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CORTISOL AS A MEDIATOR OF METABOLIC DISTURBANCES AND SLEEP QUALITY IN HEALTHY ADULTS – A REVIEW OF CURRENT EVIDENCE

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ABSTRACT

Background: Contemporary lifestyles, characterized by chronic stress and disruptions of circadian rhythms, significantly affect the functioning of the hypothalamic–pituitary–adrenal (HPA) axis. Cortisol, the primary effector hormone of this axis, exhibits a well-defined circadian pattern that is closely linked to the sleep–wake cycle. Under physiological conditions, cortisol levels decline during the evening hours; however, a range of behavioral and environmental factors may contribute to nocturnal hypercortisolemia. The impact of elevated nighttime cortisol on both subjective and objective sleep quality, as well as on metabolic health in otherwise healthy adult individuals, remains an area of active and ongoing investigation
Objective: The aim of this literature review is to analyze the relationship between cortisol concentrations and sleep quality and architecture in healthy adult individuals, as well as to evaluate the health consequences associated with increased nocturnal activity of the hypothalamic–pituitary–adrenal (HPA) axis.
Methods: The literature review was conducted using the PubMed database and applied strict inclusion criteria: studies involving human participants, adult populations (≥19 years), individuals considered to be in good general health, and the absence of pharmacological interventions known to affect sleep architecture. A total of ten selected publications—including experimental studies, cohort studies, and systematic reviews—were analyzed. These studies directly examined the association between cortisol biomarkers (plasma, salivary, and hair cortisol) and sleep parameters assessed using polysomnography, actigraphy, and the Pittsburgh Sleep Quality Index (PSQI).
Results: The analysis of the literature indicates the presence of a "sleep quality paradox," whereby factors such as late meal timing and everyday psychosocial stress increase nocturnal cortisol levels, which correlates with a subjective perception of non-restorative sleep, as assessed by the Pittsburgh Sleep Quality Index (PSQI), despite the absence of marked alterations in objective sleep architecture measured by polysomnography. Elevated evening and nighttime cortisol levels were shown to promote sleep fragmentation and to induce insulin resistance as well as a pro-inflammatory immune response, including increased interleukin-6 (IL-6) levels. A bidirectional relationship was also identified, whereby sleep deprivation enhances hypothalamic–pituitary–adrenal (HPA) axis reactivity on the following day, thereby creating a self-perpetuating vicious cycle.
Conclusions: A normal circadian cortisol profile is essential for the metabolic and immunological functions of sleep. Nocturnal hypercortisolemia in healthy adults, even if it does not markedly reduce total sleep duration, leads to significant impairments in glucose metabolism and an increase in inflammatory responses, which over the long term may elevate the risk of obesity and metabolic diseases.

KEYWORDS

Cortisol; Sleep Quality; Stress; Metabolism; Healthy Adults; Nocturnal Hypercortisolemia

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Introduction

Physiological basis of the HPA axis–sleep relationship

Sleep is a complex physiological process regulated by two main mechanisms: the homeostatic sleep drive and the circadian rhythm. One of the key hormonal markers of the latter is cortisol—a glucocorticoid secreted by the adrenal cortex in response to hypothalamic–pituitary–adrenal (HPA) axis activity. Under physiological conditions, cortisol secretion exhibits a distinct circadian pattern: it reaches its nadir around midnight and then rises sharply during the second half of the night, peaking shortly after awakening, a phenomenon known as the Cortisol Awakening Response (CAR) (Hirotsu et al., 2015). Proper suppression of HPA axis activity during the early phase of the night is essential for the onset of deep (NREM) sleep, which is critical for tissue regeneration and memory consolidation.

Bidirectional Mechanism

The relationship between cortisol and sleep is bidirectional and recursive. On one hand, psychophysiological stressors activate the HPA axis, leading to a state of hyperarousal that impairs sleep initiation and maintenance (Elder et al., 2016). On the other hand, even short-term sleep deprivation or poor sleep quality acts as a potent biological stressor, triggering cortisol release at times when its levels would normally remain low (Dahlgren et al., 2017). Such hormonal dysregulation may result in the development of glucocorticoid resistance, wherein chronically elevated stress hormone levels no longer exert their anti-inflammatory effects and instead promote inflammation and chronic fatigue (Lopresti, 2017).

Importance of Research in Healthy Adults

Most previous studies on HPA axis dysfunction have focused on clinical populations, such as individuals with insomnia or depression. However, a growing body of evidence suggests that in healthy adults—free from chronic diseases and not receiving pharmacological treatments—subtle alterations in nocturnal cortisol levels, triggered by factors such as meal timing or everyday psychosocial stress, may have significant implications for long-term health (Gu et al., 2020). Understanding these mechanisms in healthy individuals is crucial for the prevention of metabolic and lifestyle-related diseases.

Objective and Hypothesis

The aim of this review is to systematize the current knowledge regarding the impact of nocturnal cortisol on sleep quality in healthy adults. The review posits the hypothesis that elevated nocturnal cortisol levels constitute a significant risk factor for metabolic and immunological disturbances, even when objective sleep duration parameters remain within normal ranges.

Methods

Search Strategy

To gather relevant research material, a systematic literature review was conducted using the PubMed database (National Library of Medicine). The search and selection process was carried out from September 2025 to January 2026. The search strategy was based on a combination of keywords and Boolean operators: (cortisol OR hydrocortisone) AND (sleep quality OR sleep architecture OR insomnia) AND (healthy adults). Additionally, the reference lists of selected articles were examined to identify supplementary sources (manual search).

Inclusion and Exclusion Criteria: To ensure high consistency of results and minimize confounding factors, strict criteria were applied for the selection of publications.

Inclusion criteria:

- Timeframe: Articles published between 2015 and 2025 were included to capture the most recent scientific evidence while maintaining an appropriate theoretical background.
- Studies conducted exclusively on human participants.
- Adult populations (age ≥19 years).
- Participants in good general health (no diagnosed chronic diseases, psychiatric disorders, or metabolic conditions).
- Articles published in English in peer-reviewed scientific journals.
- Use of objective (e.g., PSG, actigraphy, biomarker analysis) or validated subjective (e.g., PSQI) measurement methods.

Exclusion criteria:

- Animal or in vitro studies.
- Studies including children, adolescents, or older adults with comorbidities.
- Use of any pharmacological interventions affecting sleep (e.g., hypnotics, melatonin, sedatives, steroids).
- Case reports and conference abstracts.

Data Analysis and Extraction

From the ten selected articles, key data were extracted, including participant characteristics, the method of cortisol measurement (plasma, saliva, urine, or hair), the method used to assess sleep parameters, and the main findings regarding hormonal–metabolic associations. The selected studies represent a variety of research approaches, ranging from tightly controlled laboratory experiments (e.g., meal timing control) to observational studies conducted under naturalistic conditions.

Results

1. Overview of the Reviewed Literature

A total of ten publications meeting the inclusion criteria were analyzed in this review. The following table presents a synthesis of the data extracted from the selected studies.

Table 1. Characteristics of studies included in the literature review.

Author & Year	Cortisol Measurement	Sleep Assessment	Key Observations
Gu et al. (2020)	Plasma (night profile)	PSG (Polysomnography)	Late dinner increases nocturnal cortisol without altering sleep architecture.
Hirotsu et al. (2015)	Review (various)	Various	Sleep deprivation activates the HPA axis, impairing entry into deep sleep.
Bush et al. (2016)	Saliva (CAR)	Actigraphy	Poor sleep efficiency enhances the Cortisol Awakening Response (CAR).
Vargas et al. (2020)	Saliva (evening)	Questionnaires	Elevated evening cortisol correlates with higher IL-6 levels and difficulty falling asleep.
Zhao et al. (2019)	Serum (morning)	PSQI	Significant correlation between poor sleep quality and elevated morning cortisol.
Kim et al. (2022)	Plasma	PSG / Metabolic assessments	Short sleep duration increases cortisol, inducing insulin resistance in healthy adults.
Michaud & Guay (2020)	Hair (HCC)	PSQI / Actigraphy	Chronic cortisol correlates with subjective (PSQI) but not objective sleep measures.
Lopresti (2017)	Review (HPA–Immune)	Theoretical analysis	Sleep loss → glucocorticoid resistance and chronic inflammation.
Elder et al. (2016)	Saliva (evening)	PSG	Higher evening cortisol is associated with increased wake after sleep onset (WASO).
Dahlgren et al. (2017)	Saliva (24-hour profile)	Diaries / Actigraphy	Daytime stress and short sleep elevate evening cortisol the following day.

2. Narrative Analysis of Results

2.1. Relationship Between Cortisol and Sleep Architecture (Objective vs. Subjective):

Analysis of the collected data reveals an interesting discrepancy between objective sleep parameters and participants' subjective perceptions. Gu et al. (2020) demonstrated that in healthy adults, nocturnal cortisol elevation (e.g., induced by a late dinner) did not significantly alter the structure of sleep stages (NREM/REM) measured via polysomnography. These findings are consistent with Michaud and Guay (2020), in which chronic hair cortisol concentrations showed no association with objectively measured movement via actigraphy, despite a strong correlation with negative sleep perception assessed using the PSQI. This suggests that the subjective feeling of non-restorative sleep may result from "nocturnal hormonal stress," even when sleep duration remains physiologically normal.

2.2. Influence of Time of Day and Behavioral Factors on HPA Axis Activity:

A key finding is the impact of evening cortisol levels on sleep continuity. Elder et al. (2016) and Vargas et al. (2020) reported that elevated cortisol concentrations before bedtime impair sleep initiation and increase the number of nighttime awakenings (WASO). This mechanism is further exacerbated by daytime stress and prior sleep deprivation, which Dahlgren et al. (2017) describe as a cumulative HPA axis load effect.

2.3. Metabolic and Immunological Consequences:

Elevated nocturnal cortisol in healthy adults leads to significant disturbances in carbohydrate metabolism. Kim et al. (2022) demonstrated that cortisol mediates the development of sleep deprivation–induced insulin resistance. Furthermore, findings from Vargas et al. (2020) and Lopresti (2017) indicate a correlation between HPA axis dysfunction and increased inflammatory markers, such as interleukin-6 (IL-6). These results suggest that nocturnal hypercortisolemia promotes a chronic low-grade inflammatory state, providing a physiological basis for the development of future lifestyle-related diseases.

Discussion

1. Interpretation of the Sleep Quality Paradox

A central observation of this review is that in healthy adults, elevated cortisol levels do not always manifest as drastic changes in sleep architecture measured via polysomnography (PSG), as demonstrated by Gu et al. (2020) and Michaud and Guay (2020). The fact that the brain can maintain proper proportions of sleep stages despite metabolic and hormonal stress suggests the presence of robust compensatory mechanisms. However, the subjective perception of poor sleep quality (PSQI) in these individuals indicates that cortisol may affect subtle parameters of sleep microstructure (e.g., specific brain wave patterns) that are not always captured by standard assessments.

2. The "Vicious Cycle" Mechanism and the Role of Daily Stress

The analysis highlights the inseparable link between recovery and HPA axis reactivity. Dahlgren et al. (2017) provide new insights into how a single night of sleep deprivation "programs" the body for heightened stress the following day. This creates a dangerous vicious cycle: fatigue leads to elevated evening cortisol, which in turn impairs the body's ability to downregulate before the next night, as suggested by Elder et al. (2016).

3. Implications for Metabolic Health and Preventive Strategies

The most concerning finding from the analyses by Kim et al. (2022) and Vargas et al. (2020) is that nocturnal hypercortisolemia in healthy adults serves as a direct bridge to insulin resistance and inflammatory states. This suggests that behaviors such as very late meals or poor evening stress management may represent key risk factors for the development of metabolic syndrome, even in individuals who report "sleeping" 7–8 hours per night.

4. Limitations and Directions for Future Research

Despite the application of strict inclusion criteria, this review has certain limitations. The heterogeneity of cortisol measurement methods (saliva vs. hair vs. serum) complicates direct comparisons of absolute concentrations. Future studies should focus on the long-term effects of subclinical HPA axis disturbances in young, healthy adults to more precisely identify the critical threshold at which nocturnal hormonal stress becomes an irreversible clinical concern.

Conclusions

- In healthy adults, HPA axis activity is strongly correlated with the subjective perception of sleep quality, even in cases where objective measures of sleep architecture (PSG) remain within physiological norms.
- Nocturnal hypercortisolemia, triggered by behavioral factors (e.g., late-night meals) or daytime stress, leads to significant metabolic disturbances, including the induction of insulin resistance and an increase in systemic inflammatory markers (e.g., IL-6).
- There exists a bidirectional relationship between sleep and cortisol, in which sleep deprivation enhances HPA axis reactivity the following day, thereby impairing recovery in subsequent circadian cycles (vicious cycle mechanism).

Optimizing sleep hygiene and reducing evening HPA axis–activating factors should be considered key strategies for the prevention of lifestyle-related diseases in healthy adults.

List of Abbreviations

- **ACTH** – adrenocorticotropic hormone
- **APA** – American Psychological Association
- **CAR** – cortisol awakening response
- **DOI** – digital object identifier
- **HCC** – hair cortisol concentration
- **HPA** – hypothalamic-pituitary-adrenal axis
- **IL-6** – interleukin-6 (proinflammatory cytokine)
- **NREM** – non-rapid eye movement sleep (deep sleep)
- **PSG** – polysomnography
- **PSQI** – Pittsburgh Sleep Quality Index
- **REM** – rapid eye movement sleep
- **WASO** – wake after sleep onset

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