Journal of ADHD and Care

# The Relationship Between Abnormal Sleeping Patterns and ADHD

Julia Davis[1,*], James Lampard[1], Allen Dsouza[2]

[1]LECOM Erie OMS 4 student, affiliated with Penn Highlands Dubois

[2]Attending pediatric psychiatry physician at Penn Highlands Dubois

**Abstract**

The objective of this focused literature review includes establishing and explaining the high correlation between sleep issues and ADHD. Through a systematic review of sources, a high correlation between sleep issues in those with ADHD was established with multiple theories lending explanation to this comorbidity. Neurological pathways in the brain already afflicted by the disorder of ADHD may cause direct effect on sleep pathways in their dysfunction. Other comorbid sleep conditions were found to occur in high frequency with ADHD however, a common etiology of these issues is yet to be established. The major conclusion of this study includes that there are multiple mechanisms through which sleep disturbances may be caused in ADHD patients, all of which must be considered in future research.

**Research Article**

**Open Access &**

**Peer-Reviewed Article**

**DOI:** 10.14302/issn.3066-8042.jac-24-5273

**Corresponding author:**

Julia Davis, LECOM Erie OMS 4 student, affiliated with Penn Highlands Dubois

**Keywords:**

ADHD, Sleep disorders, Chronotype, PFC, Prefrontal cortex, Sleep pathways, Attention, Neuroscience and ADHD, Neuroscience and Sleep

**Received:** August 28, 2024

**Accepted:** October 10, 2024

**Published:** January 10, 2025

**Citation:**

Julia Davis, James Lampard, Allen Dsouza (2025) The Relationship Between Abnormal Sleeping Patterns and ADHD. Journal of ADHD and Care - 1(2):1-6. https://doi.org/10.14302/issn.3066-8042.jac-24-5273

## Introduction

People with ADHD are known to have difficulties with various aspects of everyday routines, adequate and appropriate sleep being one of them. In this project we decided to take a deeper dive into the co-occurrence of sleep issues and ADHD in patients by looking at both genetic factors and neurological pathways playing a part in these common issues

There are two prevalent theories addressing the interplay between ADHD and insomnia. The first theory asserts that a common source is responsible for both conditions [6]. The idea that dysfunctional neurological circuits implicated in the etiology of ADHD affect the pathways for falling asleep will be further explored here as it relates to this primary theory. In contrast, the second school of thought as to this relationship, is that these issues are not interconnected in etiology but rather cyclically exacerbate one another, while coming from different sources in the body [6]. A look at the comorbidity of evening chronotype and ADHD will be explicated further to address this latter theory. It is the purpose of this paper to provide a basis of understanding of the connection between sleep and ADHD to further future research efforts in this field, which is not only multifactorial, but also an area of ongoing study.

## Methods

A systematic search and review of academic articles related to ADHD and sleep

using key words listed below was performed. Overlap in etiology, neurology and genetic factors were then assessed from the gathering of this information.

**Background**

In order to be diagnosed with ADHD an individual must show signs before age 12 years and specifically must show "Six or more symptoms of inattention and/or hyperactivity-impulsivity for children up to age 16 years or, five or more symptoms of inattention and/or hyperactivity-impulsivity for adolescents ages 17 years and older and adults." The list of symptoms applicable to ADHD as outlined by the DSM-V include: [4]

"Often fails to give close attention to details or makes careless mistakes in schoolwork, at work, or with other activities.

Often has trouble holding attention on tasks or play activities.

Often does not seem to listen when spoken to directly.

Often does not follow through on instructions and fails to finish schoolwork, chores, or duties in the workplace (e.g., loses focus, side-tracked).

Often has trouble organizing tasks and activities.

Often avoids, dislikes, or is reluctant to do tasks that require mental effort over a long period of time (such as schoolwork or homework).

Often loses things necessary for tasks and activities (e.g. school materials, pencils, books, tools, wallets, keys, paperwork, eyeglasses, mobile telephones).

Is often easily distracted.

Is often forgetful in daily activities.

Often fidgets with or taps hands or feet, or squirms in seat.

Often leaves seat in situations when remaining seated is expected.

Often runs about or climbs in situations where it is not appropriate (adolescents or adults may be limited to feeling restless).

Often unable to play or take part in leisure activities quietly.

Is often "on the go" acting as if "driven by a motor."

Often talks excessively.

Often blurts out an answer before a question has been completed.

Often has trouble waiting their turn.

Often interrupts or intrudes on others (e.g., butts into conversations or games)." [4]

NOTE: Symptoms of inattention have been present for at least 6 months, and they are inappropriate for developmental level. [4]

These symptoms must also be present in two or more settings and clearly interfere with the patient's ability to function properly in said settings. These symptoms have to not be explicable by another source like drug intoxication or another mental illness". [4]

The underlying source of dysfunction in patients with ADHD originates in the frontal lobe resulting in dysfunction of executive functioning. Here, disorganization in neural pathways and neurotransmitters

results in difficulty engaging in "directed attention". This is because it is difficult for individuals with ADHD to suspend their "automatic attention" or in other words block out attention drawing stimuli, to directly focus on something specific. This etiology contributes then to the hyperactivity, task jumping, low frustration tolerance, poor concentration and distractibility characteristic of individuals with ADHD. [2]

## Results

In this review, two major factors were found to contribute to the high frequency of sleep issues co-occurring with ADHD. The major inducer of sleep disturbance in ADHD patients was found to be explained by the direct effect of the underactivity of the inhibitory pathways within the prefrontal cortex (PFC) of the brain. The underactive PFC directly effects input to the suprachiasmatic nucleus and causes underactivity of the basal forebrain, dampening both major sleep-inducing pathways. Additionally, the prevalence of the evening chronotype genes to be found in 73-78% of ADHD patients further describes an abnormal circadian rhythm seen in these patients. The sleep issues observed in these subjects ultimately causes short sleep and sleep deprivation, resulting in decreased functionality in the patients' daily lives, regardless of underlying etiology.

## Discussion

*How ADHD causes poor sleep through direct neurological influence*

*The pathways behind ADHD*

In studying the neurological mechanisms through which ADHD and sleep disturbances function, it can be understood how the condition of ADHD can affect sleep directly. It is understood that the major mechanism causative of ADHD symptomatology is a lack of NE, DA and other catecholamines acting in the prefrontal cortex (PFC). These neurotransmitters primarily function to maintain inhibitory functions of the PFC to other areas of the brain. In their absence, the brain struggles to: "focus on the understanding and consolidation of important, but not necessarily intuitive information, to block out both internal and external distractions and to inhibit inappropriate behaviors." [11].

Additionally, it has been found that the basal ganglia, an important structure linking the PFC and hypothalamus, is also dysfunctional in ADHD patients[13]. Pathological support for this understanding is shown in how MRI studies have demonstrated reduced volume in the PFC, basal ganglia, dorsal anterior cingulate cortex, corpus callosum, and the cerebellum in ADHD patients[5]. Additionally, diffusion tensor imaging (DTI) those with ADHD has revealed evidence of decreased myelination in the axons of these pathways, demonstrating a decrease in the speed of neuronal communication in inhibitory pathways due to low catecholamines[5]. The basal ganglia function to help facilitate attention based on the processing of important visual cues. Later we will discuss how damage to this region would affect the retino-hypothalamic tract and thus sleep patterns.

*The Pathways Behind Sleep*

There are two major ways through which sleepiness is achieved in the individual, and the induction of these align with light input from the sun's 24-hour cycle resulting in proper circadian rhythm. The primary pathway which aids in sleep includes the suprachiasmatic nucleus (SCN) within the hypothalamus which, when it ceases to be stimulated by light by glutaminergic input through the retino -hypothalamic pathway, ceases to exert GABAergic input to the superior cervical ganglion (SCG), allowing for the SCG to then become active. Norepinephrine release from the SCG then induces

melatonin release from the pineal gland, initiating sleepiness [3].

Additionally, it is known that sleep is also induced by adenosine release from the basal forebrain which quiets the activity of the midbrain (an arousal center) thus allowing for sleep to occur by helping to "turn off" the arousal center [11]. It is important here to note that the basal forebrain consists of many structures but most importantly, this region contains the medial forebrain bundle [12]. The combination of the induction of these two pathways beginning a few hours before bedtime and persisting consistently and in a strong enough manner until sleep occurs, is how one falls asleep.

*How one Pathway Impacts the Other*

Underactive input to the hypothalamus from the PFC could directly result in underactivity of the hypothalamus, where the SCN resides. Since it is through a buildup of signals from the PFC to the SCN that the SCN is eventually silenced and melatonin secretion is ultimately induced, underactivity of input from the PFC to the hypothalamus would likely inhibit this sleep-inducing pathway. The underactivity of the PFC would equate to an increased duration of time needed to mount a proper sleep-inducing response from the pineal gland. This is because underactivity of the PFC would translate directly to a lower frequency of signals sent to the hypothalamus and thus increased latency in the time it takes to induce pineal gland activity. This interaction can be observed symptomatically as the increased sleep latency often noted in ADHD patients.

It is known that "The ventromedial PFC monitors and inhibits emotions and emotional habits through extensive projections to the amygdala, hypothalamus, and nucleus accumbens, as well as to brainstem nuclei mediating the stress response." [1].So, since the entire PFC is underactive in ADHD due to the lack of proper dopaminergic and catecholaminergic activity, the ventromedial PFC (part of the basal forebrain) is directly understood to be underactive as well. This indicates an effectual underactivity of secretion of adenosine by the basal forebrain to quiet the midbrain, thus hindering the process of the body transitioning from awake to sleep through ceasing of the midbrain activity. Additionally, underactivity of the nucleus accumbens, (part of the basal forebrain) and hypothalamus due to decreased input to the region by the PFC, secondary to global underactivity of the PFC, will further result in a lack of adenosine secreted by the basal forebrain. Thus, further supporting the idea that the arousal signals from the midbrain would fail to be inhibited, furthering the awake state in an individual.

Lastly, dysfunction in the basal ganglia directly as is discussed earlier in this paper in regard to damaged structures seen in the brains of ADHD patients, can influence sleep. Dysfunction in the basal ganglia indicates further dysfunction of the basal forebrain-midbrain pathway resulting in poor sleep through inability to quiet signals of arousal. Additionally, as signal travels through the basal ganglia when coursing through the retino-hypothalamic tract, altered input to the SCN through this tract could cause abnormality in the sleep/wake cycle when there is dysfunction in the basal ganglia.

*Cyclic Exacerbation Sleep dysfunction and ADHD; the comorbidity of Evening Chronotype with ADHD*

Research shows that between 73-78% of people with ADHD have the evening chronotype [8]. This chronotype causes increased sleep latency and habitually leads to increased "short sleep", two sleep issues commonly described by those afflicted with ADHD, leading to chronic sleep debt in these individuals [7]. The gene mutations attributed to this chronotype include: 3111C allele on the CLOCK gene, as well as mutations in the PER3 gene while the gene mutations attributed to the ADHD phenotype include chromosomal regions 5p13, 16p13 and 17p11 [9]. While there is virtually no overlap in the genetic source of each issue, comorbidity of these problems is still high, pointing to a correlation

without a common cause, unlike the neurological pathways discussed earlier.

These two highly comorbid conditions likely impact each other greatly as the exacerbation of one could certainly be understood to worsen the phenotype of the opposing condition. The evening chronotype causes individuals to not be able to fully fall asleep until late into the night with many patients describing the inability to fully fall asleep until about 3-4 am most nights. As the average person's workday starts around 8 am or 9 am, the patient with the evening chronotype is often forced to then wake up from a deep sleep only hours after finally falling asleep, resulting in less restorative sleep. They habitually experience sleep deprivation through consistent nights of "short sleep" which is all shown to increase mortality in a variety of ways [10]. While some may catch up on lost hours of sleep during the weekends, many cannot recover fully from the level of sleep deprivation they experience with such a sleeping pattern. This deprivation of sleep leads to the cognitive and attention difficulties we know result from a lack of sleep which only worsens the already existing impulsivity and attention deficit phenotype seen in ADHD patients.

Furthermore, it is known that many people with the evening chronotype do not experience peak performance in terms of focus and cognitive functioning until later in the day [8]. As most jobs require peak functioning in the morning and then allow for a steady decline into the evening, the patient with an evening chronotype is already at a disadvantage before the further deficits from ADHD in this realm of functioning is factored in [8]. The anxiety that then comes from the inability to focus at work secondary to ADHD symptomatology further perpetuates the inability to calm racing thoughts in the evening and worsens the ability to sleep. The distraction of stress over the inability to sleep then causes further distraction and exacerbation of ADHD symptoms at work and as such the vicious cycle repeats itself resulting in sleep impairment nightly and attention impairments daily for the patient.

**Conclusion**

As is clearly depicted by the previous assertions, there are multiple ways in which ADHD causes and interacts with sleep disorders. It may be understood how ADHD and certain sleep disorders occur comorbidly and cyclically exacerbate one another. While the major etiology explaining the co-occurrence of these diseases may be debated, it is hard to refute the comorbidity of such disorders. Additionally, one cannot ignore the possible negative repercussions which neglecting to treat either disorder in an affected individual may have on their well-being globally.

It can be understood that direct interaction of the neuronal pathways involved in ADHD and sleep may etiologically explain the concordance of sleep problems and ADHD. Additionally, the correlation between carrying the gene for the evening chronotype and a diagnosis of ADHD may also help to explain the comorbidity of ADHD and disrupted sleep. Our hope is that this review helps further direct studies into treatment solutions for sleep issues experienced by those with ADHD.

**References**

1. Arnsten A. F. (2009). The Emerging Neurobiology of Attention Deficit Hyperactivity Disorder: The Key Role of the Prefrontal Association Cortex. *The Journal of pediatrics*, *154*(5), I–S43. https:/ doi.org/10.1016/j.jpeds.2009.01.018
2. *Attention-deficit/hyperactivity disorder (ADHD)*. Cleveland Clinic. (2024b, May 1). https:// my.clevelandclinic.org/health/diseases/4784-attention-deficithyperactivity-disorder-adhd
3. Borjigin, J., Zhang, L. S., & Calinescu, A. A. (2012). Circadian regulation of pineal gland

rhythmicity. *Molecular and cellular endocrinology*, *349*(1), 13–19. https://doi.org/10.1016/j.mce.2011.07.009

4. Centers for Disease Control and Prevention. (n.d.). *Diagnosing ADHD*. Centers for Disease Control and Prevention. https://www.cdc.gov/adhd/diagnosisindex.html#cdc_testing_resources-resources
5. Curatolo, P., D'Agati, E., & Moavero, R. (2010). The neurobiological basis of ADHD. *Italian journal of pediatrics*, *36*(1), 79. https://doi.org/10.1186/1824-7288-36-79
6. [European College of Neuropsychopharmacology]. (2017, September 28). *30th ECNP Congress webcast: Circadian rhythm and sleep in ADHD – cause or life style factor?* [Video]. Youtube. https://www.youtube.com/watch?v=SkuIL-Ljmwo
7. Frontiers (2022, August 10). *Chronotype, circadian rhythm, and psychiatric disorders: Recent evidence and potential mechanisms*. Frontiers in Neuroscience. Retrieved July 29, 2024, from https:/ www.frontiersin.org/journals/neuroscience/articles/10.3389/fnins.2022.811771/full
8. Gabay, L., Miller, P., Alia-Klein, N., & Lewin, M. P. (2022). Circadian Effects on Attention and Working Memory in College Students With Attention Deficit and Hyperactivity Symptoms. *Frontiers in psychology*, *13*, 851502. https://doi.org/10.3389/fpsyg.2022.851502
9. Kalmbach, D. A., Schneider, L. D., Cheung, J., Bertrand, S. J., Kariharan, T., Pack, A. I., & Gehrman, P. R. (2017). Genetic Basis of Chronotype in Humans: Insights From Three Landmark GWAS. *Sleep*, *40*(2), zsw048. https://doi.org/10.1093/sleep/zsw048
10. MDPI (2021, March 24). *Biological Rhythm and Chronotype: New Perspectives in Health*. Retrieved July 29, 2024, from https://www.mdpi.com/2218-273X/11/4/487
11. NIH (2024, June 18). *Brain Basics: Understanding Sleep*. National Institute of Neurological Disorders and Stroke. Retrieved July 29, 2024, from https://www.ninds.nih.gov/health-information/public-education/brain-basics/brain-basics-understanding-sleep#:~:text=The%20brain%20stem%2C%20at%20the,t%20act%20out%20our%20dreams
12. Roland Zahn, Ricardo de Oliveira-Souza, Jorge Moll, Moral Motivation and the Basal Forebrain, Neuroscience & Biobehavioral Reviews, Volume 108, 2020, Pages 207-217, ISSN 0149-7634, https://doi.org/10.1016/j.neubiorev.2019.10.022. (https://www.sciencedirect.com/science/article/pii/S0149763419300776)
13. Shaw, P., De Rossi, P., Watson, B., Wharton, A., Greenstein, D., Raznahan, A., Sharp, W., Lerch, J. P., & Chakravarty, M. M. (2014). Mapping the development of the basal ganglia in children with attention-deficit/hyperactivity disorder. *Journal of the American Academy of Child and Adolescent Psychiatry*, *53*(7), 780–9.e11. https://doi.org/10.1016/j.jaac.2014.05.003