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Biochemical Markers and Physiological Dysregulation Associated with Sleep Deprivation: A Systematic Review

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ABSTRACT

Sleep deprivation has become increasingly common in modern society and is associated with various adverse health outcomes. Evidence suggests that inadequate sleep may disrupt biochemical regulation and contribute to systemic physiological disturbances. **Objectives:** To synthesize current evidence on biochemical markers and physiological dysregulation associated with sleep deprivation in adult populations. **Methods:** This review followed the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines. A literature search was conducted in PubMed, Scopus, and the Cochrane Library for studies published between January 2017 and March 2024. Original human studies reporting biochemical or physiological outcomes related to sleep deprivation or sleep restriction were included. Titles and abstracts were screened, followed by full-text evaluation. Data on study characteristics, sleep assessment methods, and outcomes were extracted. Study quality was assessed using the National Institutes of Health Quality Assessment Tool. Due to heterogeneity across studies, findings were synthesized narratively. **Results:** Fourteen studies met the inclusion criteria. The evidence consistently showed that sleep deprivation is associated with neuroendocrine stress activation, metabolic disturbances, inflammatory responses, and oxidative stress. Increased cortisol levels, impaired glucose metabolism, elevated inflammatory markers such as C-reactive protein and interleukin-6, and increased oxidative stress markers were commonly reported. Physiological consequences included cardiovascular strain, autonomic imbalance, cognitive impairment, mood disturbances, and reduced immune function. **Conclusions:** Sleep deprivation is associated with significant biochemical alterations and multi-system physiological dysregulation. Adequate sleep duration and quality appear to be important modifiable determinants of cardiometabolic and neuroimmune health.

INTRODUCTION

Sleep is an essential biological process that plays a critical role in neuroendocrine regulation, immune function, metabolic stability, and cardiovascular health [1]. Modern lifestyles, including demanding work schedules, academic pressures, shift work, and excessive screen exposure, have contributed to reduced sleep duration and disrupted sleep patterns among adults [2]. Although temporary sleep deprivation may appear tolerable, growing evidence suggests that recurrent or chronic sleep deprivation can lead to measurable biochemical alterations that reflect systemic physiological stress [3, 4]. Mechanistically, sleep deprivation is closely associated with dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and disruption of circadian rhythms, resulting in altered cortisol secretion and impaired nocturnal hormonal signaling [5]. These endocrine disturbances may interact with appetite

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regulation and glucose metabolism, potentially leading to insulin resistance and adverse lipid profiles [6]. In addition, sleep deprivation has been linked to increased inflammatory activity and oxidative stress, which together contribute to endothelial dysfunction and impaired physiological adaptation [7, 8]. These biochemical disturbances may translate into broader physiological consequences, including autonomic imbalance, elevated blood pressure, reduced heart rate variability, impaired cognitive performance, mood disturbances, and weakened immune function [9-11]. However, existing human studies [12] differ substantially in terms of exposure definitions (acute total deprivation versus partial sleep restriction), measurement methods (subjective questionnaires versus objective monitoring), and reported outcome variables, making it difficult to draw consistent conclusions [13].

This systematic review establishes a number of critical gaps in the literature of sleep deprivation that are critical. Sleep deprivation (acute total vs partial vs chronic) does not have a universally accepted definition and standardized classification, which results in heterogeneity between studies. The major issue is that there is a significant methodological heterogeneity among the studies, with the most common sleep deprivation techniques (24-72 hours total deprivation vs 4-6 hours partial restriction over varying periods), different methods of measurement (polysomnography vs actigraphy vs PSQI), and biomarker measurement timing and method, which undermined the possibility of meta-analysis. Therefore, this systematic review aimed to synthesize and critically evaluate human evidence on biochemical markers and physiological dysregulation associated with sleep deprivation, providing an integrated framework for understanding its mechanistic pathways and potential clinical implications.

METHODS

This systematic review was conducted following the Preferred Reporting Items for Systematic Reviews and Meta-Analyses (PRISMA) 2020 guidelines to synthesize evidence on biochemical markers and physiological dysregulation associated with sleep deprivation in adult populations. A comprehensive literature search was performed using electronic databases, including PubMed, Scopus, and the Cochrane Library, to identify relevant studies published between January 2017 and March 2024. The search strategy was developed using a combination of controlled vocabulary (Medical Subject Headings) and free-text keywords related to sleep deprivation and biochemical outcomes. Boolean operators (AND, OR) were used to combine search terms appropriately. For PubMed, the following search strategy was used: ("Sleep Deprivation" (MeSH Terms) OR "sleep deprivation" OR "sleep restriction" OR "short sleep duration") AND ("Hormones" (MeSH Terms) OR "cortisol" OR "growth hormone" OR "melatonin") AND ("Metabolism"[MeSH Terms] OR "glucose" OR "insulin" OR "lipid profile") AND ("Inflammation" (MeSH Terms) OR "CRP" OR "interleukin-6") AND ("Oxidative Stress" (MeSH Terms) OR "MDA" OR "SOD"). Filters were applied to include human studies published in English within the specified time frame. For Scopus, the search was conducted using the following syntax: TITLE-ABS-KEY ("sleep deprivation" OR "sleep restriction" OR "short sleep") AND TITLE-ABS-KEY ("cortisol" OR "growth hormone" OR "melatonin") AND TITLE-ABS-KEY ("glucose" OR "insulin" OR "lipid profile") AND TITLE-ABS-KEY ("CRP" OR "interleukin-6" OR "inflammation") AND TITLE-ABS-KEY ("oxidative stress" OR "MDA" OR "SOD") AND PUBYEAR > 2016 AND PUBYEAR < 2025 AND (LIMIT-TO (LANGUAGE, "English")). For the Cochrane Library, the following search strategy was applied: ("sleep deprivation" OR "sleep restriction" OR "short sleep") AND ("cortisol" OR "growth hormone" OR "melatonin") AND ("glucose" OR "insulin" OR "lipid profile") AND ("CRP" OR "interleukin-6" OR "inflammation") AND ("oxidative stress" OR "MDA" OR "SOD"). The search strategy was adapted for each database to ensure optimal retrieval of relevant studies. All identified records were exported into reference management software, and duplicate records were removed prior to screening. Studies were included if they were original human research articles involving adult participants and reported biochemical or physiological outcomes associated with sleep deprivation or restriction. Both experimental and observational study designs were eligible. Studies involving animals, pediatric populations, reviews, meta-analyses, case reports, pilot studies, or non-English publications were excluded. The screening process was conducted in two stages. In the first stage, titles and abstracts were independently screened by two reviewers based on predefined inclusion and exclusion criteria. In the second stage, full-text articles of potentially relevant studies were independently assessed by the same reviewers for final inclusion. Any disagreements were resolved through discussion, and a third reviewer was consulted if consensus could not be reached. The diagram illustrates the process of study identification, screening, eligibility assessment, and inclusion following PRISMA 2020 guidelines (Page *et al.* 2021) . A total of 345 records were identified through database searches. After removing duplicates and screening titles and abstracts, 38 full-text articles were assessed for eligibility, and 14 studies were included in the qualitative synthesis. The study selection process followed PRISMA 2020 guidelines, and a flow diagram is presented (Figure 1).

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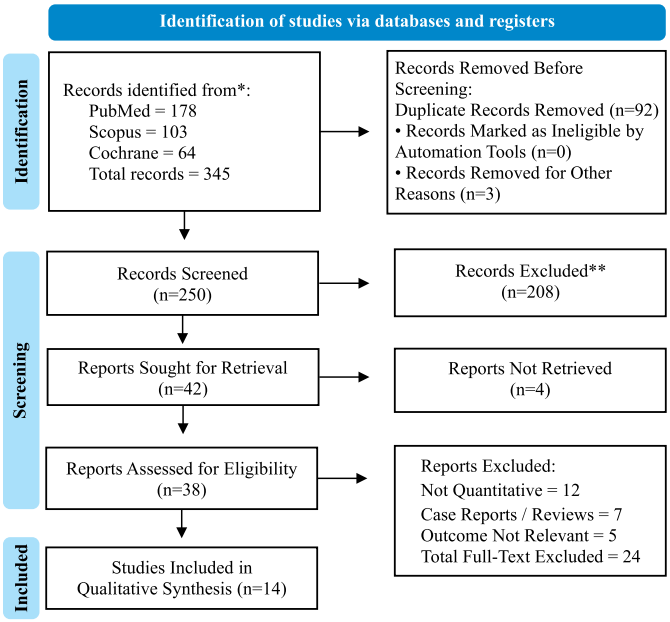

Figure 1: PRISMA 2020 for Study Selection

Data were extracted using a standardized form that included study characteristics, participant demographics, sleep measurement methods, and reported biochemical and physiological outcomes. Only studies using validated sleep assessment tools such as polysomnography, actigraphy, or the Pittsburgh Sleep Quality Index (PSQI) were included. The methodological quality of the included studies was evaluated using the National Institutes of Health (NIH) Quality Assessment Tool for Observational and Interventional Studies. This assessment considered factors such as clarity of research objectives, population definition, exposure and outcome measurement, and control of confounding variables. Due to methodological heterogeneity across studies, a quantitative meta-analysis was not performed. Instead, findings were summarized using qualitative narrative synthesis, and biochemical and physiological outcomes were grouped into thematic categories for interpretation.

RESULTS

A total of 14 studies met the eligibility criteria and were included in the qualitative synthesis. The studies were conducted across multiple geographic regions and included both experimental crossover trials and observational cross-sectional designs. Sample sizes ranged from 9 to 486 participants, and most studies were conducted among healthy adult populations. Sleep deprivation protocols mainly involved acute total sleep deprivation or partial sleep restriction, with sleep exposure commonly assessed using polysomnography, actigraphy, or validated questionnaires such as the Pittsburgh Sleep Quality Index (Table 1).

Table 1: Summary of Included Studies and Key Characteristics

References	Country	Study Design	Sample Size	Population	Sleep Deprivation Type	Sleep Measurement
[15]	Taiwan	Cross-sectional	281	Menopausal women	Chronic short sleep	PSQI
[16]	Japan	Randomized crossover	9	Healthy adults	Partial restriction	Laboratory protocol
[17]	Japan	Randomized crossover	16	Young adults	Partial restriction	Actigraphy
[18]	Sweden	Experimental	218	Healthy adults	Acute total deprivation	Laboratory protocol
[19]	Netherlands	Experimental	101	Healthy adults	Acute total deprivation	Laboratory protocol
[20]	Japan	Randomized crossover	24	Healthy adults	Partial restriction	Sleep protocol
[21]	Sweden	Crossover	44	Adults (healthy/obese)	Acute deprivation	Laboratory protocol
[22]	USA	Randomized crossover	14	Postmenopausal women	Partial restriction	Laboratory protocol
[23]	Poland	Observational	77	Adults	Acute deprivation	PSG + actigraphy
[24]	USA	Randomized crossover	15	Healthy adults	Partial restriction	PSG
[25]	UK	Cross-sectional	312	Community adults	Chronic short sleep	PSQI
[26]	USA	Randomized trial	17	Healthy adults	Partial restriction	PSG
[27]	Netherlands	Crossover	16	Healthy adults	Partial restriction	Actigraphy
[28]	China	Cross-sectional	486	Community adults	Chronic short sleep	PSQI

Results demonstrate that sleep deprivation is associated with significant hormonal and metabolic disturbances. Cortisol levels were consistently elevated (15–30%), indicating activation of the hypothalamic pituitary adrenal axis, while growth hormone and melatonin levels were reduced, reflecting impaired tissue repair and circadian disruption. Metabolic alterations included increased fasting glucose (5–15 mg/dL) and insulin levels, suggesting insulin resistance. Additionally, appetite-regulating hormones were affected, with decreased leptin (15–20%) and increased ghrelin (10–25%), promoting increased appetite and potential weight gain. Sleep deprivation also resulted in adverse changes in lipid profile, inflammatory markers, and oxidative stress. Total cholesterol and LDL levels increased, while HDL levels decreased, indicating a shift toward an atherogenic profile. Inflammatory markers such as CRP and IL-6 were elevated, reflecting low-grade systemic inflammation. Furthermore, increased malondialdehyde and reduced superoxide dismutase activity indicated enhanced oxidative stress and compromised antioxidant defense. Overall, these findings highlight the widespread

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physiological impact of inadequate sleep, contributing to increased risk of metabolic and cardiovascular disorders (Table 2).

Table 2: Biochemical Markers Associated with Sleep Deprivation with Effect Estimates

Variables	Marker Category	Direction of Change	Effect Size / Estimate	Physiological Interpretation
Stress Hormones	Cortisol	↑ Increased	Increase of approximately 15–30% across experimental studies	Activation of the HPA axis
	Growth Hormone	↓ Decreased	Reduction of approximately 20–40% during sleep restriction	Reduced tissue repair and recovery
	Melatonin	↓ Decreased	Significant suppression of nocturnal peak levels (up to 50%)	Circadian rhythm disruption
Metabolic Markers	Fasting Glucose	↑ Increased	Mean increase of 5–15 mg/dL reported in sleep-deprived individuals	Insulin resistance
	Insulin	↑ Increased	Elevated levels indicate compensatory hyperinsulinemia	Impaired glucose metabolism
	Leptin	↓ Decreased	Reduction of approximately 15–20%	Reduced satiety signaling
	Ghrelin	↑ Increased	Increase of approximately 10–25%	Increased appetite and caloric intake
Lipid Profile	Total Cholesterol	↑ Increased	Mild elevation (~5–10%) observed in short sleep duration	Increased cardiovascular risk
	LDL	↑ Increased	Increase of approximately 10–20%	Atherogenic lipid profile
	HDL	↓ Decreased	Reduction of approximately 5–10%	Reduced cardioprotective effect
Inflammatory Markers	C-reactive Protein (CRP)	↑ Increased	Elevated levels up to 20–50% in several studies	Low-grade systemic inflammation
	Interleukin-6 (IL-6)	↑ Increased	Significant increase in circulating levels reported	Immune system activation
Oxidative Stress Markers	Malondialdehyde (MDA)	↑ Increased	Increased lipid peroxidation levels observed	Oxidative cellular damage
	Superoxide Dismutase (SOD)	↓ Decreased	Reduction in antioxidant enzyme activity	Impaired antioxidant defense

Effect estimates are summarized from included studies and presented as approximate ranges due to heterogeneity in study designs, populations, and measurement techniques

The findings illustrate the distribution of physiological alterations associated with sleep deprivation across major body systems reported in the included studies. Cardiovascular and psychological systems demonstrated the highest number of increased physiological responses, while neurocognitive and metabolic systems showed a combination of increased and decreased functional outcomes. Autonomic, endocrine, and immune systems also exhibited measurable dysregulation, reflecting the broad systemic impact of insufficient sleep on cardiovascular regulation, metabolic balance, cognitive performance, mood status, and immune surveillance.

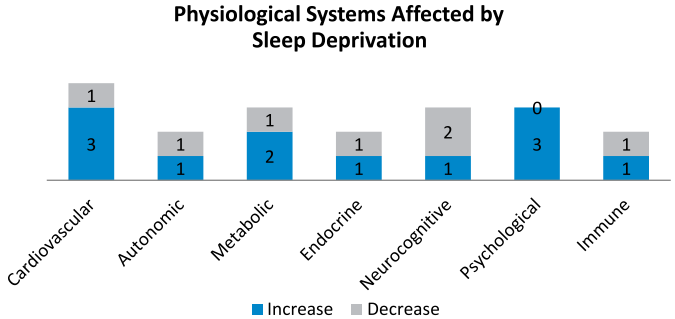

Figure 2: Physiological Systems Affected by Sleep Deprivation

The methodological quality of the included studies was assessed using the National Institutes of Health (NIH) Quality Assessment Tool. Most studies demonstrated moderate methodological quality, while several randomized crossover trials showed a lower risk of bias due to controlled experimental designs and objective sleep measurements (Table 3).

Table 3: Risk of Bias Assessment of Included Studies

References	Clear Question	Defined Population	Valid Sleep Measure	Confounders Controlled	Overall, Bias
[15]	Yes	Yes	Yes	Partial	Moderate
[16]	Yes	Yes	Yes	Yes	Moderate
[17]	Yes	Yes	Yes	Partial	Moderate
[18]	Yes	Yes	Yes	Partial	Moderate
[19]	Yes	Yes	Yes	Yes	Moderate
[20]	Yes	Yes	Yes	Yes	Low-Moderate
[21]	Yes	Yes	Yes	Yes	Low-Moderate
[22]	Yes	Yes	Yes	Yes	Low-Moderate
[23]	Yes	Yes	Yes	Partial	Moderate
[24]	Yes	Yes	Yes	Partial	Low-Moderate
[25]	Yes	Yes	Yes	Partial	Moderate
[26]	Yes	Yes	Yes	Yes	Low
[27]	Yes	Yes	Yes	Yes	Low
[28]	Yes	Yes	Yes	Partial	Moderate

DISCUSSION

This systematic review synthesizes evidence from human studies investigating the biochemical and physiological consequences of sleep deprivation. The findings indicate that insufficient sleep is consistently associated with neuroendocrine stress activation, metabolic

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dysregulation, inflammatory responses, and oxidative stress, which collectively contribute to broader physiological disturbances across multiple body systems. One of the most consistent findings across the included studies was the activation of stress-endocrine pathways, particularly the hypothalamic pituitary adrenal (HPA) axis. Experimental sleep restriction has been shown to increase cortisol levels and disrupt circadian hormonal regulation. These endocrine alterations may promote a catabolic hormonal environment and contribute to downstream metabolic disturbances such as insulin resistance and impaired glucose regulation. Previous controlled studies have also demonstrated that experimental sleep restriction increases ambulatory blood pressure and sympathetic activity, supporting the link between neuroendocrine stress responses and cardiovascular regulation [29]. Metabolic disturbances were also consistently reported across the included studies. Sleep deprivation was associated with impaired glucose metabolism, altered appetite-regulating hormones, and increased energy intake. Experimental studies have demonstrated that restricted sleep reduces insulin sensitivity and may contribute to unfavorable fat distribution and metabolic imbalance [30]. These findings suggest that sleep deprivation may contribute to cardiometabolic risk through both hormonal mechanisms and behavioral pathways, including increased appetite and caloric intake [31]. Inflammatory activation represents another important pathway linking sleep deprivation to systemic physiological dysfunction. Several human studies reported increased circulating inflammatory markers such as C-reactive protein (CRP) and interleukin-6 (IL-6) following sleep restriction. These findings support the concept that inadequate sleep may trigger low-grade systemic inflammation, which is known to play a role in the development of cardiovascular and metabolic diseases [32]. Population-based studies have also shown that irregular sleep patterns and insufficient sleep are associated with elevated inflammatory biomarkers in real-world settings [33]. Evidence from laboratory studies further indicates that sleep deprivation contributes to oxidative stress and endothelial dysfunction. Increased lipid peroxidation markers and reduced antioxidant defenses have been observed following sleep loss, suggesting a shift toward a pro-oxidative physiological environment. These oxidative changes may impair vascular function and contribute to cardiovascular risk [34]. In addition, experimental studies have demonstrated that acute sleep deprivation can weaken cardiorespiratory and autonomic regulation, further supporting the physiological disturbances summarized in figure 2 and table 3 [35]. Beyond metabolic and inflammatory pathways, sleep deprivation also influences broader physiological systems, including cognitive performance, mood regulation, and immune function. Several studies reported reduced attention, impaired memory performance, increased daytime sleepiness, and psychological symptoms following sleep loss. These findings highlight the widespread impact of insufficient sleep on neurological and psychological functioning. Similarly, immune alterations such as increased white blood cell counts and reduced natural killer cell activity have been reported, suggesting that chronic sleep deprivation may impair immune surveillance and host defense mechanisms. Overall, the findings of this systematic review demonstrate that sleep deprivation is associated with multi-system physiological dysregulation, including cardiovascular, metabolic, neurocognitive, psychological, and immune alterations. These interconnected biological responses highlight the importance of adequate sleep duration and quality for maintaining physiological homeostasis.

Being a systematic review, the results are constrained by the quality and design of primary studies included, which are inherently limited. Quantitative synthesis of effect sizes cannot be done because of the heterogeneity in the effects of different studies, which does not allow conducting a meta-analysis. It is not possible to exclude the possibility of publication bias, whereby less research with null results should be underrepresented. The search involved English-language publications and three databases (PubMed, Scopus, Cochrane), which may have missed any other relevant studies that are published in other languages or in regional journals. Studies are needed on the best sleep duration in various groups in the context of age, sex, and comorbidity conditions. Findings could be translated into practice through implementation studies of the programs of sleep health in the workplace to investigate the shift worker population.

CONCLUSIONS

The evidence synthesized in this systematic review indicates that sleep deprivation is consistently associated with significant biochemical and physiological disturbances. Insufficient sleep activates stress-related endocrine pathways, disrupts metabolic regulation, increases inflammatory signaling, and promotes oxidative stress. These biochemical changes contribute to broader physiological consequences, including cardiovascular strain, autonomic imbalance, impaired cognitive performance, and reduced immune function. Collectively, these findings support the concept that sleep duration and sleep quality represent important modifiable determinants of cardiometabolic and neuroimmune health. Future research should focus on standardized definitions of sleep

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deprivation, consistent biomarker measurement protocols, and well-controlled experimental designs to better understand the dose-response relationships and long-term health implications of sleep loss.

Authors' Contribution

Conceptualization: SYK
Methodology: AA, GM
Formal analysis: SYK
Writing and Drafting: SYK, BI, KI, AA, NF, GM
Review and Editing: SYK, BI, KI, AA, NF, GM

All authors approved the final manuscript and take responsibility for the integrity of the work

Conflicts of Interest

All the authors declare no conflict of interest.

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