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Relationship between Sleep Deprivation and Risk of Cardiovascular Diseases:

– A Narrative Review

by

Dr. Apurva Ahuja

Under the guidance of

Dr. Sukesh Bhardwaj

PGDM (Hospital and Health Management)

2023-25

International Institute of Health Management Research New Delhi

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TO WHOMSOEVER IT MAY CONCERN

This is to certify that DR APURVA AHUJA student of PGDM (Hospital & Health Management) from International Institute of Health Management Research, New Delhi has undergone internship training at CARPL.ai from 24TH FEB 2025 to 24TH May 2025.

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This is to certify that the dissertation titled "**Relationship between Sleep Deprivation and Risk of Cardiovascular Diseases: A Narrative Review**" and submitted by **DR APURVA AHUJA** Enrollment No. **PG/23/023** under the supervision of **DR SUKESH BHARDWAJ** for award of PGDM (Hospital & Health Management) of the Institute carried out during the period **from 24TH FEB 2025 to 24TH MAY 2025** embodies my original work and has not formed the basis for the award of any degree, diploma associate ship, fellowship, titles in this or any other Institute or other similar institution of higher learning.

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Certificate of Approval

The following dissertation titled **"Relationship between Sleep deprivation and risk of cardiovascular diseases - a narrative review"** at **IIHMR Delhi** is hereby approved as a certified study in management carried out and presented in a manner satisfactorily to warrant its acceptance as a prerequisite for the award of **PGDM (Hospital & Health Management)** for which it has been submitted. It is understood that by this approval the undersigned do not necessarily endorse or approve any statement made, opinion expressed or conclusion drawn therein but approve the dissertation only for the purpose it is submitted.

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Dr. Shiv	
Dr. Sumant Swain	

Certificate from Dissertation Advisory Committee

This is to certify that **DR. APURVA AHUJA,** a graduate student of the **PGDM (Hospital & Health Management)** has worked under our guidance and supervision. She is submitting this dissertation titled "**Relationship between Sleep Deprivation and Risk of Cardiovascular Diseases: A Narrative Review**" at "CARPL.ai" in partial fulfillment of the requirements for the award of the **PGDM (Hospital & Health Management).**

This dissertation has the requisite standard and to the best of our knowledge no part of it has been reproduced from any other dissertation, monograph, report or book.

Dr. Sukesh Bhardwaj
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FEEDBACK FORM

Name of the Student: Dr. Apurva Ahuja

Name of the Organisation in Which Dissertation Has Been Completed: CARPL.AI

Area of Dissertation: Customer Success and Account Management (CSAM)

Attendance: 100%

Objectives achieved:

1. Ensured smooth AI deployment post-sales by coordinating with clients and internal technical teams.
2. Liaised with deployment, tech, and AI teams to resolve bugs, address feature requests, and support validation project needs.
3. Participated in client-facing calls, including tri-party validation discussions and weekly syncs, ensuring timely resolution of client requirements.
4. Maintained internal trackers, documents, and project plans to monitor progress, identify discrepancies, and enable informed decision-making.

Deliverables:

1. Coordinated the go-live for a new client site, ensuring all technical, validation, and communication steps were in place for a smooth launch.
2. Regularly compiled and shared comprehensive monthly updates for all US region accounts.
3. Raised and tracked issue tickets to closure, ensuring minimal client-side disruption and clear documentation for internal teams.

Strengths: Demonstrates strong ownership in ensuring smooth AI deployments and validation projects across US-based accounts.
Highly organized in maintaining trackers, documentation, and follow-ups across internal and external stakeholders.
Proactive in identifying and resolving issues, ensuring minimal disruption to client workflows.

Suggestions for Improvement: Increase direct interactions with customers to better understand their ongoing needs.

Suggestions for Institute (course curriculum, industry interaction, placement, alumni):

Signature of the Officer-in-Charge/ Organisation Mentor (Dissertation)

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This is to certify that the dissertation titled, "**Relationship between Sleep Deprivation and Risk of Cardiovascular Diseases: – A Narrative Review**," submitted by Dr. Apurva Ahuja Enrollment No. PG/23/023, under the supervision of Dr. Sukesh Bhardwaj, for the Institute's PGDM (Hospital & Health Management) award between February 24, 2025, and August 24, 2025, this is my original work. It has not been used as the basis for any degree, diploma, associateship, fellowship, or title from this Institute or any other comparable higher education institution.

Dr. Apurva Ahuja

Acknowledgement

Working on this dissertation has been a truly enriching experience, both in terms of learning and personal growth. I feel fortunate to have had the chance to carry out my research at **CARPL.AI**, where the practical exposure and guidance from experienced professionals added immense value to my work.

I'm genuinely thankful to the entire team at **IIHMR Delhi**, the faculty and staff, whose constant support and positive academic environment made it easier for me to focus on my research and expand my skill set. Their professionalism and encouragement created the right setting to explore my interests.

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To everyone who helped me along the way—thank you. Your contributions, whether big or small, truly made a difference.

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LIST OF ABBREVIATIONS

Abbreviation	Full Form
AHA	American Heart Association
CAD	Coronary Artery Disease
CBT-I	Cognitive Behavioural Therapy used for Insomnia
CHD	Coronary Heart Disease
CI	Confidence Interval
CPAP	Continuous Positive Airway Pressure
CVD	Cardiovascular Disorders
HF	Heart Failure
hs-CRP	High-sensitivity C-Reactive Protein
IL-6	Interleukin 6
MI	Myocardial Infarction
MR	Mendelian Randomization Technique
MACE	Major Adverse Cardiac Events
MSNA	Muscle Sympathetic Nerve Activity
NO	Nitric Oxide
OSA	Obstructive Sleep Apnea
RCT	Randomized Controlled Trial
ROS	Reactive Oxygen Species
RR	Relative Risk Ratio
SNS	Sympathetic Nervous System
TNF-α	Tumor Necrosis Factor Alpha

ABOUT THE ORGANIZATION

About CARPL

CARPL.ai is the world's first testing and deployment platform for medical imaging AI applications, which connects healthcare providers to third party AI applications, helping improve access, affordability, and quality of medical care.

It bridges the gap between healthcare providers and AI developers by serving as a gatekeeper that seamlessly connects both sides of the ecosystem. In essence, it is a single interface to access AI algorithms, validate and test them, and subsequently embed them into radiology workflows.

It is used by some of the world's leading healthcare providers, AI researchers, industry teams and startups. It is built with the single goal of making it easy for clinicians to get access to advanced analytics tools.

Vision

The goal of CARPL is to become the go-to-market strategy of choice for AI developers and the single interface for AI deployment at healthcare providers, serving as the back-end platform for all medical imaging AI deployments worldwide.

Features

Easy to Access: There is a fragmented ecosystem out there if any HCP wanted to deploy any radio diagnostic AI solution how they can contact AI developers providing similar solutions individualistically, but CARPL platform have 100+ AI solutions of different AI developer on a simple single user- friendly platform.

Easy to Assess: CARPL is the first company to provide the feature of Validation and Testing & Monitoring of the AI solutions for the HCPs so they can analyze the efficiency of the AI model over their own Patient data.

Easy to Integrate: If the HCPs starts deploying AI solutions related to radiodiagnosis by multiple AI developers for different use cases they would require different RIS-PACS integrations but for accessing multiple AI solutions via CARPL requires single RIS-PACS setup.

Reliability: All the AI solutions onboarded on CARPL's platform are regulatory compliant including FDA(US), CE(Europe), HSA(Singapore), ANVISA(Brazil) and TGA (AUS).

1. PROJECT TITLE

Relationship between Sleep Deprivation and Risk of Cardiovascular Diseases:– A Narrative Review

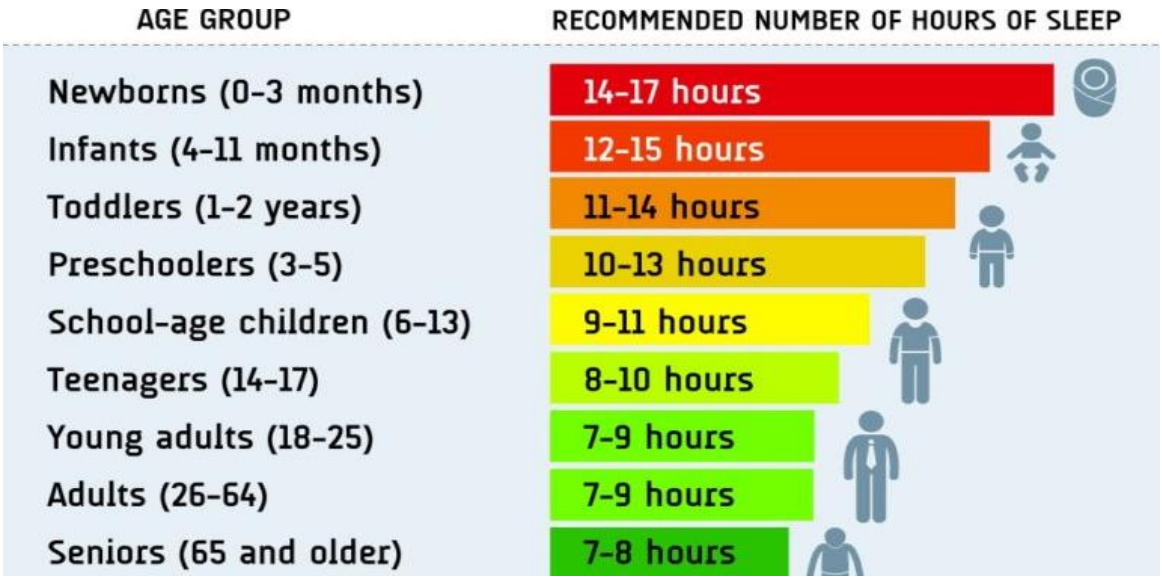

Figure1.1

2. ABSTRACT

- **Background of the study-**

Cardiovascular disease (CVD) is one of the leading causes of illness and mortality globally, resulting from a complex interplay of genetic predispositions, environmental exposures, and lifestyle habits. Traditionally, prevention strategies have centred on well- known risk factors include maintaining a healthy diet, regular physical activity, stopping smoking, and managing conditions like diabetes and high blood pressure. However, sleep has become an important yet often ignored factor in keeping heart health. More research shows a strong link between not getting enough sleep or having poor-quality sleep and developing cardiovascular disease (CVD). The American Heart Association (AHA) recognized this by making sleep duration a key part of its updated 2022 "Life's Essential 8" guidelines for cardiovascular health. Still, sleep gets too little attention in clinical practice and public health policies.

- **Objective-**

This narrative review seeks to consolidate current research findings on the epidemiological

relationship, underlying biological mechanisms, and clinical consequences that connect sleep deprivation with cardiovascular disease.

- **Methods-**

A literature search using keywords was performed across databases such as PubMed, Scopus, and Google Scholar. This search focused on peer-reviewed publications from 1990 to 2024. Official guidelines and scientific reports from top health organizations were also reviewed.

- **Results-**

Both insufficient and excessive sleep durations are linked to a U-shaped trend of increased cardiovascular risk. Not getting enough sleep causes various physiological issues, including metabolic imbalance, problems with blood vessel function, chronic low-grade inflammation, and irregularities in the autonomic nervous system. Clinically, this is connected to a higher risk of conditions like heart failure, stroke, coronary artery disease, and hypertension. These risks increase when coupled with sleep-related disorders, especially obstructive sleep apnea and insomnia. Other factors, such as hormonal changes, shift work schedules, and demographic variables, also affect an individual's vulnerability.

- **Conclusion-**

Sleep is a key component of heart health. It is important to identify and treat sleep deprivation in both medical and public health contexts to lessen the worldwide impact of cardiovascular disease (CVD). Future studies should concentrate on intervention trials and include sleep data in regular heart risk assessments.

INTRODUCTION:

The understanding of cardiovascular health and its determinants has undergone a significant transformation in the 21st century. Historically, the cornerstones of cardiovascular disease (CVD) prevention have been diet, physical activity, smoking cessation, and the management of clinical risk factors such as hypertension and dyslipidemia.

Sleep, while acknowledged as important for general well-being, was often relegated to a secondary role—a passive state of recovery rather than an active, modifiable determinant of health. However, a robust and growing body of scientific evidence has compelled a fundamental paradigm shift, elevating sleep to a status co-equal with other major lifestyle factors.

The most definitive signal of this paradigm shift came by the American Heart Associations (AHA), which, in 2022', updated its metrics for the ideal cardiovascular healthy from "Life's Simple 7" to "Life's Essential 8," with the novel inclusion of sleep duration.[1] This landmark decision was the culmination of decades of accumulating evidence, formally recognizing that the quantity and quality of sleep are foundational inputs that actively regulate cardiovascular function.[1] This urgency is underscored by the widespread insufficiency of sleep in modern society, a trend often described as an public health epidemic.[5] In the United States, about one-third of adults fail to achieve the recommended amount of sleep on a regular basis, a phenomenon driven by the pressures of a 24/7 society.[6]

A sophisticated understanding of the relation between sleep deprivation and CVD requires moving beyond the simple metric of sleep duration to a multidimensional model of "sleep health" that encompasses duration, quality, regularity, and architecture.[3] This review synthesizes the evidence establishing sleep as an indispensable pillar of cardiovascular health, details the pathophysiological mechanisms linking sleep loss to vascular injury, and examines the clinical manifestations and modulating factors that influence this complex relationship.

3. OBJECTIVES:

Primary Objective:

1. To assess the correlation between sleep deprivation and the risk of developing cardiovascular disease, based on current evidence.

Secondary Objectives:

1. To investigate how certain aspects of sleep, including duration, quality, and conditions like obstructive sleep apnoea and insomnia, affect cardiovascular risk.
2. To identify and analyze population-level modifiers (age, gender, occupation) and underlying biological mechanisms (autonomic, inflammatory, metabolic) influencing this relationship.

4. RESEARCH QUESTION:

Is there a relationship between sleep deprivation and the risk of developing cardiovascular diseases?

5. RESEARCH METHODOLOGY

Type of Study: Narrative Review

Search Strategy:

1. A term-based literature search was carried out in Google Scholar and PubMed, two significant databases, taking into account articles published between 1990 and 2024.
2. Peer-reviewed journal articles, systematic reviews, and meta-analyses were the primary search criteria.

Keywords Used:

1. "Sleep deprivation," "insufficient sleep," "insomnia," "obstructive sleep apnea," "cardiovascular diseases," "hypertension," "coronary artery diseases," "stroke."
2. Boolean operators (AND, OR) were utilized to refine searches.

Filters Applied:

1. Publication years: **Last 15 years**
2. Language: **English**
3. Population: **Human adults (18+)**
4. Article types:

1. Meta-analyses
2. Systematic Reviews
3. Randomized Control Trials (RCTs)
4. Observational studies
5. Scoping reviews

Article Selection Process

1. Initial Yield: ~1,000 articles identified through keyword search

2. Screening Process:

- Title screening → shortlisted 150 articles
- Abstract screening → selected 20 core articles

3. Inclusion Criteria:

- Direct relevance to cardiovascular impact of sleep deprivation.

- The review focused on synthesizing evidence regarding the epidemiological associations, pathophysiological mechanisms, and clinical outcomes linking sleep health to cardiovascular status.
- Relevant guidelines and scientific statements from organizations such as the American Heart Association (AHA) were also manually searched and incorporated to provide a current and authoritative perspective.

4. **Saturation Point:** Further literature showed repetitive findings

6. RESULTS:

Section 1: Understanding the U-Shaped Link Between Sleep Duration and Cardiovascular Risk

Research over the years has consistently indicated that the length of sleep can affect cardiovascular health. Numerous extensive studies across various demographic groups have identified a distinct, curved relationship between an individual's sleep duration and their risk of cardiovascular issues or mortality. Notably, this connection does not adhere to a linear pattern; it frequently appears as a "U-shaped" or at times a "J-shaped" curve, as stated by specialists. In basic terms, adequate sleep is generally the most advantageous, whereas extremely short and excessively long sleep periods are linked to increased health risks. This trend is now commonly acknowledged in the industry and has a major impact on health recommendations related to sleep.

1.1 The Cornerstone of Evidence: The U-Shaped and J-Shaped Relationships

The main conclusion from decades of observational research is that poor health outcomes are linked to both short and long sleep durations. People who consistently sleep for short periods, usually less than 6 or 7 hours per night, or for long periods, typically more than 8 or 9 hours per night, face a much higher risk of cardiovascular death, coronary heart disease (CHD), stroke, and death from all causes than people who sleep for moderate periods of time. The nadir of this risk curve, which represents the optimal amount of sleep for cardiovascular health, is typically found to be between 7 and 8 hours per night.[9]

The magnitude of this risk is clinically significant. Cappuccio et al . performed a groundbreaking analysis in 2011 that merged data of 15 studies with nearly 475,000

participants, yielding robust estimates of these associations. A 15% higher risk of stroke (RR=1.15, 95% CI 1.00–1.31) and a 48% higher risk of getting or dying from CHD (RR=1.48, 95% CI 1.22–1.80) were associated with short sleep duration. Additionally, a startling 65% increased risk of stroke (RR=1.65, 95% CI 1.45–1.87) and a 38% increased risk of CHD (RR=1.38, 95% CI 1.15–1.66) were associated with long sleep duration.[10]

Subsequent meta-analyses have confirmed these findings. In a 2017 dose-response analysis, Yin et al. calculated the incremental risk and discovered that there was a 6% increase in all-cause mortality for every hour of sleep that was less than seven hours per day and a 13% increase for every hour of sleep that was more than seven hours.[20] These consistent results from numerous extensive evidence syntheses establish the U-shaped relationship as a key idea in the epidemiology of cardiovascular disease and sleep.

1.2 The Conundrum of Long Sleep

While the epidemiological relation between long sleep and adverse cardiovascular outcomes is robust, its interpretation is a subject of considerable scientific debate. Unlike for short sleep, where clear pathophysiological mechanisms are being elucidated, the link between long sleep and CVD is less mechanistically understood. The prevailing hypothesis is that habitual long sleep may not be a direct cause of cardiovascular disease but rather a sensitive *marker* or epiphenomenon of underlying, often undiagnosed, health problems.[25]

Several factors could confound the observed association. Pre-clinical or subclinical diseases, which may not be captured in baseline health assessments, can cause fatigue and increase sleep need. For instance, conditions such as chronic low-grade inflammation, early-stage heart failure, cancer, or anemia could manifest as longer sleep duration before a formal diagnosis is made. Furthermore, psychosocial factors are strongly linked to both long sleep and CVD risk. Depressive symptoms are a strong predictor of long sleep and are themselves a potent risk factor for cardiovascular disease.[27] Other potential confounders include physical inactivity, unemployment, and low socioeconomic status, all of which are associated with both increased sleep duration and poorer health outcomes.[27]

This puzzling hypothesis is backed by Mendelian randomization (MR) studies. MR uses genetic variations as tools to evaluate the causal relationship between an exposure, such as sleep duration, and an outcome, like cardiovascular disease (CVD). This method helps

eliminate confusion from lifestyle and environmental factors. Several MR studies have found a causal link between genetically predicted short sleep and CVD, they have often failed to show a similar link between long sleep and CVD.[28] While the negative effects of short sleep are more likely to be causal, the association for long sleep may actually be driven by reverse causation or confounding, according to this disparity between observational and genetic evidence.

Section 2: Pathophysiological Mechanisms: From Sleep Loss to Vascular Injury

The consistent epidemiological link between sleep deprivation and cardiovascular disease prompts a fundamental question: how does a behavioral state translate into cellular and organ-level pathology? The answer lies in a complex interplay of interconnected physiological systems that are profoundly dysregulated by the absence of sufficient sleep. Normal sleep is not merely a period of inactivity but a highly organized state of cardiovascular quiescence and repair. Sleep deprivation disrupts this essential process, initiating a cascade of maladaptive responses across the autonomic nervous, inflammatory, and metabolic systems. This section will dissect these intricate biological pathways, moving from the macro-level observation of risk to the micro-level mechanisms of vascular injury.

2.1 Autonomic Nervous System Dysregulation: The Sympathetic Overdrive

During the deep, slow-wave phases of sleep, there is a synchronized drop in central sympathetic outflow and a rise in parasympathetic vagal tone. This leads to cardiovascular quiescence, which is a reduction in heart rate, blood pressure, and total cardiac workload. One This healing process is disrupted by sleep deprivation, which keeps the body's sympathetic nervous system (SNS) hyperactive. 19. This "sympathetic overdrive" is thought to be a key mechanism connecting hypertension and sleep deprivation.[19]

One of the most critical consequences is the disruption of the normal nocturnal "dipping" of blood pressure, a 10–20% drop during sleep that is a vital protective mechanism.[1] Sleep deprivation significantly attenuates or eliminates this dip, a "non-dipping" profile that is a powerful predictor of future cardiovascular events.[1]

However, a pivotal study that used direct microneurographic recording discovered that muscle sympathetic nerve activity (MSNA) was significantly lower after one night of

complete sleep deprivation, even though blood pressure was higher. This casts doubt on a straightforward model of global sympathetic overdrive and raises the possibility that non-adrenergic pathways or locally specific sympathetic activation that is not detected by limb muscles could be responsible for the pressor effect.[37]

2.2 The Inflammatory Cascade and Endothelial Dysfunction

The vascular endothelium is the single layer of cells that lines all blood vessels. Sleep deprivation affects this layer and causes a serious, low-grade inflammatory response. This pathway is important in the development of atherosclerosis. Physiological stress brought on by sleep deprivation increases key inflammatory biomarkers, especially high-sensitivity C-reactive protein (hs-CRP), a good predictor of future cardiovascular events.[39] A landmark experimental study demonstrated that both acute total and chronic partial sleep deprivation cause a dose-dependent increase in hs-CRP, along with other pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-α).[1]The vascular endothelium is a primary target of these insults. The hallmark of endothelial dysfunction is impaired bioavailability of nitric oxide (NO), a critical molecule for vasodilation and vascular health. Sleep deprivation compromises this protective function, as confirmed by systematic reviews showing impaired endothelial function across various forms of sleep insufficiency.[43] Lack of sleep causes more reactive oxygen species (ROS) to be produced, which damage vascular cells, deactivate NO, and prolong inflammation. Oxidative stress is the link between these pathways. As a result, atherogenesis is accelerated in a vicious cycle where inflammation is fueled by inadequate sleep, which in turn causes sleep disturbances.

2.3 Metabolic and Endocrine Derangements

Sleep deprivation profoundly disrupts metabolic and endocrine homeostasis, creating a state conducive to type 2 diabetes and obesity, both major CVD risk factors. One of the most robust consequences is the rapid development of insulin resistance. Laboratory studies show that restricting sleep to 4–5 hours for even a few nights is sufficient to decrease insulins sensitivities and impair glucose tolerant in healthy elder adults.[26] This is driven by elevated levels of circulating free fatty acids and altered secretion of hormones like growth hormone and noradrenaline, which have anti-insulin effects.[32]

Sleep loss also disrupts the rhythm of key hormones. Cortisol, the primary stress hormone,

becomes elevated in the evening when it should be low, contributing to hypertension and insulin resistance.[12] Melatonin, a cardioprotective hormone, is suppressed, which may contribute to increased sympathetic activity.[19] Finally, lack of sleep causes the satiety hormone leptin to decrease and the hunger hormone ghrelin to increase, leading to an increase in appetite, a preference for foods high in energy, and ultimately weight gain.[27]

Section 3: Clinical Manifestations and the Role of Co-Morbid Sleep Disorders

An elevated incidence of overt cardiovascular disease is the ultimate manifestation of the pathophysiological disruptions brought on by sleep deprivation. Additionally, it is crucial to differentiate the risks associated with primary clinical sleep disorders, such as obstructive sleep apnea (OSA) and insomnia, which have distinct risk profiles, from the effects of behavioral sleep deprivation.

3.1 Sleep Deprivation and the Risk of Specific Cardiovascular Diseases

- **Hypertension:** The link between high blood pressure and sleep deprivation is one of the most often cited. Meta-analyses have shown that habitually sleeping fewer than six hours is associated with a 20% to 60% increased risk of incident hypertension, with middle-aged adults and women experiencing the most pronounced effect.[9]
- **Coronary Artery Disease (CAD) and Myocardial Infarction (MI):** A strong U-shaped relationship indicates that people who sleep less than six hours a night are about 48% more likely to have a CHD event.[10] Even after taking duration into consideration, poor sleep quality remains a risk factor in and of itself.[15] Mechanistically, a higher burden of subclinical atherosclerotic plaque is linked to shorter sleep duration.[9]
- **Stroke:** The evidence for stroke risk also follows a U-shaped pattern, though the data are more complex.[10] Some meta-analyses suggest long sleep may confer a greater relative risk for stroke than short sleep, and studies using Mendelian randomization have produced conflicting findings about causality.[10]
- **Heart Failure (HF):** The relationship is characterized by strong bidirectionality. Sleep disturbances, particularly sleep-disordered breathing, are strong risk factors for heart failure, and the clinical syndrome of heart failure itself is a major cause of sleep disruption.[26]

3.2 Distinguishing from Primary Sleep Disorders: A Critical Clarification

- **Insomnia:** Insomnia is a clinical disorder ordered by persistent difficulty with sleep *despite adequate opportunity*, accompanied by daytime impairment.[34]It is an risk factor of hyper tensioning, MI, and HF, with a risk increase of up to 45%.[70] The underlying mechanism arises from continuous psychophysiological hyperarousal over a 24-hour period, which differs from mere sleep restriction.[34]The phenotype of insomnia with objective short sleep duration carries the greatest risk.[34]
    - **Obstructive Sleep Apnea (OSA):** The respiratory condition known as OSA is typified by recurrent upper airway collapse while you sleep. It affects up to 80% patients are with resistant hypertension and a potent, independent risk factor for CVD.[76] Its unique pathophysiology involves intermittent hypoxia, large intrathoracic pressure swings, and frequent arousals, which lead to severe sympathetic activation, inflammation, and endothelial dysfunction.[17] OSA is strongly linked to resistant hypertension, atrial fibrillation, HF, and CAD.[65]

Section 4: Modulating Factors, Special Populations, and Future Directions

The relationship between sleep and cardiovascular diseases is not uniform. The risk is modulated by demographic characteristics, life stage, and co-existing lifestyle behaviors. Understanding this heterogeneity is crucial for identifying vulnerable populations and tailoring preventive strategies.

4.1 Heterogeneity of Risk: The Influence of Age, Gender, and Lifestyle

- **Gender and Sex Differences:** In addition to reporting lower-quality sleep, women may be more susceptible to the inflammatory and hypertensive consequences of sleep deprivation. 90 But risk profiles can vary depending on the endpoint; one meta-analysis found shorter sleep was more prominently linked to CHD risk in women, while longer sleep was more consequential for men.[93] Hormonal fluctuations during pregnancy and menopause represent periods of heightened vulnerability.[91]
- **Risk Across the Lifespan:** The impact of sleep deprivation varies with age. Adolescents face a "perfect storm" of biological shifts and societal pressures (e.g., early school start

times) leading to chronic sleep debt, which is linked to early cardiovascular risk factors.[96] Middle-aged adults often show the strongest associations between short sleep and incident CVD.[19] In the elderly, the relationship becomes more complex, possibly due to survivor effects and confounding from multiple comorbidities.[19]

- **The Unique Challenge of Shift Work:** Shift work serves as a real-world model of combined sleep deprivation and circadian disruption. It is a robust, independent risk factor for CVD, with an associated risk increase of up to 40%, linked to hypertension, type 2 diabetes, and accelerated atherosclerosis.[13]

4.2 Synthesis, Clinical Implications, and a Research Agenda for the Future

The cumulative evidence leads to an unequivocal conclusion: sleep is a biological necessity for cardiovascular health. The primary clinical implication is that sleep health must be integrated into routine cardiovascular risk assessment. A selection of the key studies discussed throughout this review is summarized in Table 4.

Patients with sleep disorders can benefit from evidence-based treatments like cognitive behavioral therapy for insomnia (CBT-I) and continuous positive airway pressure (CPAP) for OSA.[106] However, their ability to reduce hard cardiovascular endpoints has yielded mixed results in large clinical trials, often due to poor patient adherence, highlighting a major challenge.[108] There are still important knowledge gaps despite advancements. Compared to short sleep, the causality of the link between poor sleep quality and prolonged sleep is less clear. The separate effects of regularity and quality of sleep are not as well defined as duration.[2] Large-scale randomized controlled trials (RCTs) are desperately needed to find out if better sleep can stop or reverse CVD.[114]

Study (First Author, Year)	Design	Participants	Key Findings
Cappuccio et al. 2011 [10]	Meta-analysis of prospective studies	474,684 participants from 15 studies	Sleep durations on either end of the spectrum, short (5–6 hours or less) or long (8–9 hours or more), are linked to a significantly higher risk of developing or dying from coronary heart disease and stroke. This indicates a clear U-shaped relationship.
Meier-Ewert et al. (2004) [39]	Experimental study	20 healthy adults in two separate protocols	High-sensitivity C-reactive protein (hs-CRP) is an important marker of inflammation connected to cardiovascular risk. It increases significantly and in a dose-dependent way after both complete sleep loss (88 hours) and ongoing partial sleep restriction (4.2 hours per night over 10 days).
Vgontzas et al. (2009) [75]	Prospective cohort study (Penn State Cohort)	1,741 men and women	Compared to people who sleep normally, those who have insomnia and objectively measured short sleep duration (less than six hours via polysomnography) are much more likely to develop incident hypertension.
Huang et al. (2020) [122]	Prospective cohort study (Multi-Ethnic Study of Atherosclerosis)	1,992 participants	During a 5-year follow-up, greater irregularity in sleep timing (more than 90 minutes of variation) and in duration (more than 2 hours of variation over the week) was independently linked to a higher risk of future cardiovascular events.

McEvoy etal. (2016) [111]	Randomized Controlled Trial (SAVE trial)	The study included 2,717 participants with pre-existing cardiovascular disease and moderate to severe obstructive sleep apnea.	Continuous positive airway pressure (CPAP) treatment did not significantly reduce the rate of major adverse cardiovascular events compared to usual care, even though patient adherence to the therapy was a recognized limitation.

Table 1.1: Summary of Select Studies Examining the Association Between Sleep and Cardiovascular Disease and its Risk Factors

A future research agenda should prioritize:

1. Moving beyond duration to multidimensional sleep health, incorporating objective measures like actigraphy and emerging diagnostic tools like advanced wearable sensors and biomarkers.[123]
2. Conducting rigorous intervention trials to test whether improving sleep reduces major adverse cardiovascular events (MACE).[115]
3. Deepening mechanistic research into novel molecular pathways, including epigenetics, the gut microbiome, and intercellular signaling.[17]
4. Paying attention to health inequalities among vulnerable groups, such as racial/ethnic minorities and particular age and gender cohorts.[91]
5. Informing public health policy with actionable recommendations, such as advocating for later school start times and healthier shift work schedules.[97]

In conclusion, the scientific journey has firmly established sleep as a cornerstone of cardiovascular health. The task ahead is to refine this understanding, prove the efficacy of interventions, and translate this critical knowledge into clinical practice and public policy.

7. CONCLUSION:

The information in this review indicates that sleep plays a crucial role in affecting cardiovascular health. The length of sleep is merely one aspect of the complex relationship between sleep and heart disease. Additional factors encompass the quality of sleep, its regularity, and the presence of concurrent sleep disorders. Research has indicated that insufficient sleep can negatively impact inflammation, vascular function, metabolic regulation, and autonomic control, yet population studies have consistently demonstrated a U-shaped relationship.

In clinical environments, insufficient sleep greatly elevates the likelihood of heart failure, stroke, coronary artery disease, and hypertension. These results highlight the importance of prioritizing sleep health in preventive cardiology and public health initiatives. Dealing with insufficient sleep should be an ongoing aspect of evaluating and controlling cardiovascular risk.

8. STUDY LIMITATIONS

There are a number of restrictions on this narrative review. First, there may be selection bias in the included studies due to the review design's lack of a systematic methodology. Second, even though observational studies provide compelling evidence, their inherent limitations—such as confounding, reverse causality, and self-reported sleep measures—make it difficult to draw conclusions about causality, particularly when considering extended periods of sleep. Third, population differences across studies in terms of age, sex, socioeconomic status, and comorbid conditions may limit the findings' ability to be applied broadly. Furthermore, only a brief discussion was given to a few significant new topics, including the contribution of socioeconomic disparities, epigenetic modifications, and circadian misalignment to sleep-related cardiovascular risk.

9. RECOMMENDATIONS FOR IMPROVEMENT

This narrative review has several limitations.

1. First, review design does not follow a systematic methodology and therefore may be subject to selection bias in the studies included.
2. Second, while the evidence from observational studies is strong, the inherent limitations of these studies, like confounding, reverse causality, and self-reported

sleep measures, limit the ability to infer causality. This is particularly true in the context of long sleep duration.

3. Third, the generalizability of findings may be restricted due to population differences across studies in terms of age, sex, socioeconomic status, and comorbid conditions. Additionally, some important emerging areas such as the role of circadian misalignment, epigenetic changes, and socioeconomic disparities in sleep-related cardiovascular risk were only briefly discussed.

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Apurva Ahuja D report

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