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The Silent Risks of Sleepless Nights: Can short sleep duration be considered a significant risk factor for hypertension? - A literature review

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Abstract

Introduction and purpose

Hypertension is one of the most common diseases worldwide affecting more than 30% of the global adult population. It can lead to serious complications. Making it a major concern for modern medicine. While traditional risk factors such as obesity, diet and lifestyle choices are well known, recent evidence suggests that short sleep duration may also play a significant role in the development of hypertension. The aim of this study is to summarise information on the impact of short sleep duration on the incidence and pathophysiology of hypertension.

Methods

A systematic review of scientific articles published between 2015 and 2024 was conducted using PubMed. Articles were searched using the following terms: hypertension, high blood pressure, short sleep duration, sleep deficiency.

Brief description of the state of knowledge

Current literature suggests that sleep deprivation influences blood pressure regulation through multiple pathways, including autonomic nervous system dysfunction, increased inflammation, oxidative stress, and metabolic disturbances. Observational studies indicate a higher incidence of hypertension among individuals sleeping less than six hours per night, with variations based on age and sex. Meta-analyses confirm the relationship, where short sleep durations correlate with increased hypertension risk.

Summary

Short sleep duration is a modifiable yet often overlooked risk factor for hypertension. Given the high prevalence of sleep deficiency in modern society, integrating sleep hygiene recommendations into hypertension prevention strategies may be beneficial. Further research utilizing objective sleep measurements is essential to establish causality and develop targeted interventions.

Keywords: hypertension; high blood pressure; sleep deficiency; short sleep duration

Introduction

High blood pressure poses a major worldwide health issue, impacting more than 30% of adults across the globe.[1] Hypertension is diagnosed when resting blood pressure is consistently 140/90 mm Hg or higher in a clinical or office setting.[2] Hypertension is a medical condition characterised by consistently elevated arterial blood pressure. Although typically asymptomatic, hypertension can lead to serious complications including: atrial fibrillation, coronary artery disease, heart failure, stroke, dementia, chronic kidney disease, peripheral vascular disease etc.[3,4] The role of hypertension in the onset of heart failure is well established. It is mainly associated with long-term left ventricular pressure overload and elevated blood volume, which impact the heart's structure and function through various molecular pathways.[5] Risk factors for high blood pressure are well known and mainly include obesity and unhealthy lifestyle behaviours, such as alcohol consumption and high meat consumption.[6] The available evidence from both observational and experimental studies indicates that insufficient sleep duration is also a significant risk factor for cardiovascular disease and mortality. It appears that

sleep deprivation can lead to adverse, long-lasting physiological changes that are likely to work together to increase the risk of developing cardiovascular pathology.[7,8]

It is important to look at a regular night's sleep before understanding how sleep disturbances facilitate the onset of hypertension. According to evidence-based recommendations, the appropriate sleep duration for adults is between 7 and 9 hours per night.[9] Each stage of sleep is characterized by a specific duration, distinct physiological processes, and unique blood pressure patterns.[8]

Given the widespread problem with sleep deficit and the scientific evidence linking sleep deprivation to the onset of hypertension, it is worth taking a closer look at this issue.[8]

Objective of this work

This study aims to review the impact of short sleep duration on the incidence and pathophysiology of hypertension.

Materials and methods

A systematic review of scientific papers with full text was carried out, based on the PubMed. The following search terms were used: ‘hypertension, ‘high blood pressure’, ‘short sleep duration’ and ‘sleep deficiency’ in appropriate combinations. Articles and research papers published between 2015 and 2024 were the focus of the analysis. The titles and abstracts were manually screened to assess their relevance and the origin of the article. The collected data were analyzed and summarized. Since this study was not designed as a meta-analysis, no statistical methods were applied.

State of knowledge

The issue of sleep restriction and insomnia

Sleep disorders encompass a broad spectrum of conditions. Insomnia is defined as a difficulty in initiating or staying asleep leading to daytime impairments, despite having adequate sleep conditions and sufficient time allocated for rest. Insufficient sleep has become more pervasive, but its effects on health and overall quality of life remains incompletely understood. This issue has become a prevalent health problem in developed countries. Roughly one-third of Americans report sleeping less than 7 hours per night. Since the 1980s, average

self-reported sleep duration has declined, as evidenced by a 30% increase in the number of adults sleeping less than 6 hours per night. Regions with the highest rates of cardiometabolic diseases tend to have noticeable patterns of shorter sleep durations.[10–12] Insomnia, as a condition, affects up to 50% of the general population at any given time and chronically impacts approximately 10%–20% of adults. People with insomnia often report difficulties falling asleep or staying asleep and face a 23% higher risk of cardiovascular mortality, primarily due to a 50% increased likelihood of developing hypertension.[13,14] Insomnia disorder characterized by objectively short sleep duration (less than 6 hours of measured sleep or sleep efficiency below 85%) is recognized as a biologically severe subtype of insomnia, linked to a heightened risk of cardiometabolic morbidity.[13]

Tab 1. Criteria for the diagnosis of insomnia: [15,16]

I: Required all 4 major criteria:
1. Early awakening 2. Difficulty falling asleep 3. Difficulty staying asleep 4. Daytime impairment manifested by at least one of the following : daytime sleepinees, fatigue or malaise, mood disturbance, reduced motivation or energy, poor concentration, increased accidents, ongoing worry about sleep, educational or social dysfuncion
II: Occurs at least 3 times per week for at least 1 month
III: Not related to inadequate opportunity, an inappropriate sleep environment or another sleep disorder

The physiology of sleep and the cardiovascular changes that occur during sleep

While the exact biological significance of sleep remains a topic of debate, it is clear that sleep is a complex physiological phenomenon. It engages a variety of biological pathways, ranging from neural cortical circuits to cardiovascular systems. Compared to wakefulness, most biological functions of the body undergo significant changes during sleep. When we sleep, blood pressure and heart rate decrease. Blood pressure and heart rate decrease, along with

changes in body temperature, hormone secretion, and immune function. Sleep has a profound effect on cardiovascular regulation. This relationship is bidirectional. Cardiovascular disease can disrupt normal sleep patterns, while sleep disorders can have a significant impact on cardiovascular function, increasing the risk of cardiovascular disease.[17,18] It is important to note that each stage of sleep has a unique duration, physiology and blood pressure pattern. Sleep can be divided into two main stages. The first, Non-Rapid Eye Movement (NREM) sleep, is characterised by a progressive decrease in blood pressure, heart rate, cardiac output and sympathetic nerve activity.[19] During NREM sleep, the least time is spent in stage 1 (N1), while stage 2 (N2) is the longest, regardless of age. Stage 3, or slow-wave sleep, decreases with age, mainly because of more frequent waking after falling asleep.[19] Characteristics of rapid eye movement (REM), the second main part of sleep, include blood pressure and heart rate levels similar to waking levels, accompanied by a marked increase in sympathetic nerve traffic.[8]

Potential pathomechanisms linking hypertension and short sleep duration

Tab 2. Areas of pathophysiological change resulting in hypertension, that may be caused by short sleep duration.

1. The autonomic nervous system.
2. Inflammation.
3. Oxidative stress and endothelial dysfunction.
4. Metabolic and endocrine functions.

1. The autonomic nervous system (ANS) has been extensively studied as a potential factor in the heightened risk of cardiovascular disease following sleep deprivation. Nearly all evidence points to an increase in sympathetic activity after partial or complete acute sleep deprivation or sleep fragmentation. The autonomic nervous system, consisting of the sympathetic and parasympathetic (vagal) branches, maintains blood pressure stability through various reflex mechanisms, with the baroreflex playing a central role.[20,21] After a full night of sleep deprivation, a decrease in overall variability was observed, indicating a reduced ability of the cardiovascular system to respond to stress. Additionally, there is a shift in the balance between the sympathetic and parasympathetic nervous systems, with the sympathetic system becoming more dominant. Sleep deprivation indicating baroreflex dysfunction may contribute

to elevations in blood pressure. That is why sleep deprivation has important effects on haemodynamic and autonomic parameters.[22]

2. Inflammation plays a key role in the development of cardiovascular diseases. It is a complex process involving the overproduction of various adhesion molecules, chemokines, cytokines, and growth factors, which is central to the development of atherosclerosis. Additionally, high levels of pro-inflammatory biomarkers have been found in obesity and other conditions linked to an increased risk of cardiovascular disease. As a result, a pro-inflammatory state is believed to be a possible mechanism contributing to the heightened cardiovascular risk in individuals who get insufficient sleep.[22] The study found that restricting sleep to four hours resulted in increased production of IL-6 and TNF by monocytes, which are pro-inflammatory cytokines involved in the initiation of the acute phase reaction.[23] It would appear that acute sleep deprivation affects the circadian pattern of IL-6 secretion, resulting in excessive secretion during the day and insufficient secretion at night. There is a negative correlation between diurnal IL-6 levels and the amount of sleep obtained the previous night. In rodent studies, IL-6 has been shown to inhibit vasodilation and enhance systemic vasoconstriction. Therefore, high levels of IL-6 may explain the increase in blood pressure found after sleep deprivation. Furthermore, high levels of IL-6 have been identified as one of the mechanisms underlying daytime sleepiness and fatigue.[21,22]

C-reactive protein (CRP), an acute phase reactant, is also a biomarker that increases after time without sleep according to some of the studies. It is a protein synthesised in the liver mainly under the control of IL-6. It has been thoroughly investigated as an indicator of cardiovascular events.[24] Elevated CRP levels were observed in 10 healthy individuals following either total sleep deprivation (88 hours) or partial sleep deprivation (4 hours of sleep per night for 10 consecutive days).[25] While results regarding individual pro-inflammatory biomarkers are sometimes inconsistent - mainly due to variations in experimental conditions and differences in blood collection methods - multiple studies consistently demonstrate a pro-inflammatory response associated with both partial and total sleep deprivation. This state may contribute to increased oxidative stress, endothelial dysfunction, the release of prothrombotic factors and ultimately an increased risk of atherosclerosis. While the precise mechanisms underlying the inflammatory response associated with short sleep duration remain unclear, increased sympathetic activity is thought to play a crucial role.[18,22]

3. Oxidative stress and endothelial dysfunction is a potential mechanism that may explain the heightened risk of cardiovascular disease linked to short sleep durations too. Temporary sleep deprivation in experimental settings has been associated with a rise in

myeloperoxidase, an enzyme that contributes to the generation of oxidizing agents capable of converting LDL into its atherogenic form. Furthermore, sleep deprivation has been shown to increase levels of insulin-like growth factor I (IGF1), which is known to trigger the release of oxidant agents from activated neutrophils. This may help explain the observed elevation in myeloperoxidase levels.[26,27] Reactive oxygen species are important for vascular cell function, including endothelial cell growth and migration, vascular tone, apoptosis and angiogenesis. They also influence cardiac contractility, remodelling by hypertrophic signalling or necrosis. However, excessive levels of reactive oxygen species can induce vascular disease through direct and irreversible oxidative damage.[27,28]

4. Metabolic and endocrine functions: Sleep and circadian rhythms are essential in managing metabolic and hormonal processes. It is also a well-established fact that metabolic changes, such as increased weight, are among the causes of hypertension. Short sleep duration appears to be associated with negative changes in the secretion of the hormones leptin and ghrelin, which we link to increased incidence of obesity. This assertion has been corroborated by numerous research conducted on rats.[22] These hormones are involved in controlling satiety and hunger. Specifically, short sleep duration has been associated with decreased leptin levels and increased ghrelin levels. This has been demonstrated to lead to activation of the hunger and satiety centre, which, in turn, has been shown to result in increased food intake, including fast food.[29,30] Calvin et al. show an increase in total calorie intake of 500 kcal per day after 1.5 hours less sleep over 8 days.[31] It is important to note that there are additional studies that support the notion of increased total food intake after sleep deprivation. However, it should be acknowledged that the data are not always consistent, and no changes in leptin and ghrelin levels have been reported after several days of partial sleep deprivation.[31]

Similar scientific studies have also shown elevated fasting glucose levels in adolescents after sleep deprivation. This correlated with reduced insulin sensitivity.[32] The study revealed an elevated low-frequency to high-frequency ratio, pointing to sympathetic overactivity as a potential driver of insulin resistance and diminished pancreatic insulin secretion. In summary, some data indicate that we can link sleep deprivation to increased glucose levels, insulin levels and increased calorie intake. And changes in metabolism as well as weight gain will be associated with increased blood pressure.[22,32,33]

What meta-analyses show us?

In the scientific literature, multiple meta-analyses have examined the relationship between hypertension and short sleep duration. Researchers have conducted studies on diverse age groups, including both males and females. The results varied depending on age group,

gender, and study design—whether cross-sectional or prospective. Nevertheless, evidence suggests a potential causal relationship between short sleep duration and the subsequent development of hypertension.[34]

In a comprehensive meta-analysis, M. Grandner et al. evaluated data from over 700,000 participants, all of whom were adults over the age of 18 and residents of the USA. Survey respondents were randomly selected based on age, gender, and race to best reflect a representative cross-section of the population. The data were primarily collected via telephone interviews and included questions regarding participants' health and risk factors. Respondents provided detailed information on sleep duration, self-reported high blood pressure, and other relevant characteristics. The data were gathered over a 10-year period, with a total sample size of 728,717 participants. The study concluded that there was a strong association between subjective short sleep duration and hypertension in a large sample of US adults. Further refined analyses and categorized sub-analyses indicated that the link between insufficient sleep and hypertension was more robust and consistent than the association between excessive sleep and hypertension. Moreover, short sleep duration was consistently more strongly associated with hypertension risk in women compared to men across the adult lifespan.[35]

The meta-analysis by Li et al. was based on nine high quality prospective cohort studies that were published between 2004 and 2017 and included 48525 subjects (including 15276 hypertensive cases). It was mentioned that the lowest risk of hypertension in the study was observed in people sleeping between 7 and 7.9 hours per night. As one moves away from this reference point, there is in turn an increase in risk. The researchers concluded that the elevated risks associated with both short and long sleep were consistent, irrespective of multifactorial adjustments for lifestyle, clinical and sleep-related concomitant variables. It has been established that in younger individuals, a comparatively brief duration of sleep is a notable risk factor for hypertension. However, this study posits that such a correlation is not evident in older populations. In elderly subjects, both wrist actigraphy-assessed sleep duration and self-reported sleep duration demonstrate an absence of correlation with the incidence of hypertension. In their conclusion, the authors add that the risk of hypertension was reduced by 0.3207% when sleep time was increased by 1 hour.[4]

In another study, Wang et al. also investigated the relationship between sleep duration and hypertension in adults. The authors emphasised the role of sleep in our lives and highlighted the problem of decreasing average sleep duration per day in the adult population. For example, the National Sleep Foundation reported an increase from 12% to 16% of subjects sleeping less than 6 hours on workdays between 1998 and 2005. The study also addressed the issue of

defining short sleep duration. The available literature was reviewed, and the following definitions were identified: ≤ 5, 4-5, ≤ 6, or < 7 hours per night. The study focused exclusively on nighttime sleep duration, rather than total 24-hour sleep, which could be a crucial factor for the elderly population that takes regular naps. The discrepancies in the definitions could potentially lead to difficulties in accurately interpreting the results. The study comprised 347,759 participants, 115,007 of whom had hypertension. The number of cases and total participants for each sleep duration category were as follows: 7,452 of 19,695 had ≤ 5 hours of sleep; 17,524 of 53,603 had 6 hours; 26,648 of 92,895 had 7 hours; 41,073 of 126,544 had 8 hours; and 22,310 of 54,534 had ≥ 9 hours. All participants were > 18 years old. Their analysis revealed that, compared to individuals who slept 7 hours per day, all other sleep duration groups were associated with an increased risk of hypertension. The highest odds ratio (OR) was observed for sleep duration of ≤ 5 hours compared to 7 hours, and this remained statistically significant even after adjustment for age and sex. These findings suggest that both insufficient and excessive sleep may be potential risk factors for hypertension, especially in women. Researchers also suggest that, although sleep deprivation appears to cause hypertension, the mechanisms underlying this association are not yet fully comprehended. The principal mechanism appears to be the effect of sleep deprivation as a stressor on the body. This activates the sympathetic nervous system, which can trigger a series of events responsible for the increase in pressure, including stimulation of the renin-angiotensin-aldosterone system and increased synthesis of central catecholamines.[36]

Conclusions

Short sleep duration is an emerging yet frequently overlooked risk factor for hypertension. Evidence from epidemiological and mechanistic studies suggests that insufficient sleep contributes to elevated blood pressure mainly through autonomic nervous system dysregulation, increased inflammatory response, oxidative stress, and metabolic disturbances. The association between short sleep duration and hypertension appears to be particularly significant among younger individuals and women. Given the widespread prevalence of sleep deficiency in modern society, sleep hygiene should be recognized as an essential component of cardiovascular disease prevention. Future research should focus on prospective cohort studies and interventional trials utilizing objective sleep assessments, including polysomnography, to further elucidate the causal relationship between sleep duration and hypertension. Additionally, it is imperative to investigate the pathomechanisms that remain to be elucidated regarding the

association between sleep disorders and hypertension. Exploring the role of circadian rhythm disruptions in hypertension pathogenesis may provide valuable insights into personalized therapeutic strategies. Finally, the integration of sleep assessment into routine clinical practice and public health recommendations holds significant potential for enhancing the effectiveness of hypertension prevention and management.

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References

1. Egan BM, Kjeldsen SE, Grassi G, Esler M, Mancia G. The global burden of hypertension exceeds 1.4 billion people: should a systolic blood pressure target below 130 become the universal standard? *J Hypertens*. 2019;37(6):1148-1153. doi:10.1097/HJH.0000000000002021

2. Poulter NR, Prabhakaran D, Caulfield M. Hypertension. *The Lancet*. 2015;386(9995):801-812. doi:10.1016/S0140-6736(14)61468-9

3. Lackland DT, Weber MA. Global Burden of Cardiovascular Disease and Stroke: Hypertension at the Core. *Can J Cardiol*. 2015;31(5):569-571. doi:10.1016/j.cjca.2015.01.009

4. Li H, Ren Y, Wu Y, Zhao X. Correlation between sleep duration and hypertension: a dose-response meta-analysis. *J Hum Hypertens*. 2019;33(3):218-228. doi:10.1038/s41371-018-0135-1

5. Gallo G, Savoia C. Hypertension and Heart Failure: From Pathophysiology to Treatment. *Int J Mol Sci*. 2024;25(12):6661. doi:10.3390/ijms25126661

6. Ondimu DO, Kikuvi GM, Otieno WN. Risk factors for hypertension among young adults (18-35) years attending in Tenwek Mission Hospital, Bomet County, Kenya in 2018. *Pan Afr Med J*. 2019;33. doi:10.11604/pamj.2019.33.210.18407

7. Covassin N, Singh P. Sleep Duration and Cardiovascular Disease Risk. *Sleep Med Clin*. 2016;11(1):81-89. doi:10.1016/j.jsmc.2015.10.007

8. Bock JM, Vungarala S, Covassin N, Somers VK. Sleep Duration and Hypertension: Epidemiological Evidence and Underlying Mechanisms. *Am J Hypertens*. 2022;35(1):3-11. doi:10.1093/ajh/hpab146

9. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's sleep time duration recommendations: methodology and results summary. *Sleep Health*. 2015;1(1):40-43. doi:10.1016/j.sleh.2014.12.010

10. St-Onge MP, Campbell A, Aggarwal B, Taylor JL, Spruill TM, RoyChoudhury A. Mild sleep restriction increases 24-hour ambulatory blood pressure in premenopausal women with no indication of mediation by psychological effects. *Am Heart J*. 2020;223:12-22. doi:10.1016/j.ahj.2020.02.006

11. Ford ES, Cunningham TJ, Croft JB. Trends in Self-Reported Sleep Duration among US Adults from 1985 to 2012. *Sleep*. 2015;38(5):829-832. doi:10.5665/sleep.4684

12. Liu Y, Wheaton AG, Chapman DP, Cunningham TJ, Lu H, Croft JB. Prevalence of Healthy Sleep Duration among Adults — United States, 2014. *MMWR Morb Mortal Wkly Rep*. 2016;65(6):137-141. doi:10.15585/mmwr.mm6506a1

13. Johnson KA, Gordon CJ, Chapman JL, et al. The association of insomnia disorder characterised by objective short sleep duration with hypertension, diabetes and body mass index: A systematic review and meta-analysis. *Sleep Med Rev*. 2021;59:101456. doi:10.1016/j.smrv.2021.101456

14. Bollu PC, Kaur H. Sleep Medicine: Insomnia and Sleep.

15. Sateia MJ. International Classification of Sleep Disorders-Third Edition. *Chest*. 2014;146(5):1387-1394. doi:10.1378/chest.14-0970

16. Roth T. Insomnia: Definition, Prevalence, Etiology, and Consequences. *J Clin Sleep Med*. 2007;3(5 suppl). doi:10.5664/jcsm.26929

17. Kendzerska T, Mollayeva T, Gershon AS, Leung RS, Hawker G, Tomlinson G. Untreated obstructive sleep apnea and the risk for serious long-term adverse outcomes: A systematic review. *Sleep Med Rev*. 2014;18(1):49-59. doi:10.1016/j.smrv.2013.01.003

18. Tobaldini E, Costantino G, Solbiati M, et al. Sleep, sleep deprivation, autonomic nervous system and cardiovascular diseases. *Neurosci Biobehav Rev*. 2017;74:321-329. doi:10.1016/j.neubiorev.2016.07.004

19. Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV. Meta-Analysis of Quantitative Sleep Parameters From Childhood to Old Age in Healthy Individuals: Developing Normative Sleep Values Across the Human Lifespan. *Sleep*. 2004;27(7):1255-1273. doi:10.1093/sleep/27.7.1255

20. Ogawa Y, Kanbayashi T, Saito Y, et al. Total Sleep Deprivation Elevates Blood Pressure Through Arterial Baroreflex Resetting: a Study with Microneurographic Technique. *Sleep*. 2003;26(8):986-989. doi:10.1093/sleep/26.8.986

21. Tobaldini E, Cogliati C, Fiorelli EM, et al. One night on-call: Sleep deprivation affects cardiac autonomic control and inflammation in physicians. *Eur J Intern Med*. 2013;24(7):664-670. doi:10.1016/j.ejim.2013.03.011

22. Tobaldini E, Fiorelli EM, Solbiati M, Costantino G, Nobili L, Montano N. Short sleep duration and cardiometabolic risk: from pathophysiology to clinical evidence. *Nat Rev Cardiol*. 2019;16(4):213-224. doi:10.1038/s41569-018-0109-6

23. Irwin MR. Sleep Deprivation and Activation of Morning Levels of Cellular and Genomic Markers of Inflammation. *Arch Intern Med*. 2006;166(16):1756. doi:10.1001/archinte.166.16.1756

24. Ridker PM. C-Reactive Protein and the Prediction of Cardiovascular Events Among Those at Intermediate Risk. *J Am Coll Cardiol*. 2007;49(21):2129-2138. doi:10.1016/j.jacc.2007.02.052

25. Meier-Ewert HK, Ridker PM, Rifai N, et al. Effect of sleep loss on C-Reactive protein, an inflammatory marker of cardiovascular risk. *J Am Coll Cardiol*. 2004;43(4):678-683. doi:10.1016/j.jacc.2003.07.050

26. Zouaoui Boudjeltia K, Faraut B, Esposito MJ, et al. Temporal Dissociation between Myeloperoxidase (MPO)-Modified LDL and MPO Elevations during Chronic Sleep Restriction

and Recovery in Healthy Young Men. Goel N, ed. *PLoS ONE*. 2011;6(11):e28230. doi:10.1371/journal.pone.0028230

27. Münzel T, Camici GG, Maack C, Bonetti NR, Fuster V, Kovacic JC. Impact of Oxidative Stress on the Heart and Vasculature. *J Am Coll Cardiol*. 2017;70(2):212-229. doi:10.1016/j.jacc.2017.05.035

28. Paneni F, Diaz Cañestro C, Libby P, Lüscher TF, Camici GG. The Aging Cardiovascular System. *J Am Coll Cardiol*. 2017;69(15):1952-1967. doi:10.1016/j.jacc.2017.01.064

29. St-Onge MP, O'Keeffe M, Roberts AL, RoyChoudhury A, Laferrère B. Short Sleep Duration, Glucose Dysregulation and Hormonal Regulation of Appetite in Men and Women. *Sleep*. 2012;35(11):1503-1510. doi:10.5665/sleep.2198

30. Broussard JL, Castro AVB, Iyer M, et al. Insulin access to skeletal muscle is impaired during the early stages of diet-induced obesity. *Obesity*. 2016;24(9):1922-1928. doi:10.1002/oby.21562

31. Calvin AD, Covassin N, Kremers WK, et al. Experimental Sleep Restriction Causes Endothelial Dysfunction in Healthy Humans. *J Am Heart Assoc*. 2014;3(6):e001143. doi:10.1161/JAHA.114.001143

32. De Bernardi Rodrigues AM, Da Silva CDC, Vasques ACJ, et al. Association of Sleep Deprivation With Reduction in Insulin Sensitivity as Assessed by the Hyperglycemic Clamp Technique in Adolescents. *JAMA Pediatr*. 2016;170(5):487. doi:10.1001/jamapediatrics.2015.4365

33. Tasali E, Leproult R, Ehrmann DA, Cauter EV. Slow-wave sleep and the risk of type 2 diabetes in humans.

34. Gangwisch JE. A Review of Evidence for the Link Between Sleep Duration and Hypertension. *Am J Hypertens*. 2014;27(10):1235-1242. doi:10.1093/ajh/hpu071

35. Grandner M, Mullington JM, Hashmi SD, Redeker NS, Watson NF, Morgenthaler TI. Sleep Duration and Hypertension: Analysis of > 700,000 Adults by Age and Sex. *J Clin Sleep Med*. 2018;14(06):1031-1039. doi:10.5664/jcsm.7176

36. Wang Y, Mei H, Jiang YR, et al. Relationship between Duration of Sleep and Hypertension in Adults: A Meta-Analysis. *J Clin Sleep Med*. 2015;11(09):1047-1056. doi:10.5664/jcsm.5024