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The impact of night shift work on cortisol secretion

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Abstract

Night-shift work poses significant challenges to human health, largely through disruption of circadian rhythms and dysregulation of cortisol secretion. Cortisol, a key glucocorticoid, regulates metabolism, immune function, and stress responses, following a distinct diurnal pattern characterized by a morning peak and gradual decline throughout the day. Night-shift work alters this rhythm, often causing blunted cortisol awakening responses, elevated evening cortisol levels, and overall circadian misalignment. These hormonal disturbances contribute to a spectrum of adverse health outcomes, including metabolic dysregulation (insulin resistance, hyperglycemia, obesity), cardiovascular disorders (hypertension, atherosclerosis, increased

cardiovascular mortality), impaired cognitive function, and heightened susceptibility to mood disorders such as anxiety and depression. Sleep disruption further exacerbates cortisol dysregulation and associated physiological and psychological effects. Understanding the mechanisms linking night-shift work to cortisol rhythm alterations is crucial for developing interventions aimed at mitigating health risks in shift-working populations. This review synthesizes current evidence on the effects of night-shift work on cortisol dynamics and the broader implications for metabolic, cardiovascular, and mental health.

Keywords: night-shift work; cortisol circadian rhythm; cortisol; cortisol awakening response (CAR); hypothalamic-pituitary-adrenal (HPA) axis; hypercortisolism, metabolic dysregulation, cardiovascular diseases, sleep debt

Introduction

Night-shift work can negatively affect workers' health and overall well-being. These impacts include: disruptions to normal circadian rhythms, psychological and physiological functions; reduced performance and efficiency which can lead to mistakes and accidents; difficulties in maintaining family and social relationships; a gradual decline in health, often beginning with sleep and eating problems and, over the long term, progressing to serious gastrointestinal issues, neuropsychological problems (like chronic fatigue, anxiety, and depression), and cardiovascular diseases (including hypertension and ischemic heart conditions) [1]. This review explores the impact of night-shift work on circadian rhythm regulation, with particular attention to alterations in cortisol secretion patterns and their wider consequences for metabolic and general health. It also evaluates the health risks arising from circadian misalignment, with a specific focus on metabolic disorders, cardiovascular disease, and cognitive functioning.

Aim of the study

The aim of this study is to review and synthesize current evidence on the effects of night-shift work on circadian rhythm regulation, with particular emphasis on alterations in cortisol secretion patterns. This study seeks to examine the mechanisms linking circadian misalignment and dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis to metabolic,

cardiovascular, cognitive, and mental health outcomes in night-shift workers. Additionally, the study aims to highlight the role of sleep disruption in exacerbating cortisol dysregulation and to discuss potential implications for risk mitigation and preventive strategies in shift-working populations.

Material and methods

This study was conducted as a narrative review of peer-reviewed scientific literature addressing the effects of night-shift work on circadian rhythm regulation and cortisol secretion. Relevant publications were identified through comprehensive searches of electronic databases, including PubMed, Scopus, and Google Scholar. The literature search was performed using combinations of predefined keywords related to night-shift work, circadian rhythms, cortisol, cortisol awakening response, and the hypothalamic–pituitary–adrenal axis. Only articles published in English were considered for inclusion. Both original research articles and review papers were included to ensure a broad overview of available evidence. Studies were selected based on their relevance to the study aim, clarity of methodology, and scientific quality. The selected literature was systematically reviewed, compared, and synthesized to provide an integrated summary of current findings and to identify common patterns and knowledge gaps related to cortisol dysregulation and circadian misalignment in night-shift workers.

Cortisol physiological functions

Cortisol performs numerous roles in the body, including managing the stress response, controlling metabolic processes, and regulating both inflammation and immune system activity. Cortisol influences multiple tissues, including the liver, muscles, fat, and pancreas. In the liver, elevated cortisol promotes gluconeogenesis while reducing glycogen formation. In muscles, it lowers glucose uptake and usage and accelerates protein breakdown, providing amino acids for glucose production. In adipose tissue, cortisol stimulates lipolysis. Additionally, in the pancreas, cortisol decreases insulin secretion and increases glucagon release, with both hormones together enhancing gluconeogenesis, glycogen breakdown, ketone production, and lipolysis in the liver [12]. Cortisol also has anti-inflammatory functions, contributing to the regulation of immune function and supporting immune system balance over the course of the day [2].

Cortisol circadian rhythm regulation

Cortisol is a major glucocorticoid hormone whose levels follow a distinct daily rhythm crucial for preserving physiological balance. Produced and released by the adrenal cortex’s zona

fasciculata. It is involved in regulating energy use, modulating immune activity, and coordinating the response to stress. Its production is controlled by the hypothalamic-pituitary-adrenal (HPA) axis, which operates on a carefully timed circadian schedule. Cortisol displays a distinct daily cycle, rising to its highest levels in the early morning and steadily decreasing as the day progresses. This rhythmic pattern can be evaluated using several diagnostic methods, including saliva, blood, or urine testing [2][3]. Salivary cortisol is widely utilized because it is easy to collect and accurately represents the physiologically active free cortisol fraction. In contrast, blood tests measure total cortisol, including both free and protein-bound hormone, while urinary cortisol reflects cumulative cortisol output across a 24-hour period [4].

The suprachiasmatic nucleus (SCN), known as the body's central circadian clock, governs this daily pattern of cortisol release. Situated in the hypothalamus, the SCN synchronizes internal physiological rhythms with environmental cues - most notably light, which is transmitted from specialized retinal ganglion cells through the retinohypothalamic tract. Light sensed by the retina triggers the suprachiasmatic nucleus (SCN), the central circadian pacemaker, which regulates the organism's cycles of activity and rest. By aligning peripheral cellular clocks throughout the body, the SCN indirectly drives cortisol secretion by stimulating the pituitary to release adrenocorticotropic hormone (ACTH). Produced and released by corticotrophs in the anterior pituitary, ACTH travels through the bloodstream to activate specific receptors on the surface of adrenal cortical cells. These receptors are located in the adrenal zona fasciculata, where further cortisol synthesis takes place [5]. Through this pathway, cortisol secretion stays aligned with the natural light–dark cycle. When the SCN is disrupted or becomes desynchronized from external stimuli, this coordination can break down, resulting in altered cortisol rhythms [2].

The Cortisol Awakening Response (CAR)

The cortisol awakening response (CAR) is marked by a swift increase in cortisol shortly after waking, generally rising 38–75% above baseline and reaching its peak approximately 30 minutes later. This surge can be significantly reduced by low-dose dexamethasone, a synthetic glucocorticoid that imitates cortisol's negative feedback on ACTH release, demonstrating that CAR is controlled by HPA axis feedback mechanisms [6]. The morning cortisol peak helps the body get ready for the day's challenges by mobilizing energy stores, increasing glucose availability, and regulating immune activity. After this early surge, cortisol levels steadily decrease over the course of the day, reaching their lowest levels during the night [2].

In addition to the cortisol awakening response (CAR), other cortisol-based circadian measures offer important information about the HPA axis's daily dynamics. Monitoring cortisol over a full 24-hour period enables a detailed view of secretion patterns beyond the morning peak. Key chronobiological metrics, such as acrophase (the timing of the cortisol peak) and mesor (the 24-hour average cortisol level), are often used to detect shifts in circadian timing, for example, in individuals working night shifts. These approaches provide a broader understanding of circadian rhythm disturbances, complementing insights gained from CAR [7].

Impact of working night shifts on cortisol secretion patterns

Circadian desynchronization arises when the body's internal clock becomes out of sync with external cues which is commonly seen in night-shift workers. This misalignment interferes with the physiological functions controlled by the circadian system and can contribute to negative health effects discussed further [9]. The suprachiasmatic nucleus (SCN) in the hypothalamus acts as the body's central clock, aligning physiological functions, including cortisol secretion with the external light–dark cycle. Normally, the SCN maintains a clear daily rhythm of cortisol release, marked by a strong rise after awakening followed by a steady decline across the day. In night-shift workers, however, exposure to artificial light at night and daytime sleep disturb this alignment, leading to substantial circadian desynchronization [10]. Circadian misalignment lead to shifts in the timing of peak cortisol release, increased cortisol levels in the evening, or a reduced cortisol awakening response (CAR) [8]. This dysregulation causes a shift in the timing of cortisol release, with peak concentrations appearing at inappropriate times - such as during the night or early morning when cortisol levels are normally low in individuals who follow a typical day–night schedule [11]. The cortisol awakening response (CAR) typically helps the body get ready for daily activities by enhancing energy supply, supporting cognitive performance, and regulating stress responses. A blunted CAR refers to a noticeably smaller rise in cortisol during the first 30–45 minutes after waking. Under normal conditions, cortisol levels increase by about 50–150% from waking baseline. When this rise is below 50%, the response is considered blunted, suggesting reduced HPA axis activity [2]. As the diurnal cortisol rhythm becomes less distinct, the timing and amplitude of cortisol secretion are impaired, weakening the body's capacity to regulate energy balance, stress responses, and immune function [11].

Health consequences of changes in cortisol secretion patterns

Ongoing misalignment between the body's circadian clock and external schedules in night-shift workers frequently results in chronic hypercortisolism, characterized by persistently elevated

cortisol levels. Unlike the normal daily rhythm, which supports metabolism, immune function, and stress regulation, sustained high cortisol disrupts these processes and poses serious health risks. Sustained high cortisol levels can disrupt metabolism, causing increased gluconeogenesis, insulin resistance, and accumulation of visceral fat. These metabolic disturbances raise the risk of obesity, type 2 diabetes, and metabolic syndrome. Additionally, chronic hypercortisolism contributes to dyslipidemia by elevating triglycerides and lowering HDL cholesterol, thereby increasing the likelihood of cardiovascular disease [13]. Beyond its metabolic consequences, chronic hypercortisolism also adversely affects the cardiovascular system. Persistently elevated cortisol can raise blood pressure by promoting sodium retention and increasing vascular sensitivity to catecholamines, contributing over time to hypertension, endothelial dysfunction, and atherosclerosis, thereby elevating the risk of stroke and heart attack. The immune system is similarly compromised: although cortisol's immunosuppressive effects help limit excessive inflammation during acute stress, chronic elevation weakens immune surveillance, making individuals more prone to infections and slowing recovery. Disrupted cortisol rhythms can also drive low-grade chronic inflammation, which has been linked to autoimmune disorders and cancer. Psychological health is affected as well, with prolonged high cortisol associated with mood disorders such as depression and anxiety. Night-shift workers often experience fatigue, irritability, and impaired cognitive performance, symptoms that are intensified by cortisol dysregulation and disturbed sleep [2] [13].

Health implications of chronic hypercortisolism

Metabolic dysregulation

Sustained exposure to elevated cortisol leads to profound disturbances in glucose regulation by affecting multiple metabolic tissues simultaneously, including the liver, skeletal muscle, adipose tissue, and pancreatic islets. In the liver, glucocorticoid excess enhances gluconeogenic activity and weakens insulin-mediated inhibition of glucose production, resulting in increased hepatic glucose output and contributing to hyperglycemia. [14] In peripheral tissues, cortisol reduces insulin sensitivity through distinct mechanisms. In skeletal muscle, it interferes with post-receptor insulin signaling, limiting glucose transporter mobilization and reducing glucose uptake. [15] In adipose tissue, cortisol alters adipocyte function by stimulating lipolysis and increasing the release of free fatty acids, which further impair insulin action in liver and muscle and promote a lipotoxic metabolic environment. These effects are intensified by local regeneration of active cortisol through tissue-specific enzymatic pathways, particularly within visceral fat depots. [16] Chronic glucocorticoid exposure also compromises β-cell function,

diminishing insulin secretory capacity and restricting the ability to compensate for rising insulin resistance. In parallel, dysregulation of central neuroendocrine pathways—most notably persistent activation of the hypothalamic-pituitary-adrenal axis and altered glucocorticoid receptor signaling - reinforces peripheral metabolic defects. Disruption of normal circadian cortisol patterns further exacerbates insulin resistance and impairs metabolic adaptability. [17] Together, these interacting peripheral and central mechanisms support the view that hypercortisolism functions as a primary driver of glucose dysregulation, rather than merely a secondary consequence of metabolic disease, and should be considered an integral component of contemporary models of type 2 diabetes pathophysiology. [18]

Cardiovascular diseases

Chronic hypercortisolism is strongly associated with elevated cardiovascular morbidity and mortality. Hypertension is one of the most common manifestations. The pathophysiology is multifactorial, including activation of mineralocorticoid receptors due to saturation of 11β-hydroxysteroid dehydrogenase type 2, increased vascular responsiveness to catecholamines and angiotensin II, endothelial dysfunction with decreased nitric oxide and prostacyclin, and overactivation of the renin–angiotensin–aldosterone system. [21][22] Cortisol excess profoundly affects glucose metabolism. Cortisol also diminishes incretin hormone signaling and stimulates glucagon secretion, further aggravating hyperglycemia. As a result, impaired glucose tolerance and overt diabetes are common. [19][23] Dyslipidemia is a frequent complication, characterized by elevated triglycerides, increased LDL cholesterol, reduced HDL cholesterol, and accumulation of visceral adipose tissue. Cortisol enhances peripheral lipolysis while promoting central fat deposition and hepatic lipid synthesis, contributing to insulin resistance and atherogenesis. Chronic hypercortisolism also promotes systemic inflammation, endothelial dysfunction, and pro-thrombotic states, further amplifying cardiovascular risk. [20][23] Structural cardiovascular alterations are significant. Patients frequently develop left ventricular hypertrophy, myocardial fibrosis, and diastolic dysfunction, along with accelerated atherosclerosis and increased carotid intima-media thickness. Clinically, these changes are associated with elevated rates of myocardial infarction, stroke, heart failure, and cardiovascular death. Persistent cardiovascular abnormalities are often observed even after successful treatment, reflecting the long-term effects of chronic cortisol exposure. [21][22]

Mental health disorders

The dysregulation of cortisol dynamics has been linked to altered emotional regulation and increased susceptibility to mood disorders. Elevated or mistimed cortisol levels can enhance reactivity within the limbic system, particularly the amygdala, which is critically involved in processing fear and emotional responses. Such alterations may underlie heightened anxiety, irritability, and stress sensitivity observed in individuals with chronic circadian misalignment. A sustained blunted cortisol awakening response (CAR) has been correlated with symptoms of fatigue, reduced stress resilience, and cognitive impairment, all of which are common precursors or features of anxiety and depressive disorders. [2][24][25] Moreover, night-shift workers often experience persistent circadian misalignment that exacerbates sleep disturbances, creating a bidirectional relationship between cortisol rhythm disruption and mental health: poor sleep quality worsens mood disturbances, while psychological stress and depressive symptoms further fragment sleep architecture, compounding HPA axis dysfunction. This reciprocal interaction may contribute to chronic stress states that further dysregulate cortisol rhythms and elevate the risk of clinically significant mood disorders, including major depressive disorder (MDD) and generalized anxiety disorder (GAD). [25] Elevated cortisol levels outside the normal diurnal range have been associated in other research with impaired affective regulation and increased psychiatric morbidity, suggesting that HPA axis hyperactivity and altered circadian cortisol patterns constitute key biological mediators linking shift work to mental health outcomes. [26]

Impact of night-shift work on sleep and cognitive performance

Night shift schedule significantly disrupts normal sleep patterns and circadian physiology. Workers attempting daytime sleep experience shorter and more fragmented rest periods due to misalignment with the body's natural circadian rhythm and environmental disturbances such as light and noise. On average, daytime sleep is 1–4 hours shorter than nocturnal sleep, with longer sleep onset latency and frequent awakenings, which cumulatively contribute to chronic sleep debt. [27][28] The architecture of sleep is also adversely affected. Night and rotating shift workers show reductions in slow-wave sleep (SWS) and rapid eye movement (REM) sleep, which are essential for physical restoration, memory consolidation, and emotional regulation. Loss of SWS undermines recovery from physical and cognitive fatigue, whereas diminished REM sleep can impair mood stability and executive function. Repeated exposure to rotating shifts without sufficient recovery prevents normalization of sleep stages, compounding fatigue

and reducing alertness [29][30] Disruption of sleep timing further interferes with hormonal regulation, particularly cortisol secretion. Normally, cortisol peaks shortly after awakening to support alertness and cognitive function. In shift workers, this morning surge is often delayed or blunted, while cortisol may remain elevated during attempted daytime sleep, leading to lighter, less restorative sleep and difficulty achieving full recovery. [31][32] These disturbances have downstream effects on fatigue and cognitive performance. Chronic sleep loss and circadian misalignment impair psychomotor function, attention, and short-term memory. The resulting deficits in alertness and vigilance increase the risk of errors and accidents, with implications for both occupational safety and overall health. [31]

Conclusion

Night-shift work disrupts the body's natural circadian rhythms, leading to significant physiological and psychological consequences. Altered sleep–wake cycles, exposure to artificial light at night, and misaligned cortisol secretion collectively impair restorative sleep, reduce slow-wave and REM sleep, and contribute to chronic sleep debt. These disturbances weaken energy regulation, cognitive function, and stress resilience, increasing fatigue, reducing attention, and elevating the risk of workplace errors. Chronic disruption of cortisol rhythms, including blunted cortisol awakening response and elevated evening levels, promotes metabolic dysregulation, insulin resistance, visceral fat accumulation, and dyslipidemia, thereby increasing the risk of obesity, type 2 diabetes, and cardiovascular diseases such as hypertension, atherosclerosis, and myocardial injury. Elevated or mistimed cortisol also adversely affects the central nervous system and limbic structures, heightening vulnerability to mood disorders, anxiety, and impaired emotional regulation. The combination of disrupted sleep, altered hormonal patterns, and cognitive impairment underscores the multifaceted health risks of night-shift work. Interventions aimed at stabilizing circadian rhythms, optimizing sleep timing, and mitigating cortisol dysregulation may help preserve metabolic, cardiovascular, and mental health, as well as support cognitive performance and overall well-being in night-shift workers.

Disclosure

The authors declare that they have no relevant financial or non-financial interests to disclose.

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Conflict of interest statement

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