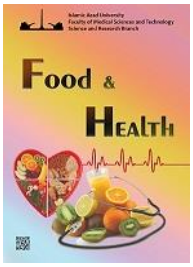

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Studies on sleep disorders in relation to health

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ABSTRACT

Sleep disorders are very prevalent in the general population and are associated with significant medical, psychological, and social disturbances. A multidimensional approach is recommended for its assessment covering sleep hygiene measures, psychotherapy, and medication. The parasomnias, including sleepwalking, night terrors, and nightmares, have benign implications in childhood but often reflect psychopathology or significant stress in adolescents and adults and organicity in the elderly. Excessive daytime sleepiness is typically the most frequent complaint and often reflects organic dysfunction. Narcolepsy and idiopathic hypersomnia are chronic brain disorders with an onset at young age, whereas sleep apnea is more common in middle age and is associated with obesity and cardiovascular problems. Therapeutic naps, medications, and supportive therapy are recommended for narcolepsy and hypersomnia; continuous positive airway pressure, weight loss, surgery, and oral devices are common treatments for sleep apnea. There is a need for identifying the association of sleep and general health so as to provide a better and safe mechanism of management of insomnia since the pharmacological therapy adds the risk of side effects and addiction problems. The data is collected based on the internet search and papers submitted in PubMed, ScienceDirect, Web of Science, and Google Scholar. Sleep assessment is vital to physical and mental health. Having difficulty in getting proper sleep could be the base of initiation of many health problems According to our study in this article we suggest the importance of sleep to be measured and evaluated by the health care system for the individuals whether in hospitals or institutes or any organization.

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1. Introduction

Sleep-related disorders are major health issues, the estimated prevalence of sleep problems in the general population range between 15 and 24%, but prevalence as high as 62% has been reported in the elderly population (1). Poor sleep quality is associated with increased tension, irritability, depression, confusion and a general low life satisfaction (2) Many factors may contribute to insomnia such as environmental factors (e.g., uncomfortable, noisy, or hot/cold surroundings; stressful life events), medical treatment, alcohol, or dietary stimulants such as caffeine. Behavioral processes may lead to conditions wherein patients convince themselves that they will not be able to sleep; such behaviors may lead to insomnia and in long-term occurrences cause severe insomnia. With the realization that sleep problems are on the rise in populations, sleep quality and various components of sleep that are affected due to environmental, psychophysiological and pharmacological factors are in focus in recent times. The fundamental question of why human being requires sleep is largely unanswered. Despite this, scientists are providing more information regarding how humans sleep. As the duration and timing of sleep are tightly regulated, it is assumed that sleep provides a number of important psychological functions (3). Insomnia, as defined by the DSM-IV-TR, is “difficulty initiating or maintaining sleep or experiencing non-restorative sleep that results in clinically significant distress or impairment in functioning”. Insomnia, therefore, is a subjective clinical diagnosis, and there is a lack of objective measurements that correlate well with the patient’s subjective symptoms. It is obvious that sleep disorders have negative consequences for health, functioning, and overall quality of life, especially in elderly people. Therefore, it will be important to improve the quality of sleep,

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which may improve the individual`s energy/vitality levels and daytime quality of life. Therefore, the present review was conceived to investigate sleep disorders and their effects on health.

2. Sleep physiology

Sleep is characterized by reduced response to stimuli; reversibility; minimal movement; species-specific stereotypic posture; and species-specific diurnal timing and duration (4). The two characteristics that clearly distinguish sleep from other non-wake states (e.g., coma and hibernation) are responsiveness and reversibility; responsiveness to endogenous and exogenous stimuli is reduced, but not absent, during sleep. The reversibility is also a ready response variation incurring due to individual characteristics. The immobility of the sleep state is relative to species, fish swim in place while sleep. A man sleeps in a recumbent position with eyes closed, but some mammals sleep with eyes open (e.g., cattle), some while standing (e.g., elephants), and some while hanging by their feet (e.g., parrots). Mammals move periodically during sleep, and some humans walk and talk in their sleep. The typical total daily sleep time varies l0-folds among species, from approximately 2 hours in the giraffe to 20 hours in the little brown bat; it is 8 hours for man. Sleep is timed to occur predominantly in the dark hours for man and many other mammals, but for certain mammals like rodents, it is linked to the light period. The scientific study about sleep requires characterization in order to identify sleep disorders, sleep from non-wake up status.

Sleep can be characterized behaviorally as well as using electrophysiological gadgets such as electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG). Behavioral measurements are less reproducible and therefore problematic. The instrumental measurements are more precise and less obtrusive especially in testing the threshold level for sleep. However, there are good correlates between instrumental and behavioral measures. These measures indicate that sleep is a highly organized process, consisting of 2 distinct brain states having a 6 profound impact on other physiological functions. The continuous recordings of electroencephalography (EEG), electrooculography (EOG), and electromyography (EMG) revealed the 2 distinctly different sleep states i.e., rapid eye movement (REM) and non-rapid eye movement (NREM) sleep (5). These statuses appear alternatively in cyclic fashion and reflect different levels of brain nerve cell activity. A typical night of normal sleep has 4 to 6 cycles, with each cycle lasting 90 to 110 minutes, alternating between REM and NREM sleep (6). Fig. 1 illustrates the progression of NREM and REM sleep over an 8-hour night in a healthy young adult.

3. Sleep cycles

3.1. Non-rapid eye movement sleep (Non-REM)

Non-REM sleep is also termed quiet sleep. It (75-80% of total sleep) is subdivided into four stages of sleep progression: Stage 1 NREM sleep (2-5% of total sleep) is a transitional stage occurring at the onset of sleep and briefly during the transition between other stages. In stage 1 NREM sleep, individuals can be easily awakened. Stage 2 NREM sleep typically lasts for ~ 10 to 25 minutes, during which there is a higher arousal threshold. Stages 3 and 4 (-13-23% of total sleep) are defined as slow-wave sleep (SWS) and are the periods of deepest sleep. With each descending stage, awakening becomes more difficult. It is not known what governs Non-REM sleep in the brain. A balance between certain hormones, particularly growth and stress hormones are considered important for deep sleep (8).

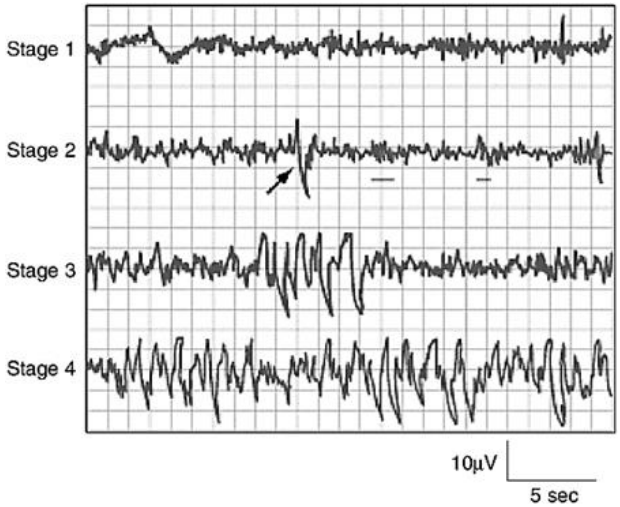

Fig. 1. Characteristic EEG activity of each of the four stages of NREM sleep. NOTE: In stage 2, the arrow indicates a K-complex, and the underlining shows two sleep spindles. According to American Academy of Sleep Medicine (7).

3.2. Rapid eye-movement sleep (REM)

REM sleep is termed active sleep and follows Non-REM. Most vivid dreams are known to occur during REM sleep. Brain activity during this state is comparable to that in waking time, but there is a noticeable decrease in muscle tone, possibly prevent people from acting out of their dreams. In fact, except for vital organs like the lungs and heart, the only muscles not paralyzed during REM are the eye muscles. REM sleep is said to be critical for daytime activities such as learning and day-to-day mood regulations. Those people who are sleep-deprived, their brains work harder than when they are well rested (9).

3.3. The REM/NREM cycle

The cycle between quiet (Non-REM) and active (REM) sleep generally follows this pattern: -After about 90 minutes of Non-REM sleep, eyes move rapidly behind closed lids, giving rise to REM sleep. -As sleep progresses the Non-REM/REM cycle repeats. With each cycle, Non-REM sleep becomes progressively lighter, and REM sleep becomes

progressively longer, lasting from a few minutes early in sleep to perhaps an hour at the end of the sleep episode.

3.4. Electromagnetic characteristics

The cortical EEG of NREM sleep is characterized by frequency slowing and increased voltage relative to the low voltage (10 to 30 µV) and fast frequency (16 to 25 Hz) of activated wakefulness. Relaxed wakefulness with eyes closed exhibits an 8 to 12 Hz EEG pattern of 20 to 40 µV, which further slows and decreases during drowsy sleep. When the arousal threshold is highest, the EEG of NREM sleep has 0.5 to 2 Hz waves of 27.5 µV, which is typically termed slow-wave sleep. The EOG tracings exhibit rapid eye movements during wakefulness that become slow rolling movements at the transition to NREM sleep and, finally, are quiescent during slow-wave NREM (10). The EMG, high in amplitude during wakefulness, is gradually reduced in NREM sleep, although body repositioning and motor events occur during NREM. NREM sleep is classified in 4 stages: Stage 1 (drowsy sleep), stage 2 (intermediate sleep) and stages 3 and 4 (slow wave sleep); (Fig. 1). The cortical EEG of REM sleep is in dramatic contrast reverts to the low-voltage, mixed-frequency pattern seen during drowsy sleep. The EMG is reduced to its lowest level for the night; in fact, there is paralysis of most major voluntary muscle groups through a process of postsynaptic inhibition of motor neurons in the spinal cord. The EOG displays the bursts of rapid eye movements that give this stage its name (10).

3.5. Tonic versus phasic features

REM sleep is characterized by its tonic and phasic features. Tonic features include the persisting muscle atonia and the desynchronized EEG. Phasic REM events are intermittent. The eye movements of REM occur in bursts followed by periods of EOG quiescence. Bursts of eye movements are due to phasic muscle twitches, typically involving peripheral muscles that are superimposed on the tonic atonia (10).

4. Physiologic function during sleep

4.1. Autonomic nervous system

Synchronization of ANS with sleep has been well established. The response of both parasympathetic and sympathetic nerves varies in wakefulness and sleep status. The parasympathetic nerves are more active and predominate during sleep. Sympathetic activity remains relatively constant during both statuses of wakefulness and sleep (11). However, in REM status a dramatic increase and decrease of the sympathetic and parasympathetic activity occur alternately during the tonic and REM respectively (12, 13).

4.2. Respiratory system

Sleep alters breathing pattern, thereby controls breathing. The major physiological factor during sleep that leads to disturbed breathing is the altered skeletal muscle tones. A 13-15% reduction in ventilation occurs during NREM. The tonic, cortical and non-metabolic drive to breathing characteristic to wakefulness, is abolished with the onset of NRAM, further, low muscle tone during sleep causes reduction of upper airway dilator muscle tone and adds resistance to air flow. Atonia characteristic to REM further decreases airflow, this produces irregular breathing patterns the irregularity is greatest in phase REM (14). The metabolic control of breathing is also altered during sleep. The hypoxic ventilatory drive is reduced in NREM sleep and declines further in REM. The hypercapnic ventilatory response is also reduced in NREM but is virtually absent in REM. Breathing during NREM sleep appears to be primarily controlled by arterial levels of CO_2, and at levels below the CO_2, threshold, breathing effort ceases. With the changed CO_2, set point in NREM sleep relative to waking, breathing often can become periodic at transitions between wakefulness and sleep. Thus, in elderly persons who often have fragmented, discontinuous sleep (e.g., frequent wake-sleep transitions) central apnea is produced. Finally, given the virtual absence of hypercapnic drive in REM, obstructive apnea in patients is prolonged (15).

4.3. Body temperature control

Thermoregulation is also altered while sleep; during NREM sleep there is a lower set point, i.e., the temperature is maintained at a lower level than in waking, this cause sweating and shivering at lower temperatures than in waking. Conversely, in REM sleep, there is no temperature regulation, sweating and shivering to cease, hence body temperature equilibrates to the ambient temperature in REM sleep. But typically, REM episodes last 30 minutes or less, and dramatic changes in body temperature do not occur (16).

5. Regulation of sleep and wake

Sleep is an organized biologic function that is actively regulated. It comprises 3 hypothesized processes: (a) a homeostatic process determined by the amount of prior sleep and waking; (b) a circadian process that organizes alternations of sleep and waking over the 24-hour day; and (c) an ultradian process within sleep that controls the alternation between the 2 sleep states, NREM and REM sleep (17). The homeostatic and circadian processes are considered independent although interacting to certain extent.

Whereas most information indicates that REM and NREM sleep are interdependent. The neurobiology of the circadian process has been understood better than that of the homeostatic and ultradian processes or the interaction among these processes. The presence of homeostatic control of sleep is inferred from measurement of sleep latency at night and during the daytime consequences and from EEG slow-wave activity during sleep. The prior amount of sleep and wake is directly related to the degree of daytime sleepiness (18). Reduction of sleep time the previous night by 2 to 8 hours linearly increases

the speed of falling asleep throughout the following day on a standardized measure of daytime sleepiness. Reductions of sleep time by as little as 1 to 2 hours accumulate overnights, reduce sleep latency, conversely, increased nocturnal sleep time, results in increased sleep latencies. Consequently, reductions or increases of sleep time systematically alter homeostatic sleep drive. The other indicant of sleep homeostatic balance is EEG slow-wave activity. Quantification of slow-wave activity during sleep has repeatedly shown a predominance of slow-wave activity during the first hours of sleep and further decline toward the last hours. (See Fig. 1 for stages 3 and 4 sleep).

During the day, after increasing durations of waking, slow-wave activity reappears on naps (19). Total and partial sleep deprivation produce increased slow-wave activity during recovery sleep. Prevention of slow-wave activity with acoustic stimuli during the first 3 hours of sleep, without increasing wake time, results in increased slow-wave activity during the last hours of sleep. The output of these systems suggests a very precise homeostatic regulation of sleep. Independent of the homeostatic process is a circadian process that organizes sleep and waking in phase with 24-hour light-dark cycles. The light-dark cycle, through the retinohypothalamic tract, exerts a direct synchronizing effect on the suprachiasmatic nucleus (SCN), which is considered to be the biologic clock. The efferent projections from the SCN or SCN-triggered hormonal signals regulating the various physiologic rhythms establishing circadian timing, this is not completely understood, but the output of this system is well characterized (20).

Fig. 2 illustrates the human circadian rhythm. Circadian period, phase, and temperature, which in human (between 3 AM and 5 AM), a peak in the early evening (between 5 PM decline at the mid-day (between 1 PM and 3 PM) measured by speed of falling asleep or rhythm, with sleep propensity being greatest around the temperature the temperature peak, hormonal and metabolic rhythms are growth hormone, and melatonin that strongly released of these hormones. Cortisol, in the timing and duration of sleep; its release begins Melatonin release begins with the onset of darkness, and l amplitude are often documented by recording body s is characterized by a daily nadir in the early morning (Fig. 3). Sleep propensity in the duration of sleep parallels the temperature nadir, least during and moderate over the midday (21).

6. Disorders of sleep

The diagnosis and treatment of medicine dating to the mid information about sleep physiology patients getting afflicted Surveys of the US population complain of chronic insomnia and 0.5 sleepless or sleepiness complain - to 120-minute cycle of NREM be interdependent in that during cycles the amount of slow-wave sleep (stages of REM sleep increases) (24). Sleep disorders is a relatively new field in dating to - 1970s. The emergence of a core of basic scientific its pathophysiology and an increasing number of with sleep complaints fostered the development indicate that approximately 10 to 15% of to 5% of excessive sleepiness (25) these are symptoms that can be caused by a number of and(REM 3 and 4) of this field. Respondents (25). These different disorders such as medical and psychiatric reasons including primary sleep disorders for which nutrition could be one of the causes.

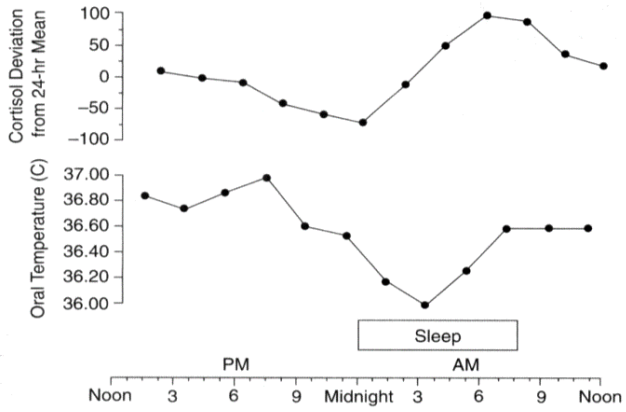

Fig. 2. The human circadian rhythm of oral temperature and cortisol, reprinted with permission from Kryger et al. (22).

In addition, a driven by the SCN. Cortisol, etc., all display a circadian rhythm. Prolactin and linked to sleep, that is, delayed sleep onset contrast displays circadian variation regardless of around the temperature nadir. Levels remain elevated until and 8 PM), and a human, variety of TSH, prolactin, delay the sunrise. Finally, the ultradian rhythm is a 90 sleep. The ultradian and homeostatic processes appear to successive NREM-REM progressively declines, while the amount of REM sleep increases.

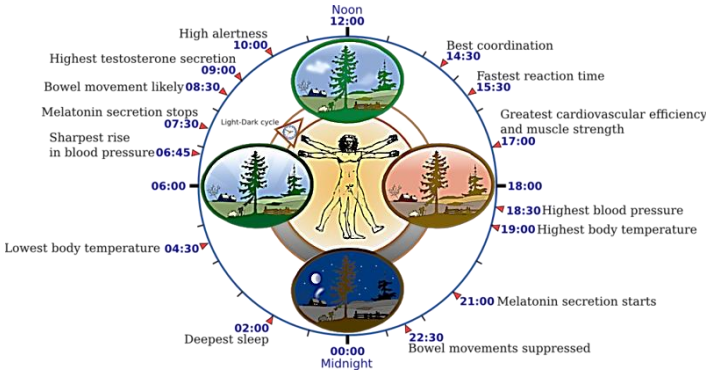

Fig. 3. The body clock guide to better health" by Smolensky and Lamberg (23).

6.1. Disorders of sleep initiation and maintenance (Insomnia)

Insomnia is defined as difficulty initiating or maintaining sleep or non-restorative sleep associated with impairment during the following day (26). Insomnia can occur as a transientand short-term, intermittent or persistent sleeping difficulty. The transient and short-term insomnia may be

caused by disruptions of one's sleep schedule, a non-conducive sleep environment, or a stressful life experience and which last for days or weeks. Insomnia persisting for months is often secondary to various medical and psychiatric disorders. Also, primary sleep-related pathologies such as apnea and periodic limb movements, and behavioral processes like hyperarousal and conditioning can produce chronic insomnia. Some insomniac people with chronic circadian rhythm disorders have also been identified (24).

6.2. Primary insomnia

Primary insomnia is learned, conditioned insomnia. Subjects with primary insomnia have associated physiologic and cognitive arousal with sleep and the sleep environment. The subjects typically report difficulty falling asleep or middle-of-the-night awakenings, or both. Sometimes they describe racing thoughts and anxieties when trying to fall asleep. In other cases, subjects have no explanation as to why they lie awake or awaken in the night, which troubles them further, particularly the middle-of-the-night awakenings. This has been related to the brain activities; two different conditions are proposed to occur: (1) When homeostatic sleep drive is insufficient at sleep onset to overcome the drives to arousal, (2) in case of middle-of-the-night awakenings, a prior sleep of 1 or 2 causes the arousal drives to predominate. Treatment strategies focus on reducing arousal drives through relaxation techniques and on increasing the sleep drive through reduced bedtime and napping. Hypnotics are often used as a symptomatic treatment, typically short- or intermediate-acting drugs (27).

6.3. Secondary insomnias

Although insomnia can be secondary to a variety of medical and psychiatric disorders, the most frequently encountered secondary insomnia is that due to depression. 80% of depressed patients are reported to have insomnia, and recently it is indicated that insomnia can be a prelude to the recurrence of a depressive episode intensifying each other (28). The characteristic of insomnia typically in depression is the problem of early morning awakening. The prominent defective sleep that occurs is with REM sleep, the shortened latencies to REM sleep, an altered distribution of REM sleep across the night, and dense eye movements during REM sleep. The treatment choice is a sedating antidepressant or a hypnotic as an adjunct to a non-sedating antidepressant (29).

6.4. Primary Sleep-Related Pathologies

The 2 sleep-related pathologies typically associated with insomnia are central sleep apnea and periodic leg movements during sleep (30). Central sleep apnea is distinctly different from obstructive sleep apnea; central apnea is primarily a physical positioning of the head during sleep. Periodic leg movements are slow (0.5-second) dorsiflexions of the leg at the knee or ankle that occur with a 15- to 45-second rhythmicity during sleep. The movements are associated with brief arousals and awakenings and, in cases where the movements and arousals occur at particularly high rates, there may be excessive daytime sleepiness. Due to the atonia of REM sleep, the movements usually cease during REM. Treatments with dopaminergic agents, opiates, and benzodiazepines have all been reported to be successful (31).

7. Disorders associated with excessive sleepiness

Excessive daytime sleepiness is defined as an increased likelihood of falling asleep when not intending to do so. It should be differentiated from fatigue or malaise; it is the behavior of falling asleep. Excessive sleepiness is reported by 0.5 to 5% of the population (18). It is the most frequent complaint among the insomniacs. Excessive sleepiness can be caused by habitually inadequate time in bed, such as various drugs and specific sleep disorders. The most common sleep disorder associated with excessive sleepiness is obstructive sleep apnea syndrome (32).

7.1. Obstructive sleep apnea syndrome

In addition to excessive sleepiness as an identifying characteristic, the subjects with sleep apnea syndrome snores, are obese, and is typically male, by a 2: 1 ratio (33).

During sleep, apneas that are usually 10 to 30 seconds long and as frequent as 1 to 2 per minute occur because of obstruction of the upper airway. The airway becomes obstructed from a combination of factors such as excessive fatty tissue in the airway and, related reduced muscle tonus of NREM sleep, the atonia of REM sleep, and the altered metabolic control of breathing during sleep all combined to produce apnea. Brief arousals from sleep occur to break the obstruction and open the airway. These frequent arousals fragment sleep, disrupt its restorative capacity and produce excessive sleepiness. The apnea episode itself is associated with oxygen desaturation and sometimes with cardiac arrhythmias. The standard treatment for obstructive sleep apnea is continuous positive airway pressure (CPAP), which provides a pneumatic splint to the airway, blocking the increasing negative pressure of the obstruction. Properly set CPAP pressures will reverse the apneas, the frequent arousals from sleep and ultimately, the daytime sleepiness (34).

7.2. Narcolepsy

Narcolepsy also has excessive and persistent sleepiness as is the most prominent symptom. The narcoleptic syndrome consists of cataplexy, sleep paralysis, and hypnagogic hallucinations. Cataplexy is a reversible loss of muscle tone, usually triggered by intense emotion that occurs during the waking state. At transitions from wake to sleep, the patient with narcolepsy often experiences sleep paralysis, an inability to move or speak, and visual hallucinations. The clinical symptoms of cataplexy-sleep paralysis and hypnagogic hallucinations are all pathologic manifestations of REM sleep.

Sleep-onset REM periods are now considered to be the pathologic sign of narcolepsy. It is a genetic disorder and research has identified an autosomal recessive mutation of the hypocretin receptor 2 genes as responsible for narcolepsy in dogs (35). Although there is a very close similarity between human and canine narcolepsy, non-genetic factors and other genes may be important as well. Only 1 in 4 identical twins share the disorder, and the symptoms of the disorder are not expressed until the second and third decades of life. Earlier human genetic studies reported a tight association between class II human (36). Leukocyte antigen haplotypes and narcolepsy, suggesting that autoimmunity plays a role Thus, many think it is multigenic and environmentally influenced stimulants for the excessive sleepiness and antidepressants for the cataplexy-most antidepressants are profound REM suppressing drugs.

8. Circadian rhythms and its disorders

In mammals, the most evident manifestation of adaptation to night and day is the temporal organization of behavior into periods of rest and activity, sleep and waking. The generation and regulation of circadian rhythms in human is a function of a specific neural system, the circadian timing system (CTS). The CTS is responsible for providing not only a temporal organization for behavior but also for a large series of physiological and endocrine functions that form a critical substrate for behavior. The ultimate function of the CTS is to enhance the adaptive value of both sleep and waking behavior to promote survival and reproduction. To understand the neurobiology of normal and disordered circadian function, consider the two fundamental properties of circadian rhythms, generation by endogenous pacemakers and entrainment by environmental stimuli (37). The obvious explanation of a daily rhythm in sleep-wake behavior is that it represents a simple, passive response to the environmental light-dark cycle, but the rhythm persists for a period that differs from 24 hours a day. The maintenance of free running rhythms is accountable only by the action of an intrinsic clock, or pacemaker (38). A circadian rhythm disorder refers to a condition in which an individual's circadian rhythm of sleep and wakefulness is out of phase with the sleep and wake periods necessary to conform with conventional environmental patterns (Fig. 4). Normally, when sleep and wake periods are altered, the biologic rhythms readjust to the new pattern if there is a parallel change in the light-dark cycle (40). In circadian rhythm disorders, a persistent misalignment exists, and adjustment never seems to occur, or, at best, is most difficult. The rhythm can be advanced (i.e., early sleep onset) or delayed (i.e., late sleep onset) relative to the environment. The persisting circadian rhythm disorder should be contrasted with a transient circadian rhythm disturbance, such as in jet lag or with certain work schedules. These are transient disturbances, and, when a stable sleep schedule is established, adjustment occurs. Light has been shown to be the major synchronizing stimulus, operating through the SCN. As noted earlier, the SCN receives projections from the retina, and this track with others, transmit photic stimuli synchronizing sleep (41). Thus, the pathophysiology of the circadian rhythm disorders is presumed to be associated with the pacemaker (i.e., the SCN), the coupling mechanisms, or the synchronizing mechanisms. Bright light treatment, properly timed to produce a phase advance (i.e., early morning) or delay (i.e., early evening), has been used with some success (42).

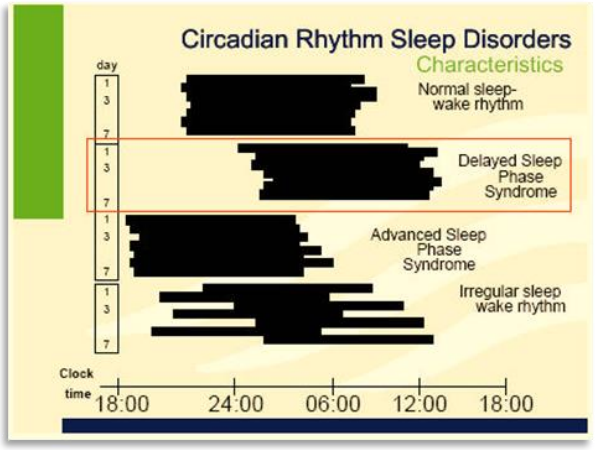

Fig. 4. Circadian sleep-wake rhythm disorders (39).

9. Sleep syndromes

9.1. Work-shift syndrome

In industrialized countries, many occupations require unusual work hours. This is particularly true for health care workers, police and security guards, truck drivers, and workers in heavy industry (43). The factors involved in adjusting to shifting work are reported to be complex and disturbs circadian mechanisms (44). The major symptom of work-shift disorder is impaired sleep. There have been recent descriptions of the use of bright light and melatonin to treat the circadian abnormalities associated with shift work (45, 46).

9.2. Delayed phase sleep syndrome

Delayed phase sleep syndrome is characterized by a persistent inability to fall asleep and arise at conventional clock times; the phase of the sleep-wake cycle is delayed (See Fig. 4). Sleep onset is usually delayed until early morning, with a consequent delay in rising (47). Individuals attempting to go to bed earlier are unable to fall asleep until their usual time and an attempt to maintain a normal schedule cause insomnia and daytime fatigue. This syndrome has customarily been viewed as a consequence of choice, or lifestyle. Although the symptoms clearly represent the lifestyle in some individuals, in others it is clearly a disorder of entrainment, a failure of pacemaker sensitivity to light in the phase-advance portion of the PRC (48).

9.3. Irregular sleep-wake pattern syndrome

Affected individuals have an irregular distribution of sleep and wake with numerous interruptions, which are relatively less common. These individuals generally complain of delayed sleep latency, insomnia, and daytime fatigue, which appear to be arrhythmic. Studies in which 24-hrs body temperature recordings have been made and found no discernable rhythm in core body temperature (49). Therefore it is possible to reoccur in two situations either with evident hypothalamic pathology, such as a tumor compressing the chiasmatic region of the hypothalamus or a spontaneous appears of the problem without evident neuropathology (7).

10. Sleep Hygiene

Sleep hygiene encompasses environmental conditions and lifestyle practices that influence sleep (50). Lifestyle practices which have major influence on sleep include: ingesting caffeine, drinking alcohol, smoking, exercising (amount and timing), napping, bedtime routine, and consistency of getting-up. Proper sleep hygiene can be defined as "controlling all behavioral and environmental factors preceding sleep that interfere sleep (51). It is the practice of following simple and sensible guidelines to ensure restful and effective sleep, which can promote daytime alertness as well as help treat or avoid sleep disorders. Trouble falling asleep and daytime sleepiness are indicators of poor sleep hygiene. The International Classification of Sleep Disorders Revised (ICSD-R) specified the importance of assessing the contribution of inadequate sleep hygiene in maintaining sleep (52). The effects of optimizing conditions and behaviors to facilitate cyclical, restorative sleep, particularly with insomnia, have been the subject of the only limited investigation. Conceptually, insomnia occurs when a combination of predisposing, precipitating and perpetuating factors reach a threshold (51). Predisposing or constitutional factors that render people prone to not sleeping are those features that are relatively permanent like age, genes, physiology, medical conditions, or personality characteristics. Precipitating factors are those that trigger the onset of insomnia such as stressful life events like grief, divorce, or acute illness. Perpetuating factors are those lifestyle factors that reinforce or sustain insomnia. For example, caffeine consumption or an irregular sleep-wake schedule perpetuates insomnia precipitate in the presence of other events. Theoretically, factors or combination of factors that increase arousal, prevent sleep generation or disrupt circadian rhythms bringing people closer to their insomnia thresholds. Common features of insomnia are prolonged time to go to sleep, awakening prematurely during the sleep and feeling un-refreshed after sleeping. Midlife women frequently report difficulty sleeping as they transition through menopause especially if they have hot flushes or night sweats (53, 54). Poor sleep has been linked to poor physical health, loss of memory, and poor work performance; compromised interpersonal relationships; diminished coping ability and quality of life; impaired concentration; difficulty accomplishing tasks; greater drowsiness when driving; and more automobile accidents (55). Poor sleep, as well as habitually short sleep has been associated with increased risk of mortality. Abstaining from caffeine or limiting caffeine in the afternoon and/or evening is recommended for those with insomnia to avoid its invigorating effects. Caffeine is a commonly used stimulant that increases physiological arousal and prevents sleep onset. As a function, caffeine found in coffee, tea, soft drinks, and medications the effect is already known. Tobacco is another strong risk factor for insomnia. Insomniacs are recommended to avoid smoking for improving sleep. Smoking is associated with increased sleep-onset latency and increased awake time (56). Although alcohol expedites sleep onset, even a moderate amount of alcohol (three servings) disturbs sleep throughout the night, making alcohol abstinence a sleep hygiene recommendation (57). In general, alcohol increases non–rapid eye movement sleep (NREM) and decreases rapid eye movement (REM) sleep in the first half of the night and increases REM sleep and sympathetic nervous system arousal in the last half of the night. After alcohol is metabolized, rebound arousal occurs that can lead to awakening from sleep. The amount of alcohol, nicotine, and caffeine consumed and their rate of metabolism, along with individual sensitivity effects sleep and the effect may last more than one night (58). Regular exercise is recommended to obtain better-quality sleep, although the mechanism by which this occurs has not been clearly delineated (59). Exercise is associated with increased total sleep time and slow-wave sleep, perhaps as a function of raising body temperature, as well as decreased sleep-onset latency, duration of REM sleep, and waking after sleep onset (60). Reported from the effect of Physical fitness, explain the benefit regularly exercises on sleep (61). Exercise causes arousal, the basic recommendation regular exercise in the morning hours and at least 3 hr prior to sleep alleviate delayed sleep onset. Having consistent bed and getting up is believed to strengthen the circadian timing of sleep through a homeostatic mechanism and by habitual rhythmic exposure to environmental light and darkness, enabling the brain and body to regulate sleep and awaking time (62). Getting up at the same time each day is more important than having a regular bedtime for keeping sleep synchronized with the light and dark cycle. Generally, when people have difficulty falling asleep they tend to sleep longer in the morning. Such pattern gives rise to a reregulating sleep schedule. Hauri and et.al explain the possible mechanism of such reregulation leading to delayed sleep onset. In a cyclic fashion, the duration of wakefulness prior to the next sleep bout (after approximately 12-16 hr) creates pressure to sleep, leading to normal refreshing sleep. When getting up late in the morning, and getting to bed at the usual bedtime the following night means a shorter wake duration, thus cause Less building up of pressure to sleep. Therefore lifestyle recommendations insist to avoid the sleep-disruptive substances the caffeine, nicotine, and alcohol; to engage in well-timed exercise and to pursue regular retiring and awakening times as daily habits to positively modulate sleep in people with insomnia (63). Although sleep hygiene education is included in cognitive-behavioral treatments for insomnia, actual sleep hygiene practices and their consistent

use by adults, specifically women, with insomnia have been documented sparsely or not at all. Research on whether groups with and without insomnia differ in sleep hygiene practices has produced mixed results, suggesting that people either are unaware of sleep hygiene principles or are aware but do not practice them (64).

11. Prevalence of Insomnia

An international collaborative study conducted in several countries revealed that physicians could detect only 50 % of patients among those who had sleep problems. Despite this high prevalence rate evidence suggest that insomnia is under diagnosed and under treated globally (65). Statistics, in general, indicate that 10- 15% of the adult American population suffers from chronic insomnia, an additional 25 – 35% has transient or occasional insomnia. A higher percentage (57%) was observed in the elderly (66). Approximately 40% of community-dwelling adults and 69% of primary care patients report sleep problems in the form of difficulty falling asleep, discontinuous sleep, and/or non-restorative sleep (67). America being an exception in identifying insomnia as a risk factor of public health importance is a leading country in providing a solution for its management. Detailed statistics about the prevalence of insomnia and other related information is made available only in the USA. The occurrence of insomnia in other parts of the world other than America indicates that people experience insomnia symptoms of various degrees of severity, difficulty initiating sleep (15.3%) and non-restorative sleep (19%). However, it is commonly believed that sleep duration in the population has declined over the decades. Common sleep difficulties include sleep initiation and maintenance. The statistic Gallup Organization from the United States indicate 49% of adults “do not sleep well” for 5 nights per month, 10–40% suffer from intermittent insomnia, and 10–15% have long-term sleep difficulties (68). A survey reported from Australia, suggesting insomnia prevalence among urban men and women to be 17% and 25% respectively (69).

12. Contributory factors for lack of statistic

Effective behavioral and pharmacologic therapies exist for insomnia, only 31% of poor sleepers discuss their insomnia with a physician (70). Experts in sleep medicine have accordingly called for improved insomnia recognition and treatment. However, consideration of the patient’s perspective suggests that the problem is not simply physician is failure to recognize sleep problems; many patients with defined insomnia do not view their sleep problem as clinically significant. Of the 9% of American adults reporting the most severe sleep loss, one third do not consider their problem to be serious, and only 24% of British adults who report insomnia in surveys also describe themselves as dissatisfied about their sleep (71). Thus, more information is needed to understand the patient-centered factors involved in insomnia presentation. This information may help explain why only some poor sleepers seek help, identify subgroups of poor sleepers to target for intensive intervention efforts and identify intervention targets (beyond sleep quality) of primary importance to patients (71). It is estimated that between 5% and 36% of individuals with insomnia have at some time consulted a physician specifically for their sleep problem, while 27–55% have discussed it with a physician in the course of a consultation for another problem (72). Higher consultation rates in people with insomnia may be partly due to the fact that they suffer from a greater number of co-morbid physical and/or psychological health complaints.

13. Consequences of disturbed sleep

It has been demonstrated that sleep is vital for physical and mental functioning. Lack of sleep is associated with daytime fatigue, impaired cognitive functioning, mood disturbances, increased rates of infections, depression, and impaired productivity at work (73). Furthermore, lack of sleep has been found to be predictive of traffic and occupational accidents and has deleterious consequences on body systems. Moreover, it has been implicated in decreased immune functioning and increased risk of cardiovascular disease and diabetes (74). This indicates that sleep disorders have negative consequences for health, functioning, and overall quality of life, especially in elderly people. Therefore, it will be important to improve the quality of sleep, which may improve the individual energy/vitality levels and daytime quality of life (75). Research also reveals higher hospitalization rates in individuals with insomnia although the specific contribution of insomnia to hospitalization is poorly understood. People with insomnia often use prescription medications or over-the-counter (OTC) products or alcohol to manage their sleep difficulties (76). Insomnia can have a negative impact on various aspects of daytime functioning, including work performance. Several cross-sectional studies of working-age adults have found a link between poor sleep and absenteeism, reduced work capacity/ productivity, and low levels of work satisfaction and performance (45). Schweitzer et al. estimated that individuals reporting poor sleep miss at least five more days of work per year than good sleepers, while Leigh reported monthly absence rates 1.4 times higher in poor sleepers than in workers with no sleep difficulties. The specific causes of these absences are not clear. A recent study suggests that depressive, behavioral and other complaints that may accompany insomnia explain work absenteeism and not insomnia per se. These results raise an important issue concerning the specific role of insomnia relative to other co-morbid problems in accounting for work absenteeism (77). Fatigue, combined with other cognitive difficulties associated with insomnia (e.g., poor attention), can lead to serious consequences when individuals are carrying out tasks such as driving. Individuals with sleep problems have been found to be three to four times more likely than good sleepers to experience a motor-vehicle or other serious accident (78). Similarly, decision-making errors and on-the-job accidents are also more frequent in individuals with sleep problems.

Interpreting such results is complicated by the fact that the largest proportions of sleep-disorder-related accidents occur for people suffering from sleep disorders other than insomnia (i.e., sleep apnea), which are usually not considered separately from insomnia. As a result, the relationship between insomnia alone and accident rates is not clear. Insomnia is found to be more common in females especially during menopausal age, elderly people with medical and psychological conditions (25, 78). It is documented that insomnia per se affects the quality of life, the insomniacs tend to exhibit compromised activity profile, frequent illness and require greater health care costs. The general consensus is that without sleep, physical, mental, emotional functions are impaired, initiate gradually, reach peak with depression and mania at severe conditions. The quality of life of an insomniac is described as poor as the people with heart failure (79).

14. Conclusion

Sleep quality represents a complex phenomenon that is difficult to define and measure objectively. It includes quantitative aspects of sleep, such as sleep duration and sleep latency as well as more subjective aspects such as depth or restfulness of sleep. Polysomnography provides accurate information on the physiologic indices of quality of sleep. However, it is expensive, and it requires much time for testing and interpretation of data. Actigraphy is another method for objective sleep monitoring that measures sleep activity around the clock. Alternatively, self-reported methods such as sleep diaries, sleep logs and sleep questionnaires provide a measurement of quality of sleep experienced by the patient. These subjective methods attempt to measure both quantitative as well as qualitative aspects of sleep and are easily administered, inexpensive and have wide applicability. The current treatment options for insomnia are pharmacotherapy and cognitive behavioral therapy. Treatments are considered effective if they shorten sleep onset latency (SOL) or increase total sleep time by 30 minutes. Cognitive behavioral therapy is considered the best practice. Other popular remedies used to treat sleep difficulties include prescribed sedatives and tranquilizers, herbal extracts and complementary medicines, massage and relaxation techniques, regular physical activity, and avoidance of stimulants such as caffeine before sleeping.

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