BIOLOGICAL RHYTHMS

A biological rhythm is a cyclical variation over some period of time in psychological or physiological processes.

An endogenous pacemaker is an internal factor. In the absence of a zeitgeber, rhythmicity is driven by internal devices or internal biological clock. E.g. NATURE: hormones.

An exogenous zeitgeber is an environmental factor. E.g. NURTURE: light, noise, stress.

CIRCADIAN RHYTHMS:

A circadian rhythm is a cycle that runs to about 24 hours. E.g. the sleep-wake cycle

Exogenous Research:

Campbell and Murphy (1988): 15 volunteers slept in a lab and were woken at different times. A light pad was shone on the back of their knees. Results showed that circadian rhythms fluctuated by as much as 3 hours from the normal cycle, depending on the time it was shone. This suggests that we don’t solely rely on light entering the eyes, and blood may be the messenger carrying signals to the brain.

Klein (1993): studied a blind man with a rhythm of 24.5 hours, which eventually got out of sync with the 24 hour day. Time cues didn’t help, and he had to take stimulants and sedatives to regulate his cycle, meaning that light acts as a zeitgeber.

Evaluation:

Studies like Campbell’s haven’t been replicated, so we can’t say results are reliable and consistent.

Lab studies – lacks ecological validity since it was an artificial environment. It was unnatural since participants wouldn’t have been in their own rooms, and would have been wired up – not an ordinary and comfortable nights sleep.

Czeizler (1999): p’s lived for a month with subdued lights. They were placed on a 28 hour sleep-wake cycle. Body temp monitored which marks action of body clock. Found that sleep clock generates on a schedule of 24 hours 11 mins, not 25 hours (more in line with the norm).

Practical Imps/apps: We shouldn’t use technology at night – struggle to sleep since the penal glands take longer to start to secrete melatonin. Also, wearing knee pads on night flights will stop light from sending signals to the brain, and therefore, not wake you up.

Endogenous Research:

Aschoff and Wever (1962): P’s lived in a underground bunker for 3-4 weeks. They could turn lights on and off, but had no idea of the time of day. All P’s followed a sleep-wake cycle of 25
ULTRADIAN RHYTHMS

An Ultradian rhythm is a cycle that happens more than once every 24 hours, but with shorter periods.

The Sleep cycle (the regularity of the 90 minute cycle suggests there must be a brain mechanism alternating causing REM and NREM sleep.

There are 2 basic forms of sleep/phases:

- Rapid eye movement (REM) sleep – where dreaming is most likely to occur. (4+)
- Non-rapid eye movement (NREM) sleep (1-4)

Sleep is measured using EEGs (brain), EMGs (muscles) and EOGs (eyes). EEG is the most popular but others compliment it.

Sleep stages:

Awake:

- Brain is active – full consciousness and high level of alertness.
- Beta waves show when fully awake, and some alpha when starting to feel relaxed and drowsy.

Stage 1:

- Eyes close and roll slightly.
- This is the transition stage between wakefulness and sleep – only lasts a few minutes.
- Body is likely to jerk, these are called myoclonic jerks, and are frequently accompanied by sensations of falling.
- On EEG, alpha waves show since it resembles relaxed wakefulness. But also there are some theta waves.

Stage 2:

- Light sleep – muscles are relaxed except for the occasional twitches.
- Heart rate + blood pressure increases, and breathing is shallow.
- Vivid dreaming can occur
- Can be easily woken in this stage, and will claim not to have slept.
- Sleep spindles and K complexes appear on EEG, which show sporadic bursts of activity.

Stage 3 + 4:

- This is deep sleep, also known as slow wave sleep (SWS).
- Dreaming can occur, but when woken, dreams will be disorganised and less easily remembered.
- Heart rate and blood pressure reduces and muscles relax further.
- Irritated and disoriented when woken and hard to get back to sleep.
- Delta and theta waves present on EEG since sleep is deep.

Stage 4+

- Dreaming is most frequent here, and we are most likely to remember our dreams.
- Paradoxical state: muscles are relaxed, the brain is alert just like the awake stage, but the body is paralysed (movement inhibition).
- Sensory blockade: no senses and don’t wake up when touched. Would need to be vigorously shaken to wake.
Rechtschaffen at al (1983) – Rotating rats

- Two rats placed on a disc above a container of water. One was able to sleep, but the other wasn’t since when the EEG indicated sleep, the disc began to rotate so the rat fell into the water. All of the sleep deprived rats died within 33 days; the rates that were not sleep deprived appeared to suffer no ill effects as a result of the study.

- Shows that disruptions to the circadian rhythm sleep-wake cycle can lead to fatal incidents in animals, such as death.

Evaluation

Case study – low population validity as it is only done on one person so we can’t generalise it to the whole population. People may have reacted differently due to individual differences such as current sleeping pattern, sleep disorders, age etc. However, a case study is very detailed.

Animal based research – troubles with extrapolation since humans are much more complex. It is also unethical since it caused distress and harm to the cats, eventually leading to death. However, it allows us to do research that we can’t do on humans.

Scientific measurements of sleep – EEGs are very objective.

Overall Evaluation of Disrupting Biological Rhythms

There is a lot of research to show that disrupting rhythms has potentially serious consequences. This means that research is reliable – we need to be able to sleep well, as most studies show consistent findings.

Lots of studies on shift work and jet lag were naturalistic so it has high ecological validity as there was nothing artificial or set up – using real people with real jobs. High mundane realism.

However a problem with these studies is that you cannot control EVs such as lifestyle (smoking, stress, alcohol etc) and genetics that could also cause these consequences. Also, it doesn’t account for individual differences, e.g. the ability of a sports team. With jet lag, there are mediating variables, e.g. hydration levels, age.

Lab based research – lacks ecological validity since EEGs were used (wired up), and p’s were in an artificial environment, they had no home comfort. However, this did mean that EVs were easier to control, and the study will be easier to replicate.

The research helps us to understand real life incidents (practical imps), such as why there are more accidents on night shifts than day shift. Also, Challenger rocket incident – people involved were deprived of sleep.

Practical applications: can prevent jet lag with techniques such as melatonin supplement. Also, in an ideal world, no shift work. Or if working shifts, it should be non-fluctuating and clockwise.

Research is deterministic since it suggests that if you get jet lagged a lot, or work shifts, you will get cancer. However, we do have some extent of free will, e.g. choosing a clockwise non-fluctuating shift pattern to reduce the risk, or even moving jobs.

A lot of individual differences in people’s reactions to shift work, jet lag and sleep deprivation, which means we can’t generalise results to all – some people aren’t affected by jet lag, everyone reacts differently depending on age, gender, lifestyle, personality.
• **Sleep-state misperception**
  - People sleep adequately but feel that they don’t – they underestimate the total sleep time and overestimate the time it takes them to fall asleep.
  - It results from an unclear perception and consciousness and difficulty distinguishing sleep from waking – faulty perceptions.

**Causes of primary insomnia:**

**Genetics**

*Dauvilliers (2005):*

- 256 primary insomniacs completed clinical interviews, psychometric questionnaires and a questionnaire on the family history of insomnia.
- A control group was also used to obtain an estimated base-rate incidence of insomnia in their families.
- Results showed that of those patients with primary insomnia, 72.7% reported familial insomnia (a family history with insomnia) compared to 24.1% in the non-insomnia control group.
- These findings support a familial link to primary insomnia.

**Evaluation**

Large sample – nomothetic – a lot of data to analyse.

Clinical interviews and psychometric questionnaires used – carried out in hospitals/clinics – fairly objective measures but demand characteristic – unreliable.

Nature, biological and deterministic due to our genes.

**Cognitive behaviour**

*Morin et al (2003):*

- Examined the role of stress, coping skills, and pre-sleep arousal in insomnia.
- Good sleepers and insomniacs were asked to record daily measures of stressful events, pre-sleep arousal, as well as retrospective measures of depression, anxiety, stressors and coping skills for 21 consecutive days.
- They found good sleepers and insomniacs recorded the same number of daily stressors but insomniacs rated the impact of the stressors higher.
- Insomniacs also relied on emotion-orientated coping strategies and higher levels of pre-sleep arousal.
- This supports the cause is cognitive because it suggests that the key common factor in insomnia is the way people appraise stressors and the perceived lack of control, rather than the number of stressful events.
**Evaluation**

Self reports – subjective measures, participant could exaggerate, unreliable results.

Subjective definition of ‘stress’ – doesn’t take into account individual differences, can’t generalise.

Practical applications – stress coping strategies to treat insomnia

Nurture – the stressors are external.

Unethical – could cause distress having to talk about and elaborate on stressful things in their lives.

**Brain physiology**

Smith *et al* (2002):

- Studies neuro-images of the brains of 9 females (5 insomniacs and 4 controls) over 3 nights in a lab.
- The insomniacs showed consistent and significant decreases in blood flow in several brain areas during NREM sleep compared to good sleepers.
- This supports that one cause of primary insomnia is brain physiology since insomnia is due to abnormalities in the brain.

**Evaluation**

Lab study, lacks ecological validity since the situation was very artificial – may sleep differently in a lab to at home. Scientific, objective measures used – neuro-images.

Correlational study – can’t see cause and effect and can’t control extraneous variables that may be causing insomnia, e.g. lifestyle. But can make predictions and use results in real life situations.

Gender (beta) bias since it was done on females, so ignores differences between males and females. With females, there may be external factors such as menstrual cycle/menopause affecting sleep.

**Faulty perceptions**

Dement (1999):

- A patient who complained of severe insomnia was asked to sleep for 10 consecutive nights in the sleep lab
- Each morning, he was asked to complete a questionnaire where he had to estimate how long it took him to fall asleep each night.
- He reported times ranging for 1-4 hours to fall asleep, with a mean on 90 minutes.
- However, according to the recordings, he never took more than 30 minutes to fall asleep and the mean was 15 minutes.
- This supports that one cause of primary insomnia is faulty perceptions as they perceive the time wrong – they have difficulty distinguishing wakefulness to sleep.

**Evaluation**