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ABSTRACTS

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The future of sleep medicine

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New management of common sleep disorders will be discussed, with a focus on sleep-related breathing disorders. Consumer technologies, such as wearables, nearables, and airables will be described, and how they will be used in the future by sleep specialists. The integration of tools for the diagnosis and management of sleep disorders into sleep medicine clinical and laboratory settings will also be covered.

Redox signaling orchestrates subcellular circadian harmony in plant cells

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Through endosymbiosis, ancestral cyanobacteria lacking a nucleus were engulfed by primordial eukaryotes and underwent a series of adaptive modifications, eventually culminating in chloroplast organelles within plant cells. The two-circadian rhythms in plant cells, consisting of the metabolite-dependent chloroplast redox rhythm and the transcription/translation feedback loop (TTFL)-based nuclear oscillators, are harmoniously orchestrated. However, the synchronization mechanism of these two circadian rhythms remains unknown. We elucidate that a chloroplast redox protein (CRP) modulates the synchronization between nuclear circadian clocks and chloroplast redox rhythms. The hyperoxidation/reduction cycle of chloroplast 2-Cys peroxiredoxins (Prxs) is a sensitive marker of chloroplast redox state. CRP facilitates this circadian rhythmic coordination by regulating intracellular ROS and sucrose concentrations, establishing a molecular bridge between these distinct subcellular compartments. CRP plays a critical role in modulating the amplitude of nuclear circadian rhythms, as exemplified by CCA1 and TOC1. CRP-mediated fluctuations in cytosolic metabolites significantly dampen the amplitude of core clock genes without altering their periodicity. The absence of CRP in mutant plants leads to a profound disruption of the chloroplast redox rhythm and subsequent impairment of TTFL-driven circadian cycles. A reciprocal regulatory mechanism exists, whereby the canonical clock component TOC1 modulates the diel redox cycle of chloroplasts by controlling the expression of CRP, as exemplified by redox oscillation of 2-Cys Prxs. Collectively, our findings demonstrate that CRP serves as a pivotal circadian integrator, orchestrating the synchronization of chloroplast redox dynamics with the temporal regulation of nuclear circadian oscillators.

Mental disorders hidden behind sleep disorders

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Sleep disorders often co-occur with mental health conditions. Patients frequently present to clinics with sleep symptoms as their primary complaint. Insomnia is a common manifestation, but careful inquiry into other symptoms can reveal underlying psychiatric disorders. Depression is particularly prevalent in this population. Conversely, sleep disorders can be masked by mental health conditions. Sleep apnea syndrome, for instance, may be concealed by depression. Many individuals with depression experience sleep apnea either before or after the onset of depressive symptoms. Sleep apnea can increase the risk of depression by contributing to daytime attention difficulties and heightened stress levels. In recent years, our clinic has observed a rise in young patients with hypersomnia who also exhibit symptoms of attention-deficit hyperactivity disorder (ADHD). Many of these patients demonstrate positive results on the Multiple Sleep Latency Test (MSLT), while others do not, yet continue to complain of excessive sleepiness. These findings will be presented and discussed in detail during the lecture.

Oscillator networks in the suprachiasmatic nucleus for behavior rhythm regulation

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Mammalian circadian system is composed of the central clock in the suprachiasmatic nucleus (SCN) of the hypothalamus and peripheral clocks throughout the body. Every cell of the mammalian body, except germ cells, has its own cellular clock based on the transcription and translation feedback of clock genes. These cellular clocks form peripheral clocks in each tissue or organ to exhibit local circadian functions. Then, what makes the SCN distinct from other peripheral clocks? The SCN has two characteristics critical for the central clock. First, the SCN is the only clock that receives external photic information and drives light-entrained circadian rhythms to peripheral clocks to coordinate physiological functions and behavior with day–night cycles. Second, the SCN clock has a hierarchical multi-oscillator structure with networks at different levels. Heterogeneous SCN neurons with individually specific circadian periods synchronize to each other to form regional clocks, such as Evening and Morning clocks, that further couple to organize the central clock. Importantly, not only neuronal but also various diffusible factors are involved in these networks, such as neuropeptides, cytokines, growth factors and gasses. Such structure makes the clock with remarkable stability and wide range of adaptability to the day–night and seasonal changes in environments.

Towards the mystery of sleep—how sleep homeostasis is governed in mammals

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Homeostatic sleep response is a universal phenomenon, whereby sleep deprivation (SD) causes an increase in the amount and/or intensity of recovery sleep to maintain sleep homeostasis. Despite identification of dozens of sleep/wake centers in the mouse brain, the homeostat neurons that govern homeostasis of non-rapid eye movement sleep (NREMS) remain unclear. Here, we find that calcineurin—Ca2 + /calmodulin-dependent phosphatase—is required in the noradrenergic (NA) neurons of locus coeruleus (LC)—a well-known wake center—for the homeostatic regulation of NREMS. Chemogenetic activation of the LC, but not ventral tegmental area (VTA), recapitulates the homeostatic sleep response by causing prolonged wakefulness followed by calcineurin-dependent recovery NREMS. Accordingly, LC exhibits calcineurin-dependent plasticity and transcriptional changes including persistent c-Fos

expression after SD. Chemogenetic suppression of LCNA neurons after SD diminishes recovery NREMS, whereas ablation of LCNA neurons abolishes the homeostatic recovery NREMS. In contrast to previous reports, both chemo- and optogenetic activation of LCNA neurons increase the amount and/or intensity of NREMS especially during the dark phase. Therefore, we propose that LCNA neurons serve as a dual sleep/wake center and govern the homeostatic regulation of NREMS in mice. We anticipate that our findings will inspire more sophisticated future investigations to elucidate the molecular and neural pathways that govern NREMS homeostasis in mammals.

Future of Asian forum on chronobiology

Ken-ichi Honma

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Asian Forum on Chronobiology was built 10 years ago in order to facilitate mutual communication among scientists in the neighbouring countries in Asia. Fortunately, the forum survived the COVID19 pandemic and today we get together in New Delhi, India. In Asia, we have a large number of human resources which is the basis of future sciences. In addition, we had great predecessors in our field such as Maroli Chandrashekaran in India and Syuiti Mori in Japan. Although their pioneering achievements are not evaluated fairly enough in the western world, we are proud of them and admire their contributions to our science. Asian Forum on Chronobiology is like the caravanserais on the silk roads, where we exchange our valuable information. Today I like to talk about an unsettled question in our field which should be the target of our future study. Chronobiology is the science to understand the mechanism by which we adapt with the rotating terrestrial environments and the pathology of maladaptation. By doing so, we will prevent and conquer the related health problems. We now know the clock genes play critical roles in the generation of the circadian oscillation, but circadian rhythms in some body functions persist without clock genes. What is lacking in our knowledge is the mechanisms by which a number of cellular circadian oscillations are integrated into the pacemakers and the internal organization of multiple pacemakers. The oscillatory couplings are the key issue to understand the integration but we are totally ignorant about them.

Sleep problems in neurodevelopmental disorders

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Sleep problems are a common symptom of neuropsychiatric disorders. For this reason, sleep problems in traditional psychiatric disorders have been the subject of a long body of research. Neurodevelopmental disorders, on the other hand, are not new concepts in themselves, but the development of their treatment and research has advanced considerably in the past decade. In parallel, it has come to our attention that sleep problems in neurodevelopmental disorders may also be universal and essential. For the past 5 years, we have been working with a grant from a national project in Japan to focus on the problem of hypersomnia in attention-deficit hyperactivity disorder (ADHD), one of the most common neurodevelopmental disorders. In that project, PET of dopamine and noradrenaline, fMRI, and polysomnography were measured before and after methylphenidate administration in one ADHD patient, and we have been working to clarify the pathophysiology of ADHD and the mechanism of drug treatment. I would like to present some of our results and review the literature.

Asian Sleep Research Society: looking back and moving forward

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Till about the early 1990s, the scientists and practitioners of sleep medicine of most Asian countries, even neighboring countries, had not co-operated in sleep research and sleep medicine, as there was no forum for them to meet and interact. Sleep research and sleep medicine in Asia got a significant boost with the formation of the Asian Sleep Research Society (ASRS), primarily through the efforts of Prof. Shojiro Inoue of Japan.

In 1991, Prof. Inoue introduced the idea during a Satellite Symposium on International Co-operation in Sleep Research at Otaru, Japan. Subsequent interaction between the scientists who attended the meeting in Japan led to the formation of the Founding Committee of ASRS on 10th September, 1992 at New Delhi. The Asian Sleep Research Society was founded on June 16, 1994 during the Founding Congress of Asian Sleep Research Society at Tokyo, Japan. After that, the subsequent ASRS Congresses were held every 3 years. Another major venture that was undertaken by ASRS was the holding of the World Federation meetings, one in Delhi during 22–26 September, 2005, and another one in Kyoto, Japan during 16–20 October 2011. These international gatherings had brought together established sleep scientists, physicians, as well as students from around the world.

The prime objective of these ASRS meetings had been to enable people to present their individual research, and to provide an insight into the success stories of renowned sleep investigators. Moving forward, ASRS should enable talented young scientists from different countries to collaborate to produce high-quality work.

Diagnostic delay in narcolepsy in Japan—a sense of urgency to educate health professionals

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Although it has been reported that there is long diagnostic delay in narcolepsy in various countries, the causes of this delay have not been studied enough except for European counties. According to the study using the European Narcolepsy Network database, the mean diagnostic delay was 9.7 ± 11.5 years. Insufficient knowledge of narcolepsy in medical professionals and difficult access to expert sleep centers account for this delay to some extent, but the real-world practice seems to be complicated. Japan is in a unique position in which all the people have public medical insurance coverage that allows them to have unlimited direct access to any medical specialists; therefore, Japanese citizens could consult their symptoms to sleep specialists with no financial burden. In addition, narcolepsy type 1 (NT1) is characterized by the pathognomonic symptom of cataplexy, so we assumed that cataplexy might contribute to the accelerating the diagnostic process. This study aims at characterizing and comparing the status of diagnostic delay in NT1 and NT2 with special emphasis on cataplexy. This retrospective study dealt with consecutive 115 patients with narcolepsy (69 men/46 women, age: 28.5 ± 11.9 years, 52 NT1/63 NT2), whose diagnosis was made from

April 2006 to March 2024 based on the PSG/MSLT criteria of the International Classification of Sleep Disorders, 3rd edition. We performed the chart review to retrieve demographic and clinical information, and the patients' journey to obtain proper diagnosis before coming to our sleep clinic. The overall diagnostic delay was 11.2 ± 9.4 years (range 1–47 years, median 9.0 years). There was no significant difference between the diagnostic delay in patients with NT1 and NT2 (14.9 ± 11.2 vs 8.2 ± 6.2 years). The age of onset also had no significant difference between NT1 and NT2 patients (18.1 ± 10.0 vs 16.6 ± 4.9 years of age). In NT1 patients, cataplexy was the first manifestation in 11 out of 52 (21.2%), and excessive daytime sleepiness (EDS) preceded cataplexy in 39 (75.0%) patients. 24 (46.2%) patients considered cataplexy disturbing and/or of pathological nature, but for the remaining 28 patients, they did not pay attention to cataplexy until EDS became problematic for their daily life. Seven (13.5%) patients had been diagnosed as having epilepsy, and the first medical contact of 4 patients was emergency room visit due to cataplexy, but none of them was diagnosed as having narcolepsy. There was substantial diagnostic delay in patients with narcolepsy, and cataplexy did not seem to be properly evaluated in Japan. We need to increase attention and awareness of narcolepsy as well as sleep physiology among health professionals, and ultimately to promote systematic education about sleep disorders.

Sleep–wake phase and circadian rhythm: reevaluation

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Diurnal animals, like humans, are awake during the day and sleep at night. The sleep–wake phases usually synchronize with the day–night cycle, which regulates the circadian rhythm of many physiological functions including body temperature (BT) and melatonin. When the circadian rhythm loses synchronization with the day–night cycle, the sleep–wake rhythm, along with the BT and melatonin rhythm, also becomes desynchronized. This is typically found as a non-24-h sleep–wake rhythm disorder (non-24) in totally blind people. Which is one of circadian rhythm sleep–wake disorders (CRSWDs). However, it is known that in isolation experiments, some subjects show internal desynchronization, where the sleep–wake rhythm becomes desynchronized from the circadian rhythm of BT and melatonin. In addition, recent findings unexpectedly revealed that many patients with CRSWDs show a normal melatonin rhythm, prompting a reevaluation of the relationship between sleep–wake phases and circadian rhythm. To better understand the pathophysiology and explore treatment options, we utilized a mouse model with continuous administration of methamphetamine. These mice exhibited a longer wake phase and a shorter sleep phase, which gradually became delayed from the day–night (light–dark) cycle. Some mice even began to run freely, despite being under light–dark conditions. Aripiprazole was effective in reversing these phenotypes. This model may provide insights into human CRSWD patients with a normal melatonin rhythm.

New insights into REM sleep

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The neuronal circuitry underlying REM sleep control is undergoing major changes based on recent studies increasing the granularity of brainstem nuclei. Hayashi et al. have recently reported Crhbp as a genetic marker of a group of glutamatergic neurons in the sublaterodorsal tegmentum (SLD) that are sufficient to elicit REM sleep even from wakefulness. Seigneur et al. will report a new circuit projecting from the SLD to spinal cord that is active during non-REM sleep and accurately predicts the onset and duration of subsequent REM sleep episodes. Work by the Peever group has also shown optogenetic stimulation of SLD neurons increases REM sleep. Together, these findings significantly add to our understanding of REM sleep regulation and function.

Can meditation be the model to study sleep consciousness

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Meditation practices are ideal tools to study the neurophysiological aspects of cognition, sleep and consciousness as the practices induce various aspects of neuroplasticity and brain network modulation that helps in achieving a perfect body mind harmony. Meditation practices modulate all physiological functions in such a way that it enhances cognition, sleep organization, regulate hormonal and immune functions and induces positive affect and reduces negative affect which is essential to achieve inner peace and happiness. Most studies have focused mainly to study the impact of meditation practices on attention, awareness, and how it modulates brain functions to enhance cognition, self-awareness and emotion regulation. Not many studies are available on the role of meditation practices on sleep organization, and understanding consciousness from the perspectives of sleeping brain. Our studies demonstrated that meditation practices help to establish a perfect a sleep organization even among older practitioners of meditation with enhanced N3, REM sleep states, reduced WASO, appropriate sympathovagal balance indicative of perfect cardiovascular regulations, enhanced DHEA and melatonin levels. Microsleep architecture details such as sleep spindle parameters; amplitude, relative power, frequency, oscillation and spindle density was significantly reduced among meditation practitioners when compared to non-meditating control subjects though they had comparable sleep efficiency index. Meditation practices thus ensure a proper macrosleep architecture.

Looking beyond powers spectral measures to assess subjective sleep experiences

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Brain is a complex and dynamical non-linear system; therefore, nonlinear approaches are more appropriate for assessing the intrinsic dynamics of EEG and exploring the physiological mechanisms of brain activity during sleep. In the present study, non-linear approaches EEG are explored to assess differences in sleep dynamics between controls and meditators, explore any signatures of meditation are reflected during sleep as well. Healthy controls (n = 21) and vipassana meditators (n = 23) volunteered for two-day whole night

polysomnography recordings and sleep was scored as per ASSM criteria. EEG power spectrum, non-linear and fractal measures were assessed for every 30 s epochs across sleep stages and averaged and was compared between 2 groups with two-way ANOVA and post hoc corrections with $p < 0.05$ significant level. Subjective sleep quality, mood scales were assessed after sleep. Meditators showed comparable sleep states with controls. Beta1 activity was high in all states except N3. Further, meditators demonstrated less DFA, autocorrelation, lepulziv compressibility across NREM sleep and higher entropy values across all measures. Meditators demonstrated less coherent and a-periodic neural activity during sleep when compared to controls. This observation is in contrast to familiar notion that meditation should induce more coherency. How these could be translated into subjective measures of sleep quality will be discussed.

Incorporation of sensory stimuli into dreams: how targeted memory reactivation affects dream content, dream length, and conscious experience

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Dreams have long been a subject of scientific inquiry, with the continuity hypothesis suggesting that waking experiences significantly influence dream content. Our study explores how audio–visual learning impacts dream content through targeted memory reactivation (TMR) during sleep. Using polysomnography, we analyzed dream reports from 29 participants across REM and N2 sleep stages, following auditory cues linked to prior audio–visual learning. Dreams were categorized using the Hall and Van de Castle method, focusing on characters, interactions, activities, settings, and emotions. Over three nights, the first served as adaptation, the second night had multiple awakenings without stimuli, and the third included sensory stimuli during awakenings. Dream recall remained steady, though REM dream lengths decreased on Night 3 when auditory cues were introduced. Beta activity increased in N2 and REM stages, particularly linked to dream recall. Incorporation of learned content was more frequent in N2 and REM, although unintended incorporations of unrelated content were also observed. Emotionally, dreams on Night 3 displayed fewer negative emotions, with a rise in happiness and apprehension. Findings suggest TMR influences dream content and emotional tone, with incorporation of learned events.

The association between social jet lag on cognitive motor function in Japanese workers

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Social jetlag (SJL) refers to the misalignment between an individual's biological rhythm and societal time schedules, leading to decreased performance and various physical and mental health issues. SJL is associated with chronic sleep deprivation and is particularly recognized as a significant issue among young individuals with evening chronotypes, affecting their sleep–wake rhythms. The implications of SJL on athletic performance are crucial for conditioning athletes, yet while cognitive decline due to SJL has been reported, research focusing specifically on athletic function remains limited. Athletes are shown to require longer sleep durations than the non-athletes; however, they often suffer from sleep deprivation due to demanding schedules. For these athletes, excessive sleep during off-periods may serve as a compensatory strategy, though the scientific understanding of SJL's impact within this population is still unclear. This presentation will explore research investigating the fluctuations in exercise tasks related to SJL and seek to delve into its implications for young individuals with active lifestyles. We aim to enhance the discourse surrounding the pros and cons of SJL.

Optimal total sleep duration to maintain health-related quality of life in Japanese young adults

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Almost all Japanese college students faced chronic insufficient sleep. Particularly, among collegiate athletes, it is speculated that they have more serious sleep loss because, in addition to training, they also need to spend additional time in classes, studying, and doing part-time jobs. In addition, considering the number of daily exercises that athletes undertake, it is assumed that they need longer sleep durations than non-athletes to recover from exhaustion. This study investigated the optimal total sleep duration per day required by collegiate athletes to maintain the physical and mental health-related quality of life (HRQOL), compared with non-athlete students. Furthermore, the effects of optimal sleep extension were verified. A questionnaire survey was conducted to assess demographic variables, lifestyle and sleep habits, and HRQOL in 392 collegiate students. Physical component summary (PCS) and mental component summary (MCS) were assessed using the Short-form-8 health survey. Participants with both good PCS and MCS were defined as having a good HRQOL. Receiver operating curve (ROC) analyses were performed for detection of the cutoff point of total sleep duration per day sufficient to maintain a good HRQOL. Subsequently, twenty-three male collegiate soccer player having sleep loss extended their nocturnal sleep duration to optimal sleep duration for 3 weeks. The cutoff point of total sleep duration per day to maintain good HRQOL for collegiate athletes was 7.92 h, which was longer than 6.79 h for non-athlete students. Their PCS score improved after 3-week sleep extension.

Sleep interventions for children and adolescents with autism spectrum disorders

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Sleep disturbances represent one of the most common problems among children and adolescents with autism spectrum disorder (ASD), affecting between 37 and 93% of them (Schwichtenberg et al. 2022). These sleep problems are associated with the severity of social dysfunction, sensory sensitivity, and internalizing and externalizing behavior problems in children with ASD (Whelan et al. 2022). These sleep problems often persist into adulthood (Morgan et al. 2020). Recent systematic reviews suggest the efficacy of melatonin (Nogueira et al. 2023) and behavioral interventions such as stimulus control, extinction, or reinforcement for sleep problems among youth

with ASD (Phillips et al. 2020). The American Academy of Neurology (AAN) developed a comprehensive practice guideline based on its systematic review, recommending the combination of melatonin and cognitive behavioral therapy (CBT) or melatonin alone as probably effective interventions for bedtime resistance, sleep-onset latency, sleep continuity, and total sleep time (Williams Buckley et al. 2020). However, the comparative efficacy of each intervention is unclear. To provide effective interventions, we should compare the number of interventions. We conducted a network meta-analysis (NMA) of sleep interventions for children and adolescents with ASD. We searched and selected randomized controlled trials on pharmacological, psychological, and physical interventions that addressed sleep problems among youth with ASD. The primary outcome was sleep-onset latency, and secondary outcomes included sleep parameters and daytime functioning. We synthesized the studies included in our review with the efficacy ranking. We will present the preliminary reports of our findings on the efficacy of each sleep intervention.

Physical exercise enhances light sensitivity of the circadian pacemaker in humans

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Human physiology and behavior (sleep and wakefulness) show a 24-h circadian rhythm in the real world. The circadian rhythms are generated by the circadian pacemaker in the suprachiasmatic nucleus (SCN) of the brain's hypothalamus. The SCN pacemaker entrains an environmental light–dark cycle and coordinates a temporal order among physiology and behavior by regulating the peripheral clocks in the extra-SCN brain regions and peripheral organs. Although light is a primary zeitgeber for the circadian pacemaker in humans, non-photic time cues such as physical exercise and meal schedule act as the non-photic zeitgeber for the sleep–wake cycle. Previously, we could demonstrate that exercise under bright light elicits a larger phase-advance shift in the circadian rhythm of plasma melatonin after an 8-h advance shift of sleep schedule in subjects under a temporal isolation facility. Recently, we examined whether exercise could enhance light perception in the eye. We measured pupil size under different light conditions, both with and without exercise intervention at different intensities. We found that exercise under bright light diluted pupil size, and exercise under bright light suppressed the melatonin level as compared with the light alone. These results support the idea that exercise could enhance light perception which may induce light-induced phase-shift. In this symposium, we will discuss the recent progress of the human circadian system and the next step to uncover the mechanism regulating the human sleep–wake cycle.

Neuronal outputs from the suprachiasmatic nucleus that regulate torpor in mice

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In mammals, the circadian rhythms of physiology and behavior are regulated by the central circadian clock located in the hypothalamic suprachiasmatic nucleus (SCN). Environmental conditions on Earth fluctuate in 24-h cycles, including changes in temperature and light/dark cycles, which can present significant survival challenges for animals. For example, animals must endure extreme ambient temperatures and limited food availability during winter. To survive such conditions, animals engage in adaptive strategies such as hibernation and torpor, which are specialized mechanisms for energy conservation through metabolic suppression. While the circadian clock is known to regulate hibernation and torpor, the specific neural mechanisms and circuits that mediate these processes remain largely unresolved. To investigate the circadian regulation of torpor, we induced torpor in both wild-type (WT) and Cry1/Cry2-deficient mice and monitored core body temperature (Tb) using temperature loggers implanted in the abdominal cavity. We induced torpor under conditions of constant darkness. Under these conditions, WT mice displayed a reduction in Tb from the mid-subjective night to the early subjective day, reflecting a temporal pattern similar to that seen under LD cycles. In contrast, Cry1/Cry2-deficient mice exhibited reduced Tb throughout the entire 24-h period, indicating that torpor induction in these mice is not restricted to specific circadian phases. These results suggest that Cry1/Cry2-deficient mice have an impaired ability to regulate torpor in a circadian-dependent manner. The underlying neuronal mechanisms by which the circadian clock modulates torpor in mice will be explored in further detail.

The Period gene in Drosophila was identified in three different behavioural circadian rhythm mutants

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The Period gene in Drosophila was identified in three different behavioral circadian rhythm mutants: short period, long period, and arrhythmic. In mammals, three homologs have been identified. The three mouse Period genes (mPer1, mPer2, and mPer3) play distinct roles in regulating circadian clock function. This study compared circadian pacemakers controlling wheel-running activity rhythms in various per-less mice, observed as free-running systems in constant darkness (DD) and phase response curves (PRCs) for 6-h light pulses. Entrainment of the Pers-less (mPer1–/–, mPer2–/–, mPer3–/–) mice was unstable due to individual circadian rhythm instability. In addition, the Pers-less mice that became arrhythmic upon transfer from LD to DD responded to a 6-h light pulse with transient reorganization of short-period circadian activity rhythm. Bilateral electrolytic lesions in the suprachiasmatic nuclei (SCN) eliminated this reorganization. Multiunit neural activity (MUA) recordings in freely moving mice showed elevated MUA during the day in the SCN of the Pers-less mice. Although they failed to display a daytime increase in DD, a 6-h light pulse reorganized the short-period circadian rhythmicity in both locomotor activity and MUA in the SCN. In vitro multi-electrode array (MEA) recordings of dispersed SCN neurons revealed circadian rhythms in spontaneous firing activities in the Pers-less mice. Physiological analysis showed blunted estrus cycles in the behavior of the Pers-less females. However, by simply manipulating the LD cycle, regular rhythmicity was restored. This suggests that circadian entrainment of the endogenous rhythm to environmental cycles is crucial for maintaining regular estrous cycles even in the Pers-less animals.

The central circadian clock of mammals is located in the suprachiasmatic nucleus (SCN) of the hypothalamus

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The central circadian clock of mammals is located in the suprachiasmatic nucleus (SCN) of the hypothalamus. The SCN regulates the circadian rhythms of various behavioral and physiological functions. In rodents, the luteinizing hormone (LH) surge that induces ovulation is time-dependent and occurs in the evening of proestrus. In addition, SCN-lesioned rats and hamsters do not produce the LH surge. The projection pathway from the SCN to the gonadotropin-releasing hormone (GnRH) neurons that drive the LH surge in the hypothalamus has been studied. However, the functional neural circuit that sends the timing information remains unclear. In the present study, we focused on the AVP, a neuropeptide abundant in the SCN, and conducted experiments using Avp-Cre mice. In mice where GABA release was specifically deficient in AVP neurons (Avp-Cre; Vgat flox/flox mice: AVP-Vgat –/– mice), disturbances in the estrous cycle, usually occurring for 4 or 5 days, were observed. Cre-dependent restorations of Vgat expression in the SCN-AVP neurons recover their estrous cyclicity in Avp-Vgat –/– mice. Furthermore, the estrous cycle was disrupted when AVP neurons in the SCN were suppressed using chemogenetic and optogenetic techniques just before the LH surge occurred during proestrus. However, this was not observed on days other than proestrus. Disturbances in the estrous cycle mean that regular ovulation is not occurring, and these results suggest that GABAergic transmission from AVP neurons in the SCN has essential roles in female reproductive functions.

Prevalence of sleep problems in sportsperson is common

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The correct way ahead is to identify sportsperson with sleep problems and correct them beforehand. Also helping the sportsperson sleep better in pre-competition time, during competition time and after the competition is over is of extreme importance. The sportsperson often even when given the opportunity to sleep are unable to sleep due to individual or peer group effects. Awareness about good sleep at individual level, coaches and administration level is vital. The effort taken by our group would be discussed. The relevant studies where sleep strategies were used would be elaborated. Different disciplines are known to have predilection to certain disorders, e.g., wrestlers are often associated with sleep apnea but a comprehensive evaluation by sleep medicine expert can rule out insomnia and others which can co-occur and affect the performance of the individual. Thus, a comprehensive assessment of the sportsperson and an individualised prescription for the elite performer is the only way forward.

Performance anxiety is the common reasons for sleep problems in athletes

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More than 60% of the athletes reported insomnia the night before competition. Even without this, the elite-athlete lifestyle includes frequent travel, variable schedules, and injury or pain, which can predispose individuals to insomnia. Recently, the International Olympic Committee (IOC) has addressed, for the first time, sleep as a major contributor to athletic performance and as a fundamental feature of athlete mental health (Reardon et al. 2019). Poor sleep can also affect the immune system leaving athletes more susceptible to illness. Our bodies do most of their rest and repairing during the night sleep to help us retain information and maintain memories. This is essential for athletes learning new skills and plays. It is also linked to mental health issues such as anxiety, depression, and stress. Sleep deprivation can exacerbate inflammation, leading to increased pain and decreased performance. Athletes in training should sleep about an hour extra. During sleep, release of hormones, i.e., testosterone, GH and IGF-1, and anabolic effect occur, which are known to increase the activity of protein synthesis hence muscle growth, muscle building, bone growth, and promoting the oxidation of fats. It improves glucose metabolism, which leads to increased energy and a better repair. On the other hand, sleep debt results in corticosterone/cortisol increases and catabolic effect, and a degradation pathway increases protein degradation, impairing skeletal muscle integrity, leading to injury. Insufficient sleep also causes reduced insulin sensitivity resulting in poor glucose metabolism, early fatigue, more injuries and poor and incomplete recovery.

Assessment and monitoring of sleep in sports: an opportunity to enhance performance

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Sleep plays a vital role in optimizing performance, recovery, and injury prevention in sportspersons. Considering the importance of sleep in sports performance, accurate assessment and monitoring methods are crucial. Various modalities such as polysomnography (PSG), clinical sleep interviews, actigraphy, smartwatches/wearables, sleep diaries and sleep questionnaires can be used for sleep evaluation in sports persons. Polysomnography remains the gold standard for objective sleep assessment; however, its high cost, need for trained personnel and laboratory setting limit its utility for large-scale or routine use in sportspersons. Clinical sleep interviews, while useful, are limited by the availability of sleep medicine specialists. Actigraphy, widely utilised in studies of elite sports persons, provides an accessible, ambulatory option for monitoring sleep–wake cycles, but it has limitations in accuracy, particularly for sleep stage differentiation. Sleep questionnaires are useful when studying a large population but questionnaires designed for the general population may not adequately address the specific sleep challenges sportspersons face. The Athlete Sleep Screening Questionnaire (ASSQ),

designed specifically for sportspersons, is a more useful screening tool, identifying sleep disturbances, sleep-disordered breathing, circadian misalignment, and chronotype. Wearable devices, such as smartwatches and fitness trackers, allow continuous long-term sleep monitoring and provide metrics such as sleep duration and heart rate variability. These devices provide valuable data on sleep duration and heart rate variability but lack the accuracy of PSG in assessing sleep stages, highlighting the need for further validation.

The role of astrocytes in sleep–wake control: focusing on sleep disturbances in Alzheimer's disease

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Alzheimer's disease (AD) is frequently associated with disruptions in sleep–wake cycles. While 40-Hz sensory stimulation has been shown to reduce amyloid-beta (Aβ) pathology, its role in modulating sleep–wake control remains unclear. In this study, we highlight the critical role of astrocytes in regulating sleep disturbances in AD. We found that repeated 40-Hz auditory steady-state responses (rASSR) not only alleviated Aβ pathology but also improved sleep in 5xFAD mice. Reactive astrocytes produced aberrant levels of GABA, which inhibited nNOS-positive cortical neurons that promote sleep. This dysfunction was reversed by rASSR stimulation. In addition, rASSR enhanced astrocytic Ca2 + influx and volume changes in response to neuronal activity, accompanied by shifts in oxidative stress-related gene expression. Increased astrocytic Ca2 + influx by optogenetic stimulation with MonSTIM1 replicated the beneficial effects of rASSR on sleep and Aβ pathology. Our findings emphasize the pivotal role of astrocytes in sleep–wake regulation, particularly in the context of AD-related sleep disturbances.

Sleep is an essential behaviour across species, regulated by complex molecular and cellular processes

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Sleep is an essential behavior across species, regulated by complex molecular and cellular processes. Among these, salt-inducible kinase 3 (SIK3) has emerged as a key regulator of sleep homeostasis. Our forward genetics study identified the Sleepy splice mutant allele of Sik3 increases total NREMS time and EEG delta power, indicating its role in sleep depth regulation. Similarly, the Sleepy2 mutant allele in Hdac4 demonstrates a hypersomnic phenotype, further emphasizing the critical interaction between SIK3 and histone deacetylase 4 (HDAC4). We found that SIK3 phosphorylates HDAC4, promoting its cytoplasmic localization, while nuclear HDAC4 represses sleep-promoting genes. Phosphor-deficient Hdac4S245A mice, resistant to SIK3 phosphorylation, show reduced NREMS time and delta power. In addition, somatic expression of Hdac4S245A in Sik3Sleepy mutants alleviates their hypersomnic phenotype, confirming the direct role of SIK3-HDAC4 signaling in regulating sleep depth and duration. Neuron-specific manipulations reveal that SIK3-HDAC4 signaling in cortical excitatory neurons regulates sleep depth, while in hypothalamic neurons, it modulates NREMS quantity. SIK3 is also crucial for circadian rhythm regulation. Deficiency of SIK3 in GABAergic and neuromedin S (NMS)-producing neurons in the suprachiasmatic nucleus (SCN) lengthens the circadian period and delays arousal timing without affecting total sleep. These findings establish the SIK3-HDAC4 pathway as essential for synchronizing sleep homeostasis and circadian rhythms, offering new insights into molecular mechanisms governing sleep and circadian regulation, and identifying potential therapeutic targets for sleep-related disorders.

Sleep presents a significant, yet under explored, biomarker for advancing precision medicine

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This symposium explores the integration of AI-driven analysis with polysomnography (PSG) to harness sleep data for predictive diagnostics. Moving beyond traditional metrics like the Apnea–Hypopnea Index (AHI), novel biomarkers such as ventilatory burden (VB) offer superior predictive capabilities for cardiovascular and all-cause mortality. Leveraging machine learning algorithms, automated sleep scoring platform like Neurobit PSG improves diagnostic accuracy and reduces manual scoring time by 99%, facilitating personalised treatment for obstructive sleep apnea (OSA) and other sleep-related disorders. These AI-enabled biomarkers—including VB, hypoxic burden, and arousal burden—provide clinicians with actionable insights, enabling a more personalized approach to managing OSA and optimizing treatment outcomes. This approach reduces inter-rater variability and enables consistent, reproducible data analysis. The symposium will discuss how Neurobit's vector embedding techniques generate a comprehensive representation of sleep physiology, enabling unbiased, data-driven biomarker discovery. By clustering patients based on treatment response, AI models further refine disease prognostication and therapeutic decision-making. Recent studies validate these innovative biomarkers, demonstrating their integration into clinical workflows and highlighting the potential of sleep as a cornerstone for predictive, personalised medicine.

RLS with its comorbidities and mimics: search for comprehensive tools of assessment—focus on 'm-RLS-DQ' and 'RLS Diary'

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Restless legs syndrome is sleep-related neurological disorder that is diagnosed on the clinical information. However, diagnosis of RLS is often confounded by RLS mimics. A few questionnaires are available for the diagnosis of RLS. Existing tools have some limitations, e.g., absence of items to rule out mimics and co-morbid diagnosis of RLS mimics. We will be presenting the development and validation of a new tool-modified RLS Diagnostic Questionnaire that overcomes some of the limitations of the existing tools. In the patient population, it was found to have acceptable psychometric properties.

Why sleep is altered across a wide range of neuropsychiatric disorders (NPD)?

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Sleep disturbances are prevalent in a variety of neuropsychiatric disorders such as depression, bipolar disorder, anxiety, psychosis, and substance use disorders. This proposal explores the complex relationship between sleep and these psychiatric conditions, focusing on shared genetic factors, neurobiological underpinnings, circadian rhythm disruptions, and the influence of psychotropic medications. Emerging evidence suggests that sleep disturbances are not merely symptoms of psychiatric disorders but may exist as co-occurring conditions requiring independent treatment. The proposal will discuss how neurotransmitters like dopamine, involved in both arousal and mental health disorders, and neurotrophic factors like BDNF, critical for neuroplasticity and sleep regulation, serve as common pathways between sleep disorders and psychiatric illnesses. It will also highlight the overlap in neural circuits, particularly the default mode network (DMN), which is implicated in both insomnia and psychiatric conditions such as anxiety and depression. The role of disrupted circadian rhythms, influenced by genetic variations in clock genes, will be explored, demonstrating how these disruptions contribute to mood disorders and schizophrenia. In addition, the impact of psychiatric medications on sleep architecture will be analyzed, emphasizing the need for tailored treatment strategies that address both sleep and psychiatric symptoms simultaneously. This submission aims to provide a deeper understanding of the bidirectional relationship between sleep disturbances and neuropsychiatric disorders, with the ultimate goal of informing better clinical practices and improving patient outcomes.

Sleep apnea syndrome and psychiatric disorders

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Sleep-disordered breathing (SDB) and psychiatric disorders do not seem to be closely related, but it is known that the prevalence of SDB is significantly higher in people with psychiatric disorders than in people without such disorders, and conversely, the prevalence of psychiatric disorders is significantly higher in people with SDB than in people without such disorders. SDB and psychiatric disorders have an influence on each other. We would like to discuss the diagnosis and treatment of SDB in psychiatric disorders. One of the difficulties in diagnosing SDB in psychiatric disorders is that the subjective symptoms are surprisingly similar. The most common subjective symptoms of SDB is daytime sleepiness. However, when some patients with depression do not perceive their symptoms as "sleepiness," they often describe their symptoms as "foggy and unable to think," or "lack of motivation. These are symptoms of depression. Some drugs for psychiatric pharmacotherapy may increase the risk of SDB with the medications used to treat them. Some of antipsychotic, antidepressant, and mood-stabilizing medications are prone to metabolic abnormalities and weight gain side effects. In addition, benzodiazepines have muscle relaxant effects, which tend to relax the muscles of the tongue and pharynx during sleep, causing airway narrowing. In addition, many psychotropic drugs are known to have some effect on sleep architecture. This may make PSG analysis difficult.

Recent advances in symptomatic narcolepsy

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We have been measuring orexin levels in CSF and have used this to diagnose narcolepsy. In addition to essential narcolepsy, there are rare but symptomatic forms of narcolepsy. Among these, neuromyelitis optica is the most common. We have seen over 50 cases to date, and this is one of the diagnostic criteria for neuromyelitis optica. (2) Treatment of morning arousal disorders in adolescents: circadian rhythm sleep–wake disorders (CRSWDs), especially delayed sleep–wake phase disorder (DSWPD), are common among young people and are a major problem in sleep medicine. They are usually treated with melatonin, bright light therapy, and lifestyle guidance, but nighttime sleep time is often extended, making them difficult to treat. We have reported that administration of 0.5–3 mg aripiprazole to 12 patients with CRSWDs resulted in sleep onset time 1 h earlier, sleep time 1.3 h shorter, and wake time 2 h earlier. A meta-analysis of 45 cases confirmed that aripiprazole is highly effective as a treatment option for CRSWDs. However, the mechanism of action is unknown, which is a drawback in clinical application. Mice were given a 6-h early jet lag and then given aripiprazole, which rapidly resynchronized the shifted light–dark cycle. Ex vivo experiments with suprachiasmatic nucleus (SCN) slice cultures have revealed that aripiprazole causes desynchronization of SCN neurons via the serotonin 1A receptor. Melatonin as a therapeutic drug promotes sleep onset, but aripiprazole promotes early awakening, so it is expected that its effectiveness will be enhanced using it in combination.

Addiction related to digital device use and its impact on sleep and mental health among Japanese students

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Behavioral addiction related to digital device use can lead to mood and behavioral problems such as fatigue, academic decline, and depressive symptoms partly due to sleep deprivation and delayed sleep phase. Given the high prevalence of smartphone use among adolescents, this study aimed to examine the characteristics of these issues by comparing patients and healthy individuals using smartphone screentime records, and by conducting a survey on the relationship between internet usage and sleep. Participants were recruited from the general public and our outpatient clinic. The patient group included those diagnosed with impaired control over electronic device use and social dysfunction based on a physician's web interview. The healthy group consisted of individuals screened to have no severe issues with device use. Sleep and screentime were evaluated using questionnaires and smartphone screentime records. This study was approved by the ethics committee of Tokyo Medical and Dental University. Compared to healthy individuals, the patient group showed not only a longer total smartphone screentime but also a significant increase in smartphone screentime during early morning hours (3:00–5:00) and after school time (15:00–20:00). These screentime record suggest delayed or shortened sleep time. Therefore, measuring the time-of-day usage patterns of smartphone screentime could potentially be applied to predict and detect risks in individuals whose daily lives are impaired by excessive use.

Orexin and epilepsy

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Orexin, a neuropeptide primarily involved in regulating wakefulness and sleep, has garnered increasing attention for its potential role in epilepsy. Initially discovered as a key modulator of arousal, orexin antagonists have since been developed as therapeutic agents for insomnia. Recent research suggests that orexinergic dysfunction may contribute to seizure susceptibility, as orexin neurons—located in the hypothalamus—project to regions involved in seizure initiation and propagation, including the cortex, hippocampus, and amygdala. Preclinical studies using animal models have demonstrated that orexin antagonism significantly influences seizure frequency. However, alterations in orexin tone alone may not fully explain the mechanisms underlying seizure reduction; other factors, such as the rhythm of orexin secretion, may also play significant roles. Furthermore, clinical trials investigating the effects of orexin antagonism on epilepsy in humans remain limited. There is a critical need for further research to elucidate how alterations in orexin pathways, particularly in individuals with epilepsy and concurrent sleep disturbances, impact disease progression and treatment outcomes. This presentation will explore recent advances in understanding the orexin–epilepsy relationship, focusing on the therapeutic potential of targeting the orexinergic system. A comprehensive review of preclinical and clinical studies will be provided, highlighting the effects of orexin receptor agonists and antagonists on seizure frequency and severity. In addition, we will discuss orexin's role at the sleep-epilepsy interface, underscoring its regulatory potential. Emerging evidence on orexin's role in epilepsy offers a promising frontier for innovative treatment strategies that address both seizure control and sleep regulation. Future investigations are pivotal to uncovering the precise mechanisms.

How sleep effects epilepsy and vice versa

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There exist a complex and reciprocal relationship exists between sleep and epilepsy possibly due to common neuro-physiological mechanisms. Epilepsy itself can disrupt sleep architecture, namely an increase in sleep latency, and in the number and duration of awakenings, with a decrease in sleep efficiency and a reduction or fragmentation of REM sleep. Sedation is one of the most common adverse effects of the AEDs, but it may also disrupt sleep, but their effects are variable. Certain epilepsy syndromes are known to occur predominantly/exclusively during sleep. Sleep is a powerful physiological modulator of epileptic activity. Seizure semiology and frequency can also be influenced by sleep and sleep deprivation. Patients with epilepsy (PWE) have several disturbances in sleep architecture that occur despite the absence of daytime-seizures and antiepileptic drugs (AEDs), and this is suggestive of inherent sleep instability in the epileptic brain. Epileptic phenomena often occur during sleep. Studies in focal epilepsies have indicated an increased spiking rate during NREM sleep. The sleep stages represent forms of varied neuronal synchronization that influence epileptic activity. Sleep disorders may influence seizure control, and effective treatment of sleep disorders can improve seizure control. Arousal instability including cycling alternating pattern (CAP) during sleep not only contributes to sleep disorganization but also acts as a potential trigger to activate epileptiform activity. Consequently, occurrence of epileptiform activity during sleep can further hinder sleep continuity and worsen arousal instability, causing a vicious cycle of poor sleep quality and intractable seizures.

Memory encoding and sleep in older adults: a neurocognitive perspective

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Aging populations are vulnerable to sleep architecture disruptions, which impact cognitive function in memory encoding and executive processes. Sleep quality is linked to synaptic plasticity and neurogenesis. Slow-wave sleep (SWS) plays a role in consolidating hippocampal-dependent episodic memories and clearing neurotoxic waste through the glymphatic system. Disruptions in sleep spindles and delta power during non-REM sleep stages impair long-term potentiation (LTP) and memory encoding. In aging individuals, reductions in SWS and fragmented sleep impair synaptic plasticity, leading to deficits in memory encoding and recall tasks. Poor sleep quality may accelerate neurodegeneration by reducing glymphatic clearance of amyloid-beta and tau proteins, contributing to Alzheimer's disease. Decreased cholinergic activity and increased cortical hyperexcitability in aged brains, combined with sleep disturbances, affect prefrontal cortex-mediated executive functions and attentional control. Neuroimaging studies show poor sleep quality is associated with reduced functional connectivity in memory-related networks, such as the default mode network (DMN), and atrophy in the hippocampus and prefrontal regions. These changes in sleep-related neurophysiological processes are believed to accelerate cognitive decline in older adults. Improving sleep quality is needed to mitigate cognitive deterioration and preserve neural integrity. Studies show that good sleepers outperform poor sleepers in attention and executive function, as measured by the Trail Making Test Part B (TMT-B). Good sleepers also show better performance in immediate recall, linking sleep quality to memory encoding. These findings highlight the importance of interventions to protect against age-related cognitive decline.

Insights into sleep cognition from EEG connectivity dynamics during sleep states

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Emerging evidence suggest that sleeping period is cognitively rich and yet distinct from waking cognition. This should reflect in distinct brain synchrony patterns during sleep stages, which can be evident in sleep EEG connectivity dynamics. Unfortunately, few studies have been tapping into this potential. During this talk, I will discuss some of the recent studies in this area and showcase few novel findings from our lab in this regard. This will cover the gradual change in EEG connectivity patterns and their dynamics across light to deep sleep stages, across the illness to wellness spectrum (schizophrenia, normal subjects and meditators). The talk will provide insights into how sleeping brain offers a unique opportunity in understanding an

individual's brain health, through the study of EEG connectivity from the several hours of rich brain activity. The talk will be important to sleep researchers and clinicians to appreciate the potential of using sleep EEG connectivity patterns and their dynamics to investigate sleep architecture, complementing the conventional hypnogram-based approaches. It may also open ideas for future non-pharmacological therapeutic/performance-enhancement interventions during sleep using real-time EEG connectivity monitoring.

Restless legs syndrome: mimics and comorbidities

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There are certain conditions which have some of their features akin to symptoms of restless legs syndrome (RLS). These conditions, which might have symptoms roughly similar to RLS, can confuse the clinicians, leading to diagnostic inaccuracies, are known as RLS mimics. The fifth criterion of essential criteria was added to the diagnostic criteria for RLS, specifically to rule out these mimics of RLS. There are another set of disorders, which have been suggested to be associated with RLS, often labeled as symptomatic or secondary cases. RLS in these patients may develop temporally after the associated or co-morbid disorders, thus sometimes implying a causal relationship. We aimed to find out the prevalence of such RLS mimics and co-morbidities through OPD services. RLS mimics were found to be present among $\sim$ 35% patients, whereas chronic insomnia, obesity, OSA and HTN were the major co-morbidities present. Arthritis, as a mimic, was found to be significantly more in older and female patients, whereas exertional myalgia was predominantly found in younger patients. Among the co-morbidities, HTN and obesity was significantly more in females, whereas DM, HTN and OSA were predominantly more in older population.

A tool for the longitudinal assessment of RLS: development and validation of RLS diary

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Due to the daily fluctuations in restless legs syndrome (RLS) symptoms, it is advisable to track daily progress instead of relying on retrospective weekly or monthly assessments. In this study, we developed, verified, and evaluated a restless legs syndrome diary for patients with RLS. The RLS diary was designed to evaluate symptoms about timing, severity, topography, drug efficacy, and daytime health effects. The diary attained a score of 1 for content validation by universal agreement. The diary had a Cronbach's alpha of 0.92, and the composite score demonstrated a weak to moderate positive correlation with the IRLS score at week 4. We determined the RLS diary to be a valid and psychometrically superior instrument for a comprehensive evaluation of the intensity and impact of RLS symptoms.

Restless legs syndrome and coronary artery disease

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The prevalence of RLS in coronary artery disease (CAD) is 8–65%, depending upon the ethnicity, and type and severity of CAD. RLS disrupts night-time sleep, daytime dysfunction, and is associated with adverse cardiovascular consequences even after revascularization, with a significant impact on sleep and life quality. RLS in the CAD population has a distinct clinical profile, it is late onset and possibly secondary to cardiovascular pathology. In the last decade, many authors focused on the potential consequences of RLS on cardio-cerebrovascular diseases, by investigating the autonomic nervous system (ANS), mainly during sleep. The recently observed changes in sympathovagal balance during sleep in RLS patients using heart rate variability (HRV) spectral analysis and complaints of autonomic system disorders in patients suffering from RLS suggest that RLS increases the risk of cardio-vascular diseases. In addition, it is a relatively underrecognized and underestimated condition because of its unclear pathophysiology, low morbidity, and common misdiagnosis. Importantly, RLS is a treatable sleep disorder that causes disturbed sleep, and treating RLS is associated with reduced risk of myocardial infarction and angina. Hence, identification and optimal treatment of RLS in this population through pharmacological (alpha2 delta legends and dopamine agonists) and non-pharmacological (yoga, exercise, and massage) interventions could improve their quality of life and reduce serious clinical consequences.

Stroke-related restless legs syndrome—phenotypes and course

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The third part of the symposium focuses on the emerging entity of stroke-related restless legs syndrome (sRLS), characterized by new-onset or worsening RLS symptoms following a stroke. sRLS symptoms typically appear shortly after stroke onset but can occasionally manifest beforehand. The stroke's topography significantly influences sRLS development, with higher prevalence in supratentorial and brainstem infarctions. Especially subcortical lesions may lead to RLS. While rare cases of RLS regression following stroke have been reported, the neuroanatomical pathways remain unclear, suggesting involvement of multiple brain structures. Small lesion-induced sRLS could serve as a valuable model for studying RLS mechanisms. This presentation will explore the clinical presentation, course, response to treatment, and underlying pathophysiology of sRLS, offering insights into this underrecognized yet impactful condition.

Excessive sleepiness in acute stroke patients: causes and outcome

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Acute stroke patients often suffer from excessive daytime sleepiness (EDS). The estimated prevalence rate of EDS in stroke survivors ranges between 18 and 72%. EDS in acute stroke patients may interfere with their rehabilitation program, affects daytime functional performance, lower overall wellbeing, and impacts cognitive functioning. Most studies used the Epworth Sleepiness Scale (ESS) as a measure for sleepiness in acute stroke. The problem with using ESS is that it is a subjective measure and may not be suitable to use in bedbound acute stroke patients. Many factors may contribute to EDS in acute stroke, such as homeostatic and circadian factors, stroke type and severity, anatomical site of stroke, psychological factors, medication, also sleep disorders particularly sleep apnea. Acute stroke patients with EDS are usually associated with worse outcome compared to those without EDS. These patients have worse neurocognitive, functional and health-related outcomes. Mitigation of EDS in acute stroke may improve patients' survival and outcome. That is why it is very important recognize EDS in acute stroke.

The impact of excessive sleepiness in medical residents

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Excessive daytime sleepiness (EDS) is a common problem that occurs in health workers, especially residents. EDS can have a significant impact on attention, concentration, and psychomotor skills. Residents generally work quite long hours with irregular shifts and have high demands on clinical responsibilities, where all these factors can reduce the quality of sleep. Several studies show a high prevalence of daytime sleepiness in medical residents and health workers that is correlated with decreased academic performance and clinical services to patients. The Psychomotor Vigilance Test (PVT) is a test developed to objectively evaluate a person's ability to maintain attention and respond in a timely manner to salient signals or cues. This study aims to determine the relationship between excessive daytime sleepiness and attention as measured by the PVT. Epworth sleepiness scale scores and PVT were assessed among medical residents in Airlangga University Hospital and Dr. Sutomo Hospital Surabaya.

Navigating the health consequences of shift work and strategies to mitigate the effects

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Introduction: shift work, especially night shifts, disrupts the circadian rhythm and is linked to negative health effects. This study evaluates the impact of shift work on sleep quality and cognitive performance among security guards at AIIMS Gorakhpur on a shift-duty roster. This cross-sectional study assessed 22 male security guards. Sleep quality was measured using the Pittsburgh Sleep Quality Index (PSQI), while cognitive performance was evaluated using the Stroop Color and Word Test (SCWT) and the Psychomotor Vigilance Test (PVT) on Day 1 and Day 10 of each shift block. Overall, 68% of the participants reported poor sleep quality (PSQI > 5), highlighting significant sleep disturbances among night shift workers. No significant correlation was found between sleep quality and cognitive performance, indicating potential adaptive mechanisms. On Day 1, evening shifts showed better cognitive performance on the SCWT congruent task than morning shifts ($p = 0.038$), while reaction times were slower during the morning shift ($p = 0.029$), indicating decreased cognitive efficiency. By Day 10, no significant differences were observed in SCWT correct responses across shifts, suggesting adaptation; however, reaction times were consistently slower in the morning ($p = 0.015$ and $p < 0.001$). PVT results on Day 1 indicated slower reaction times in the morning shift compared to night shifts ($p = 0.011$), highlighting reduced cognitive alertness in early morning shifts. Shift work is associated with poor sleep quality, while cognitive performance shows a mix of adaptation and persistent deficits. The findings highlight the need for interventions to improve sleep health and optimize cognitive function among shift workers.

Shift work hazard

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To maintain pace of providing access to goods and services 24 × 7, many organizations have altered normal working schedule of 9 AM–5 PM to regular evening or night shift, other shifts, viz., rotation, split, on-call or casual, 24 h, and other non-day schedule. 20–25% of the global workforce is engaged in shift work pattern, which disrupts the biological circadian rhythm and natural sleep–wake cycle leading to short sleep times and fatigue, adversely affecting different domains of health and lifestyles. Apart from affecting physical, mental and social health, shift schedules are associated with decline in occupational performance and increased risk of workplace or road traffic accidents, leading to popular terminology "Circadian Rhythm Sleep Disorder"/ "Shift worker disorder"/"shift worker sleep disorder", posing both individual and social risk. Evidence suggests single night without sleep requires approximately 1–2 nights of sleep to restore baseline level of performance. Long-term shift works have been documented resulting in prostate cancer and breast cancer. Physical health issue includes decrease glucose tolerance, weight gain, metabolic syndrome, MI and type 2 diabetes mellitus, while mental ailments include social-family conflict out of non-traditional work timing, cognitive decline, burnout, depression, anxiety, insomnia, decreased alertness, day napping and suicidal tendencies. Education, strategies and its implementation are required to address shift work hazards at or outside workplace.

Sleep micro-architecture in children with obstructive sleep apnea syndrome

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Sleep is essential in child's growth and development. Polysomnography (PSG) is a method to evaluate micro-architecture of sleep. The microstructure of sleep and its electrophysiological features have

been investigated in only a few studies. Sleep spindles protect the sleeping brain from external sensory stimuli and can serve as markers of sleep integrity. The present review is a summary of our studies about spindles and alpha waves in OSAS children. In the first cross-sectional study, we compared microstructural features of sleep in patients with different severities of OSAS and investigated the relationship between sleep microstructural fragmentation and cognitive impairment, as well as daytime sleepiness. It showed thalamocortical dysfunction during sleep in OSAS patients and as the severity of apnea increases, the speed of the spindles decreases. In the second study, we designed to evaluate brain rhythm waves pattern 15 s before waking up in 42 children. It has revealed that awakening happened with a specific trigger of three 10 HZ frequency alpha waves in the occipital lobe in the N2 phase. In third study, we compared sleep spindles activity and its relationship with daytime functioning in 265 children with neuromuscular disorder (NMD) and children with OSAS. NMD children had less number and lower amplitude of spindles in stages N2 and N3 and higher Epworth Sleepiness Scale. Reduction in sleep spindles increases sleep disruption and perhaps has the negative effects on quality of life.

Sleep parameters in pediatric sleep disorders

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The purpose of this lecture is to review macro- and microstructural parameters of sleep in some sleep disorders such as OSA, RLS, and sleep parameters in children with ASD, epilepsy, ADHD and some others by objective and subjective tools. Macrostructure parameters consist of: time in bed (TIB), sleep period time (SPT), total sleep time (TST), sleep latency (SL), sleep efficiency (SE), wakefulness after sleep onset (WASO%), percentage of rapid eye movement sleep (REM%), rapid eye movement sleep latency (RL), percentage of non-rapid eye movement stage 1 (N1%), stage 2 (N2%) and slow wave sleep (SWS%) and microstructural parameters include: cyclic alternating pattern (CAP), spectral analysis, and EEG band power and the detection of sleep electrophysiological features, such as sleep spindles, k-complex, and Mu rhythm. These parameters are different in various sleep disorders and we are going to discuss about them.

Patterns of sleep disorders with regards to site of stroke and hemispheric involvement

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Sleep disturbances are common after stroke and can significantly impact recovery and quality of life. Sleep architecture is frequently disrupted in stroke patients with reduced sleep efficiency, fragmented sleep, and altered sleep stages. Hermann et al. (2008) found that stroke patients tend to spend more time in lighter sleep stages (NREM 1 and 2) and experience reduced deep sleep and REM. The site of the stroke is crucial in determining the type of sleep disruption. Thalamic and brainstem strokes are associated with reduced sleep efficiency and central sleep apnea, while frontal lobe strokes can lead to insomnia and fragmented sleep (Bassetti and Aldrich 2001, Klingelhofer et al. 1992). The hemisphere affected by the stroke also plays a role in the type of sleep disturbances experienced. Right-sided strokes are linked to sleep-disordered breathing, such as obstructive sleep apnea, while left-sided strokes are more associated with insomnia (Vock et al. 2002). Our study on the relationship between hemispheric involvement and sleep disturbances in patients with middle cerebral artery (MCA) territory strokes found that right MCA stroke patients exhibited lower sleep efficiency compared to left MCA patients. However, there were no significant differences in other measures such as sleep-onset latency and deep sleep latency between right and left hemispheric strokes. The relationship between site of location of lesions in stroke patients, and sleep architecture is complex and multifaceted.

Technical nuances of sleep evaluation in patients with stroke

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Sleep problems are common among individuals who have had a stroke, affecting 40% to 70% of the patients. Issues such as sleep-disordered breathing (SDB), insomnia, and hypersomnia are linked to poorer recovery, cognitive decline, increased risk of another stroke, and reduced quality of life. Obstructive sleep apnea (OSA) has been found to be connected to post-stroke fatigue and worsened functional outcomes. However, evaluating sleep disorders in stroke patients involves challenges. Diagnostic tools like polysomnography (PSG) require patient cooperation, which can be difficult for those with hemiparesis or cognitive and motor impairments. Wearable devices and AI-driven models are less invasive and more personalized monitoring options, but they require more validation. Treating sleep disorders after a stroke is equally complicated. While continuous positive airway pressure (CPAP) therapy is effective for sleep apnea, it is often poorly tolerated by many stroke survivors. Modified cognitive-behavioral therapy for insomnia (CBT-I) has shown significant improvement, but there are limited resources and there is a need for specialized personnel. In our research, we encountered difficulties with polysomnography, as there was a poor correlation between subjective measures such as the Pittsburgh Sleep Quality Index (PSQI) and objective PSG-derived data. Screening tools such as the Berlin Questionnaire and STOP-BANG showed modest sensitivity and specificity, indicating the need for improved assessment techniques.

Spectrum of sleep disorders in patients with stroke

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Sleep disorders are increasingly recognized as both a risk factor for and a consequence of stroke. Obstructive sleep apnea (OSA) is the most studied sleep disorder in stroke patients, with research showing a bidirectional relationship between stroke and sleep-disordered breathing. OSA is prevalent in up to 72% of stroke patients, increasing the risk of ischemic stroke by 2- to threefold and worsening post-stroke outcomes. Central sleep apnea (CSA), prevalent in brainstem infarctions, also leads to poor recovery due to fragmented sleep and daytime fatigue. Insomnia affects 20–40% of the stroke

survivors and is linked to depression, anxiety, and impaired cognitive function, complicating rehabilitation. Restless legs syndrome (RLS) and periodic limb movement disorder (PLMD) are reported by 8–12% of the stroke patients, causing sleep fragmentation and excessive daytime sleepiness. Circadian rhythm disturbances are common in patients with hypothalamic or brainstem lesions, disrupting sleep–wake cycles and rehabilitation schedules. Hypersomnia, associated with thalamic and pontine strokes, leads to excessive sleepiness and worse functional outcomes. In our research, we observed that 81.9% of the stroke patients had sleep disordered breathing with an Apnea–Hypopnea Index (AHI) of 5 or more, with 38% experiencing severe apnea (AHI > 30). The median Arousal Index (ArI) was 5.5 (IQR: 0.5–49.5), increasing with apnea severity. Sleep disorders in stroke patients are critical to address for optimal recovery, requiring early detection and a multidisciplinary approach to treatment. Managing these conditions can enhance rehabilitation and improve quality of life.

Preclinical evidences on maternal sleep loss and poor cognitive outcomes in offspring

Kamalesh K Gulia

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Sleep is a dynamic process that undergoes changes from conception to death. In recent years, there has been growing interest to study the ontogenetic development to find correlation between sleep during pregnancy and the fetal outcomes. Novel evidences suggest that sleep during pregnancy plays an important role in shaping optimal neurocognitive development of offspring in human and other altricial species. Restriction of REM sleep during late pregnancy in a rodent model resulted in postnatal age-matched immature sleep networks in brain, reduction in crying (ultrasonic vocalizations obtained in isolation paradigm) patterns in neonates and depression like traits during adolescence. However, total sleep restriction during pregnancy also resulted in immature sleep networks but with increase in ultrasonic vocalizations in neonates, and hyperactivity and increased risk taking behavior in offspring. From the perspective of prenatal origin of adult diseases such as depression and anxiety, it is utmost crucial to understand potential link and optimal tuning of state-dependent changes, i.e., sleep–wakefulness (S–W) networks with autonomic nervous system (ANS) for attaining emotional regulation of behavior, since both these networks (S–W, ANS) are underdeveloped at birth making them vulnerable to any activation or perturbation during perinatal window, thereby posing increasing risk of dysregulated emotions and cognitive decline in growing babies. In modern sleepless society, the role of prenatal sleep in shaping the health outcomes in offspring are discussed through a complex interaction of heart–brain in state-dependent changes in heart rate variability during early development.

Highlights of artificial intelligence in sleep medicine: an appraisal of current literature

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Rapid evaluation in the field of artificial intelligence is expected to improve sleep medicine practices in terms of diagnosis, underpinnings of sleep pathophysiology, treatment targets, and prognosis of sleep disorders. It is foreseeable that many knowledge gaps are likely to become narrower with the application of AI technology. In this seminar, participants review status of AI technology in sleep medicine, organizational positions and anticipated management recommendation in near future. Speakers focus on many poorly understood areas of sleep medicine with the hope of revealing specific treatment targets with the help of AI technology. Women usually have better quality of sleep compared to men until perimenopausal period. Significant changes in the hormonal level occur at puberty, during menstruation, pregnancy and perimenopausal time. Sleep patterns correspond to the different hormonal milieu. The significance of the sex hormones in sleep regulation is the new area of interest for sleep scientists. While different sleep patterns have been traditionally evaluated by history and physical examination occasional laboratory testing, polysomnography, actigraphy, multiple sleep latency test, and maintenance of wakefulness test, the rapid identification of different patterns corresponding to different stages of menstrual cycle, pre- and post-menopausal state using a larger database can facilitate better phenotypical definition of the sleep disorders and provide insight into their pathophysiology. Such AI-assisted programming and protocols can facilitate identifying personalized management protocols based on deeper understanding of multivariate pathophysiologic mechanisms, which are likely to be more effective. The authors in this symposium attempt to present pilot data on the subject.

Revisit AI to understand intricacies of sleep analysis and diagnosis: emerging trends

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The historical discovery of rapid eye movement (REM) sleep in 1953, which brought a revolution in the history of the modern sleep medicine, parallel to emergence of artificial intelligence (AI) also dates back to 1956 when the term AI was coined by John McCarthy during the famous Dartmouth workshop. Interestingly, with the digitization of traditional overnight polysomnography datasets in late nineties, the potential application of machine learning tools (subset of AI) for analysis of sleep data were carried out but mandatory manual rechecking of entire data is yet required to eliminate various errors of the programed analysis. Digitized polysomnographic data are crucial as they contain complex information about several vital parameters of body recorded simultaneously for 6–8 h of sleep, which can be used to train various deep learning algorithms to extract the key features of various sleep disorders. With increasing market size of consumer sleep technologies, AI algorithms have huge potential using direct (EEG based) or indirect (heart rate variability measures, movement, respiration rate, oxygen saturation, etc.) parameters for sleep detection. Though these consumer technologies are far from reaching a clinical grade, their future will be defined by the strength of uniformly approved AI algorithms. Moreover, AI-based consumer sleep technologies which may become affordable in years to come definitely holds promise to meet the sustainable developmental goal of good health and wellbeing to play a significant role in modulating the surge in various non-communicable diseases.

The trajectory of the circadian rhythm of heart rate in ICU patients is associated with increased mortality: a multicenter retrospective cohort study

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The circadian clock, a fundamental biological mechanism present in nearly all organisms, enables the anticipation of daily fluctuations and regulation of homeostasis. Disruptions to this clock, such as from shift work or jet lag, can lead to significant physiological and behavioral deficits. Numerous methods have been developed to identify robust human circadian biomarkers. In this study, we introduced an analytical strategy to examine diurnal patterns in heart rate (HR) and their association with cardiovascular disease (CVD) development. We also characterized circadian rhythm trajectories in vital signs, explored their associations with survival outcomes in intensive care unit (ICU) patients. Our multicenter retrospective analysis evaluated dynamic changes in circadian rhythms in ICU patients. Contrary to expectations, circadian rhythm alone did not predict favorable outcomes. Instead, an arrhythmic HR trend in the first three ICU days was associated with a lower mortality risk. These findings suggest that dynamic circadian changes may reflect adaptive responses, potentially decoupling the circadian clock from external cues to prioritize internal homeostasis. Under severe stress or organ damage, circadian mechanisms may hinder the body's ability to maintain equilibrium. Our results suggest that the HR trajectory of circadian rhythm represents an unmodifiable clock function, predicting adverse outcomes.

Function of circadian core oscillators in land plants

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The circadian clock is a timekeeping mechanism synchronizing self-sustained physiological rhythms to the 24-h environmental cycles. We found the detached shoot and root possess 24-h rhythmic protein–protein interactions between clock core components, in which circadian periodicity exhibits a difference in organs. Compared to wild type, the period length difference between shoot and root was not remarkable in prr7-3 and prr7-3 prr9-1 mutants. Further, the phase transition curve (PTC) indicated that shoot and root clock respond differently to the resetting cues of ambient temperature. PRR9 and PRR7 compensate circadian period between 22 and 28 °C in shoot, not in root. In addition, we found that a subfamily of zinc finger transcription factors, B-box (BBX)-containing proteins, have a critical role in fine-tuning circadian rhythm. Overexpressing *Arabidopsis thaliana* BBX19 and BBX18 significantly lengthened the circadian period, and the null mutation of BBX19 accelerated the clock pace. Moreover, BBX19 and BBX18 protein, which are expressed during the day, physically and dynamically interacted with PRR9, PRR7, and PRR5 in the nucleus in precise temporal ordering from dawn to dusk, consistent with the respective protein accumulation pattern of PRRs. Collectively, our findings demonstrate the circadian rhythmicity and tissue specificity of interactions between clock proteins, which determine the regulation of their target genes to perform physiological functions and stress responses at specific times of a day and specific organs.

The role of an evolutionarily young testicular gene in modulating circadian rhythms in human

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Circadian clock controls a variety of physiology and behavior processes which endowing the capabilities to adapt to the cycling environmental cues. In addition to core circadian clock components, many regulators are implicated in the fine controlling of circadian rhythms. Here we report the identification of a PER2 antisense gene (Testicular TDP-43 Related Protein gene, TTRP), which is a human testis-specific gene owing to lower methylation at its promoter in testis. TTRP overlaps with the core clock gene PER2 which interferes with the transcription of the latter. In addition, TTRP encodes a protein which interacts with clock proteins PER2, BMAL1 and CRY2 and attenuates the interaction between BMAL1 and CLOCK, thus regulates the circadian negative feedback loop. Overexpression of TTRP led to reprogramming of circadian rhythms of global gene expression, including those genes associated with circadian clock and miosis. TTRP appears exclusively in Homininae that is derived from homologous pseudo gene $\sim$ 18 Mya ago. Overexpression of gorilla TTRP changed the period and amplitude of circadian rhythms in U2OS cells. Knock-in and expression of human TTRP in mouse testis led to prolonged brood-interval time and increased ratio of sperms with high motility in epididymides. These findings highlight the important function of TTRP as a novel regulator of circadian rhythms and suggest that human circadian clock may be undergoing continuous evolution by recruiting novel factors.

Effects of oral appliance therapy in patients with obstructive sleep apnea: data presentation

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Oral appliance helps maintain an open airway and prevent collapse by positioning the mandible forward. We investigated the relationship between tongue volume (TV), oral cavity volume (OCV), and their ratio (TV/OCV) with upper airway dimensions using cone-beam computed tomography (CBCT). We performed manual segmentation of the oral cavity, tongue, and upper airway volumes from CBCT scans of 15 subjects, with a mean age of 21.86 years (range 15–33 years). The segmentation process utilized Mimics 11.0 software (Materialise, Leuven, Belgium) with varying thresholds for air and tongue tissue. The Hounsfield units (HU) for airway volume ranged from $-$ 1024 to $-$ 500, while those for tongue volume ranged from $-$ 200 to 200. Our analysis demonstrated a significant negative correlation between the ratio of tidal volume to oral cavity volume (TV/OCV) and oropharyngeal volume ($r = -0.51$; $P = 0.04$), as well as between TV/OCV and oral cavity airway volume ($r = -0.74$; $P = 0.002$). Conversely, a significant positive correlation was observed between TV/OCV and tongue volume ($r = 0.65$; $P = 0.009$). The results indicate a significant negative correlation between the TV/OCV ratio and both oropharyngeal and oral cavity airway volumes, suggesting that the volumes of the tongue and oral cavity, along with their ratio, may significantly influence oropharyngeal patency.

Obstructive sleep apnea pathophysiology and treatment overview

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Multiple factors determine upper airway patency during sleep. Patients with obstructive sleep apnea (OSA) tend to have small upper airways either secondary to a small bony enclosure or due to increased soft tissue surrounding the airway. The upper airways of OSA patients are narrower and more collapsible. Men have a smaller difference in their sleeping $PaCO_2$ and apneic threshold, predisposing them to central apnea or hypopnea after periods of hyperventilation. There are few experimental considerations for study to prove the benefit of CPAP treatment on cardiovascular outcomes. During normal, non-rapid eye movement (NREM) sleep, there is a decrease in metabolic rate, sympathetic nerve activity, blood pressure, and heart rate, whereas vagal tone increases compared with wakefulness. OSA changes this normal pattern considerably. Treatment options for mild OSA included the side sleep position, treatment of nasal congestion, weight loss (adjunctive), an oral appliance (OA), or upper airway surgery. Studies found an increase in upper airway size in the lateral position. Changes in the airway shape or size with changes in posture could be due to an effect of gravity on the tissue surrounding the upper airway or to posterior movement of the tongue. Lower lung volume in the supine position may also reduce upper airway size. Treatment with an OA is indicated for patients with primary snoring and mild to moderate OSA who prefer OAs to CPAP. Oral appliance devices include MRAs and tongue-retaining devices.

Surgical decision making on sleep apnea and sleep disorders

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Obstructive sleep apnea (OSA) is confirmed by polysomnography, which shows continued inspiratory effort despite airway obstruction, evidenced by abdominal and thoracic muscle contraction. Surgical intervention for snoring and obstructive sleep apnea is considered when: (1) a surgically correctable abnormality is identified as the primary cause. (2) Continuous positive airway pressure (CPAP) therapy has failed. In addition, patients may opt for surgery after non-invasive treatments prove ineffective or intolerable. Surgical procedures typically involve modification of one or more upper airway structures, such as: nasal septum–inferior nasal turbinates–adenoids–tonsils–anterior and posterior tonsillar pillars–uvula–soft palate–base of the tongue craniofacial abnormalities (acquired or congenital) may also be surgically corrected. In rare cases, obstruction occurs at the laryngeal level (e.g., tumor and laryngomalacia). Depending upon the level of obstruction usually confirmed by DISE and radiology and need of patient and severity, the surgical decision is made.

Math + AI + wearables for treatment of circadian and sleep disorders

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The circadian clock, an internal time-keeping system orchestrates 24-h rhythms in physiology and behavior by regulating rhythmic transcription in cells. Astrocytes, the most abundant glial cells, play crucial roles in central nervous system (CNS) functions, but the impact of the circadian clock on astrocyte functions remains largely unexplored. In this study, we identified 412 circadian rhythmic transcripts in cultured mouse cortical astrocytes through RNA sequencing. Gene Ontology analysis indicated that genes involved in Ca2 + homeostasis are under circadian control. Notably, Herpud1 (Herp) exhibited robust circadian rhythmicity at both mRNA and protein levels, a rhythm disrupted in astrocytes lacking the circadian transcription factor, BMAL1. HERP regulated endoplasmic reticulum (ER) Ca2 + release by modulating the degradation of inositol 1,4,5-trisphosphate receptors (ITPRs). ATP-stimulated ER Ca2 + release varied with the circadian phase, being more pronounced at subjective night phase, likely due to the rhythmic expression of ITPR2. Correspondingly, ATP-stimulated cytosolic Ca2 + increases were heightened at the subjective night phase. This rhythmic ER Ca2 + response led to circadian phase-dependent variations in the phosphorylation of Connexin 43 (Ser368) and gap junctional communication. Given the role of gap junction channel (GJC) in propagating Ca2 + signals, we suggest that this circadian regulation of ER Ca2 + responses could affect astrocytic modulation of synaptic activity according to the time of day. Overall, our study enhances the understanding of how the circadian clock influences astrocyte function in the CNS, shedding light on their potential role in daily variations of brain activity and health.

Evoked brain during sleep

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Sleep is not a homogeneous state. It is characterized and studied by brain wave patterns obtained through electroencephalographic (EEG) methods. In addition to changes in EEG activity during sleep, there are also alterations in neuromodulation and cognition between the sleep stages. These changes affect the processing of external stimuli in the brain. Evoked potentials are one of the main research tools used to assess information processing. Evoked potentials are small amplitude changes in the EEG induced by an external physical stimulus. The shape, amplitude, and timing of these potentials differ across sleep stages, providing crucial information on how external stimuli are processed during sleep. In this presentation, brain responses to auditory or non-painful tactile stimuli administered to individuals during sleep will be evaluated and presented, considering both topological and hemispheric aspects.

The brain responsiveness across the brain states

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Measuring consciousness presents unique challenges, requiring robust methods across various domains to capture the brain's complexity. The brain's functional organization is a multidirectional process, where inputs and outputs are regulated, while preserving the continuum of sleep. Traditional concepts, such as cognition or behavioral outputs, serve as key metrics for evaluating the wakeful brain in psychological contexts. However, using these tools to assess cognition across the ever-changing states of the brain during sleep is nearly impossible. In this context, brain responsiveness emerges as a promising tool for gauging vigilance and operational capacity across different states of consciousness. This presentation will explore examples from both clinical and experimental settings, demonstrating how brain responsiveness can provide critical insights into brain function from milliseconds to extended time periods, helping to better understand biological rhythms and transitions between states of consciousness. By concurrently applying these methods, we can begin to uncover the brain's intricate workings, offering a more dynamic and comprehensive approach to studying sleep and consciousness.

Circadian clock, cancer, and chronotherapy

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The circadian clock, regulated by the suprachiasmatic nucleus (SCN) in the hypothalamus, is critical for maintaining human physiological processes, metabolic cycles, and behavioral patterns. Acting as the body's master clock, the SCN synchronizes peripheral clocks in various organs with external environmental cues, particularly the light–dark cycle. At the cellular level, molecular clocks are composed of interdependent clock genes that operate through complex feedback loops to maintain circadian rhythm. Studies on both human and animal models have shown that disruptions to these rhythms can lead to a wide range of health issues, such as metabolic syndrome, cardiovascular diseases, and notably, cancer. These circadian systems also play a significant role in modulating drug metabolism and action, laying the foundation for the emerging field of cancer chronotherapy. Cancer chronotherapy aims to exploit the relationship between circadian rhythm disruption and tumor progression to optimize cancer treatment. By applying the principles of circadian biology, treatments can be precisely timed to maximize drug efficacy while minimizing side effects. Monitoring the circadian rest-activity cycles in cancer patients serves as a reliable marker of internal clock function, guiding the timing of therapeutic interventions. Administering anticancer agents at specific points in the circadian cycle has been shown to reduce toxicity and enhance therapeutic outcomes. However, variability in individual circadian rhythms can result in different responses to treatment. Therefore, personalized chronotherapy, which integrates chrono-pharmacological principles, mathematical modeling, and advanced technology, holds great promise for addressing these inter-individual differences.

Differential neuronal responses of diurnal and nocturnal rodents to chronic altered light–dark cycles

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Sleep, clocks, and immunity are intertwined. Sleep loss, and circadian dysfunction impairs the immune response. Conversely, immune activation alters sleep. To better understand the relationship between sleep, clocks, and immunity, we studied flies carrying mutations in different anti-microbial peptides (AMPs). These AMPs fall in five classes—Group A (Defensin), Group B (Attacins, Diptericins, and Drosocin), Group C (Drosomycin and Metchnikowin), Cecropins and Bomanins. We find that deletion of AMPs reduced sleep time and, in some cases, increased sleep latency (a function of the circadian clock). In addition to differences in baseline sleep, we also observed differences in responses to sleep modulatory inputs e.g. social enrichment and starvation. Further, different classes of AMPs differently impacted learning and synapse abundance. Interestingly, the Group C mutant flies that slept the least retained the ability to learn, and downscale synapses—two reported functions of sleep. This suggests that these flies are able to carry out sleep functions despite not getting much sleep. Further, Group C mutant flies also displayed a slightly shortened circadian period. Together, these data are a thorough characterization of the effects of AMPs on sleep, clocks, and sleep and clock-dependent outputs.

Neuropathy and insomnia

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The prevalence of sleep disturbance in patients with chronic pain ranges from 50 to 80%, and the severity of sleep disturbance is related to pain intensity. The effects of neuropathic pain on sleep quality have been examined directly. The relationship between neuropathic pain and sleep disturbances is bidirectional. Patients with neuropathic pain are more likely to develop sleep disorders, and in turn, pain is exacerbated by the lack and/or poor quality of sleep. Diabetic neuropathy is one of the neurological complications in diabetic type 2. Painful diabetic neuropathy, depression and insomnia are part of the symptoms of diabetic neuropathy. The aim of a study was to measure correlation of painful diabetic neuropathy and depression compared to insomnia on diabetic neuropathy patients. In a cross-sectional study in Dr. Hasan Sadikin and Dr. M. Salamun Hospital Bandung, Indonesia (2018) with total of 60 diabetic neuropathy patients, we found 71.6% with painful diabetic neuropathy (Douleur Neuropathique 4 Question Score > 4), 71,6% with depression (Hamilton Depression Rating Scale > 7), and 58.33% with insomnia (Insomnia Severity Index > 7). In the conclusion of this study, there was correlation between painful diabetic neuropathy and insomnia and between depression and insomnia.

Nocturnal epilepsy and its mimickers in sleep

Benjamin Ng Han Sim

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Paroxysmal events during sleep are always a clinical challenge to treating clinicians, as these events are often not observed and only told by the eyewitness; be it bed partner or concerned parents. Empirical treatment of these events may miss treating the underlying cause and exposing patients to the potential side effects of treatment. In this talk, I would like to share my clinical perspective on parasomnias and how to differentiate these from nocturnal epileptic attack. Management of each would be discussed. The aim of my talk is to bridge what we learnt from sleep research and the clinical practice of sleep medicine. It is hope that this talk would increase the interest in parasomnias and their mimics among the clinicians.

The reciprocal relationship between stroke and sleep disorders

Zamroni Afif

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A wide range of sleep disorders are associated with stroke, including sleep-disordered breathing, insomnia, parasomnia, circadian rhythm disorder, sleep-related movement disorder, hypersomnia and excessive daytime sleepiness. Most stroke patients (20–63%) experience sleep disorders. Sleep disturbance can be a risk factor for stroke, for example, by significantly increasing the risk of ischemic stroke. Impaired sleep disturbances arise due to factors associated with stroke, such as sleep-disordered breathing, medication use, inactivity, environment, depression, stress and premorbid health problems. Insomnia can impact upon a patient's functioning, including excessive daytime sleepiness, fatigue, memory and attention potentially leading to stress and depression. Sleep disorder maybe related with location of stroke. Sleep problems can arise as a consequence of the stroke itself, in which brain areas involved in sleep regulation, including the hypothalamus, brainstem and thalamus. Severe sleep disturbance suggests bilateral paramedian thalamic, mesencephalic or brainstem infarction, but may also be seen in large hemispheric ischemia. Insomnia is present when the thalamic infarction involves the posterolateral, ventral posterolateral, dorsomedial and centromedial nuclei. Obstructive sleep apnea (OSA) is an independent risk factor for stroke but can also develop or progress as a result of stroke. Effective management of post-stroke patients requires identifying and treating sleep quality disorders and associated risk factors to improve quality of life and reduce complications associated with stroke.

Future methodology prospects and emerging fields in sleep

Murat Ozgoren

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Measuring consciousness presents unique challenges, necessitating the development of robust, adaptable methods. One of the main difficulties lies in the inherent complexity and dynamic nature of brain function. While scientific methods traditionally rely on fixed, well-defined parameters to measure specific phenomena, this approach proves inadequate when applied to the brain. The brain's functional states can shift dramatically within seconds, making it impossible to rely on static parameter sets for accurate measurements. This talk will explore the limitations and constraints posed by current methodological approaches in assessing consciousness, particularly in relation to the brain's ever-changing complexity during sleep. We will delve into the boundaries of existing algorithms, highlighting both their shortcomings and potential solutions. In addition, with the rapid advancements in computational power, sensor technology, and techniques derived from diverse scientific fields, we need to rethink and redesign our research strategies. Incorporating these innovations into a more flexible and comprehensive framework will be crucial for improving the accuracy and depth of our understanding of the sleeping brain. This presentation will provide insights into how these advancements can be orchestrated to develop more effective tools for sleep research.

Brain measurement and limitations

Adile Oniz Ozgoren

Near East University, Cyprus, Turkey

Memory encompasses a series of cognitive processes related to the storage and retrieval of information, while learning refers to the acquisition and application of this information. Learning and memory are widely studied topics in different states of consciousness. However, clarity has yet to be achieved in this area. It is possible to measure learning in sleep and other non-waking states of consciousness (such as under anesthesia) through psychometric tests and electrophysiological methods. The most important memory processes in the approach to learning are explicit and implicit memory processes. Explicit memory is defined as the type of memory that is voluntarily recalled and verbally expressed, whereas implicit memory refers to the retrieval of information without voluntary and conscious recall. In this context, explicit, implicit memory, and learning processes during sleep, wakefulness, and anesthesia can be examined through a battery capable of measuring different memory subdomains. Examples from studies that investigate types of memory and the effect of learning during anesthesia and sleep using a developed battery will be presented.

Setting up auditory and tactile modalities for sleep

Gonca INANC

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Sleep is a vital process for the brain and body, and understanding how responses to external stimuli change during this period can provide important insights for both neuroscientific research and clinical applications. In this talk, the changes in brain response components to auditory and tactile stimuli during sleep, particularly in different stages of sleep (e.g., REM and NREM sleep), will be discussed. Significant differences are observed in how stimuli are perceived and processed by the brain in these stages. Brain waves measured using electroencephalography (EEG) reveal how brain activity changes in response to stimuli. This allows for the investigation of differences in brain activity in response to auditory and tactile stimuli during sleep. Finally, the focus will be on how these findings can be applied to sleep research and clinical practice. Analyzing responses to stimuli may contribute to the development of new approaches for diagnosing and treating sleep disorders. Examples will be presented on how the brain perceives and processes different external stimuli during sleep.

What does big data say about narcolepsy?

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Narcolepsy is a rare and debilitating condition, making accurate identification of patients crucial for understanding the disease's epidemiology, clinical features, and response to treatment. Large-scale electronic medical record (EMR) data sources, such as the Veterans Health Administration (VHA) database, which contains millions of health records, provide an opportunity to identify and study such conditions. Traditionally, chart review has been the gold standard for uncovering similarities and differences in patient presentations, as well as for identifying comorbidities or potential subtypes of diseases. However, this method is often labor-intensive, time-consuming, and can be biased by geography or institutional practices. These challenges are exacerbated in the study of less common diseases like narcolepsy, where the limited number of available charts can further impede research. AI tools that identify correct patients can be of help. In this symposium, we will review the establishment of a registry as a prospective mean to collect information and help with diagnosis and management of patients with narcolepsy. We will also discuss the use of AI in extracting information on a large cohort of patients with narcolepsy. Finally, we present data on 25,000 patients with narcolepsy using AI analysis of VHA big data.

Turkish Narcolepsy Network—current directions and future perspectives

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The term narcolepsy is a combination of the Greek words "narko" meaning "sleep" and "lepsy" meaning "possession". The main clinical finding is excessive daytime sleepiness, which is often accompanied by findings originating from the rapid eye movements (REM) sleep phase. These findings include cataplexy, sleep paralysis (nightmare), hypnagogic/hypnopompic hallucinations and disturbed night sleep. Narcolepsy type 1 is characterized by the presence of cataplexy as an accompanying clinical finding and/or low levels of hypocretin in cerebrospinal fluid (CSF). Cataplexy describes a sudden and transient complete or partial loss of power in striated muscles in which consciousness is not affected; it may occur spontaneously or is often triggered by an emotional stimulus. The diagnosis of narcolepsy type 1 (NT1), formerly known as narcolepsy with cataplexy, can be made without cataplexy if the CSF hypocretin level can be measured and is found to be 110 pg/mL or less or less than one third of the average laboratory data. The prevalence of NT1 is very low, around 0.02–0.18%. Narcolepsy should be differentiated from other more common causes of hypersomnolence, such as sleep deprivation, sleep-related respiratory disorders and other sleep-related disorders. Conditions such as revealing secondary causes, interrogation of neurologic and psychiatric diseases, drug and substance use or discontinuation should also be excluded. Treatment options include non-pharmacologic and pharmacologic approaches. Turkish Narcolepsy Network, a study group of the Turkish Sleep Medicine Society, aims to increase the awareness of narcolepsy and other central disorders of hypersomnolence.

Understanding clock dynamics in aging

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Aging is associated with changes in several basic parameters of circadian timing system (CTS) leading to circadian dysfunction. The suprachiasmatic nucleus (SCN) in hypothalamus in brain contains a light-entrained circadian clock involved in regulation of neuronal, endocrine and behavioural rhythms. It regulates the rhythmic production and release of melatonin (messenger of darkness) from pineal gland involving a network of interconnected transcriptional and translational feedback loops. It is suggested to have a link with alterations and disruptions occurring during aging and age-related diseases such as Alzheimer's disease, Parkinson's disease, dementia, sleep and metabolic disorders. We have reported from our group changing dynamics of clock function with aging in various behavioural, histological, biochemical and molecular parameters. Our studies have demonstrated alterations in daily locomotor rhythms, expression of various clock genes, 5-HT metabolism components, protein profiles, antioxidant enzymes, immune genes as well as molecular markers of inflammation, learning and memory affecting sleep and mental health. We further studied therapeutic effects of melatonin and herbal nutraceuticals Curcumin and Ashwagandha on age induced perturbances in stoichiometry of daily chronomics in aging influencing functional integrity of CTS. In addition, we have observed ameliorating effects of Time restricted feeding (TRS) and Ketogenic diet intervention in restoration of Circadian function on age-induced desynchronization. This work may prove useful towards targeting novel treatments for circadian dysfunction, good health and longevity.

Integrating circadian biology into our understanding of aging for potential health and therapeutic benefits

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With increasing age, most likely, our unequivocal timekeeping machinery breaks down, which leads to low adaptability to the periodic changes in the environment. However, the precise molecular pathways and mechanisms linking circadian dysfunction and aging are largely obscure. We curated a comprehensive map of human aging markers (besides homologs in mice and Drosophila) from publicly available databases. Furthermore, we created a tissue/organ-specific rhythmic aging gene map by tracing our circadian datasets in the Human Protein Atlas. We performed metabolomics of young and old Drosophila, the fruit fly, revealing significant variations in differentially abundant metabolites. The activity-based behavioral analysis indicates that the young flies displayed distinct increased activity at dawn and dusk. In contrast, older flies exhibited multiple period peaks, reduced amplitude, and erratic behavior, indicating a perturbed self-anticipatory day–night cycle. We have also investigated the diurnal rhythmicity in various physiological parameters and electrolyte levels in elderly human participants (age > 60). Most parameters exhibited diminished rhythmicity, likely attributing to the attenuation of inherent rhythmic patterns with aging. However, serum

cortisol, sodium, and creatinine rhythms were still rhythmic in the aged cohorts. The findings may offer new clues to target our time-keeping machinery for gaining therapeutic benefits in aging attenuation.

Restless legs syndrome and stroke—association and role in risk stratification

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The introductory talk will feature lessons from our work on prevalence and characterization of pre-stroke restless legs syndrome with unique features, leading up to determination of specific associations with and differentiation from other cardiocerebrovascular risk factors like resistant hypertension and also with cerebral small vessel disease. Evidence suggests that pre-existing RLS (and possibly association periodic limb movements) could exacerbate stroke risk through mechanisms such as sympathetic overactivity and sleep disruption. While more common in subcortical strokes, RLS's role in post-stroke recovery remains underexplored. This talk will review the current evidence, emphasizing the need for incorporating RLS into risk stratification models for better stroke prevention and management.

COMISA: it is complicated! A real-world data set and treatment plan

Supriya Singh

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The goal of this proposal aims to discuss traditional evaluation of COMISA using big data. Furthermore, we will discuss innovations with large language models and machine learning in assessment and diagnostic considerations. Our goal is to show beneficial outcomes in patients in corroboration with comorbid diseases. In addition, the proposal aims to discuss a model for effective clinical management. Finally, we are utilizing a patient-centered care approach.

Embracing the night: how AI and machine learning is revolutionizing COMISA and cognitive behavioral treatment

Tara Thomas

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Large language models (LLMs) and machine learning are integrated to identify epidemiological factors and clinical correlates/outcomes for patients with COMISA. Furthermore, we will discuss clinical considerations for patient-centered evaluation and treatment in patients with COMISA. We will develop interdisciplinary treatment plans for patients with COMISA that integrate both medical and cognitive-behavioral therapy. AI is allowing us to provide a variation in CBT-I; therefore, we are tailoring our treatment for COMISA patients. We are optimizing CBT-I interventions. It can help us to recruit the patients who benefit the most, and therefore, it will help us allocate our resources effectively. Lastly, we will discuss the current state-of-the science and best practices regarding interventions for COMISA and apply this when sequencing interventions during routine clinical management. Using these innovative processes, we are hoping to advance the future of sleep medicine.

Utilize artificial intelligence to optimize clinical interventions for COMISA

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The integration of large language models (LLMs) and machine learning (ML) into sleep medicine is revolutionizing the study of conditions like COMISA (co-occurring insomnia and sleep apnea), particularly when applied to large national retrospective databases. These advanced tools enable the extraction, organization, and analysis of vast amounts of unstructured data, such as electronic medical records (EMR) and polysomnography (PSG) reports, to form comprehensive, high-quality big data cohorts. By leveraging LLMs and ML, researchers can uncover complex interactions between insomnia and sleep apnea, identify population-level trends, and explore their impact on health outcomes. With several publications in sleep medicine utilizing machine learning and natural language processing (N ha LP), my work s demonstrated the potential of these methods in advancing the understanding and management of COMISA, enabling evidence-based approaches to improve patient care.

Advancing precision medicine: the role of automated PSG scoring in AI-driven biomarker discovery and clinical applications

Kishan

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Advancements in precision medicine require innovative tools to analyse complex physiological data, particularly from polysomnography (PSG), the gold standard for assessing sleep disorders. Manual scoring, while accurate, is time-intensive and prone to variability, limiting its scalability for clinical research and biomarker discovery. Automated PSG scoring overcomes these barriers by enabling consistent, efficient, and accurate analysis, transforming sleep data into actionable insights for personalized healthcare. The Neurobit Hub clinical trial platform enhances biomarker discovery by integrating AI-driven algorithms with standardized workflows, supporting research in conditions such as narcolepsy, hypersomnia, and CPAP compliance. For narcolepsy, the platform facilitates the identification of biomarkers linked to sleep architecture abnormalities and REM onset latency. In hypersomnia, automated analysis enables researchers to quantify excessive sleep duration and fragmented sleep patterns, offering a data-driven approach to refine diagnostic criteria. In CPAP compliance research, the Neurobit Hub supports the evaluation of treatment efficacy by analysing longitudinal sleep metrics, helping clinicians optimize patient management strategies. This comprehensive approach empowers researchers to uncover novel biomarkers tailored to specific disorders, bridging the gap between sleep physiology and clinical applications. By integrating automated PSG scoring with a robust clinical trial platform, Neurobit accelerates the discovery of actionable biomarkers, advancing personalized

treatments and improving patient outcomes across a range of sleep-related conditions.

Leveraging AI-driven multimodal sleep data analysis: from complex PSG patterns to predictive clinical outcomes

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Polysomnography (PSG) is the gold standard for diagnosing and understanding sleep disorders, yet its potential remains underutilized due to the limitations of manual scoring and reliance on oversimplified metrics such as the Apnoea–Hypopnea Index (AHI). This session explores how AI-driven multimodal analysis transforms complex PSG patterns into actionable clinical insights, enabling the identification of novel biomarkers for predictive and personalized healthcare. Key focus areas include ventilatory burden (VB), hypoxic burden, and arousal burden—biomarkers that go beyond traditional indices to offer a comprehensive view of sleep physiology. VB, which quantifies the proportion of impaired breaths, has demonstrated strong predictive value for cardiovascular and all-cause mortality. Hypoxic burden captures the total oxygen desaturation experienced during sleep, providing insights into the systemic impacts of sleep-disordered breathing. Arousal burden measures the frequency and intensity of sleep disruptions, contributing to the understanding of fragmented sleep and its physiological consequences. This presentation will delve into the development and clinical applications of these biomarkers, illustrating their utility in diagnosing and managing conditions such as obstructive sleep apnea, hypersomnia, and narcolepsy. By leveraging AI to uncover patterns across these multidimensional data streams, clinicians can achieve more precise disease characterization, optimize treatment strategies, and improve patient outcomes. This session underscores the transformative potential of AI in advancing sleep medicine toward a new era of precision diagnostics and predictive healthcare.

Translating AI-driven sleep analytics to clinical research: experiences and insights from an Indian academic institution

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Our research leverages AI-driven sleep analytics through wearable devices to investigate the impact of night shift work on healthcare workers in a tertiary care hospital in West Bengal. This study represents a crucial intersection of workplace health, sleep medicine, and AI-enabled diagnostics in an Indian healthcare setting. Using continuous sleep monitoring, we analyse sleep architecture, circadian disruptions, and recovery patterns among healthcare workers across different shift schedules. The AI-powered wearable analytics enable us to capture comprehensive sleep metrics including sleep-onset latency, sleep efficiency, sleep stage transitions, and heart rate variability. This granular data collection, previously impossible with traditional methods, allows us to identify subtle patterns in sleep disruption and their correlation with various health parameters. This presentation will discuss our experience implementing AI-driven sleep monitoring in a resource-conscious setting, including challenges in data validation, user compliance, and integration with existing health monitoring systems. We will also share insights on adapting these technologies for Indian healthcare workers, considering unique cultural and institutional factors affecting sleep patterns and shift work adaptation.

Obstructive sleep apnea among commercial drivers in North India

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Obstructive sleep apnea (OSA) is the most common medical cause of excessive daytime sleepiness and identified worldwide as risk factors for road accidents. To estimate the risk of (OSA) in commercial drivers in North India and its correlation with vehicle accidents or near miss accidents. Cross-sectional study was done in a metropolitan city. 153 commercial drivers with minimum 5 years of driving experience with age ranging between 24 and 65 years were enrolled by simple random sampling technique. All the demographic, driving history including total years of driving, vehicular collisions encountered due to sleepiness, near miss accidents due to sleep, and anthropometric and historical data were obtained. Sleep quality was assessed using PSQI. The risk of OSA and excessive daytime sleepiness was assessed using the STOP BANG questionnaire and Epworth Sleepiness Scale, respectively. On the basis of questionnaire, 21.56% of the drivers were at risk of OSA. There was no significant correlation between OSA and collisions. 28.10% of the commercial drivers had poor sleep quality, whereas 15.16% of the high-risk OSA commercial drivers had excessive daytime sleepiness. Prevalence of risk for OSA is high among commercial drivers. The sleep quality of commercial drivers was poor. In country of 1.3 billion population, the studies pertaining to OSA in commercial drivers are scarce. There are no guidelines for OSA detection in commercial drivers in India despite the fact OSA is a major cause of excessive daytime sleepiness and may lead to sleeping on wheels.

Melatonin supplementation in substance addiction

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Recent evidence links melatonin hormone and its receptor to the etiology and behavioural manifestation of addiction. The role of exogenous melatonin in addiction treatment is still inconsistent and unclear. The present study aimed to review the literature on randomized clinical trials that evaluated the effect of melatonin supplementation, compared to placebo, in the treatment of various substance addictions. The main outcome measure of this systematic review was the assessment of sleep quality which is the major predictor of treatment outcome. In this review, we have included randomized clinical trials published in MEDLINE and Google Scholar databases, investigating the effect of melatonin treatment, compared to placebo, on substance addiction-related parameters. Non-randomized clinical trials, observation studies, and animal studies were excluded. Of 537 articles, 12 randomized control trials (RCT) met our inclusion criteria. Studies have been conducted on substances of addiction including benzodiazepine (BZD), alcohol, nicotine, and opioids. Our results indicated that melatonin treatment

had mixed results in improving sleep quality and was not found beneficial in BDZ cessation/discontinuation rate among patients with BDZ dependence. Sleep quality and mental health had improved by melatonin supplements in opioid addiction. In nicotine addiction, melatonin treatment showed effectiveness only on mood changes but not in performance tests. In patients with alcohol use disorder (AUD), melatonin treatment did not show any improvement in sleep quality. We found that the use of exogenous melatonin in substance addiction has mixed results which do not provide sufficient evidence, relative to randomized clinical trials, to establish its role.

Is there a phantom of dementia in OSA?

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Obstructive sleep apnea (OSA) is a common sleep disorder linked to daytime sleepiness, however can impair cognitive function, including memory and attention. CPAP therapy, the standard treatment for OSA, improves sleep quality. This study aimed to investigate whether CPAP therapy can enhance cognitive function in OSA patients. This prospective single-centre observational study evaluated the impact of CPAP therapy on cognitive function in OSA patients. Patients were diagnosed based on clinical history and polysomnography. A comprehensive neuropsychological battery assessed cognitive function before and after one year of CPAP therapy. Of the 45 patients (80% male), AHI significantly decreased from 31.4 to 6.2 post-1 year of CPAP therapy ($p < 0.001$). Global cognitive function improved, with a 2-point increase in ACE score ($p = 0.003$). Significant improvements were observed in: attention: digit span forward (median of 7–8.5, $p < 0.001$), executive function: trail making B (median duration pre-test 210 s and post-test 186 s, $p = 0.019$), WMS paired association test (mean pre-test 13.01 ± 3.78–14.52 ± 4.04 post-test, $p < 0.001$), language: confrontation naming (mean: 49.73 ± 6.38–50.67 ± 7.07, $p = 0.006$), letter fluency ($p < 0.001$), category fluency($p < 0.001$); memory: RAVLT total, immediate, and delayed recall (all $p < 0.001$). While psychological well-being showed a trend of improvement, it was not statistically significant. CPAP therapy significantly improved cognitive function in OSA patients, including attention, executive function, language, and memory. While psychological well-being showed a trend of improvement, it was not statistically significant. Long-term CPAP adherence with significant AHI reduction can positively impact overall cognitive and psychological well-being.

Factors associated with restorative sleep in patients with depression: a focus on sleep–wake state discrepancy

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Sleep disturbance is a common symptom in patients with depression, and non-restorative sleep can negatively impact the recovery process. While factors associated with restorative sleep have been investigated in healthy subjects and patients with insomnia, there is a lack of research evaluating these factors in patients with depression. Depressed patients often experience sleep–wake state discrepancy, which may further influence their restorative sleep. We aim to investigate restorative sleep in depressed patients and identify associated factors, with a particular focus on sleep–wake state discrepancy. In this cross-sectional study, 91 participants completed questionnaires assessing their symptoms before undergoing polysomnography (PSG). The morning following the PSG, subjective sleep duration and restorative sleep were assessed. Multiple regression analysis was performed to examine the relationship between restorative sleep and associated factors, including demographic characteristics, sleep–wake state discrepancy, depression severity, daytime sleepiness, and PSG findings. The study was approved by the Bioethics Review Committee of the Nagoya University School of Medicine. Multiple regression analysis revealed an association between restorative sleep and depression severity ($\beta = -0.055$, $p = 0.007$), daytime sleepiness ($\beta = -0.106$, $p = 0.020$), and overestimation of wake after sleep onset ($\beta = -0.006$, $p = 0.030$). The impact of sleep–wake state discrepancy and depression on the perception of restorative sleep may be greater than that associated with sleep stages in individuals with depression. To improve the restorative sleep of depressed patients, clinicians should implement sleep interventions to address sleep–wake state discrepancy alongside depression treatment.

Clinical characteristics and risk factors of sleep-related eating disorder: a study in psychiatry outpatients

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Sleep-related eating disorder (SRED) is a parasomnia characterized by involuntary eating behavior during non-REM sleep, and often with impairment of consciousness level. SRED is sometimes associated with psychotropic medications, in particular sedative hypnotics, and comorbid with psychiatric disorders. Although SRED has significant negative effects on general health, its precise pathophysiology is still unclear. We examined clinical profiles in patients with SRED to clarify the risk factors and comorbid psychiatric conditions. We examined 3371 patients who visited our psychiatry outpatient clinic during October 2023 to September 2024. SRED was diagnosed according to the third edition of the International Classification of Sleep Disorders. Age, sex, psychiatric comorbidities, medications, and clinical symptoms were also evaluated. Eleven of 3,371 (0.3%) patients met the diagnostic criteria for SRED. The mean age of SRED patients was 44.7 ± 6.5 years and nine (81.8%) were female. Among 376 patients with bipolar disorder, five (1.3%) had SRED. All SRED patients reported experiencing depressive symptoms during their clinical course. At least nine cases exhibited SRED symptoms while using benzodiazepine receptor agonists (BzRA) as sedative agents. There was no increase in the BzRA dose at the previous visit when SRED symptoms appeared. In three cases, SRED symptoms improved following a reduction of BzRA. These results suggested that mood disorders, depressive symptoms and dosage of BzRA are associated with SRED.

A cross-sectional study on the impact of pre-sleep smartphone usage on sleep quality among undergraduate students

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Smartphone use is increasingly necessary among medical students, with high pre-sleep usage causing disruptions in biological rhythms. The blue light from screens can lead to sleep disorders, fatigue, and mental health issues. This study investigates the effect of pre-sleep smartphone usage on sleep quality in MBBS students. Healthy MBBS undergraduates aged 18–24 ($n = 70$) with smartphone ownership participated. A customized questionnaire and Pittsburgh Sleep Quality Index (PSQI) were used. Participants were grouped as: Group A (Good Sleepers; PSQI ≤ 4) and Group B (Poor Sleepers; PSQI ≥ 5). Data were analyzed using a paired t-test and Pearson regression. The mean age of participants was 19.68 ± 1.51 years. Group A included 13 females and 20 males, while Group B had 14 females and 23 males. Both groups showed significant Instagram usage ($p < 0.05$), with Group B using more apps overall. A strong correlation was found between increased smartphone use and daytime lethargy ($r^2 = 0.89$). Second-year MBBS students exhibited high smartphone usage before sleep, resulting in decreased daytime performance, lethargy, and sleepiness. These patterns could negatively impact their health and cognitive abilities, potentially affecting their academic performance and well-being.

The prevalence of obstructive sleep apnea in treatment-naive pulmonary sarcoidosis and its impact on fatigue and quality of life

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Patients with sarcoidosis experience fatigue and poor quality of life. Studies suggested undiagnosed obstructive sleep apnea (OSA) to be an important contributor, but reliable data on the prevalence and impact of OSA in sarcoidosis are scarce. Subjects with newly diagnosed pulmonary sarcoidosis were included in the study and underwent evaluation using in-lab polysomnography. Fatigue Assessment Scale (FAS) and Functional Outcome of Sleep Questionnaire (FOSQ10) were also recorded. A total of 30 subjects (18 men, 12 women) with a mean (SD) age of 40.4 (10.31) years were included. Twenty-one (70%) were detected with OSA (11 with mild OSA, 10 with moderate-severe OSA). The mean AHI was 12 ± 12.7 in subjects diagnosed with OSA, and rapid eye movements (REM)-dominant OSA was diagnosed in 52% of the OSA subjects. The mean FAS score was significantly higher in OSA subjects (31 ± 4.5 vs 24 ± 5.24, $p = 0.004$). FOSQ10 was numerically higher in the OSA group as compared to non-OSA (13.7 ± 2.09 vs 12 ± 3.76, $p = 0.231$) but did not reach statistical significance. OSA is common in sarcoidosis subjects and leads to more fatigue. Sarcoidosis subjects should be evaluated for OSA at diagnosis.

Adult-onset, isolated, nocturnal seizures associated with REM dominant sleep apnea: a report of twelve cases

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Adult-onset seizures are the usual consequence of an acquired, enduring, structural or electrical alteration in neural networks. Isolated nocturnal seizures are known to occur in early childhood or adolescence as result of specific syndromes (JME, BCECTS, etc.) or channel mutations (e.g. ADNFLE). We encountered twelve patients over 10 years who presented with isolated, nocturnal, and sporadic seizures interspersed by long intervals of time. Diligent history taking revealed that the seizures tended to occur in the early morning hours of sleep between 4 and 6 am. The eyewitness account was characterized by generalized tonic–clonic events occasionally preceded by a vocalization. Five of the twelves experienced bony dislocations or fractures during the seizures. None of the patients had potentially epileptogenic biological antecedents like trauma or infantile encephalitis/seizures. In all patients, neurological examination, interictal EEGs, and epilepsy protocol MRIs were normal. In all patients, the seizures persisted despite institution of appropriate anti-seizure medications. In all patients, sleep history and polysomnographic evaluation revealed hitherto unrecognized obstructive sleep apnea. In all patients, the REM apnea–hypopnea indices (AHI) exceeded the NREM AHI by at least two times. In all patients, the PSG demonstrated prolonged apnea durations and steep oxygen desaturations. In seven of the twelve patients, the seizures have remitted for over 3 years after institution of CPAP therapy. In the others, the follow-ups have not completed 3 years. In some patients, adult-onset sporadic, early morning, unexplained (on routine investigations) nocturnal seizures may herald a hitherto undetected REM-dominant obstructive sleep apnea syndrome.

Sleep disturbances in children with respiratory infections: patterns of night wakings and maternal sleep associations

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Sleep disturbances are common in children suffering from respiratory infections, affecting both their rest quality and recovery. This study aims to assess the sleep patterns, night waking behaviors, and associations influencing sleep in children with respiratory infections, based on the provided dataset. Data were collected on sleep habits from a sample of children ($n = 118$), documenting key sleep parameters, including nap durations, time to fall asleep, night wakings, and sleep associations. Descriptive analysis was conducted to assess average nap duration, night waking frequency, and how the children were put back to sleep. Results: the age range of the patients is from 9 to 15 years, with a mean age of 11.8 years and a median of 12 years. The average nap duration was 2.25 h, ranging from 1 to 3.5 h. Children took an average of 9 min to fall asleep, with times ranging from 1 to 12.5 min. On average, children woke up 2 times per night

(range 1–3), with waking episodes lasting between 1 and 5 min (mean: 4 min). The most common wake-up time was 7:00 AM, and naps occurred predominantly in the afternoon. Most bedtime routines began at 11:00 PM, and children were typically in bed by 11:00 PM. Maternal presence ("sleeping with mother") was the most frequent sleep association, while "feeding" was the primary method used to settle children after waking. The results suggest that children with respiratory infections experience disrupted sleep, with frequent night wakings and reliance on maternal presence or feeding.

Evaluation of sleep spindle characteristics and slow-wave activity in post-stroke patients

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Stroke is a leading cause of disability, often accompanied by sleep disturbances that negatively impact neurological recovery. Sleep spindles and slow-wave activity (SWA), crucial elements of sleep microarchitecture, play essential roles in cognitive functions such as memory consolidation and brain plasticity. This study investigates the effects of ischemic stroke on spindle density and SWA compared to healthy. Thirty-five patients and thirty controls were screened, and fifteen participants from each group consented. Socio-demographic and clinical data were collected using case record forms. Overnight polysomnography was performed, with sleep architecture scored using Polyman software in accordance with AASM 2017 guidelines. Spindle density and SWA were calculated using Python scripts based on the YASA package. Stroke patients had significantly lower spindle density and amplitude compared to controls across all channels. In the C4 channel, stroke patients had a mean spindle density of 3.58 (SD: 1.46) and a mean amplitude of 13.9 (SD: 0.373) μV, whereas controls had a mean spindle density of 6.62 (SD: 2.06) and a mean amplitude of 14.9 (SD: 0.378) μV. Stroke patients had significantly lower average delta frequency (2.27 vs. 4.92) and amplitude (73.02 vs. 81.78) compared to controls ($p < 0.01$). This study highlights a significant disruption in spindle density, spindle amplitude, and slow-wave activity among stroke patients compared to the controls. Further exploration and characterization of these findings are required, particularly in larger cohorts of stroke patients.

Exploring the relationship between sleep disturbances and headache patterns in migraine patients

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The aim is to understand the relationship between sleep disturbances and headache in individuals with migraine. Methods: in this cross-sectional case–control study, migraine patients were divided into either episodic (EM) or chronic migraine (CM) based on their headache frequency. They were screened for sleep apnea, insomnia, and excessive daytime sleepiness using self-rated questionnaires. Their sleep quality was assessed by the Pittsburgh Sleep Quality Index (PSQI). Out of 253 migraine patients recruited in the study, 103 had episodic migraine (EM) and 150 had chronic migraine (CM), with a mean age of 36.5 ± 10.9 years and 37.5 ± 9.5 years, respectively. A total of 158 age-and gender-matched healthy controls (HCs) were also recruited with a mean age of 37.6 ± 11.1 years. Severe disability (MIDAS score above 21) was present in 50.5% of the patients in the EM group, and 87.3% of the patients in the CM group. Clinical insomnia (ISI score above 14) was present in 5.8% and 22.3% of the EM and CM patients, respectively. Poor sleep quality (PSQI global score above 5) was present in 21.4% and 64% of EM and CM patients, respectively. Migraine disability and insomnia severity showed a positive correlation in both the groups. Sleep disturbances can trigger headaches and vice versa, especially when migraine is chronic. But a proportion of patients have sleep disturbances that are independent of their headache. Migraine disability and poor sleep quality are positively related. Sleep disturbances may be considered a concordant comorbidity in migraine patients.

Circadian rhythm of activity and ambient light in association with sleep variables of asthma patients

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In the background of early morning exacerbation in asthma patients with sleep–wakefulness disturbance, it is worthy to study the circadian (24 h) rhythm in activity (a rhythm marker) and light exposure (a major time cue) in asthma patients along with their sleep-activity profile. The aim is to assess the circadian rhythm in activity and ambient light along with sleep variables in asthma patients and control. The rest activity and ambient light exposure rhythm were analyzed for circadian rhythm using the cosinor method and sleep variables were assessed for 5–7 days using the actigraphy technique in 50 asthma patients and 45 control male subjects of 20–50 years. Significant circadian rhythm in rest activity and ambient light exposure in both the groups with afternoon acrophase was observed. Also significantly higher light exposure from evening to midnight in control as compared to patients was evident. Significantly higher assumed sleep, with higher sleep latency and total sleep activity score in patients along with more sleep fragmentation, low 24-h rest (%) and central phase measure (CPM) was also observed in patients. The young and middle-aged asthma patients, though can maintain more or less similar circadian rhythm in the background of their societal responsibilities, appear to show definite symptoms of disturbed state of sleep profile due to their underlying disease with a major potential to develop rhythm di-synchronization. Decreased activity and ambient light exposure from evening to night in the patients group may indicate higher fatigability in them.

Stratifying OSA patients with elevated cardiovascular risk using the AI-powered Belun® Ring through comorbid insomnia symptom phenotyping in a monodisciplinary setting

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Co-morbid insomnia and obstructive sleep apnea (COMISA) present a significant healthcare challenge due to their association with worse cardiovascular outcomes than either condition alone. AI-powered Belun® Ring (BR) show potential in stratifying OSA severity identification and aiding COMISA management. This study evaluates the

feasibility of using BR, for OSA diagnosis and sleep stage classification, to triage OSA patients with insomnia symptoms under monodisciplinary clinical settings. 257 participants (median age: 54 years, BMI: 27.9, 40% female) from Neurology & Sleep Centre, Delhi, India used the BR for 1–3 nights. Based on BR-derived AHI4% ($\geq$ 15 events/h) and sleep efficiency ($<$ 80%), participants were categorized as Normal, OSA (AHI $\geq$ 15), COMISA (AHI $\geq$ 15 and SE $<$ 80%), or Insomnia (SE $<$ 80%). Sleep metrics were compared using the Mann–Whitney test, and co-morbidity frequencies were analyzed with the Chi-squared test. Among participants, 49% were Normal, 25% OSA, 10% COMISA, and 16% Insomnia. COMISA and Insomnia groups had significantly higher wake after sleep onset (WASO) than OSA ($p < 0.001$). The Insomnia group exhibited longer sleep-onset latency (SOL) compared to OSA and COMISA ($p < 0.001$, $p < 0.01$) and the lowest SDNN ($p < 0.01$, $p < 0.05$). Among 155 patients with co-morbidities, COMISA had the highest rates of co-morbidities ($p < 0.01$) among OSA-alone ($p < 0.05$) and Insomnia-alone ($p < 0.05$) (diabetes: 41%, 21%, 26%; hypertension: 53%, 42%, 42%; $\geq$ 2 comorbidities: 35% vs 12% vs 26%) compared to normal, respectively (diabetes: 15%; hypertension: 32%, $\geq$ 2 comorbidities: 12%). The BR provides a reliable, scalable tool to precisely phenotype OSA.

Deep learning for automated detection of sleep-disordered breathing using SpO_2 data in ischemic stroke patients

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Sleep-disordered breathing (SDB), including obstructive sleep apnea (OSA), is prevalent among ischemic stroke patients and negatively impacts recovery and cognitive outcomes. Polysomnography, the gold-standard diagnostic tool, is resource-intensive and impractical for widespread use, particularly in low-resource settings. This study explores the potential of deep learning models trained on oxygen saturation (SpO_2) signals to provide scalable and accessible SDB detection in stroke patients. A deep learning framework combining convolutional and recurrent neural network architectures was developed to analyze SpO_2 fluctuations indicative of SDB. The single-channel approach simplifies diagnostics while addressing challenges such as fragmented sleep and comorbidities in stroke patients. Attention mechanisms were incorporated to align predictions with known physiological patterns, enhancing interpretability and clinical relevance. Preliminary results indicate that SpO_2-based models effectively detect SDB events, offering a promising alternative to traditional diagnostics. This approach improves accessibility and scalability, particularly in settings with limited resources. The model's interpretability supports clinical integration, enabling earlier detection and management of SDB in ischemic stroke patients. This scalable solution has the potential to enhance health outcomes and recovery trajectories in this population.

Sleep microarchitecture in essential tremor and ET-Plus: a focus on spindles and delta waves

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Sleep spindles are brief bursts of neural activity (11–16 Hz) generated by thalamocortical circuits, crucial for memory consolidation and cortical reorganization. Delta waves, dominant in stage 3 NREM sleep, are vital for restorative sleep and brain recovery. Altered spindle and delta activity is associated with sleep disturbances and may indicate neurodegeneration, impacting cognitive and neurological health. Essential tremor (ET), particularly ET-Plus, is thought to exhibit neurodegenerative features. This study examines sleep spindle and delta wave abnormalities in ET and ET-Plus as potential biomarkers of degeneration. To compare sleep spindle and delta wave characteristics among individuals with ET, ET-Plus, and healthy controls. A cross-sectional observational study at NIMHANS, Bengaluru, included 45 patients (26 ET, 19 ET-Plus) and 45 healthy controls. Diagnoses followed IPMDS criteria. Sleep spindle (density, frequency, amplitude, duration, and scoherence) and delta characteristics during stage 3 NREM sleep were analyzed using overnight PSG per AASM 2022 guidelines. Statistical analysis was conducted using R Studio. ET and ET-Plus groups showed significantly reduced spindle density, frequency, coherence, and amplitude, with increased duration, compared to controls ($p < 0.001$). Abnormalities were more pronounced in ET-Plus ($p < 0.001$). Delta frequency was reduced in both the groups ($p < 0.001$), with lower amplitude in ET-Plus ($p = 0.041$). Marked sleep spindle and delta wave patterns in ET and ET-Plus, particularly ET-Plus, suggest impaired sleep quality and support their role as potential biomarkers for neurodegeneration.

Magnitude and evolution of sleep apnea and arousals in patients with ischemic stroke

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Sleep-disordered breathing (SDB) in ischemic stroke patients is linked to poorer neurological outcomes and increased risk of stroke recurrence. Study objective: to evaluate the severity, type, and evolution of SDB and arousals in ischemic stroke patients. Ischemic stroke patients were recruited from in-patient and out-patient neurology services to evaluate sleep-disordered breathing. Patients underwent overnight polysomnography twice initially within 1 month of stroke onset and again after 3 months. Clinical outcome in the form of telephonic mRS and recurrence of vascular events or death were evaluated at 6, 18, and 24 months. Out of 141 screened patients with acute ischemic stroke, 111 were enrolled, and 105 underwent technically adequate polysomnography. The mean age was 50.5 years (SD: 12.30), with 78.4% male and a mean BMI of 26 (SD: 5.4). 81.9% had an apnea–hypopnea index (AHI) $\geq$ 5, with 12.5% exhibiting an AHI $>$ 30. The median arousal index (ArI) was 15.4 (IQR: 0.5–50.5), increasing with apnea severity. At follow-up, 56.8% had AHI $\geq$ 5, including 12.5% with severe apnea. The median ArI decreased from 15.4 (IQR: 0.5–50.5) to 11.54 (IQR: 0.5–21.1).

Changes in AHI severity categories were seen (mild: 8 vs. 15; moderate: 17 vs. 11; severe: 19 vs. 7). In those with AHI > 30, ArI declined from 24.63 (IQR: 0.7–50.05) to 10.54 (IQR: 7.1–15.07). The study identified a significant prevalence of sleep-disordered breathing (SDB) in acute ischemic stroke patients, with improvements during follow-up.

Prevalence of sleep paralysis (SP) among the medical and paramedical students of Government Medical College (GMC) Srinagar: a cross-sectional study

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Sleep paralysis is the phenomenon in which resumption of consciousness occurs, while muscle atonia of rapid eye movement (REM) sleep is maintained, leaving person paralyzed. The experience can last for few seconds to several minutes. This phenomenon is termed as SP when it occurs in person without a diagnosis of narcolepsy. A cross-sectional study sample size was calculated taking a prevalence of 25% (n = 300) and the questionnaire was made using data from the unusual sleep experiences questionnaire(USEQ) and distributed among students of GMC Srinagar and using an online Google form(consent form included) in snowball technique and then the statistical analysis was done. 352 responses were received with 124 males (M) and 228 females (F), out of which 176 students (50%) reported having episodes of SP. In females, prevalence is 50.8%, while in males, it is 48%. Maximum time noted for SP is 15 min. Majority of individual 45% reported SP at the time of sleep. 11% have only single episode during their life, while 38% have greater than 10 episodes. The study was conducted in the age group of 15–40 years and majority of the individuals have first episode during adolescence. The study reveals that the half of the medical students have suffered from SP and females are more prevalent than males. In conclusion, this study highlight the need of early diagnosis of SP and to improve the sleep cycle and stress management among the medical students to reduce the risk of SP, therefore, improving the patient care.

Effect of serum from obstructive sleep apnea patients on human coronary artery endothelial cell viability

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Obstructive sleep apnea (OSA) is characterized by repetitive episodes of complete (apnea) or partial (hypopnea) upper airway obstruction occurring during sleep. Intermittent hypoxia, a hallmark of OSA, triggers oxidative stress and inflammatory pathways, potentially impacting endothelial cell viability. The cell viability can be assessed using the MTT assay, which provides a quantitative measure of mitochondrial activity, reflecting the metabolic health of cells. The aim is to assess the impact of serum from OSA patients on the viability of human primary coronary artery endothelial cells. Participants underwent overnight polysomnography, and OSA was diagnosed based on AHI, RDI, and clinical symptoms. They were grouped into: OSA with diabetes, OSA without diabetes, and controls. In the morning, blood samples were collected, and serum was separated. The human primary coronary artery endothelial cells were cultured to reach 80% confluency. The cell viability was assessed using the MTT assay. The cells were plated in a 96-well plate and were allowed to adhere overnight. They were exposed to varying concentrations of the serum (2%, 5%, 10%, and 15%) for different durations (2 h, 4 h, 6 h, and 12 h). The MTT solution was added, followed by SDS-HCl solution to dissolve crystals. The absorbance was measured at 570 nm by spectrophotometry. Serum from OSA patients significantly impacted cell viability: at 2 h (15%), absorbance was 0.728; at 4 h (15%), 1.577; at 6 h (10%), 1.238; and at 12 h (10%), 0.802. Serum from OSA patients affects the viability of human primary coronary artery endothelial cells, highlighting the OSA-related vascular dysfunction.

Leveraging circadian processes to mitigate phenotypes associated with accelerated ageing in a fly model of Huntington's disease

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Disruption in rhythmic phenomena such as sleep, and other circadian clock-regulated behaviours and physiological processes are often associated with many neurodegenerative conditions. On the other hand, lifestyle-induced abnormal sleep patterns are known to contribute to chronic conditions including neurodegenerative phenotypes. We use the highly genetical and behavioural tractable fruit fly *Drosophila melanogaster* model to dissect out the likely bidirectional relationship between accelerated ageing phenotypes and sleep disruption. Specifically, we create a model of the autosomal dominant human condition, Huntington's disease in the fly. We examine whether improvement in circadian processes may be able to mitigate some of the toxic phenotypes induced by the expression of the mutant Huntingtin protein. I will present the results of ongoing studies in our laboratory which suggest that providing strong diurnal cues can slow down the progression of neuronal dysfunction.

Proposing the potential of machine learning algorithms in predicting sleep–wake disorders related to the circadian rhythm, a promising avenue in the field of sleep medicine

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Circadian rhythm sleep–waking disorders (CRSWDs) are caused by a persistent mismatch between an individual's unique sleep patterns and their body's natural sleep cycles. They impact global health. High-throughput machine learning algorithms are essential to identify correlations between CRSWDs and behavioral, genetic, and environmental factors. The convergence of machine learning and sleep medicine in the modern era of sleep-omics has made it possible to analyze enormous amounts of big data to predict sleep disorders. However, we are still in the early phases of implementing algorithm-based predictions for CRSWD. This extensive study will explore the use of cutting-edge machine learning algorithms to forecast CRSWDs by delving into the field of pertinent literature. This investigation involves reviewing data from relevant questionnaires such as the

Pittsburgh Sleep Quality Index, polysomnograms, sleep logs, ambulatory assessments like actigraphy, and genomic research on clock genes and circadian phase indicators. This critical literature review reveals the need for more reliable prediction models for people with chronic sleep disorders (CRSWDs) to improve sleep health and overall well-being. Utilizing advanced technologies can reduce strain and ensure CRSWD predictions are accepted, potentially revolutionizing sleep medicine.

Differential neuronal responses of diurnal and nocturnal rodents to chronic altered light–dark cycles

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Disruption of the circadian system due to prolonged altered light–dark (LD) cycles exerts profound effects on physiological and behavioral functions. This study explores how diurnal (squirrel) and nocturnal (mouse) mammals respond differently to constant light (LL), constant darkness (DD), and varying photoperiods, with a particular focus on mood-associated endocrine signaling, inflammatory markers, antioxidant defenses, and hippocampal neuronal plasticity. Constant light exposure consistently induced depressive-like behavior, elevated corticosterone levels, and proinflammatory cytokine (TNF-α) expression in both species, while reducing melatonin, antioxidant enzymes (SOD, CAT), and compromising hippocampal neuronal structure. In contrast, the response to constant darkness revealed a divergence: squirrels displayed increased corticosterone, diminished antioxidant capacity, and reduced sucrose preference, whereas nocturnal mice showed elevated melatonin and antioxidant activity, highlighting species-specific mechanisms of circadian resilience and vulnerability. In addition, altered photoperiods (8:16 h short and 16:8 h long) in diurnal squirrels caused mood disturbances and impaired hippocampal dendritic complexity and spine density. This was accompanied by decreased neurotrophic growth factors (BDNF, NGF), along with heightened oxidative stress and inflammation. These findings underscore the distinct neuroendocrine and neuroplastic adaptations of diurnal and nocturnal species to disrupted LD cycles, emphasizing the critical influence of environmental light exposure on mood regulation, cognitive function, and hippocampal integrity. The work highlights the intricate role of circadian rhythms in maintaining brain health and its disruption as a potential driver of neurobehavioral dysfunction.

Ameliorating effects of NF-κB inhibitor on circadian dysfunction in SCN and microglia in rotenone-induced Parkinson's disease (RIPD) male Wistar rats

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The suprachiasmatic nucleus (SCN) in the hypothalamus serves as the central pacemaker in mammals, synchronizing peripheral clocks via core clock genes. Circadian rhythm disruptions can impair metabolism and cellular functions, contributing to age-related neurodegeneration such as Parkinson's disease (PD). PD involves degeneration of the dopaminergic neurons in the substantia nigra pars compacta (SNpc). Nuclear factor kappa-light-chain-enhancer of activated B cells (NF-κB) is a family of nuclear transcription factors that regulate immunity and inflammation by controlling pro-inflammatory gene expression. Microglia, resident macrophages of the central nervous system (CNS), are regulated by SCN. NF-κB plays a key role in inflammasome regulation and nitric oxide synthase production in microglia. Circadian clock components influence NF-κB activity. Pyrrolidinedithiocarbamate (PDTC) is a potent inhibitor of NF-κB that blocks inhibitor of NF-κB (IκB) phosphorylation, preventing NF-κB nuclear entry and reducing cytokine expression. PDTC has shown therapeutic potential by inhibiting inflammation and oxidative stress in various diseases. Therefore, the present study aims to investigate the effect of PDTC on expression of clock, PD associated and immune genes in the central clock/SCN and extra-SCN clock such as microglia in rotenone-induced Parkinson's disease male Wistar rat model (RIPD). Partial and significant restoration in mean 24-h levels of few of the clock genes studied was observed in SCN in RIPD + PDTC group. PDTC resulted in significant restoration in mean 24-h levels of rPark2 in SCN. Significant reduction in mean 24-h levels of rCox2 and rIl1β were observed in SCN and microglia with PDTC treatment.

The influence of feeding states on executive control and response inhibition in morning and evening chronotypes

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Chronotype impacts cognitive functions such as decision-making and response inhibition. Morning and evening chronotypes have distinct cognitive profiles, particularly sensitive to feeding states. The study evaluates neurocognitive performance using Montreal Cognitive Assessment (MOCA) Questionnaire and event-related potentials (ERP) in fasting and fed states, of morning and evening chronotypes. Healthy volunteers (ages 19–28) classified as morning (n = 32) and evening (n = 28) types by MEQ score were studied over two days (07:00 and 10:00) after an overnight fast and post-breakfast. Neurocognitive assessment included the MOCA, P300 visual ERP with Go–NoGo test at Fz, Cz, and Pz cortical sites, with recorded omission, commission errors, and response times. Of 360 volunteers screened, most were intermediate (70.3%), followed by morning (20.3%), and evening (9.4%). types. Post-meal, both chronotypes showed significant hyperglycemia and increased MoCA scores. Significant NoGo anteriorization improved P300 amplitude, faster response times, and fewer commission errors indicating enhanced attention, impulse control, and decision-making in the fed state in both the groups. These effects were stronger in evening types, who matched morning type performance during the morning session, despite it not being their preferred time. In evening types, lower latency in the fed state suggests better cognitive processing unlike in morning types, which indicates short-term metabolic status may have less impact on cognition at preferred circadian times. Evening chronotypes excelled on MoCA, response inhibition, and attention control post-meal, indicating improved cognitive resource allocation. Thus, evening types might benefit from scheduling demanding tasks after meals for optimal performance.

Effect of chronic sleep deprivation on rhythms of SD marker genes with aging: therapeutic effects of melatonin

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Sleep deprivation (SD) disrupts both sleep homeostasis and circadian regulation, particularly with aging which makes the bidirectional relationship between sleep–wake cycles and circadian rhythms increasingly vulnerable to desynchronisation. The molecular mechanisms underlying disrupted sleep homeostasis and circadian misalignment remain poorly understood. This study investigated the rhythmic expression patterns of key sleep-regulatory genes in different brain regions with ageing in Wistar rats and evaluated melatonin's therapeutic potential in mitigating these effects. We examined the hypothalamic prepro-orexin (Hcrt) gene, crucial for sleep–wake regulation, alongside activity-dependent genes (Arc, cFos, and Fra-2) in the cerebral cortex, which serve as reliable markers of neuronal activation and plasticity. SD was induced by gentle handling method. We analysed these various gene expression rhythms in variable age groups such as 3-, 12-, and 24-month-old rats upon chronic SD. We demonstrated significant phase shifts and altered transcript levels across all studied genes during SD, indicating disrupted circadian regulation upon SD. Further as melatonin is sleep synchroniser, we studied effects of melatonin administration on these genes and demonstrated the therapeutic efficacy by restoring rhythmicity. These findings provide novel insights into age-related changes in sleep-regulatory gene expression and highlight melatonin's potential as a therapeutic intervention for sleep disorders across different age groups.

Early time-restricted eating enhances sleep duration after transmeridian travel: a preliminary study

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Jet lag, characterized by sleep disruption and impaired performance due to circadian desynchrony, poses significant challenges for travelers. This pilot study investigates the impact of early time-restricted eating (eTRE) on sleep recovery following rapid transmeridian travel. Six participants traveling between the United States and China were enrolled, with four in the eTRE intervention group and two in the control group. One participant was excluded due to illness during the study. The eTRE protocol involved a daily fasting period of $\geq$ 16 h, followed by a meal in the morning of the destination time zone. Total sleep time (TST) was monitored using wrist-worn actigraphy and sleep diaries during the week post-travel. Participants in the eTRE group exhibited a significant increase in TST, averaging an improvement of 1.1 h per night. In contrast, control group participants experienced a mean decrease of 1.205 h per night. Overall, the eTRE group achieved 2.305 h more TST across the recovery week compared to the controls. These preliminary findings suggest that eTRE may accelerate circadian adaptation and improve sleep outcomes during jet lag recovery. Further research with larger sample sizes is warranted to confirm these results and explore the potential of leveraging food entrainable oscillators to improve human entrainment. eTRE represents a promising, non-invasive intervention for mitigating the negative effects of circadian desynchrony, with potential applications for business travelers and other sleep disorders.

Enhanced immobility state in *Drosophila melanogaster* under the presence of predators

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Sleep is generally considered a disadvantage for survival due to immobility and reduced responsiveness to external stimuli. Nonetheless, animals require sleep for its critical functions in the maintenance of central nervous system. On the other hand, recent research has revealed that sleep-like immobility state is conserved even in species lacking a central brain, such as hydra, suggesting physiological significance for sleep beyond advanced brain functions. We hypothesize that a state of quiescence is an adaptive behavior for small organisms during periods not dedicated to activities such as feeding and reproduction, and that immobility itself constitutes a part of sleep's functions. This study aims to elucidate the role of immobility in *Drosophila melanogaster* using the jumping spider, a natural predator of fruit fly. Flies exposed to the spider showed increased immobility time which is considered as sleep during daytime. In addition, short-sleep mutant, fmn showed increased immobility time during both the daytime and nighttime in the presence of predators. Furthermore, we found that a similar phenotype was induced by spider cue, probably spider odor. These data suggest that flies do not simply respond to spider movement but recognize predator's presence and increase their immobility time. We are investigating the neural circuits and genes involved in this phenotype. In addition, to explore the impact of the active/immobile states of flies on their susceptibility to predation, we examined the behavioral changes of the spiders which was presented with flies whose mobility was artificially regulated by optogenetics.

Leptin and ghrelin levels in short and long sleepers in obese students

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Short sleep duration is associated with obesity. Sleep duration plays a crucial role in regulating appetite and metabolic processes, with leptin and ghrelin being key hormones involved in these mechanisms. This study examines the levels of leptin and ghrelin in individuals categorized as short sleepers (9 h of sleep per night). Methods: data from 100 obese subjects aged between 18 and 25 years (mean $\pm$ SD 19.63 $\pm$ 1.68) and BMI was 33.84 $\pm$ 4.28 kg/m^2 was analyzed and divided into two groups consisting of short sleepers (sleep duration 9 h/day). Sleep duration was studied using Pittsburgh Sleep Quality Index questionnaire (PSQI). Blood samples were collected for estimation of serum leptin and ghrelin levels using enzyme-linked immunosorbent assay techniques. Results: out of 100 obese, 75% were short sleepers, 10% were long sleepers and 15% were adequate sleepers. The results indicated that short sleepers in obese had leptin levels of 15.27 $\pm$ 2.16 ng/mL and ghrelin levels was 2.79 $\pm$ 1.45 ng/mL. Long sleepers showed leptin levels of 14.94 $\pm$ 1.76 ng/mL and ghrelin levels of 3.92 $\pm$ 2.27 ng/mL. Normal sleepers showed leptin

levels of 14.82 ± 2.03 ng/mL and ghrelin levels of 3.21 ± 2.24 ng/mL. One-way ANOVA indicates that there is no statistically significant difference in leptin levels among the three groups (short, long, and adequate sleepers). Ghrelin level was increased in long sleepers when compared to normal sleepers but not was not statistically significant.

Glycaemic control in diabetics with obstructive sleep apnea in relation to two predominant stages of sleep

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Increasing severity of obstructive sleep apnea (OSA) is known to be associated with poor control of blood glucose levels in type II diabetes mellitus. Raised AHI in REM sleep as seen in mild-to-moderate cases of OSA may also have an impact on glycemic control in type II diabetes. We aimed to find the effect of REM-predominant OSA and NREM-predominant OSA on HbA1c in subjects with type II diabetes. All participants underwent polysomnography, and glycemic control was assessed by HbA1c. Thirty diabetic participants underwent PSG, four of whom had REM -predominant OSA, nineteen had NREM-predominant OSA and seven had normal AHI level. The mean HbA1c in REM predominant OSA group was 5.8% and that in NREM predominant OSA was 7.3%. REM predominant OSA was found in mild and moderate OSA. Nadir oxygen saturation was 82.5% and 71.9% in REM-related OSA and NREM-related OSA, respectively. The present study reveals that both REM predominant OSA and NREM predominant OSA are significantly associated with type II diabetes mellitus. The significantly higher AHI in REM stage of sleep calls for adequate CPAP usage covering the whole sleep time rather than typical use for 3 to 4 h and to treat judiciously even the milder cases of OSA to achieve the metabolic effects of CPAP therapy.

Acute sleep deprivation exacerbates microglial and astroglia reactivity by increasing the oxidative stress in the hypothalamus of rats

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Sleep deprivation (SD) is known to modulate inflammatory and oxidative stress markers. Therefore, in the present study, we examined the effects of 24-h acute sleep deprivation on dendritic arborization, glial architecture of the hypothalamus. The study was conducted as per the guidelines of the Institutional Animal Ethics Committee. Total 18 rats were randomly divided into three groups: Control, 24-h sleep deprivation (SD), 24-h recovery sleep (Rec). After 24-h SD using modified multiple platform method, animals were killed and tissues were collected and processed either for GFAP, IBA1, PSD-95, mt-COX, Golgi-Cox staining and oxidative stressors (SOD, CAT, MDA and GSH) measurement using ELISA. We have observed reduction in the REM sleep on 24-h acute sleep deprivation. Immunofluorescence imaging showed increase in number and intensity of microglial and astrocyte markers in sleep deprived as compared to other counterparts. Sholl analysis exhibited a significant increase in the number of intersections, bifurcations and length of dendrites after 24-h SD as compared to control and recovery sleep. Further, mt-COX and PSD-95 positive cell density was found to be increased in SD group as compared to control and 24-h recovery group. In addition, oxidative and antioxidative markers were found to be downregulated in recovery sleep as compared to sleep deprivation. Present study suggests that sleep debt can alter dendritic plasticity through remodelling energy metabolism.

Multivariate EEG pattern during dreaming shows higher non-linear feature contribution and inter-individual variability in a high-density PSG data

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The study of sleep and dreams relies on subjective reports, as direct observation is often unreliable, and challenging to control by exposure to external stimuli. To gain empirical evidence on dream phenomena, the use of brain waves to decode dreams has become popular. Previous studies have linked transient phasic activities—such as eye movements, PGO waves, and EEG power changes—with dreaming, identifying markers like local decreases in low-frequency activity in posterior cortical regions. However, the complex, nonlinear dynamics of EEG activity and the global nature of sleep suggest that a univariate and region-specific approach may be inadequate for identifying EEG markers of dreaming. In this work, we focus on multivariate characteristics derived from EEG data across scalp regions to identify potential indicators for different dream situations. We extracted features (IRASA, FOOOF, non-linear features, and PSD) from epochs of whole night PSG recordings, employing a serial awakening paradigm to capture dream experiences and trained on a random forest algorithm to classify between dream and no dream. Our findings show that the multivariate approach significantly enhances classification accuracy between dreaming and non-dreaming states, with nonlinear features contributing more than conventional spectral features. Notably, the patterns observed are spread across different scalp regions rather than confined to isolated areas. In addition, we found that dream markers can be individualistic, suggesting that emotional and contextual characteristics of dreams may lead to more effective markers in future research.

Development of a novel tool for detection of daytime sleepiness

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Daytime sleepiness, a common issue in individuals with disrupted sleep cycles or disorders, has been identified as a major contributor to functional impairments and accidents. Traditional methods, such as the Multiple Sleep Latency Test are effective but require extensive time and resources. The Karolinska Sleepiness Scale (KSS) is an established subjective measure for assessing sleepiness, yet it may be susceptible to self-assessment biases. Although EEG offers an objective alternative, its use as a sleepiness measure remains under-validated. This study aims to explore correlations between KSS scores and daytime EEG to validate EEG as a measure for assessing sleepiness in healthy individuals. Methods: this is a cross-sectional observational study in which 50 healthy participants (age 18–30 years) have a regular sleep pattern and no history of sleep disorders, chronic diseases, substance use, or recent time zone shifts were included. Daytime sleepiness was evaluated using the 9-point KSS questionnaire. Objective EEG measurements included the Karolinska Drowsiness Test (KDT) and Alpha Attenuation Test (AAT). The EEG data were extracted and subjected to FFT to decompose it into delta to gamma frequency bands via SOMNO software. After removing the outliers, the frequency bands were correlated with KSS and RT using GRAPHPAD with significance set at a p-value of < 0.05. Results in the eyes open (EO) condition and CZGamma demonstrated a significant negative correlation with KSS, $r(48) = -0.30$, $p = 0.036$. Conclusion: this study demonstrated CZ Alpha (eyes open) and C4 Gamma (eyes closed), as effective tools for assessing daytime sleepiness.

Comparative efficacy of yoga therapy and CBT-I for chronic insomnia: insights from subjective measures

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Chronic insomnia is a prevalent sleep disorder associated with significant health and functional impairments. Cognitive behavioural therapy for insomnia (CBT-I) is the gold-standard treatment, while yoga therapy is emerging as a promising alternative. This study aims to compare the efficacy of these interventions using subjective measures of sleep, associated psychological outcomes, and medication use. Methods: a total of 106 participants diagnosed with chronic insomnia were recruited, with 88 completing the intervention (yoga group: $n = 45$; CBT-I group: $n = 43$). Participants were randomized to receive either yoga therapy or CBT-I along with Lorazepam, and data were collected at baseline and week 16. Subjective assessments included the Insomnia Severity Index (ISI), Pittsburgh Sleep Quality Index (PSQI), Generalized Anxiety Disorder-7 (GAD-7), Patient Health Questionnaire-9 (PHQ-9), Dysfunctional Beliefs and Attitudes about Sleep Scale (DBAS), Hyperarousal Scale (H Scale), and daily sleep diaries. Objective measures included polysomnography (PSG) and heart rate variability (HRV). Weekly cumulative doses of Lorazepam were recorded to evaluate changes in hypnotic medication use. Baseline characteristics revealed that the yoga group had a mean age of 42.04 years (SD = 9.25) and a mean BMI of 24.98 (SD = 3.16), while the CBT-I group had a mean age of 43.51 years (SD = 13.57) and a mean BMI of 25.16 (SD = 3.56). Both groups were comparable in terms of education, sex, and socioeconomic status. Results: both Yoga and CBT-I groups showed significant improvements across all measures (SOL, WASO, PSQI, ISI, GAD, PHQ, DBAS, and H-Scale) from baseline to week.

An unconventional way of scoring sleep–wake cycle in rats

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Twelve-hour sleep data from 6 rats were manually scored by the conventional rule of designating a single 30-s epoch as one sleep stage if 50% or more of the epoch fell in that sleep stage and designating each sleep–wake stage separately within that 30-s epoch. The total sleep time (TST) and total wake time (TWT) were (5.1 ± 0.5) h and (6.9 ± 2.1) h for the conventional method of scoring, while the TST and TWT were (4.8 ± 1.2) h and (7.4 ± 1.4) h when scored as denoting exact time spent in each SW stage. The comparison of both the scoring methods was done using paired t-tests. It was found that there was no significant difference between the percentage of TST (57.2 ± 7.8) % vs. (61.7 ± 4.8) %) and TWT (42.8 ± 19.8) % vs. (39.8 ± 5.8) %) in both the scoring methods. However, when microanalysis of the individual sleep–wake stage was done, it was seen that the exact time in the percentage spent in REM sleep (R, 10.5 ± 1.9 vs. 0.7 ± 0.3) and quiet wake (QW, 14.3 ± 2.2 vs 0.5 ± 0.2) was significantly higher ($p < 0.05$) when compared to the scoring done by conventional designation of 50% and > 50% of the 30-s epoch as a single sleep stage. Active wake and slow wave sleep scoring in both scoring methods showed no significant change. This unique way of re-analyzing the sleep data and comparing the two scoring methods gives us insight into the fact that some of the actual sleep stages are masked when following the conventional scoring method, more so because the sleep stages in rodents are polyphasic.

Somatosensory information processing in patients of chronic insomnia

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Sleep plays a pivotal role in the central nervous system's ability to process and integrate sensory information. Disruptions in sleep architecture as seen in insomnia can impair nociceptive processing and alter sensory perception, thereby influencing pain modulation. Quantitative sensory testing (QST) provides a standardized methodology for assessing sensory processing and pain phenotyping by measuring thresholds for detecting and tolerating thermal stimuli, which are often dysregulated in insomnia. Method: this observational study includes 6 patients diagnosed with chronic insomnia and 6 age- and sex-matched healthy controls. The parameters evaluated using QST were warm detection threshold, cold detection threshold, hot pain threshold, cold pain threshold, hot pain tolerance threshold and cold pain tolerance threshold (°C). Results: patients with insomnia demonstrated significantly higher warm detection thresholds

($p = 0.0008$) and cold detection thresholds ($p = 0.02$) compared to controls. The mean hot pain threshold in insomnia patients was 43.73 °C, significantly higher than 38.54 °C in controls ($p = 0.0012$). Similarly, cold pain thresholds were elevated in insomnia patients ($p = 0.002$). Cold pain tolerance thresholds were markedly higher in the insomnia cohort ($p = 0.0001$). However, no statistically significant difference was observed in hot pain tolerance thresholds between the two groups. Conclusion: the findings indicate attenuated sensory responsiveness in individuals with insomnia. Chronic insomnia is associated with dysregulation of the central pain modulation systems, particularly within the descending inhibitory pathways originating in the brainstem and modulated by the periaqueductal gray (PAG) and rostroventromedial medulla (RVM). Insomnia may impair the release of endogenous opioids reducing the efficiency of descending pain inhibition.

REM sleep restriction during late pregnancy disrupts the optimal development of sympatho-vagal balance in developing rat pups

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At the time of birth, brain networks for sleep and autonomic nervous system (ANS) are immature, thus making them more vulnerable to any sleep loss. This study is aimed to evaluate changes in the developmental pattern of cardiac ANS of rat pups born to REM sleep restricted (REMSR) rat dams during the last trimester of pregnancy taking heart rate variability (HRV) measures. After confirmation of pregnancy, Wistar rats were REMSR using platform method from gestation day 15–20 for 20 h/day. The pups born to control and REMSR groups were acutely implanted for ECG electrodes for HRV and EMG, and were recorded in a customized thermostat chamber. Assessment of Active sleep (AS), Quiet sleep (QS) and wakefulness (W) were done on four different post-natal days (D1, D5, D10 and D15). HRV analysis was carried out using the software Kubios scientific taking low-frequency cutoff as 0.25–0.8 Hz and high-frequency cutoff as 0.8–2.5 Hz in 5-s epochs on D1 and D5, and 30-s epochs on D10 and D15. The results showed significant increase in HR in the REMSR group compared to the control group in all post-natal days. Moreover, in frequency domain, the higher HF power during AS compared to QS and lower LF power in the REMSR pups during D1-10 suggested impaired development of vagal tone during QS. These findings indicate that prenatal REM sleep loss delays and dysregulates sleep state-dependent cardiac autonomic balance in the developing offspring that potentially contribute to the development of depression behaviour.

Study of sleep–wakefulness associated changes in heart rate variability in adult female rat to model the sleep-cardiac autonomic functional dynamics

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Heart rate variability (HRV) is a widely used non-invasive technique to assess the sympatho-vagal balance during the awake resting state. However, studies on the steady-state HRV changes during sleep stages when the complex interplay of two branches of cardiac autonomic nervous system takes place are not common. This study was aimed to model HRV dynamics during sleep–wake (S-W) stages in control adult female Wistar rat. To record S-W, EEG and EMG electrodes and recording heart rate, ECG electrodes were chronically implanted under anaesthesia. After post-operative recovery, these signals were acquired continuously for 72 h. Offline HRV analysis during W, NREM and REM sleep stages were carried out taking 30 s epoch using kubios software. Statistical analysis of the time and frequency domain HRV parameters among S-W states were done using student's t-test and also for day vs night. In the time domain comparisons, RR interval was increased during NREM sleep compared to W/REM sleep, while HF power was increased during NREM sleep compared to wake state. There was an increase in LF power during NREM compared to W/REM sleep. In this nocturnal species, HR during W was higher during night compared to daytime, while for NREM sleep, it was lowest during the daytime, indicating a high nighttime sympathetic index. This study provided insights into the changing HRV dynamics during S-W states in a rodent model, which can be used to understand the relationship between sleep and autonomic regulation for emergence of neurodevelopmental disorders.

Prevalence of excessive daytime sleepiness and self-reported sleepiness on the wheel among drivers victims of road traffic accidents

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Excessive daytime sleepiness (EDS) often resulting from sleep deprivation and sleep disorders is a recognized factor for road traffic accident (RTA). However, these factors are mostly overlooked when investigating the causes of RTAs. This study aims to assess the proportion of EDS and self-reported sleepiness on the wheel (SAW) in drivers victims of RTA. It also aims to explore the role of factors such as demographic, chronotype, functional outcome of sleep, substance use, road and environmental factors. A cross-sectional study conducted among 383 driver victims of RTA presented to the emergency department. Participants' demographic, medical and driving experiences were collected, along with assessments of EDS, SAW, chronotype and substance use. Statistical analysis included Chi-square, Mann–Whitney U and Kruskal–Wallis H test with multivariate logistic regression to identify predictors of RTA-SAW. EDS and SAW was reported by 21.7% and 20.6% of the participants, respectively, but no association was found between them. Comparison between participants with and without RTA-EDS and RTA-SAW showed no significant differences in vehicular, road-related, and driving durations nor in countermeasures of sleepiness, substance use or injury severity. Lastly, there was also no association between EDS or SAW and chronotype, injury severity and the functional outcome of sleepiness. SAW and EDS appear to be distinct constructs, with SAW being a more reliable predictor of accident risk under specific situational and environmental conditions. This suggests the need for targeted strategies for SAW detection and management among drivers, including educational programs and sleep monitoring interventions.

Inspiratory muscle training for respiratory muscle strength and pulmonary function in female breast cancer patients: a systematic review

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Background: Breast cancer, the most prevalent cancer among women globally, often leads to weakened respiratory muscles and impaired pulmonary function due to treatments such as chemotherapy, radiotherapy, and surgery. These affects the inspiratory muscles, lung mechanics and increase respiratory effort during physical activity. Research indicates that IMT, especially when combined with aerobic exercise, enhances respiratory muscle strength and reduces dyspnea. IMT has shown potential in improving the quality of life for breast cancer survivors by addressing fatigue, stress, and post-treatment complications. This study aims to evaluate the impact of IMT on respiratory mechanics and pulmonary function in breast cancer patients, focusing on mitigating respiratory challenges associated with the disease and its treatments.
Method: The review included randomized controlled trials (RCTs) and clinical studies adhering to the PICO framework. Participants were women with stable breast cancer diagnoses who had completed adjuvant treatments and exhibited reduced inspiratory muscle strength or dyspnea. Interventions included IMT combined with aerobic or other physical exercises, with lower intensity IMT used in control groups. Primary outcomes were respiratory muscle strength and pulmonary function, while data extraction captured study characteristics, participant details, and intervention specifics. Trial quality was assessed using the 11-point PEDro scale. The findings support incorporating IMT into breast cancer rehabilitation to improve respiratory function, reduce stress, and enhance physiological performance. Further multicenter studies are needed to confirm these benefits and strengthen IMT's role in breast cancer recovery.

Can elderly individuals use peripheral arterial tonometry devices without difficulty?

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It remains unclear whether individuals aged 65 and older can use peripheral arterial tonometry (PAT) devices without difficulty. This pilot study aims to explore this question. Between December 2022 and December 2024, individuals aged 65 and over who consented to participate in the study were asked to use a PAT device. Their experiences and any issues encountered were documented through interviews. This study was approved by the Institutional Review Board. Eight participants were enrolled, with a mean age of 72.5 years; all participants were male. Most participants reported being able to use the device without major issues. Although limited, this pilot study suggests that individuals aged 65 and older may be able to use PAT devices without significant difficulty.

Sleep architecture in patients with alcohol dependence syndrome

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Regular alcohol consumption may alter the normal circadian cycle. Alcohol consumption and abuse disrupt central nervous system transmission processes, impair the activity of several biological systems, and cause major health problems. There are reports of insomnia and sleep disturbances in acute alcohol dependence which may be the cause of relapse sometimes. The present study is designed to understand the subjective complaints of sleep disturbance in alcohol-dependent patients in an objective manner using polysomnography. After obtaining Institute Ethics approval, ten alcohol addicts admitted for de-addiction were recruited for the study. After the patients are detoxified, and ensuring the effect of benzodiazepines is washed off and they do not have withdrawal symptoms. They were called to physiology lab for an initial screening and general examination; they were assessed for sleep architecture using polysomnography SOMNO HD PSG with camera compact wS (NGDV202Aws). The mean age and BMI of recruited patients were 38.3 years and 21.4. The average sleep efficiency was 77.25%, and sleep onset latency was 27.5 min. The N1, N2, N3, and REM percentages were 15.82%, 54.5%, 9.85%, and 19.8%, respectively. There is a slight decrease in sleep efficiency and sleep-onset latency and a considerable decrease in N3 sleep. Along with de-addiction, the patients can undergo cognitive behaviour therapy for insomnia to reduce the probability of relapse.

Machine learning approaches to evaluate EEG correlates of relaxation between supine and sitting postures in eyes closed condition

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To attain brain relaxation, various techniques are commonly employed while sleep remains nature's ultimate remedy. Various machine learning (ML) tools are employed to understand the neural correlates of relaxation from the EEG bands. However, majority of the previous studies focused on comparing the EEG bands during eyes-open (EO) and eyes-closed (EC) resting condition paradigm to train the datasets in ML. Moreover, several yogic techniques are performed using sitting and supine positions, and we investigated the short-term changes in EEG activities between supine and sitting postures during EC condition in healthy participant using ML classifiers. EEG was recorded using nine unipolar electrodes, including O1, OZ, O2, C3, CZ, C4, F3, FZ, and F4 for 25 min during EC supine and EC sitting postures each on five different days, along with ECG for heart rate variability (HRV) analysis. Relaxation was analysed by examining the relative power of the alpha and theta waves extracted from the EEG data and corroborated with the HRV parameters. These EEG metrics were analysed by leveraging ML classifiers (Random Forest-RF; XGBoost; K-Nearest Neighbors-KNN, and Support

Vector Machine-SVM) for relaxation states under these two conditions (sitting and supine). Statistical tests including ANOVA and t-tests were also used to compare parameters between these conditions. Our findings indicated that SVM particularly excelled in classifying relaxation states from the EEG alpha and theta band data. The study demonstrates ML techniques effectively classified relaxation states based on EEG and HRV metrics during different postures, with SVM performing particularly well in this classification task.

The effect of integrated approach of yoga therapy (IAYT) on stress and sleep quality among night shift IT professionals: a pre–post-test interventional study

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The Indian IT industry is marked by its professionals' alignment with global customer requirements, adapting to varied time zones, and dealing with short-term contracts. This often leads to job stress, poor sleep, and a host of health issues. Sleep quality is greatly affected by work stress, potentially causing a variety of physical and mental health challenges. Yoga fosters self-awareness and mindfulness and emerges as a potential solution to reduce stress and anxiety and enhance sleep quality, health, and well-being. Despite growing research, specific studies on stress and poor sleep faced by Indian IT professionals working night shifts remain limited. Therefore, this study aims to assess the effectiveness of the integrated approach of yoga therapy (IAYT) on stress and sleep quality among night-shift IT professionals. A pre- and post-interventional study was conducted among 60 IT professionals working on a night shift. Baseline assessments collected age, body mass index (BMI), and sleep quality using the Pittsburgh Sleep Quality Index (PSQI) Perceived Stress Scale (PSS). A 6-week IAYT workplace yoga intervention was conducted by a trained yoga professional, followed by posttest assessments. Results revealed that in the intervention group (YG), stress levels as measured with PSS reduced significantly (14.75%, $P < 0.05$) and sleep quality as measured with PSQI also improved significantly (20.79%, $P < 0.05$). IAYT yoga intervention appears to be a promising approach to alleviating stress and enhancing sleep quality among IT professionals working night shifts. Implementing tailored workplace yoga programs can play a crucial role in improving sleep and promoting the well-being.

Aripiprazole treatment on mouse model for circadian rhythm sleep–wake disorders

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Circadian rhythm sleep–wake disorder (CRSWD) causes social problems because the patient's sleep–wake rhythm deviates from the normal day–night rhythm. CRSWD were traditionally thought to result from a phase shift in the circadian rhythm center, located in the brainstem. On the other hand, recent studies showed that one-third or more of CRSWD patients have normal melatonin rhythms, but their sleep–wake rhythms are sifted. The pathophysiology is very similar to internal desynchronization. There is no specific treatment for the disease because the standard therapies targeting circadian rhythms are ineffective for patients with internal desynchronization. Aripiprazole (APZ) is broadly used as an antipsychotic in psychiatry and is recently started to be applied to CRSWD. However, it is unclear how APZ acts on those patients. In this study, we established a mouse model of CRSWD involving chronic administration of Methamphetamine in drinking water and attempted treatment with APZ in these mice to analyze the effectiveness and therapeutic mechanisms of APZ. As a result, we found that administration of APZ to CRSWD mice model synchronized their sleep–wake rhythm to the light–dark cycle. Moreover, we confirmed that this entrainment was accompanied by changes in sleep architecture. Our results may provide a stepping stone to understanding the pathophysiology of CRSWD with internal desynchronization and suggest that APZ target modification of sleep–wake rhythms rather than circadian rhythms.

Study of whether intrinsically photosensitive ganglion cells (ipRGCs) activity contribute to changes in human rectal temperature after morning light exposure

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The intrinsically photosensitive retinal ganglion cells (ipRGCs) play a role of non-visual function such as the regulation of circadian rhythm using light information. However, there is no clear evidence as to how differences in the amount of stimulation of ipRGCs affect in humans. The purpose of this study is to clarify whether the morning light exposure, which stimulates ipRGCs by different amounts, affects human rectal temperature. Seventeen healthy adult males (mean ± SD: 21.7 ± 2.2 yr. old) with normal color vision and intermediate-type (chronotype) participated in three-days and two-night laboratory experiments. The subjects were exposed to one of three types of light (A: 500 lx, 6,500 K, 620 mW/m^2, B: 500 lx, 23,000 K, 1090 mW/m^2, C: 880 lx, 6500 K, 1090 mW/m^2) between 0700 and 0800 on the second and third days. In all three conditions, the subjects spent time in the same light environment, except for this morning light exposure. A two-way repeated ANOVA, comparing the rectal temperatures from 1000 to 1200 on the third day of A as the control with the same time in B, demonstrated a significant interaction of time × condition ($F(4, 120) = 6.737$, $p < 0.001$). Same analysis, comparing A as control with C, demonstrated a significant interaction of time × condition ($F(4, 124) = 7.054$, $p < 0.001$). From these results, the morning light with high stimulus-level for the ipRGC, with high color-temperature and high illuminance, might increase the subsequent morning rectal temperature.

Effect of melatonin on daily rhythms of oxidative stress markers in amyloid-β induced Alzheimer disease's (AβIAD) male Wistar rat model

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Suprachiasmatic nucleus (SCN), in mammals, is considered as a master clock which consists of core clock genes that regulate the circadian rhythms. Disruption of the circadian rhythms is prevalent in the early stages of Alzheimer's disease (AD). AD is an age-related most common type of neurodegenerative disorder characterized by cognitive decline, memory loss, and accumulation of amyloid-beta plaques and tau protein tangles in the brain. Oxidative stress plays a crucial role in AD pathogenesis, contributing to neuronal damage and cognitive impairment. Alterations in daily rhythms of lipid peroxidation (LPO) and antioxidant enzymes such as superoxide dismutase (SOD), catalase (CAT), and glutathione-s-transferase (GST) have been demonstrated from our laboratory earlier in amyloid-β induced AD rat model (AβIAD). Melatonin is an antioxidant, free radical scavenger, rhythm synchronizer, and has neuroprotective properties. Therefore, we studied the effect of melatonin on the daily rhythms of LPO, GST, SOD, and CAT in the AβIAD rat model. Current studies suggest amelioratory effects of melatonin on AβIAD rat model in the daily rhythms of oxidative stress markers, potentially offering a novel therapeutic approach for AD management.

Daily rhythms of oxidative stress markers in peripheral clocks of STZ and high-fat diet-induced type 2 diabetic (T2D) male Wistar rat model

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The master clock or circadian pacemaker, suprachiasmatic nucleus (SCN) present in the hypothalamus coordinates and entrains circadian oscillations in the peripheral tissues such as the liver, kidney, lungs, and pancreas that are called peripheral clocks. These also have endogenous circadian oscillations. Circadian rhythms significantly influence several metabolic processes in type 2 diabetes (T2D). Lipid peroxidation and nitric oxide (NO) are markers of oxidative stress and cellular damage. We have observed circadian rhythmic alterations in these oxidative stress markers in peripheral clocks of streptozotocin (STZ) and high-fat diet-induced T2D male Wistar rat model. Daily rhythms of lipid peroxidation and NO levels were studied in the liver and pancreas at variable time points (Zeitgeber Time (ZT) 0, 6, 12 and 18) in male Wistar rats. Disrupted patterns of daily rhythms of lipid peroxidation and NO in peripheral tissues such as the liver and pancreas were observed in T2D condition. These findings suggest that the altered daily rhythms of lipid peroxidation and NO may contribute to the pathophysiology of T2D. Understanding these mechanisms could provide insights into potential therapeutic interventions targeting circadian rhythms to mitigate oxidative damage in T2D conditions.

Sleep pattern in coronary artery disease patients and "Dosa kalas" of "Dinacharya"

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Short sleep duration is a risk for cardiovascular events, and early morning transient myocardial ischemia was observed in coronary artery disease (CAD). Ayurveda consider CAD under "vata dosha" and early morning before 6 AM is known as "vata kala". The aim of current study is to identify "kalas" of sleep wake pattern and its association with duration of total sleep time. The study enrolled patients with coronary artery disease. We took a detailed clinical and sleep history, administered PSQI, and classified their sleep and wake times using the Ayurvedic concept of "dosa kala" from "Dinacharya." "Dosa kala" was defined as follows: Period from 6 to 10 am is known as "kapha kala," while the period from 10 am to 2 pm is known as "pitta kala," 2 pm to 6 pm is known as "vata kala," from 6 to 10 pm as "kapha kala," from 10 pm to 2 am as "pitta kala," and from 2 to 6 am as "vata kala." 182 patients (129 males, 23 females) of coronary artery disease with mean age of patients 53.11 ± 8.13 years were enrolled in study. 85.71% patients went to bed in "pitta kala", and out of them, 64% patients wake up in "vata kala". Patients waking up in "vata kala" have reported significantly decreased sleep duration in comparison to others (5.59 ± 1 vs 6.46 ± 1 h, $p < 0.001$). Early morning awakening in "vata kala" is significantly associated with short sleep duration in coronary artery disease patients.