Independent Research & further reading

Guest: Dr Matthew Walker

Disclaimer 1: The sources presented here, directly (or as closely as possible), look at statements made by the guest in this episode. In order to report each topic thoroughly, an extensive search and review (beyond the scope of this document) would be required.

Disclaimer 2: The information provided in this podcast and any associated materials is not intended to replace professional medical advice. For any medical concerns, it is essential to consult a qualified health professional.

Contents

Effects of Sleep on DNA Expression, Belief, and Social Connection

> *"even down to the level of your DNA, your sleep and how you are sleeping or not sleeping will change the very DNA nucleic alphabet that spells out your daily health narrative (...) It can change our belief systems. It can change how lonely or sort of hyper social we are."*

Evidence shows a clear relationship between sleep and DNA expression. Research indicates that both sleep and sleep deprivation alter the activity of large numbers of genes across the brain and peripheral tissues, including genes that regulate circadian rhythms such as BMAL1, CLOCK, and PER1. Sleep loss also affects epigenetic mechanisms (processes that regulate gene expression without changing the DNA sequence) through measurable changes in DNA methylation and histone modifications. These alterations can shift chromatin accessibility, particularly in enhancer regions, influencing which genes are available for transcription. The effects are highly specific to tissue type, brain region, and cell population, with neurones and astrocytes showing distinct patterns of response. Overall, the evidence demonstrates that sleep plays a central role in regulating gene expression and epigenetic states, helping to shape physiological, cognitive, and circadian functions.

Relationship Between Sleep and Belief

Evidence indicates a bidirectional relationship between sleep and belief. Research shows that sleep-related beliefs, such as unrealistic expectations about sleep or misinterpretations of insomnia symptoms, are strongly associated with poorer sleep quality. These dysfunctional beliefs can be improved through cognitive behavioural therapy for insomnia, which often leads to better sleep and

mental health outcomes. Individuals who endorse sleep myths or feel they lack control over their sleep tend to report more disturbances and less healthy sleep behaviours, particularly when psychological flexibility is low. Broader belief systems also relate to sleep: people with more positive worldviews, for example, often experience better subjective sleep quality, while poorer sleep is associated with stronger endorsement of paranormal and conspiracy beliefs, with anxiety and depression frequently mediating these effects. Belief in a just world can further moderate the relationship between sleep quality and subjective well-being, contributing to resilience and more positive outcomes.

Relationship Between Sleep, Loneliness, and Pro-Sociality

Evidence shows a strong connection between sleep, loneliness, and pro-social behaviour. Research indicates that sleep problems, such as insomnia or poor sleep quality, are consistently associated with higher levels of loneliness, and this relationship appears to be bidirectional. Loneliness can heighten nighttime vigilance and arousal, leading to fragmented sleep, while insufficient sleep can increase feelings of social withdrawal, creating a reinforcing cycle. Factors such as depression, anxiety, and daily stress often mediate this relationship, although the association remains significant when these variables are considered. Studies also show that sleep loss reduces pro-social behaviour, including empathy and willingness to help others, and these effects are linked to impaired functioning in brain regions involved in social cognition, such as the temporoparietal junction. The depth and quality of sleep, particularly slow-wave activity, appear to support prosocial tendencies, suggesting that sleep plays an important role in sustaining healthy social connection and cooperative behaviour.

References 1-16.

Brain and Body Activity During Sleep

> *“sleep is not a passive state. It's an incredibly active state, both in terms of the brain and the body.”*

Evidence indicates that both the brain and body remain highly active during sleep, although the nature of this activity varies across sleep stages. During non-REM sleep, the brain generates slow

oscillations, sleep spindles, and sharp-wave ripples, which support memory consolidation and neural homeostasis. REM sleep shows patterns that resemble wakefulness, including theta oscillations and increased blood flow in regions involved in emotion and memory. Even during the deepest stages of sleep, the brain continues to process some external information, although with reduced responsiveness.

The body also maintains active physiological regulation throughout sleep, with ongoing autonomic control of heart rate, respiration, and temperature. REM sleep includes rapid eye movements, muscle twitches, and variability in cardiovascular and respiratory patterns, while non-REM sleep presents more stable but still active physiological processes. Additional changes in blood flow, cerebrospinal fluid movement, and metabolic waste clearance occur across sleep stages, contributing to overall restoration and health. Observations across species, including invertebrates, further suggest that these active sleep processes are evolutionarily conserved.

References 17-24.

Sleep Education in Medical Training

> *"And doctors themselves, you know, there's a great study and what they found was that medical doctors across, I think it was about 11 different curricula around the world, they will only receive about 1.2 hours of education on sleep, but it's a third of their patients' lives."*

Evidence shows that physicians receive very limited formal education on sleep and sleep disorders. Surveys from multiple countries indicate that medical students typically receive between 2 and 4 hours of sleep education, with most receiving fewer than 3 hours in total. In the United States, the average is under 2 hours, and a substantial proportion of medical schools provide no dedicated sleep education at all, while only a small minority offer 4 or more hours, usually as elective content. International data are similar, with averages under 2.5 hours globally and most schools in the United Kingdom offering less than 2 hours. Other regions, including Saudi Arabia, Brazil, and Lebanon, also report low exposure, with most students receiving fewer than 3 hours of instruction. Residency training adds only modest additional coverage, averaging 4 to 5 didactic hours per year. Overall, the evidence indicates that sleep education for physicians remains minimal despite the significant health burden associated with sleep disorders.

References 25-31.

Sleep Apnea and Restless Leg Syndrome

> *"Those people who have other sleep disorders like sleep apnea,*
>
> *there's another disorder called restless leg syndrome. It's a terrible disorder where your legs start to feel as though they've got this sort of creepy crawly feeling and you've constantly got a massage them and move them. It's a terrible disruptor of your sleep."*

Features of Sleep Apnea

Evidence shows that sleep apnea is a common sleep disorder involving repeated pauses in breathing, referred to as apnea, or shallow breathing episodes, referred to as hypopnea, during sleep. The most prevalent form is obstructive sleep apnea, which occurs when the upper airway collapses or becomes blocked. Less common forms include central sleep apnea, in which the brain does not send appropriate signals to the breathing muscles, and complex sleep apnea syndrome, which combines features of both obstructive and central types. Symptoms often include loud snoring, gasping during sleep, daytime sleepiness, headaches on waking, irritability, and difficulty concentrating.

Risk factors include obesity, male gender, older age, family history, alcohol use, smoking, and anatomical features such as large neck circumference or craniofacial structure. Sleep apnea is associated with substantial health consequences, including hypertension, cardiovascular disease, stroke, and metabolic dysfunction. Diagnosis typically requires a sleep study, such as polysomnography, and treatment most often involves continuous positive airway pressure, with alternatives including oral appliances, surgical interventions, and lifestyle modification.

Restless Leg Syndrome

Evidence shows that restless leg syndrome, also called Willis-Ekbom disease, is a neurological sensorimotor disorder defined by an uncontrollable urge to move the legs, typically accompanied by uncomfortable sensations such as creeping, crawling, tingling, or pain. Symptoms generally begin or intensify during periods of rest or inactivity, are partially or completely relieved by movement, and tend to be worse in the evening or at night. Diagnosis requires that these symptoms are not better

explained by another medical or behavioural condition. The disorder may be primary, with a genetic contribution, or secondary to factors such as iron deficiency, pregnancy, chronic kidney disease, or certain neurological or metabolic disorders.

Research indicates that dopaminergic dysfunction and brain iron dysregulation play central roles in the condition. Prevalence estimates in Western populations range from 2 to 5 percent, with higher rates among women and older adults. Restless leg syndrome can substantially impair sleep and daytime functioning. Treatment typically includes dopaminergic agents, alpha-2-delta calcium channel ligands, and iron supplementation when deficiency is present, alongside lifestyle strategies and, in severe cases, opioid therapy. Long-term dopaminergic treatment can sometimes lead to symptom worsening, referred to as augmentation.

References 32-41.

Effects of Alcohol, Caffeine, and THC on Sleep

> *"And then you've got another collection of individuals who, they don't have any sleep disorders, but they are either doing things that will dismantle their sleep. So either the taking on things into themselves like alcohol, caffeine, THC, that aren't the sleep helpers, even though sometimes they think they are. And then you've got perhaps the internal things that will prevent you from sleeping well, things like stress and anxiety. "*

Effects of Alcohol on Sleep Quality and Architecture

Evidence indicates that alcohol has a disruptive effect on sleep despite its short-term sedative properties. Acute alcohol intake can shorten sleep onset, but it reduces REM sleep and increases slow-wave sleep early in the night, followed by more awakenings and lighter sleep in the later hours. Chronic or heavy alcohol use is linked to long-lasting sleep disturbances, including persistent insomnia, alterations in sleep architecture, and disruptions to circadian rhythms. Research also shows that alcohol use can increase the risk of sleep disorders such as sleep apnea, particularly in individuals with alcohol use disorder.

The relationship between alcohol and sleep is bidirectional, since disturbed sleep can increase alcohol consumption, and alcohol use can worsen sleep quality. These effects are observed

across age groups, with older adults and men appearing more vulnerable to sleep-related disruption from heavy drinking. Overall, alcohol may initially seem to promote sleep, but its long-term impact is consistently associated with poorer sleep quality and greater risk of ongoing sleep problems.

Effects of Caffeine on Sleep

Evidence indicates that caffeine disrupts sleep quality and sleep architecture, particularly when consumed later in the day or in higher doses. Caffeine acts by blocking adenosine receptors, which reduces sleep pressure and promotes wakefulness. Research shows that caffeine intake decreases total sleep time, increases the time required to fall asleep, lowers sleep efficiency, and reduces both slow-wave sleep and REM sleep.

Meta-analytic findings suggest that caffeine can reduce total sleep time by approximately 45 minutes and sleep efficiency by around 7 percent, while increasing wakefulness after sleep onset and time spent in lighter sleep stages. Higher doses, such as 400 milligrams, and consumption within 8 to 12 hours of bedtime have the most substantial effects, whereas lower doses taken more than 4 hours before bedtime typically have minimal impact. Regular daytime consumption can still delay REM sleep and affect the quality of awakening. Individual sensitivity varies, with older adults and certain genetically predisposed individuals showing greater vulnerability to sleep disruption. Even habitual users may experience reduced subjective sleep quality despite smaller objective effects. The relationship is bidirectional, since poor sleep can lead to increased caffeine use, which then further disturbs sleep.

Effects of THC on Sleep

Evidence indicates that Tetrahydrocannabinol (THC) alters sleep architecture, particularly by suppressing REM sleep. Controlled studies show that THC, whether used alone or with CBD, reduces the amount of REM sleep and increases the time required to enter REM. Acute administration can also decrease total sleep time, with some findings in insomnia patients showing reductions of approximately 25 minutes compared with placebo. Research on chronic or high-dose use suggests small decreases in nighttime sleep duration, possibly related to tolerance. THC may shorten the time it takes to fall asleep, although subjective sleep quality shows mixed outcomes. Some individuals report better sleep, while others experience no improvement or worsening sleep, especially with longer-term use or higher doses.

Withdrawal following regular use is linked to significant sleep disturbances, including insomnia and REM rebound, which involves increased REM sleep and vivid dreaming. Additional findings show that THC can produce next-day sleepiness and cognitive impairment at higher doses, and pre-sleep use can lower heart rate variability, indicating increased cardiovascular stress. Effects vary by age, baseline sleep quality, and conditions such as pain or anxiety. Overall, the evidence does not support THC as a reliable or safe long-term sleep aid.

References 42-54.

Compensatory Weekend Sleep and Cardiovascular Risk

> *"there was a study published from what's called the UK Biobank (...) they studied in this particular research paper over 90,000 individuals. And what they did was they essentially split them down into those individuals who were short sleeping during the week and then short sleeping at the weekend. And they compared them to those individuals who were short sleeping during the week, but then long sleeping at the weekend, they were doing catchup sleep (...) in the people who were short sleeping during the week, but long sleeping at the weekend, they had a 20% reduced cardiovascular disease risk relative to the people who were short sleeping during the week, but also short sleeping at the weekend."*

Evidence from the UK Biobank indicates that compensatory weekend sleep, measured using wrist-worn accelerometers in a cohort of 90,903 adults, is associated with a lower risk of cardiovascular disease. Participants were grouped by the amount of additional weekend sleep, and those in the highest compensatory sleep category showed reduced risk across a median follow-up of 13.8 years. This reduced risk remained stable after accounting for genetic predisposition to ischemic heart disease, heart failure, and atrial fibrillation. The association was even stronger among individuals who routinely slept too little on weekdays. These findings suggest that extending sleep on weekends may help offset some cardiovascular risk associated with chronic weekday sleep restriction.

Reference 55.

Effects of Pre-Emptive Sleep Extension on Sleep Restriction

“But here's this new remarkable data from Walter Reed Medical Army Institute, a researcher called Thomas Balkan. And what he was interested in was flipping the direction of the question, not if I go into debt, can I pay back with credit? But what if I know in future I'm going to face a debt, an upcoming debt? Can I do sleep banking? (...) It's a sleep saving system (...) what he found was that people who had built up this credit, this savings account of sleep in the days in the week beforehand, they suffered about 40% less of an impairment relative to the people who had not created any savings plan (...)

athletes sleep terribly typically the night before. A huge performance, no matter, you know, of course they're nervous. But what you can do is you can have them bank sleep in the days before when they're not as nervous and therefore their performance doesn't suffer as much, even though they know they're going to be deprived”

Evidence indicates that pre-emptive sleep extension, sometimes called sleep banking, can reduce certain negative effects of subsequent sleep restriction. Research shows that increasing sleep duration before anticipated sleep loss improves alertness, psychomotor vigilance, reaction time, and overall cognitive performance during periods of restricted sleep. Studies in laboratory environments and operational settings report fewer attention lapses, reduced fatigue, and in some cases faster recovery following sleep deprivation. Research involving shift workers, military personnel, and healthy adults shows that sleep banking may reduce acute fatigue and improve performance in safety-critical tasks, and some studies report potential metabolic advantages such as improved insulin sensitivity and lower intake of free sugars in chronically sleep-restricted individuals. However, the overall quality of evidence is low, with many studies using small samples or designs at risk of bias.

Findings in athletes suggest possible benefits for motor performance, accuracy, and mood, although evidence in this group remains limited.

Sleep Extension Before Competition and Athletic Performance

Evidence suggests that extending sleep in the days leading up to competition can improve certain aspects of athletic performance, although findings vary by sport, performance metric, and study quality. Research indicates that aiming for longer sleep durations, often nine to ten hours per night, can enhance sport-specific skills such as sprint speed, shooting or serving accuracy, and

reaction time. Benefits have been reported across several sports, including basketball, tennis, swimming, and rugby, and appear strongest in activities that rely on technical skill, speed, or cognitive processing. Sleep extension may also support better mood and stress resilience during periods of intense training or competition. Pre-emptive sleep extension may mitigate performance deficits associated with pre-competition sleep loss caused by factors such as anxiety, travel, or early event times. Studies show that athletes who extend sleep before expected sleep restriction tend to maintain better motor performance and demonstrate less cognitive impairment than those who do not. However, much of the evidence comes from small or laboratory-based studies, and real-world competition data remain limited.

References 56-69.

Effects of Electronic Device Use on Sleep Quality

"most impactful things that you can start doing tonight to start sleeping better? (...) digital detox (...) it's not a problem of blue light (...) we've been taught this myth of the blue light effect (...) after a very influential paper, which is a great paper. And what they showed was that one hour of iPad reading before bed ended up impairing your melatonin. It disrupted sleep, it reduced the amount of dream sleep (...) But then Michael Gradisar, this incredible Australian researcher, started to say, well, I can't replicate these findings. And what he was discovering is that it's not the blue light that's the problem (...) It's a combination of first these devices that we use are attention capture devices and they are designed to fleece you of your attention economy (...) So what happens is that these devices become hugely activating and as a result, they essentially will be a mute button on your sleepiness. So you could be there, you get into bed, it's 11:00 PM you think, I am so tired, I was falling asleep on the, the, the couch watching television. And then you get to bed, you start going onto social media and then you start doom scrolling. And then you get into this, what we call bed rotting, where you just sit there and now you look at the clock and it's no longer 11:00 PM it's 1:00 AM and you've just done sleep procrastination. Now it turns out that it's yes, that, that these are attention grabbing devices that will mute your sleepiness, but you have to be of a certain personality type (...) perhaps neurotic, someone who has high impulsivity, or someone who is perhaps high anxious (...)"

Evidence consistently shows that using electronic devices before bedtime is associated with poorer sleep quality across children, adolescents, and adults. Large reviews and meta-analyses report that evening screen use increases the likelihood of insufficient sleep duration, lower sleep efficiency, and greater daytime sleepiness, and that even the presence of a device in the bedroom is linked to worse sleep outcomes. The mechanisms underlying these effects include melatonin suppression and circadian delay from blue light exposure, increased cognitive or emotional arousal from engaging content, and displacement of sleep time due to prolonged device use. Interactive activities and extended screen exposure appear particularly disruptive. Experimental studies indicate that reducing or eliminating device use before bed improves sleep latency, sleep duration, and overall sleep quality.

Cognitive and Light-Related Causes of Delayed Sleepiness

Evidence indicates that both blue light exposure and cognitive or emotional engagement with electronic devices can delay sleepiness, although blue light has a more consistently demonstrated physiological effect. Research shows that blue light from screens suppresses melatonin, delays circadian rhythms, and increases alertness, which leads to later sleep onset. Interventions such as blue light–blocking glasses or screen filters produce modest improvements in sleep, supporting this mechanism.

Cognitive and emotional engagement, such as exposure to stimulating content, gaming, or social interaction, can also delay sleep by increasing arousal and mental activation, though these effects are more variable and less directly quantified in controlled studies. Current evidence therefore suggests that blue light plays a more reliably measurable role in delaying sleepiness, while cognitive or emotional engagement remains an additional contributing factor, particularly when the content is highly stimulating.

Sleep and Technology: A Bidirectional Framework

A 2024 review proposes a bidirectional model describing how technology use and sleep influence one another, emphasising that the relationship is not explained by blue light exposure alone. The authors detail four pathways through which evening technology use can delay or disrupt sleep: cognitive and emotional arousal generated by engaging content, the displacement of sleep time due to prolonged use, circadian effects from light exposure, and physiological activation associated with stimulation or stress responses. The paper argues that earlier research

overemphasised blue light's role while underestimating the impact of cognitive engagement and behavioural displacement.

At the same time, sleep itself shapes technology use, since inadequate or poor-quality sleep can increase evening screen time, lower self-control, and heighten reward sensitivity, thereby reinforcing late-night device engagement. The authors conclude that interventions should target both sides of this relationship, reducing stimulating or time-consuming technology use before bed while also improving sleep to weaken the feedback loop.

References 70-79.

Advanced Circadian Phase Disorder

> *"if you have a circadian rhythm disorder, let's say that you're someone who has an advanced circadian phase, what that means is you're someone who really can't get sleepy until three or four in the morning, and you would prefer to be sleeping throughout most of the day.*
> *So you, you are almost nocturnal (...) it's a genetic disorder in part (...) probably somewhere between, probably one to 2% of the population have a very severe, what we call an advanced circadian phase disorder."*

Advanced circadian phase disorder, also called advanced sleep–wake phase disorder, is a circadian rhythm condition in which the timing of sleep is shifted several hours earlier than desired. Individuals typically become sleepy in the early evening and wake up very early in the morning, reflecting an advance in underlying circadian rhythms such as core body temperature and melatonin secretion. Prevalence estimates range from approximately 0.04 to 1 percent, and some cases follow an autosomal dominant pattern known as familial advanced sleep phase, which has been linked to mutations in clock genes including PER2, PER3, CSNK1D, CRY2, and CACNA1D. The condition is often identified in middle-aged or older adults, although genetically driven cases can appear earlier in life. Diagnosis is based on clinical history supported by sleep diaries, actigraphy, and occasionally melatonin phase assessment. Management approaches include evening light therapy, phase-delay strategies, and timed melatonin, though responses vary and evidence remains limited.

References 80-85.

Melatonin Use, Sleep Outcomes, and Health Risks

“if you look at what we call meta-analysis, where we gather together all of the individual studies on a topic and we put them in a big statistical bucket, what they found is that melatonin [supplementation] will only improve the speed with which you fall asleep by about 3.4 minutes, and it will only increase the efficiency of your sleep by about 2.2% (...)

it's not a peer review, it's not reviewed yet by scientists. It's not published in a journal, so you've got to treat it at preliminary. But there was a recent conference where someone published, literally 72 hours ago a paper showing that people who were taking melatonin had double the risk of cardiovascular disease across the lifespan that they studied than people who were not taking melatonin (...)

There was a study that was published about three years ago that showed here in America over the past 10 years, there has been a 503% increase in poisonous overdose admissions to hospitals of melatonin in the past 10 years, 503% increase. So firstly, we've got to be a bit careful. The second reason is that melatonin is a bioactive hormone and it's also involved in reproductive development. And those studies done back in the 1970s, I think, where they were looking at juvenile male rats, which is to say male rats who were going through adolescence. And they were dosing them with high amounts of melatonin. And what they found is that that stunted the development of the testes of the testicles, and it caused testicular atrophy.”

Effects of Melatonin on Sleep Onset Latency

Evidence shows that melatonin supplementation produces a modest reduction in sleep onset latency (the time it takes to transition from full wakefulness to sleep) in adults. Meta-analyses report average decreases ranging from approximately 4 to 10 minutes compared with placebo, with findings such as a 4-minute reduction in one large review, reductions of 5 to 6 minutes in others, and estimates around 7 minutes in studies using prolonged-release formulations. Larger effects are reported in specific sleep disorders, particularly delayed sleep phase disorder, where reductions in sleep onset latency can exceed 20 minutes. Factors influencing the magnitude of benefit include dose, timing of administration, and baseline sleep quality, with greater improvements observed when melatonin is taken 1 to 3 hours before bedtime and among individuals with poorer sleep quality or circadian rhythm disorders.

Effects of Melatonin on Sleep Efficiency

Evidence shows that melatonin supplementation produces a small but measurable improvement in sleep efficiency in adults. Meta-analyses in general adult samples report average increases of approximately 2 to 4 percent, with one pooled estimate showing a 2.2 percent improvement and analyses restricted to healthy adults or those with insomnia showing increases around 3.1 percent. Findings in older adults are mixed, with some studies showing no significant change and others reporting small improvements, particularly at higher doses. Research in specific clinical populations, such as adults with neurocognitive disorders or individuals taking beta-blockers, shows slightly larger improvements ranging from about 3 to 7 percent. Overall, the magnitude of improvement is modest, and the clinical significance may be limited for healthy adults, although benefits may be more meaningful in groups with sleep disruption or medical comorbidities.

Melatonin Supplementation and Cardiovascular Disease Risk

Evidence from large prospective cohort data indicates that melatonin supplementation is not associated with an increased risk of cardiovascular disease in adults. A pooled analysis of more than 159,000 participants followed for up to 23 years reported a multivariable-adjusted hazard ratio of 0.94 with a 95 percent confidence interval of 0.83 to 1.06 when comparing melatonin users with non-users, showing no significant increase or decrease in cardiovascular disease risk. This null association was consistent across men and women and remained stable after extensive adjustment for confounding factors. Additional clinical and mechanistic research suggests that melatonin may exert cardioprotective effects, including lowering blood pressure, improving endothelial function, reducing oxidative stress, and decreasing platelet aggregation. Low endogenous melatonin levels have been linked to higher cardiovascular risk, and supplementation may help mitigate related risk factors such as hypertension and inflammation.

Trends in Melatonin Overdose and Poisoning Reports

Evidence shows a substantial rise in melatonin-related overdose reports and medical encounters in the United States, particularly among children. Data from US poison control centers indicate that pediatric melatonin ingestions increased by approximately 530 percent between 2012 and 2021, growing from over eight thousand to more than fifty-two thousand annual reports, and melatonin became the most frequently ingested substance reported for children in 2020. Hospitalizations and serious medical outcomes also increased over this period, with the largest rise seen in unintentional ingestions among children five years of age and younger. Emergency

department visits for unsupervised melatonin exposures in young children likewise increased by more than 400 percent from 2009 to 2020. These trends appear to be influenced by greater melatonin sales, broad over-the-counter availability, and child-attractive formulations such as gummies.

Effects of High-Dose Melatonin on Testicular Development in Juvenile Rodents

Evidence from animal research indicates that high-dose melatonin does not cause testicular atrophy or impair testicular development in juvenile male rodents. Studies administering melatonin alone at doses ranging from 10 to 25 milligrams per kilogram report no reductions in testicular weight, no abnormalities in seminiferous tubule structure, and no impairments in sperm parameters when compared with controls. In experimental models where testicular injury is induced by toxins, chemotherapy agents, radiation, or endocrine disruptors, melatonin consistently shows protective effects, including reduced oxidative stress and apoptosis, preservation of testicular weight, and improved sperm count and motility.

One early study from 1969 reported a slight reduction in testicular weight in prepubertal rats given very high melatonin doses under constant light exposure, although this was not accompanied by histological atrophy or hormonal disruption and has not been replicated in modern research. Overall, the current body of evidence shows that melatonin does not harm testicular development in juvenile rodents and is more often protective in contexts of testicular stress or injury.

References 86-106.

Effects of Anticipatory Anxiety on Sleep

> *"when we create this anticipatory anxiety, the amount of deep sleep that you have drops significantly, you don't sleep as well."*

Evidence shows that anticipatory anxiety disrupts sleep by increasing both physiological and cognitive arousal before bedtime. Heightened worry about upcoming events is associated with elevated heart rate, mental rumination, and emotional distress, all of which make it more difficult to initiate and maintain sleep. Studies indicate that higher levels of evening anticipatory stress predict longer sleep onset latency and poorer subjective sleep quality, particularly in individuals who do not

have chronic insomnia or depression. Research also suggests that pre-sleep cognitive and emotional arousal can fragment REM sleep, contributing to more frequent awakenings and negatively toned dreaming. These disruptions often lead to next-day fatigue, increased sleepiness, and impaired emotion regulation. The relationship between anticipatory anxiety and sleep is bidirectional, since disrupted sleep heightens stress reactivity and can intensify anticipatory anxiety, creating a reinforcing cycle.

References 107-113.

The QQRT Framework (Quantity, Quality, Regularity, and Timing)

> *"Relative to your own subjective perception of did I sleep well or not. Science really teaches, at least in my mind, that there are four pillars, four macros of good sleep. And you can think of this under the acronym QQRT (...) Quantity, Quality, Regularity and Timing (...)*
>
> *There's a study that also came out of that same data set that I described. It's called the UK Biobank Data (...) they looked at 60,000 individuals and they decided that they were going to compare and split them into quartiles. So the most regular to the second, most regular to then sort of the third, most regular. And then the final quartile was those who were the least regular (...)*
>
> quality seems to be as predictive as quantity in making a difference, not just to your all cause mortality, but quality even more than quantity when it comes to mental health (...)"

The QQRT Framework for Evaluating Sleep Health

The QQRT framework describes four core dimensions of healthy sleep: quantity, quality, regularity, and timing.

- Quantity refers to obtaining an age-appropriate amount of sleep, with most adults requiring seven to nine hours per night and a smaller proportion functioning well at slightly lower or higher durations.
- Quality reflects how restorative sleep is and includes fewer awakenings, minimal time spent awake after sleep onset, strong deep non-REM sleep, and a subjective sense of refreshment on waking.

- Regularity concerns going to bed and waking at the same times each day, since consistent sleep–wake patterns support circadian stability and are associated with better long-term health outcomes, including lower mortality risk.

- Timing involves sleeping in alignment with one's chronotype, whether lark, owl, or neutral, because misalignment between biological preference and daily schedule can degrade sleep quality and contribute to fatigue, emotional difficulties, metabolic disruption, and increased substance use.

Together, these four macros offer a simple structure for understanding what constitutes healthy sleep.

Sleep Regularity and Mortality Risk

A 2024 UK Biobank study examined 60,977 adults who wore wrist accelerometers for one week to determine how regular their sleep–wake patterns were, quantified using the Sleep Regularity Index. Participants were divided into five groups from least to most regular sleepers, and mortality outcomes were tracked for up to eight years. Individuals in the least regular group had significantly higher risks of all-cause, cardiovascular, and cancer mortality compared with those whose sleep was more regular. After full adjustment for demographic, lifestyle, and health factors, people in the top four regularity groups showed 20 to 48 percent lower mortality risk relative to the least regular group. The study also compared sleep regularity with average sleep duration and found that regularity was a stronger predictor of mortality than sleep duration alone. The findings suggest that maintaining consistent bedtimes and wake times may be an important and independent marker of long-term health.

Sleep Regularity and Risk of Depression and Anxiety

A 2025 UK Biobank study followed 79,666 adults without depression or anxiety at baseline to examine whether sleep regularity (measured objectively over seven days using the Sleep Regularity Index) predicts later mental-health outcomes. Participants were classified as irregular, moderately irregular, or regular sleepers based on the consistency of their sleep–wake timing across the week. During a median follow-up of 6.6 years, individuals with regular sleep patterns had substantially lower risks of developing depression and anxiety. After full adjustment for sleep duration, chronotype, sociodemographic factors, lifestyle behaviours, and health status, regular sleepers showed a 38 percent lower risk of incident depression and a 33 percent lower risk of incident anxiety

compared with irregular sleepers. Importantly, these associations persisted even among people who already met recommended sleep-duration guidelines, indicating that regularity contributes independently to mental-health protection. The authors conclude that maintaining consistent sleep timing may be a critical factor for long-term psychological wellbeing.

References 114-116.

Short Sleep Duration and All-Cause Mortality

> *"the shorter your sleep. The shorter your life. Short sleep predicts all cause mortality."*

Evidence from multiple meta-analyses and large prospective cohort studies shows that short sleep duration predicts a higher risk of all-cause mortality. Adults who regularly sleep fewer than seven hours per night have an estimated 7 to 14 percent higher mortality risk compared with those sleeping seven to eight hours, even after adjusting for factors such as age, sex, lifestyle behaviours, and comorbid health conditions. This association is part of a broader U- or J-shaped pattern in which both short and long sleep durations are linked to elevated mortality, with the lowest risk occurring in the midrange. Some studies suggest that the effect of short sleep may be stronger in women and in people with underlying medical conditions. Proposed mechanisms linking short sleep to mortality include increased cardiovascular risk, metabolic disruption, and impaired immune function. Recent research also indicates that sleep regularity may be an even stronger predictor of mortality risk than sleep duration alone.

References 117-123.

Effects of Reducing Evening Light on REM Sleep

> *"There's a great study where they did something similar to what I'm telling you now. 90 minutes before bed, they turned down the lights to below 30 lux and they pulled out all of the blue light and just that trick of dropping the lights down 90 minutes before bed below 30 lux making it warm yellow light, increase their REM sleep by 18%."*

Research indicates that lowering evening light levels and removing blue wavelengths before bedtime can increase REM sleep and improve its consolidation in healthy adults. A randomised crossover trial found that spending five evenings in a blue-depleted light environment increased REM sleep duration and reduced REM sleep fragmentation and microarousals when compared with normal indoor lighting of similar brightness. These effects occurred without reducing deep sleep and were partly independent of circadian phase shifts. Additional evidence shows that evening exposure to blue light disrupts sleep by suppressing melatonin, increasing alertness, and delaying sleep onset, while interventions that block blue light can improve subjective sleep quality and total sleep time. Effects on REM sleep are strongest in healthy adults, with mixed but generally positive findings in individuals with sleep disturbances..

References 124-129.

Neural Mechanisms Regulating Sleep and Circadian Rhythms

"this is the thalamus, this is the sensory gait of your brain. So all of your five senses, sound, touch, taste, smell, they all flood into this gate called the thalamus. And then the thalamus will decide whether it sends those sensory signals up to your cortex. And when it sends the SA signals up to your cortex, you start processing them and you become consciously aware of the external world. Now, as we're falling asleep, just as an aside, what's interesting is that this gate, the sensory gait, the thalamus, once we start to fall asleep, the gait will close shut. Now your eyes are technically still seeing, your ears are still hearing, your tongue is still tasting. But because the gate of the thalamus, the sensory gait closes shut, those signals that are coming into your brain are no longer sent up to your cortex (...)

You've fallen asleep. Now, the hypothalamus (...) within that structure contains a nucleus and that group of cells, the nucleus has a fancy term and it's called the supra charismatic nucleus (...) the supra charismatic nucleus is your master 24 hour clock (...) in the super charismatic nucleus, you get the 24 hour rhythm of being awake and being asleep, being awake and being asleep (...) The way it does that is that it uses signals such as light and dark (...) So every time that you are going to bed at the same time and waking up at the same time, you are feeding the supra charismatic nucleus, the master 24 hour clock in your brain, you are feeding it signals of regularity, and when it feeds on signals of regularity, it improves the quantity and the quality of your sleep."

Thalamic Relay of Sensory Information

Evidence shows that the thalamus is responsible for relaying sensory information from the senses to the cortex, with the exception of olfactory inputs. Sensory pathways for vision, hearing, and somatosensation project to specific thalamic nuclei, which then transmit these signals to corresponding cortical regions in organised topographic patterns. The thalamus is not only a relay but also an active regulator, integrating ascending sensory inputs with descending cortical signals and modulating the flow of information according to behavioural context. This modulation supports processes such as attention and arousal. Olfactory information is the primary exception, since it bypasses the thalamus and projects directly to the olfactory cortex.

Thalamic Gating During the Transition to Sleep

Evidence indicates that thalamic gating of sensory input increases as humans fall asleep, resulting in reduced transmission of sensory signals to the cortex. During the transition from wakefulness to non-REM sleep, the reticular thalamic nucleus inhibits thalamocortical relay cells, decreasing the flow of sensory information and contributing to sleep onset. Electrophysiological studies show reduced relay neuron output and increased inhibitory activity during non-REM sleep, and human imaging studies demonstrate decreased thalamocortical connectivity that corresponds to higher sensory thresholds. Although some thalamic-cortical connectivity returns during REM sleep, external sensory input remains largely suppressed. This enhanced gating is considered a core mechanism supporting sleep maintenance and the reduction of responsiveness to the external environment.

Circadian Regulation by the Suprachiasmatic Nucleus

Evidence shows that the suprachiasmatic nucleus in the hypothalamus is the master regulator of the brain's 24 hour circadian rhythm. This cluster of neurons generates intrinsic daily oscillations in gene expression and electrical activity, and it synchronises peripheral clocks throughout the body to maintain coordinated physiological timing. Its timekeeping mechanism is supported by transcriptional-translational feedback loops involving core clock genes, as well as intercellular signalling through neuropeptides neuropeptides (signalling molecules used by neurons) and GABA (an inhibitory neurotransmitter that reduces neuronal activity). The suprachiasmatic nucleus receives light input from the retina, allowing environmental light-dark cycles to entrain internal rhythms, and it communicates timing information to other brain regions and organs through neural and hormonal pathways.

References 130-149.

Imagery-Based Distraction and Sleep Onset

> *"the next thing you can do is take yourself on a mental walk. There's a great study from my university, the University of Berkeley, California (...) Alison Harvey did it (...) what she found was that (...) if you think about a walk that you know in great vivid detail (...) you get your mind off itself (...) What she found is that that increases the speed with which you fall back asleep. significantly."*

A 2002 study showed that engaging in a vivid, interesting mental image before sleep can reduce the time it takes to fall asleep in individuals with insomnia. In this study, participants with long-standing sleep difficulties were randomly assigned to one of three strategies: an imagery distraction task, a general distraction instruction, or no instruction. The imagery distraction group was trained to select and elaborate a pleasant, engaging scene in detail, which they then used during the pre-sleep period. Participants who used imagery reported significantly shorter sleep onset latency than those who received no instructions, and they also rated their thoughts as less distressing during the pre-sleep period. The imagery task was experienced as more engaging than the strategies used in the other groups, and most participants reported that their visual imagery was moderately vivid or clearer. The results suggest that a specific and engaging mental image can effectively occupy cognitive resources that would otherwise be taken up by worries or intrusive thoughts, thereby helping individuals fall asleep more quickly.

Reference 150.

Structure of Human Sleep Architecture

> *"So we go from light non-REM and then we go down into stage two non-REM. Then we go down into deep sleep and then we're going to rise back up into light stage two non-REM sleep. We're going to stay there and after about 70, 80 minutes you're going to pop up and you'll have a short REM sleep period (...) You go down into non-REM sleep and then up into rem, down into non-REM sleep, and you'll go up into rem, down into non-REM sleep and up into rem (...) so we go down into non-REM sleep and then up into REM in this 90 minute cycle. The problem is that it's on average 90 minutes. It ranges from 70 minutes to, to 120 minutes from one individual to the next (...)"*

Human sleep architecture consists of a repeating pattern of non REM sleep and REM sleep that cycles throughout the night. Each cycle typically lasts 90 to 110 minutes and occurs four to six times per night.

- Non REM sleep contains three stages: N1, which is the lightest stage and marks the transition from wakefulness; N2, which features sleep spindles (brief bursts of fast brain activity) and K complexes (large single waves that help protect sleep),

represents over half of total sleep time; and N3, also called slow wave sleep, which is the deepest and most restorative stage.

- REM sleep follows these stages and is characterised by rapid eye movements, vivid dreaming, and near complete muscle atonia.

The distribution of these stages changes across the night, with slow wave sleep dominating the early cycles and REM sleep periods lengthening in later cycles. Sleep architecture is dynamic and shaped by factors such as age, sex, circadian rhythms, and individual traits, meaning that the exact sequence and duration of each stage can vary. Sleep structure is measured using polysomnography, and its patterns are often visualised in a hypnogram that shows how the brain moves through each stage across the night.

Variation in the Duration of NREM–REM Sleep Cycles

Evidence shows that the duration of a full NREM–REM sleep cycle varies considerably across individuals. Large studies demonstrate substantial interindividual differences, with average cycle lengths centred around 90 to 100 minutes but ranging widely from about 70 to 150 minutes. These differences are stable over time within a person and are considered phenotypic, meaning they reflect trait-like characteristics rather than night-to-night fluctuations. Factors such as age, sex, genetics, sleep pressure, and circadian rhythms all influence the duration of NREM and REM periods. For example, older adults often show longer NREM episodes and shorter REM episodes, and genetics can shape the length of both NREM and REM stages, sometimes in sex-specific ways. The overall pattern indicates that sleep cycle duration is not fixed but varies in predictable ways across individuals.

References 151-161.

The Functional Roles of Dreaming

“these deep emotional centers of the brain (...) called the amygdala (...) Those can be up to 40% more active when you're in dream sleep than when you're awake. Dream sleep is rapid eye movement sleep, REM sleep (...)

So we dream for at least two different reasons. The first reason is emotional first aid. REM sleep is what I've defined as overnight therapy. And it's during dream sleep where your brain takes difficult, painful, emotional experiences, and it acts like a nocturnal soothing balm. And it just takes the sharp edges off those difficult, painful experiences (...) REM sleep is the only time during the 24 hour period where the brain shuts off a stress related neurochemical called noradrenaline. Now you've heard of the sister chemical downstairs in your body called adrenaline. Well, upstairs in the brain, we don't have adrenaline. We have no adrenaline. REM sleep is the only time where it's completely shut off. And what we put forward is a theory called overnight therapy, where your brain has, as I told you, these emotional centers and these memory centers, the emotional centers, the amygdala, the memory centers, the hippocampus. You reactivate those structures when you go into dream sleep. So your brain gets the chance to reactivate and replay and reprocess emotional experiences (...)

But there's a second completely independent benefit of dreaming. Dreaming is a form of informational alchemy. Dreaming is creativity (...) It's almost as though we go to sleep with the pieces of the jigsaw, but by way of dreaming, we wake up with a puzzle complete. And when you start to fuse things that shouldn't, should not normally go together, but when they do every now and again cause a marked advance in evolutionary fitness, that is the biological basis of creativity.”

Activation of Emotional Brain Regions During REM Sleep

Research shows that emotional brain regions are more active during REM sleep than during wakefulness. Neuroimaging studies report increased activity in structures such as the amygdala, which is involved in processing emotional significance, the anterior cingulate cortex, which helps regulate emotional conflict, the medial prefrontal cortex, which supports emotional control, and the insula, which contributes to internal emotional awareness. REM sleep is also characterised by prominent theta and beta oscillations, which are patterns of brainwave activity that support learning and emotional memory consolidation. These patterns allow for synaptic plasticity, meaning that

connections between neurons can strengthen or weaken in ways that help reshape emotional responses. Together, these findings indicate that REM sleep provides a neural environment that supports the processing, consolidation, and regulation of emotional experiences.

REM Sleep and the Processing of Emotional Experiences

Evidence shows that REM sleep supports the processing of emotional experiences and can reduce their emotional intensity over time. Studies demonstrate that REM sleep facilitates the consolidation of emotional memories, meaning that emotionally significant events become more stable and easier to recall after REM-rich sleep. Research also indicates that REM sleep is associated with decreased amygdala reactivity and lower subjective emotional responses to previously encountered emotional stimuli, suggesting a reduction in emotional charge. This effect is linked to characteristic features of REM sleep, including reduced adrenergic activity and specific patterns of brain oscillations that support emotional memory consolidation. Some findings show that emotional responses may be heightened shortly after sleep but decline over longer periods, indicating a dynamic process in which REM sleep both activates and ultimately helps resolve emotional experiences. Disrupting REM sleep impairs emotional regulation and increases next-day emotional reactivity, highlighting the role of REM sleep in adaptive emotional processing.

Dreaming and the Formation of Novel Creative Associations

Evidence indicates that dreaming can enhance creative problem solving and promote novel associations between ideas. Studies show that REM sleep, which is the stage of sleep richest in vivid dreaming, supports the formation of associative networks and the integration of information that is not normally linked, leading to improved performance on creative problem solving tasks. Research also highlights the role of the sleep onset period, known as N1 or hypnagogia, where targeted dream incubation has been shown to increase creative output and expand the semantic distance of ideas, meaning that people generate responses that are more conceptually distant from one another. Higher dream recall frequency is associated with greater creativity and divergent thinking, and individuals who frequently remember or elaborate on their dreams often report that dreams inspire creative ideas. Together, these findings suggest that dreaming provides a mental state in which the brain combines memories and concepts in unusual ways that can support creative insight.

Noradrenaline Suppression During REM Sleep

Evidence shows that noradrenaline levels in the brain are sharply reduced during REM sleep. Noradrenergic neurons in regions such as the locus coeruleus, which normally release noradrenaline to support alertness and arousal, become almost completely inactive during REM sleep. This leads to a significant decline in noradrenaline concentrations across brain areas including the cortex, hippocampus, and thalamus. Microdialysis and other neurochemical studies confirm this drop in noradrenaline during REM. The suppression of noradrenergic activity is essential for the generation of REM sleep and its characteristic features, since artificially increasing noradrenaline can block REM sleep while reducing it can help initiate REM.

Adrenaline in the Brain Compared With Noradrenaline

Evidence shows that adrenaline is present in the human brain, but in far smaller amounts and with a more limited role than noradrenaline. Adrenaline is found mainly in specific brainstem and hypothalamic regions and is produced only in areas that contain phenylethanolamine N methyltransferase, the enzyme required for its synthesis. Noradrenaline, by contrast, is widely distributed throughout the brain and is released primarily by neurons in the locus coeruleus, a region involved in arousal and attention. Noradrenaline functions as a major neurotransmitter in the central nervous system and plays key roles in regulating vigilance, mood, and stress responses. Adrenaline can act on adrenergic receptors within the brain, but its influence is minor compared with that of noradrenaline, since most adrenaline in the body functions as a hormone released from the adrenal medulla into the bloodstream.

References 162-194.

Nightmares, Psychological Wellbeing, and Emotional Regulation

“there is now a better treatment for nightmares if you are suffering from nightmares. And it's called image rehearsal therapy or IRT. And it works through an incredible mechanism of memory (...) called Memory Reconsolidation (...)

The definition of nightmare disorder is that you have to have these dreams frequently, maybe at least twice a week. They have to wake you up outta your sleep, and you have vivid recall. Plus, it has to cause you some kind of daytime distress where you're not feeling good about the day. It's causing you mental anguish. And furthermore, if it starts to lead to hopelessness or a sense that your life is not worth living, you absolutely need to go and see someone. And here's what we've discovered about nightmares. Short sleep duration, not getting enough sleep, sleeping less than six hours, predicts by about a hundred to 150% higher percentage chance of you having suicidal thoughts attempting suicide or tragically suicide completion (...) So if short sleep has 150% higher likelihood of suicidality, having nightmares has an 800% higher likelihood of suicidal tendencies associated with it (...)

Normative dreaming, even if they're bad dreams, is beneficial (...) There was a study done by a late sleep scientist, a woman called Rosalind Cartwright, and she was studying people who'd gone through a difficult period, let's say bereavement or very bitter divorce, and they had become depressed because of that experience. And around the time of the experience, she was seeing them as a therapist and she was having them do dream recall. So she was having them create dream diaries every single night. And then she tracked these patients for a year, and it turned out about roughly half of them by 12 months later, had remitted from their depression (...) Those individuals that went on to gain remission to their depression and get better, those people were dreaming of the events itself at the time that they were happening. Whereas those other people who didn't go on to gain clinical remission, they were dreaming, they just weren't dreaming of the experiences itself.”

Imagery Rehearsal Therapy for the Treatment of Nightmares

Evidence shows that imagery rehearsal therapy, often called IRT, is an effective treatment for reducing the frequency and distress of nightmares. Research demonstrates that IRT leads to meaningful improvements in adults, children, and individuals with psychiatric conditions such as post traumatic stress disorder and borderline personality disorder. Meta analyses and systematic reviews

report large and sustained reductions in nightmare frequency and better sleep quality, with improvements lasting for up to a year after treatment. IRT is as effective as prazosin, a commonly used medication for trauma related nightmares, and works well when delivered individually, in groups, or in brief and self guided formats. Around 30 percent of patients may not fully respond and dropout rates can be higher in populations with severe trauma, but overall the evidence supports IRT as a first line psychological treatment recommended by major sleep and psychiatric organisations.

Definition and Features of Nightmare Disorder

Nightmare disorder is a recognised sleep disorder marked by frequent and distressing nightmares that significantly disrupt sleep and daily functioning. These nightmares are typically long, vivid, and emotionally negative, often involving threats to safety or survival. They usually occur during rapid eye movement, or REM, sleep and lead to awakenings in which the person is quickly alert and aware of their surroundings. A diagnosis requires that the nightmares cause clinically significant distress or impairment in social, occupational, or other important areas of life, and that the symptoms are not better explained by medication, substance use, or another mental or medical condition. Nightmare disorder affects an estimated 2 to 8 percent of the general population, with higher rates in people with post traumatic stress disorder or mood disorders, and is associated with difficulties such as insomnia, anxiety, depression, and reduced quality of life.

Short Sleep Duration and Risk of Suicidal Thoughts and Behaviours

Evidence shows that short sleep duration is a significant predictor of suicidal ideation and suicide attempts in both adolescents and adults. Large scale and longitudinal studies consistently find that sleeping less than about six hours per night is associated with higher rates of suicidal thoughts, and this relationship remains even after accounting for factors such as depression and anxiety. Research also indicates that short sleep increases the likelihood of suicide attempts, with some studies showing a dose-response pattern in which progressively shorter sleep is linked to greater risk. Although the evidence for suicide completion is less extensive, meta analyses and longitudinal studies identify sleep disturbance as a predictor of suicide deaths with a medium effect size. The relationship between sleep duration and suicidality is often U shaped, meaning that both short and long sleep durations can increase risk, though short sleep shows the most consistent association. Vulnerable groups, including adolescents, sexual and gender minorities, and individuals with psychiatric comorbidities, appear particularly affected.

Dream Content and Emotional Adjustment After Marital Separation

A 2006 study led by Rosalind Cartwright examined whether the dream content of recently separated individuals predicted their emotional adjustment over the course of a year. Participants spent one night in the sleep laboratory during the early stages of their separation, where their REM sleep dream reports were collected following monitored awakenings. Dream samples were scored for the presence and frequency of the spouse image, the intensity of sadness, and the degree of intra-dream memory integration. Participants were then tracked for one year, during which their depressive symptoms were reassessed.

The researchers found that those who subsequently showed improvement in depression had dreams that contained more developed and personally relevant imagery, including more frequent appearances of the spouse and stronger emotional processing within the dream. In contrast, participants whose depression persisted tended to produce more bland, vague, or less emotionally engaged dreams. Regression analyses showed that individuals who retained higher levels of depression at follow-up reported dreams that were less well developed, contained more neutral emotional content, and showed weaker integration of the stressor into dream imagery. These results suggest that the way stressful experiences are incorporated into dreams may support emotional adjustment, and that richer, more emotionally engaged dream content is associated with better long-term mood outcomes.

References 195-214.

Magnesium

> *“there is one form of magnesium that seems to have some evidence in favor of it. It's called Magnesium L-Threonate (...)*
>
> *there may be an indirect benefit of magnesium in that it does seem to relax muscles. And when the body is in a state of relaxation, it sends a signal of relaxation back up a branch of nerves called the vagus nerve that goes up to the brain and signals to your brain. You are starting to relax down and you get the state of quiescence, and that's very helpful for sleep.”*

Magnesium L-Threonate and Sleep Quality

Current evidence suggests that magnesium L-threonate can improve sleep quality in adults who report sleep difficulties. A recent randomised, double blind, placebo controlled trial found that taking magnesium L-threonate for 21 days led to significant improvements in both subjective and objective sleep measures. Participants receiving the supplement showed better deep sleep and REM sleep on wearable device recordings, along with improved readiness and daytime functioning, whereas sleep quality declined in the placebo group. Self-reported measures also indicated better morning alertness, energy, mood, and overall sleep satisfaction. Broader research on magnesium supplements in general shows mixed results, with inconsistent findings across different forms of magnesium, but magnesium L-threonate may have stronger effects because of its ability to reach the brain more effectively. More large scale and independent studies are needed, but current evidence supports a beneficial effect of magnesium L-threonate on sleep quality in people with self-reported sleep problems.

Magnesium and Muscle Relaxation

Research shows that magnesium plays an essential physiological role in muscle function, including the regulation of muscle contraction and relaxation, but evidence that magnesium supplementation reliably enhances muscle relaxation in the general population is mixed. Large systematic reviews and randomised controlled trials indicate that magnesium does not meaningfully reduce muscle cramps or produce consistent muscle-relaxing effects in healthy adults or older individuals. Benefits appear more likely in specific contexts, such as magnesium deficiency or certain clinical settings, where magnesium's action as a calcium antagonist can reduce neuromuscular excitability. Some studies in athletes and physically active individuals also report modest reductions in muscle soreness after supplementation, although direct effects on muscle relaxation remain uncertain. Overall, magnesium contributes to muscle physiology, but routine supplementation has not been shown to consistently promote muscle relaxation in the general population.

Vagal Activity, Relaxation, and Sleep Onset

Research shows that increased vagal activity, which reflects greater activation of the parasympathetic nervous system, promotes relaxation and supports the transition into sleep. The parasympathetic system is the branch of the autonomic nervous system responsible for calming the body, lowering heart rate, and reducing physiological arousal. Studies demonstrate that techniques known to enhance vagal tone, such as slow, deep breathing or interventions that stimulate the vagus nerve, lead to increased feelings of relaxation and measurable decreases in stress markers, including

heart rate and blood pressure. Higher vagal activity is also linked to faster sleep initiation, better sleep quality, and fewer nighttime awakenings. During normal sleep onset, the body naturally shifts from sympathetic dominance, associated with alertness, to parasympathetic dominance, which supports the onset and maintenance of sleep. These findings indicate that strengthening parasympathetic activity can be beneficial for relaxation and for falling asleep more easily.

References 215-233.

Effects of Ashwagandha and Phosphatidylserine on Stress

> *"ashwagandha and another compound called phosphatidylserine. Both of these supplements seem to (...) ratchet down the fight or flight branch of your nervous system, and they can also reduce the amount of cortisol that the body is releasing (...) both push you back over into the more quiescent, what we call parasympathetic nervous system branch."*

Ashwagandha and Stress Physiology

Research shows that ashwagandha supplementation can lower cortisol, the body's main stress hormone, and may also shift the nervous system toward a more relaxed state. Multiple high-quality reviews and clinical trials report that adults taking ashwagandha experience meaningful reductions in cortisol levels, particularly those under chronic stress. Some studies also find increases in heart rate variability, a physiological indicator of greater parasympathetic activity. The parasympathetic nervous system is the branch responsible for rest, recovery, and relaxation, and higher heart rate variability generally reflects reduced activation of the sympathetic, or "fight-or-flight", system. These findings suggest that ashwagandha not only lowers cortisol but may also help rebalance the autonomic nervous system in a way that promotes a calmer physiological state.

Phosphatidylserine and Stress Physiology

Studies show that phosphatidylserine can reduce the body's cortisol response to stress, though its effects on the sympathetic, or "fight-or-flight", branch of the nervous system are less consistent. Clinical trials using doses between 400 and 800 milligrams per day report that phosphatidylserine dampens the typical surge in cortisol that occurs during physical or psychological

stress. This suggests a stabilising effect on the hypothalamic-pituitary-adrenal axis, which regulates hormonal stress responses. In contrast, most studies find little evidence that phosphatidylserine directly reduces sympathetic activity, as measures such as heart rate and blood pressure generally remain unchanged. A small number of studies note more relaxed brain activity patterns after supplementation, but these findings are indirect. Overall, the strongest evidence indicates that phosphatidylserine helps control cortisol reactivity rather than directly lowering sympathetic nervous system activation.

References 234-250.

Insomnia, Cortisol, and Cognitive Behavioural Therapy

> *"that insomnia we can classify usually as at least one of two different types (...) One is called sleep onset insomnia. I can't fall asleep. The other is sleep maintenance insomnia. I can't stay asleep (...)*
>
> *With insomnia patients, they show (...) a beautiful rise [in cortisol] in the late morning hours. It starts to come down, but then you see two anomalies right before bed. It goes back up and then in the middle of the night, even when it's starting to rise, it will have this abnormal spike right in the middle of the night too. And what we believe is that that may in part, explain sleep onset insomnia. Cortisol should be coming down and it should stay low right before we go to bed (...)*
>
> *one of the most effective ways to help people sleep is called cognitive behavioral therapy for insomnia"*

Sleep Onset Insomnia and Sleep Maintenance Insomnia

Sleep-onset insomnia refers to difficulty falling asleep at the beginning of the night. People with this form of insomnia typically take more than about 30 minutes to fall asleep on a regular basis, often because of heightened mental or physiological arousal near bedtime. Sleep maintenance insomnia refers to difficulty staying asleep, such as waking repeatedly during the night or struggling to fall back asleep after an awakening. This pattern leads to fragmented sleep and reduced overall sleep efficiency. Both types of insomnia can occur separately or together, and each has different

underlying contributors, ranging from stress and circadian rhythm disruption to medical or age-related factors. Understanding the distinction helps guide treatment, since different mechanisms may require different therapeutic approaches.

Cortisol Patterns in Insomnia

Evidence shows that insomnia is associated with abnormal increases in cortisol, the body's primary stress hormone, particularly during the evening and night. Clinical studies and meta-analyses find that people with chronic insomnia tend to have higher cortisol levels than good sleepers, reflecting greater activation of the hypothalamic-pituitary-adrenal axis, the system that governs hormonal stress responses. These elevations often occur at times when cortisol should normally be low, especially before bedtime and in the early part of the night, which may contribute to difficulties falling asleep or staying asleep. Research also shows that insomnia is linked to a disrupted daily rhythm of cortisol secretion, including a flatter decline across the day or an exaggerated rise in the morning. Not all individuals with insomnia display these changes, but overall the evidence supports a pattern of heightened physiological arousal that interferes with normal sleep regulation.

Effectiveness of Cognitive Behavioural Therapy for Insomnia

Cognitive behavioural therapy for insomnia, often called CBT-I, is one of the most effective treatments for improving sleep in people with chronic insomnia. Large reviews of clinical trials show that CBT-I leads to meaningful improvements in the time it takes to fall asleep, the ability to stay asleep, overall sleep quality, and daytime functioning. These benefits are long-lasting and often continue for a year or more after treatment, making CBT-I more durable than sleep medications for long-term management. Both traditional face-to-face CBT-I and digital or internet-based versions are effective, with digital programmes offering similar improvements and greater accessibility. CBT-I also works well for people whose insomnia occurs alongside other conditions such as depression, PTSD, chronic pain, or cancer. Research in adolescents shows comparable benefits, including shorter time to fall asleep and longer total sleep time.

References 251-266.

Genetic Short Sleepers

> *"… genetic short sleepers. These are individuals who by way of a genetic mutation, can survive on as little as 6.25 hours of sleep. In other words, six hours and 15 minutes of sleep, and they show zero impairment in their brain or their body (...) They are more efficient sleepers, they can get done what you and I take eight hours to do. They can do it in six. "*

Genetic short sleepers, sometimes called natural short sleepers, are individuals who naturally sleep far less than average, often about four to six hours per night, yet function normally without the cognitive impairments, health problems, or daytime fatigue that typically accompany chronic sleep loss. This pattern is lifelong and arises from specific genetic variants that alter the brain's regulation of sleep need. Several rare mutations have been identified in families with this trait, including variants in genes such as DEC2/BHLHE41, NPSR1, GRM1, ADRB1, and SIK3. These genes influence cellular pathways involved in sleep regulation, and studies using animal models show that the same mutations reliably produce reduced sleep need without negative consequences. Genetic short sleeping is therefore understood as a biologically distinct sleep phenotype, rather than a form of insomnia or voluntary sleep restriction.

References 267-271.

Effects of Sleep Loss on Metabolism and Eating Patterns

> *"certainly what you eat will change how you sleep, but perhaps more powerfully is how you sleep dramatically changes how you eat and how you dispose of those calories and what happens inside of your body (...) when you are undersleep and taking on board calories, the way that you dispose of that energy is different (...)*
>
> *We then did a brain imaging study where we deprived you of sleep and we put you inside of a scanner and we showed you a whole range of foods from really unhealthy things to really healthy things, to low calorie, high calorie. When you are undersleep, despite them being the same exact food stimuli, your brain responded far more powerfully to the unhealthy sort of high carbohydrate, high sugar foods.*
>
> *there was a great study where they looked at people who were dieting and either getting sufficient sleep or not getting sufficient sleep. What was fascinating is that both of those groups, whether you are well slept or not well slept, you both lost weight. In fact, you lost about the same amount of weight. So you'd think, okay, so that's fine. The problem was if you looked at what you were losing, there was an issue. Those people who were dieting but not getting sufficient sleep, 70% of all the weight that they lost came from lean muscle mass and not fat."*

Sleep Loss and Eating Behaviour

Research shows that insufficient sleep reliably alters appetite and eating behaviour in ways that promote overeating. Studies find that short sleep disrupts the balance of appetite-regulating hormones, including increases in ghrelin, which stimulates hunger, and decreases in leptin, which signals fullness. However, recent evidence indicates that the strongest effects come from changes in the brain's reward and self-control systems rather than hormonal shifts alone. When people are sleep deprived, they show stronger cravings for energy-dense foods that are high in fat and sugar, increased snacking, and poorer overall diet quality. Neuroimaging studies demonstrate heightened activity in reward-related brain regions when viewing food cues, along with reduced activity in brain regions responsible for restraint and decision-making. These combined effects make unhealthy foods more appealing and harder to resist, leading many individuals to eat more than they would when fully rested.

Sleep Deprivation and Metabolic Function

Evidence shows that sleep deprivation has significant effects on metabolism, influencing how the body regulates glucose, manages hormones, and processes nutrients. Even short periods of restricted sleep reduce the body's sensitivity to insulin, the hormone that helps move glucose into cells. This leads to higher blood sugar levels and metabolic patterns that resemble those seen in type 2 diabetes. Sleep loss also disrupts hormones involved in appetite and energy balance, including increases in ghrelin, which stimulates hunger, decreases in leptin, which signals fullness, and rises in cortisol, a stress hormone that can promote fat storage. Studies examining blood chemistry after sleep deprivation find increases in circulating lipids and certain amino acids, which reflect broader disturbances in metabolic pathways. Although being awake longer slightly increases energy expenditure, this effect is small compared to the increase in calorie intake that typically follows sleep loss. As a result, sleep deprivation creates a positive energy balance that can contribute to weight gain and impaired metabolic health over time.

Sleep Loss and Neural Responsivity to High-Calorie Foods

Evidence from neuroimaging research shows that acute sleep deprivation alters the way the brain evaluates food, biasing individuals toward higher-calorie options. In a controlled crossover study, healthy adults completed two conditions: one night of normal sleep and one night of total sleep deprivation. After each condition, participants underwent functional MRI while viewing and rating a wide range of foods varying in caloric density. The study found that insufficient sleep reduced activity in cortical regions involved in higher-order evaluation and decision-making, while increasing activation in the amygdala, a subcortical structure associated with emotional and reward-driven responses. This shift in neural responsivity was accompanied by a heightened behavioural desire for calorie-dense foods. The findings indicate that lack of sleep weakens top-down regulatory control over food choices and enhances bottom-up reward signals, increasing the appeal of energy-rich foods even when the visual stimuli are identical across conditions.

Sleep Restriction and Dieting Outcomes

A 2018 study examined how insufficient sleep influences body-composition changes during intentional weight loss. In this 8-week randomised study, adults with overweight or obesity were assigned either to caloric restriction alone or to caloric restriction combined with repeated sleep restriction, in which time in bed was reduced on five nights per week by roughly one hour per night relative to the control condition. Both groups lost a similar amount of total body weight over the

intervention, but the composition of that weight loss differed. Evidence from the trial shows that participants who maintained adequate sleep lost a greater proportion of fat mass, whereas those undergoing sleep restriction lost less fat and proportionally more lean mass despite equivalent weight change. These findings indicate that reduced sleep can impair the metabolic quality of weight loss during dieting by shifting losses away from fat and toward fat-free mass.

References 272-282.

References

1. Hor, C., Yeung, J., Jan, M., Emmenegger, Y., Hubbard, J., Xenarios, I., Naef, F., & Franken, P. (2019). Sleep–wake-driven and circadian contributions to daily rhythms in gene expression and chromatin accessibility in the murine cortex. Proceedings of the National Academy of Sciences of the United States of America, 116, 25773 - 25783.

2. Jan, M., Jimenez, S., Hor, C., Dijk, D., Skeldon, A., & Franken, P. (2024). Model integration of circadian- and sleep-wake-driven contributions to rhythmic gene expression reveals distinct regulatory principles.. Cell systems.

3. Gaine, M., Chatterjee, S., & Abel, T. (2018). Sleep Deprivation and the Epigenome. Frontiers in Neural Circuits, 12.

4. Vanrobaeys, Y., Peterson, Z., Walsh, E., Chatterjee, S., Lin, L., Lyons, L., Nickl-Jockschat, T., & Abel, T. (2023). Spatial transcriptomics reveals unique gene expression changes in different brain regions after sleep deprivation. Nature Communications, 14.

5. Da Costa Souza, A., & Ribeiro, S. (2015). Sleep deprivation and gene expression.. Current topics in behavioral neurosciences, 25, 65-90.

6. Lyons, L., Vanrobaeys, Y., & Abel, T. (2023). Sleep and memory: The impact of sleep deprivation on transcription, translational control, and protein synthesis in the brain. Journal of Neurochemistry, 166, 24 - 46.

7. Thakral, M., Von Korff, M., McCurry, S., Morin, C., & Vitiello, M. (2019). Changes in dysfunctional beliefs about sleep after cognitive behavioral therapy for insomnia: A systematic literature review and meta-analysis.. Sleep medicine reviews, 49, 101230 .

8. Faccini, J., Joshi, V., Graziani, P., & Del-Monte, J. (2023). Beliefs about sleep: links with ruminations, nightmare, and anxiety. BMC Psychiatry, 23.

9. Rauf, B., Perach, R., Madrid-Valero, J., Denis, D., Sharpless, B., Poerio, G., French, C., & Gregory, A. (2023). The associations between paranormal beliefs and sleep variables. Journal of Sleep Research, 32.

10. Jolley, D., Dinnick, I., Burgin, L., Ryan, S., Morgan-Finn, O., & Muncer, S. (2025). Investigating the link between sleep quality and belief in conspiracy theories.. Journal of health psychology, 13591053251320598 .

11. Su, P., & He, M. (2023). Relationship between sleep quality and subjective well-being: resilience as a mediator and belief in a just world as a moderator. Frontiers in Psychiatry, 14.

12. Hom, M., Chu, C., Rogers, M., & Joiner, T. (2020). A Meta-Analysis of the Relationship Between Sleep Problems and Loneliness. Clinical Psychological Science, 8, 799 - 824.

13. Gordon, A., Carrillo, B., & Barnes, C. (2021). Sleep and social relationships in healthy populations: A systematic review.. Sleep medicine reviews, 57, 101428 .

14. Matthews, T., Danese, A., Gregory, A., Caspi, A., Moffitt, T., & Arseneault, L. (2017). Sleeping with one eye open: loneliness and sleep quality in young adults. Psychological Medicine, 47, 2177 - 2186.

15. Griffin, S., Williams, A., Ravyts, S., Mladen, S., & Rybarczyk, B. (2020). Loneliness and sleep: A systematic review and meta-analysis. Health Psychology Open, 7.

16. Studler, M., Gianotti, L., Lobmaier, J., Maric, A., & Knoch, D. (2024). Human Prosocial Preferences Are Related to Slow-Wave Activity in Sleep. The Journal of Neuroscience, 44.

17. Girardeau, G., & Lopes-Dos-Santos, V. (2021). Brain neural patterns and the memory function of sleep. Science (New York, N.Y.), 374, 560 - 564.

18. Al-Shama, R., Uleman, J., Pereira, M., Claassen, J., & Dresler, M. (2024). Cerebral blood flow in sleep: A systematic review and meta-analysis.. Sleep medicine reviews, 77, 101977 .

19. Lewis, L. (2021). The interconnected causes and consequences of sleep in the brain. Science, 374, 564 - 568.

20. Fultz, N., Bonmassar, G., Setsompop, K., Stickgold, R., Rosen, B., Polimeni, J., & Lewis, L. (2019). Coupled electrophysiological, hemodynamic, and cerebrospinal fluid oscillations in human sleep. Science, 366, 628 - 631.

21. Andrillon, T., Poulsen, A., Hansen, L., Léger, D., & Kouider, S. (2016). Neural Markers of Responsiveness to the Environment in Human Sleep. The Journal of Neuroscience, 36, 6583 - 6596.

22. Dang-Vu, T., Schabus, M., Desseilles, M., Sterpenich, V., Bonjean, M., & Maquet, P. (2010). Functional neuroimaging insights into the physiology of human sleep.. Sleep, 33 12, 1589-603.

23. Stevner, A., Stevner, A., Vidaurre, D., Cabral, J., Cabral, J., Rapuano, K., Nielsen, S., Tagliazucchi, E., Tagliazucchi, E., Laufs, H., Vuust, P., Deco, G., Woolrich, M., Someren, E., Someren, E., & Kringelbach, M. (2019). Discovery of key whole-brain transitions and dynamics during human wakefulness and non-REM sleep. Nature Communications, 10.

24. Whitehurst, L., Subramoniam, A., Krystal, A., & Prather, A. (2022). Links between the brain and body during sleep: implications for memory processing. Trends in Neurosciences, 45, 212-223.

25. Salas, R., Strowd, R., Ali, I., Soni, M., Schneider, L., Safdieh, J., Vaughn, B., Avidan, A., Jeffery, J., & Gamaldo, C. (2018). Incorporating sleep medicine content into medical school through neuroscience core curricula. Neurology, 91, 597 - 610.

26. Almohaya, A., Qrmli, A., Almagal, N., Alamri, K., Bahammam, S., Al-Enizi, M., Alanazi, A., Almeneessier, A., Sharif, M., & Bahammam, A. (2013). Sleep medicine education and knowledge among medical students in selected Saudi Medical Schools. BMC Medical Education, 13, 133 - 133.

27. Romiszewski, S., May, F., Homan, E., Norris, B., Miller, M., & Zeman, A. (2020). Medical student education in sleep and its disorders is still meagre 20 years on: A cross-sectional survey of UK undergraduate medical education. Journal of Sleep Research, 29.

28. Smith, G. (2018). A sleep medicine medical school curriculum. Neurology, 91, 587 - 588.

29. Mindell, J., Bartle, A., Wahab, N., Ahn, Y., Ramamurthy, M., Huong, H., Kohyama, J., Ruangdaraganon, N., Sekartini, R., Teng, A., & Goh, D. (2011). Sleep education in medical school curriculum: a glimpse across countries.. Sleep medicine, 12 9, 928-31 .

30. Novais, Y., Borges, J., Ferreira-Moraes, F., & Tamura, E. (2024). Sleep medicine and chronobiology education among Brazilian medical students. Brazilian Journal of Medical and Biological Research, 57.

31. Aoun, R., Zibara, V., Costanian, C., Attarian, H., & Bahous, S. (2021). Sleep Medicine Knowledge Among Graduating Medical Students in Lebanon During an Economic and Political Crisis: A Preliminary Survey. Sleep Science, 16, e300 - e309.
32. Gharibeh, T., & Mehra, R. (2010). Obstructive sleep apnea syndrome: natural history, diagnosis, and emerging treatment options. Nature and Science of Sleep, 2, 233 - 255.
33. Rundo, J. (2019). Obstructive sleep apnea basics. Cleveland Clinic Journal of Medicine, 86, 2 - 9.
34. Abbasi, A., Gupta, S., Sabharwal, N., Meghrajani, V., Sharma, S., Kamholz, S., & Kupfer, Y. (2021). A comprehensive review of obstructive sleep apnea. Sleep Science, 14, 142 - 154.
35. McNicholas, W., & Pevernagie, D. (2022). Obstructive sleep apnea: transition from pathophysiology to an integrative disease model. Journal of Sleep Research, 31.
36. Hirani, R., & Smiley, A. (2023). A Scoping Review of Sleep Apnea: Where Do We Stand?. Life, 13.
37. Laratta, C., Ayas, N., Povitz, M., & Pendharkar, S. (2017). Diagnosis and treatment of obstructive sleep apnea in adults. Canadian Medical Association Journal, 189, E1481 - E1488.
38. Trenkwalder, C., & Paulus, W. (2010). Restless legs syndrome: pathophysiology, clinical presentation and management. Nature Reviews Neurology, 6, 337-346.
39. Garcia-Malo, C., Romero-Peralta, S., & Cano-Pumarega, I. (2021). Restless Legs Syndrome - Clinical Features.. Sleep medicine clinics, 16 2, 233-247 .
40. Guo, S., Huang, J., Jiang, H., Han, C., Li, J., Xu, X., Zhang, G., Lin, Z., Xiong, N., & Wang, T. (2017). Restless Legs Syndrome: From Pathophysiology to Clinical Diagnosis and Management. Frontiers in Aging Neuroscience, 9.
41. Gossard, T., Trotti, L., Videnovic, A., & St. Louis, E. (2021). Restless Legs Syndrome: Contemporary Diagnosis and Treatment. Neurotherapeutics, 18, 140 - 155.
42. Koob, G., & Colrain, I. (2019). Alcohol use disorder and sleep disturbances: a feed-forward allostatic framework. Neuropsychopharmacology, 45, 141-165.

43. He, S., Hasler, B., & Chakravorty, S. (2019). Alcohol and sleep-related problems.. Current opinion in psychology, 30, 117-122 .

44. Gardiner, C., Weakley, J., Burke, L., Roach, G., Sargent, C., Maniar, N., Huynh, M., Miller, D., Townshend, A., & Halson, S. (2024). The effect of alcohol on subsequent sleep in healthy adults: A systematic review and meta-analysis.. Sleep medicine reviews, 80, 102030 .

45. Thakkar, M., Sharma, R., & Sahota, P. (2015). Alcohol disrupts sleep homeostasis.. Alcohol, 49 4, 299-310 .

46. Park, S., Oh, M., Lee, B., Kim, H., Lee, W., Lee, J., Lim, J., & Kim, J. (2015). The Effects of Alcohol on Quality of Sleep. Korean Journal of Family Medicine, 36, 294 - 299.

47. Gardiner, C., Weakley, J., Burke, L., Roach, G., Sargent, C., Maniar, N., Townshend, A., & Halson, S. (2023). The effect of caffeine on subsequent sleep: A systematic review and meta-analysis.. Sleep medicine reviews, 69, 101764 .

48. Clark, I., & Landolt, H. (2017). Coffee, caffeine, and sleep: A systematic review of epidemiological studies and randomized controlled trials.. Sleep medicine reviews, 31, 70-78

49. Weibel, J., Lin, Y., Landolt, H., Berthomier, C., Brandewinder, M., Kistler, J., Rehm, S., Rentsch, K., Meyer, M., Borgwardt, S., Cajochen, C., & Reichert, C. (2021). Regular Caffeine Intake Delays REM Sleep Promotion and Attenuates Sleep Quality in Healthy Men. Journal of Biological Rhythms, 36, 384 - 394.

50. Watson, E., Coates, A., Kohler, M., & Banks, S. (2016). Caffeine Consumption and Sleep Quality in Australian Adults. Nutrients, 8.

51. Weibel, J., Lin, Y., Landolt, H., Kistler, J., Rehm, S., Rentsch, K., Slawik, H., Borgwardt, S., Cajochen, C., & Reichert, C. (2021). The impact of daily caffeine intake on nighttime sleep in young adult men. Scientific Reports, 11.

52. Velzeboer, R., Malas, A., Boerkoel, P., Cullen, K., Hawkins, M., Roesler, J., & Lai, W. (2022). Cannabis Dosing and Administration for Sleep: A Systematic Review.. Sleep.

53. Babson, K., Sottile, J., & Morabito, D. (2017). Cannabis, Cannabinoids, and Sleep: a Review of the Literature. Current Psychiatry Reports, 19, 1-12.

54. Kuhathasan, N., Dufort, A., Mackillop, J., Gottschalk, R., Minuzzi, L., & Frey, B. (2019). The use of cannabinoids for sleep: A critical review on clinical trials.. Experimental and clinical psychopharmacology.

55. European Society of Cardiology. (2024, August 29). Catching up on sleep on weekends may lower heart disease risk by up to 20%.

56. Patterson, P., Ghen, J., Antoon, S., Martin-Gill, C., Guyette, F., Weiss, P., Turner, R., & Buysse, D. (2019). Does evidence support "banking/extending sleep" by shift workers to mitigate fatigue, and/or to improve health, safety, or performance? A systematic review.. Sleep health.

57. Vitale, K., Owens, R., Hopkins, S., & Malhotra, A. (2019). Sleep Hygiene for Optimizing Recovery in Athletes: Review and Recommendations. International Journal of Sports Medicine, 40, 535 - 543.

58. Rupp, T., Wesensten, N., Bliese, P., & Balkin, T. (2009). Banking sleep: realization of benefits during subsequent sleep restriction and recovery.. Sleep, 32 3, 311-21.

59. Parker, R., & Parker, P. (2016). The impact of sleep deprivation in military surgical teams: a systematic review. Journal of the Royal Army Medical Corps, 163, 158 - 163.

60. Axelsson, J., & Vyazovskiy, V. (2015). Banking Sleep and Biological Sleep Need.. Sleep, 38 12, 1843-5.

61. Leproult, R., Deliens, G., Gilson, M., & Peigneux, P. (2015). Beneficial impact of sleep extension on fasting insulin sensitivity in adults with habitual sleep restriction.. Sleep, 38 5, 707-15. .

62. Arnal, P., Sauvet, F., Léger, D., Van Beers, P., Bayon, V., Bougard, C., Rabat, A., Millet, G., & Chennaoui, M. (2015). Benefits of Sleep Extension on Sustained Attention and Sleep Pressure Before and During Total Sleep Deprivation and Recovery.. Sleep, 38 12, 1935-43 .

63. Famodu, O.; Montgomery-Downs, H.; Thomas, J.; Gilleland, D.L.; Bryner, R.W.; Olfert, M.D. Impact of a single week of sleep extension on performance, mood, and nutrition among female college track athletes. Sleep 2017, 40, A32.

64. Mah, C. D., Mah, K. E., Kezirian, E. J., & Dement, W. C. (2011). The effects of sleep extension on the athletic performance of collegiate basketball players. Sleep, 34(7), 943-950.

65. Ritland, B. M., Simonelli, G., Gentili, R. J., Smith, J. C., He, X., Mantua, J., ... & Hatfield, B. D. (2019). Effects of sleep extension on cognitive/motor performance and motivation in military tactical athletes. Sleep medicine, 58, 48-55.

66. Bonnar, D., Bartel, K., Kakoschke, N., & Lang, C. (2018). Sleep Interventions Designed to Improve Athletic Performance and Recovery: A Systematic Review of Current Approaches. Sports Medicine, 48, 683-703.

67. Cunha, L., Costa, J., Marques, E., Brito, J., Lastella, M., & Figueiredo, P. (2023). The Impact of Sleep Interventions on Athletic Performance: A Systematic Review. Sports Medicine - Open, 9.

68. Charest, J., & Grandner, M. (2020). Sleep and Athletic Performance: Impacts on Physical Performance, Mental Performance, Injury Risk and Recovery, and Mental Health.. Sleep medicine clinics, 15 1, 41-57.

69. Gwyther, K., Rice, S., Purcell, R., Pilkington, V., Santesteban-Echarri, O., Bailey, A., & Walton, C. (2022). Sleep interventions for performance, mood and sleep outcomes in athletes: A systematic review and meta-analysis. Psychology of Sport and Exercise.

70. Carter, B., Rees, P., Hale, L., Bhattacharjee, D., & Paradkar, M. (2016). Association Between Portable Screen-Based Media Device Access or Use and Sleep Outcomes: A Systematic Review and Meta-analysis.. JAMA pediatrics, 170 12, 1202-1208 .

71. Hysing, M., Pallesen, S., Stormark, K., Jakobsen, R., Lundervold, A., & Sivertsen, B. (2015). Sleep and use of electronic devices in adolescence: results from a large population-based study. BMJ Open, 5.

72. He, J., Tu, Z., Xiao, L., Su, T., & Tang, Y. (2020). Effect of restricting bedtime mobile phone use on sleep, arousal, mood, and working memory: A randomized pilot trial. PLoS ONE, 15.

73. Hartstein, L., Mathew, G., Reichenberger, D., Rodriguez, I., Allen, N., Chang, A., Chaput, J., Christakis, D., Garrison, M., Gooley, J., Koos, J., Van Den Bulck, J., Woods, H., Zeitzer, J., Dzierzewski, J., & Hale, L. (2024). The impact of screen use on sleep health across the lifespan: A National Sleep Foundation consensus statement.. Sleep health.

74. Perrault, A., Bayer, L., Peuvrier, M., Afyouni, A., Ghisletta, P., Brockmann, C., Spiridon, M., Vesely, S., Haller, D., Pichon, S., Perrig, S., Schwartz, S., & Sterpenich, V. (2019). Reducing the use of screen electronic devices in the evening is associated with improved sleep and daytime vigilance in adolescents.. Sleep.

75. Shechter, A., Kim, E., St-Onge, M., & Westwood, A. (2018). Blocking nocturnal blue light for insomnia: A randomized controlled trial.. Journal of psychiatric research, 96, 196-202 .

76. Chinoy, E., Duffy, J., & Czeisler, C. (2018). Unrestricted evening use of light-emitting tablet computers delays self-selected bedtime and disrupts circadian timing and alertness. Physiological Reports, 6.

77. Dresp-Langley, B., & Hutt, A. (2022). Digital Addiction and Sleep. International Journal of Environmental Research and Public Health, 19.

78. Chang, A., Aeschbach, D., Duffy, J., & Czeisler, C. (2014). Evening use of light-emitting eReaders negatively affects sleep, circadian timing, and next-morning alertness. Proceedings of the National Academy of Sciences, 112, 1232 - 1237.

79. Bauducco, S., Pillion, M., Bartel, K., Reynolds, C., Kahn, M., & Gradisar, M. (2024). A bidirectional model of sleep and technology use: a theoretical review of how much, for whom, and which mechanisms. Sleep Medicine Reviews, 76, 101933.

80. Sack, R., Auckley, D., Auger, R., Carskadon, M., Wright, K., Vitiello, M., & Zhdanova, I. (2007). Circadian rhythm sleep disorders: part II, advanced sleep phase disorder, delayed sleep phase disorder, free-running disorder, and irregular sleep-wake rhythm. An American Academy of Sleep Medicine review.. Sleep, 30 11, 1484-501 .

81. Lee, E. (2020). Advanced Sleep-Wake Rhythm Disorder. , 109-122.

82. Reid, K., Chang, A., Dubocovich, M., Turek, F., Takahashi, J., & Zee, P. (2001). Familial advanced sleep phase syndrome.. Archives of neurology, 58 7, 1089-94 .

83. Curtis, B., Ashbrook, L., Young, T., Finn, L., Fu, Y., Ptáček, L., & Jones, C. (2019). Extreme morning chronotypes are often familial and not exceedingly rare: the estimated prevalence of advanced sleep phase, familial advanced sleep phase, and advanced sleep-wake phase disorder in a sleep clinic population.. Sleep.

84. Hirano, A., Shi, G., Jones, C., Lipzen, A., Pennacchio, L., Xu, Y., Hallows, W., McMahon, T., Yamazaki, M., Ptáček, L., & Fu, Y. (2016). A Cryptochrome 2 mutation yields advanced sleep phase in humans. eLife, 5.

85. Pavlova, M. (2017). Circadian Rhythm Sleep-Wake Disorders. CONTINUUM: Lifelong Learning in Neurology, 23, 1051–1063.

86. Cruz-Sanabria, F., Bruno, S., Crippa, A., Frumento, P., Scarselli, M., Skene, D., & Faraguna, U. (2024). Optimizing the Time and Dose of Melatonin as a Sleep-Promoting Drug: A Systematic Review of Randomized Controlled Trials and Dose–Response Meta-Analysis. Journal of Pineal Research, 76.

87. Brzezinski, A., Vangel, M., Wurtman, R., Norrie, G., Zhdanova, I., Ben-Shushan, A., & Ford, I. (2005). Effects of exogenous melatonin on sleep: a meta-analysis.. Sleep medicine reviews, 9 1, 41-50 .

88. Auld, F., Maschauer, E., Morrison, I., Skene, D., & Riha, R. (2017). Evidence for the efficacy of melatonin in the treatment of primary adult sleep disorders.. Sleep medicine reviews, 34, 10-22 .

89. Ferracioli-Oda, E., Qawasmi, A., & Bloch, M. (2013). Meta-Analysis: Melatonin for the Treatment of Primary Sleep Disorders. PLoS ONE, 8.

90. Maruani, J., Reynaud, E., Chambe, J., Palagini, L., Bourgin, P., & Geoffroy, P. (2023). Efficacy of melatonin and ramelteon for the acute and long-term management of insomnia disorder in adults: A systematic review and meta-analysis. Journal of Sleep Research, 32.

91. Duffy, J., Wang, W., Ronda, J., & Czeisler, C. (2022). High dose melatonin increases sleep duration during nighttime and daytime sleep episodes in older adults. Journal of Pineal Research, 73.

92. Marupuru, S., Arku, D., Campbell, A., Slack, M., & Lee, J. (2022). Use of Melatonin and/on Ramelteon for the Treatment of Insomnia in Older Adults: A Systematic Review and Meta-Analysis. Journal of Clinical Medicine, 11.

93. Xu, H., Zhang, C., Qian, Y., Zou, J., Li, X., Liu, Y., Zhu, H., Meng, L., Liu, S., Zhang, W., Yi, H., Guan, J., Chen, Z., & Yin, S. (2020). Efficacy of melatonin for sleep disturbance in middle-aged primary insomnia: a double-blind, randomised clinical trial.. Sleep medicine, 76, 113-119 .

94. Scheer, F., Morris, C., Garcia, J., Smales, C., Kelly, E., Marks, J., Malhotra, A., & Shea, S. (2012). Repeated melatonin supplementation improves sleep in hypertensive patients treated with beta-blockers: a randomized controlled trial.. Sleep, 35 10, 1395-402 .

95. Hsu, W., Anderson, A., Rowles, W., Peters, K., Li, V., Stone, K., Ashbrook, L., Gelfand, A., & Bove, R. (2021). Effects of melatonin on sleep disturbances in multiple sclerosis: A randomized, controlled pilot study. Multiple Sclerosis Journal - Experimental, Translational and Clinical, 7.

96. Li, Y., Huang, T., Redline, S., Willett, W., Manson, J., Schernhammer, E., & Hu, F. (2024). Use of melatonin supplements and risk of type 2 diabetes and cardiovascular diseases in the USA: insights from three prospective cohort studies.. The lancet. Diabetes & endocrinology.

97. Pandi-Perumal, S., Bahammam, A., Ojike, N., Akinseye, O., Kendzerska, T., Buttoo, K., Dhandapany, P., Brown, G., & Cardinali, D. (2017). Melatonin and Human Cardiovascular Disease. Journal of Cardiovascular Pharmacology and Therapeutics, 22, 122 - 132.

98. Tobeiha, M., Jafari, A., Fadaei, S., Mirazimi, S., Dashti, F., Amiri, A., Khan, H., Asemi, Z., Reiter, R., Hamblin, M., & Mirzaei, H. (2022). Evidence for the Benefits of Melatonin in Cardiovascular Disease. Frontiers in Cardiovascular Medicine, 9.

99. Franco, C., Sciatti, E., Favero, G., Bonomini, F., Vizzardi, E., & Rezzani, R. (2022). Essential Hypertension and Oxidative Stress: Novel Future Perspectives. International Journal of Molecular Sciences, 23.

100. Ozkalayci, F., Kocabaş, U., Altun, B., Pandi-Perumal, S., & Altun, A. (2021). Relationship Between Melatonin and Cardiovascular Disease. Cureus, 13.

101. Lelak, K., Vohra, V., Neuman, M., Toce, M., & Sethuraman, U. (2022). Pediatric Melatonin Ingestions — United States, 2012–2021. Morbidity and Mortality Weekly Report, 71, 725 - 729.

102. Moradi, M., Goodarzi, N., Faramarzi, A., Cheraghi, H., Hashemian, A., & Jalili, C. (2021). Melatonin protects rats testes against bleomycin, etoposide, and cisplatin-induced toxicity via mitigating nitro-oxidative stress and apoptosis.. Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie, 138, 111481 .

103. Habiba, E., Harby, S., El-Sayed, N., Omar, E., Bakr, B., Augustyniak, M., El-Samad, L., & Hassan, M. (2023). Sericin and melatonin mitigate diethylnitrosamine-instigated testicular impairment in mice: Implications of oxidative stress, spermatogenesis, steroidogenesis, and modulation of Nrf2/WT1/SF-1 signaling pathways.. Life sciences, 122220.

104. Bahrami, N., Goudarzi, M., Hosseinzadeh, A., Sabbagh, S., Reiter, R., & Mehrzadi, S. (2018). Evaluating the protective effects of melatonin on di(2-ethylhexyl) phthalate-induced testicular injury in adult mice.. Biomedicine & pharmacotherapy = Biomedecine & pharmacotherapie, 108, 515-523.

105. Wang, Y., Zhao, T., Zhao, H., & Wang, H. (2018). Melatonin protects methotrexate-induced testicular injury in rats.. European review for medical and pharmacological sciences, 22 21, 7517-7525 .

106. Debeljuk, L. (1969). Effect of melatonin on the gonadotrophic function of the male rat under constant illumination.. Endocrinology, 84 4, 937-9.

107. Goldstein, A., Greer, S., Saletin, J., Harvey, A., Nitschke, J., & Walker, M. (2013). Tired and Apprehensive: Anxiety Amplifies the Impact of Sleep Loss on Aversive Brain Anticipation. The Journal of Neuroscience, 33, 10607 - 10615.

108. Block, V., Meyer, A., Miché, M., Mikoteit, T., Hoyer, J., Imboden, C., Bader, K., Hatzinger, M., Lieb, R., & Gloster, A. (2019). The effect of anticipatory stress and openness and engagement on subsequently perceived sleep quality–An Experience Sampling Method study. Journal of Sleep Research, 29.

109. Kalmbach, D., Anderson, J., & Drake, C. (2018). The impact of stress on sleep: Pathogenic sleep reactivity as a vulnerability to insomnia and circadian disorders. Journal of Sleep Research, 27.

110. Cox, R., & Olatunji, B. (2020). Sleep in the anxiety-related disorders: A meta-analysis of subjective and objective research.. Sleep medicine reviews, 51, 101282 .

111. Oh, C., Kim, H., Na, H., Cho, K., & Chu, M. (2019). The Effect of Anxiety and Depression on Sleep Quality of Individuals With High Risk for Insomnia: A Population-Based Study. Frontiers in Neurology, 10.

112. Brown, W., Wilkerson, A., Boyd, S., Dewey, D., Mesa, F., & Bunnell, B. (2018). A review of sleep disturbance in children and adolescents with anxiety. Journal of Sleep Research, 27.

113. Zhang, J., Li, X., Tang, Z., Xiang, S., Tang, Y., Hu, W., Tan, C., & Wang, X. (2024). Effects of stress on sleep quality: multiple mediating effects of rumination and social anxiety. Psicologia, Reflexão e Crítica : revista semestral do Departamento de Psicologia da UFRGS, 37.

114. Walker, M. (2024). What is good sleep? Sleep Diplomat.

115. Li, D. R., Li, Z. X., Li, M. H., Liu, B. Q., Fang, Q., Liu, J. C., ... & Wu, Q. J. (2025). Regular sleep patterns, not just duration, critical for mental health: association of accelerometer-derived sleep regularity with incident depression and anxiety. Psychological Medicine, 55, e239.

116. Windred, D. P., Burns, A. C., Lane, J. M., Saxena, R., Rutter, M. K., Cain, S. W., & Phillips, A. J. (2024). Sleep regularity is a stronger predictor of mortality risk than sleep duration: a prospective cohort study. Sleep, 47(1), zsad253.

117. Cappuccio, F., D'elia, L., Strazzullo, P., & Miller, M. (2010). Sleep duration and all-cause mortality: a systematic review and meta-analysis of prospective studies.. Sleep, 33 5, 585-92 .

118. Grandner, M., Hale, L., Moore, M., & Patel, N. (2010). Mortality associated with short sleep duration: The evidence, the possible mechanisms, and the future.. Sleep medicine reviews, 14 3, 191-203 .

119. Yin, J., Jin, X., Shan, Z., Li, S., Huang, H., Li, P., Peng, X., Peng, Z., Yu, K., Bao, W., Yang, W., Chen, X., & Liu, L. (2017). Relationship of Sleep Duration With All-Cause Mortality and Cardiovascular Events: A Systematic Review and Dose-Response Meta-Analysis of Prospective Cohort Studies. Journal of the American Heart Association: Cardiovascular and Cerebrovascular Disease, 6.

120. Kim, Y., Wilkens, L., Schembre, S., Henderson, B., Kolonel, L., & Goodman, M. (2013). Insufficient and excessive amounts of sleep increase the risk of premature death from cardiovascular and other diseases: the Multiethnic Cohort Study.. Preventive medicine, 57 4, 377-85 .

121. Liu, T., Xu, C., Rota, M., Cai, H., Zhang, C., Shi, M., Yuan, R., Weng, H., Meng, X., Kwong, J., & Sun, X. (2017). Sleep duration and risk of all-cause mortality: A flexible, non-linear, meta-regression of 40 prospective cohort studies.. Sleep medicine reviews, 32, 28-36 .

122. Yang, L., Xi, B., Zhao, M., & Magnussen, C. (2021). Association of sleep duration with all-cause and disease-specific mortality in US adults. Journal of Epidemiology & Community Health, 75, 556 - 561.

123. Da Silva, A., De Mello, R., Schaan, C., Fuchs, F., Redline, S., & Fuchs, S. (2016). Sleep duration and mortality in the elderly: a systematic review with meta-analysis. BMJ Open, 6.

124. Vethe, D., Drews, H., Scott, J., Engstrøm, M., Heglum, H., Grønli, J., Wisor, J., Sand, T., Lydersen, S., Kjørstad, K., Faaland, P., Vestergaard, C., Langsrud, K., & Kallestad, H. (2022). Evening light environments can be designed to consolidate and increase the duration of REM-sleep. Scientific Reports, 12.

125. Shechter, A., Kim, E., St-Onge, M., & Westwood, A. (2018). Blocking nocturnal blue light for insomnia: A randomized controlled trial.. Journal of psychiatric research, 96, 196-202 .

126. Knufinke, M., Fittkau-Koch, L., Møst, E., Kompier, M., & Nieuwenhuys, A. (2018). Restricting short-wavelength light in the evening to improve sleep in recreational athletes – A pilot study. European Journal of Sport Science, 19, 728 - 735.

127. Münch, M., Kobialka, S., Steiner, R., Oelhafen, P., Wirz-Justice, A., & Cajochen, C. (2006). Wavelength-dependent effects of evening light exposure on sleep architecture and sleep EEG power density in men.. American journal of physiology. Regulatory, integrative and comparative physiology, 290 5, R1421-8 .

128. Shechter, A., Quispe, K., Barbecho, J., Slater, C., & Falzon, L. (2020). Interventions to reduce short-wavelength (“blue”) light exposure at night and their effects on sleep: A systematic review and meta-analysis. Sleep Advances: A Journal of the Sleep Research Society, 1.

129. Chellappa, S., Steiner, R., Oelhafen, P., Lang, D., Götz, T., Krebs, J., & Cajochen, C. (2013). Acute exposure to evening blue-enriched light impacts on human sleep. Journal of Sleep Research, 22.

130. McCormick, D., & Krosigk, M. (1992). Corticothalamic activation modulates thalamic firing through glutamate "metabotropic" receptors.. Proceedings of the National Academy of Sciences of the United States of America, 89 7, 2774-8 .

131. Sherman, S. (2017). Functioning of Circuits Connecting Thalamus and Cortex.. Comprehensive Physiology, 7 2, 713-739.

132. Halassa, M., & Sherman, S. (2019). Thalamocortical Circuit Motifs: A General Framework. Neuron, 103, 762-770.

133. Sherman, S., & Guillery, R. (2002). The role of the thalamus in the flow of information to the cortex.. Philosophical transactions of the Royal Society of London. Series B, Biological sciences, 357 1428, 1695-708 .

134. Mease, R., Metz, M., & Groh, A. (2016). Cortical Sensory Responses Are Enhanced by the Higher-Order Thalamus.. Cell reports, 14 2, 208-15 .

135. Coenen, A. (2024). Sensory gating and gaining in sleep: the balance between the protection of sleep and the safeness of life (a review). Journal of Sleep Research, 33.

136. Overwiening, J., Tesler, F., Guarino, D., & Destexhe, A. (2024). A multi-scale study of thalamic state-dependent responsiveness. PLOS Computational Biology, 20.

137. Anderson, M., Mochizuki, T., Xie, J., Fischler, W., Manger, J., Talley, E., Scammell, T., & Tonegawa, S. (2005). Thalamic Cav3.1 T-type Ca2+ channel plays a crucial role in stabilizing sleep.. Proceedings of the National Academy of Sciences of the United States of America, 102 5, 1743-8

138. Picchioni, D., Pixa, M., Fukunaga, M., Carr, W., Horovitz, S., Braun, A., & Duyn, J. (2014). Decreased connectivity between the thalamus and the neocortex during human nonrapid eye movement sleep.. Sleep, 37 2, 387-97 .

139. Masson, G., Masson, S., Debay, D., & Bal, T. (2002). Feedback inhibition controls spike transfer in hybrid thalamic circuits. Nature, 417, 854-858.

140. Hale, J., White, T., Mayhew, S., Wilson, R., Rollings, D., Khalsa, S., Arvanitis, T., & Bagshaw, A. (2016). Altered thalamocortical and intra-thalamic functional connectivity during light sleep compared with wake. NeuroImage, 125, 657-667.

141. Brancaccio, M., Patton, A., Chesham, J., Maywood, E., & Hastings, M. (2017). Astrocytes Control Circadian Timekeeping in the Suprachiasmatic Nucleus via Glutamatergic Signaling. Neuron, 93, 1420 - 1435.e5.

142. Vadnie, C., & Mcclung, C. (2017). Circadian Rhythm Disturbances in Mood Disorders: Insights into the Role of the Suprachiasmatic Nucleus. Neural Plasticity, 2017.

143. Herzog, E., Hermanstyne, T., Smyllie, N., & Hastings, M. (2017). Regulating the Suprachiasmatic Nucleus (SCN) Circadian Clockwork: Interplay between Cell-Autonomous and Circuit-Level Mechanisms.. Cold Spring Harbor perspectives in biology, 9 1.

144. Hastings, M., Maywood, E., & Brancaccio, M. (2018). Generation of circadian rhythms in the suprachiasmatic nucleus. Nature Reviews Neuroscience, 19, 453 - 469.

145. McMahon, W., Ftouni, S., Phillips, A., Beatty, C., Lockley, S., Rajaratnam, S., Maruff, P., Drummond, S., & Anderson, C. (2020). The impact of structured sleep schedules prior to an in-laboratory study: Individual differences in sleep and circadian timing. PLoS ONE, 15.

146. Sletten, T., Weaver, M., Foster, R., Gozal, D., Klerman, E., Rajaratnam, S., Roenneberg, T., Takahashi, J., Turek, F., Vitiello, M., Young, M., & Czeisler, C. (2023). The importance of sleep regularity: a consensus statement of the National Sleep Foundation sleep timing and variability panel.. Sleep health.

147. Phillips, A., Clerx, W., O'Brien, C., Sano, A., Barger, L., Picard, R., Lockley, S., Klerman, E., & Czeisler, C. (2017). Irregular sleep/wake patterns are associated with poorer academic performance and delayed circadian and sleep/wake timing. Scientific Reports, 7.

148. Meyer, N., Harvey, A., Lockley, S., & Dijk, D. (2022). Circadian rhythms and disorders of the timing of sleep. The Lancet, 400, 1061-1078.

149. Zisapel, N. (2018). New perspectives on the role of melatonin in human sleep, circadian rhythms and their regulation. British Journal of Pharmacology, 175, 3190 - 3199.

150. Harvey, A. G., & Payne, S. (2002). The management of unwanted pre-sleep thoughts in insomnia: distraction with imagery versus general distraction. Behaviour research and therapy, 40(3), 267-277.

151. Yetton, B., McDevitt, E., Cellini, N., Shelton, C., & Mednick, S. (2018). Quantifying sleep architecture dynamics and individual differences using big data and Bayesian networks. PLoS ONE, 13.

152. Burman, D., & Muzumdar, H. (2020). Sleep Architecture and Physiology. , 12-22.

153. Rama, A., Cho, S., & Kushida, C. (2021). Normal human sleep. Reference Module in Neuroscience and Biobehavioral Psychology.

154. Stevner, A., Stevner, A., Vidaurre, D., Cabral, J., Cabral, J., Rapuano, K., Nielsen, S., Tagliazucchi, E., Tagliazucchi, E., Laufs, H., Vuust, P., Deco, G., Woolrich, M., Someren, E., Someren, E., & Kringelbach, M. (2019). Discovery of key whole-brain transitions and dynamics during human wakefulness and non-REM sleep. Nature Communications, 10.

155. Voss, U. (2004). Functions of Sleep Architecture and the Concept of Protective Fields. Reviews in the Neurosciences, 15, 33 - 46.

156. Kong, G., Li, C., Peng, H., Han, Z., & Qiao, H. (2023). EEG-Based Sleep Stage Classification via Neural Architecture Search. IEEE Transactions on Neural Systems and Rehabilitation Engineering, 31, 1075-1085.

157. Kishi, A., & Van Dongen, H. (2023). Phenotypic Interindividual Differences in the Dynamic Structure of Sleep in Healthy Young Adults. Nature and Science of Sleep, 15, 465 - 476.

158. Cajochen, C., Reichert, C., Münch, M., Gabel, V., Stefani, O., Chellappa, S., & Schmidt, C. (2023). Ultradian sleep cycles: Frequency, duration, and associations with individual and environmental factors-A retrospective study.. Sleep health.

159. Sawai, H., Matsumoto, M., & Koyama, E. (2021). The relationship between each length of REM - NREM sleep cycle and sleep stage. 2021 IEEE 3rd Global Conference on Life Sciences and Technologies (LifeTech), 171-172.

160. Carskadon, M., & Dement, W. (2005). Chapter 2 - Normal Human Sleep : An Overview. , 13-23.

161. Bon, O., Lanquart, J., Hein, M., & Loas, G. (2019). Sleep ultradian cycling: Statistical distribution and links with other sleep variables, depression, insomnia and sleepiness—A retrospective study on 2,312 polysomnograms. Psychiatry Research, 279, 140-147.

162. Hutchison, I., & Rathore, S. (2015). The role of REM sleep theta activity in emotional memory. Frontiers in Psychology, 6.

163. Genzel, L., Spoormaker, V., Konrad, B., & Dresler, M. (2015). The role of rapid eye movement sleep for amygdala-related memory processing. Neurobiology of Learning and Memory, 122, 110-121.

164. Brown, R., Basheer, R., McKenna, J., Strecker, R., & McCarley, R. (2012). Control of sleep and wakefulness.. Physiological reviews, 92 3, 1087-187.

165. Scarpelli, S., Bartolacci, C., D'Atri, A., Gorgoni, M., & De Gennaro, L. (2019). The Functional Role of Dreaming in Emotional Processes. Frontiers in Psychology, 10.

166. Walker, M., & Van Der Helm, E. (2009). Overnight therapy? The role of sleep in emotional brain processing.. Psychological bulletin, 135 5, 731-48.

167. Wassing, R., Lakbila-Kamal, O., Ramautar, J., Stoffers, D., Schalkwijk, F., & Van Someren, E. (2019). Restless REM Sleep Impedes Overnight Amygdala Adaptation. Current Biology, 29, 2351-2358.e4.

168. Rosales-Lagarde, A., Armony, J., Del Río-Portilla, Y., Trejo-Martínez, D., Conde, R., & Corsi-Cabrera, M. (2012). Enhanced emotional reactivity after selective REM sleep deprivation in humans: an fMRI study. Frontiers in Behavioral Neuroscience, 6.

169. Vijayan, S., Lepage, K., Kopell, N., & Cash, S. (2017). Frontal beta-theta network during REM sleep. eLife, 6.

170. Werner, G., Schabus, M., Blechert, J., & Wilhelm, F. (2020). Differential Effects of REM Sleep on Emotional Processing: Initial Evidence for Increased Short-term Emotional Responses and Reduced Long-term Intrusive Memories. Behavioral Sleep Medicine, 19, 83 - 98. .

171. Helm, E., Yao, J., Dutt, S., Rao, V., Saletin, J., & Walker, M. (2011). REM Sleep Depotentiates Amygdala Activity to Previous Emotional Experiences. Current Biology, 21, 2029-2032.

172. Tempesta, D., Socci, V., De Gennaro, L., & Ferrara, M. (2017). Sleep and emotional processing.. Sleep medicine reviews, 40, 183-195.

173. Groch, S., Wilhelm, I., Diekelmann, S., & Born, J. (2013). The role of REM sleep in the processing of emotional memories: Evidence from behavior and event-related potentials. Neurobiology of Learning and Memory, 99, 1-9.

174. Miller, K., & Gehrman, P. (2019). REM Sleep: What Is It Good For?. Current Biology, 29, R806-R807.

175. Nishida, M., Pearsall, J., Buckner, R., & Walker, M. (2008). REM Sleep, Prefrontal Theta, and the Consolidation of Human Emotional Memory. Cerebral Cortex (New York, NY), 19, 1158 - 1166.

176. Werner, G., Schabus, M., Blechert, J., Kolodyazhniy, V., & Wilhelm, F. (2015). Pre- to postsleep change in psychophysiological reactivity to emotional films: Late-night REM sleep is associated with attenuated emotional processing.. Psychophysiology, 52 6, 813-25.

177. Goldstein, A., & Walker, M. (2014). The role of sleep in emotional brain function.. Annual review of clinical psychology, 10, 679-708 ..

178. Osorio-Forero, A., Cardis, R., Vantomme, G., Guillaume-Gentil, A., Katsioudi, G., Fernandez, L., & Lüthi, A. (2021). Noradrenergic circuit control of non-REM sleep substates. Current Biology, 31, 5009-5023.e7.

179. Bellesi, M., Tononi, G., Cirelli, C., & Serra, P. (2016). Region-Specific Dissociation between Cortical Noradrenaline Levels and the Sleep/Wake Cycle.. Sleep, 39 1, 143-54 .

180. Gottesmann, C. (2011). The Involvement of Noradrenaline in Rapid Eye Movement Sleep Mentation. Frontiers in Neurology, 2.

181. Léna, I., Parrot, S., Deschaux, O., Muffat-Joly, S., Sauvinet, V., Renaud, B., Suaud-Chagny, M., & Gottesmann, C. (2005). Variations in extracellular levels of dopamine, noradrenaline, glutamate, and aspartate across the sleep–wake cycle in the medial prefrontal cortex and nucleus accumbens of freely moving rats. Journal of Neuroscience Research, 81.

182. Jones, B. (2019). Arousal and sleep circuits. Neuropsychopharmacology, 45, 6-20.

183. Stanford, S. (2013). Adrenaline and noradrenaline: introduction.

184. Goldstein, D. S. (2010). Adrenaline and noradrenaline. eLS.

185. Mefford, I., Oke, A., Keller, R., Adams, R., & Jonsson, G. (1978). Epinephrine distribution in human brain. Neuroscience Letters, 9, 227-231.

186. Gorain, B., Dutta, S., Nandy, U., Sengupta, P., & Choudhury, H. (2020). Pharmacology of adrenaline, noradrenaline, and their receptors. In Frontiers in Pharmacology of Neurotransmitters (pp. 107-142). Singapore: Springer Singapore.

187. Horowitz, A., Esfahany, K., Gálvez, T., Maes, P., & Stickgold, R. (2023). Targeted dream incubation at sleep onset increases post-sleep creative performance. Scientific Reports, 13.

188. Barrett, D. (2017). Dreams and creative problem-solving. Annals of the New York Academy of Sciences, 1406.

189. Sierra-Siegert, M., Jay, E., Flórez, C., & Garcia, A. (2019). Minding the Dreamer Within: An Experimental Study on the Effects of Enhanced Dream Recall on Creative Thinking. Journal of Creative Behavior, 53, 83-96..

190. Cai, D., Mednick, S., Harrison, E., Kanady, J., & Mednick, S. (2009). REM, not incubation, improves creativity by priming associative networks. Proceedings of the National Academy of Sciences, 106, 10130 - 10134..

191. Deperrois, N., Petrovici, M., Jordan, J., Huber, L., & Senn, W. (2024). How Adversarial REM Dreams May Facilitate Creativity, and Why We Become Aware of Them. Clinical and Translational Neuroscience..

192. Lewis, P., Knoblich, G., & Poe, G. (2018). How Memory Replay in Sleep Boosts Creative Problem-Solving. Trends in Cognitive Sciences, 22, 491-503.

193. Klepel, F., Schredl, M., & Göritz, A. (2019). Dreams stimulate waking-life creativity and problem solving: Effects of personality traits. International Journal of Dream Research, 95-102.

194. Vallat, R., Türker, B., Nicolas, A., & Ruby, P. (2022). High Dream Recall Frequency is Associated with Increased Creativity and Default Mode Network Connectivity. Nature and Science of Sleep, 14, 265 - 275.

195. Schwartz, S., Clerget, A., & Perogamvros, L. (2022). Enhancing imagery rehearsal therapy for nightmares with targeted memory reactivation. Current Biology, 32, 4808-4816.e4.

196. Lancee, J., Effting, M., & Kunze, A. (2020). Telephone-guided imagery rehearsal therapy for nightmares: Efficacy and mediator of change. Journal of Sleep Research, 30.

197. Sayk, C., Koch, N., Stierand, J., Timpe, F., Ngo, H., Wilhelm, I., & Junghanns, K. (2024). Imagery rehearsal therapy for the treatment of nightmares in individuals with borderline personality disorder - A pilot study.. Journal of psychiatric research, 182, 34-41 .

198. Krakow, B., Kellner, R., Pathak, D., & Lambert, L. (1995). Imagery rehearsal treatment for chronic nightmares.. Behaviour research and therapy, 33 7, 837-43 .

199. Van Schagen, A., Lancee, J., De Groot, I., Spoormaker, V., & Van Den Bout, J. (2015). Imagery rehearsal therapy in addition to treatment as usual for patients with diverse psychiatric diagnoses suffering from nightmares: a randomized controlled trial.. The Journal of clinical psychiatry, 76 9, e1105-13

200. Nappi, C., Drummond, S., Thorp, S., & McQuaid, J. (2010). Effectiveness of imagery rehearsal therapy for the treatment of combat-related nightmares in veterans.. Behavior therapy, 41 2, 237-44 .

201. Casement, M., & Swanson, L. (2012). A meta-analysis of imagery rehearsal for post-trauma nightmares: effects on nightmare frequency, sleep quality, and posttraumatic stress.. Clinical psychology review, 32 6, 566-74 .

202. Gill, P., Fraser, E., Tran, T., De Sena Collier, G., Jago, A., Losinno, J., & Ganci, M. (2023). Psychosocial treatments for nightmares in adults and children: a systematic review. BMC Psychiatry, 23.

203. St-Onge, M., Mercier, P., & De Koninck, J. (2009). Imagery Rehearsal Therapy for Frequent Nightmares in Children. Behavioral Sleep Medicine, 7, 81 - 98.

204. Gieselmann, A., Aoudia, M., Carr, M., Germain, A., Gorzka, R., Holzinger, B., Kleim, B., Krakow, B., Kunze, A., Lancee, J., Nadorff, M., Nielsen, T., Riemann, D., Sandahl, H., Schlarb, A., Schmid, C., Schredl, M., Spoormaker, V., Steil, R., Van Schagen, A., Wittmann, L., Zschoche, M., & Pietrowsky, R. (2019). Aetiology and treatment of nightmare disorder: State of the art and future perspectives. Journal of Sleep Research, 28.

205. Swart, M., Van Schagen, A., Lancee, J., & Van Den Bout, J. (2013). Prevalence of Nightmare Disorder in Psychiatric Outpatients. Psychotherapy and Psychosomatics, 82, 267 - 268.

206. Stefani, A., & Högl, B. (2020). Nightmare Disorder and Isolated Sleep Paralysis. Neurotherapeutics, 18, 100 - 106.

207. Spoormaker, V., Schredl, M., & Van Den Bout, J. (2006). Nightmares: from anxiety symptom to sleep disorder.. Sleep medicine reviews, 10 1, 19-31 .

208. Schredl, M., & Goeritz, A. (2019). Nightmare frequency and nightmare distress: Socio-demographic and personality factors. Sleep Science, 12, 178 - 184.

209. Saulnier, K., Singh, R., Lenker, K., Calhoun, S., He, F., Liao, D., Vgontzas, A., Bixler, E., & Fernandez-Mendoza, J. (2022). Association of insomnia phenotypes based on polysomnography-measured sleep duration with suicidal ideation and attempts.. Sleep health, 712 .

210. Dolsen, M., Prather, A., Lamers, F., & Penninx, B. (2020). Suicidal ideation and suicide attempts: associations with sleep duration, insomnia, and inflammation. Psychological Medicine, 51, 2094 - 2103.

211. Littlewood, D., Kyle, S., Carter, L., Peters, S., Pratt, D., & Gooding, P. (2018). Short sleep duration and poor sleep quality predict next-day suicidal ideation: an ecological momentary assessment study. Psychological Medicine, 49, 403 - 411.

212. Pereira, T., Martins, S., & Fernandes, L. (2017). Sleep duration and suicidal behavior: A systematic review. European Psychiatry, 41, s854 - s854.

213. Liu, R., Steele, S., Hamilton, J., B, Q., Furbish, K., Burke, T., Martinez, A., & Gerlus, N. (2020). Sleep and suicide: A systematic review and meta-analysis of longitudinal studies.. Clinical psychology review, 81, 101895.

214. Cartwright, R., Agargun, M. Y., Kirkby, J., & Friedman, J. K. (2006). Relation of dreams to waking concerns. Psychiatry research, 141(3), 261-270.

215. Hausenblas, H., Lynch, T., Hooper, S., Shrestha, A., Rosendale, D., & Gu, J. (2024). Magnesium-L-threonate improves sleep quality and daytime functioning in adults with self-reported sleep problems: A randomized controlled trial. Sleep Medicine: X, 8.

216. Arab, A., Rafie, N., Amani, R., & Shirani, F. (2022). The Role of Magnesium in Sleep Health: a Systematic Review of Available Literature. Biological Trace Element Research, 201, 121-128.

217. Mah, J., & Pitre, T. (2021). Oral magnesium supplementation for insomnia in older adults: a Systematic Review & Meta-Analysis. BMC Complementary Medicine and Therapies, 21.

218. Zhang, Y., Chen, C., Lu, L., Knutson, K., Carnethon, M., Fly, A., Luo, J., Haas, D., Shikany, J., & Kahe, K. (2021). Association of magnesium intake with sleep duration and sleep quality: findings from the CARDIA study.. Sleep.

219. Luo, X., Tang, M., Wei, X., & Peng, Y. (2024). Magnesium depletion score and sleep quality in U.S. adults, NHANES 2005-2014.. Journal of affective disorders.

220. Khalid, S., Bashir, S., Mehboob, R., Anwar, T., Ali, M., Hashim, M., Waseem, H., & Basharat, S. (2024). Effects of magnesium and potassium supplementation on insomnia and sleep hormones in patients with diabetes mellitus. Frontiers in Endocrinology, 15.

221. Nielsen, F., Johnson, L., & Zeng, H. (2010). Magnesium supplementation improves indicators of low magnesium status and inflammatory stress in adults older than 51 years with poor quality sleep.. Magnesium research, 23 4, 158-68 .

222. Saba, S., Faizi, F., Sepandi, M., & Nehrir, B. (2022). Effect of short-term magnesium supplementation on anxiety, depression and sleep quality in patients after open-heart surgery. Magnesium research, 35 2, 62-70 .

223. Carlos, R., Matias, C., Cavaca, M., Cardoso, S., Santos, D., Giro, R., Vaz, J., Pereira, P., Vicente, F., Leonardo-Mendonça, R., Ganhão-Arranhado, S., Santos, H., Reiter, R., & Teixeira, F. (2024). The effects of melatonin and magnesium in a novel supplement delivery system on sleep scores, body composition and metabolism in otherwise healthy individuals with sleep disturbances. Chronobiology International, 41, 817 - 828.

224. Garrison, S., Korownyk, C., Kolber, M., Allan, G., Musini, V., Sekhon, R., & Dugré, N. (2020). Magnesium for skeletal muscle cramps.. The Cochrane database of systematic reviews, 9, CD009402 .

225. Tarsitano, M., Quinzi, F., Folino, K., Greco, F., Oranges, F., Cerulli, C., & Emerenziani, G. (2024). Effects of magnesium supplementation on muscle soreness in different type of physical activities: a systematic review. Journal of Translational Medicine, 22.

226. Garrison, S., Allan, G., Sekhon, R., Musini, V., & Khan, K. (2012). Magnesium for skeletal muscle cramps.. The Cochrane database of systematic reviews, 9, CD009402 .

227. Liguori, S., Moretti, A., Paoletta, M., Gimigliano, F., & Iolascon, G. (2024). Role of Magnesium in Skeletal Muscle Health and Neuromuscular Diseases: A Scoping Review. International Journal of Molecular Sciences, 25.

228. Reno, A., Green, M., Killen, L., O'Neal, E., Pritchett, K., & Hanson, Z. (2020). Effects of Magnesium Supplementation on Muscle Soreness and Performance. Journal of Strength and Conditioning Research, 36, 2198 - 2203.

229. Steward, C., Zhou, Y., Keane, G., Cook, M., Liu, Y., & Cullen, T. (2019). One week of magnesium supplementation lowers IL-6, muscle soreness and increases post-exercise blood glucose in response to downhill running. European Journal of Applied Physiology, 119, 2617 - 2627.

230. Ertürk, Ç., & Özden, A. (2025). Comparison of the Acute Effects of Auricular Vagus Nerve Stimulation and Deep Breathing Exercise on the Autonomic Nervous System Activity and Biomechanical Properties of the Muscle in Healthy People. Journal of Clinical Medicine, 14.

231. El-Sheikh, M., Erath, S., & Bagley, E. (2013). Parasympathetic nervous system activity and children's sleep. Journal of Sleep Research, 22.

232. Werner, G., Ford, B., Mauss, I., Schabus, M., Blechert, J., & Wilhelm, F. (2015). High cardiac vagal control is related to better subjective and objective sleep quality. Biological Psychology, 106, 79 - 85.

233. Tsai, H., Kuo, T., Lee, G., & Yang, C. (2015). Efficacy of paced breathing for insomnia: enhances vagal activity and improves sleep quality.. Psychophysiology, 52 3, 388-96 .

234. Bachour, G., Samir, A., Haddad, S., Houssaini, M., & Radad, M. (2025). Effects of Ashwagandha Supplements on Cortisol, Stress, and Anxiety Levels in Adults: A Systematic Review and Meta-Analysis. BJPsych Open, 11, S39 - S39.

235. Lopresti, A., Smith, S., & Drummond, P. (2021). Modulation of the hypothalamic–pituitary–adrenal (HPA) axis by plants and phytonutrients: a systematic review of human trials. Nutritional Neuroscience, 25, 1704 - 1730.

236. Lopresti, A., Smith, S., Malvi, H., & Kodgule, R. (2019). An investigation into the stress-relieving and pharmacological actions of an ashwagandha (Withania somnifera) extract. Medicine, 98.

237. Remenapp, A., Coyle, K., Orange, T., Lynch, T., Hooper, D., Hooper, S., Conway, K., & Hausenblas, H. (2021). Efficacy of Withania somnifera supplementation on adult's cognition and mood. Journal of Ayurveda and Integrative Medicine, 13.

238. Wiciński, M., Fajkiel-Madajczyk, A., Kurant, Z., Kurant, D., Gryczka, K., Falkowski, M., Wiśniewska, M., Słupski, M., Ohla, J., & Zabrzyński, J. (2023). Can Ashwagandha Benefit the Endocrine System?—A Review. International Journal of Molecular Sciences, 24.

239. Arumugam, V., Vijayakumar, V., Balakrishnan, A., Bhandari, R., Boopalan, D., Ponnurangam, R., Thirupathy, V., & Kuppusamy, M. (2024). Effects of Ashwagandha (Withania Somnifera) on stress and anxiety: A systematic review and meta-analysis.. Explore, 20 6, 103062 .

240. Speers, A., Cabey, K., Soumyanath, A., & Wright, K. (2021). Effects of Withania somnifera (Ashwagandha) on Stress and the Stress-Related Neuropsychiatric Disorders Anxiety, Depression, and Insomnia. Current Neuropharmacology, 19, 1468 - 1495.

241. Gopukumar, K., Thanawala, S., Somepalli, V., Rao, T., Thamatam, V., & Chauhan, S. (2021). Efficacy and Safety of Ashwagandha Root Extract on Cognitive Functions in Healthy, Stressed Adults: A Randomized, Double-Blind, Placebo-Controlled Study. Evidence-based Complementary and Alternative Medicine : eCAM, 2021.

242. Salve, J., Pate, S., Debnath, K., & Langade, D. (2019). Adaptogenic and Anxiolytic Effects of Ashwagandha Root Extract in Healthy Adults: A Double-blind, Randomized, Placebo-controlled Clinical Study. Cureus, 11.

243. Pandit, S., Srivastav, A., Sur, T., Chaudhuri, S., Wang, Y., & Biswas, T. (2024). Effects of Withania somnifera Extract in Chronically Stressed Adults: A Randomized Controlled Trial. Nutrients, 16.

244. Hellhammer, J., Vogt, D., Franz, N., Freitas, U., & Rutenberg, D. (2014). A soy-based phosphatidylserine/ phosphatidic acid complex (PAS) normalizes the stress reactivity of hypothalamus-pituitary-adrenal-axis in chronically stressed male subjects: a randomized, placebo-controlled study. Lipids in Health and Disease, 13, 121 - 121.

245. Hellhammer, J., Fries, E., Buss, C., Engert, V., Tuch, A., Rutenberg, D., & Hellhammer, D. (2004). Effects of Soy Lecithin Phosphatidic Acid and Phosphatidylserine Complex (PAS) on the Endocrine and Psychological Responses to Mental Stress. Stress, 7, 119 - 126.

246. Monteleone, P., Beinat, L., Tanzillo, C., Maj, M., & Kemali, D. (1990). Effects of phosphatidylserine on the neuroendocrine response to physical stress in humans.. Neuroendocrinology, 52 3, 243-8 .

247. Stout, J., Graves, B., Smith, A., Hartman, M., Cramer, J., Beck, T., & Harris, R. (2008). Journal of the International Society of Sports Nutrition Open Access the Effects of Phosphatidylserine on Endocrine Response to Moderate Intensity Exercise.

248. Starks, M., Starks, S., Kingsley, M., Purpura, M., & Jäger, R. (2008). The effects of phosphatidylserine on endocrine response to moderate intensity exercise. Journal of the International Society of Sports Nutrition, 5, 11 - 11.

249. , X., Li, X., Wang, W., Zhang, M., Yang, B., & Miao, Z. (2022). Phosphatidylserine, inflammation, and central nervous system diseases. Frontiers in Aging Neuroscience, 14.

250. Cicero, A., Sahebkar, A., Fogacci, F., Bove, M., Giovannini, M., & Borghi, C. (2019). Effects of phytosomal curcumin on anthropometric parameters, insulin resistance, cortisolemia and non-alcoholic fatty liver disease indices: a double-blind, placebo-controlled clinical trial. European Journal of Nutrition, 59, 477 - 483.

251. Lack, L., Micic, G., & Lovato, N. (2023). Circadian aspects in the aetiology and pathophysiology of insomnia. Journal of Sleep Research, 32.

252. Chung, K. (2005). Insomnia Subtypes and Their Relationships to Daytime Sleepiness in Patients with Obstructive Sleep Apnea. Respiration, 72, 460 - 465.

253. Waters, W., Adams, S., Binks, P., & Varnado, P. (1993). Attention, stress and negative emotion in persistent sleep-onset and sleep-maintenance insomnia.. Sleep, 16 2, 128-36 .

254. Bjorøy, I., Jørgensen, V., Pallesen, S., & Bjorvatn, B. (2020). The Prevalence of Insomnia Subtypes in Relation to Demographic Characteristics, Anxiety, Depression, Alcohol Consumption and Use of Hypnotics. Frontiers in Psychology, 11.

255. Dressle, R., Feige, B., Spiegelhalder, K., Schmucker, C., Benz, F., Mey, N., & Riemann, D. (2022). HPA axis activity in patients with chronic insomnia: A systematic review and meta-analysis of case-control studies.. Sleep medicine reviews, 62, 101588 .

256. Vargas, I., Vgontzas, A., Abelson, J., Faghih, R., Morales, K., & Perlis, M. (2018). Altered ultradian cortisol rhythmicity as a potential neurobiologic substrate for chronic insomnia.. Sleep medicine reviews, 41, 234-243 .

257. Vgontzas, A., Bixler, E., Lin, H., Prolo, P., Mastorakos, G., Vela-bueno, A., Kales, A., & Chrousos, G. (2001). Chronic insomnia is associated with nyctohemeral activation of the hypothalamic-pituitary-adrenal axis: clinical implications.. The Journal of clinical endocrinology and metabolism, 86 8, 3787-94 .

258. Abell, J., Shipley, M., Ferrie, J., Kivimäki, M., & Kumari, M. (2016). Recurrent short sleep, chronic insomnia symptoms and salivary cortisol: A 10-year follow-up in the Whitehall II study. Psychoneuroendocrinology, 68, 91 - 99.

259. Passos, G., Youngstedt, S., Rozales, A., Ferreira, W., De-Assis, D., De-Assis, B., & Santana, M. (2022). Insomnia Severity is Associated with Morning Cortisol and Psychological Health. Sleep Science, 16, 92 - 96.

260. Van Der Zweerde, T., Bisdounis, L., Kyle, S., Lancee, J., & Van Straten, A. (2019). Cognitive behavioral therapy for insomnia: A meta-analysis of long-term effects in controlled studies.. Sleep medicine reviews, 48, 101208.

261. Soh, H., Ho, R., Ho, C., & Tam, W. (2020). Efficacy of digital cognitive behavioural therapy for insomnia: a meta-analysis of randomised controlled trials.. Sleep medicine, 75, 315-325 .

262. Hertenstein, E., Trinca, E., Wunderlin, M., Schneider, C., Züst, M., Fehér, K., Su, T., VanStraten, A., Berger, T., Baglioni, C., Johann, A., Spiegelhalder, K., Riemann, D., Feige, B., & Nissen, C. (2022). Cognitive behavioral therapy for insomnia in patients with mental disorders and comorbid insomnia: A systematic review and meta-analysis.. Sleep medicine reviews, 62, 101597 .

263. Selvanathan, J., Pham, C., Nagappa, M., Peng, P., Englesakis, M., Espie, C., Morin, C., & Chung, F. (2021). Cognitive behavioral therapy for insomnia in patients with chronic pain - A systematic review and meta-analysis of randomized controlled trials.. Sleep medicine reviews, 60, 101460 .

264. Zachariae, R., Lyby, M., Ritterband, L., & O'Toole, M. (2016). Efficacy of internet-delivered cognitive-behavioral therapy for insomnia - A systematic review and meta-analysis of randomized controlled trials.. Sleep medicine reviews, 30, 1-10 .

265. Simon, L., Steinmetz, L., Feige, B., Benz, F., Spiegelhalder, K., & Baumeister, H. (2023). Comparative efficacy of onsite, digital, and other settings for cognitive behavioral therapy for insomnia: a systematic review and network meta-analysis. Scientific Reports, 13.

266. Alimoradi, Z., Jafari, E., Broström, A., Ohayon, M., Lin, C., Griffiths, M., Blom, K., Jernelöv, S., Kaldo, V., & Pakpour, A. (2022). Effects of cognitive behavioral therapy for insomnia (CBT-I) on quality of life: A systematic review and meta-analysis.. Sleep medicine reviews, 64, 101646.

267. Ashbrook, L., Krystal, A., Fu, Y., & Ptáček, L. (2019). Genetics of the human circadian clock and sleep homeostat. Neuropsychopharmacology, 45, 45-54.

268. Shi, G., Yin, C., Fan, Z., Xing, L., Mostovoy, Y., Kwok, P., Ashbrook, L., Krystal, A., Ptáček, L., & Fu, Y. (2020). Mutations in Metabotropic Glutamate Receptor 1 Contribute to Natural Short Sleep Trait. Current Biology, 31, 13-24.e4.

269. Yook, J., Rizwan, M., Shahid, N., Naguit, N., Jakkoju, R., Laeeq, S., Reghefaoui, T., Zahoor, H., & Mohammed, L. (2021). Some Twist of Molecular Circuitry Fast Forwards Overnight Sleep Hours: A Systematic Review of Natural Short Sleepers' Genes. Cureus, 13.

270. Austin-Zimmerman, I., Levey, D., Giannakopoulou, O., Deak, J., Galimberti, M., Adhikari, K., Zhou, H., Denaxas, S., Irizar, H., Kuchenbaecker, K., McQuillin, A., Concato, J., Buysse, D., Gaziano, J., Gottlieb, D., Polimanti, R., Stein, M., Bramon, E., & Gelernter, J. (2023). Genome-wide association studies and cross-population meta-analyses investigating short and long sleep duration. Nature Communications, 14.

271. Ashbrook, L., Krystal, A., Fu, Y., & Ptáček, L. (2021). 388 Familial natural short sleepers have greater resilience than unaffected family members. Sleep, 44.

272. Morrison, S., Jackson, R., Haszard, J., Galland, B., Meredith-Jones, K., Fleming, E., Ward, A., Elder, D., Beebe, D., & Taylor, R. (2023). The effect of modest changes in sleep on dietary intake and eating behavior in children: secondary outcomes of a randomized crossover trial.. The American journal of clinical nutrition, 117 2, 317-325 .

273. Gomes, S., Ramalhete, C., Bicho, M., & Valente, A. (2023). Sleep Patterns, Eating Behavior and the Risk of Noncommunicable Diseases. Nutrients, 15.

274. Papatriantafyllou, E., Efthymiou, D., Zoumbaneas, E., Popescu, C., & Vassilopoulou, E. (2022). Sleep Deprivation: Effects on Weight Loss and Weight Loss Maintenance. Nutrients, 14.

275. Markwald, R., Melanson, E., Smith, M., Higgins, J., Perreault, L., Eckel, R., & Wright, K. (2013). Impact of insufficient sleep on total daily energy expenditure, food intake, and weight gain. Proceedings of the National Academy of Sciences, 110, 5695 - 5700.

276. Chaput, J., McHill, A., Cox, R., Broussard, J., Dutil, C., Da Costa, B., Sampasa-Kanyinga, H., & Wright, K. (2022). The role of insufficient sleep and circadian misalignment in obesity. Nature Reviews. Endocrinology, 19, 82 - 97.

277. Mullington, J., Haack, M., Toth, M., Serrador, J., & Meier-Ewert, H. (2009). Cardiovascular, inflammatory, and metabolic consequences of sleep deprivation.. Progress in cardiovascular diseases, 51 4, 294-302.

278. Schmid, S., Hallschmid, M., & Schultes, B. (2015). The metabolic burden of sleep loss.. The lancet. Diabetes & endocrinology, 3 1, 52-62 .

279. Briançon-Marjollet, A., Weiszenstein, M., Henri, M., Thomas, A., Godin-Ribuot, D., & Polák, J. (2015). The impact of sleep disorders on glucose metabolism: endocrine and molecular mechanisms. Diabetology & Metabolic Syndrome, 7.

280. Reutrakul, S., & Van Cauter, E. (2018). Sleep influences on obesity, insulin resistance, and risk of type 2 diabetes.. Metabolism: clinical and experimental, 84, 56-66 .

281. Greer, S. M., Goldstein, A. N., & Walker, M. P. (2013). The impact of sleep deprivation on food desire in the human brain. Nature communications, 4(1), 2259.

282. Wang, X., Sparks, J. R., Bowyer, K. P., & Youngstedt, S. D. (2018). Influence of sleep restriction on weight loss outcomes associated with caloric restriction. Sleep, 41(5), zsy027.