

Impact of chronic and pathological stress on sleep architecture

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Abstract. Chronic and pathological stress significantly disrupt sleep architecture, leading to profound consequences for mental and physical health. This review synthesizes current literature to examine the neurobiological mechanisms linking stress to sleep disturbances, focusing on dysregulation of the HPA axis, hyperarousal mediated by the amygdala and prefrontal cortex, and disruptions in REM and NREM sleep stages. Chronic stress alters cortisol rhythms, impairs sleep continuity, and increases the risk of insomnia, cognitive deficits, and long-term health conditions such as cardiovascular disease and metabolic disorders. Moderating factors like resilience and gender further influence these interactions, with women being disproportionately affected. Despite advancements, gaps remain in understanding longitudinal effects and the role of digital stressors. This review highlights the need for targeted interventions to mitigate stress-induced sleep disruptions and calls for further research to address these gaps.

Keywords. chronic stress, sleep architecture, HPA axis, REM sleep, NREM sleep, hyperarousal, insomnia, cognitive impairment

1. Introduction

Chronic stress has emerged as a defining public health challenge of modernity, disrupting physiology and eroding mental resilience. According to the Ipsos World Mental Health Day 2024 report spanning 31 countries, 62% of participants reported stress-related disruptions to daily routines, with younger generations disproportionately affected – 54% required work leave due to stress [1]. While acute stress serves as an adaptive human response, chronic stress elevates risks for cardiovascular disease [2], impairs memory, cognition, and learning [3], and destabilizes the foundation of restorative health: sleep [4].

This review examines how chronic and pathological patterns of three non-rapid eye movement (NREM) stages and rapid eye movement (REM) sleep [5]. We analyzed neurobiological mechanisms, moderating factors, and consequences of sleep impairment, while identifying research gaps. By integrating clinical and molecular perspectives, we aim to highlight pathways for targeted interventions.

2. Methodology

This review synthesizes the literature on the impact of chronic and pathological stress on sleep architecture, drawing primarily from publications indexed in the PubMed database. A targeted search was conducted using keywords such as chronic stress, pathological stress, sleep architecture, HPA axis, and REM/NREM sleep. Articles were selected based on their relevance to neurobiological mechanisms and measurable sleep outcomes. Priority was given to studies published within the last fifteen years, though foundational older studies were included to provide context.

3. Neurobiological mechanisms linking stress and sleep disruption

3.1 Hypothalamic-Pituitary-Adrenal axis dysregulation and cortisol release

Humans exhibit rhythmic cycles that govern physiological processes, operating over varying

timescales (hours, days, or months) to modulate bodily functions and establish consistent, reproducible patterns – such as sleep-wake cycles and menstruation. [6] These rhythms are organized by biological systems featuring central pacemakers, synchronized output signals to cells, tissues, and organs, and feedback loops that regulate activity while integrating external and internal cues. [7]

A key regulatory system is the Hypothalamic-Pituitary-Adrenal (HPA) axis, which controls the secretion of corticotropin-releasing hormone (CRH), adrenocorticotropic hormone (ACTH), and cortisol. The process begins in the hypothalamus, which releases CRH into the hypothalamic-pituitary portal system. CRH then stimulates the anterior pituitary (adenohypophysis) to secrete ACTH, which travels through the bloodstream to the adrenal cortex, where it triggers cortisol production in the zona fasciculata. The HPA axis self-regulates via negative feedback, suppressing further CRH and ACTH release to maintain homeostasis. [7] (See fig. 1)

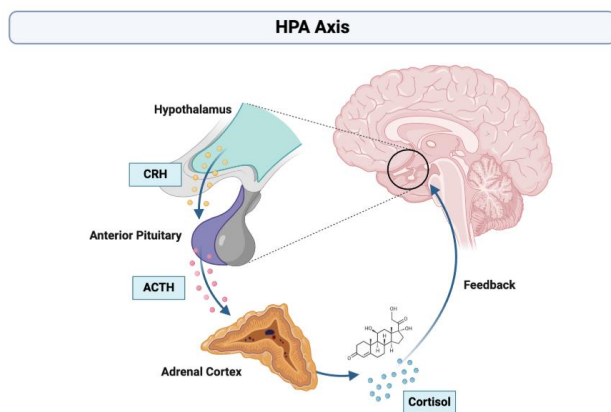


Fig. 1 - Kim, Y. (2024). Hypothalamic-Pituitary-Adrenal (HPA) Axis. Biorender.

Beyond intrinsic regulation, the HPA axis is influenced by external factors, such as photic entrainment – the synchronization of biological rhythms with the light-dark cycle, which includes cortisol release. Specialized retinal photoreceptors detect light and relay this information to the suprachiasmatic nucleus (SCN), the body’s master circadian clock. The SCN then signals the paraventricular nucleus (PVN) of the hypothalamus to modulate CRH release, thereby adjusting HPA axis activity in response to environmental light. [8] Disruption in any component of the HPA axis – such as glucocorticoid receptor desensitization, which impairs negative feedback and leads to sustained overactivation of the axis – can destabilize fundamental physiological processes. In acute stress, the limbic system activates the hypothalamus, triggering the HPA axis to initiate adaptive physiological responses crucial for survival. However, chronic stress disrupts this balance, dysregulating other systems – such as sleep, metabolism, and immune function – due to prolonged cortisol exposure and impaired feedback mechanisms [9].

Cortisol concentrations in blood plasma have been extensively profiled, demonstrating a distinct circadian rhythm that precisely coordinates with sleep-wake cycles. Research confirms cortisol levels remain markedly low throughout most nocturnal periods, followed by a characteristic surge preceding dawn that stimulates cortical activation and facilitates natural awakening. [10] (See Fig. 3)

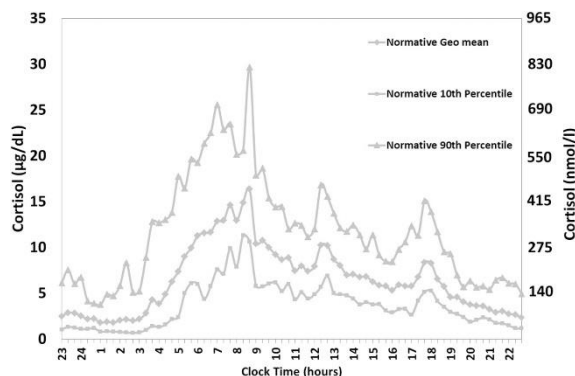


Fig. 2 - Fig. 2. Schematic of cortisol release in a day. Reprinted from Liyanarachchi et al. [3], with permission from Elsevier.

3.2 Role of the amygdala and prefrontal cortex in stress-induced hyperarousal

Hyperarousal is a heightened physiological and emotional state often observed during and after stress exposure. It is characterized by increased alertness, emotional reactivity, disrupted sleep, and autonomic hyperactivity [11]. At the neurobiological level, this state is primarily mediated by limbic structures such as the amygdala, and regulatory regions like the prefrontal cortex (PFC), whose interactions are critically altered under stress.

The amygdala plays a central role in initiating and maintaining stress responses. Under acute stress, it exhibits increased extracellular concentrations of excitatory neurotransmitters, including dopamine, noradrenaline, glutamate, and serotonin [12]. These neurochemical shifts heighten the amygdala’s excitatory output, enhancing the salience of threatening stimuli and contributing to behavioral hypervigilance and emotional overactivity. The amygdala also facilitates activation of the HPA axis, thereby promoting systemic stress responses, including glucocorticoid release.

The PFC, particularly its medial subdivisions, normally exerts inhibitory control over the amygdala, enabling emotional regulation and executive decision-making. However, stress exposure disrupts PFC function through neurochemical alterations such as increased dopamine and acetylcholine levels. These changes impair synaptic plasticity and reduce the efficacy of top-down inhibition [12]. Consequently, the amygdala’s excitatory drive is left unmodulated, allowing it to dominate behavioral and physiological responses.

Corticosterone, the primary glucocorticoid released during stress in rodents, has region-specific effects: it enhances excitatory signaling in the amygdala while compromising synaptic regulation in the PFC. This differential impact further skews the balance toward amygdala-driven hyperarousal, sustaining a state of heightened emotional and autonomic activation [12].

The imbalance between amygdala excitation and PFC inhibition is a hallmark of stress-induced hyperarousal. This shift favors immediate, emotionally charged responses at the expense of cognitive regulation, contributing to symptoms commonly observed in stress-related disorders such as PTSD, anxiety, and depression, even in early childhood [12,13].

4. Effects of pathological stress on sleep architecture

4.1 REM sleep disruptions

As a critical phase of the sleep cycle, Rapid Eye Movement (REM) sleep is characterized by heightened brain activity, muscle atonia, and vivid dreaming. It plays a vital role in emotional regulation, memory consolidation, and neurophysiological restoration [14]. However, REM sleep is highly vulnerable to pathological stress, which can disrupt its duration, latency (time to onset), and density (frequency of rapid eye movements) [15]. Chronic stress may lead to either an increase or decrease in REM sleep, depending on the stressor's nature, intensity, and individual susceptibility [16]. Fragmented REM sleep, marked by frequent awakenings and instability, compromises its restorative functions and exacerbates emotional reactivity [17]. These findings suggest that REM sleep disturbances not only serve as biomarkers of psychological distress but may also perpetuate and amplify maladaptive emotional states.

4.2 NREM sleep disruptions

Non-rapid eye movement (NREM) sleep is particularly susceptible to disruption by pathological stress. NREM sleep, which accounts for roughly 75–80% of total sleep time, is composed of three stages (N1 to N3), each progressively deeper. The third stage, known as slow-wave sleep (SWS) or deep sleep, is marked by the presence of high-amplitude, low-frequency delta waves (0.5–4 Hz) and is considered the most restorative phase of sleep [18]. It is during this stage that the brain engages in essential processes such as synaptic downscaling, metabolic clearance, and, crucially, memory consolidation.

Experimental evidence highlights the vulnerability of SWS to stress-induced alterations. For instance, animals subjected to fear conditioning exhibit changes in memory strength and specificity depending on the timing and presence of replay during SWS. A study showed that replay during post-training SWS significantly enhances the precision

and strength of odor-based fear memories, while disruption of this process or replay during wakefulness can lead to generalization or extinction of memory traces [19]. This emphasizes SWS's role in preserving the fidelity of memory consolidation and indicates how stress-induced alterations may degrade this function. Moreover, the link between sleep architecture and memory was explored in mice lacking adult hippocampal neurogenesis. These mice, modeling a pathological disruption of neural plasticity, exhibited reduced NREM sleep duration, altered slow-wave activity, and impaired sleep spindles, all of which correlate with poor spatial memory consolidation. [20] These findings suggest that hippocampal integrity and neurogenesis are closely tied to NREM-dependent memory processing and are particularly susceptible to stress-related pathologies.

Altogether, pathological stress reduces the quality and quantity of SWS, impairing its role in synaptic downscaling and the replay mechanisms essential for memory consolidation.

4.3 Sleep continuity disruptions

Stress-induced alterations in sleep patterns are often characterized by disruptions in the natural progression through the various sleep stages, with a particular focus on impairing the deeper restorative stages of sleep. Frequent awakenings during the night and extended sleep latency are two of the most common manifestations of these disruptions, which prevent the individual from achieving adequate sleep consolidation. These disturbances are not merely transient but can lead to long-term consequences for both mental and physical health [21].

Among the most notable effects of stress on sleep is the heightened incidence of insomnia, which is considered a primary comorbidity in individuals suffering from chronic stress conditions. Studies have shown that individuals experiencing high levels of pathological stress are more likely to report difficulties with both sleep onset and sleep maintenance, thus fragmenting their overall sleep continuity [22]. In particular, the physiological stress response - marked by the activation of the HPA axis and increased cortisol levels - can directly impact the neurobiological mechanisms responsible for regulating sleep cycles. The presence of insomnia further complicates these issues, as individuals with stress-induced insomnia tend to experience a vicious cycle, where poor sleep exacerbates stress and vice versa [23].

Additionally, these sleep disturbances not only affect the quality of rest but also hinder the healing process, potentially delaying cognitive and physical recovery. Furthermore, individuals from lower socioeconomic backgrounds, who may experience chronic stress due to factors such as financial insecurity and poor living conditions, are at an even greater risk for these sleep disturbances [25,26].

5. Moderating factors influencing stress-sleep interactions

Resilience. An individual's intrinsic dexterity to cope and/or adapt in the face of stressors, maintaining psychological and physical well-being, is part of a multifaceted process influenced by neurobiological, social, and personal experiences. A recent study on the interplay between sleep quality, stress, and coping mechanisms revealed that individuals considered as part of the resilient personality type reported less perceived stress and better subjective sleep quality in comparison to other personality categories. Additionally, resilience was correlated with reduced incidence of depression and anxiety symptoms – which, as previously discussed in this review, assists in the preservation of physiological sleep architecture. [27] However, resilience has its limits. Another study associating resilience, perceived stress, and depressed mood in women under in-vitro fertilization (IVF) treatment, noted that while resilience may contribute to rapid recovery after a negative IVF outcome, it can diminish under chronic stressors, particularly among those with preexisting depressive symptoms [28]. Thus, these findings suggest that although resilience plays an important role as an adaptive mechanism in face of stress, it can be exhausted, leaving individuals vulnerable to the sustained effects of stress on both mind and body, such as sleep disruptions, especially in the context of mental health disorders.

Gender. Existing literature highlights the disproportionate impact of sleep disruptions, and consequent alterations in sleep architecture, on women compared to men. Women are 1.5 times more likely to experience insomnia and twice as likely to suffer from depression as their male counterparts [29-31]. A recent review on the interplay between sex hormones, sleep disturbances, and depression reiterated the well-documented bidirectional relationship between poor sleep and depression while shedding light on the emerging role of endogenous sex hormones in sleep regulation. Notably, higher endogenous estrogen levels appeared protective against sleep disturbances, whereas exogenous hormonal sources were associated with worsened sleep quality and exacerbated psychological symptoms, particularly in individuals with preexisting depressive symptoms. [32] Whilst menopausal women, marked by the decline in estrogen, are biologically predisposed to experiencing insomnia, younger women are vulnerable to sleep and mood disruptions from exogenous hormones (e.g., hormonal contraceptives). This hormonal volatility, combined by women's higher baseline susceptibility to stress-related conditions, emphasizes the need for sex-specific interventions in sleep and mental health management.

6. Consequences of stress-induced sleep disruptions

6.1 Cognitive impairments and emotional dysregulation

The consequences of poor-quality or insufficient sleep are wide-ranging, affecting even mental health – from cognitive performance to emotional regulation [33]. The human body relies on sleep for its memory consolidation properties, rest (derived from the temporary absence of external stimuli), among others. As a result, even a single night with fewer than five hours of uninterrupted sleep can lead to noticeable declines in cognitive performance [34]. A recent study on the cognitive effects of chronic sleep deprivation among medical residents – a population whose work inherently demands attention, memory, and decision-making under stress – used a comparative methodology to assess test results across groups with varying levels of sleep deprivation. The findings revealed cognitive decline and increased impulsivity in workers subjected to more severe sleep deprivation [35]. These negative patterns also extend to emotional regulation. Studies indicate heightened generalized emotional reactivity [35] and increased attention to negative stimuli [37] in cases of chronic sleep deprivation.

6.2 Long-term health risks

Sleep deprivation, particularly the loss of slow-wave sleep essential for physiological and immune restoration, demonstrates strong associations with sustained elevation of inflammatory markers such as C-reactive protein and interleukin-6 [38]. This pro-inflammatory state from fragmented sleep may drive the pathogenesis of chronic inflammatory diseases, including rheumatoid arthritis and autoimmune disorders. In terms of metabolic consequences, those include dysregulated glucose metabolism and energy balance, which can precipitate insulin resistance and weight gain - mechanisms that contribute directly to the development of type 2 diabetes and obesity [39]. Within the cardiovascular system, prolonged deep-sleep deprivation induces endothelial dysfunction, along with heightened sympathetic activity and impaired vascular tone, ultimately increasing the risk of hypertension [40].

7. Gaps in research and future directions

Longitudinal studies. Most evidence linking chronic stress to sleep disorders derives from cross-sectional studies. While valuable for identifying correlations, such designs cannot track temporal progression - a critical limitation given the cumulative, worsening nature of stress-related sleep deterioration. Longitudinal implementations could directly observe subjects across months/years, revealing how sleep architecture degrades with prolonged stress exposure. This approach would better map individual disease progression, identify vulnerability

windows, and precisely evaluate preventive interventions. Crucially, it would clarify causal direction – determining whether stress triggers insomnia or whether chronic insomnia increases stress susceptibility – while detecting delayed effects often missed in cross-sectional analyses. [41-43]

Digital stressors. Existing studies highlight that excessive screen use, particularly before bedtime, can disrupt sleep patterns by delaying sleep onset, reducing sleep duration, and fragmenting sleep continuity [44-46]. However, much of the current research remains focused on isolated digital behaviors rather than the cumulative effects of multiple digital stressors across the lifespan. Furthermore, while psychological factors such as increased mental arousal and delayed melatonin release are known to disrupt sleep [44,46], the underlying biological mechanisms are not fully understood, highlighting the need for research into the neurophysiological processes involved. It is equally critical to explore individual differences in susceptibility to digital stress, as not all individuals experience the same level of sleep disruption, which calls for tailored interventions like digital detox strategies and cognitive-behavioral therapies. Future research should aim to clarify the interactions between different types of digital stressors, explore long-term effects across the lifespan, and investigate the underlying biological mechanisms.

8. Conclusion

Chronic stress disrupts sleep by impairing HPA axis function and limbic activity, leading to fragmented sleep, reduced slow-wave sleep, and REM disturbances. These disruptions harm emotional, cognitive, and physical health, with women and chronically stressed individuals at higher risk. A stress-sleep deprivation cycle worsens mental and physical health, requiring integrated interventions. Future research should focus on longitudinal studies and digital stressors to improve sleep health strategies.

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