quite ill. This may be another example of the disruption of biological rhythms, because raised body temperature entrains some oscillators causing desynchronisation (Herzog, 2003).

PHASE ADVANCE AND PHASE DELAY

The notion of phase advance and phase delay applies to all circadian disruptions, including both shift work and jet lag as well as simply staying up late and/or getting up early. In terms of jet lag, most travellers report less difficulty in adjusting when they are flying west (e.g. London to New York) than when they are flying east (e.g. New York to London). The diagram below illustrates what happens.

We all know that it is easier to stay up later than usual rather than to get up earlier than normal – if you have to get up early you feel slightly out of sorts and have difficulty concentrating. Why should phase delay (east to west travel or staying up late) be easier than phase advance? It is probably because phase delay means, on the first morning, you get up when your body is already quite awake – a bit like having a lie-in. On the other hand, with phase advance, you have to get up when you are in a circadian trough.

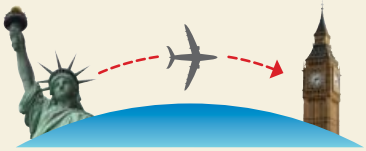
East to West Phase delay; easier (like going to bed later than usual)



New York
Arrive 6 hours later
London time = 10pm
Minus 5 hours time difference
New York time = 5pm

London
Leave 4pm

West to East Phase advance; more difficult (like having to get up earlier than usual)



New York
Leave 10pm

London
Arrive 6 hours later
New York time = 4am next day
Plus 5 hours time difference
London time = 9am

CONSEQUENCES OF DISRUPTION

Shift work and shift lag

Night workers are required to be alert at night and so must sleep during the day, which is the reverse of our natural rhythms and out of line with most of the available cues from zeitgebers.

Decreased alterness – Nightworkers often experience a circadian 'trough' of decreased alertness during their shifts (Boivin *et al.*, 1996). This occurs between midnight, when cortisol levels are lowest, and 4.00 am, when core body temperature is at its lowest.

Sleep deprivation – Workers who have to sleep by day often experience sleep problems because when they finish work it is daytime and there are other interruptions (e.g. noises outside) and daylight reduces sleep quality. Daytime sleep is typically between one and two hours shorter than a nocturnal sleep period; REM in particular is affected (Tilley and Wilkinson, 1982). Poor quality daytime sleep then makes it even more difficult for shift workers to stay awake through the night, especially when they hit the circadian trough.

Effects on health – There is a significant relationship between shift work and organ disease. For example, Knutsson *et al.* (1986) found that individuals who worked shifts for more than 15 years were three times more likely to develop heart disease than non-shift workers. Martino *et al.* (2008) linked shift work to a range of organ diseases including kidney disease. This may be due to the direct effects of desynchronisation or indirect effects such as sleep disruption.

Jet travel and jet lag

The term 'jet lag' is generally used to refer to the physiological effects of disrupted circadian rhythms – even those that do not arise from jet travel. Our biological rhythms are not equipped to cope with sudden and large changes; it is estimated that the dorsal portion of the SCN takes several cycles to fully resynchronise to abrupt large changes in environmental time – a process we experience as jet lag. Winter *et al.* (2008) calculated that this is equivalent to one day to adjust to each hour of time change. Symptoms of jet lag include loss of appetite, nausea, fatigue, disorientation, insomnia and mild depression.

Performance decrement – A popular way to demonstrate the debilitating effects of jet lag has been to study American major league baseball teams who have to travel from coast to coast to play league games. The west coast of America is three hours behind the east coast so, when east coast teams have to play on the west coast, they experience their clocks going backwards – a phase delay; west coast teams experience phase advance when they play teams on the east coast. In one such study Recht *et al.* (1995) analysed US baseball results over a three-year period. Teams that travelled east to west won (on average) 44% of their games; whereas, when travelling from west to east, the percentage of games won dropped to just 37%.

INDIVIDUAL DIFFERENCES

The effects of circadian disruption vary considerably between individuals. It is possible that those people whose circadian rhythms change least are the ones who cope best overall. Reinberg *et al.* (1984) found that people who gave up shift work because they couldn't cope tended to have rhythms that changed a lot while on shift while the 'happy shift workers' had unchanging rhythms.

▶ The *Exxon Valdez* oil tanker ran aground at 12.04 am in 1989, dumping more than 10 million gallons of oil into Prince William Sound, Alaska, and killing thousands of marine animals and sea birds. It was by no means the only major accident to occur in the middle of the night – the Chernobyl nuclear power station disaster began at 1.23 am, and Three-Mile Island nuclear power station accident at 4.00 am. Most lorry accidents occur between 4.00 am and 7.00 am. Moore-Ede (1993) estimated the cost of shift worker fatigue in the US to be $77 billion annually as a result of both major accidents and ongoing medical expenses due to shift work-related illnesses.

COMMENTARY

Our society cannot function without night working (and hence shift lag) and jet travel (and hence jet lag) so we need to understand the consequences in order to find ways to deal with such disruptions of biological rhythms, both to protect individuals and to protect all of us from associated accidents (see above).

Other factors

Shift work effects are not just due to the disruption of biological rhythms. They may be due to the lack of sleep associated with having to go to bed at unusual times. There are other factors too. One is that shift workers experience social disruption as well as disruption to their biological rhythms. It is difficult to meet friends and spend time with family when working on shift; divorce rates may be as high as 60% among all-night shift workers (Solomon, 1993).

Jet lag may also be affected by factors other than the disruption of biological rhythms. An individual may sleep badly the night before travelling because of worry. Travel itself is tiring and many holidays involve long hours getting to the airport before flying. Drinking alcohol or coffee, constant noise, low-oxygen cabin air and annoying passengers can be cumulative factors.

Reducing the harmful effects

Rotating shifts – Research indicates that more problems occur when people have to do rotating shifts, where shifts alternate every few days (Gold *et al.*, 1992). Non-fluctuating shifts (where an individual always works nights) are less disruptive because the individual can get used to one sleep-wake pattern – though days off are likely to mean temporary changes in sleeping patterns which will disrupt the biological rhythms.

Forward-rotating shifts follow the logical order of the day (phase delay) and may be easier on the body and less damaging to worker health, according to a review of research by Bambra *et al.* (2008). An example of such a shift pattern would be a shift in the morning for one week, then an afternoon shift the next week and finally a night shift for the third week. Bambra also concluded that rotating workers through shift changes more quickly (such as every three to four days, as opposed to every seven days) is better for health and work-life balance.

Artificial lighting is moderately effective in re-setting the rhythm, as found in the study by Boivin *et al.* (see top right). This study also found that dim lighting actually had the opposite effect (i.e. it didn't re-set the rhythnm)! Gronfier *et al.* (2007) were able to entrain circadian rhythms to longer than 24 hours just by using bright light pulses – modulated light exposure (MLE).

Melatonin has been put forward as a 'miracle' cure for shift lag and jet lag. This makes sense, because it is the natural hormone that induces sleep. Herxheimer and Petrie (2001) reviewed 10 studies and found that where melatonin was taken near to bed-time, it was remarkably effective. However, if taken at the wrong time of day it may actually delay adaptation.

Social customs can help to entrain biological rhythms. For example, when travelling, it helps to eat at the right time and go to sleep when the clock says it is time to go to sleep. Recent research suggests that a period of fasting followed by eating on the new time schedule should help entrain biological rhythms (Fuller *et al.*, 2008), possibly because some of our body clocks are reset by food intake.

LAB EXPERIMENTS

The strength of lab experiments, such as the one by Boivin *et al.* (top right), is that extraneous variables can be carefully controlled to isolate causal variables. However, there is a question over whether the same 'laws' will apply in everyday life. It is therefore important to conduct field experiments as well to confirm the findings. Boivin and James (2002) used intermittent bright lights in a field study of nurses which confirmed the effectiveness of bright lighting to promote circadian adaptation.

There is a further problem in generalising this research to everyday life – people who are willing to take part in psychology experiments are likely to be quite motivated, especially as they know their behaviour is being monitored. Less motivated shift workers may not do as well with light therapies.

RESETTING BIOLOGICAL CLOCKS

Boivin *et al.* (1996) investigated the power of artificial light in resetting our biological clocks. Thirty-one male subjects were divided into four groups and put on an inverted sleep-wake cycle for three days (kept awake at night and allowed to sleep during the day). Each 'day' when they woke they were exposed to five hours of very dim light, followed by one of four conditions: Group 1 was exposed to very bright light (10,000 lux), group 2 to bright light (1,260 lux), group 3 had ordinary room light (180 lux) and group 4 remained in dim light. Core body temperature was used to assess each person's current circadian rhythm.

After three days, members of group 1 (very bright light) had advanced five hours earlier; group 2 (bright light) had advanced by three hours; group 3 had advanced by one hour; and group 4 had drifted one hour *later* in their circadian rhythms. This shows that even room lighting can have an effect on the circadian rhythm and very bright light has a significant effect.

CAN YOU...? No.1.4

...1 Write a description, in about 100–150 words, of the *consequences* of shift work.

...2 Write a description, in about 100–150 words, of the *consequences* of jet travel (i.e. jet lag).

...3 Describe two studies related to the consequences of disrupting biological rhythms and present **two** detailed criticisms of the methods used in these studies.

...4 Present **four** critical points about the consequences of shift work and jet lag (e.g. how such consequences can be avoided). This should include including **at least two** topics from the synoptic toolkit (see introduction). Each criticism should be about 50 words.

...5 Use all this material to write a 600-word answer to the question: *'Discuss the role of endogenous pacemakers and exogenous zeitgebers in biological rhythms'*. (9 marks + 16 marks)

THE NATURE OF SLEEP AND LIFESPAN CHANGES

What is sleep?

- Sleep usually involves being very still. However, dolphins and other marine mammals are not still, they must come up to the surface regularly to breathe and, to do this, they sleep one hemisphere (half of the brain) at a time.
- Sleepers are usually quite unresponsive, though they are not unconscious. Most animals are woken by significant noises. For example, most parents will wake if they hear their baby crying.
- Animals usually sleep in a quiet, private, secure place and they usually do it lying down in the dark, although it is even possible to sleep on a busy underground train sitting up. Cows sleep standing up (except during REM sleep) and cats snooze in the sun.
- The time spent sleeping varies widely. The two-toed sloth sleeps for 20 hours whereas the giraffe sleeps only 3 hours a day.
- The most distinctive thing about sleep is that it is accompanied by a characteristic pattern of electrical activity in the brain (as described on page 4). However REM activity is absent in some animals, such as reptiles.

What does this tell us? There are no certain features of sleep, but all animals sleep and they all do it differently. This suggests two things: (1) sleep is necessary for survival (otherwise why would all animals do it?), and (2) the actual patterns of sleep are adaptive to each particular species (otherwise why are the patterns so different?). On the next two spreads we will look at explanations of the function of sleep, but here we start with a look at human sleep patterns over the human lifespan.

► *Night with her train of stars and her great gift of sleep* (by Edward Hughes, 1912)

THE DEVELOPMENTAL APPROACH

Developmental psychology considers human behaviour from the perspective of how it changes over the course of a person's lifespan. At one time developmental psychologists focused just on the changes leading up to adulthood, assuming that things stayed pretty much the same thereafter – but we can tell you they don't! Developmental psychology also focuses on averages and it is important to remember that there are significant individual differences at all ages.

LIFESPAN CHANGES IN SLEEP

As humans grow from infancy to old age there are major changes in the amount and kind of sleep experienced.

Infancy

Babies sleep a lot more than children and adults, and also have different sleep patterns and different stages of sleep. They tend to sleep about 16 hours a day, but their sleep is not continuous. They usually wake up every hour or so because their sleep cycles are shorter than the adult 90-minute cycle. Infants have sleep stages which are similar to adult stages, called quiet sleep and active sleep; these are immature versions of **SWS** and **REM** sleep respectively. At birth there is more active sleep than adult REM sleep; about half of infant sleep is spent in active sleep. Another difference relates to going to sleep; adults can usually go fairly directly into the state of deep sleep (quiet sleep), whereas infants in the early months enter sleep through an initial period of light sleep. After twenty minutes or more they gradually enter deep sleep.

By the age of six months a circadian rhythm has become established (one main sleep-wake cycle) and by the age of one year infants are usually sleeping mainly at night, with one or two naps during the day. The periods of deep sleep lengthen and there is a reduction in the amounts of active/REM sleep. It is not known whether REM activity is accompanied by dreaming, as babies and young children cannot provide reliable subjective reports.

Childhood

By the age of five children have EEG patterns that look like those of an adult but they are still sleeping more (about 12 hours per day) and having more REM activity (about 30% of total sleep time). Boys sleep slightly more than girls. During childhood it is not uncommon for children to experience a variety of **parasomnias** – sleep disorders such as sleep walking or night terrors. These are described on pages 16–19.

Adolescence

During childhood the need for sleep decreases but in adolescence it increases slightly, to about nine or ten hours a night. **Circadian rhythms** also change so that teenagers feel naturally more awake later at night and have more difficulty getting up early (a phase delay). One distinguishing feature of adolescent REM sleep is that in males it is sometimes accompanied by orgasm and ejaculation, which is significantly less likely at other ages.

Adulthood and old age

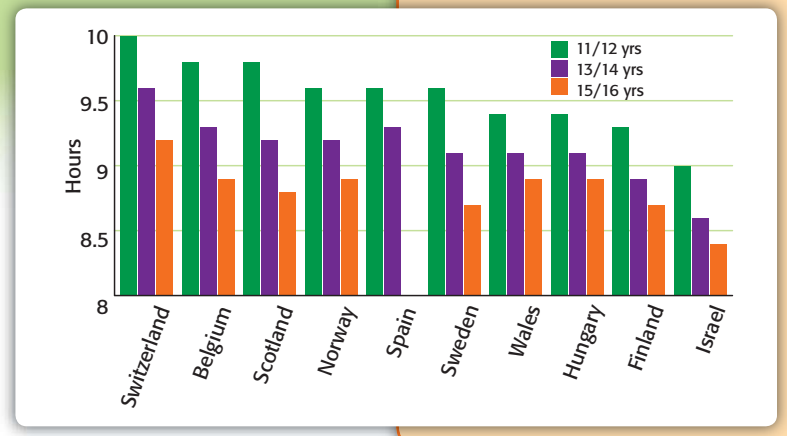
'Normal' adult sleep is typically for about eight hours per night, with 25% in REM sleep. Childhood **parasomnias** such as **sleep walking** are more rare in adulthood but there is an increasing frequency of other sleep disorders, such as **insomnia** and **apnoea** (see page 16).

With increasing age, total sleep time remains about the same, though older people have more difficulty going to sleep and wake up more frequently (up to six times a night). This means they may have a nap during the day to satisfy their sleep needs. Even more significant is the fact that the pattern of sleep changes; REM sleep decreases to about 20% of total sleep time and the amount of slow wave sleep is also considerably reduced to as little as 5% or even none (other kinds of NREM sleep increase). Older people also experience a phase advance of circadian rhythms – feeling sleepier early in the evening and waking up earlier.

CULTURAL DIFFERENCES IN SLEEP PATTERNS

The research reported on this spread shows that sleep patterns vary considerably with age but these patterns are also influenced by cultural values as well as lifestyle habits (such as consumption of alcohol and caffeine, amount of exercise taken, etc). A number of studies have recorded sleep duration in adolescents from different countries/cultures. For example, Tynjälä *et al.* (1993) surveyed 11–16-year-olds from 11 European countries; altogether over 40,000 children responded. Israeli children slept least (average of about 8.5 hours) and Swiss children slept most (average of about 9.5 hours). An important contributory factor was numerous evenings spent outside the home which meant the young people went to bed late and had less sleep because they had to get up early.

In Korea mean sleep time was recorded as about 6.5 hours for adolescents (Shin *et al.*, 2003) and a recent study in Iran found a mean time of 7.7 hours (Ghanizadeh et *al.*, 2008), both supporting the view that sleep duration is shorter in Asia than Europe.



◀ Graph showing mean sleep duration for most of the countries in the study by Tynjälä et al. (1993).

COMMENTARY

One general comment to be made is that there are significant individual differences at any age, as well as cultural differences (see above).

Newborn babies

Why are babies' sleep patterns so different from those of adults? One suggestion has been that babies' sleep is an adaptive mechanism to make their parents' life easier – daytime sleeping means that parents can get on with their chores which enhances survival. Nightwaking has adaptive benefits too. Babies have small stomachs and need to be fed regularly. A baby who has slept soundly through the night might not be woken by feeling cold or hungry.

Infants' greater amount of active/REM sleep may be explained in terms of the relative immaturity of the infant brain, and is related to the considerable amount of learning that is taking place. REM sleep has been linked to the production of neurotransmitters and to consolidation of memories. This explains why babies have a significantly greater amount of active/REM sleep. It is further supported by the fact that premature babies (whose brains are even less mature) spend 90% of their time in active sleep. Some psychologists even suggest that REM activity may present imagery which further stimulates the brain.

Adolescence

The change of sleep patterns in adolescence may be linked to changes in hormone production at this age. These hormones are primarily released at night and therefore sleep patterns are disturbed leading to sleep deprivation. Interestingly, many of the correlates of sleep deprivation are similar to those thought to be 'typical' of puberty: irritability, moodiness, changes in school performance and changes in motivation.

Hormonal changes can also explain the upset to the circadian clock, which has been described as a **delayed sleep phase syndrome** (Crowley *et al.*, 2007). In fact some researchers have recommended that schools should begin their day later to accommodate the poor attention spans of adolescents in the early morning (e.g. Wolfson and Carskadon, 2005).

Adulthood

The common perception is that a good night's sleep is related to good health, but several studies have found that there is an increased mortality risk associated with *too much* sleep. For example Kripke *et al.* (2002) surveyed over one million adult men and women and found that people sleeping for only six or seven hours had a reduced mortality risk, whereas those sleeping for an average of eight hours had a 15% increase in risk of death, and the risk was over 30% for people sleeping 10 hours. It is important to recognise that this is correlational data and there may be other intervening variables that cause the link between sleep duration and mortality. For example, underlying illness may lead to increased sleep needs and to increased mortality.

Old age

Reduced sleep in old age is partly a consequence of physiological changes but may also be explained in terms of actual problems staying asleep, such as sleep **apnoea** or medical illnesses. Problems staying asleep are also explained by the fact that deep sleep (**SWS**) is reduced in old age, so the older sleeper is more easily woken. The reduction in SWS leads to reduced production of **growth hormone** (because this is mainly produced in SWS), which may explain some of the symptoms associated with old age – such as lack of energy and lower bone density (van Cauter *et al.*, 2000).

The resulting sleep deficit in old age might explain why older people experience impaired functions, for example to their alertness. Various treatments can be used to increase sleep at night, including using relaxation techniques and taking **melatonin**.

There are useful applications of this research in finding ways to reduce the effects of ageing by improving sleep 'hygiene', i.e. improving the healthiness of sleep.

CAN YOU...? No.1.5

...1 Outline, in about 200–300 words, the lifespan changes in patterns of sleep.

...2 Select **one** study of lifespan changes in sleep and present **one** detailed criticism of the research methods used in this study.

...3 Present **six** critical points, including **at least two** topics from the synoptic toolkit (see introduction). Each criticism should be about 50 words.

...4 Use all this material to write a 600-word answer to the question: *'Describe and evaluate lifespan changes in sleep'*. (9 marks + 16 marks)

FUNCTIONS OF SLEEP: RESTORATION THEORY

Sleep must have some benefit, otherwise why would humans and other animals spend so much time sleeping? Animals cannot eat or mate while asleep, and are vulnerable to predation, so sleep must have some adaptive advantage or it would not be present in any animals whereas, in fact, it is found in virtually all animals. A likely explanation is that, during sleep, important biological functions take place, restoring the biological system to better working order; this is the basis of restoration theory which we will explore on this spread. This approach makes intuitive sense because most people feel refreshed after a good night's sleep.

The alternative view is that sleep actually has no specific benefit except to conserve energy or keep an animal safe from predators; this is regarded as the evolutionary approach which is considered on the next spread.

RESTORATION THEORY

Sleep is divided into several different stages, as we have seen earlier in this chapter. Two of the stages – **slow-wave sleep (SWS)** and **REM sleep** – are associated with particular benefits. Oswald (1980) proposed that these each had different functions – SWS enables body repair and REM enables brain recovery.

Slow wave sleep (SWS)

Growth hormone is secreted during SWS. Growth hormone (GH) stimulates growth and is therefore particularly important during childhood. It is also important in adulthood because it enables protein synthesis and cell growth to take place. This is vital in the restoration of body tissue because proteins are fragile and must be constantly renewed. This constant restoration of vital proteins is part of the body's natural recovery process.

GH is secreted in pulses through the day but a significant amount is released at night and mainly during SWS. Sassin *et al.* (1969) found that, when sleep-waking cycles are reversed by 12 hours (i.e. a person goes to sleep in the morning and gets up at night), the release of GH with sleep is also reversed. This shows that GH release is controlled by neural mechanisms related to SWS. Further evidence comes from research which found that the amount of GH released correlates with the amount of SWS (van Cauter and Plat, 1996) and the decline of GH in older age has also been associated with reduced SWS (van Cauter *et al.*, 2000).

The immune system – Lack of SWS has also been associated with reduced functioning of the immune system – the body's system of defence against viruses and bacteria (Krueger *et al.*, 1985). The immune system consists of various protein molecules – antibodies – which are regenerated during cell growth and protein synthesis in SWS.

Have you seen that weedy little rat we're using to test our new growth hormone?

REM sleep

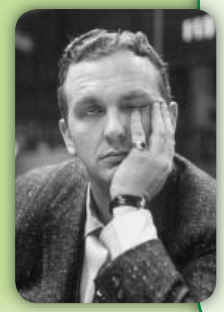
Brain growth – As we saw on the previous spread, the percentage of active/REM sleep is far higher in babies than adults, and even higher in premature babies. This has been explained in terms of their rapid brain growth. It has been suggested that the amount of REM sleep in any species is proportional to the immaturity of the offspring at birth; for example the platypus is immature at birth and has about eight hours of REM sleep per day, whereas the dolphin, which can swim from birth, has almost no REM sleep (Siegel, 2003). This suggests a relationship between neural development and REM sleep.

Neurotransmitter activity may be affected by REM sleep. Siegel and Rogawski (1988) suggest that REM sleep allows for a break in neurotransmitter release which in turn permits neurons to regain their sensitivity and allow the body to function properly. Support for this comes from the action of antidepressant drugs such as **MAIO**s. These drugs aim to increase the levels of neurotransmitters of the monoamine group (such as **dopamine** and **serotonin**). A side effect is that MAOIs abolish REM activity completely. One suggestion is that these two effects are linked – the increase in monoamines means that monoamine receptors don't have to be revitalised and therefore there is no need for REM sleep.

REM sleep and memory – For a long time psychologists have proposed a link between memory and REM sleep. For example Crick and Mitchison (1983) proposed that during REM sleep, unwanted memories are discarded thus making more important memories accessible. A recent explosion of research on sleep and memory has found a more complex relationship between memory and sleep (Stickgold, 2005). The evidence currently suggests that REM may be important in the consolidation of procedural memory (related to skills such as riding a bicycle), whereas SWS sleep is important for the consolidation of semantic memory (related to knowledge and the meaning of things) and episodic memory (memory for events).

Did you know that alcohol suppresses REM sleep? This might explain why people often feel very tired after a night out drinking, despite having apparently had plenty of sleep – the lack of sufficient REM sleep leads to REM deficit and tiredness.

STUDIES OF TOTAL SLEEP DEPRIVATION



In order to raise money for charity, American DJ, Peter Tripp, stayed awake for a total of 201 hours, even managing to perform live during his 'wakeathon'. Three days into the experiment Tripp became unpleasant and abusive, and after five days he began to hallucinate (seeing spiders in his shoes) and to become paranoid (believing people were drugging his food). Throughout the experiment Tripp showed a continuous decline in body temperature, and by the end his waking brain-wave patterns were virtually indistinguishable from those of a sleeping person. After 24 hours of sleep, Tripp awoke and reported himself feeling perfectly normal.

In 1965, Tripp's record was shattered by a 17-year-old American student, Randy Gardner, who managed to stay awake for an astonishing 260 hours (11 days). Unlike Tripp, he displayed no significant psychotic symptoms during his epic period of wakefulness and, like Tripp, he appeared perfectly normal after a lengthy sleep.

There are reports of individuals who have gone without sleep for years. For example a Vietnamese man, Hai Ngoc, is reported to have stopped sleeping altogether in 1973 with no apparent ill effects.

COMMENTARY

The effects of sleep deprivation

If sleep has an important restorative effect, then sleep deprivation should have clear consequences. The findings provide mixed support for this, suggesting that some kinds of sleep (REM sleep and SWS) are critical but that this is not true of all sleep

The effects of total sleep deprivation – The data has tended to be fairly anecdotal and based on individual case studies, such as those described above. The studies suggest that lack of sleep doesn't always result in long-term damage and that there is no need to recover anything like the amount of sleep that was lost. However, when participants have been deprived of sleep for more than 72 hours while being closely monitored, they invariably had short periods of **microsleep** while apparently awake. EEG recordings show that microsleep is the same as sleep (Williams *et al.*, 1959). It could be that apparent 'non-sleepers' are in fact getting the benefits of sleep while appearing to be awake.

Various studies of non-human animals suggest that sleep deprivation may have fatal consequences. For example, Rechtschaffen *et al.* (1983) forced rats to remain physically active by rotating a disc that they were standing on every time the rat started to go to sleep. After 33 days all sleep-deprived rats died. It is possible that stress rather than lack of sleep was the direct cause of death. However, Rattenborg *et al.* (2005) conducted a similar experiment with pigeons and the pigeons suffered no ill effects.

The effects of partial sleep deprivation may lead to what is called '*REM rebound*' – the need for more REM sleep after a night deprived of REM sleep. When a person is simply deprived of sleep there is not the same kind of rebound effect; it appears to be solely related to REM and SWS. To achieve REM sleep deprivation, researchers wake sleeping volunteers as soon as their eyes start to dart about. The result is that people show an increased tendency to go into REM sleep when they go back to sleep and, on recovery nights, the proportion of time spent in REM sleep increases, with this REM rebound being as much as 50% higher than normal (Empson, 2002).

SWS rebound effects have been demonstrated using acoustic stimulation which suppresses all SWS sleep by arousing a participant whenever their EEG appears to be going into deep sleep/SWS (Ferrara *et al.*, 1999).

CASE STUDIES

The study of sleep deprivation is generally restricted to case studies or observational studies of a small group of participants. The main difficulty with such studies is that participants are likely to be unique. For example someone who has difficulty sleeping is less likely to volunteer for studies, and people who do volunteer are probably highly motivated to try to cope well with sleep deprivation.

Other research into sleep deprivation has used animals because of the obvious ethical issues in depriving people of sleep. Animals have different sleep requirements and different sleep patterns from humans, and therefore such research may not be directly generalisable to humans.

Exercise and the need for sleep

A second consequence of 'sleep as restoration' is that increased physical exercise should lead to increased sleep in order to restore the proteins and biochemicals used, which appears to be the case in some studies. Shapiro *et al.* (1981) found that runners in a marathon race slept for about an hour more on the two nights following the race. SWS increased in particular, which fits the view that NREM sleep appears to be more associated with physical recovery.

However, in general, research has found that intense exercise does little more than make you fall asleep faster. For example, Horne and Minard (1985) gave participants numerous exhausting tasks to see if this increased their sleep duration, but it didn't. The participants went to sleep faster than usual but not for longer.

Comparative studies

Even though sleep is universal throughout the animal kingdom, sleep needs vary considerably. For example, EEG studies of dolphins have found no evidence of REM sleep; if REM sleep is vital to restoration then why don't dolphins need it as well? Like dolphins, fur seals sleep one hemisphere of the brain at a time when at sea, and have no REM. When back on land they switch to sleep patterns similar to those of other small mammals.

All this leads us to question why it is necessary to become almost unconscious (i.e. sleep) in order for these vital biological processes to take place. Young (2008) suggests that the more we know about the sleep patterns of other species, the more it becomes apparent that environmental pressures rather than restoration provide the key to understanding sleep, as we will see on the next spread.

CAN YOU...? No.1.6

...1 Outline, in about 200–300 words, why restoration is the function of sleep.

...2 Select **three** studies related to restoration theory and (a) state the conclusions that can be drawn from these studies and (b) give one methodological criticism of each study.

...3 Outline **two** arguments supporting restoration theory and two arguments against restoration theory.

...4 Use all this material to write a 600-word answer to the question: *'Outline and evaluate restoration theory as an explanation of the function of sleep'*. (9 marks + 16 marks)

FUNCTIONS OF SLEEP: EVOLUTIONARY EXPLANATIONS

We know that sleep must be adaptive in some way, otherwise why do all animals do it despite substantial costs? Either it provides some vital biological function, as explored on the previous spread, or it provides some other benefit. Evolutionary explanations aim to suggest what other benefits might be associated with sleep. The evolutionary approach has also been called the **ecological approach**. It is called 'ecological' because it is based on observations of animals in their natural environment; 'ecology' is the study of animals in relation to their environment.

The evolutionary approach to explaining behaviour is outlined in the introduction.

EVOLUTIONARY (ECOLOGICAL) EXPLANATIONS

Energy conservation

Warm-blooded animals (mammals), such as ourselves, need to expend a lot of energy to maintain a constant body temperature. This is particularly problematic for small animals with high metabolic rates, such as mice (metabolism refers to the chemical processes taking place in the body). All activities use energy, and animals with high metabolic rates use even more energy. Sleep, however, serves the purpose of providing a period of enforced *inactivity* (therefore using less energy) much as hibernation is a means of conserving energy. Webb (1982) described this as the **hibernation theory** of sleep.

Foraging requirements

If sleep is a necessity, the time spent sleeping may be constrained by food requirements. An animal has to gather food. Herbivores, such as cows and horses, spend their time eating plants (such as grass) that is relatively poor in nutrients. As a result, they must spend a great deal of time eating and consequently cannot 'afford' to spend time sleeping. Carnivores, such as cats and dogs, eat food that is high in nutrients, and so do not need to eat continuously. Therefore they can 'afford' to rest much of the time, and by resting they can conserve energy.

Predator avoidance

A further likelihood is that sleep is constrained by predation risk. If an animal is a predator, then it can sleep for longer, whereas for prey species, their sleep time is reduced as they must remain vigilant to avoid predators. Logically, to be safe they shouldn't sleep at all but if sleep is a vital function then they are best to sleep when least vulnerable.

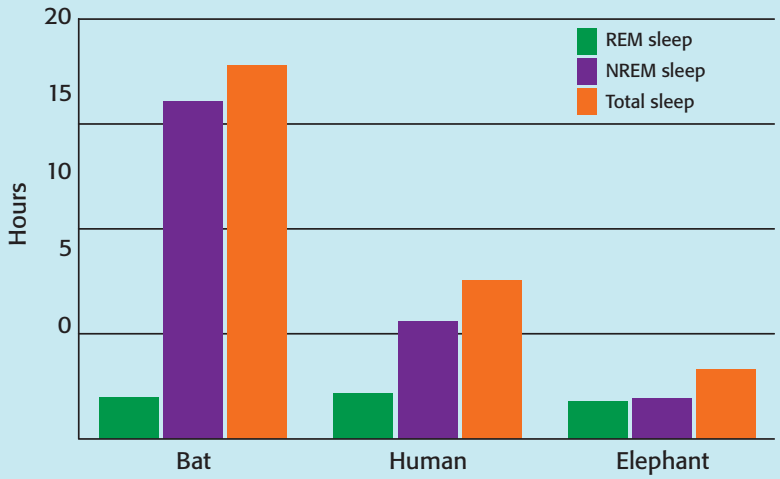
Waste of time

Meddis (1975) was the first to propose the 'waste of time' hypothesis. He suggested that sleep helps animals to stay out of the way of predators during the parts of the day when they are most vulnerable. For most animals, this means sleeping during the hours of darkness. It also means sleeping in places where they will be hidden. According to Meddis, sleep may simply ensure that animals stay still when they have nothing better to do with their time.

Siegel (in Young, 2008) concurs with this view and points out that in fact, being awake is riskier than sleeping because an animal is more likely to be injured. Siegel's view, based on what we currently know about sleep patterns (see 'Cutting Edge'), is that the only possible explanation for sleep is that it enables both energy conservation and keeping an individual out of danger: 'in the wild, the best strategy for passing on your genes is to be asleep for as long as you can get away with … and that is exactly what you see' (Young, 2008). For example, the little brown bat is awake for a few hours each day, just when the insects that it lives on are awake. It might be expected that the bat would sleep little because it is a small mammal – but it doesn't. It is awake when it needs to be.

CUTTING EDGE

We are finding out about animal sleep habits all the time. Young (2008) reports that out of 5000 mammal species we have information about the sleep patterns of less than 150. Even this knowledge is incomplete. For example we do not have reliable evidence of the different amounts of REM and SWS sleep, and of sleep patterns at different ages.



◀ Graph showing sleep patterns in different animals from the Phylogeny of Sleep project database.

The **Phylogeny of Sleep project** (see www.bu.edu/phylogeny/) aims to collect data from different sources to help future understanding of sleep. The database is available for anyone to search, and highlights some of the problems with the data, e.g. it is not always reliable. In some cases there is data for one species from a number of different studies and the records are conflicting – for example the various studies of giraffes rate NREM sleep somewhere between 1.5 and 3.6 hours and REM sleep somewhere between 0.4 and 1.0 hour. The problem is that often the data is based on studies of fewer than five animals and their sleep has been studied under lab conditions where they may not be displaying natural behaviour. The animals may also have only been observed for 12 hours. The database includes information about whether data was collected in the wild or in a lab, and also rates the quality of the lab research. Until we have a fuller and more accurate record of animal sleep patterns it won't be possible to fully understand the evolution of sleep.

UNILATERAL SLEEP

It isn't just sleep that is adaptive but also the pattern of sleeping. Sleep patterns differ from one species to another (though they are similar in genetically-related species). Such differences tell us something about the different **selective pressures** facing a particular species, with different sleep patterns being the adaptive response.

One example of an adaptive response is **unilateral sleep** – one hemisphere of the brain being asleep while the other is awake. This form of sleep has evolved in some marine mammals and also in migrating birds to cope with particular selective pressures. Dolphins must swim to the surface every time they need to take a breath. A dolphin that fell into a deep sleep (**SWS**) while underwater, would drown. The two hemispheres of the brain swap over about every two to three hours (Mukhametov, 1987). Migrating birds must remain awake for long periods of time when they are migrating and therefore unilateral sleep is important.

The fact that unilateral sleep has separately evolved in both of these groups of animals shows that it is a means of solving the evolutionary pressures facing these animal groups.

▲ Mallard ducks sleep with one eye open. Recordings of brain activity show that the brain hemisphere corresponding to the open eye is awake, while the hemisphere for the closed eye is in a deep sleep.

COMMENTARY

Comparative research

Energy, foraging or predation? One way to investigate the comparative costs and benefits of sleep is to compare sleep habits across different species. Zepelin and Rechtschaffen (1974) found that smaller animals, with higher metabolic rates, sleep more than larger animals. This supports the view that energy conservation might be the main reason for sleep. However there are many exceptions, such as sloths, which are very large yet sleep for 20 hours a day.

Allison and Cicchetti (1976) found that species who had a higher risk of predation did sleep less, though again there were exceptions, such as rabbits who had a very high danger rating yet slept as much as moles who had a low danger rating.

However, recent research by Capellini *et al.* (see right) suggests that the energy conservation hypothesis may be wrong, whereas the foraging and predator avoidance explanations are right.

REM and NREM sleep – When considering energy conservation there may be an important distinction between **NREM** and **REM** sleep. Interestingly the energy consumption of the brain drops only in NREM sleep; during REM sleep the brain is still relatively active. This leads to the view that it is only NREM sleep that has evolved for energy conservation, which was supported by Allison and Cicchetti (1976) who found that larger animals had less NREM sleep but not less REM sleep. This shows that it is NREM sleep that is important to energy conservation. However the data from Capellini *et al.* found no correlation between body size and NREM sleep.

There is a further argument for the REM/NREM distinction. Animals that are more 'primitive', such as most reptiles, only have NREM sleep. REM sleep appears to have evolved about 50 million years ago in birds and mammals. It might be that NREM sleep evolved first for energy conservation, whereas REM sleep may have evolved later to maintain brain activity. This is supported by the greater need for REM sleep in infants whose brains are developing. However, again this hypothesis is not supported by Capellini *et al.* (on the right).

A combined approach

The evolutionary approach fails to address some of the key aspects of sleep, such as why we have such a strong drive for sleep when sleep-deprived. Perhaps the resolution lies in a combined approach which recognises that some elements of sleep are for restoration whereas other aspects of sleep behaviour are related to the function of occupying unproductive hours e.g. conserving energy in small mammals.

Horne (1988) proposed a theory that combines elements from both restorative and adaptive theories. He suggested a distinction between **core** and **optional sleep**. Core sleep is equivalent to SWS sleep and is the vital portion of sleep that an organism requires for essential body and brain processes. Optional sleep (REM sleep and some portions of NREM sleep) is dispensable. Horne believes that optional sleep has the function of occupying unproductive hours and, in the case of small mammals, of conserving energy.

MAMMALIAN SLEEP

Capellini and her research team (2008) argued that previous research was flawed because the methods used to collect data on sleep in different animals were not standardised and therefore comparisons between species were meaningless. They carefully selected data from studies using only standardised procedures (e.g. animals habituated to lab conditions). The study focused on only land mammals because unilateral sleep in aquatic mammals involves different sleep patterns.

- They found a negative relationship between body mass and sleep (i.e. smaller animals slept more) which doesn't support the energy conservation hypothesis.
- However this data supports the view that there is a trade-off between sleep and foraging – greater foraging requirements create a restraint on time available for sleeping.
- The relationship between predation risk and sleep is a complex one. Animals that sleep in exposed positions sleep less, but time spent sleeping is also reduced in species that sleep socially – yet they ought to be able to sleep longer because there is safety in numbers.

Perhaps the key piece of evidence for the evolutionary approach is the existence of a strong **phylogenetic signal** for sleep among mammals. 'Phylogenetic signal' means the behavioural similarities between species that are close on the phylogenetic scale (i.e. are genetically closely related). Research has found that mammalian species that are genetically close have more similar sleep patterns than one would be expected by chance (Capellini *et al.*, 2008).

CAN YOU...? No.1.7

...1 Provide an outline, in about 200–300 words, of how evolutionary explanations might account for the function of sleep.

...2 Select **three** studies related to evolutionary explanations theory and (a) state the conclusions that can be drawn from these studies and (b) give **one** methodological criticism of each study.

...3 Outline **two** arguments supporting evolutionary explanations of sleep, and **two** arguments against.

...4 Use all this material to write a 600-word answer to the question: *'Discuss evolutionary explanations of the function of sleep'*. (9 marks + 16 marks)

SLEEP DISORDERS: EXPLANATIONS FOR INSOMNIA

A sleep disorder is any condition that involves difficulty experienced when sleeping. Such disorders often result in daytime fatigue, causing severe distress and impairment to work, and social or personal functioning. Sleep disorders can be classified as:

- Problems with falling asleep or staying asleep despite the opportunity to do so, i.e. insomnia.
- Problems staying awake, such as narcolepsy.
- Problems adhering to a regular sleep schedule due to, for example, shift work or jet travel.
- Sleep disruptive behaviours, called **parasomnias** (behaviours that occur during or around sleep) such as sleep walking, night terrors and bruxism (teeth grinding).

Insomnia affects at least 10% of the adult population, making it one of the most common psychological health complaints. Sleep deficits associated with insomnia create serious health risks such as falling asleep while driving and accidents in the workplace.

INSOMNIA

Insomnia is not defined in terms of the number of hours of sleep a person has because there are large individual differences in the amount of sleep that is 'normal' for each person and also because there are age-related differences, as we have seen. Some people who have very little sleep suffer no ill consequences whereas others who have many hours of sleep a night may feel unrefreshed and complain of insomnia. Both length and efficiency of sleep are important. Insomnia may involve trouble falling asleep (initial insomnia), trouble remaining asleep (middle insomnia) or waking up too early (terminal insomnia).

Insomnia is classified as either transient (short-term), intermittent (occasional) and chronic when it is constant and long-term (having occurred for one month or more).

Causes of insomnia

Insomnia may be either secondary to another disorder or a disorder in its own right i.e. primary.

Secondary insomnia is where there is a single, underlying medical, psychiatric or environmental cause. In such cases insomnia is a symptom of the main disorder i.e. it is secondary. For example insomnia is a characteristic symptom of illnesses such as depression or heart disease. It is also typical of people who do shift work or who have **circadian rhythm** disorders such as phase delay syndrome. Insomnia may also be the result of environmental factors such as too much caffeine (coffee, tea or even chocolate) or alcohol.

Primary insomnia describes cases where insomnia simply occurs on its own, with no known cause, for more than one month (**DSM** definition). In such cases insomnia is the person's primary problem. The individual may be feeling stressed or depressed but such psychological states are not the main problem. It may be that the individual has developed bad sleep habits (e.g. staying up late or sleeping in a room that is too light) and this has created insomnia, but insomnia is the only problem. Sometimes insomnia may have had an identifiable cause which has now disappeared, but the insomnia persists because of an *expectation* of sleep difficulty.

Risk factors influencing insomnia

There is an enormous range of factors which may influence the occurrence of insomnia, some of which, such as excessive caffeine intake, have already been mentioned.

Age and gender – Older people and women are more likely to suffer from insomnia. In older people, increasing physical problems, such as arthritis or diabetes, may disrupt sleep and lead to insomnia. It is possible that the increased incidence of insomnia in women is due to hormonal fluctuations, which are age-related, e.g. menopause.

Sleep apnoea or other **parasomnias** – Apnoea is a disorder where a person stops breathing while asleep. The pauses may last from a few seconds to minutes and may occur 5–30 times an hour, thus having a major disruptive affect on sleep. Other parasomnias include snoring, **sleep walking**, and teeth grinding. All such parasomnias increase the likelihood that a person will experience insomnia.

Personality may be a factor in insomnia. For example in one study it was found that insomniacs were more likely to internalise psychological disturbance rather than acting out problems or being aggressive (Kales *et al.*, 1976). The researchers in this study proposed that internalisation leads to higher levels of emotional arousal and increased likelihood of feeling anxious, and it is this that is a risk factor for insomnia.

COMMENTARY

Causes of insomnia

It is important to distinguish between primary and secondary insomnia because of the implications for treatment. If insomnia is a symptom of another disorder then it is important to treat the disorder rather than the insomnia. So, for example if insomnia is the result of chronic depression it would be unhelpful to simply treat the symptom.

However, recent research casts doubt on whether insomnia is just an effect. It may be the cause. A study of almost 15,000 Europeans found that insomnia more often *preceded* than followed cases of mood disorder (Ohayon and Roth, 2003). This means that, in some cases, it might be helpful to treat insomnia regardless of whether it is a primary or secondary effect.

Risk factors influencing insomnia

When discussing risk factors, Spielman and Glovinsky (1991) propose a useful distinction between predisposing, precipitating and perpetuating components. Predisposing factors include a genetic vulnerability for insomnia. Evidence for a genetic link comes from twin studies, for example in one such study Watson *et al.* (2006) found that 50% of the variance in the risk for insomnia could be attributed to genetic factors. Research also suggests that physiological factors may predispose a person to develop insomnia. For example it has been found that insomniacs are more likely to experience hyperarousal (high physiological arousal) both when awake and asleep (e.g. Bonnet and Arand, 1995). Hyperarousal would make it more difficult to get to sleep. Such factors explain why only some people develop insomnia in response to stress, jet lag and so on.

However predisposing factors alone are unlikely to explain chronic primary insomnia – the **diathesis-stress model** of mental disorders also proposes that vulnerability alone is not enough for a disorder to develop; environmental stressors are needed to trigger the disorder. In the case of insomnia, stress or environmental change may trigger episodes of insomnia.

Finally, perpetuating factors are important i.e. factors which maintain insomnia when the original causes (such as stress) have disappeared or been treated. Perpetuating factors include being tense when going to bed because of previous sleep problems. Espie (2002) suggests that such perpetuating factors are the key to chronic insomnia.

Treatment of insomnia

Treatments focus on those factors which perpetuate or maintain insomnia. Recommendations for treatment include relaxation techniques and improving sleep hygiene. 'Sleep hygiene' refers to increasing the healthiness of your sleep habits, such as reducing caffeine intake, sleeping in a darker room, cutting down on daytime naps and increasing exercise. Paradoxically some habits people develop to cope with the sleepiness arising from insomnia then block recovery e.g. taking naps because of tiredness, doing less exercise because of lethargy, drinking coffee, and so on.

Bright light therapy can be used for circadian disruption. Sedatives, anti-anxiety drugs and melatonin can be used, though these are only recommended as short-term solutions. One side effect of such drugs is daytime drowsiness.

More psychological techniques can also be used, including attribution therapy (see right), sleep restriction therapy and reconditioning. Sleep restriction involves restricting sleep to a few hours per night and gradually increasing this until a normal night of sleep is experienced. In reconditioning, the insomniac learns to associate their bed with just sleep, and should only go to bed when feeling very sleepy.

TEENAGE INSOMNIA

Rachel is in high school. She feels exhausted all day and goes to bed at 10.00 pm to try to get some much-needed sleep. She reads, she writes, she gets up again. Finally at around 1.00 am she goes to sleep. Rachel is a typical example of a teenage insomniac (Kalb, 2008). Her sleep patterns suggest that her insomnia may be due to the shift in circadian rhythms which is typical of the teenage years – circadian phase disorder.

A recent study by Roberts *et al*. (2008) found that teenage insomnia is a major problem, as common as substance abuse and depression, but given less publicity. They analysed data from over 4,000 adolescents, aged between 11 and 17, from Houston, Texas, and found that 25% of the young people had symptoms of insomnia and 5% reported that their lack of sleep interfered with their ability to function during the day. In a follow-up study of those teenagers with symptoms of insomnia, 41% were found to still have symptoms one year later. It is possible that some teenagers turn to drugs as a means of dealing with their sleep problems.

RESEARCH COMPLICATIONS

Chronic insomnia is highly complex and unlikely to be explained by one single factor. The large number of factors makes it very difficult to conduct meaningful research because studies find only small effects.

In addition there are so many different causes for insomnia – stress, depression, poor sleep hygiene, age, gender and so on, that it is very difficult to draw any firm conclusions from research.

REAL-WORLD APPLICATION

One of the causes of primary insomnia is a person's belief that they are going to have difficulty sleeping. Such an expectation becomes self-fulfilling because the person is tense when trying to sleep. One clever way to treat this is a method based on **attribution theory**. The insomniac has learned to *attribute* their sleep difficulties to 'insomnia'. If they can be convinced that the source of their difficulty lies elsewhere this will end their maladaptive attribution. In one study, insomniacs were given a pill and told either that the pill would stimulate them or act as a sedative. Those who expected arousal actually went to sleep faster because they attributed their arousal to the pill and therefore actually relaxed (Storms and Nisbett, 1970)!

CAN YOU...? No.1.8

...1 Provide an outline, in about 200–300 words, of explanations for insomnia, including the factors that influence it.

...2 Select **two** research studies and state the conclusions that can be drawn from them, including a comment on their methodological strengths or weaknesses.

...3 Present **four** further critical points including **at least two** topics from the synoptic toolkit (see introduction). Each criticism should be about 50 words.

...4 Use all this material to write an answer of 600 words in total to the following questions:

(a) *Distinguish between primary and secondary insomnia.* (2 marks + 3 marks)

(b) *Discuss explanations of insomnia.* (7 marks + 13 marks)

EXPLANATIONS FOR OTHER SLEEP DISORDERS

On this page we examine two other sleep disorders. **Insomnia** may result from behaviours which disrupt sleep, such as narcolepsy and sleep walking. **Narcolepsy** is a disorder where individuals experience sudden and uncontrollable attacks of sleep at irregular and unexpected times which may last seconds or minutes. **Sleep walking** (somnambulism) is a term that covers a range of activities which take place while sleeping but are normally associated with wakefulness (such as eating, getting dressed or walking about); the person has no conscious knowledge of what they are doing.

REAL-WORLD APPLICATION

One of the things about research is that you never know when it may prove to have a real-world application. In the case of sleep walking there have been occasional cases where expert testimony is required in order to be able to decide whether a person was actually sleep walking, for example cases of murder where the accused has claimed that the act was committed while sleep walking. One such case occurred in October 2003 when a Manchester man, Jules Lowe aged 32, attacked and killed his 82-year-old father. He claimed that he had no recollection of the attack because he was sleep walking at the time.

Dr Irshaad Ebrahim, director of the London Sleep Centre, was called in to establish whether what Mr Lowe claimed was true, based on what research has told us about sleep walking.

Tests were conducted by observing Lowe while he slept. Lowe had a history of sleep walking but he had never been violent like this previously. The tests showed that he was indeed prone to sleep walking and he was diagnosed with *insane automatism* (non-insane automatism is linked to external factors such as drinking). He was found 'not guilty' due to insanity and sent to a psychiatric hospital for an indefinite period of time.

Adapted from http://news.bbc.co.uk/1/hi/england/manchester/4337309.stm

You can see examples of narcolepsy – in dogs, babies and adults – on Youtube.

NARCOLEPSY

The two main symptoms of narcolepsy are feeling sleepy all the time and episodes of *cataplexy* (loss of muscular control) during the day. Such episodes seem to be triggered by various forms of emotional arousal such as anger, fear, amusement, or stress. Other symptoms include hallucinations and sleep paralysis, both experienced when falling asleep or waking up, and interruption of night-time sleep by frequent waking.

Narcolepsy usually begins in adolescence or early adulthood, and continues throughout life. Estimates suggest that about 1 in 2000 people are sufferers, though this may be an underestimate because many cases go undiagnosed since some people have only minor symptoms.

Explanations

REM – Over the last 50 years a variety of explanations for narcolepsy have been put forward. In the 1960s the view was that it was linked to a malfunction in the system that regulates **REM sleep**, which explained some of the classic symptoms of narcolepsy, such as the lack of muscle tone (cataplexy) which accompanies REM sleep and the intrusion of REM-type sleep (hallucinations) into daytime sleep.

HLA – In the 1980s, research appeared to indicate that narcolepsy was linked to a mutation of the immune system. Honda *et al.* (1983) found increased frequency of one type of HLA (human leukocyte antigen) in narcoleptic patients. HLA molecules are found on the surface of white blood cells and coordinate the immune response.

Hypocretin – More recently, research has uncovered a link between the **neurotransmitter hypocretin** (also called orexin) and narcolepsy. Hypocretins appear to play an important role in maintaining wakefulness. The first evidence came from narcoleptic dogs who had a mutation in a gene on chromosome 12, which disrupted the processing of the hypocretin (Lin *et al.*, 1999).

SLEEP WALKING

Sleep walking (SW) is a disorder that is most common in childhood, affecting about 20% of children and less than 3% of adults (Hublin *et al.*, 1997). In severe cases (one or more episodes a night) it may have a considerable effect on a person's life and there may be a risk of injury (see left). SW occurs only during **NREM/SWS** sleep and is related to **night terrors** which are also only found in NREM sleep and also most common in children. A sleep walker is not conscious and later has no memory of events during sleep walking.

Explanations

Incomplete arousal – Sleep walking is a disorder of arousal. EEG recordings made during sleep walking show a mixture of the *delta waves* which are typical of SWS, plus the higher frequency *beta waves* which are characteristic of the awake state. It looks as if SW occurs when a person in SWS is awakened, but the arousal of the brain is incomplete. It is likely that this abnormal arousal is genetic.

Various factors appear to increase the likelihood of SW – such as sleep deprivation, alcohol, having a fever, stress or psychiatric conditions (Plazzi *et al.*, 2005). Hormonal changes during puberty and menstruation may also be triggers for SW.

Why children? A range of explanations have been offered to explain why SW is more common in childhood. One possibility is that it happens because children have more SWS than adults. A recent suggestion by Oliviero (2008) is that the system that normally inhibits motor activity in SWS is not sufficiently developed in some children, and also may be underdeveloped in adults. This was demonstrated in a study that examined the motor excitability of adult sleep walkers during wakefulness. Compared to normal controls, the sleep walkers showed signs of immaturity in the relevant neural circuits.

COMMENTARY

Psychological explanations – Most explanations of narcolepsy have been biological, though there have been some unsuccessful attempts to provide psychological explanations. For example, Lehrman and Weiss (1943) suggested that sudden attacks of sleepiness disguise sexual fantasies.

The REM hypothesis was first proposed after Vogel (1960) observed REM sleep at the onset of sleep in a narcoleptic patient. This explanation was further supported by recordings of neuron activity in the brainstem of narcoleptic dogs that showed that cataplexy is linked to the activation of cells that in normal animals are active only during REM sleep (Siegel, 1999). However, in general, research support has not been convincing.

The narcolepsy-HLA link continues to be researched, though the specific HLA variant (HLA-DQB1*0602) found most commonly in narcoleptics is not found in all narcoleptics and is also reasonably common in the general population (Mignot *et al.*, 1997). This means that HLA cannot be the sole explanation.

Hypocretins are the most promising lead. The findings from narcoleptic dogs have been confirmed in human studies. For example, it was found that human narcoleptics had lower levels of hypocretin in their cerebrospinal fluid (Nishino *et al.*, 2000). However low levels of hypocretin are unlikely to be due to inherited factors because human narcolepsy doesn't run in families and it has not been found to be concurrent in twins where one has the disorder (Mignot, 1998). The reduction in hypocretin may be due to brain injury, infection, diet or stress, or is possibly the result of an auto-immune attack (where the body's immune system turns on itself rather than fighting external infection). This could explain the HLA link with narcolepsy because of the role of HLA in the immune response (Mignot, 2001).

▲ A time-lapse image of a woman sleep walking. Sleep walking is a term that covers any activity characteristic of the awake state, such as sitting up in bed, getting dressed, looking out of a window and so on. In other words it doesn't just involve walking.

A MEANS OF DIAGNOSING SLEEP WALKERS

A recent study experimentally demonstrated that sleep deprivation can trigger sleep walking behaviours and might provide a means of diagnosing the condition in vulnerable individuals. Zadra *et al.* (2008) studied 40 patients who were referred to a sleep lab for suspected SW, 15 of whom reported having injured themselves while sleep walking. The sleep walkers were observed while they slept and SW behaviours – such as playing with the bedsheets, sitting up and walking around – were counted. The sleep walkers were observed during one normal night's sleep and then returned to the sleep lab for a second night where they were prevented from falling asleep. After 25 hours without sleep they were allowed to sleep. On the first night 50% of the sleep walkers had showed signs of SW (total 32 episodes). After sleep deprivation this rose to 90% of the sleep walkers (total 92 episodes). Sleep deprivation does not lead to SW in normal individuals and therefore appears to be characteristic of individuals who are genetically predisposed to sleep walking.

COMMENTARY

The explanation of sleep walking (SW) can neatly fit into the **diathesis-stress** model. The diathesis (vulnerability) comes from a genetic predisposition to the disorder. Unlike narcolepsy there is strong evidence that SW has a genetic basis. For example Broughton (1968) found that the prevalence of SW in first-degree relatives of an affected subject is at least 10 times greater than in the general population. Twin studies have also been used; Lecendreux *et al.* (2003) report about 50% **concordance** in identical (MZ) twins compared to 10-15% in DZ twins, and also have identified a gene which may be critical in SW as well as *night terrors* (the DQB1*05 gene).

The stress part of the diathesis-stress model is represented either by maturity in certain key neural circuits in the brain or by the amount of slow wave sleep (SWS) experienced. One reason put forward for the increased incidence of SW in childhood is the greater amount of SWS. The same is true for the other risk factors mentioned – sleep deprivation, alcohol and fever all increase SWS and this may trigger SW. Pressman *et al.* (2007) suggest more research is needed to understand the link between factors such as alcohol and SW because voluntary intoxication has been used as a defence in criminal trials for rape and other crimes.

CAN YOU…? No.1.9

…1 Describe, in about 100–150 words, explanations of narcolepsy.

…2 Describe, in about 100–150 words, explanations of sleep walking.

…3 For each explanation of narcolepsy provide:

(a) one or more research studies that support that explanation, and

(b) two criticisms (strength or weakness) of each study.

…4 Do the same as in question 3 for the explanations of sleep walking.

…5 Use all this material to write a 600-word answer to the question: *'Outline and evaluate explanations of **two or more** sleep disorders'.* (9 marks + 16 marks)

CHAPTER SUMMARY

BIOLOGICAL RHYTHMS

CIRCADIAN RHYTHMS

SLEEP-WAKE CYCLE
- Circadian = 24 hours.
- Cycle persists despite isolation from light.

COMMENTARY
- Participants not isolated from artificial light in early research.
- Cycle length varies in individuals.

CORE BODY TEMPERATURE
- Lowest at 04:30; highest at 18:00.
- Post-lunch dip, even without food.

COMMENTARY
- Linked to cognitive abilities (Folkard *et al.*, 1977).
- Other research suggests link is spurious.

HORMONES
- Cortisol – lowest at midnight.
- Melatonin and growth hormone highest at midnight.

COMMENTARY
- Application – chronotherapeutics.

SYNOPTIC LINKS
- Biological approach = greater explanatory power.
- However, human beings have choice.
- Siffre (1975) – a case study *and* an experiment.

INFRADIAN AND ULTRADIAN RHYTHMS

ULTRADIAN RHYTHMS
- Less than one day.
- Sleep stages: First four stages = NREM.
- Fifth stage = REM.
- BRAC = 90 minutes within 24-hour rhythm.

COMMENTARY
- REM does not equal dreaming.
- BRAC important because it ensures biological processes work in unison.

INFRADIAN RHYTHMS
- Female menstrual cycle regulates ovulation.
- Males have 20-day cycle of body temperature and alertness.
- SAD – caused by melatonin during winter months.

COMMENTARY
- Menstrual cycle also subject to exogenous cues.
- Some women suffer from PMS.
- SAD could also be consequence of disrupted circadian rhythms.

SYNOPTIC LINKS
- PMS and SAD represent determinist approach.
- Evidence that rhythms can change through willpower (Born *et al.*, 1999).

ENDOGENOUS PACEMAKERS AND EXOGENOUS ZEITGEBERS

ENDOGENOUS PACEMAKERS
- SCN – main endogenous pacemaker.
- SCN contains protein mechanism.
- Pineal gland controls melatonin secretion.

COMMENTARY
- SCN evidence – 'mutant' hamsters (Morgan, 1995).
- Desynchronisation leads to symptoms similar to jet lag.

EXOGENOUS ZEITGEBERS
- Light is dominant zeitgeber.
- Social cues also important.
- Biological rhythms can be entrained by temperature.

COMMENTARY
- Artificial lighting may also reset biological clock.
- Failure of biological clock leads to sleep-phase disorders.
- Biological clock is really a blend of endogenous and exogenous factors.

SYNOPTIC LINKS
- Biological rhythms have adaptive value (SCN lesions in chipmunks – DeCoursey *et al.*, 2000).
- Non-human animal studies evaluated in terms of ethics and relevance.

SLEEP STATES

LIFESPAN CHANGES

CHILDREN
- Babies sleep 16 hours a day, but not continuous.
- Babies have shorter sleep cycles than adults.
- Circadian rhythm established at six months.
- Age 5, sleep patterns like those of adults, but sleep longer.
- May experience parasomnias.

COMMENTARY
- Sleep differences in babies – adaptive (for parents), and due to immature brain.

ADOLESCENCE
- Need for sleep increases (9–10 hours a night).
- Circadian rhythms change – slight phase delay.

COMMENTARY
- Changes may be linked to hormone production.
- Implications for school day.

ADULTHOOD AND OLD AGE
- Increase in sleep disorders (e.g. insomnia).
- Pattern of sleep changes in old age (reduction of REM and SWS).
- Older people experience phase advance effect.

COMMENTARY
- Increased mortality rate with too much sleep (Kripke *et al.*, 2002).
- Sleep deficit in old age may explain impaired functioning in other areas.

SYNOPTIC LINKS
- Developmental perspective important in the highlighting of changes across lifespan.
- Sleep patterns may also reflect cultural differences (Tynjälä *et al.*, 1993).

RESTORATION THEORY

SWS
- SWS = Stages 3 and 4.
- Growth hormone (GH) secreted during SWS.
- Decline of GH in older age because reduced SWS.
- Lack of SWS = poor immune functioning.

COMMENTARY
- Total sleep deprivation studies suggest no long-term damage, but studies often anecdotal (e.g. Peter Tripp).
- Total sleep deprivation fatal in rats (Rechtschaffen *et al.*, 1983), but not in pigeons (Rattenborg *et al.*, 2005).
- Partial deprivation leads to REM rebound.

SYNOPTIC LINKS
- Studies of sleep deprivation mainly case studies.
- Problems of individual differences and volunteer bias.

SLEEP DISORDERS

CONSEQUENCES OF DISRUPTION

SHIFT WORK AND SHIFT LAG

- Nightworkers experience 'trough' of decreased alertness.
- Sleep deprivation due to sleeping problems during day.
- Relationship between shift work and organ disease.

COMMENTARY

- Shift work effects not solely due to disruption of biological rhythms.
- More problems with rotating shifts, but forward rotating less harmful.
- Artificial lighting can reset rhythm (Boivin *et al.*, 1996) but not dim lighting (Gronfier *et al.*, 2007).

JET TRAVEL AND JET LAG

- Jet lag caused by disruption of circadian rhythms.
- Phase delay less disruptive than phase advance.
- Demonstrated in performance decrement studies (e.g. Recht *et al.*, 1995).

COMMENTARY

- Jet-lag symptoms may be caused by other factors associated with air travel.
- Melatonin may reduce symptoms.
- Social customs (e.g. eating at right time) also help.

SYNOPTIC LINKS

- Individual differences exist in coping with disruption.
- Strength of laboratory experiments – control of extraneous variables.
- But, volunteer bias in sample.

INSOMNIA

CAUSES

- Transient, intermittent and chronic insomnia.
- Secondary insomnia – symptom of an underlying disorder.
- Primary insomnia – occurs on its own, e.g. because of bad sleep habits.

COMMENTARY

- Different types, different treatments.
- Insomnia may be cause or effect – implications for treatment.

RISK FACTORS

- Age and gender – older people and women more likely to suffer.
- Sleep apnoea and other parasomnias.
- Personality – insomnia linked to tendency to internalise problems.

COMMENTARY

- Difference between predisposing factors (e.g. genetics); precipitating factors (e.g. environmental stressors); and perpetuating factors (e.g. tension from sleep problems).

SYNOPTIC LINKS

- Difficult to conduct meaningful research because of multiplicity of causal factors.
- Treatment using attributional retraining (cognitive therapy).

OTHER SLEEP DISORDERS

NARCOLEPSY

- Triggered by emotional arousal.
- Early explanations – failure of REM regulation.
- May be caused by increased frequency of HLA in immune system.
- Link between narcolepsy and hypocretin.

COMMENTARY

- Psychological explanations largely unsuccessful.
- REM hypothesis – some research evidence but not convincing.
- HLA variant cannot be sole explanation because common in general population.
- Hypocretins most promising explanation.

SLEEP WALKING (SW)

- Most common in childhood, affecting 20% of children.
- Person wakes during SWS but brain arousal incomplete.
- Other factors – sleep deprivation, alcohol, hormone changes.
- May affect children more because underdeveloped SWS inhibition.

COMMENTARY

- Diathesis-stress model.
- Diathesis – genetic link supported by Lecendreux *et al.* (2003).
- Stress – leads to e.g. sleep deprivation, which increases SWS.

SYNOPTIC LINKS

- Real-world application – accepted as defence in some crimes.
- Experimental study useful for diagnosing SW in vulnerable individuals (Zadra *et al.*, 2008).

RESTORATION THEORY cont'd

REM

- Important for brain growth.
- Important for restoring neurotransmitter sensitivity.
- Link between REM and procedural memory.

COMMENTARY

- Sleep-as-restoration view supported by Shapiro *et al.* (1981).
- Intense exercise leads only to faster sleep onset, not increased duration.
- Some animals have no REM so cannot be vital for restoration.
- Results from animals may not generalise to humans.

EVOLUTIONARY EXPLANATIONS

ENERGY CONSERVATION

- Sleep provides a period of inactivity to conserve energy, essential for animals with high metabolic rates.

FORAGING

- Herbivores spend less time sleeping; carnivores more, because food rich in nutrients.

PREDATOR AVOIDANCE

- Sleep constrained by predation risk. Predators sleep more, prey less.

WASTE OF TIME

- Sleep may simply be a way of staying still at times when an animal cannot forage and would be subject to predation.

COMMENTARY

- Evidence suggests claim that species with a higher metabolic rate sleep more (Zepelin and Rechtschaffen, 1974).
- Evidence suggests that species with higher predation risk sleep less (Alison and Cicchetti, 1976).
- NREM evolved first for energy conservation, then REM to maintain brain activity.
- Supported by Capellini *et al.* (2008) research.

SYNOPTIC LINKS

- Evolutionary approach does not explain why we need sleep when sleep-deprived.
- Horne suggests distinction between core and optional sleep, a combined approach.

EXAM QUESTION WITH STUDENT ANSWER

Question **Discuss the consequences of disrupting biological rhythms.** *(9 marks + 16 marks)*

STUDENT ANSWER	EXAMINER COMMENTS
There are three kinds of biological rhythm: circadian rhythms that occur around a day such as the sleep-wake cycle; ultradian rhythms that occur more than once a day, such as the stages of sleep and infradian rhythms that occur with less frequency, such as the menstrual cycle. All these rhythms are governed by a combination of internal mechanisms and external cues.	This introductory paragraph is a form of scene-setting but not actually relevant to the question here. It is best to avoid wasting precious examination time.
Many aspects of modern-day life disrupt these rhythms and cause considerable problems. It is reckoned that most major industrial accidents are caused at night which is due to the fact that people are not fully awake because they are doing shift work. For example the Bhopal accident occurred at night and so did the Exxon Valdez disaster. It is also true that many car accidents occur at night.	The second paragraph describes some of the consequences of disrupting biological rhythms. It may read like **AO2** because it is about consequences but, in the case of this essay, consequences are the main narrative (**AO1**).
One of the ways to study the consequences of disruption is to look at the effects of shift work on body rhythms. When people work shifts they usually do a shift for a few days at a time. One effect of this is that shift workers become sleep deprived because they have to sleep during the day when it is light outside and more noisy. Another effect of shift working is that people become ill, for example they develop heart disease. In one study they found that individuals who worked shifts for more than 15 years were three times more likely to develop heart disease.	This paragraph contains further description which is **detailed and accurate**. Notice, at the end of the paragraph, how a research study has been used as part of the description. No names have been given for this study; such information increases the **'detail'** of the answer (an important **AO1** criterion) but there is no penalty for omitting names.
However the effects of shift work may be due to factors aside from the disruption of biological rhythms. For example shift work disrupts the pattern of family life. People who work shifts are sleeping when everyone else is awake and this may cause stress in the family. Some of the effects of shift work may be reduced if rotating shifts are used, which is an example of phase delay. A further way to improve the effects of shift work is to use pulses of bright light, as shown in a study by Gronfier et al. (2007) to entrain circadian rhythms and therefore avoid longer periods of desynchronisation.	The consequences of shift work are evaluated by considering other explanations for the effects, and also considering methods of reducing some of the negative effects. The commentary provided is **sound** and the material is **coherently elaborated** (**AO2** criteria). The **line of argument** could be made clearer if specific links were made, for example pointing out how stress in the family might lead to heart disease.
Jet travel, like shift work, is likely to disrupt biological rhythms and lead to desynchronisation which is experienced as jet lag. The symptoms of jet lag include feeling disoriented, nauseous, tired or depressed. In fact these same symptoms are felt whenever the body's clocks are desynchronised. Jet lag is caused when a large change of time zones means that some of the body's clocks change with the exogenous cues such as daylight, whereas other body clocks are slower to change (though they could be changed by strong light).	Jet travel and jet lag offer a chance to further describe the consequences of disruption and desynchronisation, demonstrating a good **understanding** of a **range of relevant material**.
Evidence to demonstrate the effects of these changes comes from travelling baseball teams in America. Some of them travel from the east coast to the west coast, or vice versa, to play games. According to the belief that phase delay should be easier we would expect the teams that have a delay to do better – the teams that go from east to west. This is what the study found. This is a natural experiment, which means that we cannot draw causal conclusions from it. However it is supported by other research.	In this paragraph research has been used as commentary on the consequences, providing **AO2** material. It is important to focus on what this research shows rather than getting too bogged down in the details of the actual study. More **AO2** opportunities can be taken by considering the methodology of the studies which is also important evidence of **synopticity**.
Suggested treatments to alleviate the consequences of jet lag include the use of bright lights and also melatonin to induce sleepiness, though it is important that melatonin is taken just before bedtime or otherwise it may have a detrimental effect. *The importance of this research lies in finding ways to reduce the effects of shift work and jet lag because of the potential dangers to individuals and society at large. Our society needs shift work and jet travel so the problems are not going to go away. One thing to bear in mind, however, is that there are important individual differences. Some people are less badly affected than others by desynchronisation. One explanation is that the people who cope better are those whose rhythms are slower to adjust so they experience less desynchronisation.* **[653 words]**	The final paragraphs provide further commentary (**AO2**), including a consideration of the real-world applications and the effect of individual differences (an 'issue'), providing additional **synopticity**. The answer is **well-structured** and **ideas are expressed clearly**. The **line of argument** is weak at the outset but improves throughout the essay. **AO1** – **Sound**, **accurate** and **well-detailed**. **AO2/AO3** – **Reasonable** analysis and line of argument but lacks elaboration in places and therefore **not effective**. **Evidence of synopticity** but issues/debates/approaches rather **basic**.