

## A Confront on Sleep Disorders Linked to Addiction and Mental Health

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### Abstract

Children and adolescents who suffer from mood disorders may benefit from adequate sleep to help develop, progress, and maintain symptoms. Although most existing research on mood disorders is focused on insomnia, other sleep disorders, such as delayed sleep phase, inadequate total sleep duration, and bedtime resistance in younger children, may also contribute to mood disorders. Fortunately, behavioral sleep interventions work well for children and adolescents with sleep disorders, although no food and drug administration (FDA)-approved medications are available. These behavioral interventions are effective in improving sleep in children and adolescents with depression, and they appear to play an important role in depression treatment outcomes. Currently, there is a scarcity of literature that examines the relationship between bipolar disorders among youth and sleep, addiction to alcohol usage, and how usage of smart phones, internet addiction also affects sleep disorders. Further research is needed to examine the potential impact of sleep interventions on mood episodes in young people.

### Keywords

Sleep disorder, Mental health, Addiction

### Introduction

This paper highlights the three most prominent sleep disorders associated with depression among youth: insomnia, delayed sleep phase, and hypersomnia. In addition to nightmares and obstructive sleep apnea, bipolar disorders among youth and sleep, addiction to alcohol usage, and how usage of smart phone, internet addiction also affects sleep disorders/behaviors were discussed in this article [1].

#### Delayed sleep phase in children

The circadian system is also important in adolescence, particularly sleep disorders which affect the delayed sleep phase, which is the focus of this review. During adolescence, the circadian system shifts in favor of a delayed sleep phase, characterized by a preference for later sleep onset and offset and sometimes referred to as an evening chronotype [2, 3]. In addition to the biological shift at puberty resulting in a delayed sleep phase, social changes during adolescence often compound this shift, including less parental control, increased access to stimulating social activities (music, internet, text messaging, etc.). In addition to increased academic demands at school, early school start times, social pressures, alcohol and drug abuse, there is also an increase in alcohol and drug use during adolescence. In synergy, these social and hormonal influences can delay bedtimes, reduce sleep time, and make it more difficult to fall and stay asleep [4, 5]. In addition to having difficulty waking up and staying awake at school, adolescents also attempt

to “catch-up” on sleep on weekends, resulting in inconsistent sleep patterns and poor sleep quality. Consequently, adolescents with delayed sleep phases are more likely to experience insomnia, short sleep duration, poor sleep quality, and daytime sleepiness [6].

### Insomnia and hypersomnia in children

Insomnia has been the subject of most research on sleep disorders among youth. DSM-5 defines insomnia as difficulty initiating, maintaining, or returning to sleep after an early morning awakening at least three nights per week for three months or more. The condition must persist despite adequate sleep opportunities [7-11]. Adults and older children and adolescents may suffer from psychophysiological insomnia (also called “conditioned” insomnia). Physiological and emotional arousal related to sleep and the sleep environment are characteristic of psychological insomnia. Sleep anxiety in children with psychophysiological insomnia is frequently accompanied by maladaptive cognitions about sleep problems, amplifying their difficulties falling asleep. Children with behaviorally based insomnia also suffer from insomnia. A typical presentation of this condition is bedtime resistance, accompanied by prolonged sleep onset and/or night wakings. Bedtime resistance and prolonged nighttime wakings present a problem for many children, which often requires parental intervention, disrupting parents’ sleep as well. Insomnia caused by behavioral factors is often a result of either inaccurate or inconsistent parental limit-setting or sleep-onset associations that are maladaptive [12, 13]. Limit-setting behavioral insomnia involves verbal protests and repeated requests and demands at bedtime (“curtain calls”). Sleep deprivation caused by bedtime resistance can result in inadequate sleep, moodiness, and irritability the next day. There is no reason why children cannot experience some transient bedtime resistance or insomnia during their development [14]. It is necessary for the symptoms to occur frequently (at least three times per week) and persistently (for at least three months) and to result in significant impairment to the child, parent(s), or family member’s functioning. The prevalence of insomnia is higher among children than among adolescents [15].

Hypersomnia (or hypersomnolence) is an essential sleep disorder for youth to consider, despite not being as well-defined or thoroughly researched as insomnia and delayed sleep phase. Hypersomnia is not synonymous with excessive daytime sleepiness [16]. Due to the fact that excessive daytime sleepiness is linked with so many other conditions, idiopathic hypersomnia is less common than EDS, which is a symptom reported at a relatively high rate among both adults and children, because it is not a result of sleep deprivation, substance abuse, or other medical or psychiatric conditions [17]. Adolescence or early adulthood are commonly affected by idiopathic hypersomnia, which is chronic. Idiopathic hypersomnia patients frequently report sleep drunkenness, which is a symptom that may include excessive daytime sleepiness coupled with a normal or prolonged main sleep period greater than 10 h. Hypersomnia has been studied epidemiologically inconsistently, focusing on one of two primary symptoms: excessive sleep or excessive daytime sleepiness. There are a number of factors that predispose adolescents to excessive daytime sleep-

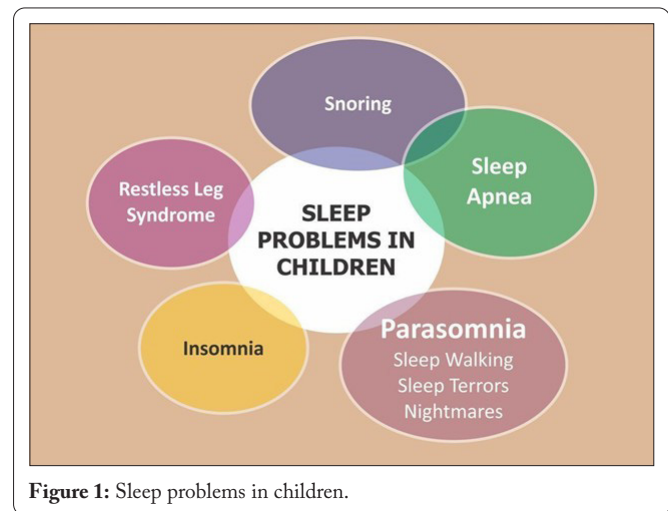


Figure 1: Sleep problems in children.

ness, including physiological changes associated with maturation, sociocultural factors, and certain pathologies. EDS seems to be both prevalent and influenced by age in children and adolescents: middle schoolers were twice as likely to suffer from EDS as preschoolers, and prevalence rates among preadolescents were 6% compared to 22% among high school seniors in pediatric clinics (Figure 1) [18-22].

### How are children with mood disorders more likely to suffer from sleep disorders?

Among children in community samples, insomnia, delayed sleep phase, and hypersomnia are fairly common, but among mood-disordered youth the prevalence rates are even higher. Major depression in adolescents is associated with insomnia [2, 23]. While fewer studies have estimated rates of evening circadian preference among depressed youth, numerous studies indicate a strong association between depression and evening circadian preference. Most of the studies suggest that sleep problems may be more common in children than adolescents, therefore less significant or indicative of a problem when they are part of a typical development trajectory [24]. There is an inextricable link between bipolar disorder and sleep disorders among children and adolescents, with a weighted average prevalence rate across subtypes. 82% of children with early-onset bipolar spectrum disorders report sleep problems related to depression, with insomnia being the most common. It is also important to note that major depression and subsyndromal depression during adolescence and prepuberty appear to precede the onset of bipolar disorder in emerging adulthood. Children’s affective development may be affected by maternal perinatal sleep quality. Despite adjusting for confounding factors, large birth research found a prospective association between prenatal insomnia symptoms and social-emotional development at 2 years of age [25]. Postnatal factors - specifically insomnia and depression symptoms - mediate the effect of perinatal insomnia on social-emotional child development, suggesting that this phenomenon is characterized by a series of mechanisms. Canan et al. [26] concluded, after reviewing the relationship between sleep quality and depression during the perinatal period, that prenatal sleep disturbance increases the risk of developing postpartum depression [26-28]. Several studies mentioned significant genetic influences in the prospective relationship between insomnia and depression. An-

other study showed that insomnia preceded depression in 69% of adolescents aged 13 to 16 who had comorbid insomnia and depression; even after adjusting for gender, race/ethnicity, and prior anxiety disorder, authors found a significant association between prior insomnia and depression onset. A bidirectional relationship exists between sleep and depression, as evidenced by the Great Smoky Mountains study. Sleep problems in childhood are associated with increased depression and anxiety symptoms in adolescents later in life, and depression in childhood is associated with a later increase in sleep problems. A representative sample of adolescents was studied by our research group<sup>1</sup> to determine which sleep parameters were associated with future depression [29]. In middle school, late bedtime was associated with depression symptoms in young adulthood. In this study and others, late bedtime appears to be an important determinant of depression as well as evening circadian preference. It is possible to detect depression as early as the perinatal period by identifying sleep problems. It is also possible that sleep problems can be used as a potential prevention intervention target for depression in youth. It is also possible that sleep disorders may signal a later development of bipolar disorder. Researchers have found that induced sleep deprivation is associated with mania or hypomania in adults. Bipolar disorder is also often thought to be preceded by sleep disturbances in childhood and adolescence. Bipolar disorder is nearly twice as likely to affect children of parents with bipolar disorder as those who sleep well or are variable sleepers. Offspring with at least one parent with bipolar disorder with disturbed sleep patterns were found to account for almost a third of the variation in psychiatric symptoms. As sleep disturbance has been linked to depression and bipolar disorder mood episodes, sleep disturbance offers an opportunity for early detection and intervention for youth at risk for mood disorders [30].

### Depression and sleep disturbances: a relationship

As reviewed above, sleep problems are associated with mood disorders in a unidirectional manner. However, there is also evidence that mood disorders are risk factors for sleep problems in the future, as well as a bidirectional relationship between the two. Several studies have shown that maternal perinatal depression may predict sleep problems among children, although it is unclear to what extent this association is mediated by concurrent maternal depression [31]. Prenatal mood disturbance was associated with offspring sleep problems at 18 and 30 months and children with clinically significant depressive symptoms during pregnancy had shorter sleep durations, longer sleep latency, and more night awakenings and were more likely to suffer from sleep disorders. Sleep disorders were more likely to occur in children whose mothers had clinically significant depressive symptoms both during pregnancy and at the time of assessment, in addition to the effects of prenatal depression [32]. Whereas adolescent sleep problems may persist beyond childhood among adolescents with maternal postnatal depression. Sleep problems do not precede mania. Patients with mood disorders and sleep problems experience severe symptoms when they are co-occurring. Sleep problems exacerbate depression symptoms [33]. A study on patients between the ages of 8 and 15 years showed a signif-

icant relationship between sleep disturbances and depression severity, as well as depressive moods, irritability, distinct sadness, psychomotor agitation, fatigue, anhedonia, inappropriate guilt, weight loss, and diurnal variations. In more than half of the samples of children and adolescents with depression disorders, insomnia symptoms were associated with an increased severity of specific depressive symptoms, such as fatigue, suicidal ideation, physical complaints, and concentration difficulties. Research on sleep and self-harm is increasingly being linked to suicidal ideation and self-harm among children and adolescents. Different study results showed a significant association between suicidal ideation and cross-sectional, national, and representation. The sleep disturbances of certain groups of adolescent suicide victims and controls were assessed using a psychological autopsy protocol. As compared to the non-suicide completers, suicide completers experienced more overall sleep disturbances during the past week and currently. As a result, youth who suffer from both sleep problems and depression are especially at risk. Suicidality and self-harm rates are higher among these youth. The severity of a patient's disease may be determined by sleep problems [34].

### The relationship between depression and anxiety and smartphone addiction

Anxiety scores and depression scores were found to be independent negative predictors of smartphone addiction, but depression scores were more powerful than anxiety scores. According to our findings, psychological traits (depression, anxiety, social phobia, and loneliness) are related to smartphone addiction in multiple studies. Several studies have identified depression as an independent positive predictor of smartphone addiction. Youth smartphone addiction was significantly reduced by mood regulation [35-38]. Researchers found that depression and anxiety scores were significantly higher among high smartphone users than low smartphone users, and that they were independent predictors of smartphone addiction severity. Survey samples showed social anxiety as a positive predictor of smartphone addiction, as well as social phobia as an independent predictor. There may not be a correlation between depression or anxiety and smartphone addiction among young university students; rather, it might apply to adults in general (Figure 2).

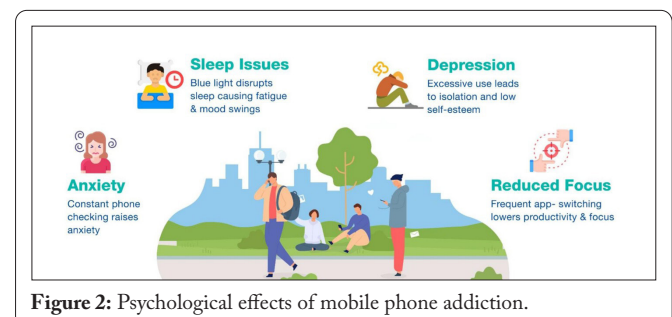


Figure 2: Psychological effects of mobile phone addiction.

### Internet addiction and sleep disorders

Globalization has been accelerated by the internet. With the internet, you can learn more, communicate more, do business, bank more, seek healthcare, and interact more socially. The number of internet users has risen dramatically in Iran,

especially among young people. Using the internet has its advantages as well as its disadvantages [39]. Both its benefits and its drawbacks can be present; both can be harmful and have negative effects on mental health.

Yang coined the term “internet addiction,” which has been embraced by psychologists, psychiatrists, therapists, scientists, and users of the internet in particular. Internet addiction can be defined as a pattern of excessive or uncontrollable preoccupations, urges, or behaviors related to the use and access of the internet. Increasing use of digital media and the internet affects sleep habits mildly, and changes in sleep quality exacerbate depression, anxiety, and stress symptoms. Using social networks more often causes medical students to be addicted to the internet, which negatively affects their sleep quality and quantity [40]. This addiction is also associated with social exclusion and other psychiatric problems, including mood disorders, anxiety, and poor impulse control. Various countries have different levels of internet addiction, but teenagers and young people are more likely to suffer from it.

Proper body function depends on adequate sleep. As smart phones and the internet have become more popular, especially among youth, using them before bedtime has become a habit which can negatively affect their quality of sleep. A study of university students was conducted to determine the relationship between internet addiction and sleep quality. According to several studies, 30% of the subjects under study were at risk for internet addiction or had it, which is higher than the results obtained from Vietnam (22%) and lower than those from countries such as Bangladesh (42%) and Jordan (38%). According to several published papers, poor sleep quality was prevalent at 15%, lower than in Iran and Bangladesh, and highest among internet addicts (62%). Although this study found lower rates of internet addiction and sleep disorders compared to other studies, the high prevalence of poor sleep quality in internet addicts in this study is in keeping with the aforementioned studies, and these results confirm that internet addiction is a contributing factor to poor sleep quality; while differences in evaluation methods, as well as cultural and social factors, may contribute to changes in internet addiction prevalence among different countries [41].

Based on a comparison between participants without internet addiction and those with various degrees of internet addiction, the global Pittsburgh Sleep Quality Index (PSQI) score was higher for participants at risk of internet addiction and internet addicts than in other participants; the result was also observed in subjective sleep quality, habitual sleep efficiency, sleep disturbances, and sleeping medication use [42]. According to this study, there was no difference between southern Taiwan and seven other countries when it came to this finding. As a result of these studies, various components of internet addiction were identified as significant predictors of subjective sleep quality, sleep disturbance, use of sleep medication, sleep duration, daytime dysfunction, and sleep latency. According to some studies, internet addiction is also associated with poor sleep quality in students, children, and teenagers. An internet addiction study indicates that students’ sleep quality is significantly affected, and an increase of one unit in internet addiction scores increases the risk of low sleep quality

by 8.7%. It is possible that the mechanism of using electronic devices in bed harms sleep by stimulating cognitive, emotional, or physiological functions [43]. It is also possible to say that exposure to intense light suppresses melatonin secretion and delays sleep, which can lead to increased consciousness and sleep disorders.

### Sleep disorders and alcoholism

Humans depend on sleep to maintain mental and physical health. There is a complex set of patterns of neurological activation and neurotransmitter release that support the different states of sleep and transitions between sleep and wakefulness. As well as affecting sleep-wake regulation, alcohol also affects a variety of neurotransmitters and brain systems. There are several ways in which alcohol affects sleep, as one might expect. The effects of alcohol on sleep can be altered by acute intoxication, but tolerance and dependence develop when repeated use occurs. Several neurotransmitter systems are altered during this process, either by modulating their release or modifying their sensitivity to stimuli. When individuals withdraw from alcohol, they may experience neurological symptoms due to a subsequent imbalance in their neurochemicals [44]. Recovery from alcohol-induced changes can occur over time, but some changes may be difficult to reverse.

A state of unconsciousness that is rapidly reversible is sleep. In fact, beliefs about substances and behaviors influencing sleep quality are based on conscious experience. These include how someone feels the next day (e.g., sleepy/rested, good/bad mood, etc.) and how they recall their wakeful consciousness around the sleep period (e.g., how long it took them to fall asleep, how long they stayed awake, etc.). Consequently, sleep onset latency (SOL), time spent awake after sleep onset, and sleep efficiency (time asleep/time in bed) based on the perception that alcohol accelerates sleep onset are more effective in developing and reinforcing beliefs than measures of rapid eye movement (REM) sleep, slow-wave sleep (SWS), or delta electroencephalogram (EEG) power during sleep. Alcohol doses that are not intoxicating have been used in several studies. Alcohol can affect the time it takes to fall asleep acutely, even at high alcohol doses. During the sleep period, the amount of time spent awake decreases early in the night and then increases late in the night when high doses of alcohol are administered before bed, causing an overall increase throughout the night [3, 45]. Therefore, somewhat paradoxically, despite alcohol’s sedative effects and the strongly held societal beliefs about its efficacy as a sleep aid, while falling asleep takes less time, the amount of sleep time decreases modestly when intoxicated alcohol is consumed before bedtime.

Those who suffer from alcohol use disorder (AUD) and their families suffer greatly from a devastating chronic disease. As a result of binge drinking to high levels of intoxication, people often develop AUD due to a compulsion to consume it, a loss of control in limiting consumption, and a shift to the “negative reinforcement side” when withdrawal begins to stave off the negative consequences. A three-stage cycle has been framed to describe this cascade of events: a) binge/intoxication, b) withdrawal/negative affect, and c) preoccupation/anticipation (“craving”). During these stages, incentive salience and habits, negative emotional states, and executive function

are dysregulated [46]. The scientific literature supports the hypothesis that AUD is a brain neurocircuitry disorder defined and perpetuated by neuroadaptations within certain motivational circuits. The pathological habits associated with binge/intoxication may be partially attributed to excessive reward circuitry activation, leading to high incentive salience for contextual cues associated with alcohol consumption [47]. Alcohol intoxication and incentive salience are mediated by circuits that converge on the nucleus accumbens and involve Gamma-aminobutyric acid (GABA), glutamate, endogenous opioids, and dopamine. As well as supporting the development of pathological habits, glutamate and dopamine act on the basal ganglia. A negative emotional state during the withdrawal/negative effect stage may involve the nucleus accumbens as well as the extended amygdala. A gain in stress reactivity may result from the loss of function of endogenous opioids, GABA, and dopamine, and the recruitment of arousal/stress factors, such as chronic renal failure (CRF), norepinephrine, hypocretin, and dynorphin. There is a violation of frontal cortical executive function and a dysregulation of substrates that regulate craving in the extended stage of preoccupation/anticipation in AUD that lasts long after abstinence. The nucleus accumbens may be dysregulated by glutamatergic projections from the frontal cortex and basolateral amygdala. Additionally, they may be associated with irreversible changes in the prefrontal cortex's structure and function, as well as neuronal connections that serve circuits that link the prefrontal cortex to other regions of the brain [48].

AUD is almost universally associated with sleep disturbances, alterations of sleep architecture, and insomnia. In the binge/intoxication stage, alcohol intoxication leads to a faster sleep onset; however, the quality of sleep afterwards is poorer than on nights when no alcohol is consumed, with a significant increase in wakefulness during sleep, especially later on. Late in the sleep cycle, REM sleep is reduced, with some limited compensation. After alcohol intoxication, SWS and delta frequency increase, however, the normal benefits of SWS may not be experienced due to a concurrent increase in alpha activity (alpha-delta sleep). Alcohol may reduce SOL and increase delta activity by acutely affecting GABAergic systems involved in sleep regulation. As well as suppressing REM sleep, alcohol also binds to GABA receptors [3, 49].

There is a great deal of variation in sleep effects during the withdrawal/negative effect stage. Within the first 30 days of abstinence, there appears to be limited recovery in sleep disturbances seen in AUD individuals, despite some decrease in SWS and limited rebound in REM sleep. Sleep variables may also be affected by withdrawal because alcohol is no longer an allosteric modulator of GABA receptors and dopamine function decreases. Acute and prolonged abstinence can both cause the overactivation of hypocretin/orexin peptides, destabilizing the boundary between arousal sleep states. Neurotransmitters and neuromodulators such as norepinephrine, CRF, and cytokines may also be involved.

Individuals who have been abstinent for a long period of time are more likely to suffer from persistent sleep problems during the extended preoccupation/anticipation stage, including extended sleep latency, increased time awake, a decrease in

SWS, a reduction in delta EEG power, an increase in REM sleep, and a decrease in delta EEG power [1, 2, 50]. There is a possibility that some of these factors, especially the reduction in spontaneous and evoked delta activity, are related to the known, largely irreversible acceleration of brain shrinkage seen in AUD, especially in the frontal and prefrontal brain areas, where delta activity is also prominent.

AUD individuals who are abstinent for a long period of time experience clinically significant insomnia, which is associated with an increased likelihood of relapsing. Prolonged and persistent sleep disorders can be attributed to dysregulation of glutamatergic systems in AUD. Drugs that are used to treat AUD may also play a role in normalizing sleep during acute withdrawal and extended abstinence periods, according to several studies [51, 52]. In fact, this is especially true for acamprostate, an allosteric modulator of glutamatergic channels, as well as gabapentin, an allosteric modulator of voltage-dependent calcium channels in neurons. In light of the link between insomnia and relapses, and the fact that sleep disturbance can exaggerate emotional dysregulation, fatigue, and cognitive withdrawal symptoms, effective treatments for insomnia in the withdrawal/negative effect and preoccupation/anticipation stages could likely assist in preventing relapse and maintaining abstinence.

## Conclusions

Children and adolescents with unipolar and bipolar depression symptoms suffer from sleep problems, which play a significant role in their development, progression, and maintenance. Depression among youth may be predicted and predated by early identification of sleep problems as early as maternal perinatal insomnia. Several independent positive predictors of smartphone addiction emerged including depression and anxiety. It could be that young adults with personality type A experiencing high stress level and low mood may lack positive stress coping mechanisms and mood management techniques and are thus highly susceptible to smartphone addiction.

In addition to depression and anxiety, smartphone addiction is associated with several independent positive predictors. Those with personality type A who experience high stress levels and low mood may lack positive stress coping mechanisms and mood management techniques, making them highly susceptible to becoming addicted to smartphones. It is important for future research to focus on the early identification and treatment of sleep problems in order to prevent depression. Among children and adolescents who develop comorbid mood symptoms and sleep problems, the risk is particularly high. Sleep problems and mood problems often coexist in children and adolescents, making them more likely to be depressed, more likely to self-harm, and more likely to commit suicide. When depression and bipolar disorder treatments are successful, sleep problems are among the most common residual symptoms, and, if untreated, are often associated with recurrent depression and/or mania. Youth with and without depression can improve their sleep problems through CBT-I treatment. Sleep improvement appears to be an important mediator of depression treatment outcomes, despite the fact that the treatment research is limited. Despite not having data on CBT-I among

youth, adult studies suggest that CBTI-BP improves mood symptoms and insomnia among adults with bipolar disorder. Besides, sleep problems like delayed sleep phase need more research, which have been under-evaluated and under-treated. If findings help national health officials and planners in Iran to design appropriate and effective interventions to improve students' health and prevent internet addiction.

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## Conflict of Interest

None.

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