

# Sleep Disorders during Adolescence

## *Ergenlik Döneminde Uyku Bozuklukları*

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### ABSTRACT

Sleep disorders during adolescence period increase each year and adversely affect the physical and mental health of adolescents. After-school social activities and various work outside the school may cause delays in bedtime. In addition, there can be shifts in the circadian rhythm due to a number of biological changes seen in the transition to adolescence, which can result in a wide range of sleep problems, such as not being able to fall asleep at night, difficulty waking up in the morning, daytime sleepiness, sleep deprivation and deterioration in sleep quality. It is important to know the causes of sleep disorders, possible effects on physical health and mental health, and protective and risk-forming factors seen in adolescent period; to intervene in these disorders and to develop preventive measures. Preventive measures, such as increasing awareness about sleep disorders in adolescents, informing families and adolescents about the issue, and organizing school start-up times for this age group, may contribute significantly to solving this important issue, which has increased year-to-year.

**Keywords:** Adolescence period, adolescents, sleep disorders, circadian rhythm

### ÖZ

Ergenlik döneminde görülen uyku bozuklukları her geçen yıl artış göstermekte ve ergenlerin fiziksel ve ruh sağlıklarını olumsuz yönde etkilemektedir. Okul sonrası sosyal aktiviteler ve okul dışında çalışılan çeşitli işler ergenlerde gece yatış saatlerinin gecikmesine sebep olabilmektedir. Ek olarak ergenlik dönemine geçiş ile birlikte görülen birtakım biyolojik değişikliklere bağlı olarak sirkadiyen ritimde kaymalar görülebilmekte; bu durum da erken okul başlangıç saatleri ile birleşince ergenlerde gece uykuya dalamama, sabah uyanmakta zorlanma, gündüz uyku hali, toplam uyku süresinde kısalma ve uyku kalitesinde bozulma gibi çok çeşitli uyku sorunları oluşabilmektedir. Ergenlerde oldukça sık görülen uyku bozukluklarının nedenlerini, fiziksel sağlık ve ruh sağlığı üzerindeki olası etkilerini ve koruyucu ve risk oluşturu etkenleri bilmek; bu bozukluklara müdahale edilmesi ve önleyici tedbirlerin geliştirilmesi açısından önemlidir. Ergenlerde görülen uyku bozukluklarına yönelik farkındalığın artırılması, ailelerin ve ergenlerin konuyla ilgili bilgilendirilmesi ve özellikle bu yaş grubu için okul başlangıç saatlerinin düzenlenmesi gibi alınabilecek önleyici tedbirler, yıldıan yıla artış gösteren bu önemli sorunun çözülmesinde önemli katkılar sağlayacaktır.

**Anahtar sözcükler:** Ergenlik dönemi, ergenler, uyku bozuklukları, sirkadiyen ritim

## Introduction

Sleep can be defined as a temporary resting state in which a person is physically inactive and its communication with the environment is lost reversibly (Carskadon and Dement 2005). Sleep has many functions such as temperature control, nutrient metabolism, adaptation ability, neural plasticity and neuronal network integration, neuronal maturation, learning and memory functions, and regulation of the immune system (Krueger et al. 2016). Adolescence is a period in which cognitive, emotional and neurobiological changes accelerate and gains new capacities (Yurgelun-Todd 2007). In this period, significant changes are observed in the sleep structure compared to the childhood period.

In this article, it is aimed to discuss the causes of sleep disorders in adolescents in the light of biological and psychosocial factors that may predispose to the development of sleep disorders. For this purpose, publications between January 1980 and April 2022 that met the eligibility criteria for this review as a result of the research conducted in Google Academic, Science-Direct and PubMed, PsycINFO, DergiPark, TÜBİTAK/ULAKBİM-Turkish Medical Index, Turkish Psychiatry Index and Turkish Medline indexes were reviewed. It is among the aims of this article to discuss the undesirable effects of sleep disorders on adolescent health by shedding light on the possible causes of sleep disorders specific to this group and to present various recommendations for the prevention and treatment of sleep disorders.

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## Sleep Structure and Age-Related Changes

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One of the most important factors affecting the structural features of sleep is age. It is known that there is a sleep-wake cycle even in intrauterine period (Parmelee Jr et al. 1964). The sleep-wake cycle is observed regularly between the 28th and 32nd weeks of intrauterine life and then rapid eye movement (REM) and non-rapid eye movement (NREM) sleep can be distinguished after the 32nd week (Pelin and Gözükırmızı 2001).

A newborn spends approximately 2/3 of the day sleeping (Parmelee Jr et al. 1961). Transitions to sleep occur with direct REM sleep and REM sleep constitutes approximately 50% of the total sleep time (Parmelee Jr et al. 1961, Pelin and Gözükırmızı 2001). After 6 months, a biphasic sleep pattern characterized by long night sleep and short daytime sleep is observed in infants (Gillin et al. 1981). During childhood, the rate of REM sleep and total sleep duration decrease, while the rate of deep slow wave sleep increases (Pelin and Gözükırmızı 2001). At the age of 8-9, only night sleep is observed (Armstrong et al. 1994).

The developmental changes brought about by the transition from childhood to adolescence cause many changes in the sleep-wake patterns of adolescents (Yang et al. 2005). Adolescents need an average sleep time of around 9 hours (Seo et al. 2010). In this period, 40% of the total sleep time consists of deep slow wave sleep, while 25% is REM sleep (Gillin et al. 1981).

### Sleep Stages

Normal sleep is divided into non-rapid eye movement (NREM) and rapid eye movement (REM) sleep (Xiao et al. 2013). NREM sleep is divided into stages: N1, N2 and N3 (deep slow wave sleep or delta wave sleep) (Wu et al. 2015). REM sleep has tonic and phasic components (Iber et al. 2007). The phasic component is characterized by rapid eye movements, respiratory variability and dominance of sympathetic activity; tonic REM is the part in which parasympathetic activity predominates. The length of REM sleep and the intensity of eye movements increase throughout the sleep (Iber et al. 2007).

Sleep begins with NREM sleep. NREM sleep typically begins in the more superficial sleep stages such as N1 and N2 and gradually deepens towards the N3 stage, where high-amplitude delta waves are seen (Stangor and Walinga 2019). N1 is considered a transition between waking and sleeping and constitutes for 2-5% of total sleep time. There are low voltage, mixed frequency waves with 2-7 Hz frequency in N1 phase. N2 is characterized by 13-14 Hz. frequency range wave patterns sleep spindles and K complexes (Chokroverty 2017, Stangor and Walinga 2019). The N2 phase consists of 45-55% of the total sleep time. Slow wave sleep (N3 stage) is present when delta waves consist for more than 20% of the sleep EEG. Stage N3 occurs mostly in the first third of the night and 5-15% of total sleep time is N3 sleep. REM represents 20-25% of total sleep time (Chokroverty 2017, Stangor and Walinga 2019).

REM sleep follows NREM sleep and occurs 4-5 times in an 8-hour sleep period. The first REM period of the night may be less than 10 minutes, while the last REM period may exceed 60 minutes. (Šušmáková and Krakovská 2008). NREM-REM cycles range from 70-100 minutes initially to 90-120 minutes during the night. Typically, N3 sleep is more prevalent in the first third of the night, while REM sleep predominates in the last third of the night (Šušmáková and Krakovská 2008).

### Regulation of The Sleep-Wake Cycle

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Sleep is occurred by numerous factors. Two of these factors are fundamental processes involved in the regulation of sleep. It is thought that the main factor in the development of the sleep-wake cycle is the interrelationship of circadian rhythm and homeostatic regulation mechanisms (Borbély 1982).

The first of the regulatory systems is the homeostatic regulation mechanism. Basically, the longer you stay awake, the more sleep pressure occurs, that is, sleep accumulates (Achermann and Borbély 2003). It is thought that this mechanism, which increases in direct proportion to the time spent awake, functions independently of circadian rhythm (Dijk et al. 1990). The biological factors involved in the homeostatic regulation mechanism can be summarized as follows:

1. Various hormones, especially adenosine; as the time spent awake increases, it accumulates and creates homeostatic sleep pressure (Greene et al. 2017).
2. The ventrolateral preoptic nucleus (VLPO) in the hypothalamus initiates sleep; The mesopontine nucleus is responsible for the NREM-REM cycle (Steriade 2003).

3. The regulation of wakefulness, REM and NREM sleep is driven by increases and decreases in the release of neurotransmitters such as acetylcholine, serotonin, norepinephrine, and histamine (Brown et al. 2012).
  - a. All these neuromediators are released at high levels during wakefulness.
  - b. During REM sleep, the release of acetylcholine alone predominates.
  - c. During NREM, all these neuromediators are released at lower levels.

The other regulatory system is the circadian system and it creates periods in which being able to fall asleep more easily or difficulty waking up at certain times of the day (Dijk and Czeisler 1995). (Dijk and Czeisler 1995). All living creatures have adapted to the dark-light cycle by developing biological clock-like mechanisms in order to adapt to the changes that occur due to the 24-hour rotation of the world around its axis. The main biological clock in the circadian system is the suprachiasmatic nucleus (SCN) in the hypothalamus (Vitaterna et al. 2001). SCN regulates the period rhythms to approximately 24 hours (Vitaterna et al. 2001).

Light exposure stimulates the SCN by the retinohypothalamic pathway and thus the SCN initiates signals that control sleep-wake cycle (Macchi and Bruce 2004). With exposure to the first light of day, the clock in the SCN is stimulated. The continuation of the circadian rhythm, even in the absence of environmental stimuli, suggests that the biological rhythm has a predetermined rhythmity and is modified by the influence of environmental factors (Sherin et al. 1996).

In addition to the created daily cycle, the biological clock ensures the intensification of wakefulness during the day and sleep at night, but the endogenous period is slightly longer than the solar day, approximately 24.2 hours (Manthena and Zee 2006). Therefore, in individuals deprived of external factors and time cues, the endogenous period is prolonged and becomes longer than 24 hours (Dijk and Czeisler 1995). Therefore, a continuous synchronization with the 24-hour solar day is required, and daylight is the most important factor that adjusts this (Czeisler et al. 1986). In addition, physical activity, dinner time, social factors and melatonin also contribute to the regulation of the endogenous clock (Czeisler et al. 1986). The intensity of the light and the time it is applied form the basis for the regulation of the biological system (Manthena and Zee 2006). Exposure of light in the morning, especially just before the waking hour, brings the biological clock forward or shortens the 24-hour day, while exposure of light at night advances the clock or lengthens the 24-hour day (Bjorvatn and Pallesen 2009).

Melatonin is a hormone secreted by the pineal gland with a circadian rhythm and its secretion depends on the sensitivity of pinealocyte cells to light (Cajochen et al. 2003). Due to the light sensitivity of pinealocyte cells, the pineal gland is inactive throughout the day and melatonin secretion is at basal level (Cajochen et al. 2003). With the disappearance of sunlight, the inhibitory signals of the SCN on melatonin secretion decrease, then the pineal gland is stimulated and melatonin secretion begins (Zisapel 2018). As melatonin levels increase, the feeling of sleep increases to. Melatonin secretion increases at 21.00-22.00 pm, reaches a maximum level at 03:00 am and then starts to decrease at 05:00 am in the morning and decreases to basal levels after 07:00 am (Çam and Erdoğan 2003). In other words, melatonin secretion is suppressed by daylight, but it is thought that not only daylight but also bright artificial light levels (200-300 lux) may have a suppressive effect on human endogenous melatonin production (Crowley et al. 2007).

According to the exposure time, light and melatonin have almost the opposite effect on the circadian rhythm and both of them have important role in the regulation of the biological clock. So they are often preferred as treatment agents (Bjorvatn and Pallesen 2009). The key point of treatment is which period of the circadian phase where these agents are applied.

## **Sleep Structure in Adolescence Period**

Physiological biphasic sleep in preschool children turns into non-physiological monophasic sleep due to the necessity of adapting to school hours with the coming of school age (Bülbül et al. 2010). In other words, adolescents' sleep patterns become a night's sleep every 24 hours. The ideal duration of night's sleep is between 8-10 hours for adolescents (Hirshkowitz et al. 2015). However, studies have showed that more than half of adolescents report less than 7 hours of sleep a day, and they tend to sleep less as they get older (Keyes et al. 2015). The frequency of adolescents reporting that they sleep less than 8 hours on school days is between 62-72% (Kansagra 2020). In addition, the fact that adolescents of the same age report lower sleep times each year raises the concern that negative consequences that may be seen due to sleep disorders may increase (Keyes et al. 2015).

The psychosocial factors that are thought to cause a tendency to sleep disorders in adolescents by delaying the onset of sleep can be summarized as follows (Carskadon, 2002).

1. Early school start times,
2. Time for social activities
3. Obligation to work after school
4. Being more tending to make their own decisions due to the autonomy brought by adolescence (such as deciding on their own bedtime)

While this delayed sleep timing can be attributed to many psychosocial factors as above, increasing evidence indicates that social factors are insufficient to explain an adolescent's delayed sleep time.

Carskadon developed a theoretical model of the delayed sleep phase syndrome in adolescence that includes homeostatic mechanisms and developmental changes in circadian rhythm. According to this model, adolescents develop a resistance to the homeostatic sleep pressure that keeps them up late at night. At the same time, their circadian phase is relatively delayed, causing them to stay awake at night and then sleep until late in the morning (Carskadon 2008). The timing of the endogenously produced circadian rhythm in adults shifts a little each day because the duration of rhythms is approximately 24.2 hours, while in adolescents this period is even longer, approximately 24.27 hours, which increases the tendency for phase delay (Carskadon 2008). Adolescents continue to show delayed circadian rhythm, even after several weeks of regular programs that allow adequate sleep. This delay continued in controlled laboratory conditions where social interaction was limited (Carskadon et al. 2004, Crowley et al. 2007). Explanations for possible causes of delayed circadian rhythm in adolescents are summarized below.

1. Changes in melatonin secretion: Changes in melatonin secretion with the adolescence period may shift the circadian rhythm forward and cause delays in the transition to sleep and difficulty in waking up in the morning (Carskadon 2002, Owens and Witmans 2004).
2. Changes in the sensitivity of the circadian rhythm to light: Studies have shown that the effect of morning light exposure on the circadian rhythm of adolescents is insufficient. It is also thought that adolescents may have an exaggerated phase delay response to exposure to light in the evening. (Hagenauer et al. 2009).
3. Secondary sexual development: It is thought that the delayed circadian phase of adolescents is associated with secondary sexual development (Carskadon et al. 2004). Some studies have provided evidence that homeostatic and circadian regulation of sleep are sensitive to gonadal hormones and they have role in phase shift in adolescents (Hagenauer et al. 2009). Some of the effects of gonadal hormones on circadian rhythm are due to modulation of the circadian rhythm in the SCN. Gonadal hormones can alter fundamental aspects of SCN physiology in terms of establishing a circadian rhythm (Roenneberg et al., 2004). Adolescent girls begin to show a delay in sleep times one year earlier than boys, due to earlier onset of puberty. (Roenneberg et al. 2004).

## **Classification of Sleep Disorders in Adolescents**

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Changes in sleep structure according to age are effective on the incidence of various sleep disorders. Due to the predominance of REM sleep in the first years of life and the partial wakefulness observed during REM sleep, disorders related to continuity of sleep are more common in infants (Türkbay and Söhmen 2001). As age progresses, the decrease in REM dominance in sleep and the increase in deep slow wave sleep duration cause NREM parasomnias to be seen more frequently in school aged children, while these disorders decrease towards adolescence. In adolescents, the increasing necessity for sleep and the time allocated to school hours and social activities make problems in terms of maintaining sleep patterns (Türkbay and Söhmen 2001, Kansagra 2020). Therefore, changes in both sleep duration and circadian rhythm of sleep are risk for dyssomnias (Carskadon 1990).

### **Insomnia**

Insomnia can be defined as a persistent deterioration in the initiation, maintenance and quality of sleep. For children and young adults, being able to fall asleep for 30 minutes or longer and waking up at night to fall asleep longer than 30 minutes are considered significant sleep disorders (Flanagan et al. 2007). Waking up early in the morning can be defined as waking up at least 30 minutes earlier than expected sleep time (Medicine 2014). Insomnia is frequently seen in adolescents as difficulty falling asleep, and its frequency varies between 7% and

40% in studies (Donskoy and Loghmanee 2018, Kansagra 2020). Psychostimulants and antidepressants, which are commonly used medications in adolescents, are associated with deterioration in sleep duration and quality (DeMartinis and Winokur 2007). Several parasomnias and sleep-related movement and respiratory disorders may also affect the sleep maintenance, leading to early-morning awakening insomnia (Barone et al. 2004). Insomnia can also occur due to circadian rhythm sleep disorders. Late-bed/Late-rise sleep-wake pattern, may cause insomnia in time (Zisapel 2001).

### **Circadian Rhythm Sleep Disorders**

Among circadian rhythm sleep disorders, delayed sleep phase syndrome is the most common type of sleep disorder in adolescents, and its prevalence is estimated to be between 7-16% (Dagan et al. 1998, Barion and Zee 2007). In patients whose main symptom is insomnia, delayed sleep phase syndrome may occur at rates as high as 16% (Regestein and Monk 1995). It is thought that the sensitivity of these individuals to light levels increases at night and decreases during the day (Aoki et al. 2001). Decreased exposure to morning light due to late awakenings affects synchronization negatively. The treatment includes compliance with sleep hygiene, morning bright light therapy, melatonin (3-6 mg/day and applied a few hours before melatonin release (4-6 hours before the onset of sleep)) and chronotherapy (Morgenthaler et al. 2007).

### **Hypersomnia**

The main problem in this group of disorders are inability to stay awake during the day and excessive daytime sleepiness. Excessive daytime sleepiness may develop due to circadian rhythm sleep disorders, insufficient sleep at night and sleep related respiratory disorders (Sowa 2016). Other than these, causes related to narcolepsy, idiopathic hypersomnia, Kleine Levin Syndrome, numerous psychiatric disorders, medication or substance use are called central disorders of hypersomnolence (Khan and Trotti 2015). Although the age of onset of narcolepsy is between 20-30, it can start at any age. Dauvilliers et al. showed that the narcolepsy had two peaks around the ages of 15 and 35 (Dauvilliers et al. 2001). Considering the case series, it is seen that 1 in 3 cases in adults begin before the age of 15 (Nevsimalova et al. 2009). Kleine-Levin Syndrome is a rare disorder with recurrent periods of excessive sleepiness and cognitive and behavioral symptoms. The first episode is often seen in male adolescents (Arnulf 2015).

In hypersomnia, the first thing to be evaluated is whether the person gets an adequate night's sleep. In addition to insufficient sleep in adolescents due to school hours and working in part-time jobs and take caffeine, which is frequently consumed to cope with daytime sleepiness, may also contribute to the disruption of night sleep and the continuation of the negative cycle. In this case, the first thing to do is to intervene in sleep hygiene and treat other disorders, if any (Boulos and Murray 2010).

### **Parasomnias**

Parasomnias are sleep disorders characterized by abnormal movements, behaviors, emotions, dreams, perceptions and impaired autonomic nervous system functions that occur during different sleep stages. The common pathology in these disorders is the deterioration in the stability of sleep (Guilleminault et al. 2006). Sleepwalking (somnambulism) and sleep terror, which are among the NREM-related parasomnias, usually start in childhood and disappear spontaneously during adolescence (Mahowald and Schenck 1996, Castelnovo et al. 2018). Confusional awakening can also be seen in adolescents, even though its frequency decreases compared to childhood, and its prevalence is around 3-4% (Ohayon and Priest 1999). REM sleep behavior disorder, one of the REM-related parasomnias, is generally seen in men over 50 years of age and is very rare in adolescence (Mahowald and Schenck 2010). The age of onset of isolated sleep paralysis is considered to be 14-17 years and the lifetime prevalence of one or more isolated sleep paralysis is between 15-40% (Ohayon and Priest 1999). Nightmare Disorder, is quite common in childhood, with prevalence of up to 75%. Its prevalence in adolescence is around 6-7%. (Krakow et al. 2001).

### **Sleep Related Respiratory Disorders**

Obstructive sleep apnea is the most common disorder among sleep related respiratory disorders, and its prevalence in children is between 1-4% (Marcus et al. 2013). The most important risk factors in children are obesity and adenotonsillar growth (Marcus et al. 2013) and It is less common in adolescence (Marcus et al. 2013). There are many neurobehavioral consequences of sleep related respiratory disorders, such as; behavioral dysregulation, increased impulsivity, hyperactivity, aggression, memory deficits (Chervin et al. 2006).

## **Sleep Problems in Common Psychiatric Disorders Among Adolescents**

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### **Attention Deficit Hyperactivity Disorder (ADHD)**

It has been reported that up to 70% of children diagnosed with ADHD have sleep problems (Sung et al. 2008). Various sleep problems such as resistance to bedtime, not being able to fall asleep at night, interruptions of night sleep, difficulty waking up in the morning and daytime sleepiness have been observed in adolescents with ADHD (Wajszilber et al. 2018). Comorbid psychiatric disorders, medical diseases, stimulants used or the diagnosis itself may cause sleep problems in adolescents with ADHD (Cortese et al. 2009). Before treatment, the cause of sleep problem should be determined first. If sleep problem is associated with stimulants first thing to do; the time and dose of the drug should be reconsidered and if necessary a reduction in dose should be tried. But the effectiveness of the treatment decreases with the reduction of the dose, it is recommended to continue the stimulant treatment at the old dose and add melatonin (Cortese et al. 2013, Mukaddes 2015). The majority of sleep-related problems in children with ADHD are delayed sleep onset and the recommended treatment is 3-6 mg/g melatonin half an hour before bedtime (Weiss and Salpekar 2010).

### **Autism Spectrum Disorders (ASD)**

Sleep problems are quite common in children diagnosed with ASD and rates of up to 80% are reported in the literature (Mannion and Leader 2014). Difficulty in initiating and maintaining sleep are the most common sleep problems (Cortesi et al. 2010). Individuals diagnosed with ASD or mental retardation may experience difficulties in creating a circadian rhythm due to problems in interpreting the social and visual cues necessary for sleep organization (Richdale 1999). As a result of sleep problems, functionality may be impaired due to the exacerbation of the classic symptoms of autism (Schreck et al. 2004). Treatment recommendations are melatonin and behavioral interventions (Mukaddes 2015). Melatonin is known to be particularly effective in adolescents who report difficulty falling asleep (Gringras et al. 2012). Risperidone can also be preferred in the treatment of sleep disorders, especially in adolescents with irritability (Johnson and Malow 2008).

### **Mood and Anxiety Disorders**

There is a bidirectional relationship between sleep problems and mood and anxiety disorders. While sleep disturbance rates of up to 80% are reported in adolescents diagnosed with depressive disorder, it is known that sleep disorders increase the severity and the risk of depression (Alvaro et al. 2013; Inkelis et al. 2021). The presence of persistent sleep problems in the treatment of depressive disorder has been associated with low treatment response (Manglick et al. 2013). A similar situation is also valid for anxiety disorders, and it has been reported that 46% of children with anxiety disorder experience sleep problems continuously and 83% periodically. (Alfano et al. 2006). It is reported that approximately 2/3 of children with sleep disorders show various symptoms of anxiety disorder (Ivanenko et al. 2004). In medical treatment, the use of antidepressants is recommended primarily for the treatment of depression and anxiety symptoms (Taylor et al. 2021) and then sleep disorders that still persist should be treated.

### **Effects of Sleep Disorders On Adolescent Health**

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There are numerous studies showing that sleep disorders in adolescents are associated with negative outcomes in various areas such as physical and psychosocial health, school performance, and risk-taking behavior (Stores, 2022). The results of these studies are given in Table 1.

### **Risk Factors and Protective Factors for Sleep Disorder in Adolescents**

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In the literature, there are several studies investigating various sleep disorders such as decreased sleep duration, difficulty falling asleep, frequent sleep interruptions and decreased sleep quality in adolescents. Table 2 presents the risk factors and protective factors for sleep disorders in adolescents.

#### **Risk Factors**

Studies have reported that sleep disorders are more common in adolescents with conflicted family relationships, domestic violence behavior and parents with alcohol-cigarette use (Carskadon et al. 2004, Giannotti and Cortesi 2009, Auvinen et al. 2010, Bülbül. et al. 2010). It is thought that the sleep quality of adolescents who report that they have social and familial problems is also impaired (Senol et al. 2012). In the meta-analysis of studies

examining sleep duration in adolescents, it is seen that there is a relationship between negative family environment and decrease in sleep duration (Bartel et al. 2015).

<b>Table 1. Effects of sleep disorders on adolescent health</b>		
Physical Health	Neck/shoulders/abdomen and waist pain	Auvinen et al., 2010, Luntamo et al. 2012
	Impairment of cardiovascular functions	Shaikh et al. 2010, Martinez-Gomez et al. 2011, Matthews et al. 2012, Narang et al. 2012, DelRosso et al. 2020
	Obesity, Insulin Resistance and type-II diabetes	Gupta et al. 2002, Spiegel et al. 2005, Calamaro et al. 2010, Garaulet et al. 2011, Al-Hazzaa et al. 2012, Geva et al. 2020
	Menstruation problems	Johnson et al. 2006
Psychosocial Health	Impairment in subjective well-being	Lemola et al. 2013
	Decreased self-esteem	Kanieta et al. 2009
	Depression and anxiety disorders	Fredriksen et al. 2004
	Aggression, attention deficit, and behavior problems	Liu and Zhou 2002, Coulombe et al. 2010
	Increase in risky behaviors such as smoking, alcohol, substance use, dangerous driving, safety violations, impulsivity, suicidal behavior	Patten et al. 2000, Roberts et al. 2001, Johnson and Breslau 2001, Lam and Yang 2007, Goldstein et al. 2008, Catrett and Gaultney 2009, Mak et al. 2010, Pasch et al. 2010
School Performance	Decreases in complex cognitive functions such as verbal fluency, creativity, computational speed, and abstract problem solving	Roberts et al. 2009
	Impaired attention and memory,	Kuula et al. 2015

Sleep quality (Şenol et al. 2012) and sleep duration (Bartel et al. 2015) were found to be significantly lower in adolescents who excessive caffeine consumption during the day. Caffeine use is associated with short sleep duration. Caffeine use may be associated with the fact that adolescents who excessive caffeine consumption during the day are more awake and unable to sleep at night (Calamaro et al. 2009) or that adolescents who sleep less at night are more tired and therefore consume more caffeinated beverages to struggle with tiredness (Bryant Ludden and Wolfson 2010). It has been reported that adolescents who smoke have more difficulty in maintaining sleep than non-smokers (Phillips and Danner 1995). In addition, it is thought that numerous physical diseases and mental disorders are also associated with sleep disorders (Şenol et al. 2012).

<b>Risk factors</b>	<b>Protective Factors</b>
Conflict family environment	Presence of regular pre-sleep rituals
Family members with alcohol-smoking use	Regular exercise habit
Excessive caffeine consumption	High education level of parents
Cigarette consumption	High parental income level
Physical and mental illness	
Use of technological devices	

The use of technological devices is another important risk factor associated with sleep disorders (Windiani et al. 2021). In a study by Owens et al. (1999), it was shown that the habits of watching television in adolescents are associated with delay in bedtime, not being able to fall asleep at night, and short sleep duration (1999). In a study by Bülbül et al., the presence of various technological devices, especially mobile phones, in the bedroom of adolescents was found to be associated with sleep disorders (2010). Exposure to light in before bed time due to computer or phone use is associated with late bedtime and therefore a decrease in sleep time (Bartel et al. 2015). It is known that artificial light in the blue spectrum, especially emitted from technological devices, poses a risk for sleep disorders (Shechter et al. 2020). In addition to light exposure, physical, emotional or psychological stimulation that may occur while using technological devices can also cause adolescents to feel less sleepy and go to bed later (Cain and Gradisar 2010, Short et al. 2013).

### **Protective Factors**

Sleep hygiene; it includes many factors such as behavioral, physiological and emotional arousal before bedtime, sleep environment and sleep stability (Storfer-Isser et al. 2013). Having low arousal before bedtime, having a consistent sleep pattern and a good sleep environment are essential for sleep hygiene. Adolescents with regular

pre-sleep rituals go to bed earlier, not being able to fall asleep more comfortably, and therefore show less sleep problems (Bülbül et al., 2010, Bartel et al. 2015).

High educational level of parents and high family income are also among the protective factors for sleep disorders (Newton et al. 2020). Regular exercise habits in adolescent were associated with earlier sleep hours and less sleep problems; however, the timing of exercise also affects sleep (Bartel et al. 2015). While exercising within one or two hours before going to bed is not suitable for good sleep hygiene, it may cause trouble falling asleep. Exercising at least 4 hours before bedtime or in the morning may be beneficial for sleep duration (Storfer-Isser et al., 2013). Similarly, the duration and frequency of exercise are also important in terms of the effects of exercise on sleep (Foti et al. 2011). It has been reported that adolescents who exercise for 60 minutes a day, four or more days a week, have less sleep problems than adolescents who do not exercise any day during this period (Foti et al. 2011).

## Recommendations for The Treatment of Sleep Disorders

We have some suggestions to reduce sleep disorders and increase sleep duration and quality in adolescents. These are summarized in Table 3. .

<b>Table 3. Recommendations for treatment of sleep disorders among adolescents</b>
Detection and treatment of factors that accompany or may cause insomnia
Providing sleep hygiene training to adolescents
Informing adolescents so that they can make informed choices
Ensuring that adolescents avoid activities that may cause pre-sleep exposure to light and may be stimulating
Ensuring that they are exposed to morning sunlight
Supporting pre-sleep rituals and relaxing activities
Getting the habit of sports
Establishing policies to delay school start times for adolescents
Reducing the activities (homework etc.) of schools that will last until late in the evening
Cognitive-behavioral treatments
Pharmacological treatments

Pharmacological treatment of sleep disorders in adolescents varies according to the type of sleep disorder and possible underlying causes. In the presence of additional psychiatric diagnosis, determining the underlying cause and providing treatment for it is a priority. Recommendations for the treatment of sleep disorders in common disorders in adolescents are discussed in the relevant section and will not be repeated here.

Especially in recent years, the use of melatonin in the treatment of insomnia in adolescents has become widespread (Taylor et al. 2021). Considering that insomnia seen in adolescents is associated with circadian rhythm problems, the role of melatonin in treatment is increasing day by day. The use of melatonin at doses of 5 mg/g in adolescents is thought to be safe and effective (Pavkovic and Kothare 2022). Possible side effects of melatonin include daytime sleepiness, restlessness, depressive symptoms, and headache (Chase and Gidal 1997, Pavkovic and Kothare 2022).

In the treatment of insomnia, should be careful because there is not enough data in the literature regarding the use of other sedative/hypnotic drugs in adolescents. Other than melatonin, sedative antidepressant drugs such as mirtazapine, trazodone, and antihistamines can be used (Chhangani et al. 2011). Alpha-2 receptor agonists can also be used off-label in the treatment of sleep disorders (Pavkovic and Kothare 2022). The use of benzodiazepines and Z-drugs such as zopiclone is not recommended due to the lack of sufficient data in terms of efficacy and safety (Chhangani et al. 2011).

The use of stimulant drugs such as methylphenidate is at the forefront in the treatment of narcolepsy and hypersomnia (Peterson and Husain 2008). In parasomnias that can be seen in adolescence, medical treatment is not recommended at the first stage. In cases where these disorders affect functionality considerably and cause intense distress, the use of clonazepam and tricyclic antidepressants may be considered by paying attention to possible side effects (Bruni et al. 2004).

## Conclusion

During adolescence period, both biological and psychosocial factors increase the risk of developing sleep disorders. While sleep disorders are associated with various mental and physical disorders in adolescents, the risk of developing sleep disorders is also higher in adolescents who already have psychiatric disorders. Paying attention to this bidirectional relationship is important to identify possible underlying causes. Sleep problems

in adolescents are getting more serious from year to year. It is expected that this review will raise awareness on the subject by drawing attention to the problems mentioned.

## References

- AASM (2014) The International Classification of Sleep Disorders:(ICSD-3). Darien, IL, American Academy of Sleep Medicine.
- Achermann P, Borbély AA (2003) Mathematical models of sleep regulation. *Front Biosci*, 8:s683-s693.
- Alfano CA, Beidel DC, Turner SM, Lewin DS (2006) Preliminary evidence for sleep complaints among children referred for anxiety. *Sleep Med*, 7:467-473.
- Al-Hazzaa HM, Msaiger AO, Abahussain NA, Al-Sobayel HI, Qahwaji DM (2012) Prevalence of short sleep duration and its association with obesity among adolescents 15- to 19-year olds: A cross-sectional study from three major cities in Saudi Arabia. *Ann Thorac Med*, 7:133-139.
- Alvaro PK, Roberts RM, Harris JK (2013) A systematic review assessing bidirectionality between sleep disturbances, anxiety, and depression. *Sleep*, 36:1059-1068.
- Aoki H, Ozeki Y, Yamada N (2001) Hypersensitivity of melatonin suppression in response to light in patients with delayed sleep phase syndrome. *Chronobiol Int*, 18:263-271.
- Armstrong KL, Quinn RA, Dadds MR (1994) The sleep patterns of normal children. *Med J Aust*, 161:202-206.
- Arnulf I (2015) Kleine-Levin Syndrome. *Sleep Med Clin*, 10:151-161.
- Auvinen JP, Tammelin TH, Taimela SP, Zitting PJ, Järvelin MR, Taanila AM et al. (2010) Is insufficient quantity and quality of sleep a risk factor for neck, shoulder and low back pain? A longitudinal study among adolescents. *Eur Spine J*, 19:641-649.
- Barion A, Zee PC (2007) A clinical approach to circadian rhythm sleep disorders. *Sleep Med*, 8:566-577.
- Barone P, Amboni M, Vitale C, Bonavita V (2004) Treatment of nocturnal disturbances and excessive daytime sleepiness in Parkinson's disease. *Neurology*, 63(suppl 3):S35-S38.
- Bartel KA, Gradisar M, Williamson P (2014) Protective and risk factors for adolescent sleep: a meta-analytic review. *Sleep Med Rev*, 21:72-85.
- Bjorvatn B, Pallesen S (2008) A practical approach to circadian rhythm sleep disorders. *Sleep Med Rev*, 13:47-60.
- Borbély AA (1982) A two process model of sleep regulation. *Hum Neurobiol*, 1:195-204.
- Boulos MI, Murray BJ (2010) Current evaluation and management of excessive daytime sleepiness. *Can J Neurol Sci*, 37:167-176.
- Brown RE, Basheer R, McKenna JT, Strecker RE, McCarley RW (2012) Control of sleep and wakefulness. *Physiol Rev*, 92:1087-1187.
- Bruni O, Ferri R, Miano S, Verrillo E (2004) L -5-Hydroxytryptophan treatment of sleep terrors in children. *Eur J Pediatr*, 163:402-407.
- Bryant Ludden A, Wolfson AR (2010) Understanding adolescent caffeine use: connecting use patterns with expectancies, reasons, and sleep. *Health Educ Behav*, 37:330-342.
- Bülbül S, Kurt G, Ünlü E, Kırılı E (2010) Adolesanlarda uyku sorunları ve etkileyen faktörler. *Çocuk Sağlığı ve Hastalıkları Dergisi*, 53:204-210.
- Cain N, Gradisar M (2010) Electronic media use and sleep in school-aged children and adolescents: A review. *Sleep Med*, 11:735-742.
- Cajochen C, Kräuchi K, Wirz-Justice A (2003) Role of melatonin in the regulation of human circadian rhythms and sleep. *J Neuroendocrinol*, 15:432-437.
- Calamaro CJ, Mason TB, Ratcliffe SJ (2009) Adolescents living the 24/7 lifestyle: effects of caffeine and technology on sleep duration and daytime functioning. *Pediatrics*, 123:1005-1010.
- Calamaro CJ, Park S, Mason TB, Marcus CL, Weaver TE, Pack A et al. (2010) Shortened sleep duration does not predict obesity in adolescents. *J Sleep Res*, 19:559-566.
- Çam A, Erdoğan MF (2003) Melatonin. *Ankara Üniversitesi Tıp Fakültesi Mecmuası*, 56:103-112.
- Carskadon MA (1990) Patterns of sleep and sleepiness in adolescents. *Pediatrician*, 17:5-12.
- Carskadon MA (2008) *Sleep in Children*, 2nd ed. Boca Raton, CRC Press.
- Carskadon MA, Acebo C (2002) Regulation of sleepiness in adolescents: update, insights, and speculation. *Sleep*, 25:606-614.
- Carskadon MA, Acebo C, Jenni OG (2004) Regulation of adolescent sleep: implications for behavior. *Ann N Y Acad Sci*, 1021:276-291.
- Carskadon MA, Dement WC (2011) Monitoring and staging human sleep. In *Principles and Practice of Sleep Medicine*, 5th ed. (Eds MH Kryger, T Roth, WC Dement ):16-26. St. Louis, Elsevier Saunders.
- Castelnovo A, Lopez R, Proserpio P, Nobili L, Dauvilliers Y (2018) NREM sleep parasomnias as disorders of sleep-state dissociation. *Nat Rev Neurol*, 14:470-481.
- Catrett CD, Gaultney JF (2009) Possible insomnia predicts some risky behaviors among adolescents when controlling for depressive symptoms. *J Genet Psychol*, 170:287-309.

- Chase JE, Gidal BE (1997) Melatonin: therapeutic use in sleep disorders. *Ann Pharmacother*, 31:1218-1226.
- Chervin RD, Ruzicka DL, Giordani BJ, Weatherly RA, Dillon JE, Hodges EK et al. (2006) Sleep-disordered breathing, behavior, and cognition in children before and after adenotonsillectomy. *Pediatrics*, 117:769-778.
- Chhangani B, Greydanus DE, Patel DR, Feucht C (2011) Pharmacology of sleep disorders in children and adolescents. *Pediatr Clin North Am*, 58:273-291.
- Chokroverty S (2017) Overview of normal sleep. In *Sleep Disorders Medicine*, 1st ed. (Eds Chokroverty S):5-27. New York, Springer.
- Cortese S, Faraone SV, Konofal E, Lecendreux M (2009) Sleep in children with attention-deficit/hyperactivity disorder: meta-analysis of subjective and objective studies. *J Am Acad Child Adolesc Psychiatry*, 48:894-908.
- Cortese S, Holtmann M, Banaschewski T, Buitelaar J, Coghill D, Danckaerts M et al. (2013) Practitioner review: current best practice in the management of adverse events during treatment with ADHD medications in children and adolescents. *J Child Psychol Psychiatry*, 54:227-246.
- Cortesi F, Giannotti F, Ivanenko A, Johnson K (2010) Sleep in children with autistic spectrum disorder. *Sleep Med*, 11:659-664.
- Coulombe JA, Reid GJ, Boyle MH, Racine Y (2011) Sleep problems, tiredness, and psychological symptoms among healthy adolescents. *J Pediatr Psychol*, 36:25-35.
- Crowley SJ, Acebo C, Carskadon MA (2006) Sleep, circadian rhythms, and delayed phase in adolescence. *Sleep Med*, 8:602-612.
- Czeisler CA, Allan JS, Strogatz SH, Ronda JM, Sánchez R, Ríos CD et al. (1986) Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. *Science*, 233:667-671.
- Dagan Y, Stein D, Steinbock M, Yovel I, Hallis D (1998) Frequency of delayed sleep phase syndrome among hospitalized adolescent psychiatric patients. *J Psychosom Res*, 45:15-20.
- Dauvilliers Y, Montplaisir J, Molinari N, Carlander B, Ondze B, Besset A et al. (2001) Age at onset of narcolepsy in two large populations of patients in France and Quebec. *Neurology*, 57:2029-2033.
- DelRosso LM, Mogavero MP, Ferri R (2020) Effect of sleep disorders on blood pressure and hypertension in children. *Curr Hypertens Rep*, 22:88.
- DeMartinis NA, Winokur A (2007) Effects of psychiatric medications on sleep and sleep disorders. *CNS Neurol Disord Drug Targets*, 6:17-29.
- Dijk DJ, Brunner DP, Beersma DG, Borbély AA (1990) Electroencephalogram power density and slow wave sleep as a function of prior waking and circadian phase. *Sleep*, 13:430-440.
- Dijk DJ, Czeisler CA (1995) Contribution of the circadian pacemaker and the sleep homeostat to sleep propensity, sleep structure, electroencephalographic slow waves, and sleep spindle activity in humans. *J Neurosci*, 15:3526-3558.
- Donskoy I, Loghmanee D (2018) Insomnia in adolescence. *Med Sci*, 6:72.
- Flanagan SR, Greenwald B, Wieber S (2007) Pharmacological treatment of insomnia for individuals with brain injury. *J Head Trauma Rehabil*, 22:67-70.
- Foti KE, Eaton DK, Lowry R, McKnight-Ely LR (2011) Sufficient sleep, physical activity, and sedentary behaviors. *Am J Prev Med*, 41:596-602.
- Fredriksen K, Rhodes J, Reddy R, Way N (2004) Sleepless in Chicago: tracking the effects of adolescent sleep loss during the middle school years. *Child Dev*, 75:84-95.
- Garaulet M, Ortega FB, Ruiz JR, Rey-López JP, Béghin L, Manios Y et al. (2011) Short sleep duration is associated with increased obesity markers in European adolescents: effect of physical activity and dietary habits. The HELENA study. *Int J Obes*, 35:1308-1317.
- Geva N, Pinhas-Hamiel O, Frenkel H, Shina A, Derazne E, Tzur D et al. (2020) Obesity and sleep disorders: A nationwide study of 1.3 million Israeli adolescents. *Obes Res Clin Pract*, 14:542-547.
- Giannotti F, Cortesi F (2009) Family and cultural influences on sleep development. *Child Adolesc Psychiatr Clin N Am*, 18:849-861.
- Gillin JC, Duncan WC, Murphy DL, Post RM, Wehr TA, Goodwin FK et al. (1981) Age-related changes in sleep in depressed and normal subjects. *Psychiatry Res*, 4:73-78.
- Goldstein TR, Bridge JA, Brent DA (2008) Sleep disturbance preceding completed suicide in adolescents. *J Consult Clin Psychol*, 76:84-91.
- Greene RW, Bjorness TE, Suzuki A (2017) The adenosine-mediated, neuronal-glia, homeostatic sleep response. *Curr Opin Neurobiol*, 44:236-242.
- Gringras P, Gamble C, Jones AP, Wiggs L, Williamson PR, Sutcliffe A et al. (2012) Melatonin for sleep problems in children with neurodevelopmental disorders: randomised double masked placebo controlled trial. *BMJ*, 345:e6664.
- Guilleminault C, Kirisoglu C, da Rosa AC, Lopes C, Chan A (2006) Sleepwalking, a disorder of NREM sleep instability. *Sleep Med*, 7:163-170.
- Gupta NK, Mueller WH, Chan W, Meininger JC (2002) Is obesity associated with poor sleep quality in adolescents? *Am J Hum Biol*, 14:762-768.
- Hagenauer MH, Perryman JI, Lee TM, Carskadon MA (2009) Adolescent changes in the homeostatic and circadian regulation of sleep. *Dev Neurosci*, 31:276-284.

- Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L et al. (2015) National Sleep Foundation's updated sleep duration recommendations: final report. *Sleep Health*, 1:233-243.
- Iber C, Ancoli-Israel S, Chesson AL, Quan SF (2007) The new sleep scoring manual—the evidence behind the rules. *J Clin Sleep Med*, 3:107-107.
- Inkelis SM, Ancoli-Israel S, Thomas JD, Bhattacharjee R (2021) Elevated risk of depression among adolescents presenting with sleep disorders. *J Clin Sleep Med*, 17:675-683.
- Ivanenko A, Barnes ME, Crabtree VM, Gozal D (2004) Psychiatric symptoms in children with insomnia referred to a pediatric sleep medicine center. *Sleep Med*, 5:253-259.
- Johnson EO, Breslau N (2001) Sleep problems and substance use in adolescence. *Drug Alcohol Depend*, 64:1-7.
- Johnson EO, Roth T, Schultz L, Breslau N (2006) Epidemiology of DSM-IV insomnia in adolescence: lifetime prevalence, chronicity, and an emergent gender difference. *Pediatrics*, 117:247-256.
- Johnson KP, Malow BA (2008) Sleep in children with autism spectrum disorders. *Curr Treat Options Neurol*, 10:350-359.
- Kaneita Y, Yokoyama E, Harano S, Tamaki T, Suzuki H, Munezawa T et al. (2009) Associations between sleep disturbance and mental health status: a longitudinal study of Japanese junior high school students. *Sleep Med*, 10:780-786.
- Kansagra S (2020) Sleep Disorders in Adolescents. *Pediatrics*, 145(Suppl 2):S204-S209.
- Keyes KM, Maslowsky J, Hamilton A, Schulenberg J (2015) The great sleep recession: changes in sleep duration among US adolescents, 1991-2012. *Pediatrics*, 135:460-468.
- Khan Z, Trotti LM (2015) Central disorders of hypersomnolence: focus on the narcolepsies and idiopathic hypersomnia. *Chest*, 148:262-273.
- Krakow B, Sandoval D, Schrader R, Keuhne B, McBride L, Yau CL et al. (2001) Treatment of chronic nightmares in adjudicated adolescent girls in a residential facility. *J Adolesc Health*, 29:94-100.
- Krueger JM, Frank MG, Wisor JP, Roy S (2016) Sleep function: Toward elucidating an enigma. *Sleep Med Rev*, 28:46-54.
- Kuula L, Pesonen AK, Martikainen S, Kajantie E, Lahti J, Strandberg T et al. (2015) Poor sleep and neurocognitive function in early adolescence. *Sleep Med*, 16:1207-1212.
- Lam LT, Yang L (2007) Short duration of sleep and unintentional injuries among adolescents in China. *Am J Epidemiol*, 166:1053-1058.
- Lemola S, Ledermann T, Friedman EM (2013) Variability of sleep duration is related to subjective sleep quality and subjective well-being: an actigraphy study. *PLoS One*, 8:e71292.
- Liu X, Zhou H (2002) Sleep duration, insomnia and behavioral problems among Chinese adolescents. *Psychiatry Res*, 111:75-85.
- Luntamo T, Sourander A, Rihko M, Aromaa M, Helenius H, Koskelainen M et al. (2012) Psychosocial determinants of headache, abdominal pain, and sleep problems in a community sample of Finnish adolescents. *Eur Child Adolesc Psychiatry*, 21:301-313.
- Macchi MM, Bruce JN (2004) Human pineal physiology and functional significance of melatonin. *Front Neuroendocrinol*, 25:177-195.
- Mahowald MW, Schenck CH (1996) NREM sleep parasomnias. *Neurol Clin*, 14:675-696.
- Mahowald MW, Schenck CH (2010) REM sleep parasomnias. In *Principles and Practice of Sleep Medicine*, fifth ed. (Eds MH Kryger, T Roth, WC Dement):1083-1097. Elsevier Inc.
- Mak KK, Ho SY, Thomas GN, Lo WS, Cheuk DK, Lai YK et al. (2010) Smoking and sleep disorders in Chinese adolescents. *Sleep Med*, 11:268-273.
- Manglick M, Rajaratnam SM, Taffe J, Tonge B, Melvin G (2013) Persistent sleep disturbance is associated with treatment response in adolescents with depression. *Aust N Z J Psychiatry*, 47:556-563.
- Mannion A, Leader G (2014) Sleep problems in autism spectrum disorder: A literature review. *J Autism Dev Disord*, 1:101-109.
- Manthana P, Zee PC (2006) Neurobiology of circadian rhythm sleep disorders. *Curr Neurol Neurosci Rep*, 6:163-168.
- Marcus CL, Moore RH, Rosen CL, Giordani B, Garetz SL, Taylor HG et al. (2013) A randomized trial of adenotonsillectomy for childhood sleep apnea. *N Engl J Med*, 368(25):2366-2376.
- Martinez-Gomez D, Eisenmann JC, Gomez-Martinez S, Hill EE, Zapatera B, Veiga OL et al. (2011) Duration and emerging cardiometabolic risk markers in adolescents: The AFINOS study. *Sleep Med*, 12:997-1002.
- Matthews KA, Dahl RE, Owens JF, Lee L, Hall M (2012) Sleep duration and insulin resistance in healthy black and white adolescents. *Sleep*, 35:1353-1358.
- Morgenthaler TI, Lee-Chiong T, Alessi C, Friedman L, Aurora RN, Boehlecke B et al. (2007) Practice parameters for the clinical evaluation and treatment of circadian rhythm sleep disorders: An American Academy of Sleep Medicine report. *Sleep*, 30:1445-1459.
- Mukaddes NM (2015) Yaşam Boyu Dikkat Eksikliği Hiperaktivite Bozukluğu ve Eşlik Eden Durumlar, 2. Baskı. Ankara, Nobel Tıp Kitapları.
- Narang I, Manhiot C, Davies-Shaw J, Gibson D, Chahal N, Stearne K et al. (2012) Sleep disturbance and cardiovascular risk in adolescents. *CMAJ*, 184:E913-E920.

- Nevsimalova S, Buskova J, Kemlink D, Sonka K, Skibova J (2009) Does age at the onset of narcolepsy influence the course and severity of the disease? *Sleep Med*, 10:967-972.
- Newton AT, Honaker SM, Reid GJ (2020) Risk and protective factors and processes for behavioral sleep problems among preschool and early school-aged children: A systematic review. *Sleep Med Rev*, 52:101303.
- Ohayon MM, Guilleminault C, Priest RG (1999) Night terrors, sleepwalking, and confusional arousals in the general population: their frequency and relationship to other sleep and mental disorders. *J Clin Psychiatry*, 60:268-276.
- Owens J, Maxim R, McGuinn M, Nobile C, Msall M, Alario A (1999) Television-viewing habits and sleep disturbance in school children. *Pediatrics*, 104:e27.
- Owens JA, Witmans M (2004) Sleep problems. *Curr Probl Pediatr Adolesc Health Care*, 34:154-179.
- Parmelee Jr AH, Schulz HR, Disbrow MA (1961) Sleep patterns of the newborn. *J Pediatr*, 58:241-250.
- Parmelee Jr AH, Wenner WH, Schulz HR (1964) Infant sleep patterns: from birth to 16 weeks of age. *J Pediatr*, 65:576-582.
- Pasch KE, Laska MN, Lytle LA, Moe SG (2010) Adolescent sleep, risk behaviors, and depressive symptoms: are they linked? *Am J Health Behav*, 34:237-248.
- Patten CA, Choi WS, Gillin JC, Pierce JP (2000) Depressive symptoms and cigarette smoking predict development and persistence of sleep problems in US adolescents. *Pediatrics*, 106:E23.
- Pavkovic IM, Kothare SV (2022) Pharmacologic approaches to insomnia and other sleep disorders in children. *Curr Treat Options Neurol*, 1:1-25.
- Pelin Z, Gözükırmızı E (2001) Uygunun ontogenetik özellikleri. *Türkiye Klinikleri Psikiyatri Dergisi*, 2:67-68.
- Peterson PC, Husain AM (2008) Pediatric narcolepsy. *Brain Dev*, 30:609-623.
- Phillips BA, Danner FJ (1995) Cigarette smoking and sleep disturbance. *Arch Intern Med*, 155:734-737.
- Regestein QR, Monk TH (1995) Delayed sleep phase syndrome: a review of its clinical aspects. *Am J Psychiatry*, 152:602-608.
- Richdale AL (1999) Sleep problems in autism: prevalence, cause and intervention. *Dev Med Child Neurol*, 41:60-66.
- Roberts RE, Roberts CR, Chen IG (2001) Functioning of adolescents with symptoms of disturbed sleep. *J Youth Adolesc*, 30:1-18.
- Roberts RE, Roberts CR, Duong HT (2009) Sleepless in adolescence: prospective data on sleep deprivation, health and functioning. *J Youth Adolesc*, 32:1045-1057.
- Roenneberg T, Kuehne T, Pramstaller PP, Ricken J, Havel M, Guth A et al. (2004) A marker for the end of adolescence. *Curr Biol*, 14:1038-1039.
- Şenol V, Soyuer F, Akça RP, Argün M (2012) Adolesanlarda uyku kalitesi ve etkileyen faktörler. *Kocatepe Tıp Dergisi*, 13:93-104.
- Seo WS, Sung HM, Lee JH, Koo BH, Kim MJ, Kim SY (2010) Sleep patterns and their age-related changes in elementary-school children. *Sleep Med*, 11:569-575.
- Shaikh WA, Patel M, Singh S (2010) Association of sleep duration with arterial blood pressure profile of gujarati Indian adolescents. *Indian J Community Med*, 35:125-129.
- Shechter A, Quispe KA, Mizhquiri Barbecho JS, Slater C, Falzon L (2020) Interventions to reduce short-wavelength ("blue") light exposure at night and their effects on sleep: A systematic review and meta-analysis. *Sleep Advances*, 1:zpa002.
- Sherin JE, Shiromani PJ, McCarley RW, Saper CB (1996) Activation of ventrolateral preoptic neurons during sleep. *Science*, 271:216-219.
- Short MA, Gradisar M, Gill J, Camfferman D (2013) Identifying adolescent sleep problems. *PLoS One*, 8:e75301.
- Sowa NA (2016) Idiopathic hypersomnia and hypersomnolence disorder: a systematic review of the literature. *Psychosomatics*, 57:152-164.
- Spiegel K, Knutson K, Leproult R, Tasali E, Van Cauter E (2005) Sleep loss: a novel risk factor for insulin resistance and Type 2 diabetes. *J Appl Physiol*, 99:2008-2019.
- Stangor C, Walinga J (2019). Sleeping and dreaming revitalize us for action. In *Introduction to Psychology*, 4th ed. (Eds Cummings JA, Sanders L):354-367. Saskatoon, University Of Saskatchewan Open Press.
- Steriade M (2003) The corticothalamic system in sleep. *Front Biosci*, 8:878-899.
- Stores G (2022) Aspects of sleep disorders in children and adolescents. *Dialogues Clin Neurosci*, 11:81-90.
- Storfer-Isser A, Lebourgeois MK, Harsh J, Tompsett CJ, Redline S (2013) Psychometric properties of the Adolescent Sleep Hygiene Scale. *J Sleep Res*, 22:707-716.
- Sung V, Hiscock H, Sciberras E, Efron D (2008) Sleep problems in children with attention-deficit/hyperactivity disorder: prevalence and the effect on the child and family. *Arch Pediatr Adolesc Med*, 162:336-342.
- Susmáková K, Krakovská A (2008) Discrimination ability of individual measures used in sleep stages classification. *Artif Intell Med*, 44:261-277.
- Taylor DM, Barnes TR, Young AH (2021) *The Maudsley Prescribing Guidelines in Psychiatry*, 14th ed. London, Wiley.
- Türkbay T, Söhmen T (2001) Çocuklar ve ergenlerde uyku bozuklukları. *Türkiye Klinikleri Psikiyatri Dergisi*, 2:86-90.
- Vitaterna MH, Takahashi JS, Turek FW (2001) Overview of circadian rhythms. *Alcohol Res Health*, 25:85-93.

- Wajszilber D, Santiseban JA, Gruber R (2018) Sleep disorders in patients with ADHD: impact and management challenges. *Nat Sci Sleep*, 10:453-480.
- Weiss MD, Salpekar J (2010) Sleep problems in the child with attention-deficit hyperactivity disorder: defining aetiology and appropriate treatments. *CNS Drugs*, 24:811-828.
- Windiani IGAT, Noviyani NMR, Adnyana IGANS, Murti NLSP, Soetjningsih S (2021) Prevalence of sleep disorders in adolescents and its relation with screen time during the COVID-19 pandemic era. *Open Access Maced J Med Sci*, 9:297-300.
- Wu HT, Talmon R, Lo YL (2015). Assess sleep stage by modern signal processing techniques. *IEEE Trans Biomed Eng*, 62:1159-1168.
- Xiao M, Yan H, Song J, Yang Y, Yang X (2013) Sleep stages classification based on heart rate variability and random forest. *Biomed Signal Process Control*, 8:624-633.
- Yang CK, Kim JK, Patel SR, Lee JH (2005) Age-related changes in sleep/wake patterns among Korean teenagers. *Pediatrics*, 115(Suppl 1):S250-S256.
- Yurgelun-Todd D (2007) Emotional and cognitive changes during adolescence. *Curr Opin Neurobiol*, 17:251-257.
- Zisapel N (2001) Circadian rhythm sleep disorders: pathophysiology and potential approaches to management. *CNS Drugs*, 15:311-328.
- Zisapel N (2018) New perspectives on the role of melatonin in human sleep, circadian rhythms and their regulation. *Br J Pharmacol*, 175:3190-3199.

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