



International Journal of Innovative Technologies in Social Science

e-ISSN: 2544-9435

Operating Publisher
SciFormat Publishing Inc.
ISNI: 0000 0005 1449 8214

2734 17 Avenue SW,
Calgary, Alberta, T3E0A7,
Canada
+15878858911
editorial-office@sciformat.ca

ARTICLE TITLE	SLEEP DISORDERS AS A RISK FACTOR FOR HYPERTENSION
----------------------	---

DOI	https://doi.org/10.31435/ijitss.3(51).2026.5923
------------	---

RECEIVED	17 July 2026
-----------------	--------------

ACCEPTED	11 August 2026
-----------------	----------------

PUBLISHED	21 September 2026
------------------	-------------------

LICENSE	
----------------	--



The article is licensed under a **Creative Commons Attribution 4.0 International License**.

© The author(s) 2026.

This article is published as open access under the Creative Commons Attribution 4.0 International License (CC BY 4.0), allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

SLEEP DISORDERS AS A RISK FACTOR FOR HYPERTENSION

Dawid Żuraw (Corresponding Author, Email: dzuraw19@gmail.com)

University Clinical Hospital No. 1, Lublin, Poland

ORCID ID: 0009-0004-3160-5659

Magdalena Kijowska

Medical University of Lublin, Poland

ORCID ID: 0009-0004-1086-7086

Karolina Klusek

Medical University of Lublin, Poland

ORCID ID: 0009-0004-7006-0188

Katarzyna Kulszo

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Poland

ORCID ID: 0000-0002-8573-9714

Martyna Rynkowska

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Poland

ORCID ID: 0009-0003-8976-2318

Magda Kowaleczko

University Clinical Hospital No. 1, Lublin, Poland

ORCID ID: 0009-0000-6055-0141

Natalia Bogumillo

University Clinical Hospital No. 1, Lublin, Poland

ORCID ID: 0009-0002-7567-2242

Maciej Wiśniecki

Stefan Cardinal Wyszyński Provincial Specialist Hospital SPZOZ in Lublin, Poland

ORCID ID: 0009-0002-1152-2835

Bartłomiej Baszun

University Clinical Hospital No. 1, Lublin, Poland

ORCID ID: 0009-0003-1694-4268

Krzysztof Mirkowski

University Clinical Hospital No. 1, Lublin, Poland

ORCID ID: 0009-0009-6976-0810

ABSTRACT

Sleep disorders are among the most prevalent health problems in modern society and are increasingly recognized as an important risk factor for cardiovascular diseases. Growing evidence indicates that insufficient sleep duration, poor sleep quality, and conditions such as insomnia or obstructive sleep apnea may contribute to the development of arterial hypertension. Sleep plays a crucial role in maintaining systemic homeostasis by regulating neurological, endocrine, and metabolic functions. Sleep disturbances may lead to chronic activation of the sympathetic nervous system and the hypothalamic–pituitary–adrenal (HPA) axis, promoting inflammatory processes and impairing blood pressure regulation. An increasing body of evidence suggests that impaired sleep negatively affects overall health and may contribute to the development of multiple chronic diseases. Sleep disturbances frequently coexist with other cardiovascular risk factors, including chronic stress, obesity, physical inactivity, and unhealthy dietary habits, thereby amplifying their adverse effects. Persistent deterioration in sleep quality may result in metabolic, hormonal, and cardiovascular dysfunction, increasing the risk of long-term health complications and reducing quality of life. In light of current evidence, adequate sleep duration and quality should be considered essential components of hypertension prevention and public health promotion strategies.

KEYWORDS

Sleep Disorders, Hypertension, Blood Pressure Regulation, Obstructive Sleep Apnea, Insomnia

CITATION

Żuraw, D., Kijowska, M., Klusek, K., Kulszo, K., Rynkowska, M., Kowaleczko, M., Bogumiłło, N., Wiśniecki, M., Baszun, B., & Mirkowski, K. (2026). Sleep disorders as a risk factor for hypertension. *International Journal of Innovative Technologies in Social Science*, 3(51). [https://doi.org/10.31435/ijitss.3\(51\).2026.5923](https://doi.org/10.31435/ijitss.3(51).2026.5923)

COPYRIGHT

© The author(s) 2026. This article is published as open access under the **Creative Commons Attribution 4.0 International License (CC BY 4.0)**, allowing the author to retain copyright. The CC BY 4.0 License permits the content to be copied, adapted, displayed, distributed, republished, or reused for any purpose, including adaptation and commercial use, as long as proper attribution is provided.

Introduction

Arterial hypertension (HTN) is one of the most prevalent chronic conditions in the adult population and represents a primary risk factor for cardiovascular complications and related mortality (Han et al., 2020). According to current guidelines, optimal blood pressure values in adults are defined as 120/80 mmHg, whereas values equal to or exceeding 140/90 mmHg are classified as hypertension. Ranges between these values are categorized as normal or high-normal blood pressure and are associated with a progressively increasing risk of cardiovascular disease. Early identification of elevated blood pressure is crucial for preventing complications and implementing appropriate preventive and therapeutic interventions (Han et al., 2020; Whelton et al., 2022). Furthermore, hypertension imposes a substantial burden on healthcare systems due to increased hospitalization rates, the requirement for long-term pharmacological treatment, and elevated medical care costs (Han et al., 2020).

Sleep is an indispensable physiological process, fundamental to maintaining systemic homeostasis. It modulates the autonomic, metabolic, and endocrine systems, which are vital for blood pressure regulation and cardiovascular function (Whelton et al., 2022). Sleep disorders, including insomnia, obstructive sleep apnea (OSA), and circadian rhythm disruptions, are an escalating public health concern among adults. Numerous studies indicate that short sleep duration and poor sleep quality may contribute to the pathogenesis of hypertension through heightened sympathetic activity, chronic low-grade inflammation, and hormonal dysregulation (Guo et al., 2013; Li & Shang, 2021). Sufficient sleep duration and quality represent a modifiable cardiovascular risk factor that should be integrated into clinical practice (Li & Shang, 2021).

The objective of this paper is to review the available literature regarding the association between sleep disorders and the risk of arterial hypertension in adults. This analysis encompasses the impact of sleep duration, sleep quality, and insomnia symptoms, as well as the physiological and hormonal mechanisms that may mediate this relationship. The synthesized data provide a deeper understanding of the importance of sleep as a modifiable risk factor for cardiovascular disease and underscore the role of prevention and health education in hypertension management.

Methodology

This study is a narrative review based on the analysis of available scientific literature concerning the association between sleep disorders and the risk of arterial hypertension, as well as the underlying pathophysiological mechanisms involved in this relationship. The literature search was conducted using electronic databases and scientific platforms, including PubMed, MDPI, and Google Scholar. The analysis included publications from 1993 to 2025, with particular emphasis placed on recent epidemiological studies, clinical trials, and meta-analyses published between 2020 and 2025. The search strategy was based on the use of the following keywords and their combinations: *sleep disorders*, *hypertension*, *blood pressure regulation*, *obstructive sleep apnea*, *insomnia*, and *short sleep duration*. The preliminary selection process involved screening the titles and abstracts of identified publications in order to exclude studies not directly related to the subject of the review or lacking sufficient scientific value. Subsequently, full-text versions of the selected articles were thoroughly analyzed. Priority was given to English-language publications, particularly clinical studies, cohort studies, meta-analyses, systematic reviews, current clinical guidelines, and review articles. The inclusion criteria comprised up-to-date publications relevant to the scope of the study, available in full text, and characterized by adequate methodological quality. Particular attention was paid to studies evaluating the impact of sleep duration and quality, insomnia, and obstructive sleep apnea on blood pressure values, hypertension risk, and autonomic, hormonal, and metabolic regulation. The exclusion criteria included publications that did not meet scientific standards, articles published in languages other than English, and studies not directly related to sleep disorders and arterial hypertension. After applying the inclusion and exclusion criteria, a total of 50 publications were selected for the final analysis. Zotero software (version 9.0.2) was used for bibliography management and duplicate removal.

Results

Foundations of Sleep Physiology

Sleep is a complex, actively regulated physiological process in which the body transitions through cyclic NREM (non-rapid eye movement) and REM (rapid eye movement) stages. These phases alternate throughout the night in cycles lasting approximately 60–90 minutes, forming the typical sleep architecture in adults (Carley et al., 2016). NREM sleep is divided into several stages, ranging from light sleep (N1) to deep slow-wave sleep (N3), reflecting varying levels of brain activity synchronization and physiological recovery (Wood et al., 2020). During REM sleep, neuronal activity resembles wakefulness, with high-frequency EEG patterns and lower synchronization compared to NREM, hence the term “paradoxical sleep” (McCarley, 2007). REM sleep is also characterized by increased variability in hemodynamic parameters, including heart rate and blood pressure, reflecting heightened metabolic and autonomic activity as well as central regulation of cardiovascular function (Wood et al., 2020; Benarroch, 2019). Sleep requirements vary with age—infants require 12–16 hours, school-aged children 9–12 hours, adolescents 8–10 hours, and adults 7–9 hours. Adequate sleep duration is essential for maintaining both physical and mental health (Hirshkowitz et al., 2015).

Analysis of heart rate variability (HRV) during sleep has shown that in healthy adults, the LF/HF ratio—where the low-frequency component (LF) reflects sympathetic and mixed autonomic activity, and the high-frequency component (HF) represents parasympathetic tone—is significantly higher during REM sleep than during NREM stages, indicating a relative dominance of sympathetic activity and greater cardiovascular variability (Makarem et al., 2019). Hemodynamic variability across sleep stages is well documented: NREM sleep is associated with reductions in blood pressure and heart rate, whereas REM sleep involves greater fluctuations due to sympathetic activation and baroreflex modulation (Vorster et al., 2024). One of the key physiological phenomena during sleep is the so-called nocturnal “dipping,” defined as a 10–20% reduction in blood pressure compared to daytime values, primarily occurring during NREM sleep (Vorster et al., 2024). Disruption of this mechanism is significantly associated with an increased risk of hypertension, left ventricular hypertrophy, and cardiovascular events (Schiffrin, EL, 2004).

An experimental study conducted in 2023 using polysomnography demonstrated that selective awakening of healthy individuals during REM sleep resulted in a significant increase in systolic and diastolic blood pressure upon awakening, indicating impaired nocturnal cardiovascular regulation in the setting of

disrupted sleep architecture (Xu et al., 2023). Additionally, REM sleep deprivation was associated with increased heart rate and cardiac output, along with decreased systemic vascular resistance, suggesting a shift toward sympathetic dominance (Xu et al., 2023). These findings indicate that proper REM sleep plays a crucial role in stabilizing hemodynamic parameters, and its disruption may lead to transient cardiovascular instability even in individuals without underlying disease (Xu et al., 2023).

Cardiovascular regulation during sleep is mediated by autonomic and hormonal mechanisms. NREM sleep is characterized by parasympathetic predominance, resulting in reduced heart rate and blood pressure, whereas REM sleep involves increased sympathetic activity and greater hemodynamic variability (Jafari, 2017). The physiological reduction of cortisol levels during the first half of the night, along with rhythmic melatonin secretion, supports proper blood pressure regulation and endothelial protection, as circadian rhythms coordinate cardiovascular function (Makarem et al., 2019). Cortisol acts via glucocorticoid receptors in vascular smooth muscle cells, increasing angiotensinogen production and activating the renin–angiotensin–aldosterone system, thereby elevating blood pressure (Makarem et al., 2019). In contrast, melatonin, through MT1 and MT2 receptors, modulates vascular tone and suppresses excessive sympathetic activity, promoting vasodilation and lowering blood pressure (Pechanova et al., 2014).

Sleep disturbances, including reduced sleep duration, sleep fragmentation, and obstructive sleep apnea, lead to chronic sympathetic activation and loss of nocturnal blood pressure decline, thereby promoting the development of hypertension (Makarem et al., 2021). In particular, obstructive sleep apnea is associated with repeated episodes of hypoxia and micro-arousals, resulting in increased sympathetic activity, oxidative stress, and endothelial dysfunction (Benjafield et al., 2025). Consequently, both the quantity and quality of sleep are critical for maintaining proper blood pressure regulation, and their disruption contributes to the development of hypertension and cardiovascular complications (Li & Shang, 2021). Furthermore, chronic sleep disturbances are associated with increased arterial stiffness, inflammation, and endothelial dysfunction, further exacerbating cardiovascular risk (Bigalke et al., 2022). These mechanisms are of particular clinical importance, as sustained sympathetic activation and the loss of nocturnal blood pressure dipping represent key pathways in the development of sleep-related hypertension (Li & Shang, 2021 ; Makarem et al., 2021).

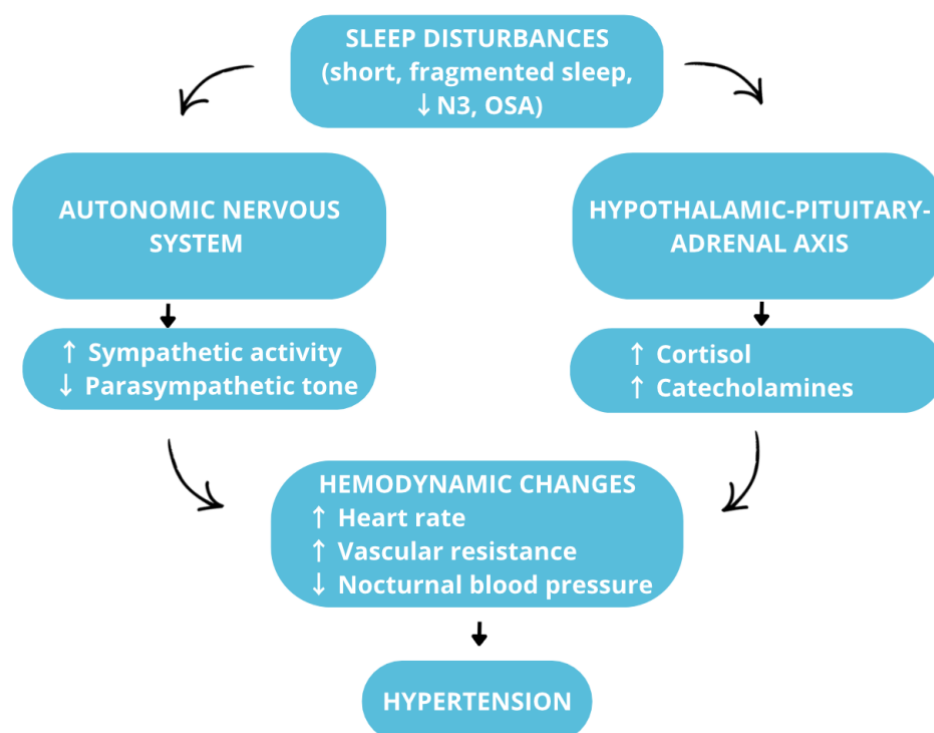


Fig. 1. Impact of Sleep Disturbances on Autonomic and HPA Regulation of Blood Pressure

Epidemiology of Sleep Disorders

Sleep disturbances related to non-optimal sleep duration are common among adults, with their prevalence showing substantial variation across countries and age groups; both short sleep (< 7 hours) and long sleep (> 9 hours) are observed in a significant proportion of the population (Fernandez-Mendoza et al., 2026). It is estimated that sleep disorders affect up to 30–40% of adults worldwide, and their prevalence is steadily increasing in parallel with population aging and lifestyle changes (Direksunthorn, T., 2025). In high-income countries, sleep-related complaints are among the most frequently reported health problems in primary care settings (Direksunthorn, T., 2025). The rising prevalence of sleep disturbances has been associated with factors such as increasing urbanization, exposure to artificial light at night, shift work, and chronic stress, all of which disrupt circadian rhythms and reduce sleep duration (Tähhämö et al., 2019).

To better characterize this phenomenon, a study conducted between 2015 and 2022 among individuals aged ≥ 40 years living in rural areas of South Africa demonstrated that 25.1% of participants reported short sleep, while 32.4% reported long sleep (> 9 hours for individuals aged 40–64 years and > 8 hours for those aged ≥ 65 years). Logistic regression analysis revealed that younger age, employment status, depressive symptoms, and hypertension were significantly associated with a higher likelihood of short sleep duration (Pengpid et al., 2025). Short sleep duration not only increases the risk of sleep disturbances but also impairs sleep quality, contributing to the development of insomnia, defined as persistent difficulty initiating or maintaining sleep accompanied by dissatisfaction with sleep quality (Benjafield et al., 2025). Importantly, the relationship between sleep duration and health often follows a curvilinear (U-shaped) pattern, indicating that both insufficient and excessive sleep are associated with adverse health outcomes (Fishbein et al., 2021).

The global prevalence of insomnia has been evaluated in a systematic review by Benjafield et al., which included 18 epidemiological studies ($n = 262,582$). Based on these data, approximately 16.2% of adults worldwide were estimated to suffer from insomnia, while 7.9% met the criteria for severe insomnia, highlighting its substantial global health burden (Benjafield et al., 2025). Furthermore, insomnia has been reported more frequently in women than in men, which may reflect biological, psychosocial, and behavioral differences (Benjafield et al., 2025). Notably, insomnia frequently coexists with psychiatric disorders, particularly depression and anxiety, further complicating its clinical course and management (Kretzschmar et al., 2026).

Sleep-disordered breathing, particularly obstructive sleep apnea (OSA), represents another important component of sleep disorder epidemiology. A 2024 systematic review reported that approximately 16% of healthy young adults meet diagnostic criteria for OSA based on objective measurements (Zasadzińska-Stempniak et al., 2024). OSA is characterized by recurrent episodes of hypoxia and micro-arousals, which disrupt autonomic and hormonal regulation, thereby contributing to the development of hypertension and cardiovascular diseases (Wang et al., 2025; Grassi et al., 2010). In a study by Yi Wang et al. (2025), involving 2,574 patients with OSA, shorter sleep duration was significantly associated with both prevalent and incident hypertension during follow-up (Wang et al., 2025). In recent years, the detection rate of OSA has increased, likely due to improved diagnostic accessibility and the growing prevalence of risk factors such as obesity and sedentary lifestyle (Jeong et al., 2025). It is also estimated that a substantial proportion of OSA cases remain undiagnosed, leading to underestimation of its true prevalence and delayed treatment initiation (Bumgarner et al., 2021).

Demographic differences in the prevalence of sleep disorders are well established. Meta-analyses including data from multiple countries indicate that sleep disturbances and insomnia are more common in women and increase with age (Benjafield et al., 2025). In contrast, OSA is more frequently diagnosed in men and older individuals, with its occurrence influenced by factors such as body mass index, fat distribution, and lifestyle (Benjafield et al., 2025). These findings highlight the importance of considering both age and sex in epidemiological analyses and clinical risk assessment. Additionally, socioeconomic disparities play a significant role, as individuals with lower socioeconomic status are more likely to experience sleep disturbances, potentially due to poorer environmental conditions, higher stress levels, and limited access to healthcare (Fishbein et al., 2021).

From an epidemiological perspective, sleep disorders constitute an important risk factor for chronic diseases, including hypertension, type 2 diabetes, obesity, and cardiovascular diseases, making them a major public health concern (Jeong et al., 2025). Moreover, both short and excessively long sleep duration have been associated with increased all-cause mortality in adult populations (Jeong et al., 2025). Consequently, sleep disturbances are increasingly recognized not only as symptoms but also as independent, modifiable risk factors for chronic diseases, underscoring the importance of their early diagnosis and management in public health strategies (Lei et al., 2024).

Pathophysiological Mechanisms Linking Sleep Disorders and Hypertension

The Autonomic Nervous System and the HPA Axis

The autonomic nervous system (ANS) plays a pivotal role in the short-term regulation of arterial blood pressure through the equilibrium between sympathetic and parasympathetic activity. Excessive sympathetic activation leads to vasoconstriction, increased peripheral resistance, and tachycardia, resulting in elevated blood pressure values (Bigalke et al., 2022; Grassi et al., 2010). During NREM sleep, parasympathetic tone predominates, causing a reduction in heart rate and a decline in blood pressure (nocturnal dipping). In contrast, the REM phase is characterized by heightened sympathetic activity and hemodynamic variability, which is essential for the cardiovascular system's adaptation to the body's metabolic demands.

Under conditions of sleep fragmentation or deprivation, the NREM-to-REM ratio is disrupted. Arousal episodes specifically curtail the N3 stage (slow-wave sleep), leading to a relative sympathetic predominance even at night, which results in increased BP and impaired parasympathetic modulation (Somers et al., 1993). In clinical practice, disrupted sleep not only blunts the physiological nocturnal "dip" in pressure but also exacerbates hemodynamic fluctuations during the day, potentially fostering chronic hypertension.

Clinical studies provide increasingly granular data on this mechanism. Grassi et al. (2010) demonstrated that augmented adrenergic activity is associated with a diminished nocturnal blood pressure fall and higher pressure values during both day and night, suggesting impaired autonomic regulation under conditions of fragmented sleep and chronic sympathetic stress (Grassi et al., 2010). A recent review by Meng, He, and Li (2025) indicates that chronic autonomic dysfunction in hypertension involves both overactivation of the sympathetic nervous system and attenuated parasympathetic modulation, leading to increased systemic vascular resistance, elevated heart rate, renal dysfunction, and neuroinflammatory processes (Meng et al., 2025).

In the context of obstructive sleep apnea (OSA), hypoxic episodes trigger acute surges of sympathetic activation and disruptions in parasympathetic modulation. This results in profound blood pressure fluctuations both during the night and in wakefulness, increasing the risk of developing and sustaining hypertension (Tobushi et al., 2024). A review by Bigalke et al. (2022) confirms that sleep debt and poor sleep quality intensify sympathetic drive, increasing vascular tone and promoting hypertension, even in individuals without other cardiovascular risk factors (Bigalke et al., 2022).

Simultaneously, sleep disturbances activate the hypothalamic-pituitary-adrenal (HPA) axis, providing an additional mechanism for hypertension risk. A review by Fernandez-Mendoza et al. (2025) indicates that insomnia with short sleep duration (<6–7 h) is linked to HPA overactivation, elevated cortisol levels, and a state of systemic physiological hyperarousal, which increases vascular tension and impairs pressure regulation (Fernandez-Mendoza et al., 2026). It is further suggested that HPA axis activation and sympathetic drive occur concurrently; elevated cortisol and catecholamines interfere with nocturnal blood pressure dipping, synergistically contributing to the pathogenesis of hypertension (Fernandez-Mendoza et al., 2026).

Metabolic and Hormonal Mechanisms

Sleep is fundamental to maintaining hormonal, metabolic, and immunological homeostasis. Sleep deprivation is associated with increased HPA axis activity, leading to the hypersecretion of stress hormones such as cortisol. This can result in sustained arterial hypertension and the exacerbation of inflammatory and metabolic responses (Narkiewicz et al., 1997).

Sleep disturbances also impact glucose metabolism and the hormonal balance of appetite-regulating hormones—leptin and ghrelin. A reduction in leptin levels weakens the satiety signal, while an increase in ghrelin stimulates appetite, promoting excessive caloric intake, weight gain, and adipose tissue accumulation, ultimately leading to insulin resistance. Insulin resistance, in turn, elevates hypertension risk by intensifying sympathetic activity, promoting renal sodium retention, and inducing structural changes in the vasculature (Direksunthorn, T., 2025; Meng et al., 2025). A narrative review by Direksunthorn (2025) corroborates these findings, showing that both acute and chronic sleep debt impair glucose metabolism and appetite-regulating hormones, thereby predisposing individuals to insulin resistance and hypertension. The author emphasizes that sleep disorders alter the leptin-ghrelin balance, increasing cravings and weight gain, thus indirectly increasing the cardiovascular load (Direksunthorn, T., 2025).

Furthermore, chronic inflammation plays a significant role in the mechanisms leading to hypertension. Shortened or poor-quality sleep elevates levels of inflammatory markers, such as C-reactive protein (CRP) and interleukin-6 (IL-6), which induce vascular endothelial dysfunction, promote increased peripheral resistance, and trigger sympathetic activation, further raising the risk of hypertension (Direksunthorn, T., 2025). In this manner, sleep disorders integrate hormonal, metabolic, and immunological pathways into a complex cardiovascular risk profile, underscoring the importance of adequate sleep duration and quality in the primary prevention of hypertension.

Epidemiological Evidence

Epidemiological evidence indicates that both sleep duration and quality exert a substantial influence on the risk of developing arterial hypertension in adults. Individuals who consistently sleep fewer than 7 hours per day exhibit a higher risk of hypertension compared to those sleeping 7–8 hours, as confirmed by a large-scale meta-analysis (2024) encompassing over one million participants across various countries (Hosseini et al., 2024). According to this analysis, short sleep duration (<7 h) was associated with a significantly higher risk of hypertension (HR = 1.07; 95% CI 1.06–1.09), with the effect being even more pronounced for sleep durations below 5 hours (HR = 1.11; 95% CI 1.08–1.14). The biological mechanisms underlying this association may include overactivation of the sympathetic nervous system, HPA axis dysfunction, and metabolic and hormonal imbalances that exacerbate blood pressure elevation (Fernandez-Mendoza et al., 2026; Somers et al., 1993). In subgroup analyses, women appear to be more susceptible to the impact of short sleep, although the precise reasons for this sexual dimorphism remain fully elucidated. In the same meta-analysis, sleep duration exceeding 8 hours was not significantly associated with hypertension risk, suggesting that very short and very long sleep may have distinct etiologies regarding metabolic risk or may serve as markers for the presence of other chronic comorbidities (Hosseini et al., 2024).

In addition to sleep duration, sleep quality plays a critical role. Cohort data based on objective polysomnography, including the Sleep Heart Health Study (2023), demonstrated that a higher proportion of the N2 phase and a reduced proportion of deep sleep (N3) were associated with a higher risk of hypertension, independent of total sleep duration (Chen et al., 2024). Clinical sleep disorders, including insomnia, also increase this risk; a meta-analysis by Dai et al. (2024) showed that the phenotype of insomnia with objective short sleep (ISSOD) was a potent predictor of both prevalent and incident hypertension (OR = 2.67; RR = 1.95), being markedly stronger than subjective insomnia without short sleep duration (Dai et al., 2024). Population data from NHANES (USA, 2005–2018), involving 33,383 adults, further indicate that prolonged sleep latency (>30 min) is associated with a higher risk of hypertension. Furthermore, the impact of sleep duration on HTN may be age-dependent: sleep shorter than 7 hours increased risk in younger participants, whereas sleep longer than 8 hours was a risk factor in the elderly (Liu et al., 2024). The significance of demographic factors is corroborated by a systematic review and meta-analysis of 23 prospective studies (2025), encompassing 173,734 participants. Sleep shorter than 7 hours was significantly linked to a higher risk of hypertension, with the effect being more pronounced in women and individuals under 60 years of age. This underscores that the HTN risk profile is contingent upon age and sex, suggesting that these groups may require targeted attention in hypertension prevention strategies (Qi et al., 2025).

Despite these consistent findings, it should be noted that the majority of evidence originates from observational studies based on self-reported data. This limits the ability to draw definitive causal inferences, and confounding factors—such as obesity, lifestyle, or comorbid chronic conditions—may partially influence the observed associations (Hosseini et al., 2024).

Interactions with Other Risk Factors

Sleep disorders, in conjunction with other risk factors, exacerbate the development of arterial hypertension, resulting from the synergistic interaction between disordered sleep patterns and an unhealthy lifestyle (Lv et al., 2022). A prospective study of 165,493 participants from the UK Biobank demonstrated that individuals with a low healthy sleep score—accounting for factors such as short or excessive sleep duration, insomnia, snoring, and daytime sleepiness—had a significantly higher risk of developing hypertension over long-term follow-up. Concurrent adherence to an unhealthy lifestyle—comprising smoking, physical inactivity, poor diet, and excessive body mass—further compounded this risk, indicating a synergistic effect between sleep disturbances and other metabolic and behavioral factors (Lv et al., 2022). The authors emphasize that both sleep disorders and unhealthy lifestyles independently and collectively influence blood pressure regulation, glucose metabolism, and autonomic function, stemming from disruptions in the biological processes governing hemodynamic and metabolic stability (Lv et al., 2022).

Obesity and Other Metabolic Disorders

Comorbidities such as type 2 diabetes and metabolic syndrome amplify the impact of sleep disorders on blood pressure, as abnormal metabolic parameters—including insulin resistance, hyperglycemia, and dyslipidemia—increase susceptibility to HTN (Jeong et al., 2025). Research indicates that acute or chronic sleep disturbances can exacerbate these abnormalities, leading to heightened activation of the sympathetic nervous system and the HPA axis, which consequently increases vascular resistance and stimulates blood pressure elevation (Jeong et al., 2025 ; Makarem et al., 2019; Vorster et al., 2024).

A 2025 cross-sectional study involving over 2.9 million adults in South Korea found that both short sleep (<6 h) and long sleep (>8 h) were associated with a higher risk of comorbid hypertension and type 2 diabetes compared to individuals sleeping 7–8 hours per day (Jeong et al., 2025). Notably, individuals with diabetes and sleep duration abnormalities exhibited significantly greater blood pressure variability, suggesting a synergistic effect of sleep disturbances and metabolic dysregulation on the cardiovascular system (Jeong et al., 2025). Concurrently, short or fragmented sleep disrupts the secretion of key appetite- and metabolism-regulating hormones, such as insulin, leptin, and ghrelin, leading to increased caloric intake, excessive weight gain, and visceral adiposity. These hormonal imbalances foster insulin resistance, elevated blood glucose levels, and lipid dysregulation, which in turn increase the risk of both visceral obesity and arterial hypertension, worsening the deleterious cardiovascular effects of sleep loss (Direksunthorn, T., 2025; Meng et al., 2025). Consequently, the combination of suboptimal sleep duration, poor sleep quality, and metabolic disease acts synergistically to potentiate the risk of developing and sustaining hypertension, while also increasing the likelihood of cardiovascular complications, including coronary artery disease, stroke, and heart failure (Jeong et al., 2025; Pechanova et al., 2014).

Obesity serves as a critical modifier of this relationship, as excessive body mass promotes metabolic dysfunction, increases the risk of obstructive sleep apnea (OSA), and intensifies chronic inflammation, ultimately elevating blood pressure and exacerbating the negative consequences of poor sleep (Battisha et al., 2025). Data from NHANES studies show that individuals with short sleep duration or impaired sleep quality have higher obesity rates, indirectly increasing their hypertension risk. A study of 4,540 adult participants utilized a sleep pattern index evaluating duration, quality, daytime sleepiness, and sleep-onset latency (Xu et al., 2025). The results indicated that individuals with unhealthy sleep patterns had a significantly higher risk of obesity, with the effect being most pronounced at the extremes of sleep duration (short or excessively long sleep) (Xu et al., 2025). Insulin resistance and hyperglycemia increase oxidative stress and the production of pro-inflammatory cytokines, leading to endothelial dysfunction, increased vascular tone, and impaired blood pressure autoregulation. In combination with obesity and disordered sleep, these mechanisms fuel chronic inflammation; elevated levels of inflammatory markers, such as CRP and IL-6, further aggravate endothelial dysfunction and increase peripheral resistance, contributing to sustained hypertension (Pechanova et al., 2014). Obesity also increases the prevalence of OSA, which generates nocturnal hypoxic episodes and abrupt sympathetic surges, intensifying pressure fluctuations and hypertension risk (Xu et al., 2025).

Therefore, promoting healthy sleep and maintaining optimal sleep duration may be a vital component of obesity prevention. Enhancing sleep quality and regularity supports hormonal balance and reduces the risk of insulin resistance and inflammation, thereby limiting the risk of developing arterial hypertension and other cardiovascular diseases in the adult population.

Stress

Chronic psychological stress and psychosocial burdens are recognized as significant, independent risk factors for the development of arterial hypertension, as they activate both the sympathetic nervous system and the hypothalamic-pituitary-adrenal (HPA) axis, leading to sustained blood pressure elevation and impaired physiological adaptation (al'Absi et al., 2000; Marwaha, 2022; Nunez et al., 2025).

Sympathetic activation in response to chronic stress triggers the release of catecholamines (epinephrine and norepinephrine), which increases heart rate and induces vasoconstriction, resulting in elevated peripheral resistance and arterial pressure (al'Absi et al., 2000; Marwaha, 2022; Nunez et al., 2025). Simultaneously, prolonged HPA axis stimulation causes a sustained rise in cortisol levels, which promotes renal sodium and water retention and elevates blood glucose concentrations—mechanisms directly linked to hypertension and metabolic dysfunction (Marwaha, 2022 ; Narkiewicz et al., 1997).

Chronic stress also affects the vascular system by promoting inflammation and endothelial injury; recurrent episodes of pressure elevation are associated with reduced nitric oxide production and increased pro-inflammatory cytokine secretion, which impairs vasodilation and maintains high arterial pressure (Malan et al., 2014 ; Marwaha, 2022 ; Meng et al., 2025; Munakata, M., 2018).

The long-term activation of the HPA axis and sympathetic drive under chronic stress is also linked to metabolic disturbances, including insulin resistance and visceral fat deposition, which indirectly increase hypertension risk through heightened peripheral resistance and chronic inflammation (Marwaha, 2022 ; Meng et al., 2025; Munakata, M., 2018). Furthermore, chronic stress may influence health behaviors, encouraging high-risk lifestyles such as smoking, physical inactivity, or poor diet—factors that independently increase

hypertension risk and may potentiate the effect of stress on the cardiovascular system (Marwaha, 2022; Munakata, M., 2018).

As a result, chronic psychological stress, acting in concert with sleep disturbances and other environmental factors, contributes to sustained blood pressure elevation, impaired vascular function, and chronic inflammation, significantly increasing the risk of hypertension and cardiovascular disease (Marwaha, 2022 Meng et al., 2025; Narkiewicz et al., 1997).

Clinical Significance and Practical Implications

In clinical practice, it is essential to recognize sleep disorders as a modifiable factor influencing the progression and management of arterial hypertension, as sleep disturbances increase the risk of an abnormal diurnal blood pressure profile and treatment resistance (Shiina,2024).

In patients with obstructive sleep apnea (OSA) and hypertension, comorbid insomnia symptoms have been associated with a three-fold increase in the risk of resistant hypertension (RH). This suggests that clinicians should actively screen for insomnia symptoms in this patient population from the moment of OSA diagnosis (Shiina,2024). A subsequent prospective study from 2025, involving 386 patients with OSA and uncontrolled hypertension, demonstrated that clinical insomnia ($ISI > 14$) was an independent predictor of RH development. Individuals with insomnia exhibited a several-fold higher risk of RH compared to those without, highlighting the clinical necessity of diagnosing and treating insomnia in this cohort (Wu et al., 2025). A systematic review by Kanclerska et al. (2023) revealed that in the majority of analyzed clinical trials, individuals with HTN exhibited more disrupted sleep architecture and greater sleep fragmentation compared to normotensive individuals, underscoring the importance of sleep quality in the diagnosis and management of hypertension (Kanclerska et al., 2023).

OSA is a major risk factor for arterial hypertension, particularly in the context of treatment-resistant cases. A 2023 literature review indicates that OSA patients more frequently exhibit a non-dipping phenotype (the absence of nocturnal blood pressure reduction) and increased variability in the diurnal pressure profile, resulting from sleep fragmentation and chronic sympathetic activation (Shiina, 2024). These characteristics have direct implications for diagnostic and therapeutic strategies, including the selection of blood pressure monitoring methods and the intensification of both pharmacological and non-pharmacological interventions in patients with comorbid OSA and hypertension (Shiina, 2024).

Consequently, routine clinical practice should incorporate screening questions regarding sleep (difficulty falling asleep, sleep maintenance issues, snoring, daytime sleepiness). Furthermore, polysomnography or ambulatory blood pressure monitoring (ABPM) should be considered, particularly for patients with resistant hypertension, to optimize pressure control and therapeutic outcomes (Shiina, 2024). In summary, contemporary clinical data emphasize that the diagnosis and treatment of sleep disorders not only facilitate the identification of high-risk patients but also exert a tangible impact on therapeutic planning and hypertension outcomes, making them an indispensable element of comprehensive care (Makarem et al., 2021).

Discussion

The findings presented in this review clearly indicate that sleep disorders play a significant role in the development and progression of arterial hypertension. Current scientific evidence consistently demonstrates that both insufficient sleep duration and poor sleep quality are associated with elevated blood pressure values and an increased risk of hypertension. Particularly strong associations have been observed in individuals sleeping less than 7 hours per night, patients with insomnia accompanied by objectively short sleep duration, and those with obstructive sleep apnea. The relationship between sleep disturbances and hypertension is complex and multifactorial, involving autonomic, hormonal, metabolic, and inflammatory mechanisms. Sleep fragmentation and REM sleep deprivation contribute to chronic activation of the sympathetic nervous system and dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis, leading to impaired nocturnal blood pressure dipping and increased vascular tone. Furthermore, hormonal disturbances involving cortisol, leptin, and ghrelin may promote insulin resistance, obesity, and other metabolic abnormalities, thereby increasing cardiovascular risk. The reviewed studies also emphasize that not only sleep duration but also sleep quality plays a crucial role in cardiovascular health. Alterations in sleep architecture, including reduced deep sleep and increased sleep fragmentation, are associated with unfavorable blood pressure profiles and a higher prevalence of resistant hypertension. Moreover, the negative impact of sleep disturbances may be further

amplified by coexisting factors such as obesity, chronic stress, type 2 diabetes, and unhealthy lifestyle habits, all of which contribute synergistically to cardiovascular complications. Despite the growing body of evidence, most available studies remain observational and are partly based on subjective sleep assessments, limiting the ability to establish definitive causal relationships. Therefore, further well-designed prospective and interventional studies are needed to better evaluate the impact of sleep improvement on blood pressure control and cardiovascular risk reduction.

Conclusions

Sleep disorders constitute an important and potentially modifiable risk factor for arterial hypertension and cardiovascular diseases. Both insufficient sleep duration and impaired sleep quality are associated with an increased risk of blood pressure dysregulation and cardiovascular complications. Insomnia and obstructive sleep apnea are of particular clinical importance, as they may not only contribute to the development of hypertension but also impair effective blood pressure control. Therefore, the assessment of sleep duration and quality should be considered an integral component of the prevention, diagnosis, and comprehensive management of patients with arterial hypertension. Early identification and treatment of sleep disorders may improve the effectiveness of antihypertensive therapy and reduce long-term cardiovascular risk. However, further studies are required to better determine the impact of sleep optimization on the prevention and management of arterial hypertension.

Conflicts of Interest: The authors declare no conflict of interest.

REFERENCES

1. al'Absi, M., & Arnett, D. K. (2000). Adrenocortical responses to psychological stress and risk for hypertension. *Biomedicine & Pharmacotherapy*, 54(5), 234–244. [https://doi.org/10.1016/S0753-3322\(00\)80065-7](https://doi.org/10.1016/S0753-3322(00)80065-7)
2. Battisha, A., Kahlon, A., & Kalra, D. K. (2025). Sleep-disordered breathing and hypertension—A systematic review. *Journal of Clinical Medicine*, 14(9), 3115. <https://doi.org/10.3390/jcm14093115>
3. Benarroch, E. E. (2019). Control of the cardiovascular and respiratory systems during sleep. *Autonomic Neuroscience*, 218, 54–63. <https://doi.org/10.1016/j.autneu.2019.01.007>
4. Benjafield, A. V., Sert Kuniyoshi, F. H., Malhotra, A., Martin, J. L., Morin, C. M., Maurer, L. F., Cistulli, P. A., Pépin, J.-L., & Wickwire, E. M. (2025). Estimation of the global prevalence and burden of insomnia: A systematic literature review-based analysis. *Sleep Medicine Reviews*, 82, 102121. <https://doi.org/10.1016/j.smrv.2025.102121>
5. Bigalke, J. A., Shan, Z., & Carter, J. R. (2022). Orexin, sleep, sympathetic neural activity, and cardiovascular function. *Hypertension*, 79(12), 2643–2655. <https://doi.org/10.1161/HYPERTENSIONAHA.122.19796>
6. Bumgarner, J. R., & Nelson, R. J. (2021). Light at night and disrupted circadian rhythms alter physiology and behavior. *Integrative and Comparative Biology*, 61(3), 1160–1169. <https://doi.org/10.1093/icb/icab017>
7. Carley, D. W., & Farabi, S. S. (2016). Physiology of sleep. *Diabetes Spectrum*, 29(1), 5–9. <https://doi.org/10.2337/diaspect.29.1.5>
8. Chen, C., Zhang, B., & Huang, J. (2024). Objective sleep characteristics and hypertension: A community-based cohort study. *Frontiers in Cardiovascular Medicine*, 11, 1336613. <https://doi.org/10.3389/fcvm.2024.1336613>
9. Dai, Y., Vgontzas, A. N., Chen, L., Zheng, D., Chen, B., Fernandez-Mendoza, J., Karataraki, M., Tang, X., & Li, Y. (2024). A meta-analysis of the association between insomnia with objective short sleep duration and risk of hypertension. *Sleep Medicine Reviews*, 75, 101914. <https://doi.org/10.1016/j.smrv.2024.101914>
10. Direksunthorn, T. (2025). Sleep and cardiometabolic health: A narrative review of epidemiological evidence, mechanisms, and interventions. *International Journal of General Medicine*, 18, 5831–5843. <https://doi.org/10.2147/IJGM.S563616>
11. Fernandez-Mendoza, J. (2026). Mechanisms linking insomnia and cardiometabolic disease risk. *Arteriosclerosis, Thrombosis, and Vascular Biology*, 46(3). <https://doi.org/10.1161/ATVBAHA.125.322880>
12. Fishbein, A. B., Knutson, K. L., & Zee, P. C. (2021). Circadian disruption and human health. *Journal of Clinical Investigation*, 131(19), e148286. <https://doi.org/10.1172/JCI148286>
13. Grassi, G., Bombelli, M., Seravalle, G., Dell'Oro, R., & Quarti-Trevano, F. (2010). Diurnal blood pressure variation and sympathetic activity. *Hypertension Research*, 33(5), 381–385. <https://doi.org/10.1038/hr.2010.26>
14. Guo, X., Zheng, L., Wang, J., Zhang, X., Zhang, X., Li, J., & Sun, Y. (2013). Epidemiological evidence for the link between sleep duration and high blood pressure: A systematic review and meta-analysis. *Sleep Medicine*, 14(4), 324–332. <https://doi.org/10.1016/j.sleep.2012.12.001>

15. Han, B., Chen, W. Z., Li, Y. C., Chen, J., & Zeng, Z. Q. (2020). Sleep and hypertension. *Sleep and Breathing*, 24(1), 351–356. <https://doi.org/10.1007/s11325-019-01907-2>
16. Hirshkowitz, M., Whiton, K., Albert, S. M., Alessi, C., Bruni, O., DonCarlos, L., Hazen, N., Herman, J., Katz, E. S., Kheirandish-Gozal, L., Neubauer, D. N., O'Donnell, A. E., Ohayon, M., Peever, J., Rawding, R., Sachdeva, R. C., Setters, B., Vitiello, M. V., Ware, J. C., & Adams Hillard, P. J. (2015). National Sleep Foundation's sleep time duration recommendations: Methodology and results summary. *Sleep Health*, 1(1), 40–43. <https://doi.org/10.1016/j.sleh.2014.12.010>
17. Hosseini, K., Soleimani, H., Tavakoli, K., Maghsoudi, M., Heydari, N., Farahvash, Y., Etemadi, A., Najafi, K., Askari, M. K., Gupta, R., Hakim, D., & Rahimi, K. (2024). Association between sleep duration and hypertension incidence: Systematic review and meta-analysis of cohort studies. *PLOS ONE*, 19(7), e0307120. <https://doi.org/10.1371/journal.pone.0307120>
18. Jafari, B. (2017). Sleep architecture and blood pressure. *Sleep Medicine Clinics*, 12(2), 161–166. <https://doi.org/10.1016/j.jsmc.2017.02.003>
19. Jeong, J., Kim, H., Park, J., Kim, H. J., Jo, H., Lee, S., Lee, K., Park, S., Yim, Y., Woo, H. G., Son, Y., Kim, S., Rhee, S. Y., & Yon, D. K. (2025). National trends in the prevalence of hypertension and diabetes stratified by sleep duration in South Korea, 2009–2022: A nationwide cross-sectional study: Trends and association of sleep duration in hypertension and diabetes. *Medicine*, 104(47), e46042. <https://doi.org/10.1097/MD.00000000000046042>
20. Kancłerska, J., Szymańska-Chabowska, A., Poręba, R., Michałek-Zrąbkowska, M., Lachowicz, G., Mazur, G., & Martynowicz, H. (2023). A systematic review of publications on the associations between sleep architecture and arterial hypertension. *Medical Science Monitor*, 29, 0–0. <https://doi.org/10.12659/MSM.941066>
21. Kretschmar, J., Rath, N., Starke, K. R., Seidler, A., & Freiberg, A. (2026). The effect of exposure to artificial light at night (ALAN) on sleep disturbance: A systematic review and meta-analysis. *Environmental Research*, 292, 123689. <https://doi.org/10.1016/j.envres.2026.123689>
22. Lei, T., Hua, H., Du, H., Xia, J., Xu, D., Liu, W., Wang, Y., & Yang, T. (2024). Molecular mechanisms of artificial light at night affecting circadian rhythm disturbance. *Archives of Toxicology*, 98(2), 395–408. <https://doi.org/10.1007/s00204-023-03647-5>
23. Li, C., & Shang, S. (2021). Relationship between sleep and hypertension: Findings from the NHANES (2007–2014). *International Journal of Environmental Research and Public Health*, 18(15), 7867. <https://doi.org/10.3390/ijerph18157867>
24. Liu, C., Ye, Z., Chen, L., Wang, H., Wu, B., Li, D., Pan, S., Qiu, W., & Ye, H. (2024). Interaction effects between sleep-related disorders and depression on hypertension among adults: A cross-sectional study. *BMC Psychiatry*, 24(1), 482. <https://doi.org/10.1186/s12888-024-05931-9>
25. Lv, Y., Jiang, G., Tan, X., Bao, W., Chen, L., & Liu, L. (2022). Association of sleep patterns and lifestyles with incident hypertension: Evidence from a large population-based cohort study. *Frontiers in Cardiovascular Medicine*, 9, 847452. <https://doi.org/10.3389/fcvm.2022.847452>
26. Makarem, N., Alcántara, C., Williams, N., Bello, N. A., & Abdalla, M. (2021). Effect of sleep disturbances on blood pressure. *Hypertension*, 77(4), 1036–1046. <https://doi.org/10.1161/HYPERTENSIONAHA.120.14479>
27. Makarem, N., Shechter, A., Carnethon, M. R., Mullington, J. M., Hall, M. H., & Abdalla, M. (2019). Sleep duration and blood pressure: Recent advances and future directions. *Current Hypertension Reports*, 21(5), 33. <https://doi.org/10.1007/s11906-019-0938-7>
28. Malan, N. T., Stalder, T., Schlaich, M. P., Lambert, G. W., Hamer, M., Schutte, A. E., Huisman, H. W., Schutte, R., Smith, W., Mels, C. M. C., Van Rooyen, J. M., & Malan, L. (2014). Chronic distress and acute vascular stress responses associated with ambulatory blood pressure in low-testosterone African men: The SABPA Study. *Journal of Human Hypertension*, 28(6), 393–398. <https://doi.org/10.1038/jhh.2013.124>
29. Marwaha, K. (2022). Examining the role of psychosocial stressors in hypertension. *Journal of Preventive Medicine and Public Health*, 55(6), 499–505. <https://doi.org/10.3961/jpmph.21.266>
30. McCarley, R. W. (2007). Neurobiology of REM and NREM sleep. *Sleep Medicine*, 8(4), 302–330. <https://doi.org/10.1016/j.sleep.2007.03.005>
31. Meng, S., He, H., Li, D., & Li, N. (2025). Advances in understanding the role of autonomic dysfunction in the pathogenesis and progression of hypertension. *Cardiology Discovery*. <https://doi.org/10.1097/CD9.0000000000000167>
32. Munakata, M. (2018). Clinical significance of stress-related increase in blood pressure: Current evidence in office and out-of-office settings. *Hypertension Research*, 41(8), 553–569. <https://doi.org/10.1038/s41440-018-0053-1>
33. Narkiewicz, K., & Somers, V. K. (1997). The sympathetic nervous system and obstructive sleep apnea: Implications for hypertension. *Journal of Hypertension*, 15(12), 1613–1619. <https://doi.org/10.1097/00004872-199715120-00062>
34. Nunez, S. G., Rabelo, S. P., Subotic, N., Caruso, J. W., & Knezevic, N. N. (2025). Chronic stress and autoimmunity: The role of HPA axis and cortisol dysregulation. *International Journal of Molecular Sciences*, 26(20), 9994. <https://doi.org/10.3390/ijms26209994>

35. Pechanova, O., Paulis, L., & Simko, F. (2014). Peripheral and central effects of melatonin on blood pressure regulation. *International Journal of Molecular Sciences*, 15(10), 17920–17937. <https://doi.org/10.3390/ijms151017920>
36. Pengpid, S., Peltzer, K., & Hajek, A. (2025). Prevalence and factors associated with short and long sleep duration among community-dwelling persons 40 years and older: Longitudinal evidence from rural South Africa, 2015–2022. *Sleep and Breathing*, 29(6), 360. <https://doi.org/10.1007/s11325-025-03547-1>
37. Qi, J., Yang, M., Zhang, S., He, C., Bao, X., He, B., Lin, Y., Chu, J., & Chen, K. (2025). The association between sleep duration and the risk of hypertension: A systematic review and meta-analysis of cohort studies. *Journal of General Internal Medicine*, 40(16), 3848–3858. <https://doi.org/10.1007/s11606-025-09398-6>
38. Schiffrin, E. L. (2004). The many targets of aldosterone. *Hypertension*, 43(5), 938–940. <https://doi.org/10.1161/01.HYP.0000123573.60340.9b>
39. Shiina, K. (2024). Obstructive sleep apnea-related hypertension: A review of the literature and clinical management strategy. *Hypertension Research*, 47(11), 3085–3098. <https://doi.org/10.1038/s41440-024-01852-y>
40. Somers, V. K., Dyken, M. E., Mark, A. L., & Abboud, F. M. (1993). Sympathetic-nerve activity during sleep in normal subjects. *New England Journal of Medicine*, 328(5), 303–307. <https://doi.org/10.1056/NEJM199302043280502>
41. Tähkämö, L., Partonen, T., & Pesonen, A.-K. (2019). Systematic review of light exposure impact on human circadian rhythm. *Chronobiology International*, 36(2), 151–170. <https://doi.org/10.1080/07420528.2018.1527773>
42. Tobushi, T., & Floras, J. S. (2024). Sleep apnea, autonomic disturbances, and blood pressure variability. *Hypertension*, 81(9), 1837–1844. <https://doi.org/10.1161/HYPERTENSIONAHA.124.20433>
43. Vorster, A. P. A., Van Someren, E. J. W., Pack, A. I., Huber, R., Schmidt, M. H., & Bassetti, C. L. A. (2024). Sleep health. *Clinical and Translational Neuroscience*, 8(1), 8. <https://doi.org/10.3390/ctn8010008>
44. Wang, Y., Chen, X. X., Lu, F. Y., Yan, Y. R., Li, S. Q., Zhang, L., Lin, Y. N., & Li, Q. Y. (2025). Association between sleep duration and hypertension risk in patients with obstructive sleep apnea. *Npj Primary Care Respiratory Medicine*, 35(1), 26. <https://doi.org/10.1038/s41533-025-00429-7>
45. Whelton, P. K., Carey, R. M., Mancia, G., Kreutz, R., Bundy, J. D., & Williams, B. (2022). Harmonization of the American College of Cardiology/American Heart Association and European Society of Cardiology/European Society of Hypertension blood pressure/hypertension guidelines. *European Heart Journal*, 43(35), 3302–3311. <https://doi.org/10.1093/eurheartj/ehac432>
46. Wood, C., Bianchi, M. T., Yun, C.-H., Shin, C., & Thomas, R. J. (2020). Multicomponent analysis of sleep using electrocortical, respiratory, autonomic and hemodynamic signals reveals distinct features of stable and unstable NREM and REM sleep. *Frontiers in Physiology*, 11, 592978. <https://doi.org/10.3389/fphys.2020.592978>
47. Wu, H., Xie, J., & Wu, C. (2025). The role of insomnia in the development of resistant hypertension in uncontrolled hypertensive patients with obstructive sleep apnea: A prospective study. *Sleep Medicine*, 131, 106508. <https://doi.org/10.1016/j.sleep.2025.106508>
48. Xu, Q., Lin, Z., Chen, Y., & Huang, M. (2025). Association between sleep duration and patterns and obesity: A cross-sectional study of the 2007–2018 national health and nutrition examination survey. *BMC Public Health*, 25(1), 1460. <https://doi.org/10.1186/s12889-025-22433-9>
49. Xu, Y., Qu, B., Liu, F., Gong, Z., Zhang, Y., & Xu, D. (2023). Sleep deprivation and heart rate variability in healthy volunteers: Effects of REM and SWS sleep deprivation. *Computational and Mathematical Methods in Medicine*, 2023(1), 7121295. <https://doi.org/10.1155/2023/7121295>
50. Zasadzińska-Stempniak, K., Zajączkiewicz, H., & Kukwa, A. (2024). Prevalence of obstructive sleep apnea in the young adult population: A systematic review. *Journal of Clinical Medicine*, 13(5), 1386. <https://doi.org/10.3390/jcm13051386>

List of abbreviations

Abbreviation	Full term
HTN	Arterial hypertension
BP	Blood pressure
OSA	Obstructive sleep apnea
NREM	Non-rapid eye movement sleep
REM	Rapid eye movement sleep
EEG	Electroencephalography
HRV	Heart rate variability
LF	Low frequency (component of HRV)
HF	High frequency (component of HRV)
LF/HF	Ratio of low- to high-frequency HRV components
ANS	Autonomic nervous system
SNS	Sympathetic nervous system
PNS	Parasympathetic nervous system
HPA axis	Hypothalamic–pituitary–adrenal axis
RAAS	Renin–angiotensin–aldosterone system
ABPM	Ambulatory blood pressure monitoring
RH	Resistant hypertension
ISI	Insomnia Severity Index
PSG	Polysomnography
ISSD / ISSOD	Insomnia with short sleep duration
CRP	C-reactive protein
IL-6	Interleukin-6
BMI	Body mass index
NHANES	National Health and Nutrition Examination Survey
HR	Hazard ratio
OR	Odds ratio
RR	Relative risk
CI	Confidence interval