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SLEEP CHANGES IN ADOLESCENCE - A REVIEW

DR ELLORA DEVI*[1] AND DR K.C.S.RAY[2]

[1]Astt. Prof. Department of Physiology, IMS & SUM Hospital, SOA University, Bhubaneswar, India
[2]Director, NIFT, Bhubaneswar, India

ABSTRACT

Sleep is a basic drive of nature. Although many questions regarding the role of sleep remain unanswered, scientific studies have shown that sleep contributes significantly to several important cognitive, emotional and performance-related functions. Deprivation of sleep causes feelings of “sleepiness,” decreased levels of alertness or concentration. Excessive sleepiness is also associated with reduced short-term memory and learning ability, negative mood, inconsistent performance, poor productivity and loss of some forms of behavioural control. The consequences of insufficient sleep among adolescents are particularly important to understand because they appear to be closely tied to key elements of human development. Achieving developmental goals during adolescence is essential for lifelong success and social competency. In addition, the transition from childhood to adulthood is a critical time for “seeding” the values and habits that will shape their lives. Therefore, understanding sleep patterns of adolescents is important so that possible strategies for intervention can be formulated.

KEYWORDS: **Sleep, Adolescence, Circadian rhythm, Development**

DR ELLORA DEVI
Astt. Prof. Department of Physiology, IMS & SUM Hospital, SOA University, Bhubaneswar, India

Corresponding author

INTRODUCTION

Sleep is a basic drive in nature of all living organisms. Adequate sleep is essential to think more clearly, complete complex tasks better and more consistently and enjoy everyday life more fully. Sleep contributes significantly to several important cognitive, emotional and performance-related functions. Sleep provides nourishment for the brain and inadequate sleep can be harmful, even life-threatening. Lack of sound sleep leads to sleepiness, decreased levels of alertness or concentration and even unexpected sleep. Excessive sleepiness may result in reduced short-term memory and learning ability, negative mood, inconsistent performance, poor productivity and loss of some forms of behavioural control. Sleep is broadly classified into two types: rapid-eye-movement (REM) sleep and non-REM sleep (NREM). Both sleep stages for an adequate amount of time is essential for complete rest to mind and body. NREM sleep consists of four stages that range from drowsiness to deep sleep. In the early stages (I and II), individuals awake easily and may not even realize that they have been sleeping. In the deeper stages (III and IV), waking is difficult. When awakened, individuals in stages III and IV may feel disoriented and confused. In NREM sleep their muscles are more relaxed than when awake. Although the sleeper in stages III and IV is able to move, this doesn't happen because the brain is not sending signals to the muscles to move. REM sleep refers to active sleep, which is when dreaming occurs. During REM sleep, the breath and heart rate become irregular, the eyes move rapidly back and forth under the eyelids. Below the neck, however, the body is essentially paralyzed because the nerve impulses to the muscles are blocked. Both REM and NREM sleep states develop before birth. Sleep cycle patterns and the amount of sleep needed changes from infancy to childhood, but at the age of four, most children sleep 10 hours a night and their sleep patterns are much like those of adults.

Our understanding of the development of sleep patterns in adolescence has advanced considerably in the last 20years. Along the way, theoretical models of the processes underlying the biological regulation of sleep have improved. Initially, it was assumed that the amount of sleep required declines with age; this was axiomatic- the older you are, the less sleep you need. The early 1970s saw a growing awareness that sleep patterns change fundamentally at the transition to adolescence – a phenomenon that is widely acknowledged today. Survey studies clearly showed then and continue to show that the reported timing of sleep begins to shift in early adolescence, with bed time and rising time both occurring at later hours. A longitudinal study begun in 1976 at the Stanford University summer sleep camp attempted to examine this axiom[1]. Another study of Strauch and Meier is the most comprehensive longitudinal survey, covering a 10-year period (1975-1985) with surveys at 2-year intervals. Most studies of adolescent sleep habits show a pattern of decreasing total sleep time, a tendency to delay the timing of sleep, and an increase level of daytime sleepiness. Laboratory tests have shown that adolescents do not have a decreased need for sleep but probably need more sleep than pre-pubertally.[2]

DEVELOPMENTAL CHANGES AFFECTING ADOLESCENT SLEEP/WAKE PATTERN

Adolescence is a crucial phase of development when profound biological, physical and social changes occur, often leading to tension, stress and other problems. Profound changes in sleep behaviour are observed during adolescence. Key changes in sleep patterns and needs that are associated with puberty include[3]:

Adolescents require at least as much sleep

Adolescents require as much sleep as they did as pre-adolescents (in general, 8.5 to 9.25 hours each night).[4] Increase in daytime sleepiness For somedaytime sleepiness increases to pathological levels — even when an adolescent's schedule provides for optimal amounts of sleep.[5]

Adolescents' sleep patterns undergo a phase delay

There is, a tendency toward later times, for both sleeping and waking. Studies show that the typical high school student's natural time to fall asleep is 11:00 pm or later.[6] A number of factors affect the development of adolescent sleep patterns:

Puberty

Older adolescents become sleepy in the daytime, even when sleeping as much as younger adolescents. Therefore, if anything, older teens may require more sleep to maintain alertness than do younger teens.

Parental influence

Among environmental factors that appear to influence the development of sleep/wake patterns early during adolescence is a change in the way parents regulate child's sleep. One major early change in the organization of sleep/wake behaviour in adolescents is an alteration in the nature of parental influences: parents of children and younger adolescents direct their attention to bedtime; in older adolescents, parental influence diminishes at bedtime and becomes more important to waking up.

School schedules

The time that school begins in the morning has an obvious impact on setting sleep/wake schedules in adolescents, presumably strongest in its effect on the time youngsters arise in the morning. Adolescents tend gradually to adjust to shifts in school start time over a series of years by narrowing the interval between getting up and going to school. The pattern of later rising times on weekend morning also suggests that school schedule impinges on the regulation of sleep/wake pattern in school mornings. A study in India clearly demonstrates that early school start timing contribute significantly to sleep deprivation among adolescents, leading to daytime sleepiness and poor academic performance. We hope that the crucial advantage, conferred by late school timings to the student's alertness and performance maybe considered while debating on the ideal school timings for children.[7]

Socio-cultural

Increased homework, extra-curricular activities, such as sports, television viewing, playing video games, and surfing the Internet had negative impact on sleep/wake parameters. Moreover, presence of a television set or video game in the child's bedroom increased their activity before bedtime. Time to return home later after-school activity, extra hours of coaching study also had a negative impact on sleep/wake patterns.[8,9,10]

Circadian rhythms

They are physical, mental and behavioural changes that follow a roughly 24-hour cycle, responding primarily to light and darkness in an organism's environment. The human sleep wake cycle is the prototype example of all the circadian physiological processes in the body. Circadian rhythms continuously drive human functioning round the clock. Sleep tendency (latency to sleep onset) was examined during extended waking in pre-pubertal and mature adolescents to determine whether sleep pressure is lower near bedtime in the latter group. Sleep tendency after 14.5, 16.5, and 18.5 hrs awake was lower in mature versus pre-pubertal adolescents, supporting the hypothesis that a developmental change of intrinsic sleep–wake regulation may provide physiologically mediated permission for later bedtimes in older adolescents.[11]

SLEEP MODELS

The primary models describing intrinsic sleep-wake regulation rely on two principal factors, one attributed to the circadian timing mechanism, the other to underlying sleep-wake mechanisms.[12] Borbély was the first to articulate clearly a model identifying these two factors, labeling the circadian process, *Process C*, and the homeostatic process, *Process S*.[13,14] In one description of the model, Process C is modeled by a daily oscillation of one threshold at which sleep can begin and a second at which sleep terminates; process C interacts with Process S, which accumulates as wakefulness is

extended and decays exponentially with sleep initiation.[15] According to the model, sleep will begin and end where the two functions cross. In the years since this model was first described, it has been refined, other models have been proposed, and more has been learned about the interaction of the circadian and homeostatic factors. For example, Åkerstedt and Folkard[16] include in their model a "sleep inertia" factor (Process W) along with the additive circadian and homeostatic factors in order to predict waking behavior better. Edgar,[17,18] by contrast, casts the circadian and homeostatic factors as opponent processes, in which a circadian (clock-dependent) alerting process opposes a wake-dependent sleep-promoting process to maintain wakefulness in primates across the subjective day. Dijk and Czeisler[19] proposed a similar model of opposing processes to describe the maintenance of sleep across the night in humans. Although these models serve as important theoretical background for our studies, most models of sleep and vigilance regulation (such as those proposed by Edgar et al,[17] Dijk and Czeisler,[19] and Jewett and Kronauer[20]) do not account for the midday increase in sleep tendency, largely because the models are based upon introspected sleepiness. Richardson and colleagues[21] showed that subjective reports do not manifest the same diurnal pattern of sleepiness as does the MSLT(Multiple sleep latency test); thus, the midday effect is not represented in most models based on subjective estimates (as, for example, in the Jewett and Kronauer model[20]). Lack and Patrick[22] have presented a model that accounts for the midday alertness trough requiring only an intrinsic circadian rhythm factor. Broughton,[23] however, modeled an afternoon "nap zone" based on an interaction between circadian and homeostatic parameters, a model that is very similar to the predictions by Mary A Carskadon[12] constructed from data of pubertal adolescents.

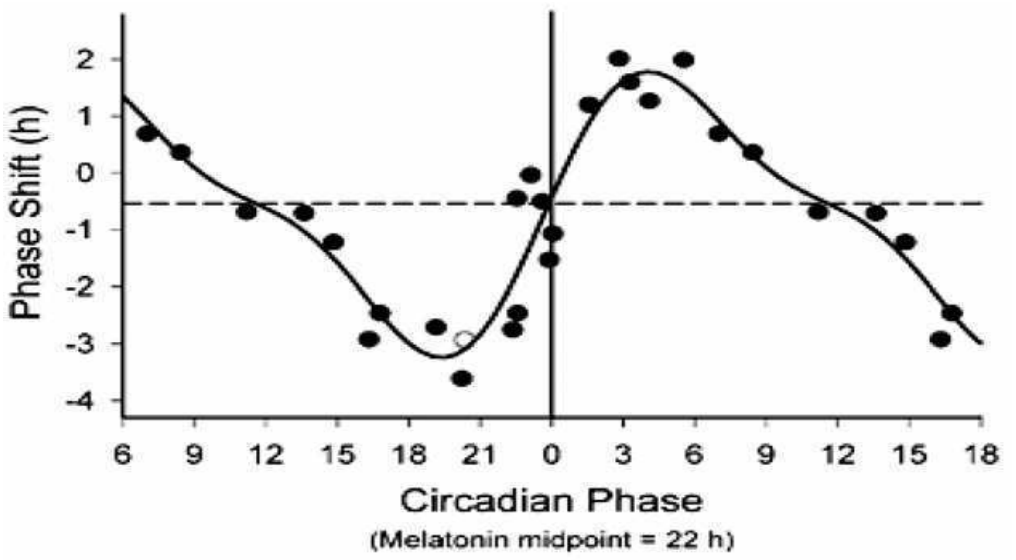
The underlying processes that can explain diurnal sleepiness come into sharp focus with MSLT data collected from adolescents undergoing a protocol that allows isolating the effects of the sleep-wake homeostatic process from those of the circadian timing system. In order to examine the independent and interactive effects of these systems, one must measure variables or systems at many times and many circadian phases. One way to accomplish such multiple measurements is to vary the length of time awake and asleep, equalizing for time of day[24]; however, such an approach is difficult to implement in a design that is orthogonal both for sleep-wake and time of day. An alternative experimental approach that has recently led to significant gains in human studies is called forced desynchrony (FD).[25]

THE CIRCADIAN TIMING SYSTEM

The circadian timing system provides temporal organization for regulatory mechanisms to facilitate adaptive behaviour, such as feeding, reproduction, and sleep/wake cycles[26]. These coordinated temporal patterns, or circadian rhythms, are self-sustained and oscillate with a period of about 24 h. The internal mechanism (pacemaker) that organizes these rhythms in mammals has been localized to a small paired nucleus in the hypothalamus, the supra-chiasmatic nucleus (SCN)[27]. Biological events or markers associated with these rhythms can be used to estimate circadian time or phase. Melatonin is a hormone secreted by the human pineal gland that oscillates with a circadian rhythm. Levels of the hormone are nearly absent during the daytime, rise in the evening near one's usual bedtime, stay relatively constant during the night time, and decline near one's habitual wake-up time. Melatonin is suppressed by light[28], and recent studies show that even room light levels (_200–300 lux) can have a suppressive effect on human endogenous melatonin production[29,30,31] . The onset of melatonin secretion, also called the dim light melatonin onset (DLMO) phase, is a marker of the circadian timing system[32] and can be measured from saliva samples collected at 30-min intervals in dim light (<30 lux)[33] . Research laboratories often define DLMO phase as the time at which melatonin concentration rises above a designated threshold (e.g.4 pg/mL for saliva and 10 pg/mL for plasma melatonin). The decline of

melatonin, also called the dim light melatonin offset (DLMOff) phase, and the midpoint between DLMO and DLMOff are other phase markers of the circadian timing system derived from the melatonin rhythm. Other rhythms such as core body temperature have also been used to mark the circadian system; however, DLMO phase is currently thought to be the most reliable marker of phase[34,35]. The circadian timing system oscillates with an intrinsic period slightly different from 24 hrs but synchronizes (entrains) to the 24-h day in response to external time givers, or zeitgebers. The primary synchronizing stimulus for the circadian timing system is the daily variation of daylight and darkness[36]. The circadian system is sensitive to light, especially during the night time, which for humans is the usual sleep period. A phase response curve (PRC) describes how light input is able to shift circadian rhythms earlier or later in time.

Graph – 1
Circadian Phase



Graph above shows a PRC to light for adult humans, constructed by Khalsa and colleagues (2003)[37]. These researchers measured circadian phase by way of plasma melatonin before and after a 6.7-hr bright light stimulus (10,000 lux) timed at various times of day. The x-axis represents the timing of the light stimulus. The melatonin midpoint phase is defined as 22 h and core body temperature minimum phase is estimated at 0 hr. (The midpoint of melatonin secretion usually occurs just after the midpoint of a regular sleep episode in adult humans sleeping 8 hr)[38]. Responses to the bright light stimulus are shown on the y-axis as a phase shift.

In general, the human system responds in a systematic and predictable manner. Light during the end of the daytime and beginning of the night time (before the estimated core body temperature minimum) shifts circadian rhythms later (phase delay); bright light during the end of the night time or at the beginning of the daytime (after the estimated core body temperature minimum) shifts circadian rhythms earlier (phase advance)[37,39]. (It is important to note that a PRC to light for adolescent humans has not yet been described and may differ from the adult PRC.) This flexibility of the system allows animals, including humans, to entrain to the 24-hr solar day. The entrainment process differs among individuals because some manifest an oscillatory period shorter than 24 hr and some longer than 24 hr. The intrinsic period of healthy human adolescents averages 24.27 hr[40]. The majority of humans entrain by a small phase advance each day and a small-proportion entrain by a small phase delay[41]. According to the adult human

light PRC, morning exposure to light will facilitate entrainment in humans who have an intrinsic period greater than 24 hr, whereas evening light exposure will entrain individuals with an intrinsic period shorter than 24 hr. The circadian timing system undergoes developmental changes during adolescence. Initially, behavioural factors (e.g., social and academic responsibilities) were thought to be entirely responsible for delay in sleep/wake patterns of adolescents. Circadian timing change was noted in a study of circadian phase preference in young adolescents.[42] Phase preference, or morningness / eveningness is a behavioural pattern related to the time of day best preferred for waking behaviour. Researchers found that phase preference was correlated with self-assessed pubertal development.[42] Subsequent studies confirmed that more mature and older adolescents prefer later timing of activities than younger, less mature adolescents.[43,44] Adolescents with evening and morning phase preference also show differences in patterns of sleep/wake; those with evening preference report later sleep schedules than those with morning phase preference.[44] These studies provide some evidence that sleep regulation mechanisms may differ between phase preference types. The adolescent phase preference shift toward eveningness is indicative of underlying changes to mechanisms regulating sleep and wake during pubertal development. Indeed, laboratory studies provide evidence to suggest that the circadian timing system changes during puberty: pubertal stage is positively associated with later circadian timing when sleep (dark) is held fixed.[45,46] Adolescent phase delay may be explained by a change in light sensitivity. Recent work shows that less total sleep time may attenuate the phase shifting response. Evidence to date supports hypotheses that circadian mechanisms change across adolescent development, including a change toward evening circadian phase preference and later circadian phase. Furthermore, adolescents may tend to have a longer intrinsic period compared to adults. Slower accumulation of homeostatic sleep pressure during puberty also permits the older adolescent to stay awake longer and, thus, delay the sleep/wake (dark/light) cycle.

Therefore, in addition to environmental factors, underlying changes in the circadian and sleep systems accompanying pubertal development may also be associated with later sleep/wake schedules often observed in adolescents.[47] Puberty is associated with enormous change to the neuroendocrine milieu. To date, however, it is unclear how these changes interact with specific sleep regulation mechanisms.[48]

The presence of irregularity of sleep–wake schedules in E-types and their tendency to extend sleep duration has been described in various studies.[49,50,51] In fact, as already pointed out by Carskadon et al[3], E-types used to go to bed and wake up later, especially on weekends, on average 2 h later than M-types, to sleep more at weekends than on school nights and to complain of a sleep debt of more than 1 h compared with the other chronotype. On the contrary, E-types stated, in one study[51] as well as in other reports, a greater need for sleep than other groups and tried to reduce their sleep debt by not only extending weekend sleep duration, but also by napping on school days. This irregularity of sleep–wake schedules may produce insufficient sleep and excessive daytime sleepiness.[52] Furthermore; Rosenthal et al. found an overall higher level of sleepiness, evaluated not only subjectively with the Sleep–Wake Activity Inventory but also objectively by means of multiple sleep latency tests in a morning group of young adults.[53] In contrast, study by Flavia giannotti et al found that E-types complained of more subjective sleepiness and obtained higher scores in Sleepiness Scale than the M-types, indicative of sleepiness problems.[51] Regression results pointed out that evening preference, as well as a problematic sleep, irregularity of sleep–wake pattern and high level of emotional problems was significantly related to daytime sleepiness. It is quite difficult to explain why adolescent E-types complained of more subjective daytime sleepiness than older ones. Older E-types appear to adapt better to sleep irregularity and sleep restriction than adolescents.

However, further studies are needed to better understand these differences.

CONCLUSION

Our understanding of changes in sleep patterns occurring in adolescence is still evolving. Maturational changes that affect the alignment of circadian and sleep-wake processes appear to underlie the reorganization of diurnal sleep tendency. The pathway from pubertal maturation to phase angle realignment is not clear, and the possibility that feedback of behavioral factors ultimately is responsible for this reorganization has not been ruled out. In terms of the practical realities of adolescents' lives, this combination of forces is particularly devastating for adjusting easily to the demands of early-morning school starting times. Developmental delays in sleep/wake behaviour across adolescence are associated with extrinsic and intrinsic factors. Investigations of circadian timing mechanisms and homeostatic sleep processes may provide insights into biological underpinnings of behavioural changes. Understanding sleep patterns of adolescents will help in developing strategies for intervention to achieve adolescents' full potential while fulfilling their academic, socio-cultural and biological demands.

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