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Impacts of chronic sleep deprivation on human health

Impactos da privação crônica de sono na saúde humana

Impactos de la privación crónica del sueño en la salud humana

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ABSTRACT

Objective: To analyze the effects of chronic sleep deprivation on human health. **Literature review:** Chronic sleep deprivation is characterized as a multisystem condition capable of compromising essential biological processes and triggering progressive cognitive deficits when wakefulness exceeds physiological limits. The findings indicate neurocognitive and neurodegenerative impairments, as well as significant cardiovascular, metabolic, and endocrine alterations. In addition, insufficient sleep negatively affects mental health and professional performance, representing an important threat to public health. **Final considerations:** Chronic sleep deprivation goes beyond simple fatigue, exerting broad and harmful effects on different body systems. Therefore, it is essential to recognize sleep as an indispensable biological component for maintaining homeostasis and quality of life, as well as to implement educational measures and public policies aimed at promoting healthy sleep and raising public awareness about the risks associated with insufficient sleep.

Keywords: Chronic sleep deprivation, Human health, Health impacts, Physiological phenomena, Metabolism.

RESUMO

Objetivo: Analisar os efeitos da privação crônica do sono sobre a saúde humana. **Revisão bibliográfica:** A privação crônica do sono configura-se como uma condição multissistêmica, capaz de comprometer processos biológicos essenciais e desencadear déficits cognitivos progressivos quando o tempo de vigília ultrapassa limites fisiológicos. Os achados evidenciam prejuízos neurocognitivos e neurodegenerativos, bem como alterações cardiovasculares, metabólicas e endócrinas significativas. Além disso, o sono insuficiente impacta negativamente a saúde mental e o desempenho profissional, caracterizando-se como uma importante ameaça à saúde pública. **Considerações finais:** A privação crônica do sono ultrapassa o simples cansaço, exercendo efeitos amplos e deletérios sobre diferentes sistemas do organismo. Dessa forma, torna-se imprescindível reconhecer o sono como um componente biológico indispensável à manutenção da homeostase e da qualidade de vida, bem como implementar medidas educativas e políticas públicas voltadas à promoção do sono saudável e à conscientização da população sobre os riscos associados ao sono insuficiente.

Palavras-chave: Privação crônica do sono, Saúde humana, Impactos na saúde, Fenômenos fisiológicos, Metabolismo.

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RESUMEN

Objetivo: Analizar los efectos de la privación crónica del sueño sobre la salud humana. **Revisión bibliográfica:** La privación crónica del sueño se configura como una condición multisistémica, capaz de comprometer procesos biológicos esenciales y desencadenar déficits cognitivos progresivos cuando el tiempo de vigilia supera los límites fisiológicos. Los hallazgos evidencian perjuicios neurocognitivos y neurodegenerativos, así como alteraciones cardiovasculares, metabólicas y endocrinas significativas. Además, el sueño insuficiente impacta negativamente la salud mental y el desempeño profesional, caracterizándose como una importante amenaza para la salud pública. **Consideraciones finales:** La privación crónica del sueño supera el simple cansancio, ejerciendo efectos amplios y perjudiciales sobre diferentes sistemas del organismo. Por lo tanto, resulta imprescindible reconocer el sueño como un componente biológico indispensable para el mantenimiento de la homeostasis y la calidad de vida, así como implementar medidas educativas y políticas públicas orientadas a la promoción del sueño saludable y a la concienciación de la población sobre los riesgos asociados al sueño insuficiente.

Palabras clave: Privación crónica del sueño, Salud humana, Impactos en la salud, Fenómenos fisiológicos, Metabolismo.

INTRODUCTION

Sleep constitutes an essential and indispensable biological function for human beings, playing a determining role in cognitive processes, emotional regulation, and the maintenance of both physical and mental health (RAHMAN SA, et al., 2020; SOUISSI W, et al., 2020). According to Rahman SA, et al. (2020), it is a well-regulated and rapidly reversible state of consciousness defined by distinct behavioral, homeostatic, and electrophysiological properties. This process facilitates critical restorative functions, including immune system performance, physiological and neural homeostasis, and the consolidation of memory (DIEKELMANN S, et al., 2024; LECLERC A, 2024). According to Barros JP e Sousa CE (2022), this process is actively governed by circadian mechanisms lasting approximately 24 hours and by homeostatic factors - such as the accumulation of adenosine-responsible for the progressive increase in sleepiness throughout the waking period.

Chaudhry N e Hussain ME (2022) emphasize the importance of distinguishing between sleep deprivation patterns based on their duration and physiological cost. Acute sleep deprivation refers to a total lack of sleep or a severe reduction in total sleep time, typically lasting 24 to 48 hours, where wakefulness extends beyond 18 hours. In contrast, Chronic Sleep Deprivation (CSD) is characterized by a routine and persistent pattern of sleeping less than the amount required for optimal functioning, occurring almost every day for at least three months (CHAUDHRY N e HUSSAIN ME, 2022). While acute loss triggers immediate systemic changes, the neurobehavioral deficits of CSD accumulate over time, and individuals often become unaware of their increasing cognitive impairment (RAHMAN SA, et al., 2020; TARTAR JL, et al., 2022).

Modern lifestyle changes and relentless work demands have deprioritized restorative rest, establishing chronic sleep loss as a widespread public health challenge with severe global dimensions (CHAUDHRY N e HUSSAIN ME, 2022; REDDY S, et al., 2023). The classification of CSD as a major clinical concern stems from its ability to generate a cascade of repercussions that transcend individual fatigue. Clinically, it acts as a precursor to metabolic dysfunction and systemic inflammation (ALJURAIBAN A, et al., 2022; HE J, et al., 2022); socially, it exacerbates health inequities and compromises educational and professional performance, creating a cycle of reduced cognitive productivity and increased vulnerability to psychiatric disorders (BARROS JP e SOUSA CE, 2022; DEVIVA JC, et al., 2021). This condition is frequently overlooked and under-reported despite its systemic nature, affecting nearly every key indicator of morbidity and mortality (REDDY S, et al., 2023).

Wheaton AG, et al. (2023) report that epidemiological data reveal that approximately 33.2% of adults experience a short sleep duration, failing to achieve the recommended minimum of 7 hours of rest per 24-hour period. Such inadequate patterns are independently associated with an increased risk of premature mortality and a higher incidence of metabolic and cardiovascular disorders, such as hypertension and coronary heart

disease (GOTTESMAN RT, et al., 2024; LIEW SC e AUNG T, 2021). He J, et al. (2022) suggest that CSD acts as a systemic stressor that triggers the activation of the hypothalamic-pituitary-adrenal (HPA) axis and promotes a persistent low-grade inflammatory state. This process is evidenced by elevated circulating markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), which correlate with a higher risk of atherosclerosis and type 2 diabetes (CHIANG JJ, 2024; HE J, et al., 2022).

In the neurocognitive domain, significant impairment is observed in sustained attention, working memory, and executive functions (SOUISSI W, et al., 2020; HABIBURRAHMAN M, et al., 2025). Furthermore, insufficient sleep weakens prefrontal-amygdala connectivity, which may lead to emotional dysregulation, increased impulsivity, and heightened symptoms of anxiety and depression (LECLERC A, 2024). Wong SY, et al. (2022) state that a primary catalyst for this crisis in contemporary society is the pervasive exposure to artificial blue light from digital devices, which disrupts circadian entrainment. This environmental interference suppresses pineal melatonin synthesis - a hormone that also acts as a potent endogenous antioxidant - and induces a circadian phase delay, which reduces sleep efficiency and impairs alertness (BOUAZZA M, et al., 2024; RAHMAN SA, et al., 2020).

According to Aljuraiban A, et al. (2022), this disruption is often compounded by circadian misalignment, where behavioral cycles occur at inappropriate biological times (MCHILL AW, et al., 2020). Such desynchronization leads to acute metabolic alterations, as food intake occurring during the biological night triggers elevated postprandial glucose responses and higher energy intake, reinforcing a link between technology use and metabolic risks (WONG SY, et al., 2022).

Beyond individual health, CSD generates profound social and economic repercussions. Insufficient sleep duration and poor rest quality are directly linked to significant productivity losses through presenteeism and increased rates of absenteeism (WONG SY, et al., 2022; GONZÁLEZ-GARCÍA M, et al., 2024). Shift workers and healthcare professionals are particularly vulnerable, with frequent night duties significantly increasing the risk of Burnout syndrome and clinical errors (LEE KY, et al., 2024; NAIR S, et al., 2024). Sleep disorders represent a massive economic burden, incurring billions in direct healthcare costs and indirect losses related to the management of associated chronic comorbidities (RATHAGIRISHNAN R e BHANGU A, 2022). The scarcity of public policies aimed at promoting sleep hygiene worsens this scenario, reinforcing the need for preventive interventions. Therefore, it is essential to deepen the scientific understanding of these impacts to support strategies that prioritize sleep as a fundamental determinant of global health.

LITERATURE REVIEW

Neurocognitive and degenerative changes

CSD has been consistently associated with neurocognitive alterations characterized by attentional lapses, microsleep episodes, reduced working memory, and impairments in long-term memory consolidation (SAWADA Y, et al., 2024). Evidence from controlled experimental studies and observational investigations demonstrates that sustained reductions in sleep duration - particularly to values equal to or below 4-6 hours per night over weeks - promote progressive cognitive deficits in a dose - response pattern, often comparable to those observed after episodes of intense acute deprivation. According to HUDSON AN, et al. (2020), these findings indicate a cumulative effect of sleep restriction, especially on executive functions and vigilant attention.

From a neurobiological perspective, these impairments can be explained by alterations in fundamental mechanisms of brain restoration that occur during sleep. Among them are dysfunction of the glymphatic system and reduced slow-wave activity during NREM sleep, processes essential for the clearance of neurotoxic metabolites and the maintenance of synaptic homeostasis (BARROS JP e SOUSA CE, 2022; SAWADA Y, et al., 2024). Experimental and translational studies associate reduced sleep duration with the accumulation of proteins related to neurodegeneration, such as β-amyloid and phosphorylated tau; however, while CSD is linked to pathological processes typical of Alzheimer's disease, definitive causal confirmation still depends on more robust longitudinal studies (LIEW SC e AUNG T, 2021; RAMOS AR, et al., 2023; BARROS JP e SOUSA CE, 2022).

Khan MS e Al-Jahdali H (2023) suggest that sleep deprivation may directly compromise neurophysiological mechanisms involved in hippocampal memory consolidation, particularly those mediated by long-term potentiation (LTP). In this context, N-methyl-D-aspartate (NMDA) receptors play a central role in stabilizing newly acquired information and facilitating its transition into durable memory traces, a process that appears significantly impaired under conditions of sleep restriction (KHAN MS e AL-JAHDALI H, 2023).

Recent investigations also describe the phenomenon known as “local sleep,” in which specific cortical regions exhibit electrophysiological patterns resembling sleep during prolonged periods of wakefulness. This mechanism may explain attentional lapses and transient losses in cognitive performance even in the absence of intense subjective sleepiness, reinforcing the need for integration between behavioral, electrophysiological, and neuroimaging assessments to better understand the progression of synaptic alterations associated with prolonged deprivation (SAWADA Y, et al., 2024).

The reduction in sleep quantity and quality significantly compromises neurobehavioral performance, leading to cumulative deficits in attention, memory encoding, and executive functions that jeopardize long-term functional health (SOUISSI W, et al., 2020; LECLERC A, 2024). Even moderate and partial sleep restriction can produce measurable effects, including increased reaction time and reduced accuracy in cognitive tasks. SAWADA Y, et al. (2024) affirm that under these conditions, initial learning may occur, but consolidation and retention become significantly compromised as deprivation becomes chronic.

Executive functions, primarily supported by the prefrontal cortex in interaction with cingulate and parietal networks, demonstrate high sensitivity to sleep restriction, presenting disorganization of neural processing that accumulates over successive days (HUDSON AN, et al., 2020; SAWADA Y, et al., 2024). Concurrently, emerging evidence suggests that sleep deprivation may promote neuroinflammatory processes and microglial activation, though the extent to which these alterations persist after subsequent periods of sleep recovery remains a subject of ongoing research (RAMOS AR, et al., 2023; BARROS JP e SOUSA CE, 2022).

In summary, chronic sleep restriction to approximately 4-6 hours per night is associated with cumulative and dose-dependent cognitive deficits, often underestimated by individuals themselves. These findings reinforce the relevance of preserving sleep duration as a fundamental strategy for preventing cognitive decline and promoting public health (HUDSON AN, et al., 2020; SAWADA Y, et al., 2024).

Cardiovascular and hemodynamic repercussions

The association between insufficient sleep and cardiovascular dysfunction is strongly supported in the contemporary literature. Evidence from systematic studies and cohort analyses demonstrates that CSD is related to the loss of the physiological nocturnal blood pressure decline (non-dipping pattern), increased prevalence of arterial hypertension, and early markers of endothelial dysfunction (FARIAS AV, et al., 2022; PALMIERI DF, et al., 2025). These effects appear particularly pronounced in populations exposed to chronic occupational stressors, such as public safety professionals and healthcare workers subjected to shift work, suggesting a complex interaction between psychosocial overload, circadian dysregulation, and impaired cardiovascular recovery during the night (PALMIERI DF, et al., 2025; SAHAROV G, et al., 2025).

According to LI X, et al. (2023), at the molecular level, experimental studies indicate that although total levels of circulating exosomes remain relatively stable, plasma exosomes derived from individuals and animal models subjected to sleep deprivation exhibit high pro-inflammatory potential, acting as important mediators of endothelial inflammation and atherogenic processes. These findings reinforce the hypothesis that qualitative alterations in intercellular signaling mechanisms, rather than merely quantitative changes, play a relevant role in cardiovascular pathophysiology associated with insufficient sleep. Furthermore, CSD has been linked to direct cellular damage, including increased oxidative DNA damage and impaired DNA repair mechanisms, which may accelerate vascular aging and the progression of chronic diseases (CHEUNG V, et al., 2019; RAMOS AR, et al., 2023).

Chiang JJ (2024) describes that the proposed pathophysiological mechanisms include the persistent activation of the hypothalamic-pituitary-adrenal (HPA) axis and the sympathetic nervous system, leading to an

elevation of systemic catecholamines and a state of low-grade systemic inflammation. This inflammatory state is evidenced by elevated circulating markers such as C-reactive protein (CRP) and interleukin-6 (IL-6), which correlate with a higher risk of atherosclerosis and type 2 diabetes (CHIANG JJ, 2024; HE J, et al., 2022). Additionally, chronic sleep deprivation promotes a prothrombotic state characterized by the elevation of biomarkers such as plasminogen activator inhibitor-1 (PAI-1) and heparanase. Collectively, such alterations contribute to increased arterial stiffness, endothelial dysfunction, and mitochondrial oxidative stress (SAHAROV G, et al., 2025; LIEW SC e AUNG T, 2021).

Concurrently, evidence synthesized in meta-analyses indicates that regular physical exercise, associated with adequate cardiovascular conditioning, may partially attenuate the negative cardiovascular impacts of sleep deprivation, contributing both to cardiovascular protection and to the reduction of anxiety-related processes (ZHAO Y, et al., 2023). However, the cumulative burden of shift work and circadian misalignment remains a significant concern, as frequent night duties significantly increase the risk of Burnout syndrome and clinical errors, reinforcing the link between professional fatigue and systemic health decay (LEE KY, et al., 2024; NAIR S, et al., 2024).

Despite the consistency of observed associations, estimates of absolute cardiovascular risk vary according to individual factors, including comorbidities, genetic predisposition, and the intensity and duration of sleep restriction. Therefore, prospective stratified studies and longitudinal interventions capable of assessing the magnitude of reversibility of cardiovascular alterations related to CSD remain necessary to support more effective public health policies (RAMOS AR, et al., 2023).

Metabolic and endocrine imbalances

Aljuraiban GS, et al. (2022) demonstrate that chronic short sleep duration is linked to energy homeostasis disruption, characterized by increased adiposity and higher caloric intake. This state is associated with low-grade systemic inflammation that may promote insulin resistance (ALJURAIBAN GS, et al., 2022). Evidence suggests that sleep restriction alters the hedonic evaluation of food and induces "social jetlag," leading to poor dietary choices and lower fiber intake (ALJURAIBAN GS, et al., 2022). Hormonally, alterations in cortisol, ghrelin, and leptin levels increase susceptibility to obesity and type 2 diabetes (LIEW SC e AUNG T, 2021). McHill AW, et al. (2020) emphasize that even moderate restriction (~6.5 hours) can elevate postprandial glucose levels, illustrating rapid metabolic destabilization (MCHILL AW, et al., 2020).

CSD compromises energy metabolism by favoring adipose tissue accumulation and visceral fat deposition through the interference of energy expenditure and caloric intake balance (LIEW SC e AUNG T, 2021; RAMOS AR, et al., 2023). These biological mechanisms are amplified by behavioral influences, such as circadian misalignment-where food intake during the biological night triggers exaggerated glucose responses and endocrine alterations (MCHILL AW, et al., 2020; ALJURAIBAN GS, et al., 2022). Beyond glucose regulation, CSD induces a catabolic state, characterized by an elevated cortisol-to-testosterone ratio that favors muscle protein breakdown (DÁTTILO M, et al., 2020; LAMON S, et al., 2021). Furthermore, suppressed slow-wave sleep is associated with reduced growth hormone (GH) secretion, potentially impairing tissue recovery and cellular repair (DÁTTILO M, et al., 2020; CHAUDHRY N e HUSSAIN ME, 2022).

According to the review by Garbarino S, et al. (2021), chronic sleep deprivation induces prolonged sympathetic activation and autonomic dysfunction, increasing circulating catecholamine levels and resulting in elevated blood pressure. These findings support the role of autonomic imbalance in the development of cardiovascular alterations associated with insufficient sleep (GARBARINO S, et al., 2021). This state is exacerbated by systemic inflammation, characterized by elevated CRP and IL-6, and oxidative DNA damage (CHEUNG V, et al., 2019; CHIANG JJ, 2024). Additionally, melatonin suppression by nighttime light exposure weakens endogenous antioxidant defenses, as melatonin plays a critical role in neutralizing free radicals (BOUAZZA M, et al., 2024).

Glymphatic system dysfunction during sleep further raises concerns regarding impaired β-amyloid clearance, a potential risk factor for neurodegenerative processes (BARROS JP e SOUSA CE, 2022). While methodological limitations exist, such as reliance on self-reported measures, Wheaton AG, et al. (2023)

maintain that chronic sleep deprivation (CSD) establishes a documented risk for cardiometabolic dysfunction. The convergence of dysregulated hunger signals, persistent low-grade inflammation, and suppressed anabolic processes reduces metabolic flexibility, acting as a central mediator for early cardiometabolic disease (MCHILL AW, et al., 2020; RAMOS AR, et al., 2023).

Impacts on mental health and the professional sphere

CSD is a significant systemic issue, particularly among professionals on on-call schedules and students (PALMIERI DF, et al., 2025). Persistent sleep disturbances are identified as critical risk factors for mental health disorders, being independently associated with psychological distress and diminished professional performance (ZHANG L, et al., 2023; DEVIVA JC, et al., 2021). According to REDDY S, et al. (2023), this phenomenon is exacerbated by sociodemographic disparities, where atypical working hours and chronic stress contribute to a higher prevalence among vulnerable groups, characterizing a major public health challenge.

CSD severely impairs executive functions mediated by the prefrontal cortex, including sustained attention and cognitive flexibility. This neurocognitive decay results in attentional lapses and reduced psychomotor performance (HUDSON AN, et al., 2020; SAWADA Y, et al., 2024). A critical aspect of this condition is the divergence between subjective perception and objective performance: while neurobehavioral deficits accumulate over weeks, individuals often fail to accurately perceive their functional decline, leading to a dissociation between subjective alertness and objective impairment (HUDSON AN, et al., 2020; TARTAR JL, et al., 2022).

From a neurobiological perspective, sleep insufficiency weakens prefrontal-amygdala connectivity, which may contribute to emotional dysregulation and symptoms of anxiety (LECLERC A, 2024). Prolonged sleep loss is associated with increased irritability and cognitive disorganization (HYNDYCH A, et al., 2025; BARROS JP e SOUSA CE, 2022). These changes are often compounded by lifestyle factors, such as nighttime digital device use, which suppresses the synthesis of melatonin - a hormone that also functions as a potent endogenous antioxidant - and impairs academic achievement (BOUAZZA M, et al., 2024; HABIBURRAHMAN M, et al., 2025).

NAIR S, et al. (2024) demonstrate that in the professional realm, fatigue impairs clinical decision-making and increases the risk of medication errors. Lee KY, et al. (2024) observe that shift workers and healthcare professionals are particularly predisposed to Burnout syndrome and significant productivity losses. Additionally, Rathagirishnan R e Bhangu A (2022) emphasize that the massive economic burden involves billions in direct healthcare costs and indirect losses related to accidents and chronic comorbidities. Restructuring work schedules and prioritizing sleep hygiene are therefore essential requirements for institutional safety (BARROS JP e SOUSA CE, 2022; RAMOS AR, et al., 2023).

In summary, CSD-induced deficits in emotional self-regulation and cognitive precision extend beyond simple fatigue (LECLERC A, 2024; SAWADA Y, et al., 2024). The accumulation of neurobehavioral impairment without adequate subjective awareness creates a scenario of critical vulnerability, increasing the risk of operational errors (HUDSON AN, et al., 2020; LEE KY, et al., 2024). CSD must be recognized as a biopsychosocial determinant that compromises occupational safety, academic performance, and the integrity of global public health (REDDY S, et al., 2023).

5. Public health implications and bioethical considerations

The unequal distribution of CSD across socioeconomic and racial strata highlights critical social determinants of health. Atypical working hours, precarious housing, and chronic stress contribute to a higher prevalence of insufficient sleep among vulnerable groups, transforming sleep into a marker of social inequity (RAMOS AR, et al., 2023; LIEW SC e AUNG T, 2021). Epidemiological data reveal approximately 33.2% of adults fail to achieve 7 hours of rest, a statistic disproportionately higher in populations facing economic instability (WHEATON AG, et al., 2023; REDDY S, et al., 2023).

Bouazza M, et al. (2024) demonstrate that modern sleep loss is fueled by environmental and structural factors, particularly nighttime blue light exposure that disrupts circadian entrainment by suppressing melatonin

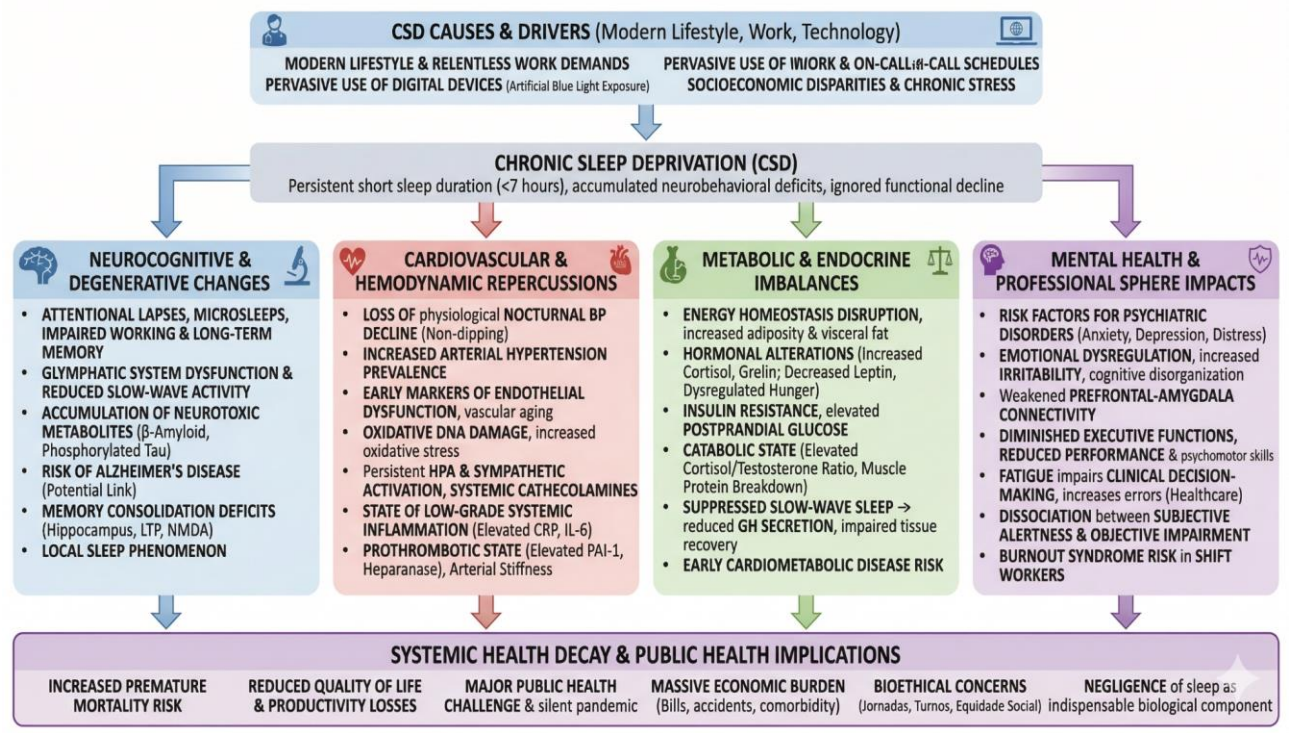
synthesis. This hormone also serves a critical role as an endogenous antioxidant (BOUAZZA M, et al., 2024). This technological interference reduces sleep efficiency and impairs next-morning alertness (BOUAZZA M, et al., 2024; HABIBURRAHMAN M, et al., 2025). Furthermore, Chiang JJ (2024) suggests that limited public awareness of metabolic and cardiovascular risks, such as systemic inflammation, prevents the adoption of preventive behaviors at the individual level (CHIANG JJ, 2024).

From a labor perspective, prolonged workdays and night shifts represent a bioethical challenge. Shift workers and healthcare professionals face chronic circadian misalignment, where biological rhythms are decoupled from social cycles (WONG SY, et al., 2022; PALMIERI DF, et al., 2025). This triggers metabolic alterations and potentially increases risks of burnout, clinical errors, and cardiovascular comorbidities (MCHILL AW, et al., 2020). The prioritization of economic productivity over biological health necessitates reassessing labor practices to protect restorative rest as a fundamental health determinant (BARROS JP e SOUSA CE, 2022; RAMOS AR, et al., 2023).

Bassetti CL, et al. (2026) demonstrate that the economic repercussions are significant, with sleep disorders incurring billions in direct healthcare costs and productivity losses. Consequently, González-García M, et al. (2024) maintain that chronic sleep deprivation must be addressed as a structural public health crisis. Barros JP e Sousa CE (2022) suggest that interventions should include implementing educational policies, restructuring shift work, and delaying school start times for adolescents to align with biological clocks. Furthermore, prioritizing longitudinal studies and cost-effectiveness evaluations is essential to support structural changes in the global health agenda (LIEW SC e AUNG T, 2021).

Barros JP e Sousa CE (2022) demonstrate that contemporary sleep deprivation involves bioethical neglect, where productivity pressures and technological presence override biological imperatives. Bassetti CL, et al. (2026) maintain that unregulated screen use and misaligned shifts create a cycle of health degradation. Furthermore, Reddy S, et al. (2023) emphasize that mitigating this widespread public health challenge requires treating sleep as a right to health and social equity, demanding structural interventions that prioritize circadian resynchronization (REDDY S, et al., 2023; BASSETTI CL, et al., 2026).

Figure 1 - Flowchart of the systemic consequences of chronic sleep deprivation on the neurological, metabolic, and cardiovascular axes.



Source: Cavalcanti RC, et al., 2026.

FINAL CONSIDERATIONS

The literature review demonstrates that CSD goes far beyond simple fatigue, causing profound impacts on cognitive functions, cardiovascular health, metabolism, mental health, and essential mechanisms of physiological regulation, especially among populations subjected to high cognitive demands and long working hours. Sleep is fundamental for homeostasis, memory consolidation, neuroendocrine balance, and cellular repair, and its neglect constitutes an important modifiable risk factor, with direct effects on quality of life, productivity, and safety. Therefore, it is essential to promote healthy sleep habits through health education, adjustment of daily routines, and public policies aimed at preventing sleep disorders, as well as to encourage further research to deepen understanding of the damage caused by insufficient sleep and to develop effective interventions.

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