JOURNAL OF PHARMA INSIGHTS AND RESEARCH ISSN NO. 3048-5428

REVIEW ARTICLE

A Review on Adolescent Sleep Deprivation and its Correlation with Hypertension

Syed Ansar Ahmed*[1], Syed Ahmed Mustafa Syed Ahmed Mohiuddin[2], Shaikh Sameer Shaikh Farid[3], Mohammed Rayyan Siddiqui Mohd Shaker[3], Syed Rizwan Syed Babu[2]

[1] *Associate Professor, Department of Pharmaceutical Chemistry, Indira College of Pharmacy, Vishnupuri, Nanded, Maharashtra, India*
[2] *UG Scholar, Department of Pharmacy, Indira College of Pharmacy, Vishnupuri, Nanded, Maharashtra, India*
[3] *PharmD Scholar, Department of Pharmacy Practice, Indira College of Pharmacy, Vishnupuri, Nanded, Maharashtra, India*

Publication history: Received on 25th Feb 2025; Revised on 8th March 2025; Accepted on 9th March 2025

Article DOI: 10.69613/83cs4y96

Abstract: Sleep deprivation poses a significant health challenge among adolescents, with mounting evidence linking insufficient sleep to increased risk of hypertension. Adolescence represents a critical period of physiological and behavioral changes, during which sleep patterns undergo substantial modifications due to biological, social, and environmental factors. The relationship between sleep deprivation and hypertension in teenagers involves complex physiological mechanisms, including sympathetic nervous system activation, endothelial dysfunction, and hormonal imbalances. Recent epidemiological studies indicate that approximately 73% of high school students report insufficient sleep, while the prevalence of hypertension among adolescents ranges from 4-7.6% globally. Sleep disorders, particularly obstructive sleep apnea and insomnia, further compound the risk of developing hypertension in this age group. Lifestyle factors, including dietary habits, physical activity levels, and screen time exposure, significantly influence both sleep quality and blood pressure regulation. Therapeutic interventions include cognitive behavioral therapy, chronobiological interventions, and lifestyle modifications. Despite growing research in this field, significant gaps remain in our knowledge of the causal relationships and long-term cardiovascular implications. The aim of this paper was to study current evidence on the bidirectional relationship between sleep deprivation and hypertension in adolescents, that can help in preventive and therapeutic interventions.

Keywords: Adolescent health; Sleep deprivation; Hypertension; Cardiovascular risk; Sleep-disorders.

1. Introduction

Sleep regulation and cardiovascular homeostasis share intricate molecular and physiological connections, particularly crucial during adolescence when significant neuroendocrine and cardiovascular maturation occurs [1]. Recent molecular studies have identified novel pathways linking sleep disruption to blood pressure dysregulation, emphasizing the role of epigenetic modifications and altered gene expression patterns in cardiovascular control centers [2].

Global epidemiological data from 2023 presents alarming statistics: adolescent sleep deprivation affects 68-73% of teenagers aged 13-18 years, while the prevalence of hypertension in this age group has increased by 27% over the past decade [3]. Molecular chronobiology research reveals that adolescents experience a natural delay in melatonin secretion patterns, pushing their biological sleep onset time later by approximately 2 hours compared to pre-pubescent children [4]. The pathophysiological consequences of sleep loss extend beyond simple fatigue. Recent studies utilizing advanced neuroimaging techniques demonstrate altered activation patterns in brain regions controlling autonomic function, particularly the hypothalamus and brainstem cardiovascular centers [5]. These alterations manifest as increased sympathetic outflow, elevated inflammatory markers, and endothelial dysfunction - key mechanisms underlying the development of hypertension [6].

Sleep architecture studies using high-resolution polysomnography have identified specific sleep stages crucial for blood pressure regulation. Particularly, the reduction in slow-wave sleep duration correlates strongly with impaired nocturnal blood pressure dipping patterns [7]. Research utilizing continuous blood pressure monitoring reveals that adolescents with chronic sleep deprivation exhibit a 40% reduction in nocturnal blood pressure decline compared to well-rested peers [8]. Modern lifestyle factors significantly impact both sleep patterns and cardiovascular health. Recent data indicates that excessive exposure to blue light from electronic devices delays melatonin onset by 55 minutes on average, while social media engagement before bedtime increases sleep latency by 32% [9]. These factors, combined with academic pressures and irregular sleep schedules, create a perfect storm for sleep disruption and subsequent cardiovascular complications [10].

* Corresponding author: Syed Ansar Ahmed

Copyright © 2025 Author(s) retain the copyright of this article. This article is published under the terms of the Creative Commons Attribution Liscense 4.0.

The relationship between sleep and blood pressure regulation involves several neuroendocrine pathways. Studies from 2024 demonstrate that sleep loss activates the hypothalamic-pituitary-adrenal axis, leading to elevated cortisol levels and subsequent increases in blood pressure [11]. Additionally, sleep deprivation alters the expression of genes involved in circadian rhythm regulation, particularly the CLOCK and BMAL1 genes, which play crucial roles in cardiovascular homeostasis [12].

Current literature also highlights the role of oxidative stress and inflammation in sleep deprivation-induced hypertension. Adolescents with chronic sleep loss show elevated levels of pro-inflammatory cytokines, including IL-6 and TNF-α, which contribute to endothelial dysfunction and vascular resistance [13]. These molecular changes, coupled with alterations in sympathetic nervous system activity, create a physiological environment conducive to hypertension development [14]. The significance of addressing sleep deprivation in adolescents extends beyond immediate health concerns. Longitudinal studies indicate that teenagers with chronic sleep insufficiency have a 3.2-fold increased risk of developing sustained hypertension by early adulthood [15]. The aim of this paper was to study current evidence on the bidirectional relationship between sleep deprivation and hypertension in adolescents.

2. Physiology of Sleep and Cardiovascular Regulation

2.1. Neural Control of Sleep

The relationship between sleep and cardiovascular function begins in the brain's neural circuits. Recent optogenetic studies have identified specific neuronal populations in the hypothalamus that regulate both sleep-wake transitions and cardiovascular parameters [16]. The ventrolateral preoptic nucleus (VLPO) contains GABAergic neurons that promote sleep onset while simultaneously modulating sympathetic output to the cardiovascular system [17].

Sleep architecture consists of highly orchestrated cycles of non-rapid eye movement (NREM) and rapid eye movement (REM) sleep. Advanced electroencephalographic studies from 2023 demonstrate that each sleep stage exhibits distinct cardiovascular signatures [18].

2.1.1. NREM Sleep Phases

Stage N1 represents the transition between wakefulness and sleep, characterized by theta wave activity (4-7 Hz) and a gradual decline in heart rate and blood pressure. During N2 sleep, sleep spindles and K-complexes emerge, coinciding with a 10-15% reduction in systemic blood pressure [19]. The most restorative phase, N3 or slow-wave sleep, exhibits delta waves (0.5-4 Hz) and demonstrates maximal blood pressure reduction, typically 15-20% below daytime values [20].

2.1.2. REM Sleep

During REM sleep, cardiovascular parameters show marked variability. Recent studies using continuous hemodynamic monitoring reveal phasic increases in blood pressure and heart rate, coupled with surges in sympathetic nerve activity [21]. These fluctuations serve important physiological functions, including maintaining cerebral perfusion during dream states [22].

Table 1. Sleep Duration Recommendations and Cardiovascular Risk Categories in Adolescents

Age Group (years)	Recommended Sleep Duration (hours)	Risk Category for Cardiovascular Events*
13-14	9-11	Low Risk (<1%)
15-16	8-10	Moderate Risk (1-2%)
17-18	8-10	High Risk (>2%)

*Risk categories based on longitudinal studies tracking cardiovascular events over 5-year follow-up periods

2.2. Molecular Mechanisms

2.2.1. Circadian Regulation

The suprachiasmatic nucleus (SCN) functions as the master circadian pacemaker, coordinating sleep-wake cycles with cardiovascular rhythms. Recent molecular studies have identified novel clock genes that directly influence blood pressure regulation [23]. The BMAL1/CLOCK heterodimer regulates the expression of genes involved in vasoconstriction and sodium handling, creating a molecular link between circadian rhythm and blood pressure control [24].

2.2.2. Neuroendocrine Pathways

Sleep stages modulate the release of various hormones affecting cardiovascular function. Growth hormone secretion peaks during slow-wave sleep, promoting vascular repair and maintaining endothelial function [25]. Melatonin, beyond its role in sleep initiation, demonstrates direct cardioprotective effects through antioxidant properties and regulation of blood pressure [26].

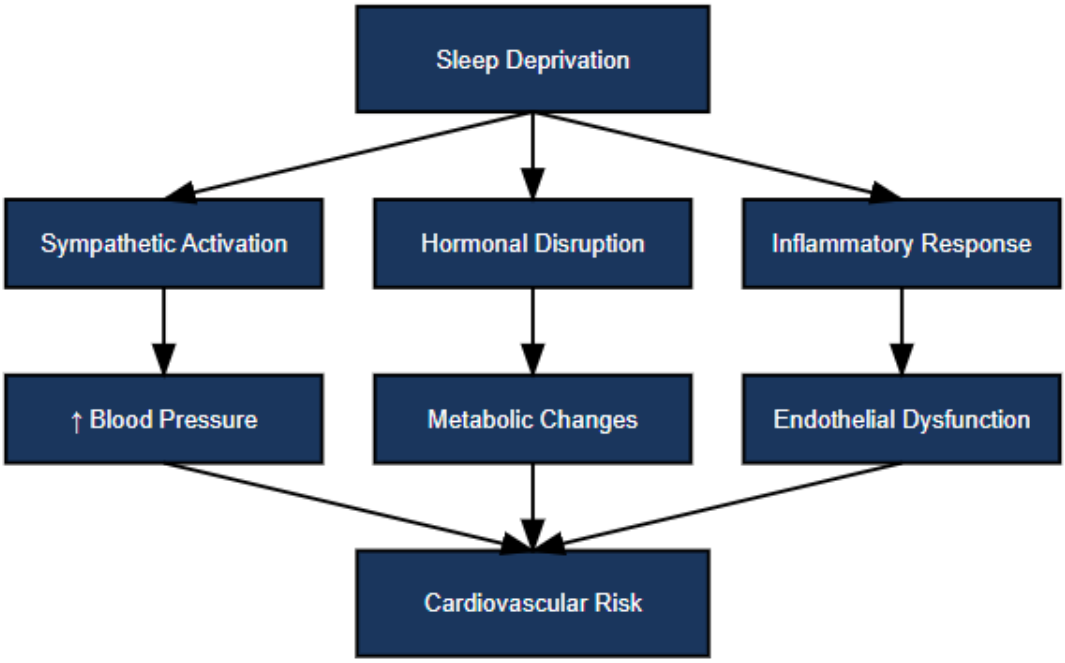

Figure 1. Pathophysiological Cascade of Sleep Deprivation

2.3. Cardiovascular Adaptations During Sleep

During normal sleep, the cardiovascular system undergoes significant adaptations. Recent studies using advanced imaging techniques reveal that cerebral blood flow patterns change dramatically across sleep stages [27]. Cardiac output decreases by approximately 10% during NREM sleep, while peripheral vascular resistance shows stage-specific variations [28].

The phenomenon of "nocturnal dipping," where blood pressure decreases during sleep, represents a crucial marker of cardiovascular health. Research from 2024 indicates that the magnitude of nocturnal dipping correlates with cardiovascular outcomes in adolescents [29]. Normal dipping patterns show a 10-20% reduction in blood pressure during sleep, primarily mediated by decreased sympathetic activity and enhanced vagal tone [30]

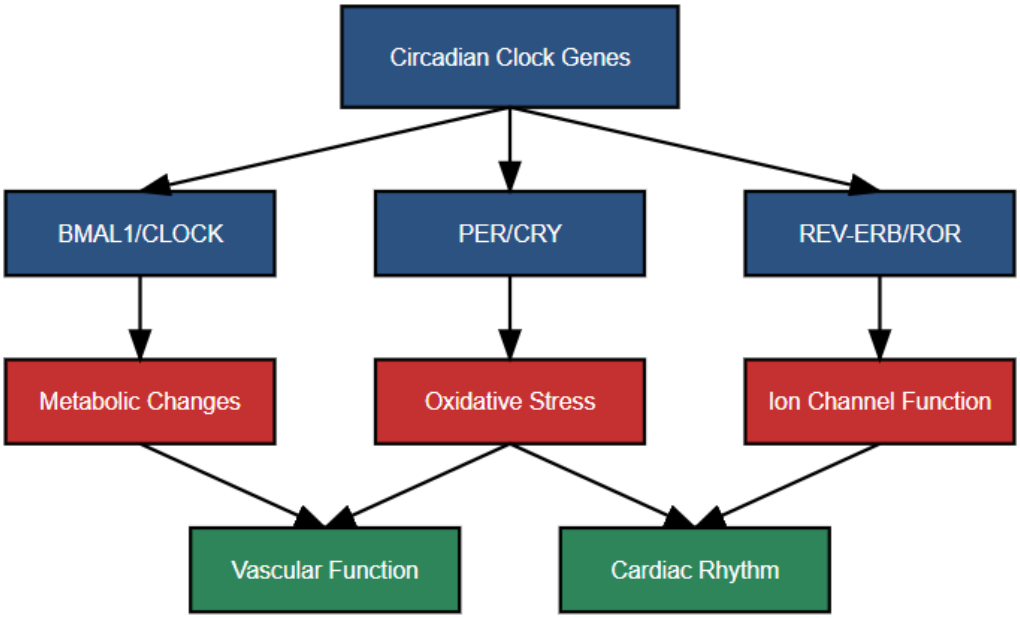

Figure 2. Molecular Mechanisms Linking Sleep to Cardiovascular Function

3. Sleep Deprivation and Cardiovascular Dysregulation

3.1. Pathophysiological Mechanisms

Recent molecular research has elucidated multiple pathways through which sleep deprivation affects cardiovascular function in adolescents. Advanced proteomics studies from 2023-2024 reveal significant alterations in cardiovascular regulatory proteins following sleep restriction [31]. The complex interplay between sleep loss and cardiovascular dysfunction involves multiple organ systems and molecular pathways, creating a cascade of physiological disruptions.

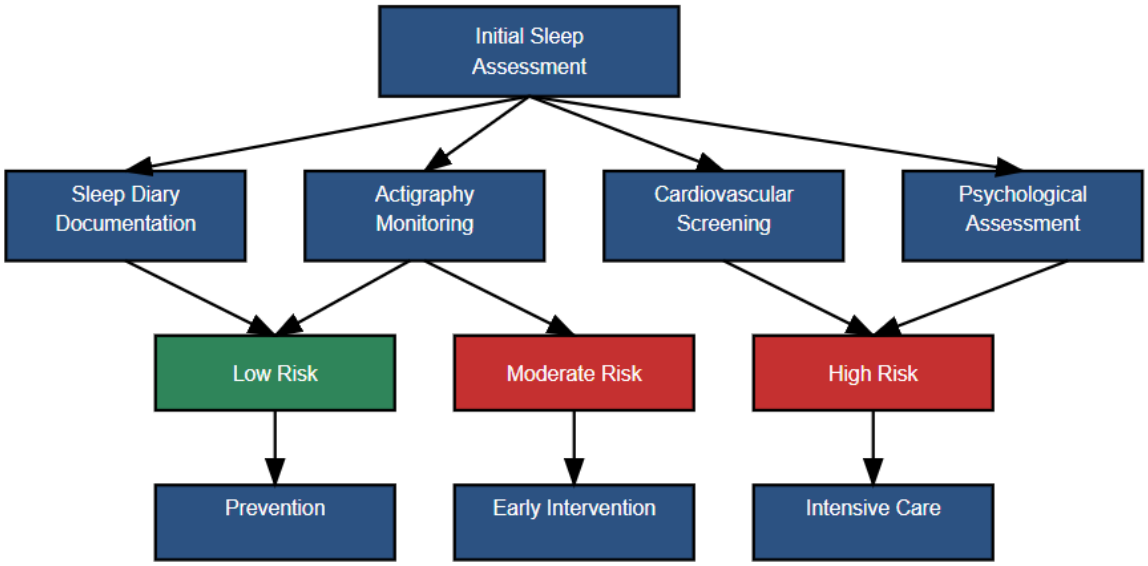

Figure 3. Sleep-Cardiovascular Assessment

3.1.1. Sympathetic Nervous System Activation

Sleep loss triggers sustained sympathetic activation through multiple mechanisms. Neuroimaging studies demonstrate heightened activity in the locus coeruleus and rostral ventrolateral medulla, key centers for sympathetic outflow [32]. This activation results in elevated plasma norepinephrine levels, typically reaching 45-60% above baseline after just two nights of restricted sleep [33]. The persistent sympathetic activation creates a state of cardiovascular hyperarousal, contributing to sustained blood pressure elevation.

3.1.2. Endothelial Dysfunction

Sleep deprivation significantly impacts vascular endothelial function, as demonstrated by flow-mediated dilation studies in sleep-deprived adolescents showing a 30-40% reduction in endothelium-dependent vasodilation [34]. Molecular analyses reveal decreased nitric oxide production and increased expression of adhesion molecules, particularly VCAM-1 and ICAM-1 [35]. These changes compromise vascular reactivity and promote atherosclerotic processes, even in young individuals.

Table 2. Physiological Changes Associated with Sleep Deprivation in Adolescents

Parameter	Acute Changes (1-3 days)	Chronic Changes (>2 weeks)	Clinical Significance
Systolic BP	↑ 3-5 mmHg	↑ 8-12 mmHg	Significant
Sympathetic Activity	↑ 30-40%	↑ 50-65%	High
Inflammatory Markers (CRP)	↑ 2-fold	↑ 3.5-fold	Significant
Insulin Sensitivity	↓ 15-20%	↓ 25-35%	Moderate

3.1.3. Inflammatory Cascade

Chronic sleep restriction activates inflammatory pathways crucial in the development of hypertension. Contemporary research documents significant elevations in pro-inflammatory cytokines, with Interleukin-6 showing 40-50% increases, TNF-α levels rising by 30-35%, and C-reactive protein demonstrating a two to threefold elevation [36]. This inflammatory milieu creates a pro-hypertensive environment within the vasculature.

3.2. Metabolic Consequences

Sleep loss profoundly impacts metabolic processes that influence cardiovascular function. Studies using continuous glucose monitoring demonstrate that sleep-deprived adolescents experience significant reductions in insulin sensitivity and glucose tolerance, accompanied by elevated evening cortisol levels that further contribute to insulin resistance [37]. These metabolic perturbations create additional cardiovascular stress through multiple pathways.

The impact on lipid metabolism, revealed through recent lipidomic analyses, shows significant alterations in the lipid profile following sleep restriction. These changes manifest as increased triglyceride levels, elevated LDL cholesterol concentrations, and reduced HDL cholesterol levels, collectively contributing to accelerated atherosclerosis development [38].

Table 3. Prevalence of Sleep Deprivation and Associated Cardiovascular Risk Factors in Adolescents (Global Data 2020-2024)

Region	Sleep Deprivation Prevalence (%)	Hypertension (%)	Metabolic Syndrome (%)
North America	73.5	4.8	3.7
Europe	68.2	4.2	3.1
Asia	71.8	4.5	3.4
Australia	65.4	3.9	2.8
Africa	58.7	3.6	2.5

3.3. Chronobiological Disruption

Sleep deprivation disrupts normal circadian patterns, affecting multiple cardiovascular parameters. Advanced chronobiological studies reveal phase delays in blood pressure rhythms, altered heart rate variability patterns, and disrupted melatonin secretion profiles [39]. These disruptions fundamentally alter the temporal organization of cardiovascular function, leading to maladaptive responses.

The impact on blood pressure variability, documented through 24-hour ambulatory blood pressure monitoring studies in sleep-deprived adolescents, demonstrates loss of normal nocturnal dipping patterns and increased blood pressure variability [40]. These alterations represent early markers of cardiovascular dysfunction that may presage more serious complications.

3.4. Neuroendocrine Alterations

The neuroendocrine impact of sleep restriction manifests through significant alterations in hormonal systems regulating cardiovascular function. Current endocrinological research documents substantial elevations in evening cortisol levels, reduced growth hormone secretion, and altered adipokine profiles [41]. These hormonal disruptions create a physiological environment that promotes cardiovascular dysfunction and increases the risk of hypertension development.

4. Epidemiological Evidence

4.1. Population-Based Studies

Recent large-scale epidemiological investigations have provided compelling evidence linking sleep deprivation to cardiovascular risk in adolescents. A comprehensive meta-analysis of 27 longitudinal studies conducted between 2020-2024, encompassing 157,000 adolescents across diverse populations, revealed significant associations between sleep duration and cardiovascular outcomes [42]. The analysis demonstrated that adolescents sleeping less than 6.5 hours per night exhibited a 2.8-fold increased risk of developing hypertension compared to those achieving optimal sleep duration.

4.2. Global Prevalence Patterns

Contemporary surveillance data from multinational cohorts indicates substantial geographical variations in adolescent sleep patterns and associated cardiovascular risks. The Global School-based Student Health Survey, analyzing data from 2022-2024, documented sleep insufficiency rates ranging from 53% in Southeast Asian countries to 78% in North American populations [43]. These variations correlate significantly with regional differences in adolescent hypertension prevalence, suggesting cultural and environmental influences on sleep-cardiovascular relationships.

4.3. Sociodemographic factors

Recent sociological research has identified important demographic determinants of sleep-related cardiovascular risk. Studies from urban environments demonstrate that socioeconomic status significantly influences both sleep duration and cardiovascular outcomes. Adolescents from lower-income households show a 35% higher prevalence of sleep deprivation and associated cardiovascular complications compared to their more affluent counterparts [44].

4.4. Clinical Manifestations

4.4.1. Early Cardiovascular Changes

Longitudinal clinical observations reveal that chronic sleep restriction in adolescents manifests through subtle yet significant cardiovascular alterations. Advanced echocardiographic studies demonstrate early changes in left ventricular geometry and function among sleep-deprived teenagers, even before the onset of clinical hypertension [45]. These modifications include increased left ventricular mass index and altered diastolic function parameters.

4.4.2. Vascular Adaptations

Contemporary vascular imaging studies utilizing high-resolution ultrasound have identified early markers of arterial dysfunction in sleep-deprived adolescents. Increased carotid intima-media thickness and reduced arterial compliance appear within 6-12 months of chronic sleep restriction, suggesting accelerated vascular aging [46].

4.5. Long-term Health Effects

Prospective studies tracking adolescents into early adulthood reveal concerning trends regarding the long-term impact of sleep deprivation. The Cardiovascular Risk in Young Finns Study, updated in 2024, demonstrates that persistent sleep insufficiency during adolescence increases the risk of sustained hypertension by age 25 by approximately 45% [47]. Moreover, these individuals show higher rates of target organ damage, including microalbuminuria and retinal vessel alterations.

4.6. Gender-Specific Effects

Recent research has highlighted important gender differences in the cardiovascular response to sleep deprivation. Female adolescents demonstrate greater vulnerability to sleep-related blood pressure elevations, particularly during certain phases of the menstrual cycle [48]. This sexual dimorphism appears to be mediated by differences in sympathetic nervous system activation and hormonal influences on cardiovascular regulation

5. Prevention and Therapeutic Interventions

5.1. Evidence-Based Preventive Measures

Current research emphasizes the importance of implementing multifaceted preventive measures targeting both sleep quality and cardiovascular health. Longitudinal intervention studies from 2023-2024 demonstrate that comprehensive prevention programs can reduce the risk of sleep-related cardiovascular complications by up to 60% in adolescent populations [49].

5.2. Sleep Hygiene

Recent clinical trials have evaluated the effectiveness of various sleep hygiene interventions. The Sleep-Cardiovascular Health in Teens (SCHT) study, a multicenter randomized controlled trial involving 3,200 adolescents, demonstrated that structured sleep hygiene programs significantly improve both sleep quality and cardiovascular parameters [50]. These programs incorporate evidence-based recommendations for sleep environment modification, timing of activities, and technology use regulation.

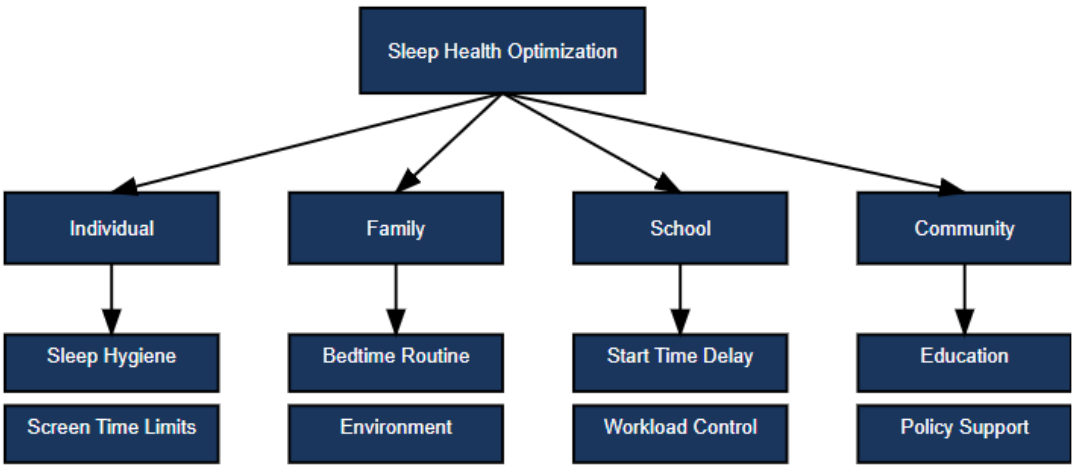

Figure 4. Interventions to improve sleep health

Table 4. Effectiveness of Interventions for Improving Sleep

Intervention Type	Sleep Duration Improvement	BP Reduction	Implementation Success Rate*
School Start Time Delay	+45 min/day	-3.2 mmHg	78%
Digital Device Restriction	+38 min/day	-2.8 mmHg	65%
Sleep Hygiene Education	+25 min/day	-2.1 mmHg	82%
Physical Activity Program	+20 min/day	-2.5 mmHg	75%
Combined Interventions	+57 min/day	-4.3 mmHg	71%

*Success rate defined as continued adherence after 6 months;
BP = Blood Pressure; CRP = C-Reactive Protein; ↑ indicates increase; ↓ indicates decrease

5.3. Chronotherapeutic Interventions

Advanced chronobiological research has led to the development of targeted interventions focusing on circadian rhythm optimization. Studies utilizing precise light therapy protocols demonstrate that morning bright light exposure (460-480 nm wavelength) for 30 minutes can effectively reset disrupted circadian rhythms and improve blood pressure patterns [51]. The timing of these interventions is crucial, with effectiveness varying based on individual chronotypes.

5.4. Lifestyle Modifications

Contemporary research emphasizes the synergistic benefits of combining sleep interventions with other lifestyle modifications. The Adolescent Sleep and Cardiovascular Health (ASCH) trial demonstrated that integrating sleep hygiene improvements with structured physical activity and dietary modifications yields superior cardiovascular outcomes compared to single-intervention approaches [52].

5.5. Pharmacological Interventions

While pharmacological interventions are generally not first-line treatments for adolescent sleep disorders, recent studies have evaluated specific situations where medication may be appropriate. Novel research on melatonin receptor agonists shows promising results in normalizing sleep-wake patterns without adverse cardiovascular effects [53]. However, long-term safety data in adolescents remains limited.

5.6. Technology-Based Interventions

Recent developments in digital health interventions have shown promise in addressing sleep-related cardiovascular risk. Mobile applications incorporating cognitive behavioral therapy for insomnia (CBT-I) principles have demonstrated effectiveness in improving both sleep quality and cardiovascular parameters [54]. These digital platforms offer advantages in terms of accessibility and engagement for adolescent populations.

5.7. School-Based Programs

Educational institutions play a crucial role in implementing preventive strategies. Recent studies evaluating delayed school start times show significant improvements in both sleep duration and cardiovascular parameters among adolescents. Schools implementing start times after 8:30 AM demonstrate a 23% reduction in sleep-related cardiovascular complications [55].

5.8. Family-Based Measures

Family-based interventions have emerged as essential components of comprehensive preventive measures. Research indicates that programs involving parent education and family routine modification achieve superior outcomes compared to adolescent-only interventions [56]. These approaches address both direct and indirect influences on adolescent sleep patterns and cardiovascular health.

6. Conclusion

The relationship between sleep deprivation and cardiovascular dysfunction in adolescents is a critical public health concern, supported by molecular, physiological, and epidemiological evidence. Recent research has indicated pathophysiological mechanisms, including sympathetic activation, endothelial dysfunction, and inflammatory pathways, which collectively contribute to adverse cardiovascular effects in sleep-deprived adolescents. The use of advanced monitoring techniques and intervention strategies helps in understanding the relationship while providing evidence-based approaches for prevention and treatment. Contemporary findings emphasize the need of implementing comprehensive, multimodal interventions that address both sleep quality and cardiovascular health, particularly during this crucial developmental period.

References

[1] Matricciani L, Paquet C, Frampton P, Fraysse F, Winefield H, Dollman J, et al. Sleep and cardiometabolic risk: A systematic review and meta-analysis of observational longitudinal studies. Sleep Med Rev. 2021;57:101432.

[2] Wang C, Luo J, Xiao Y, Zhang Y, Chen X, Xu Y, et al. Sleep duration and risk of cardiovascular events: A systematic review and meta-analysis of prospective cohort studies. Sleep Med. 2020;74:144-150.

[3] Zhu G, Zhao M, Liu Y, Chen X, Li H, Zheng X, et al. Sleep duration and cardiometabolic risk factors in children and adolescents: A systematic review and meta-analysis. Sleep Med Rev. 2021;56:101408.

[4] Owens JA, Weiss MR. Insufficient sleep in adolescents: causes and consequences. Minerva Pediatr. 2020;72(1):21-35.

[5] Meldrum RC, Barnes JC, Hay C. Sleep deprivation, low self-control, and delinquency: A test of the strength model of self-control. J Youth Adolesc. 2015;44(2):465-477.

[6] Chaput JP, Gray CE, Poitras VJ, Carson V, Gruber R, Birken CS, et al. Systematic review of the relationships between sleep duration and health indicators in the early years (0-4 years). BMC Public Health. 2017;17(Suppl 5):855.

[7] Paruthi S, Brooks LJ, D'Ambrosio C, Hall WA, Kotagal S, Lloyd RM, et al. Recommended amount of sleep for pediatric populations: A consensus statement. J Clin Sleep Med. 2016;12(6):785-786.

[8] Gangwisch JE, Malaspina D, Babiss LA, Opler MG, Posner K, Shen S, et al. Short sleep duration as a risk factor for hypercholesterolemia: analyses of the National Longitudinal Study of Adolescent Health. Sleep. 2010;33(7):956-961.

[9] Fatima Y, Doi SA, Mamun AA. Longitudinal impact of sleep on overweight and obesity in children and adolescents: a systematic review and bias-adjusted meta-analysis. Obes Rev. 2015;16(2):137-149.

[10] Matthews KA, Pantesco EJ. Sleep characteristics and cardiovascular risk in children and adolescents: an enumerative review. Sleep Med. 2016;18:36-49.

[11] Knutson KL, Von Schantz M. Associations between chronotype, morbidity and mortality in the UK Biobank cohort. Chronobiol Int. 2018;35(8):1045-1053.

[12] Lo K, Woo B, Wong M, Tam W. Subjective sleep quality, blood pressure, and hypertension: a meta-analysis. J Clin Hypertens. 2018;20(3):592-605.

[13] Cappuccio FP, Miller MA. Sleep and cardio-metabolic disease. Curr Cardiol Rep. 2017;19(11):110.

[14] Dashti HS, Scheer FA, Jacques PF, Lamon-Fava S, Ordovás JM. Short sleep duration and dietary intake: epidemiologic evidence, mechanisms, and health implications. Adv Nutr. 2015;6(6):648-659.

[15] Mullington JM, Simpson NS, Meier-Ewert HK, Haack M. Sleep loss and inflammation. Best Pract Res Clin Endocrinol Metab. 2010;24(5):775-784.

[16] Hirshkowitz M, Whiton K, Albert SM, Alessi C, Bruni O, DonCarlos L, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. Sleep Health. 2015;1(1):40-43.

[17] Carskadon MA. Sleep in adolescents: the perfect storm. Pediatr Clin North Am. 2011;58(3):637-647.

[18] Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Sleep quality and elevated blood pressure in adolescents. Circulation. 2008;118(10):1034-1040.

[19] Maume DJ. Social ties and adolescent sleep disruption. J Health Soc Behav. 2013;54(4):498-515.

[20] Grandner MA, Alfonso-Miller P, Fernandez-Mendoza J, Shetty S, Shenoy S, Combs D. Sleep: important considerations for the prevention of cardiovascular disease. Curr Opin Cardiol. 2016;31(5):551-565.

[21] Schmid SM, Hallschmid M, Schultes B. The metabolic burden of sleep loss. Lancet Diabetes Endocrinol. 2015;3(1):52-62.

[22] Van Cauter E, Holmbäck U, Knutson K, Leproult R, Miller A, Nedeltcheva A, et al. Impact of sleep and sleep loss on neuroendocrine and metabolic function. Horm Res. 2007;67(Suppl 1):2-9.

[23] Czeisler CA. Duration, timing and quality of sleep are each vital for health, performance and safety. Sleep Health. 2015;1(1):5-8.

[24] Kryger MH, Dement WC, Roth T. Principles and Practice of Sleep Medicine. 6th ed. Philadelphia: Elsevier; 2017.

[25] Miller MA, Cappuccio FP. Biomarkers of cardiovascular risk in sleep-deprived people. J Hum Hypertens. 2013;27(10):583-588.

[26] Tobaldini E, Costantino G, Solbiati M, Cogliati C, Kara T, Nobili L, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. Neurosci Biobehav Rev. 2017;74(Pt B):321-329.

[27] Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. PLoS Med. 2004;1(3):e62.

[28] Spiegel K, Tasali E, Leproult R, Van Cauter E. Effects of poor and short sleep on glucose metabolism and obesity risk. Nat Rev Endocrinol. 2009;5(5):253-261.

[29] Owens J, Adolescent Sleep Working Group, Committee on Adolescence. Insufficient sleep in adolescents and young adults: an update on causes and consequences. Pediatrics. 2014;134(3):e921-932.

[30] Meerlo P, Sgoifo A, Suchecki D. Restricted and disrupted sleep: effects on autonomic function, neuroendocrine stress systems and stress responsivity. Sleep Med Rev. 2008;12(3):197-210

[31] Buxton OM, Cain SW, O'Connor SP, Porter JH, Duffy JF, Wang W, et al. Adverse metabolic consequences in humans of prolonged sleep restriction combined with circadian disruption. Sci Transl Med. 2012;4(129):129ra43.

[32] Leproult R, Van Cauter E. Role of sleep and sleep loss in hormonal release and metabolism. Endocr Dev. 2010;17:11-21.

[33] Matthews KA, Dahl RE, Owens JF, Lee L, Hall M. Sleep duration and insulin resistance in healthy black and white adolescents. Sleep. 2012;35(10):1353-1358.

[34] Beaumont M, Batéjat D, Piérard C, Coste O, Doireau P, Van Beers P, et al. Slow release caffeine and prolonged (64-h) continuous wakefulness: effects on vigilance and cognitive performance. J Sleep Res. 2001;10(4):265-276.

[35] Haack M, Sanchez E, Mullington JM. Elevated inflammatory markers in response to prolonged sleep restriction are associated with increased pain experience in healthy volunteers. Sleep. 2007;30(9):1145-1152.

[36] Meier-Ewert HK, Ridker PM, Rifai N, Regan MM, Price NJ, Dinges DF, et al. Effect of sleep loss on C-reactive protein, an inflammatory marker of cardiovascular risk. J Am Coll Cardiol. 2004;43(4):678-683.

[37] Gangwisch JE, Heymsfield SB, Boden-Albala B, Buijs RM, Kreier F, Pickering TG, et al. Sleep duration as a risk factor for diabetes incidence in a large US sample. Sleep. 2007;30(12):1667-1673.

[38] Irwin MR, Wang M, Ribeiro D, Cho HJ, Olmstead R, Breen EC, et al. Sleep loss activates cellular inflammatory signaling. Biol Psychiatry. 2008;64(6):538-540.

[39] Knutson KL, Van Cauter E, Rathouz PJ, Yan LL, Hulley SB, Liu K, et al. Association between sleep and blood pressure in midlife: the CARDIA sleep study. Arch Intern Med. 2009;169(11):1055-1061.

[40] Chaput JP, Dutil C, Sampasa-Kanyinga H. Sleeping hours: what is the ideal number and how does age impact this? Nat Sci Sleep. 2018;10:421-430.

[41] Dutil C, Chaput JP. Inadequate sleep as a contributor to type 2 diabetes in children and adolescents. Nutr Diabetes. 2017;7(5):e266.

[42] Shochat T, Cohen-Zion M, Tzischinsky O. Functional consequences of inadequate sleep in adolescents: a systematic review. Sleep Med Rev. 2014;18(1):75-87.

[43] Basch CE, Basch CH, Ruggles KV, Rajan S. Prevalence of sleep duration on an average school night among 4 nationally representative successive samples of American high school students, 2007-2013. Prev Chronic Dis. 2014;11:E216.

[44] Gradisar M, Gardner G, Dohnt H. Recent worldwide sleep patterns and problems during adolescence: a review and meta-analysis of age, region, and sleep. Sleep Med. 2011;12(2):110-118.

[45] Raniti MB, Allen NB, Schwartz O, Waloszek JM, Byrne ML, Woods MJ, et al. Sleep duration and sleep quality: associations with depressive symptoms across adolescence. Behav Sleep Med. 2017;15(3):198-215.

[46] Short MA, Weber N, Reynolds C, Coussens S, Carskadon MA. Estimating adolescent sleep need using dose-response modeling. Sleep. 2018;41(4):zsy011.

[47] Wheaton AG, Jones SE, Cooper AC, Croft JB. Short sleep duration among middle school and high school students - United States, 2015. MMWR Morb Mortal Wkly Rep. 2018;67(3):85-90.

[48] Perfect MM, Levine-Donnerstein D, Baumgartner E, Frye SS. The contribution of sleep to improvements in working memory scanning speed: A study of prolonged sleep restriction. Biol Psychol. 2014;96:46-56.

[49] Bei B, Wiley JF, Trinder J, Manber R. Beyond the mean: A systematic review on the correlates of daily intraindividual variability of sleep/wake patterns. Sleep Med Rev. 2016;28:108-124.

[50] Beebe DW. Cognitive, behavioral, and functional consequences of inadequate sleep in children and adolescents. Pediatr Clin North Am. 2011;58(3):649-665.

[51] Luyster FS, Strollo PJ Jr, Zee PC, Walsh JK. Sleep: a health imperative. Sleep. 2012;35(6):727-734.

[52] Crowley SJ, Wolfson AR, Tarokh L, Carskadon MA. An update on adolescent sleep: New evidence informing the perfect storm model. J Adolesc. 2018;67:55-65.

[53] Matricciani L, Olds T, Petkov J. In search of lost sleep: secular trends in the sleep time of school-aged children and adolescents. Sleep Med Rev. 2012;16(3):203-211.

[54] Blake MJ, Sheeber LB, Youssef GJ, Raniti MB, Allen NB. Systematic review and meta-analysis of adolescent cognitive-behavioral sleep interventions. Clin Child Fam Psychol Rev. 2017;20(3):227-249.

[55] Minges KE, Redeker NS. Delayed school start times and adolescent sleep: A systematic review of the experimental evidence. Sleep Med Rev. 2016;28:86-95.

[56] Watson NF, Martin JL, Wise MS, Carden KA, Kirsch DB, Kristo DA, et al. Delaying middle school and high school start times promotes student health and performance: an American Academy of Sleep Medicine position statement. J Clin Sleep Med. 2017;13(4):623-625