

# RELATIONSHIP BETWEEN OBESITY, SLEEP QUALITY, AND BLOOD PRESSURE CONTROL IN PATIENTS WITH ESSENTIAL HYPERTENSION

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## ABSTRACT

Essential hypertension remains one of the leading causes of cardiovascular morbidity and mortality worldwide. Emerging evidence suggests that obesity, poor sleep quality, and physical inactivity are interrelated factors that significantly influence blood pressure (BP) control. Obesity contributes to hypertension through insulin resistance, sympathetic nervous system activation, inflammation, and altered renal sodium handling. Simultaneously, poor sleep quality has been associated with increased sympathetic activity, hormonal dysregulation, and endothelial dysfunction, all of which can elevate BP. Physical activity serves as a protective factor by improving cardiovascular fitness, promoting weight reduction, and enhancing sleep quality.

To review the relationship between obesity, sleep quality, blood pressure control, and physical activity among patients with essential hypertension, a narrative review of published literature was conducted using databases including PubMed, Scopus, Google Scholar, and Web of Science. Relevant studies evaluating body mass index (BMI), waist-hip ratio (WHR), Pittsburgh Sleep Quality Index (PSQI), blood pressure parameters, and physical activity in hypertensive adults were examined.

The literature consistently demonstrates that increased BMI and central obesity are associated with poor BP control. Higher PSQI scores indicating poor sleep quality correlate with elevated systolic and diastolic BP. Reduced physical activity exacerbates obesity and sleep disturbances, thereby contributing to uncontrolled hypertension. The interaction among these factors forms a complex cycle that adversely affects cardiovascular health.

Obesity, poor sleep quality, and physical inactivity are major modifiable determinants of blood pressure control. Comprehensive management strategies targeting weight reduction, sleep optimization, and increased physical activity may substantially improve hypertension outcomes.

**Keywords:** Essential hypertension, obesity, BMI, waist-hip ratio, sleep quality, PSQI, physical activity, blood pressure control, cardiovascular risk.

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## INTRODUCTION

Patients with hypertension have been found to be more susceptible to various cardiovascular diseases, including stroke and coronary heart disease.<sup>1,2</sup> Adequate sleep duration not only maintains normal physiological functioning but also plays a crucial role in preventing adverse cardiovascular outcomes. For instance, inadequate sleep has been associated with hypertension<sup>3</sup> and increased mortality from cardiovascular diseases.<sup>4</sup> Apart from sleep duration, sleep quality represents another important dimension of sleep; however, its

relationship with cardiovascular outcomes has received comparatively less attention.

One possible reason for the limited number of studies examining sleep quality is its complex conceptualization. Sleep quality is a multidimensional construct comprising several sleep indices, including sleep duration and the presence of sleep-related problems, which can be assessed either objectively (e.g., polysomnography or actigraphy) or subjectively (e.g., sleep diaries or self-reported questionnaires).<sup>5</sup> Objective sleep quality is characterized by a range of indices, including awakenings during sleep, quantity and proportion of sleep stages, rapid eye movement

latency, frequency of apneas or hypopneas, and periodic limb movements during sleep.<sup>5</sup> Despite their precision and reliability, these objective parameters have not been integrated into a single global sleep-quality index based on their relative clinical significance.<sup>6</sup>

Although subjective sleep measures may reflect certain objective sleep indices, such as sleep disturbances and nocturnal awakenings, subjective sleep quality specifically refers to an individual's retrospective evaluation of their overall sleep experience.<sup>5</sup> This assessment can be summarized into a global measure of sleep status. One of the most widely used instruments for this purpose is the Pittsburgh Sleep Quality Index (PSQI), which evaluates sleep latency, sleep duration, habitual sleep efficiency, sleep disturbances, use of sleep medications, and daytime dysfunction over the preceding month.<sup>7</sup> The PSQI is extensively accepted in both research and clinical settings because of its high internal consistency (Cronbach's  $\alpha = 0.83$ ), excellent test-retest reliability ( $r = 0.85$ ),<sup>7</sup> and satisfactory structural validity in identifying individuals with poor sleep quality in both clinical and non-clinical populations, using a global score  $>5$  as the diagnostic threshold.<sup>8</sup> Several systematic reviews have further demonstrated associations between PSQI-measured sleep quality and health outcomes such as obesity<sup>9</sup> and glycemic control.<sup>10</sup>

Studies conducted across Asia<sup>11</sup> and Europe<sup>12,13</sup> have reported that poor subjective sleep quality is associated with significantly increased odds of hypertension. Similarly, poor sleep quality has been linked to higher systolic blood pressure (SBP) and/or diastolic blood pressure (DBP) levels in populations from Asia<sup>14,15</sup> and Europe.<sup>12,13</sup> Nevertheless, findings remain inconsistent, as several investigations have reported non-significant associations between sleep quality and blood pressure across different geographical regions. These discrepancies suggest that the relationship between sleep quality and hypertension may vary according to population characteristics, environmental factors, and regional differences. Furthermore, several studies have shown that hypertensive individuals tend to have significantly higher global PSQI scores, indicating poorer sleep quality compared with normotensive individuals.<sup>14,15</sup>

Apart from its association with blood pressure levels, poor sleep quality may also influence the circadian pattern of blood pressure regulation. Under normal physiological conditions, blood pressure decreases by at least 10% during nighttime compared with daytime values, a phenomenon referred to as the "dipping" pattern. Patients exhibiting this normal nocturnal decline are classified as "dippers," whereas those who fail to

demonstrate such a reduction are termed "nondippers." Nondipping status is characterized by heightened sympathetic nervous system activity and reduced parasympathetic activity, contributing to persistently elevated nocturnal blood pressure. This abnormal pattern has been associated with an increased risk of target-organ damage, including left ventricular hypertrophy and cerebrovascular disease. Poor sleep quality may contribute to a nondipping blood pressure profile through disruption of normal circadian rhythms. Previous studies have supported this hypothesis by demonstrating significantly higher PSQI global scores among nondippers compared with dippers.

#### **METHODOLOGY**

This narrative review was conducted to evaluate the relationship between obesity, sleep quality, and blood pressure control among patients with essential hypertension. A comprehensive literature search was performed using electronic databases, including PubMed, Scopus, Web of Science, Google Scholar, and Embase. Relevant studies published in English between January 2000 and May 2026 were identified using combinations of the following keywords and Medical Subject Headings (MeSH) terms: "essential hypertension," "obesity," "body mass index," "waist-hip ratio," "sleep quality," "Pittsburgh Sleep Quality Index," "physical activity," "blood pressure control," "sleep disorders," and "cardiovascular risk." Additional articles were identified through manual screening of reference lists from selected publications.

The review included original research articles, observational studies, cohort studies, cross-sectional studies, case-control studies, randomized controlled trials, systematic reviews, and meta-analyses that investigated the associations among obesity-related parameters, sleep quality, physical activity, and blood pressure control in adults diagnosed with essential hypertension. Studies involving secondary hypertension, pediatric populations, pregnant women, or patients with severe psychiatric or neurological disorders affecting sleep were excluded. Particular emphasis was placed on studies assessing obesity through body mass index (BMI) and waist-hip ratio (WHR), sleep quality using validated instruments such as the Pittsburgh Sleep Quality Index (PSQI), physical activity levels, and blood pressure outcomes.

Relevant articles were screened for methodological quality, study design, sample characteristics, assessment tools, and key findings. Data were synthesized narratively to summarize the current evidence regarding the complex interplay between obesity, sleep quality, physical activity, and hypertension control. The findings were organized into thematic sections focusing on epidemiological associations, pathophysiological mechanisms,

clinical implications, and recent advances in management. This narrative approach enabled a comprehensive understanding of existing evidence and identification of knowledge gaps for future research.

#### LITERATURE REVIEW

##### **Obesity and Essential Hypertension**

Obesity is widely recognized as one of the most significant modifiable risk factors for the development and progression of essential hypertension.<sup>16</sup> The global rise in obesity prevalence has paralleled the increasing burden of hypertension, making these two conditions major public health concerns worldwide.<sup>16,17</sup> Essential hypertension, defined as persistently elevated blood pressure without an identifiable secondary cause, accounts for approximately 90–95% of all hypertension cases.<sup>17</sup> Numerous epidemiological investigations have consistently demonstrated a strong association between excess body weight and elevated blood pressure.<sup>18</sup> Individuals who are overweight or obese are estimated to have a two- to threefold greater risk of developing hypertension compared with those maintaining normal body weight.<sup>18</sup> Furthermore, obesity not only contributes to the onset of hypertension but also complicates blood pressure management, increases the likelihood of treatment resistance, and elevates the risk of cardiovascular morbidity and mortality.<sup>19</sup>

The relationship between obesity and hypertension is multifactorial and involves complex physiological, metabolic, neurohormonal, and inflammatory mechanisms.<sup>20</sup> Excess adipose tissue is now recognized as an active endocrine organ rather than a passive energy reservoir. Adipocytes secrete numerous bioactive substances, including adipokines, cytokines, and hormones that influence vascular function, sodium balance, insulin sensitivity, and sympathetic nervous system activity.<sup>20</sup> One of the principal mechanisms linking obesity to hypertension is activation of the sympathetic nervous system (SNS). Increased adiposity leads to elevated levels of leptin, an adipocyte-derived hormone that regulates appetite and energy expenditure. Chronic hyperleptinemia in obese individuals stimulates sympathetic outflow, resulting in increased heart rate, peripheral vascular resistance, and renal sodium retention, all of which contribute to elevated blood pressure.<sup>21</sup> In addition, obesity is associated with increased activity of the renin–angiotensin–aldosterone system (RAAS), which promotes vasoconstriction, sodium retention, fluid accumulation, and vascular remodeling, thereby aggravating hypertension.<sup>21</sup>

Another important mechanism involves insulin resistance, a common metabolic abnormality in obese individuals. Excess adiposity, particularly visceral fat accumulation, reduces insulin sensitivity and leads to compensatory

hyperinsulinemia. Elevated insulin levels stimulate renal sodium reabsorption and activate the sympathetic nervous system, contributing to blood pressure elevation.<sup>22</sup> Insulin resistance also impairs endothelial function by reducing nitric oxide bioavailability, thereby promoting vasoconstriction and increased vascular stiffness. Chronic low-grade inflammation associated with obesity further exacerbates endothelial dysfunction through the release of inflammatory cytokines such as tumor necrosis factor- $\alpha$  (TNF- $\alpha$ ), interleukin-6 (IL-6), and C-reactive protein (CRP).<sup>22</sup> These inflammatory mediators induce oxidative stress, impair vascular relaxation, and accelerate atherosclerotic changes, thereby contributing to sustained hypertension.<sup>20</sup>

##### **Body Mass Index (BMI)**

Body Mass Index (BMI), calculated as weight in kilograms divided by the square of height in meters ( $\text{kg}/\text{m}^2$ ), remains the most widely used and practical measure of general obesity in clinical and epidemiological settings.<sup>18</sup> BMI provides a simple and cost-effective method for assessing body fatness and categorizing individuals into underweight, normal weight, overweight, and obese groups. Numerous studies have demonstrated a strong positive relationship between BMI and blood pressure levels across different populations, age groups, and ethnicities.<sup>18,23</sup> As BMI increases, both systolic blood pressure (SBP) and diastolic blood pressure (DBP) tend to rise, indicating a dose-dependent association between body weight and hypertension risk.<sup>23</sup>

Several large-scale longitudinal studies have highlighted the importance of BMI as a predictor of hypertension. The Framingham Heart Study reported that excess body weight accounted for approximately 65–75% of the risk for developing hypertension.<sup>24</sup> Individuals who experienced weight gain during adulthood showed a significantly higher incidence of hypertension compared with those who maintained stable body weight. Conversely, weight reduction was associated with substantial decreases in blood pressure levels, underscoring the causal relationship between obesity and hypertension.<sup>24</sup> Clinical trials have demonstrated that even modest weight loss of 5–10% of body weight can result in meaningful reductions in systolic and diastolic blood pressure.<sup>25</sup> The influence of BMI on blood pressure is mediated through several physiological pathways. Increased body mass leads to greater metabolic demand, requiring enhanced tissue perfusion and oxygen delivery. This results in increased blood volume and cardiac output, which contribute to elevated arterial pressure.<sup>21</sup> Obesity also promotes renal compression due to fat accumulation around the kidneys, impairing natriuresis and facilitating sodium retention. Increased sodium and water

retention expand intravascular volume, thereby elevating blood pressure.<sup>21</sup> Furthermore, obesity-induced activation of the sympathetic nervous system and RAAS contributes to vasoconstriction and vascular remodeling.<sup>21</sup> These changes collectively increase systemic vascular resistance and sustain hypertension.

Higher BMI is also associated with structural and functional alterations in the cardiovascular system. Obese individuals frequently exhibit left ventricular hypertrophy, increased arterial stiffness, and impaired endothelial function.<sup>19</sup> These abnormalities contribute not only to hypertension but also to an increased risk of coronary artery disease, heart failure, stroke, and chronic kidney disease.<sup>19</sup> Importantly, obesity-related hypertension often develops at a younger age and tends to be more difficult to control, requiring multiple antihypertensive medications. Therefore, BMI remains a valuable screening tool for identifying individuals at increased risk of hypertension and related cardiovascular complications.<sup>23</sup>

#### **Waist–Hip Ratio (WHR)**

Although BMI is useful for assessing overall obesity, it does not provide information regarding body fat distribution. Increasing evidence suggests that the location of adipose tissue accumulation is equally important in determining cardiovascular risk. Waist–Hip Ratio (WHR), calculated by dividing waist circumference by hip circumference, serves as an indicator of abdominal or central obesity and reflects the degree of visceral fat accumulation.<sup>22</sup> Visceral adiposity has been shown to exert more detrimental metabolic and cardiovascular effects than subcutaneous fat, making WHR a particularly important predictor of hypertension and cardiovascular disease.<sup>22</sup>

Central obesity contributes to hypertension through multiple interrelated mechanisms. Visceral adipose tissue is metabolically active and secretes large amounts of pro-inflammatory cytokines, free fatty acids, and adipokines.<sup>22</sup> These substances promote systemic inflammation, oxidative stress, and endothelial dysfunction, all of which impair vascular homeostasis and contribute to elevated blood pressure.<sup>20</sup> Visceral fat also produces increased levels of angiotensinogen, leading to enhanced activation of the renin–angiotensin–aldosterone system.<sup>21</sup> The resulting vasoconstriction, sodium retention, and fluid accumulation promote sustained increases in blood pressure.

Leptin-mediated sympathetic activation is another key pathway linking central obesity to hypertension. Individuals with elevated WHR often exhibit higher circulating leptin concentrations, which stimulate sympathetic nervous system activity and increase peripheral vascular resistance.<sup>21</sup> Additionally, visceral obesity is

strongly associated with insulin resistance and metabolic syndrome, both of which contribute to hypertension through alterations in glucose metabolism, endothelial function, and renal sodium handling.<sup>22</sup> These mechanisms collectively explain why individuals with central obesity frequently develop hypertension even when their overall BMI is not markedly elevated.

Numerous studies have demonstrated that WHR is an independent predictor of hypertension and cardiovascular risk. Research has shown that elevated WHR is associated with higher systolic and diastolic blood pressure levels, greater prevalence of uncontrolled hypertension, and increased incidence of cardiovascular events.<sup>23</sup> Importantly, several investigations have reported that WHR predicts hypertension more accurately than BMI in certain populations because it specifically reflects visceral fat accumulation.<sup>23</sup> Patients with increased WHR are more likely to exhibit target organ damage, including left ventricular hypertrophy, carotid artery thickening, and renal impairment.<sup>19</sup> Furthermore, elevated WHR has been linked to poor blood pressure control despite antihypertensive therapy, suggesting that central obesity may contribute to treatment resistance.<sup>25</sup>

In clinical practice, the combined assessment of BMI and WHR provides a more comprehensive evaluation of obesity-related cardiovascular risk. While BMI quantifies overall adiposity, WHR identifies individuals with excessive visceral fat who may be at particularly high risk for hypertension and adverse cardiovascular outcomes.<sup>23</sup> Consequently, both parameters are valuable tools for risk stratification, early detection, and monitoring of obesity-related hypertension. Lifestyle interventions aimed at reducing body weight and abdominal fat, including dietary modification, regular physical activity, and behavioral counseling, remain essential components of hypertension management.<sup>25</sup> Effective control of obesity not only improves blood pressure regulation but also reduces the overall burden of cardiovascular disease, thereby enhancing long-term health outcomes in patients with essential hypertension.

#### **Sleep Quality and Hypertension**

Sleep is a fundamental biological process that plays a crucial role in maintaining cardiovascular, metabolic, neuroendocrine, and immune system homeostasis. Adequate sleep is essential for the regulation of blood pressure, heart rate, vascular function, and autonomic nervous system activity. In recent decades, growing evidence has identified poor sleep quality as an independent and significant risk factor for the development, progression, and poor control of hypertension. Sleep disturbances are increasingly recognized as contributors to

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cardiovascular disease burden worldwide, particularly among individuals with obesity and essential hypertension. Epidemiological studies have consistently shown that individuals with poor sleep quality are more likely to experience hypertension and cardiovascular outcomes compared with those who maintain healthy sleep patterns. Moreover, hypertensive patients frequently report sleep-related complaints, including difficulty initiating sleep, fragmented sleep, frequent awakenings, non-restorative sleep, and excessive daytime fatigue. The relationship between sleep quality and blood pressure regulation is complex and involves multiple physiological pathways. During normal sleep, particularly during non-rapid eye movement (NREM) sleep, there is a reduction in sympathetic nervous system activity accompanied by increased parasympathetic tone. This autonomic shift results in a physiological decline in heart rate and blood pressure, commonly referred to as “nocturnal dipping.” In healthy individuals, blood pressure decreases by approximately 10–20% during sleep compared with daytime levels. This nocturnal reduction provides an essential period of cardiovascular recovery and protection against vascular stress. However, poor sleep quality disrupts these normal physiological processes, leading to persistent sympathetic activation, hormonal imbalance, endothelial dysfunction, and chronic inflammation, all of which contribute to sustained elevations in blood pressure.

**Pittsburgh Sleep Quality Index (PSQI)**

The Pittsburgh Sleep Quality Index (PSQI) is one of the most widely used and validated instruments for assessing subjective sleep quality in both clinical and research settings. Developed to evaluate sleep patterns over the previous month, the PSQI provides a comprehensive assessment of various aspects of sleep and generates a global score that reflects overall sleep quality.

**Table. Components of the Pittsburgh Sleep Quality Index (PSQI)**

Component No.	PSQI Component	Description
1	Subjective Sleep Quality	Individual's overall sleep quality during the past month.
2	Sleep Latency	Time taken to fall asleep after going to bed.
3	Sleep Duration	Total number of hours of actual sleep. Slept approximately 7 hours and fragmented sleep reduce nitric oxide bioavailability while increasing oxidative stress, thereby impairing endothelial-dependent vasodilation. <sup>28</sup>
4	Habitual Sleep Efficiency	Percentage of time spent asleep while in bed.
5	Sleep Disturbances	Frequency of factors that interrupt or disturb sleep. Consequently, vascular stiffness and peripheral resistance increase to help induce or maintain sleep hypertension. Additionally, inadequate sleep promotes chronic low-grade inflammation characterized by elevated levels of inflammatory biomarkers such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-
6	Use of Sleeping Medication	Frequency of using medications to help induce or maintain sleep.
7	Daytime Dysfunction	Difficulties in staying awake and maintaining enthusiasm for daily activities.

**Scoring of PSQI**

Parameter

Details

Each Component	0–3 points
Sum of Component Scores	Higher scores indicate greater sleep impairment
Number of Components	7
Global PSQI Score Range	0–21 points
Interpretation of Global Score	Higher scores indicate poorer sleep quality
Cutoff for Poor Sleep Quality	<b>PSQI &gt; 5</b>
Cutoff for Good Sleep Quality	<b>PSQI ≤ 5</b>

The Pittsburgh Sleep Quality Index (PSQI) evaluates subjective sleep quality over the preceding one month and is widely used in clinical and epidemiological studies to assess overall sleep quality.

**Sleep Quality and Hypertension**

Numerous studies have demonstrated that patients with hypertension tend to have significantly higher Pittsburgh Sleep Quality Index (PSQI) scores than normotensive individuals, indicating poorer overall sleep quality.<sup>26</sup> Poor sleep quality has been associated with both increased prevalence and greater severity of hypertension.<sup>25,26</sup> Hypertensive individuals frequently experience prolonged sleep latency, reduced sleep duration, recurrent nocturnal awakenings, diminished sleep efficiency, and greater daytime dysfunction.<sup>26</sup> These disturbances contribute to chronic activation of physiological stress pathways that adversely affect cardiovascular health and blood pressure regulation.<sup>27</sup>

One of the primary mechanisms linking poor sleep quality to hypertension is increased sympathetic nervous system activity. Sleep fragmentation and inadequate sleep attenuate the normal nocturnal decline in sympathetic tone, resulting in persistently elevated levels of catecholamines such as norepinephrine and epinephrine.<sup>27</sup> Sustained sympathetic activation increases heart rate, peripheral vascular resistance, and renal sodium retention, ultimately contributing to elevated blood pressure. Furthermore, poor sleep quality stimulates the hypothalamic–pituitary–adrenal (HPA) axis, resulting in increased cortisol secretion. Elevated cortisol levels contribute to hypertension through enhanced sodium retention,

vascular constriction, and promotion of insulin resistance.<sup>27</sup>

alpha (TNF- $\alpha$ ).<sup>28</sup> These inflammatory processes further exacerbate vascular dysfunction and cardiovascular risk.

Another important consequence of poor sleep quality is disruption of normal circadian blood pressure rhythms. Individuals with sleep disturbances frequently exhibit a “non-dipping” blood pressure pattern, in which nocturnal blood pressure fails to decrease appropriately during sleep.<sup>29</sup> Non-dipping status has been associated with an increased risk of left ventricular hypertrophy, stroke, myocardial infarction, chronic kidney disease, and cardiovascular mortality.<sup>29</sup> Therefore, assessment of sleep quality using tools such as the PSQI provides valuable insight into cardiovascular risk stratification and hypertension management.<sup>26</sup>

#### **Sleep Duration and Blood Pressure**

Sleep duration represents another critical determinant of cardiovascular health. A growing body of evidence suggests that both insufficient and excessive sleep are associated with adverse cardiovascular outcomes, creating a U-shaped relationship between sleep duration and hypertension risk.<sup>25</sup> Most adults require approximately 7–8 hours of sleep per night for optimal physiological functioning. Deviations from this range have been linked to increased prevalence of hypertension, metabolic disorders, and cardiovascular disease.<sup>25</sup>

Short sleep duration, typically defined as fewer than six hours of sleep per night, has been consistently associated with elevated blood pressure and increased hypertension risk.<sup>25,27</sup> Individuals who experience chronic sleep restriction demonstrate increased sympathetic nervous system activity, elevated cortisol secretion, and enhanced activation of inflammatory pathways.<sup>27</sup> Sleep deprivation stimulates catecholamine release, resulting in vasoconstriction and increased cardiac workload. Simultaneously, elevated cortisol levels promote sodium retention and vascular resistance, further contributing to hypertension.

Short sleep duration is also closely linked to insulin resistance and metabolic dysfunction. Inadequate sleep impairs glucose metabolism and alters the secretion of appetite-regulating hormones such as leptin and ghrelin, promoting weight gain and obesity.<sup>28</sup> Since obesity itself is a major risk factor for hypertension, sleep deprivation indirectly contributes to blood pressure elevation through adverse metabolic effects. Furthermore, sleep restriction increases levels of inflammatory markers and oxidative stress, both of which contribute to endothelial dysfunction and vascular damage.<sup>28</sup>

Interestingly, prolonged sleep duration, generally defined as more than nine hours per night, has also

been associated with increased cardiovascular risk.<sup>25</sup> Although the exact mechanisms remain incompletely understood, excessive sleep duration may reflect underlying health conditions, chronic inflammation, depression, reduced physical activity, or poor sleep quality despite longer sleep periods. Several population-based studies have reported higher rates of hypertension, cardiovascular disease, and mortality among long sleepers compared with individuals maintaining normal sleep durations.<sup>25</sup> Thus, maintaining an appropriate sleep duration appears essential for optimal cardiovascular regulation and blood pressure control.

#### **Sleep Disorders and Hypertension**

Sleep disorders represent an important but often underdiagnosed contributor to hypertension. Among these disorders, Obstructive Sleep Apnea (OSA) is particularly significant because of its strong association with obesity, hypertension, and cardiovascular disease.<sup>30</sup> OSA is characterized by recurrent episodes of upper airway obstruction during sleep, leading to intermittent hypoxia, sleep fragmentation, and repeated arousals from sleep. The prevalence of OSA is substantially higher among obese individuals and patients with resistant hypertension.<sup>30</sup>

The pathophysiological mechanisms linking OSA to hypertension are multifactorial. Repeated episodes of nocturnal hypoxia trigger activation of peripheral chemoreceptors, resulting in marked sympathetic nervous system stimulation.<sup>30</sup> This sympathetic overactivity often persists during wakefulness, contributing to sustained elevations in blood pressure. Intermittent hypoxia also promotes oxidative stress through increased production of reactive oxygen species, which damage endothelial cells and impair vascular function.<sup>30</sup>

In addition, OSA activates the renin–angiotensin–aldosterone system, leading to vasoconstriction, sodium retention, and fluid accumulation.<sup>30</sup> Recurrent hypoxic episodes stimulate inflammatory pathways and increase circulating levels of pro-inflammatory cytokines, further contributing to endothelial dysfunction and vascular remodeling. These physiological alterations collectively result in persistent hypertension and increased cardiovascular risk.

OSA is particularly prevalent among obese hypertensive patients because excess adipose tissue around the neck and upper airway increases airway collapsibility during sleep.<sup>30</sup> Studies have demonstrated that the severity of OSA correlates positively with blood pressure levels and cardiovascular complications. Patients with untreated OSA are more likely to develop resistant hypertension, coronary artery disease, heart failure, stroke, and cardiac arrhythmias.<sup>30</sup>

Importantly, effective treatment of OSA can significantly improve blood pressure control. Continuous Positive Airway Pressure (CPAP) therapy remains the gold-standard treatment for moderate-to-severe OSA.<sup>30</sup> CPAP prevents airway collapse during sleep, reduces nocturnal hypoxia, improves sleep quality, and decreases sympathetic nervous system activation. Several clinical studies have demonstrated that CPAP therapy results in reductions in both systolic and diastolic blood pressure, particularly among patients with resistant hypertension.<sup>30</sup> Weight loss, lifestyle modification, positional therapy, and surgical interventions may also contribute to improved sleep quality and blood pressure regulation in selected patients.

Overall, growing evidence highlights the critical role of sleep quality, sleep duration, and sleep disorders in the pathogenesis and management of hypertension. Assessment of sleep parameters using validated instruments such as the PSQI, together with early identification and treatment of sleep disorders, should form an integral component of comprehensive hypertension care.<sup>26,30</sup> Improving sleep health may not only enhance blood pressure control but also reduce long-term cardiovascular risk and improve overall quality of life among patients with essential hypertension.

#### **Physical Activity and Blood Pressure Control**

Physical activity is widely recognized as one of the most effective non-pharmacological interventions for the prevention and management of hypertension.<sup>31</sup> Regular exercise exerts multiple beneficial effects on the cardiovascular system, contributing to improved blood pressure regulation and reduced cardiovascular risk. Current hypertension management guidelines strongly recommend incorporating physical activity as a fundamental component of lifestyle modification strategies for individuals with elevated blood pressure.<sup>31</sup>

The antihypertensive effects of physical activity are mediated through several physiological mechanisms. Regular exercise reduces sympathetic nervous system activity, leading to decreased peripheral vascular resistance and heart rate.<sup>32</sup> It improves endothelial function by enhancing the production and bioavailability of nitric oxide, a potent vasodilator that promotes vascular relaxation.<sup>32</sup> Exercise also improves insulin sensitivity, thereby reducing hyperinsulinemia-associated sodium retention and sympathetic activation. Additionally, physical activity facilitates weight reduction, decreases systemic inflammation, and improves lipid metabolism, all of which contribute to better cardiovascular health and blood pressure control.<sup>32</sup>

Numerous studies have demonstrated that aerobic exercise, such as brisk walking, cycling, jogging, and swimming, can reduce systolic blood pressure

by approximately 5–8 mmHg in individuals with hypertension.<sup>31,32</sup> Resistance training and combined aerobic-resistance exercise programs have also shown significant benefits in lowering both systolic and diastolic blood pressure.<sup>32</sup> The magnitude of blood pressure reduction achieved through regular exercise is often comparable to that observed with a single antihypertensive medication, highlighting its clinical importance in hypertension management.<sup>31</sup>

#### **Physical Activity and Sleep Quality**

Physical activity plays a crucial role in maintaining healthy sleep patterns and improving overall sleep quality.<sup>33</sup> Regular exercise influences several physiological processes involved in sleep regulation, including circadian rhythm synchronization, thermoregulation, and neuroendocrine function. Individuals who engage in regular physical activity generally experience shorter sleep latency, increased sleep efficiency, longer sleep duration, and improved subjective sleep quality.<sup>33</sup>

Exercise has been shown to reduce symptoms of anxiety and depression, which are common contributors to sleep disturbances.<sup>33</sup> Furthermore, physical activity promotes greater energy expenditure during the day, increasing the body's physiological drive for restorative sleep at night. Studies utilizing the Pittsburgh Sleep Quality Index (PSQI) consistently report lower global PSQI scores among physically active individuals compared with sedentary populations, indicating better sleep quality.<sup>33</sup> Therefore, regular physical activity serves as an important intervention not only for cardiovascular health but also for improving sleep outcomes and overall well-being.<sup>33</sup>

#### **Interrelationship Among Obesity, Sleep Quality, Physical Activity, and Blood Pressure**

The relationship among obesity, sleep quality, physical activity, and blood pressure is complex and multidirectional. These factors interact through multiple physiological and behavioral pathways, creating a cycle that may either promote health or contribute to disease progression.<sup>34</sup>

#### **Obesity and Sleep Quality**

Obesity is strongly associated with poor sleep quality. Excess adipose tissue, particularly around the neck and upper airway, predisposes individuals to sleep-disordered breathing, most notably obstructive sleep apnea (OSA).<sup>34</sup> Recurrent episodes of airway obstruction during sleep result in intermittent hypoxia, frequent arousals, and fragmented sleep architecture. Consequently, obese individuals frequently report poor subjective sleep quality, excessive daytime sleepiness, and higher PSQI scores compared with individuals of normal weight.<sup>34</sup>

#### **Poor Sleep Quality and Weight Gain**

Conversely, inadequate sleep contributes to the development and progression of obesity. Sleep

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deprivation alters the secretion of hormones involved in appetite regulation. Reduced sleep duration decreases leptin levels, which normally suppress appetite, while increasing ghrelin levels, which stimulate hunger.<sup>35</sup> These hormonal alterations promote increased caloric intake, preference for energy-dense foods, and reduced energy expenditure, ultimately leading to weight gain and obesity.<sup>35</sup> In addition, chronic sleep restriction may impair glucose metabolism and insulin sensitivity, further contributing to metabolic dysfunction.<sup>35</sup>

**Physical Inactivity, Obesity, and Sleep Disturbance**

Physical inactivity is another important contributor to both obesity and poor sleep quality. Sedentary lifestyles reduce energy expenditure, facilitating positive energy balance and fat accumulation.<sup>31</sup> Lack of regular physical activity is also associated with reduced sleep efficiency, prolonged sleep latency, and increased sleep disturbances.<sup>33</sup> Consequently, physically inactive individuals often exhibit higher body mass index (BMI), poorer sleep quality, and increased cardiovascular risk compared with their physically active counterparts.<sup>33</sup>

**Combined Impact on Blood Pressure  
Interrelationship Among Obesity, Sleep Quality,  
Physical Activity, and Hypertension**



The coexistence of obesity, physical inactivity, and poor sleep quality exerts a synergistic effect on blood pressure regulation.<sup>34</sup> Obesity promotes hypertension through activation of the sympathetic nervous system, stimulation of the renin-angiotensin-aldosterone system (RAAS), insulin resistance, and chronic low-grade inflammation.<sup>34</sup> Poor sleep quality further exacerbates sympathetic activation, increases neuroendocrine stress responses, and impairs normal nocturnal blood pressure dipping.<sup>35</sup> Physical inactivity compounds these adverse effects by reducing cardiovascular

fitness, impairing endothelial function, and limiting the beneficial effects of exercise on vascular health.<sup>32</sup>

As a result, individuals who are obese, physically inactive, and experience poor sleep quality are significantly more likely to have uncontrolled hypertension and a greater risk of cardiovascular complications than those maintaining a healthy body weight, engaging in regular physical activity, and achieving adequate sleep quality.<sup>34,35</sup> These findings highlight the importance of integrated lifestyle interventions targeting obesity reduction, enhancement of physical activity, and optimization of sleep quality as essential components of comprehensive hypertension management.

#### **Recent Advances in Management**

Recent advances in the management of obesity, sleep disturbances, and hypertension have transformed contemporary approaches to cardiovascular risk reduction by emphasizing personalized, technology-driven, and multidisciplinary care.<sup>36</sup> Traditional management strategies primarily focused on pharmacological blood pressure control; however, accumulating evidence highlights the importance of addressing underlying contributors such as obesity, poor sleep quality, physical inactivity, and behavioral risk factors.<sup>36</sup> Advances in digital health technologies, artificial intelligence (AI), telemedicine, chronotherapy, and integrated lifestyle interventions are increasingly being incorporated into comprehensive hypertension management programs.<sup>36</sup> These innovations provide opportunities for earlier detection of risk factors, continuous monitoring, enhanced patient engagement, and more effective long-term blood pressure control.

#### **Digital Sleep Monitoring**

One of the most significant developments in recent years has been the widespread adoption of digital sleep-monitoring technologies.<sup>37</sup> Wearable devices such as smartwatches, fitness bands, and sleep trackers can continuously assess sleep duration, sleep stages, heart rate variability, oxygen saturation, and nocturnal movement patterns.<sup>37</sup> Smartphone-based applications further enhance sleep assessment by providing detailed analyses of sleep quality and identifying patterns associated with sleep disturbances. These technologies enable healthcare providers to obtain objective sleep-related information outside traditional sleep laboratory settings.<sup>37</sup> Early identification of inadequate sleep, fragmented sleep, and potential sleep disorders such as obstructive sleep apnea (OSA) facilitates timely intervention and personalized treatment strategies. Continuous monitoring also increases patient awareness regarding sleep hygiene and encourages adherence

to behavioral modifications aimed at improving cardiovascular health and overall well-being.<sup>37</sup>

#### **Personalized Lifestyle Interventions**

Advancements in artificial intelligence and machine-learning technologies have facilitated the development of personalized lifestyle intervention programs.<sup>38</sup> AI-based platforms can analyze individual characteristics, including body mass index (BMI), physical activity levels, dietary habits, sleep patterns, metabolic risk factors, and blood pressure profiles, to generate tailored recommendations. Unlike conventional generalized advice, personalized interventions are designed to address the specific needs and challenges of each individual.<sup>38</sup> These systems can provide customized exercise regimens, dietary plans, weight-management strategies, and sleep-improvement recommendations. Real-time feedback, remote coaching, and adaptive behavioral support further enhance patient engagement and long-term adherence. Studies have demonstrated that personalized digital health interventions lead to greater weight reduction, improved physical activity participation, enhanced sleep quality, and more effective blood pressure control compared with traditional lifestyle counseling approaches.<sup>38</sup>

#### **Telehealth-Based Hypertension Care**

Telehealth has emerged as an invaluable tool in modern hypertension management, particularly following the rapid expansion of remote healthcare services.<sup>39</sup> Remote blood pressure monitoring systems enable patients to measure and transmit blood pressure readings from home using connected devices. Healthcare professionals can review these measurements in real time and make timely adjustments to treatment plans without requiring frequent clinic visits.<sup>39</sup> Telehealth platforms often integrate lifestyle counseling, medication reminders, educational resources, and virtual consultations, thereby improving treatment adherence and self-management. Evidence suggests that telemonitoring combined with regular healthcare-provider feedback significantly improves blood pressure control, medication compliance, and patient satisfaction.<sup>39</sup> This approach is particularly beneficial for individuals residing in rural areas, older adults, and patients with limited access to healthcare facilities.

#### **Chronotherapy**

Chronotherapy represents an emerging therapeutic strategy that considers the body's natural circadian rhythms when administering antihypertensive medications.<sup>40</sup> Blood pressure follows a physiological circadian pattern, typically decreasing during sleep and increasing upon awakening. Disruption of this rhythm, particularly the absence of normal nocturnal blood pressure dipping, has been associated with heightened cardiovascular risk.<sup>40</sup> Chronotherapy aims to

optimize the timing of antihypertensive medication administration to better align with these physiological fluctuations. Several clinical studies have suggested that bedtime dosing of selected antihypertensive agents may improve nocturnal blood pressure control and potentially reduce cardiovascular events in specific patient populations.<sup>40</sup> Although additional research is required to establish universal recommendations, chronotherapy has demonstrated considerable promise as a personalized approach to enhancing blood pressure management and cardiovascular protection.

#### **Integrated Weight-Loss Programs**

Multidisciplinary weight-management programs represent another important advancement in the treatment of obesity-related hypertension.<sup>36</sup> These comprehensive programs involve collaboration among physicians, nutritionists, sleep specialists, psychologists, physiotherapists, and exercise physiologists. Rather than focusing exclusively on weight reduction, integrated interventions simultaneously address dietary habits, physical activity, sleep quality, behavioral modification, and psychological well-being. Such programs acknowledge the complex interrelationship among obesity, sleep disturbances, and hypertension.<sup>36</sup> Evidence indicates that multidisciplinary approaches achieve greater and more sustainable weight loss, improved sleep quality, enhanced metabolic health, and superior blood pressure control compared with conventional single-component interventions.<sup>36</sup> In obese hypertensive individuals, integrated programs have also been associated with reduced medication requirements and lower long-term cardiovascular risk. As healthcare increasingly shifts toward patient-centered care, these multidisciplinary models are becoming an essential component of comprehensive hypertension management strategies.

#### **Future Directions**

Future research should focus on developing a deeper understanding of the complex interactions among obesity, sleep quality, physical activity, and blood pressure regulation in patients with essential hypertension. Longitudinal cohort studies are needed to establish causal relationships and determine how changes in body weight and sleep patterns influence hypertension development and control over time. The growing availability of wearable technologies presents an opportunity for continuous, real-world monitoring of sleep quality, physical activity, heart rate, and blood pressure, enabling more accurate assessment of cardiovascular risk and treatment responses.

Another important area of investigation is the development of personalized lifestyle intervention models that integrate individual characteristics such

as age, body composition, sleep behavior, physical activity levels, and comorbidities. Advances in genomics and precision medicine may help identify genetic determinants that predispose individuals to obesity-related hypertension and poor sleep quality, allowing for targeted preventive strategies. Artificial intelligence and machine learning algorithms also hold significant potential for predicting hypertension control outcomes, identifying high-risk patients, and optimizing individualized treatment plans.

Furthermore, large-scale randomized controlled trials are required to evaluate the effectiveness of combined interventions targeting weight reduction, sleep improvement, and increased physical activity. Such studies may provide robust evidence for integrated management approaches and contribute to the development of comprehensive clinical guidelines aimed at improving long-term cardiovascular outcomes and quality of life among patients with essential hypertension.

#### **DISCUSSION**

The findings of the present narrative review highlight the intricate and multidirectional relationship among obesity, sleep quality, physical activity, and blood pressure control in patients with essential hypertension. Current evidence indicates that these factors do not operate independently but rather interact through a complex network of physiological, metabolic, neuroendocrine, and behavioral pathways that collectively influence cardiovascular health and hypertension outcomes.<sup>41</sup> Obesity, particularly central obesity measured by waist-hip ratio (WHR), has emerged as a critical determinant of poor blood pressure control because visceral adipose tissue actively contributes to sympathetic nervous system activation, renin-angiotensin-aldosterone system (RAAS) stimulation, insulin resistance, oxidative stress, and chronic low-grade inflammation.<sup>42</sup> These mechanisms increase vascular resistance, impair endothelial function, and promote persistent elevation of blood pressure.

The present review supports the observations of Hall et al., who reported that obesity-induced hypertension results primarily from interactions among sympathetic overactivity, altered renal sodium handling, and activation of neurohormonal pathways.<sup>43</sup> Similarly, Landsberg et al. demonstrated that increased adiposity is associated with heightened sympathetic nervous system activity, which contributes significantly to the development and maintenance of hypertension.<sup>44</sup> Consistent with these findings, several large epidemiological studies have shown that increasing BMI is directly associated with progressive elevations in both systolic and diastolic blood pressure.<sup>45</sup> Moreover, abdominal obesity has been shown to possess stronger predictive value for

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hypertension than overall obesity in many populations, emphasizing the importance of assessing both BMI and WHR when evaluating cardiovascular risk.<sup>46</sup>

An important finding emerging from contemporary literature is the strong association between poor sleep quality and hypertension. Studies utilizing the Pittsburgh Sleep Quality Index (PSQI) consistently demonstrate that hypertensive patients report significantly poorer sleep quality than normotensive individuals.<sup>47</sup> These findings are in agreement with those reported by Meng et al., whose meta-analysis demonstrated that poor sleep quality was associated with a significantly increased risk of hypertension across diverse populations.<sup>48</sup> Likewise, Wang et al. observed that individuals with elevated PSQI scores had significantly higher systolic and diastolic blood pressure levels compared with those reporting good sleep quality.<sup>49</sup> The present review further supports the concept that sleep disturbances contribute to hypertension through increased sympathetic activation, hypothalamic–pituitary–adrenal axis stimulation, endothelial dysfunction, and inflammatory activation.

The bidirectional relationship between obesity and sleep quality represents another important aspect of hypertension pathogenesis. Obesity contributes to sleep disturbances through both mechanical and metabolic mechanisms. Excess adiposity, particularly around the upper airway, increases susceptibility to obstructive sleep apnea (OSA), resulting in fragmented sleep and intermittent hypoxia.<sup>50</sup> Conversely, inadequate sleep alters appetite-regulating hormones such as leptin and ghrelin, promoting increased caloric intake, weight gain, and further obesity.<sup>51</sup> This observation is comparable to the findings of Taheri et al., who reported that reduced sleep duration was associated with lower leptin levels, elevated ghrelin concentrations, and increased body mass index.<sup>52</sup> Similarly, Spiegel et al. demonstrated that sleep restriction induces metabolic and endocrine alterations that favor positive energy balance and obesity development.<sup>53</sup> Therefore, obesity and poor sleep quality appear to reinforce one another, creating a self-perpetuating cycle that contributes to blood pressure dysregulation.

Physical activity emerges as a critical modifying factor within this complex interaction. The present review indicates that regular physical activity exerts beneficial effects on both sleep quality and blood pressure regulation. These findings are consistent with those of Diaz and Shimbo, who reported that aerobic exercise significantly improves endothelial function, reduces sympathetic activity, and lowers blood pressure in hypertensive individuals.<sup>54</sup> Similarly, Kredlow et al. found that physically active individuals experience superior

sleep quality, shorter sleep latency, and improved sleep efficiency compared with sedentary populations.<sup>55</sup> Physical activity additionally facilitates weight reduction and improves insulin sensitivity, thereby simultaneously addressing multiple contributors to hypertension. Consequently, sedentary behavior may amplify the adverse effects of obesity and poor sleep on cardiovascular health.

The interrelationship among obesity, sleep quality, and physical activity has been increasingly recognized as a determinant of resistant or uncontrolled hypertension. Studies evaluating hypertensive populations have reported that individuals exhibiting combinations of obesity, sleep disturbances, and low physical activity levels are significantly more likely to have uncontrolled blood pressure despite pharmacological treatment.<sup>56</sup> This observation is supported by the work of Pedrosa et al., who identified obstructive sleep apnea, obesity, and metabolic abnormalities as major contributors to resistant hypertension.<sup>57</sup> Furthermore, Kario et al. demonstrated that disrupted sleep and abnormal nocturnal blood pressure patterns significantly increase cardiovascular risk and target-organ damage among hypertensive patients.<sup>58</sup>

Recent advances in management further emphasize the importance of addressing these interconnected factors simultaneously. Contemporary approaches incorporating digital sleep monitoring, telehealth-based blood pressure management, personalized lifestyle interventions, and multidisciplinary weight-management programs have shown encouraging results.<sup>59</sup> These integrated strategies recognize that optimal blood pressure control requires more than antihypertensive medication alone and must include interventions targeting obesity, sleep health, and physical activity. Emerging evidence suggests that combined lifestyle interventions produce greater reductions in blood pressure and cardiovascular risk than isolated interventions directed at a single risk factor.<sup>60</sup>

Overall, the available literature strongly supports the concept that obesity, poor sleep quality, and physical inactivity form a complex interdependent network that significantly influences blood pressure control in patients with essential hypertension. The findings of the present review are consistent with those reported by numerous international studies and reinforce the need for comprehensive, patient-centered management strategies. Future research should focus on longitudinal investigations exploring causal pathways among these variables and evaluating the effectiveness of integrated interventions aimed at improving weight management, sleep quality, physical activity, and long-term cardiovascular outcomes.

### CONCLUSION

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Essential hypertension remains a major global health challenge, with increasing evidence highlighting the significant roles of obesity, sleep quality, and physical activity in determining blood pressure control. The available literature demonstrates that elevated body mass index (BMI) and waist-hip ratio are strongly associated with poor blood pressure regulation, increased cardiovascular risk, and a higher likelihood of uncontrolled hypertension. Excess adiposity contributes to hypertension through multiple mechanisms, including sympathetic nervous system activation, insulin resistance, chronic inflammation, and hormonal dysregulation.

Sleep quality has emerged as another important modifiable factor influencing blood pressure outcomes. Poor sleep quality, often reflected by higher Pittsburgh Sleep Quality Index (PSQI) scores, adversely affects cardiovascular health by promoting neurohormonal imbalance, endothelial dysfunction, and inflammatory responses. Individuals with hypertension who experience inadequate or disturbed sleep are more likely to exhibit poor blood pressure control and increased cardiovascular morbidity.

Physical activity plays a crucial protective role by improving cardiovascular fitness, reducing body weight, enhancing sleep quality, and lowering blood pressure levels. Regular exercise not only mitigates obesity-related risks but also contributes to overall metabolic and vascular health. The interrelationship among obesity, sleep quality, and physical activity suggests that these factors should not be evaluated in isolation but rather as interconnected determinants of hypertension control.

Therefore, effective management of essential hypertension requires a comprehensive, multidimensional strategy that emphasizes weight reduction, optimization of sleep quality, and promotion of regular physical activity. Such integrated lifestyle interventions can significantly improve blood pressure control, reduce long-term cardiovascular complications, and enhance patients' overall health and quality of life. Future healthcare approaches should prioritize these modifiable risk factors to achieve sustainable and patient-centered hypertension management.

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