## Adolescence and Sleep

Luiz Antonio Del Ciampo ${ }^{1 *}$ and Leda Regina Lopes Del Ciampo ${ }^{2}$<br>${ }^{1}$ Department of Puericulture and Pediatrics, Faculty of Medicine of Ribeirão Preto, University of São Paulo, Brazil<br>${ }^{2}$ Department of Medicina, Federal University of São Carlos, Brazil<br>*Corresponding author: Luiz Antonio Del Ciampo, PH.D., Professor, Doctor, Department of Puericulture and Pediatrics, Faculty of Medicine of Ribeirão Preto, University of São Paulo, Brazil, Tel: 5516 36022479; E-mail: delciamp@fmrp.usp.br

Received date: July 04, 2016; Accepted date: July 13, 2016; Published date: July 20, 2016
Copyright: © 2016 Ciampo LAD, et al. This is an open-access article distributed under the terms of the Creative Commons Attribution License, which permits unrestricted use, distribution, and reproduction in any medium, provided the original author and source are credited.


#### Abstract

Sleep is a physiological condition characterized by a reversible behavioral state with changes in the level of awareness and responsiveness to stimuli and plays a fundamental role related to electrophysiological, neurochemical, anatomical and functional changes of the brain. It is controlled by chronobiological and homeostatic mechanisms and is intensely related to the periods of wakefulness. One of the most immediate results of the poor quality of sleep is the drop in performance on the next day, causing damage during wakefulness, such as drowsiness, mood fluctuations, anxiety, low self-esteem, slow thinking, memory loss, poor school and personal performance, and predisposition to accidents. Sleep plays an important role in the physical and emotional development of adolescents, who are going through a period of intense learning and differentiation. Paradoxically, however, nowadays, several factors contribute to the poor sleep of adolescents because of the social pressures that boost their activities such as excessive use of computer and phone, new affective relationships, parties, etc. All of these factors determine a reduction of nighttime sleep and consequent daytime sleepiness. The objective of the present study was to present some issues related to the physiology of the sleep-wake cycle and its implications in the daily life of teenagers, in an attempt to stimulate the thinking of professionals about the important aspects related to quality and quantity of sleep.


Keywords: Sleep; Adolescent

## Introduction

Sleep is an important physiological condition characterized by a reversible behavioral state with changes in consciousness level and in the responsiveness to internal and external stimuli. It is an active process that involves complex mechanisms in various regions of the central nervous system and is related to various development and maturation processes in the first years of life, such as homeostatic functions for energy conservation, neurotransmitter replacement, synapse and receptor remodeling, modulation of receptor sensitivity, and memory consolidation [1].

Sleeping is a basic physiological need for the body to rest and restore its energy. Sleeping improves memory recall and helps regulate the metabolism in the body and reduces mental fatigue [2]. During sleeping the brain resets itself, removes toxic products accumulated throughout the day, and repairs brain cell damage caused by free radicals [3-5].

Human beings spend about a third of your life sleeping and, although there is no unanimity about the potential functions of sleep, there are three theories that permit a better understanding of the role of this physiological state for the organism. The so-called Restorative Theory proposes that various metabolic processes occur during sleep which serves to prepare the brain and the body for the next period. The Adaptative Theory proposes that sleep can increase animal survival due to immobilization during the periods of greatest danger of the day, reducing the possibilities of their falling prey to other animals. Finally, the Energy Conservation Theory argues that sleep provides a period during which the metabolism is reduced in order to save energy $[6,7]$.

Sleep plays a fundamental role in the somatic, psychological and cognitive domains related to electrophysiological, neurochemical and anatomo-functional changes in the brain. To this end, it is controlled by homeostatic and chronobiological mechanisms. While the former determines its necessity, a circadian rhythm regulates its frequency, with the wake-sleep cycle being determined by the circadian clock [8].

The biological rhythm is important for maintaining a chronogram of hours for sleeping, studying, working, engaging in leisure activities and taking meals. Sleep is an important factor of synchronization between internal variations and environmental cycles. In humans, the best example of this synchronization is the sleep/wake cycle. According to their nature and social organization, human beings are active during the day and their physical functions are mainly oriented towards daytime activities and are related to the biological rhythm [9].

In view of the intense relation existing between the quality of sleep and wakefulness, one of the most immediate results of sleep of poor quality is low performance on the subsequent day, provoking damage during wakefulness such as somnolence, mood swings, anxiety, low self-esteem, slow reasoning, loss of memory, poor school and personal performance, and predisposition to accidents [10,11].

## Physiology of the sleep-wake cycle

The sleep-wake cycle is a circadian rhythm which, under natural conditions, oscillates along a period of 24 h . Alternance of light and dark periods, school hours, work shifts, leisure and family activities are some of the exogenous factors that synchronize this cycle. In addition to this synchronization regulated by the environment, the sleep-wake cycle is also regulated endogenously by a neural structure located in the hypothalamus-the suprachiasmatic nucleus-considered to be the
circadian biological clock for mammals. Reasons that prevent an individual from sleeping at his habitual time considerably affect the psychosomatic equilibrium, and the adverse effects of the interruption of the circadian rhythm such as night work, for example, have negative repercussions on the wakefulness period [12].

Many metabolic processes necessary for the good functioning of the organism occur during sleep. The beginning of sleep is considered to be related to the activity of endogenous metabolic factors produced during the wakefulness period. During this period, the metabolites accumulate in the brain as a consequence of the increased neuronal activity on the structures that promote wakefulness and of the overall increase of neuronal activity [1]. The slow accumulation of these metabolic factors increases the inertia of sleep and, when their level becomes critically high the brain responds by reducing the neuronal activities in the regions that promote wakefulness. The reduction of the neuronal activity determines the end of wakefulness and initiates the passive process of sleep. In turn, during sleep the metabolites reach a critically low level which causes disinhibition of neuronal activity in the brain regions that promote wakefulness, increasing the activity in the brain regions that promote behavioral states of wakefulness [13]. Over the last few years, some metabolic factors that initiate sleep have been identified, such as adenosine, the neuroinhibitory amino acids GABA and glycine, prostaglandin D2, cytokines, the alpha melanocyte stimulating hormone, somatostatin, and tumor necrosis factor alpha [14-16].

Two behavioral states are identified during sleep: synchronized or non-rapid eye movement (NREM) and desynchronized, paradoxical or rapid eye movement (REM). NREM sleep is characterized by synchronous cerebral electrical activity with its own elements and is divided into stages I, II and III which represent the progressive depth of sleep. During NREM sleep there is also a reduction of the activity of the autonomous sympathetic nervous system and an increase of parasympathetic tonus to higher levels than during wakefulness. Respiratory and heart rates, cardiac output, arterial pressure, pupil diameter, intestinal movements and galvanic skin resistance do not undergo abrupt changes. REM sleep, in turn, is characterized by the so-called electroencephalographic desynchronization, which manifests when rapid eye movements occur, as well as by brief muscle contractions of the limbs and muscular atonia, causing skeletal muscle to become paralyzed. There is instability of the autonomic sympathetic nervous system, with variations in heart and respiratory rates, cardiac output, arterial pressure, coronary and cerebral blood flow, pupil size and penile erection. In contrast, the tonus of the parasympathetic system is essentially the same in NREM sleep [17-19].

Under normal conditions, the sleep stages occur in a cyclic manner during the night (ultradian cycle), starting with the succession of one to three NREM stages [20,21].

Melatonin synchronizes the sleep-wake rhythm and various biological rhythms such as body temperature, corticotropin releasing hormone and adrenocorticotropic hormone in addition to cortisol, which show cyclic changes during the 24 h . Thus, when sleep habits become disorganized, changes in the production of these hormone may occur, with the respective clinical manifestations associated with them [ $10,22,23]$.

Two biological mechanisms acting on sleep regulation are recognized in association with environmental factors. One is called circadian (C process) and the other homeostatic (S process). The homeostatic process is related to the increase in the tendency to sleep
during the day and is subjected to the effects of lack of sleep. On the other hand, the circadian process is responsible for the predisposition to sleep during the dark phase of the day. In adolescence there is less inhibition of the secretion of melatonin at the beginning of the light phase of the day and a slower accumulation of the tendency to sleep during the day, which may lead to a phase delay, more commonly observed in more advanced pubertal periods [24]. Thus, the biological and behavioral changes that occur during adolescence lead to a phase delay that, according to the social and school context, will reflect on a reduction of hours of sleep and an increase in daytime somnolence [25,26].

Stress is a factor that strongly influences sleep because it involves an increased secretion of circulating cortisol which may lead to suppression of REM sleep, an increase of superficial sleep, and difficulty in falling asleep and staying asleep. Considering the current life conditions associated with a large amount of appointments and tasks to be performed, adolescents are subjected to one of the main effects of stress, i.e., the reduction of sleep quality and quantity [27].

Lack of sleep provokes a reduction of metabolism in frontal brain regions (responsible for the planning and execution of tasks) and in the cerebellum (the center of motor coordination), leading to difficulties in accumulating knowledge, to mood changes, and to impairment of creativity, attention, memory and equilibrium [28].

## Adolescents and Sleep

Adolescence is characterized as a phase of life during which important biopsychosocial, cognitive and behavioral changes occur, leading to repercussions on various homeostatic mechanisms, also regarding the pattern of the sleep-wake cycle. Among the various structural modifications that occur in the body, recent discoveries have pointed out that, at the beginning of puberty, the volume of gray mass existing in the frontal and parietal lobes reaches a peak, followed by a later decrease, and that this tissue is sensitive to the variations suffered by the organism, as observed for those related to sleep [29].

Sleep plays an important role in the physical and emotional development of adolescents, who are going through a period of intense learning and differentiations. Adolescent is a being biologically programmed to sleep and wake up later, with his brain not being in a wakefulness state for most of the morning. Paradoxically, however, nowadays various elements contribute to preventing the adolescent from sleeping adequately in view of the social pressures that diversify and increase his activities such as excessive use of electronic devices, new affective relationships, parties, etc. All of these factors cause a reduction of nighttime sleep, with consequent somnolence during the day $[11,12,30]$. Prolonged exposure to electronic devices can result in nocturnal melatonin suppression or a delay of melatonin release through increased nocturnal stimulation of the circadian system [31].

Social activities and habits in general have migrated to increasingly more nocturnal times, while classes start in the morning, leading to an important reduction of sleep hours and a persistent sleep debit throughout the week $[32,33]$. In turn, the technological era has caused marked transformations in today's life due to the introduction of television and, more recently, of microcomputers in the home. With the growth of the internet, the habit of surfing on the web for long periods of time is becoming increasingly more intense, especially among adolescents, who practically "surf" throughout the night at the expense of regular hours of sleep for a good physical and psychological development. One of the great current challenges for adolescents is to
try to keep a regular sleep-wake cycle, to fulfill social demands and to satisfy their sleep necessities [34,35].

Two major health problems can be associated with sleep deprivation. Sleep deprivation and epilepsy have a complex bidirectional relationship because lack of sleep can increase the likehood of seizure recurrence due the activating interictal activity and recurrent oxygen desaturation [36-38]. Chronic headache has also been associated with short sleep duration with increased frequency and severity acting as headache triggers [39-41].

Nowadays the main characteristics of the sleep-wake cycle of adolescents are to go to bed later, to get up early, and to present irregular and variable sleep patterns between week days and weekends, insufficient periods of sleep, and daytime somnolence. As a consequence, adolescents are quite vulnerable to sleep disorders, especially insomnia [42,43]. The estimate is that $14 \%$ to $33 \%$ of all adolescents complain about sleep problems and that $10 \%$ to $40 \%$ of middle school students have moderate or transitory sleep deprivation or insufficiency in addition to difficulties in school performance and behavior and mood disorders during the daytime [44].

The duration of nighttime sleep plays an important role in the health of adolescents, who are going through a period of intense learning and differentiation, has a significant impact on their physical and psychological well-being and, when reduced, is associated with behavioral and neurocognitive problems, especially disorders of learning and attention deficit, lower academic performance, mood swings and reduced opportunities for socialization and for the search of professional activities [34,35]. Studies have suggested that adolescents need 9 to $91 / 2 \mathrm{~h}$ of sleep per night and when they do not satisfy this need may have more daytime somnolence, attention and concentration difficulties, low school performance, as well mood swings, behavioral problems, depression, predisposition to accidents, delayed pubertal development, greater weight gain and greater use of alcohol and of psychostimulant drugs [ $11,35,36$ ].

In addition to the impact of these biological and environmental factors, social demands such as home tasks, extracurricular activities and work after school hours can significantly affect the sleep patterns of adolescents. A wide variability in the sleep-wake pattern is observed during the weeks, associated with the habit of going to sleep later during the weekend, as if to compensate for the accumulated sleep debt. This phenomenon, called oversleeping, contributes to a rupture of circadian rhythm and to a reduction of daytime wakefulness [45].

The increase of weight and height proportions is one of the main phenomena that occur during adolescence and is directly related to the action of growth hormone (GH), whose secretion is affected by various external stimuli, among them sleep. In adolescence, GH secretion mainly occurs during the hours of deep sleep, with $80 \%$ of its concentration being released in one or two pulses during stage III sleep each night [46]. Fewer hours and a poor quality of sleep may interfere with GH secretion, leading to delayed sexual maturation [47].

Adolescents show a delay during puberty, reaching maximum vespertine behavior close to 20 years of age, with girls reaching this peak before boys, a fact that can also be considered a marker of the end of adolescence [48].

The hours of sleep during adolescence have decreased with passing years. Dollman et al. [49] compared the duration of sleep in a sample of young Australians aged 10 to 15 years between 1985 and 2004 and observed a reduction of hours of sleep in the second evaluation
compared to the first. In addition, boys went to sleep later than girls in 2004, differences that were not observed in the first evaluation. Among adolescents, night attractions such as television, games and the internet cause a delay in bedtime during week days and weekends, and a later wake up time during the weekend. On week days, school schedules require early awakening, reducing the time in bed and sleep hours; however, the need for sleep does not decrease during adolescence [50]. A survey conducted on American adolescents showed that more than $60 \%$ reported that they slept less than seven hour per night during the week, i.e., much less than the $81 / 2$ to $91 / 2 \mathrm{~h}$ recommended [51].

## Conclusion

For a human being living in harmony with himself and with his environment it is necessary to observe a daily period of sleep. The physical and emotional events that occur during the second decade of life, associated with the stress of social demands, cause the adolescent to be an individual with difficulties in organizing his daily schedule, including the periods of sleep.
On this basis, it is important to be aware of the specific characteristics of the sleep-wake cycle and of the events that occur during adolescence so that it will be possible to offer guidelines about how to sleep well and to enjoy the benefits provided by sleep to the organism. In addition, when necessary, this life history should permit a complete understanding of the educational, social and professional activities.

It should be remembered that adolescents live in a challenging, dynamic and stimulating world that offers constant information competing with the guidelines that the hebiatrist may provide. At this time, it is fundamental for the professionals to establish an optimal relationship with their patients, with the prevalence of a frank and responsible dialogue.

Working with adolescents so that they will understand concepts such as "lost hours of sleep are not recovered" and that sleep periods programmed later will not compensate for a night of poor sleep is the key to the initiation of good sleep hygiene. In addition, adolescents should be advised that adequate practice of regular physical activity for more than 60 minutes, the reduction of idle time in front the computer and the television and a minimal routine for the night period can greatly contribute to a satisfactory period of sleep.

## References

1. Abbott SM, Reid KJ, Zee PC (2015) Circadian rhythm sleep-wake disorders. Psychiatr Clin North Am 38: 805-823.
2. Herculano-Houzel S (2013) Sleep in out. Science 342: 16-17.
3. Underwood E (2013) Neuroscience sleep: The brain's housekeeper? Science 342: 301.
4. Eugene AR, Masiak J (2015) The neuroprotective aspects of sleep. MEDtube Sci 3: 35-40.
5. de Bruin EJ, van Run C, Staaks J, Meijer AM (2016) Effects of sleep manipulation on cognitive functioning of adolescents: A systematic review. Sleep Med Rev.
6. Adam K, Oswald I (1977) Sleep is for tissue restoration. J R Coll Physicians Lond 11: 376-388.
7. Horne J (1988) Why we sleep. Oxford University Press, Oxford.
8. Brand S, Kirov R (2011) Sleep and its importance in adolescence and in common adolescent somatic and psychiatric conditions. Int J Gen Med 4: 425-442.
9. Hitze B, Bosy-Westphal A, Bielfeldt F, Settler U, Plachta-Danielzik S, et al. (2009) Determinants and impact of sleep duration in children and adolescents: data of the Kiel Obesity Prevention Study. Eur J Clin Nutr 63: 739-746.
10. Buxton OM, Chang AM, Spilsbury JC, Bos T, Emsellem H, et al. (2015) Sleep in the modern family: Protective family routines for child and adolescent sleep. Sleep Health 1: 15-27.
11. Foti KE, Eaton DK, Lowry R, McKnight-Ely LR (2011) Sufficient sleep, physical activity and sedentary behaviors. Am J Prev Med 41: 596-602.
12. Carskadon MA, Acebo C, Jenni OG (2004) Regulation of adolescent sleep: Implications for behavior. Ann N Y Acad Sci 1021: 276-291.
13. Roberts RE, Roberts CR, Duong HT (2009) Sleepless in adolescence: Prospective data on sleep deprivation, health and functioning. J Adolesc 32: 1045-1057.
14. John B, Bellipady SS, Bhat SU (2016) Sleep promotion program for improving sleep behaviors in adolescents: A randomized controlled pilot study. Scientifica (Cairo) 2016: 8013431.
15. Teixeira LR, Lowden A, Turte SL, Nagai R, Moreno CR, et al. (2007) Sleep and sleepiness among working and non-working high school evening students. Chronobiol Int 24: 99-113.
16. Campbell IG, Higgins LM, Trinidad JM, Richardson P, Feinberg I (2007) The increase in longitudinally measured sleepiness across adolescence is related to the maturational decline in low-frequency EEG power. Sleep 30: 1677-1687.
17. Gupta R, Bhatia MS, Chhabra V, Sharma S, Dahiya D, et al. (2008) Sleep patterns of urban school-going adolescents. Indian Pediatr 45: 183-189.
18. Liu X, Zhao Z, Jia C, Buysse DJ (2008) Sleep patterns and problems among chinese adolescents. Pediatrics 121: 1165-1173.
19. Kendzerska T, Mollayeva T, Gershon AS, Leung RS, Hawker G, et al. (2014) Untreated obstructive sleep apnea and the risk for serious longterm adverse outcomes: A systematic review. Sleep Med Rev 18: 49-59.
20. Pressman MR (2002) Primer of polysomnogram interpretation. Butterworth-Heinemann, Boston.
21. Russo PM, Bruni O, Lucidi F, Ferri R, Violani C (2007) Sleep habits and circadian preference in Italian children and adolescents. J Sleep Res 16: 163-169.
22. Carskadon MA (2011) Sleep in adolescents: the perfect storm. Pediatr Clin North Am 58: 637-647.
23. Ackermann K, Plomp R, Lao O, Middleton B, Revell VL, et al. (2013) Effect of sleep deprivation on rhythms of clock gene expression and melatonin in humans. Chronobiol Int 30: 901-909.
24. Owens J (2014) Adolescent sleep working group; Committee on adolescence. Insufficient sleep in adolescents and young adults: An update on causes and consequences. Pediatrics 134: e921-932.
25. Owens JA, Belon K, Moss P (2010) Impact of delaying school start time on adolescent sleep, mood and behavior. Arch Pediatr Adolesc Med 164: 608-614.
26. Liou YM, Liou TH, Chang LC (2010) Obesity among adolescents: sedentary leisure time and sleeping as determinants. J Adv Nurs 66: 1246-1256.
27. Kuula L, Pesonen AK, Martikainen S, Kajantie E, Lahti J, et al. (2015) Poor sleep and neurocognitive function in early adolescence. Sleep Med 16: 1207-1212.
28. Alhola P, Polo-Kantola P (2007) Sleep deprivation: Impact on cognitive performance. Neuropsychiatr Dis Treat 3: 553-567.
29. Dahl RE (2004) Adolescent brain development: A period of vulnerabilities and opportunities. Keynote address. Ann N Y Acad Sci 1021: 1-22.
30. Wolfson AR, Spaulding NL, Dandrow C, Baroni EM (2007) Middle school start times: The importance of a good night's sleep for young adolescents. Behav Sleep Med 5: 194-209.
31. Wood B, Rea MS, Plitnick B, Figueiro MG (2013) Light level and duration of exposure determine the impact of self-luminous tablets on melatonin suppression. Appl Ergon 44: 237-240.
32. Beebe DW (2016) The cumulative impact of adolescent sleep loss: Next steps. Sleep 39: 497-499.
33. Morrison DN, McGee R, Stanton WR (1992) Sleep problems in adolescence. J Am Acad Child Adolesc Psychiatry 31: 94-99.
34. Hawkins SS, Takeuchi DT (2016) Social determinants of inadequate sleep in US children and adolescents. Public Health S0033-3506: 30037-30043.
35. Johansson AE, Petrisko MA, Chasens ER (2016) Adolescent sleep and the impact of technology use before sleep on daytime function. J Pediatr Nurs.
36. Badawy RA, Curatolo JM, Newton M, Berkovic SF, Macdonell RA (2006) Sleep deprivation increases cortical excitability in epilepsy: Syndromespecific effects. Neurology 67: 1018-1022.
37. Vaughn BV, Ali I (2012) Sleep and epilepsy: Opportunities for diagnosis and treatment. Neurol Clin 30: 1249-1274.
38. Matos G, Tufik S, Scorza FA, Cavalheiro EA, Andersen ML (2013) Sleep and epilepsy: Exploring an intriguing relationship with a translational approach. Epilepsy Behav 26: 405-409.
39. Merikanto I, Lahti T, Puusniekka R, Partonen T (2013) Late bedtimes weaken school performance and predispose adolescents to health hazards. Sleep Med 14: 1105-1111.
40. de Tommaso M, Delussi M, Vecchio E, Sciruicchio V, Invitto S, et al. (2014) Sleep features and central sensitization symptoms in primary headache patients. J Headache Pain 15: 64.
41. Lin YK, Lin GY, Lee JT, Lee MS, Tsai CK, et al. (2016) Associations between sleep quality and migraine frequency: A cross-sectional casecontrol study. Medicine (Baltimore) 95: e3554.
42. Sadeh A, Dahl RE, Shahar G, Rosenblat-Stein S (2009) Sleep and the transition to adolescence: A longitudinal study. Sleep 32: 1602-1609.
43. Olds T, Maher C, Blunden S, Matricciani L (2010) Normative data on the sleep habits of Australian children and adolescents. Sleep 33: 1381-1388.
44. Lam LT, Yang L (2008) Duration of sleep and ADHD tendency among adolescents in China. J Atten Disord 11: 437-444.
45. Kurth S, Jenni OG, Riedner BA, Tononi G, Carskadon MA, et al. (2010) Characteristics of sleep slow waves in children and adolescents. Sleep 33: 475-480.
46. Watanabe M, Kikuchi H, Tanaka K, Takahashi M (2010) Association of short sleep duration with weight gain and obesity at 1 year follow-up: A large-scale prospective study. Sleep 33: 161-167.
47. Kern W, Dodt C, Born J, Fehm HL (1996) Changes in cortisol and growth hormone secretion during nocturnal sleep in the course of aging. J Gerontol A Biol Sci Med Sci 51: M3-9.
48. Volk C, Huber R (2015) Sleep to grow smart? Arch Ital Biol 153: 99-109.
49. Dollman J, Ridley K, Olds T, Lowe E (2007) Trends in the duration of school-day sleep among 10 to 15 year old South Australians between 1985 and 2004. Acta Paediatr 96: 1011-1014.
50. Roenneberg T, Kuehnle T, Pramstaller PP, Ricken J, Havel M, et al. (2004) A marker for the end of adolescence. Curr Biol 14: R1038-1039.
51. Fobian AD, Avis K, Schwebel DC (2016) Impact of media use on adolescent sleep efficiency. J Dev Behav Pediatr 37: 9-14.
