

Supplementary material A

The science of sleep

Sleep is regulated by the suprachiasmatic nucleus (SCN; see supplementary figure 1) which operates as a sleep pacemaker by coordination of clock gene expression.[1] Melatonin, the 'sleep' hormone released in the dark, is released as a consequence. The two-process model of sleep (see supplementary figure 2) proposes that Process C (circadian rhythm – responsible for homeostatic control of sleep wake cycles) and Process S (sleep pressure, which builds as more time is spent awake) interact, regulating the sleep/wake cycle.[2, 3] The black line in the process C and S model shown in supplementary figure 2 represents the normal circadian rhythm which is unaffected by sleep deprivation. Sleep pressure however (highlighted in green) increases as long as wake periods occur and diminishes as soon as sleep is initiated. If sleep is missed (highlighted in red) sleep pressure continues to increase.

Supplementary figure 3 shows an example of normal sleep architecture displayed as a hypnogram. Sleep is divided in two main stages: rapid eye movement sleep (REM) and non-REM sleep.

Non-REM has three stages which are N1, N2 and N3. N1 (which makes up 5-10% in adults) is the lightest stage of sleep, where sleep is initiated. Arousals are more common and slow eye rolling can also be noted. This is characterised by a combination of alpha waves (8-13 Hz) and theta waves (4-7 Hz accompanied by eye rolls, active muscle tone and hypnic jerks or myoclonus. N2 makes up 45-55% in adults. It is a light stage of sleep, though the sleeper is harder to wake. Unique features that can be observed on EEG trace during N2 are K complex waves and sleep spindles (11-16 Hz), generally thought to help sleep-based memory consolidation. N3 makes up 15-25% in adults. It is also known as slow wave sleep, with delta activity which has high amplitude waves of 0.5 - 2 Hz. N3 consists of deeper sleep and arousals are harder to precipitate. N3 is thought to have a restorative function.

Typically, after NREM stages, REM sleep (highlighted in red on the hypnogram) occurs; a stage characterized by atonia in all voluntary muscles with the exception of the eye muscles. REM is the sleep stage where we dream and plays a crucial role in mental development, memory retention and overall well-being. Each cycle lasts approximately 90 minutes (in adults) and is then repeated 4-5 times during the night. Alterations or missed cycles results in disrupted sleep.

External stimuli (zeitgebers) can adjust the timing of Process C to shift sleep/wake cycles.[4] Zeitgebers that may affect sleep, in teens especially include: academic pressures, social activities, sports, internet, smart device and television viewing, especially at night, part-time employment, peer and parental influence and home influences which may be affected by socioeconomic status.[5, 6] Changes in Processes C and S can thus precipitate sleep disruption. It is therefore important to

consider referral to a sleep laboratory to perform diagnostic tests and not simply apply the common trope of “lazy teenager.”

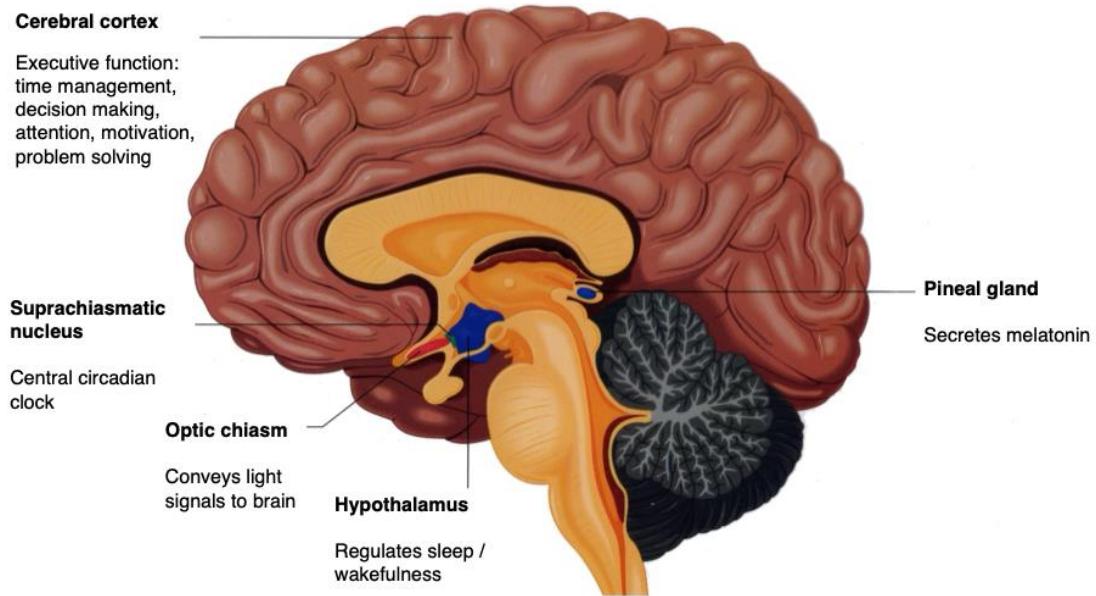
Supplementary Figure 1: Diagram of neuroanatomical structures involved in sleep

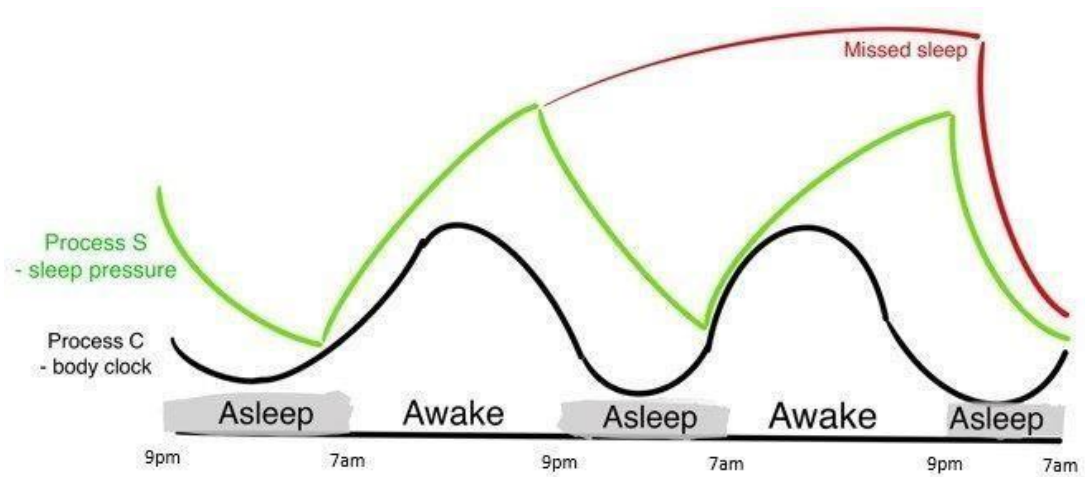
Image Credit: Illustration of the human brain showing the Cerebral Cortex, the Suprachiasmatic Nucleus, the Optic Chiasm, the Hypothalamus and the Pineal Gland

[黄雨伞](#) - Own work

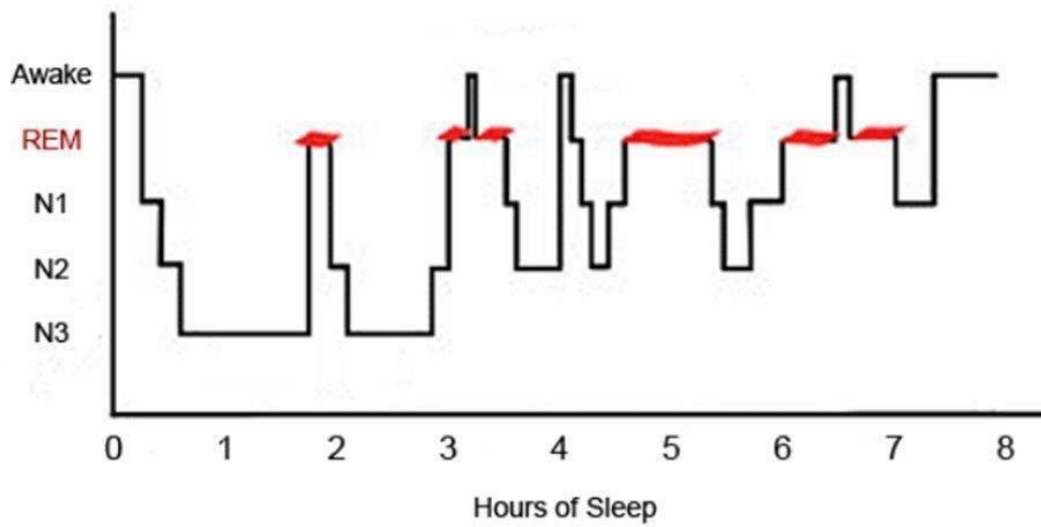
- [CC BY-SA 3.0](#)
- File:Suprachiasmatic Nucleus.jpg
- Created: 7 January 2014
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Supplementary Figure 2: The two-process model of sleep – sleep pressure and circadian rhythm (Process C and Process S)



Supplementary Figure 3: Example of normal sleep architecture displayed as a hypnogram



Supplementary material B

Description of sleep disorders seen in children and adolescents

Behaviourally Induced Insufficient Sleep Syndrome (BISS): is a condition where an individual consistently fails to achieve sufficient sleep overnight, often catching up on the missed sleep with longer nocturnal sleep over the weekend.[7] BISS is normally found in young adults with prevalence decreasing with age.[8]

Delayed Sleep Phase Disorder (DSPD): is a circadian rhythm sleep disorder and is characterized by daytime sleepiness, difficulty falling asleep and problem waking up at a set time, despite following a sleep schedule.[9] DSPD usually occurs in teenagers, with up to 16% of adolescents said to suffer from this disorder.[10] Recent studies also suggest a trend between individuals diagnosed with DSPD and ADHD.[11] As opposed to DSPD, advanced sleep phase, is where an individual falls asleep much earlier than a normal bedtime and therefore wakes up earlier than normal.[12]

Idiopathic hypersomnolence (IH): is a neurological condition, with similar symptoms to Narcolepsy type 2, where an individual presents with excessive nocturnal sleep without feeling refreshed upon waking, sleep inertia and excessive daytime sleepiness despite meeting the recommended sleep time overnight.[13] IH usually appears in younger people, and it is often diagnosed by ruling out other central disorders of hypersomnolence.[14]

Kleine-Levin syndrome (KLS): is a rare sleep disorder, where episodes of mild to extreme hypersomnolence occur recurrently often coupled with hyperphagia.[15] These episodes can last from several days to several weeks.[16] Predominantly occurring in adolescent boys, there are currently no identified causes of KLS, however documented cases suggest genetics, in particular variations in *TRANK1* gene region, may play a part in who may be affected.[17]

Narcolepsy: is a rare sleep disorder characterized by rapid REM onset, excessive daytime sleepiness and disturbed night-time sleep. In most cases narcolepsy is caused by low hypocretin level (also known as orexin), a chemical messenger in the brain which aids in regulating wakefulness and arousal.[18] Symptoms are can first be seen in childhood or adolescence, however diagnosis commonly occurs later, between the age of 20 and 40. Growing out of naps is developmentally appropriate as a child though the age this happens can vary. It is important to consider referral if a teenager has regular naps for more than 3 months after growing out of them.

Narcolepsy is grouped into two subtypes. Narcolepsy type 1 is usually associated with cataplexy, which is unique to narcolepsy type 1. Cataplexy is the sudden episode of muscle weakness provoked by emotions such as surprise, anger or laughing. Narcolepsy type 2 shares many symptoms with narcolepsy type 1 but without low hypocretin levels nor cataplexy.[18]

Supplementary material C

Strategies to manage insomnia

At the Evelina sleep clinic, the intervention for insomnia in teenagers is a form of adapted Cognitive Behavioural Therapy (CBT) for Insomnia, and includes the following components:

Sleep restriction: The sleep restriction plan is described in A's case study. The aim of this is to reduce the amount of time spent in bed not sleeping, which can give rise to frustration and weaken the link between being in bed and sleeping. Mildly restricting sleep also aims to increase sleep pressure to improve sleep latency.

Sleep hygiene: Sleep hygiene advice includes setting an electronics curfew 60 minutes before bed and incorporating a relaxing wind down routine. Additionally, it is essential to consider the sleep environment to rule out any environmental triggers/contributors to poor sleep (e.g. uncomfortable/unsuitable sleeping area, bright lights, noise). Part of good sleep hygiene includes sleep scheduling. A strict bedtime and wake time, including at weekends, helps to keep sleep pressure building in a consistent and predictable way. It is important to note that sleep hygiene in isolation does not treat insomnia.

Stimulus control: Similar to sleep restriction, the aim of stimulus control is to reduce the amount of time spent in bed not sleeping. Specifically, this is related to not doing any other activity in your bed other than sleeping. This means no TV, not messaging friends, not doing homework, not spending time worrying etc. in your bed. Furthermore, if a patient wakes for over a quarter of an hour overnight, the advice is to get out of bed, engage in a calming activity, and only return to bed when feeling sleepy and tired. This was to break associations between bed and activities and sensations non-conducive to sleep and strengthen its link with sleep.

Cognitive strategies: It is important to explore beliefs about sleep and how these might impact on sleep behaviours. Strategies to manage negative thoughts/beliefs about sleep can include CBT strategies such as thought challenging or third wave CBT approaches including Acceptance Commitment Therapy (ACT).

These strategies have been described for use with young people in several journals and books.[19-22] Tailoring the sleep plan to the individual is key to its chances of success.[23]

Supplementary material D

Description of motivational interviewing principles

Motivational interviewing (MI),[24, 25] helps participants commit to change by exploring ambivalence. A key component is on patients exploring their own arguments for change. The interviewer is aiming to elicit *change talk* and then strengthen the commitment to change. With sleep interventions with teenagers, it is important to assess the function of current sleep (for example, perhaps it means they do not have to go to school which they find stressful) and their motivations for committing to change. MI can be useful to promote healthy sleep behaviours.[26] The four main principles of MI are to express empathy, to elicit client's own ideas, to roll with resistance (e.g. not directly challenging client) and highlighting discrepancy between current behaviour and the clients hopes/values.[27]

Example questions:

What are the important reasons to change vs stay the same?

What steps are you willing to take?

In what way are you already able to make the change you wanted to make?

What is the worst/ best that can happen?

Supplementary material E

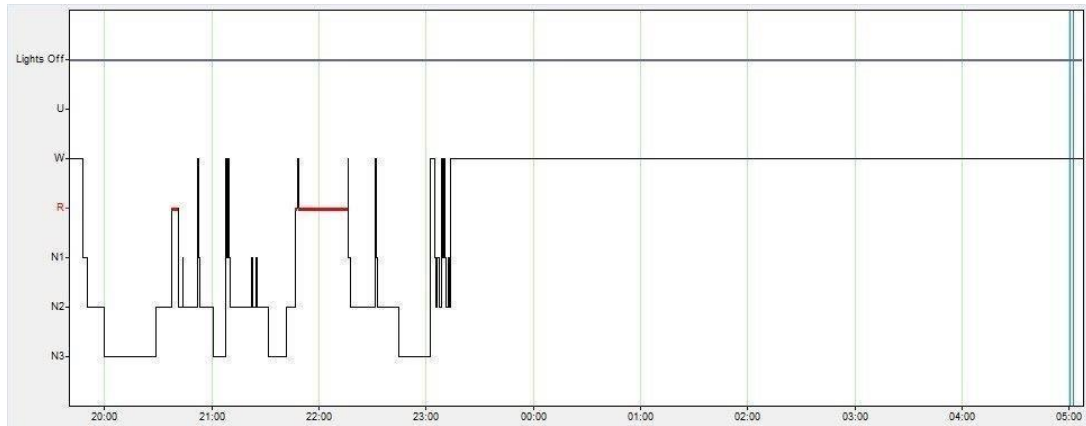
Trouble shooting plan / future planning

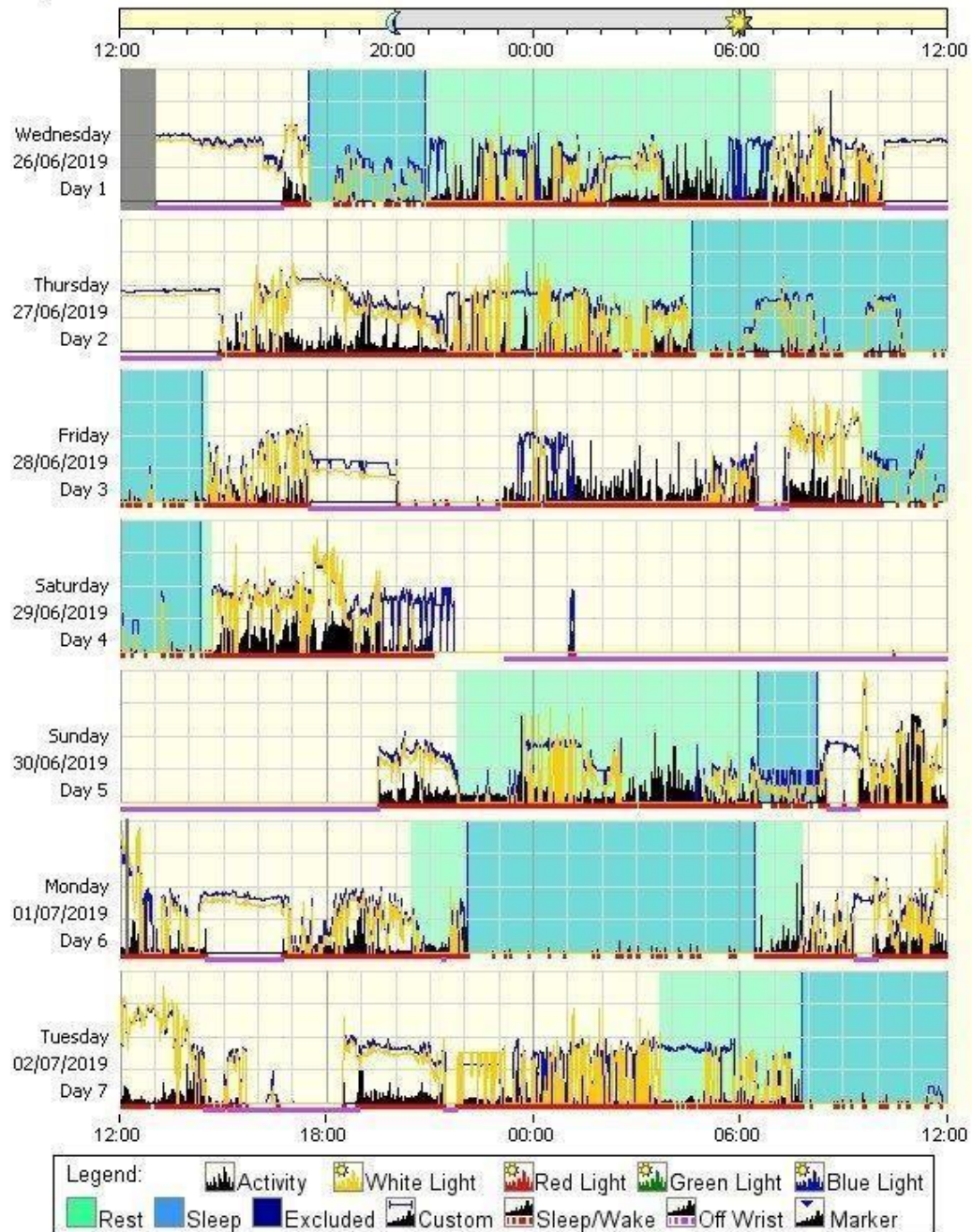
The patient is encouraged to discuss ways they would overcome difficulties that might present themselves in the future. It can be useful to make this into an action plan which can be used in the future. Questions might include

- How would I recognise that my sleep pattern is changing?
- What do I know triggers a change in my sleep?
- Who would I talk to if I thought that something was changing?
- What could I do to help myself?
- What could others do to help?

The patient also can be encouraged to write a letter to their future self as a reminder of the gains from making a change to their sleep which they would be able to refer to in the future if things began to feel more difficult.

Supplementary Figure 4: Polysomnography for case C



Supplementary Figure 5: Actigraphy for case C**Actogram:**

Supplementary material F

Details of organisations and resources with a focus on sleep in young people

<https://teensleephub.org.uk/teens-young-people/>

<https://www.evelinalondon.nhs.uk/our-services/hospital/sleep-medicinedepartment/how-to-sleep-well-for-teenagers.aspx>

If you want to know more, here are some organisations focused on sleep:

British Paediatric Sleep Society (BPSS)

Association of Clinical Psychologists in Sleep Medicine (ACPSM)

Supplementary References

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