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REVIEW

The Link Between Sleep Health Dimensions and Hypertension for Cardiovascular Health

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Abstract

Purpose of Review Hypertension, a leading modifiable risk factor for cardiovascular diseases, is projected to contribute substantially to the global burden of cardiovascular health by 2050. Sleep has emerged as a potential factor in the prevention and management of hypertension. This review synthesizes recent evidence on the relationship between sleep health dimensions and hypertension, highlighting clinical significance, identifying gaps, and outlining future research directions.

Recent Findings Prolonged sleep latency, greater wake after sleep onset, both short and long sleep duration, poor overall sleep quality, and reduced sleep efficiency are associated with a higher risk of hypertension. However, most studies are cross-sectional and rely primarily on subjective measures, often limited to sleep duration.

Summary of Findings A multidimensional approach to sleep health may reduce hypertension risk and improve blood pressure control. More longitudinal and intervention studies in varied populations are needed to develop tailored sleep health recommendations and guide clinical guidelines.

Keywords Cardiovascular health · Hypertension · Blood pressure management · Sleep health · Cardiovascular disease

Abbreviations

AHA	American Heart Association
ANS	Autonomic Nervous System
BMI	Body Mass Index
CVDs	Cardiovascular Diseases
CVD	Cardiovascular Disease
CVH	Cardiovascular Health
LE8	Life's Essential 8
NREM	Non-Rapid Eye Movement
PA	Physical Activity
PSQI	Pittsburgh Sleep Quality Index
REM	Rapid Eye Movement
WASO	Wake After Sleep Onset

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Introduction

Cardiovascular diseases (CVDs) remain one of the major causes of mortality globally and in the United States (US) [1]. In 2021, the global prevalence of CVDs was approximately 612.06 million people [1]. The overall prevalence of CVDs (including hypertension) in the US between 2021 and 2023 was 48.9%, affecting approximately 130.6 million adults [1]. One of the most prevalent modifiable risk factors for CVDs is hypertension, or high blood pressure [1]. Hypertension is characterized by a systolic blood pressure of ≥ 130 mm Hg and a diastolic blood pressure of ≥ 80 mm Hg, or using antihypertensive medication [2]. Hypertension is projected to have one of the highest burdens among cardiovascular health (CVH) metrics in the US, increasing from 51.2% in 2010 to 61.0% by 2050, underscoring the need for targeted interventions to improve CVH [1]. CVH

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is a comprehensive concept that encompasses various health factors and behaviors aimed at promoting optimal cardiovascular function, supporting healthy aging throughout the lifespan, enhancing longevity, and preventing disease [3].

In 2010, the American Heart Association (AHA) proposed the construct of ideal CVH, which is based on specific metrics and strategies designed to monitor and improve cardiovascular well-being [3]. Ideal CVH was defined as the presence of seven favorable health behaviors and health factors, coupled with the absence of clinical CVD. To promote optimal CVH, individuals were required to attain the specified levels of these seven components. These components include dietary quality, physical activity (PA), cigarette smoking exposure, measures of body mass index (BMI), total cholesterol, fasting blood glucose, and blood pressure [4]. Originally known as "Life's Simple 7," this indicator was subsequently updated to "Life's Essential 8" (LE8) with the addition of sleep as a new CVH metric [4]. The eight CVH metrics in LE8 are classified into two domains: health behaviors and health factors. The health factors domain encompasses BMI, blood lipids, blood pressure, and blood glucose, while the health behaviors domain includes diet, PA, nicotine exposure, and sleep duration [4]. Sleep was incorporated into the CVH metrics in 2022 since it has been linked to CVD events and influences the other seven components of the LE8 model [1, 4].

Sleep is a multidimensional construct encompassing multiple domains across several levels, behavioral and physiological [5]. The multidimensional constructs of sleep health include: sleep stage architecture (rapid eye movement and non-rapid eye movement stages), sleep duration, sleep quality (subjective perception), sleep continuity (sleep efficiency), sleep timing (circadian rhythm alignment), sleep regularity, absence of sleep disorders, and sleepiness/napping/alertness [5–8]. Yet, sleep duration is the only measure of sleep health included in the LE8 [5–7]. Notably, sleep has the capacity to regulate blood pressure [9, 10]. During sleep, blood pressure typically exhibits a nocturnal decline, which is known as nocturnal dipping [9–11]. Disruption of this circadian blood pressure pattern is strongly associated with cardiovascular risk [11], emphasizing the vital role of appropriate blood pressure dipping in cardiovascular function and disease prevention [10, 12]. Blood pressure dipping supports the regulation of blood pressure and is essential to prevent the risk of hypertension [9, 11]. Both short and long sleep duration, along with poor sleep quality, are linked to a higher risk of hypertension [13–15]. Similarly, negative characteristics of sleep continuity (sleep latency, sleep efficiency, wake after sleep onset) and sleep disorders increase the risk of hypertension or its prevalence [16–21], suggesting that multiple dimensions of sleep contribute to blood pressure management. A review by Buysse [5], highlighted several dimensions of sleep health and proposed a model that linked sleep dimensions to various health outcomes, such as hypertension [5]. More research on the role of sleep will be needed as sleep continues to be recognized as an essential factor impacting hypertension and ultimately CVD.

The AHA recently emphasized the need to consider multiple dimensions of sleep in the development of interventions to reduce CVD risk [8]. Therefore, understanding how various sleep dimensions jointly relate to established CVD risk factors, particularly hypertension, is critical for advancing CVH initiatives. Despite growing interest in sleep as a modifiable determinant of health, few reviews have synthesized evidence on the combined impact of multiple sleep constructs on hypertension. In this review of the past five (5) years of literature, we exclude sleep disorders that directly disrupt sleep health, such as insomnia and obstructive sleep apnea [22], and instead focus on key dimensions of sleep health, specifically sleep quality, sleep duration, and sleep continuity, which are modifiable, measurable, and relevant across populations with or without diagnosed sleep disorders [5]. These dimensions are consistent with Buysse's proposed definition of a sleep health model [5, 23], which focused on various sleep dimensions and their association with specific health outcomes [5, 23]. Although these indicators are frequently examined in isolation, considering them jointly may provide a more comprehensive understanding of sleep health by integrating both subjective and objective components. Further, we highlight the epidemiologic and clinical relevance of these sleep dimensions and identify key gaps and future directions for research on hypertension within the broader framework of CVH.

Sleep and Blood Pressure Regulation: Mechanistic Insights into Hypertension Risk

Sleep is defined as a naturally occurring and reversible state of perpetual disengagement from the external environment, decreased consciousness, immobility, and a propensity patterned by homeostatic and circadian factors [24]. Sleep consists of several cycles between three stages of non-rapid eye movement (NREM) and rapid eye movement (REM) [9], as well as transitions between wakefulness [24]. NREM sleep has three stages, and each stage has a unique physiology, duration, and blood pressure pattern [25]. The NREM stages of sleep include N1 (light stage) to N2 (intermediate stage) and N3 (deep sleep) [26]. Blood pressure gradually decreases as sleep cycles from N1 to N3 [27], commonly described as dipping. Dipping supports a decline in nocturnal systolic blood pressure by 10% (~ 10 mmHg), compared to daytime systolic blood pressure. Unfortunately, when blood pressure fails to dip, it remains high. Sleep is

essential to managing nocturnal blood pressure [26]. Two of the most common mechanisms that may link sleep and blood pressure regulation include autonomic nervous control and circadian rhythm [27]. Misalignment of the circadian system and the autonomic nervous system is linked to impaired sleep and blood pressure regulation. Disruption to blood pressure regulation increases the risk of hypertension.

Circadian System

Circadian rhythm mechanisms coordinate the functions of multiple physiological systems, including the cardiovascular system, and regulate cellular and molecular processes. Circadian rhythm refers to an intrinsic 24-hour biological cycle driven by internal oscillators, maintained independently of environmental changes, but modulated by zeitgebers (time cues) to synchronize with the 24-hour day-night cycle of the Earth [28]. A central clock within the suprachiasmatic nucleus in the hypothalamus controls the circadian rhythm. This central or master clock controls the peripheral clocks located in mammalian tissues, which ensures all the clocks synchronize. In cardiovascular tissues, peripheral clocks regulate the circadian rhythm of cardiovascular function through communication with the central clock. In cardiovascular systems, one of the most common circadian rhythms is the blood pressure rhythm [29]. Blood pressure follows a daily pattern, rising in the morning (morning surge), remaining relatively stable during the day, and decreasing at night by about 10–20% compared to daytime levels [11, 30]. Blood pressure non-dipping and reverse dipping during sleep may impair the cardiovascular system [31]. Therefore, a dip that is less than 10% is a strong predictor of adverse cardiovascular outcomes [32]. Similarly, reverse dipping increases the risk of hypertension and cardiovascular events. Reverse dipping is defined as higher nocturnal blood pressure relative to daytime levels. Reverse dipping and non-dipping may impact CVH independently. More importantly, through interactions with the cardiovascular system, circadian oscillation of the autonomic nervous system (ANS) activity controls the sleep-wake cycle [33].

Autonomic Nervous System

The ANS coordinates with the circadian system to transfer information to the peripheral organs of the body [34]. The ANS consists of two branches: the sympathetic and parasympathetic nervous systems, which together coordinate communication between the central nervous system and peripheral organs [34]. Sleep induces changes in autonomic function and other physiological processes that influence blood pressure regulation [9]. In the NREM state of sleep, there is a progressive decline in the activity of the sympathetic nerve along with an increase in parasympathetic cardiac modulation. This causes a reduction in blood pressure and heart rate variability, and the elevation of arterial baroreflex sensitivity [9]. These autonomic changes are accompanied by more regular and stable breathing patterns, which further support nocturnal regulation of cardiovascular function.

Blood pressure dipping may occur during the deep sleep stage of NREM, when blood pressure and heart rate gradually decline across the stages and cycles of sleep [9, 35]. Sympathetic nervous system activity and blood pressure levels increase during REM sleep state. However, NREM sleep state leads to a decrease in sympathetic activity and a progressive increase in parasympathetic activity, causing a decrease in cardiac output and blood pressure. Lowered sympathetic nervous system activity, reduced heart rate variability, and increased baroreflex sensitivity help to regulate blood pressure [9]. In contrast, sleep loss promotes sympathetic activation, which reduces NREM sleep, which may lead to a sustained increase in blood pressure levels [9, 35]. These changes increase the risk of serious cardiovascular events. Alterations to normal sleep physiology may contribute to the development and progression of hypertension, which negatively impacts CVH.

Sleep Dimensions and Their Association with Hypertension and Cardiovascular Health

Sleep architecture plays an essential role in blood pressure regulation and overall function of cardiovascular systems. Major sleep parameters include sleep efficiency, sleep quality, sleep latency or sleep onset latency, sleep duration or total sleep time, and wake after sleep onset. Sleep efficiency and sleep duration can be assessed using both subjective and objective sleep measures [36]. However, the objective measurements of sleep efficiency (e.g. polysomnography, actigraphy) may be more accurate [36]. Wake after sleep onset and sleep latency can be assessed subjectively [8] but are typically better measured objectively [36]. Sleep is inherently complex due to the various stages, the circadian rhythms, and physiological processes. Consequently, integrating multiple sleep parameters, captured through both subjective and objective methods, may provide deeper insights on the function of sleep in the management of blood pressure and prevention of hypertension (Table 1).

Sleep Duration

Sleep duration reflects the total amount of sleep gained over the night or across 24 h [8, 36]. Sleep duration has been widely examined as a behavioral determinant of

Table 1 Summary of Reviewed Studies on Sleep Health Dimensions and Blood Pressure Measures Over the Past Five Years[a]

Authors, Year	Study Design	Population	Sleep Dimension(s)	Sleep Measurement (Methods)	Outcome(s)
Abdalla et al. [41]	Cross-sectional	Adults aged 18–30 years, CARDIA prospective cohort, US, (n = 647)	Sleep Duration	Subjective (Sleep diaries, self-reported questionnaire) Objective (Wrist Actiwatch 2 accelerometer, actigraphy, ABPM)	Nocturnal hypertension; non-dipping, SBP; DBP
Chang et al. [39]	Cross-sectional	Adults aged 30–79 years, Sichuan province, China, (n = 55,687)	Sleep Duration	Subjective (self-reported questionnaire)	Hypertension prevalence; odds of hypertension; SBP; DBP
Li and Shang [37]	Cross-sectional	Adults aged 30–79 years, NHANES 2007–2014, US, (n = 12,166)	Sleep Duration, Sleep Quality	Subjective (self-reported questionnaire)	Hypertension; SBP; DBP
Yang et al. [45]	Cross-sectional	Adults ≥ 18 years, Emin County, Xinjiang, China, (n = 33,341)	Sleep Quality	Subjective (PSQSI)	Hypertension prevalence, SBP, DBP
Yuan et al. [15]	Prospective Cohort	Adults ≥ 18 years, Emin, Xinjiang, China, (n = 9,344)	Sleep Duration, Sleep Efficiency, Sleep Latency, Sleep Quality	Subjective (PSQI)	Incident hypertension; SBP; DBP
Zhao et al. [50]	Cross-sectional	Adults aged ≥ 40 years, Sleep Heart Health Study, US, (n = 5804)	Sleep Efficiency, Wake After Sleep Onset	Objective (PSG)	Hypertension prevalence
Lv et al. [14]	Prospective cohort	Adults aged 37–73 years; large-scale prospective cohort, UK, (n = 165,493)	Sleep Duration, Sleep Quality	Subjective (self-reported electronic questionnaire)	Incident hypertension; SBP; DBP
Xia et al. [49]	Cross-sectional	Adults > 18 years, Sleep Medical Center, Xuan Wu Hospital, (n = 3124)	Sleep efficiency, Total sleep duration, wake after sleep onset	Objective (PSG)	Hypertension prevalence
Aremu et al. [43]	Cross-sectional	Adults ≥ 18 years, Ibadan, Nigeria, (n = 3,635)	Sleep Quality	Subjective (Sleep quality scale)	Hypertension; SBP; DBP
Aqua et al. [48]	Cross-sectional	Adults aged 25–74 years; Midlife in the United States (MIDUS II) Study, US, (n = 7000)	Sleep Efficiency, Wake After Sleep Onset	Objective (wrist actigraphy)	Odds of hypertension; SBP, DBP
Pérez-Vega et al. [38]	Cross-sectional	Hospitalized adult patients, Mexico, (n = 381)	Sleep Duration, Sleep Latency	Subjective (PSQI)	SBP; DBP
Taporoski et al. 2025 [53]	Cross-sectional	Adults aged 18 to 91 years, Baependi Heart Study (BHS), (n = 1102)	Wake After Sleep Onset	Objective (PSG)	Odds of hypertension; SBP; DBP
Xiang et al. [52]	Cross-sectional	Adults aged 25–89 years, South China Cohort (SCC), China, (n = 72,476)	Sleep Latency	Subjective (PSQI)	Hypertension prevalence; odds of hypertension
Minetto et al. [54]	Cross-sectional	Adults ≥ 18 years cardiovascular risk clinic patients in Buenos Aires, Argentina, (n = 2449)	Sleep Duration, Sleep Quality	Subjective (PSQI, self-reported sleep hours)	Isolated nocturnal hypertension; daytime BP; nocturnal BP
Srimoragot et al. [44]	Cross-sectional	Woman aged 40–65 years, Thailand, (n = 125)	Sleep Quality, Sleep Efficiency	Subjective (PQSI)	DBP

Abbreviations: ABPM Ambulatory Blood Pressure Measurement, *CARDIA* Coronary Artery Risk Development in Young Adults study, DBP Diastolic Blood Pressure, *PQSI* Pittsburgh Quality Sleep Index, *PSG* Polysomnography, *SBP* Systolic Blood Pressure, *US* United States. *Notes*: The table summarizes studies published in the past 5 years that were reviewed for the link between sleep health dimensions and blood pressure measures.

cardiovascular risk, particularly in relation to hypertension [4, 8]. Early work focused primarily on insufficient sleep as a risk factor; however, more recent evidence suggests a broader pattern. Cross-sectional analyses of nationally representative samples show consistent patterns, with shorter sleep duration associated with higher odds of hypertension after adjusting for demographic and behavioral factors [37]. One cross-sectional study found a positive correlation between sleep duration and diastolic blood pressure [54]. Another study cross-sectionally examined the relationship between sleep duration (measured in hours) and blood pressure. This study

found a negative association between sleep hours and systolic and diastolic blood pressure [38]. Prospective studies further indicated that individuals with more favorable sleep patterns, including adequate duration, have a lower risk of developing hypertension over time [14]. While short sleep appears to contribute to hypertension risk, it likely operates alongside other behavioral and physiological factors. In a large population-based study of adults in southwest China, long sleep duration (≥ 9 h) was linked to higher odds of hypertension. Risk increased beyond approximately 6–7 h of sleep, demonstrating a non-linear (J-shaped) relationship, with relatively stable risk at shorter durations followed by a rise at longer durations. Among women, a U-shaped pattern was observed, suggesting elevated risk at both shorter and longer sleep durations [39]. These findings align with other population-level studies indicating that extended sleep, particularly when considered alongside sleep timing, may correspond to higher hypertension risk [40]. Despite these patterns, findings remain inconsistent; some evidence suggests no association between short sleep and nocturnal blood pressure or dipping status after accounting for relevant covariates [41]. Differences in measurement approaches, population characteristics, and co-occurring sleep disturbances may contribute to these inconsistencies. Overall, current evidence suggests that both insufficient and extended sleep may relate to hypertension risk, although the magnitude and direction of these associations vary. This variability supports examining sleep duration within the broader context of sleep patterns and blood pressure regulation rather than as an isolated exposure.

Sleep Quality

Sleep quality is a multidimensional construct characterized by sleep continuity, efficiency, disturbances, and daytime alertness. It is frequently evaluated subjectively using instruments such as the Pittsburgh Sleep Quality Index (PSQI), which integrates these domains into a global score [42]. In this section, we focus on studies examining the overall (global) sleep quality score in relation to hypertension and blood pressure, while individual components of aspects of sleep quality are addressed in subsequent sections. Although observational studies consistently associate poor sleep quality with hypertension, the relationship with mean systolic and diastolic blood pressure is less uniform. This discrepancy in the available data implies that sleep quality may be more closely related to broader cardiovascular regulatory mechanisms rather than individual blood pressure metrics [42]. Evidence on the relationship between sleep quality and measures of blood pressure remains somewhat inconsistent. A cross-sectional study examining subjective sleep quality and blood pressure measures found that moderate and poor sleep quality increase the prevalence of hypertension compared to good sleep quality [43]. Self-reported sleep measured using PSQI in a cross-sectional study on menopausal Thai women showed that sleep quality had a negative correlation with diastolic blood pressure [44]. Another cross-sectional study found a higher prevalence of hypertension with subjectively measured poorer sleep quality [45]. Although these studies highlight that sleep quality influences blood pressure outcomes, another cross-sectional study examining sleep quality and hypertension outcomes suggests that sleep quality is not independently associated with nocturnal hypertension [41]. Prospective studies offer further insight into temporal relationships between sleep quality and hypertension risk. In a large community-based cohort, poorer quality of sleep was associated with a greater incidence of hypertension over a period of three years [15]. This relationship was not uniform across individual domains. Rather than acting through a single pathway, sleep quality appears to impact blood pressure in several ways. The available evidence suggests that poor sleep quality is associated with greater hypertension risk, particularly in relation to sleep continuity.

Sleep Continuity

Sleep continuity reflects the initiation and maintenance of sleep [8]. It is typically characterized by sleep efficiency and includes other metrics, such as sleep latency, number of awakenings, and wake after sleep onset [8]. Sleep continuity provides a better understanding of sleep quality by measuring how long it takes to fall asleep, how often and for how long an individual wakes up during the night, and whether they wake up earlier than intended [46]. Sleep quality provides a perception of an individual that relies on subjective assessment. However, objective assessments provide additional insight into continuity by capturing sleep measures such as wake after sleep onset and sleep efficiency. These measures are all essential for cardiovascular health [8, 36].

Sleep Efficiency

Sleep efficiency refers to the percentage of the total period spent in bed sleeping [47]. Several studies suggest that reduced or lower sleep efficiency may increase hypertension prevalence and elevate systolic and diastolic blood pressure. Conversely, a cross-sectional analysis of menopausal Thai women based on self-reported sleep efficiency measured using PSQI showed a positive correlation between sleep efficiency and diastolic blood pressure [44].

This suggests that higher mean sleep efficiency may be linked to higher diastolic blood pressure. However, one community-based prospective cohort study found that individuals reporting subjective low sleep efficiency had a higher risk of incident hypertension compared to those with high sleep efficiency [15]. Further, objectively measured sleep efficiency using actigraphy has been associated with both daytime and nighttime blood pressure. The data from the Midlife in the United States (MIDUS II) study demonstrated that low sleep efficiency measured using actigraphy was linked to the prevalence of hypertension and higher systolic and diastolic blood pressure [48]. Sleep efficiency has shown an association with lower odds of hypertension in a cross-sectional study involving Chinese adults with obstructive sleep apnea disorder, where sleep was assessed using objective polysomnography [49]. Polysomnography is considered the gold standard to objectively assess sleep, but it does not encompass all aspects of sleep health, supporting the use of complementary assessment measures [8]. Similarly, the results of a community-based population study using objectively measured polysomnography found that high sleep efficiency lowers the prevalence of hypertension [50]. The result of subjectively measured sleep efficiency differs from that of objectively measured sleep efficiency. The studies using objective measures suggest that higher sleep efficiency lowers blood pressure levels and reduces hypertension prevalence. Importantly, these findings highlight the need for both subjective and objective measures to capture the entirety of sleep health of an individual. In addition, greater time spent asleep, rather than time spent in bed, may be beneficial for CVH by lowering hypertension risk.

Sleep Latency

Sleep latency, the time it takes one to fall asleep, is another measure of sleep continuity that has been linked to hypertension. Sleep latency is the period of time from lights out or wakefulness to the time of falling asleep [36, 47]. In a study examining hospitalized Mexican adults by subjective sleep latency assessed using the PSQI, as well as self-reported diagnosis of hypertension, individuals who had lower scores for sleep latency (shorter time to fall asleep) reported a diagnosis of hypertension compared to individuals who did not have a diagnosis of hypertension [38]. This result conflicts with evidence that suggests longer sleep latency in hypertensive individuals [51]. Additionally, the same study found that sleep latency was positively associated with systolic blood pressure. Findings in another cross-sectional study of hospitalized adults in China aged 27 to 92 years found that longer nighttime sleep latency assessed by using the PSQI questionnaire was associated with hypertension [19]. The association between sleep latency and hypertension remained after adjusting for confounders. Further, nocturnal sleep latency had a positive correlation with hypertension in both men and women in the same study. Another study of adults aged 25–89 years from a South China cohort found that prolonged self-reported sleep latency, characterized by two components: taking longer to fall asleep and difficulty initiating sleep, was associated with higher odds of hypertension [52]. Most of the results from these studies underscore the potential importance of having a shorter time to achieve sleep onset to improve blood pressure control and reduce hypertension risk.

Wake After Sleep Onset

Wake after sleep onset (WASO) refers to a period of wakefulness that occurs after initial sleep onset and before final wakening [8, 36, 47]. Evidence suggests that wake after sleep onset is associated with hypertension prevalence [50, 53]. In a community population-based Sleep Heart Health Study, objectively measured WASO had an association with a higher prevalence of hypertension [50]. Similarly, findings from the Baependi Heart Study, a Brazilian-based cohort, showed that objectively measured longer WASO was associated with elevated systolic blood pressure and higher odds of hypertension in men [53]. However, not all studies report consistent findings; one study found no significant association between objectively measured WASO and hypertension prevalence or systolic and diastolic blood pressure for participants in the MIDUS II study [48]. Overall, these findings suggest that greater wakefulness after sleep onset may contribute to higher blood pressure and hypertension risk, although evidence remains mixed. Reducing wake after sleep onset may therefore represent a potential target for improving blood pressure regulation.

Moreover, most of these studies collectively highlighted that short or long sleep duration, poor sleep quality, reduced sleep efficiency, longer sleep latency, and longer WASO contribute to negative blood pressure measures. Simultaneously, emerging evidence suggests that hypertension may also negatively impact sleep quality, sleep latency, sleep duration, sleep efficiency, and wake after sleep onset [51]. Although this review does not focus on the impact of hypertension on the dimensions of sleep health, a bidirectional relationship exists to impact CVH. Therefore, improving these essential sleep parameters may manage blood pressure outcomes to enhance CVH (Fig. 1).

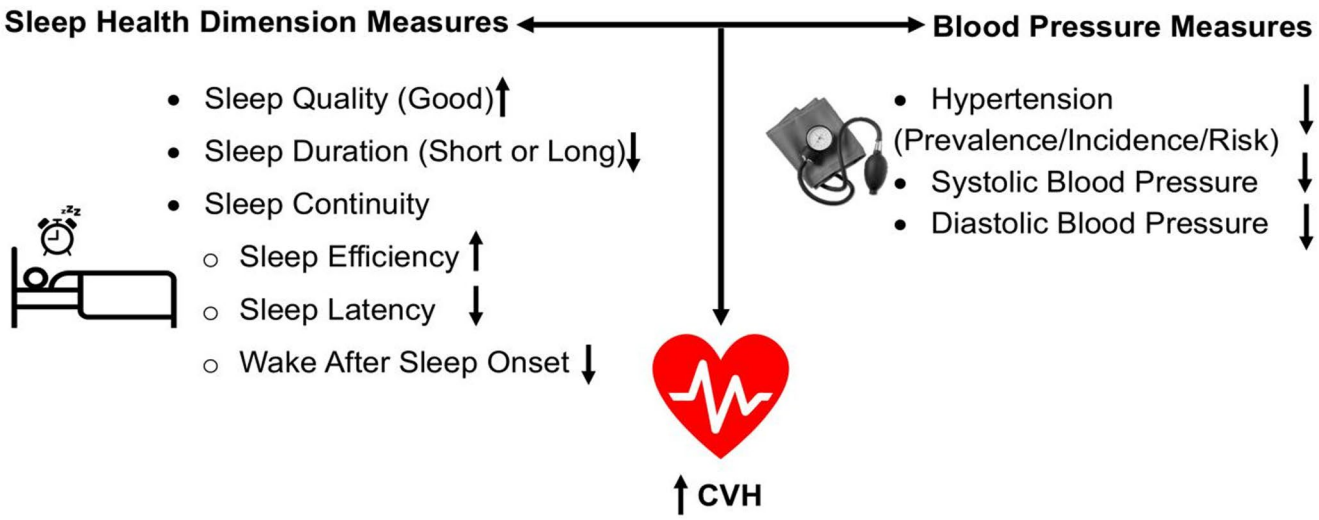


Fig. 1 Proposed Link Between Sleep Dimension and Blood Pressure Measures for CVH. Proposed Bidirectional Relationship between Sleep Health Dimension Measures and Blood Pressure Measures to Improve CVH

Conclusion

This review highlights a consistent association between poor sleep health across multiple sleep health dimensions, which includes sleep duration, continuity, efficiency, and quality, and increased risk of hypertension. Although the current evidence included in this review supports the role of sleep in blood pressure regulation, much of the literature is based on cross-sectional designs, limiting causal interpretation. In addition, much of the existing literature relies on subjective sleep measures, such as self-reported duration and quality, and is subject to recall bias and measurement error. Many of the studies reviewed had considerable heterogeneity in how the subjective measures were defined and the approach used across studies [15, 38, 44, 52, 54], identifying a need for a standardized method. Most of the studies included focused on sleep duration as the key exposure, often neglecting other important dimensions of sleep health. Inclusion of objective measures such as actigraphy and polysomnography will improve the precision and validity of the findings within the sleep health literature [55, 56]. Considering a multidimensional framework for sleep health provides a more comprehensive understanding of its contribution to hypertension risk and overall CVH and demonstrates the need to move beyond single sleep metrics in research and clinical practice.

Clinical Significance

Evidence presented here has highlighted the importance of incorporating sleep assessment into routine clinical care. Sleep is a critical modifiable health behavior to target blood pressure control and CVH. The 2025 American College of Cardiology/AHA hypertension guidelines suggest healthy lifestyle behaviors for hypertension prevention and blood pressure management [2]. Consequently, understanding various dimensions of sleep health specifically linked to blood pressure measures provides a comprehensive approach to potentially managing blood pressure and preventing hypertension for better CVH (Fig. 1). Integrating sleep health into standard risk assessment and lifestyle counseling could improve early detection and management of hypertension. Additionally, promoting healthy sleep behaviors may complement existing interventions and provide a more effective global CVD prevention approach.

Future Research Directions

Future research should prioritize longitudinal and targeted interventions to elucidate the causal relationship between sleep health and hypertension risk. There is a critical need for replication of findings across varied populations to strengthen the evidence base and generalizability. Integration of both subjective and objective sleep measures to assess the biological mechanisms and the effectiveness of sleep-related interventions for improvement of blood pressure control is warranted. Moreover, studies should focus on examining combined or multidimensional sleep factors as well as biological and behavioral mechanisms to inform targeted, evidence-based sleep interventions for hypertension prevention and management.

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  ○ This cross-sectional study assessed objectively measured sleep efficiency and WASO in participants with high WASO and low sleep efficiency. The findings suggest that low sleep efficiency has an association with higher systolic and diastolic blood pressure as well as hypertension prevalence.

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  ○ The findings of this cross-sectional study found that individuals with sleep apnea and individuals who snored had lower percentages of objectively assessed sleep efficiency and higher systolic and diastolic blood pressure levels, as well as elevated odds of hypertension.

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  ○ In this cross-sectional study, the findings of subjectively assessed sleep quality and duration suggest a correlation of both sleep measures with blood pressure. Sleep duration was correlated with diastolic pressure, while sleep quality was correlated with both systolic and diastolic pressures.

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Data Availability No datasets were generated or analysed during the current study.

Declarations

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Competing Interests The authors declare no competing interests.

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