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Psychological and Pharmacological Strategies to Address Sleep Disturbances in Chronic Illness: A Review

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ABSTRACT

Sleep disturbances are a significant and frequently neglected co-morbidity in chronic diseases that has a substantial effect on the course of the disease and patient well-being. This review describes a synthesis of evidence about the neurobiological and inflammatory processes of sleep disruption in such conditions as diabetes, cancer, cardiovascular disease, and neurodegenerative diseases. Central to this pathology is the disruption of a neurohormone system known as the hypothalamic/pituitary/adrenal (HPA), also called the "stress axis" - disruptions of the autonomic nervous system and neurotransmitters defined as gamma-aminobutyric acid (GABA), serotonin, acetylcholine. Chronic inflammation, mediated by inflammation mediators such as IL-6, TNF-α, and CRP, results in direct changes to neural circuits that control sleep-wake cycles, which then becomes a vicious cycle that worsens systemic disease. The review evaluates the available psychological treatments, especially the cognitive-behavioral treatment for insomnia (CBT-I), as well as pharmacological treatments, and their efficiency and defects. It highlights important areas for further research including lack of large-scale trials in specific disease groups, under-representation of elderly and multimorbid patients, and lack of data on longevity. Future directions focus on the concepts of precision medicine, digital therapeutics using AI, and interdisciplinary care models, as well as concentrating on the long-term quality of life outcomes to make sleep management the mainstay rather than an adjunct in chronic disease treatment.

KEYWORDS: Sleep Disturbances, Chronic Illness, HPA Axis, Inflammation, CBT-I, Precision Medicine.

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INTRODUCTION

Sleep disturbances are becoming more widely recognised as a comorbid factor found in a large number of chronic illnesses, with significant implications for both physiological and psychological outcomes of health (Budhiraja et al., 2011). Epidemiological data show that disorders like insomnia, hypersomnia, and fragmented sleep are present at disproportionately high rates in people with such chronic diseases as diabetes, cancer, cardiovascular, autoimmune, and neurodegenerative diseases. For example, almost 50-70% of patients with type 2 diabetes and up to 80% of people with cancer have sleep problems throughout the duration of their illness (Gupta et al., 2022). Similarly, patients with cardiovascular diseases tend to have poor sleep efficiency and also sleep apnea, whereas significant circadian rhythm disruptions characterize neurodegenerative diseases like Parkinson's and Alzheimer's disease (Parrino et al., 2022). One implication of these findings is the extensive burden and widespread prevalence of sleep disorders in chronic disease populations.

Clinically, sleep disturbances have far-reaching implications. Poor sleep quality and shorter sleep duration are associated with a lower quality of life, impaired cognitive and emotional functioning, and reduced ability to participate in daily activities (Ramos et al., 2023). Furthermore, insufficient sleep could impact the efficacy of treatment, exacerbating symptoms and disease progression by a number of different mechanisms including heightened inflammation, hormonal imbalance and lowered immunity. For example, for diabetes patients, sleep deprivation disrupts glucose metabolism and insulin sensitivity in the patients, and in oncology, insomnia is related to worse treatment and fatigue (Khan & Aouad, 2017). Thus, the management of sleep disturbance is not necessarily a quality of comfort but a fundamental part of holistic disease management.

From a pathophysiological perspective, the points of contact between chronic disease and sleep disruption are mediated by complex neuroendocrine and immunological mechanisms (Davis et al., 2020). Chronic inflammation, a hallmark of most non-

communicable diseases, can alter circadian regulation by affecting central clock genes and melatonin secretion, leading to dysregulated sleep-wake cycles. Conversely, disturbed sleep perpetuates systemic inflammation and oxidative stress, so that there is a vicious cycle of perpetuating disease progression (Musiek & Holtzman, 2016). Neurotransmitter imbalances that involve serotonin, dopamine, and GABA contribute further to changes in sleep patterns, particularly in conditions of neurodegeneration and psychiatric conditions (Xiao et al., 2023). Understanding these mechanisms provides a biological basis for therapeutic interventions that target them.

Given this multifaceted interplay, there is a strong need for an integrative review that explicitly addresses both psychological and pharmacological strategies for managing sleep disturbances associated with chronic illness (Maurer et al., 2018). While pharmacological agents (such as benzodiazepines, melatonin agonists or sedating antidepressants) are mainstays of treatment, non-pharmacological approaches to insomnia treatment, such as CBT-I, mindfulness-based interventions or sleep hygiene education, are sustainable, side effect free treatments (Rosenberg et al., 2021). A holistic synthesis of these approaches can help clinicians develop individual, evidence-based care programs to improve patient outcomes and overall wellness.

PATHOPHYSIOLOGY OF SLEEP DISTURBANCES IN CHRONIC ILLNESS

Neurobiological mechanisms

Dysregulation of the hypothalamic–pituitary–adrenal (HPA) axis and the autonomic nervous system.

The pathway starts with stress activating the hypothalamus, which in turn produces CRH. CRH stimulates the anterior pituitary to secrete ACTH that stimulates the adrenal gland to secrete cortisol, the primary stress hormone in Figure 1. A distinctive feature of this system is feedback inhibition: the secretion of cortisol stops both the hypothalamus and the anterior pituitary from overproducing. However, the diagram makes a special mention of "Chronic Cortisol Elevation" as a state of dysregulated response, implying that this feedback mechanism can become overwhelmed with prolonged stress. This protracted high dose of cortisol has been linked with deleterious systemic consequences, such as insulin resistance, loss of neurons in the brain, cardiovascular disease, and systemic inflammation. The inclusion of the placenta and estradiol/progesterone suggests that such HPA axis dysregulation can also influence reproductive hormones, as well as fetal development, further demonstrating that it impacts the ANS and overall health just as broadly.

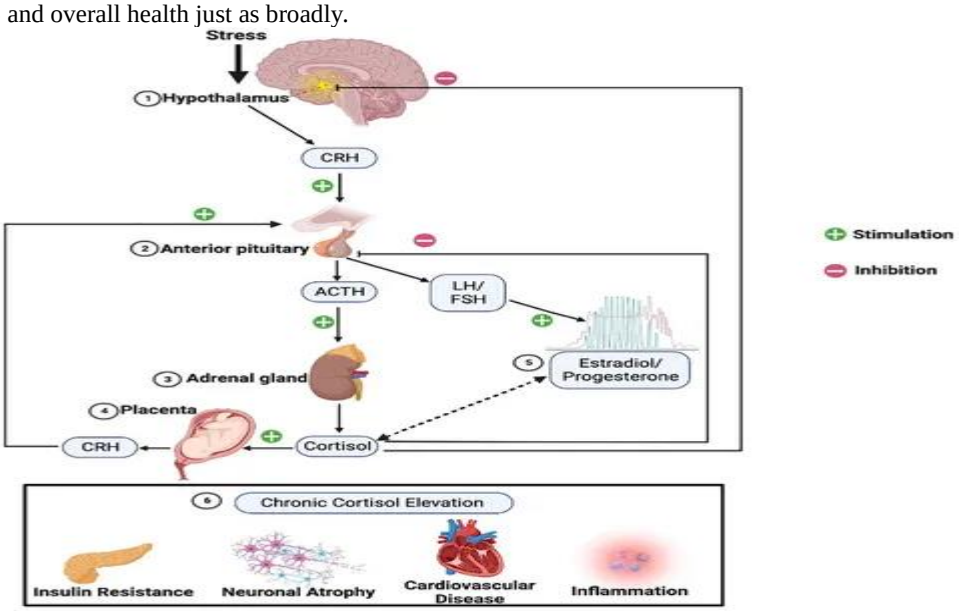

Figure 1. Dysregulation of the HPA axis and its downstream physiological consequences are linked to chronic stress. CRH - corticotropin-releasing hormone; ACTH- adrenocorticotropic hormone. (Hantsoo et al., 2023)

Altered neurotransmission (GABA, serotonin, melatonin)

This elaborate scientific drawing depicts a central neural network, surrounded by four separate panels, that describes in detail a specific type of pathway whereby key neurotransmitters interact to cause neuronal dysfunction and synaptic alteration, particularly in the context of neurodegenerative conditions, such as those involving amyloid beta (Ab). The diagram uses dotted lines to link specific regions in the central neural circuit to mechanistic explanations in the surrounding panels in Figure 2.

The first mechanism, which is labeled Panel A, describes glutamate-mediated neuronal dysfunction. It shows that the excess release of glutamate from neurons leads to neuroinflammation and the malfunction of astrocyte transporters, which are necessary for clearing glutamate from the synapse. This results in prolonged overstimulation of postsynaptic NMDA receptors and mGluR1 receptors, leading to excitotoxicity and ultimately synaptotoxicity, which causes damage to neural connections.

Panel B focuses on the topic of alterations mediated by the neurotransmitter GABA through its receptors, GIRK. Here, the neurotransmitter GABA is shown being released to activate the inhibitory GABAA receptor, which usually opens GIRK1 receptors to decrease neuronal activity. In this dysregulated state, however, the GABAA receptors are downregulated. At the same time, Kv1.4 potassium channels are up regulated, and this combination results in a decreased ability of the neuron to be inhibited, which potentially leads to pathological hyperexcitability.

Panel C presents the way in which acetylcholine signaling can be damaged by pathology to contribute to Ab pathology. Acetylcholine binding with nicotinic (nAChR) and muscarinic (M1R) receptors activates intracellular processes involving PKC. Dysregulation, particularly compromised M1R signaling, is associated with an enhanced neuronal response and a greater accumulation of Ab plaques.

Last, in Panel D, modifications mediated by adenosine are considered for Ab. Biochemical - Adenosine is produced from the breakdown of ATP, which acts on A2AR and A3R receptors. The diagram shows that substances such as caffeine and istradefylline can block these receptors, suggesting that modulation of adenosine signaling may affect neurotoxicity related to Ab. A legend at the bottom defines all the molecular symbols used throughout the figure 2, ensuring clarity to the viewer.

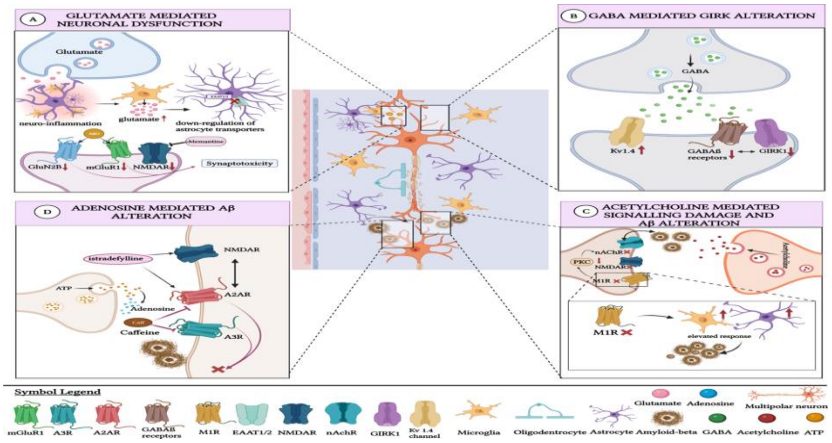

Figure 2. Multifaceted Neurotransmitter Dysregulation in Neurodegenerative Pathology: Glutamate, GABA, Acetylcholine, and Adenosine Pathways in Synaptic Dysfunction and Amyloid-Beta Alteration. Panel A. Glutamate-Mediated Neuronal Dysfunction; Panel B. GABA-Mediated GIRK Alteration; Panel C. Acetylcholine-Mediated Signalling Damage and Aβ Alteration; Panel D. Adenosine-Mediated Aβ Alteration. (Akyuz et al., 2025)

INFLAMMATORY PATHWAYS AND CYTOKINE INVOLVEMENT

Role of IL-6, TNF-α, and CRP in sleep disruption

Figure 3 illustrates a complex neuroimmune pathway through which inflammatory cytokines, particularly IL-1, interfere with sleep architecture, primarily by increasing non-rapid eye movement (NREM) sleep.

While IL-6, TNF-α, or CRP are not primary features of the image in labels, the main emphasis is on IL-1 as an essential mediator of inflammation, causing changes in sleep. This certainly provides a plausible model for the operation of other pro-inflammatory markers, such as IL-6 and TNF-α. The process begins with IL-1 acting on neurons in the DRN, which modulates the firing rate of serotonergic (5-HT) neurons and increases the inhibitory postsynaptic potential (IPSP), leading to increased NREM sleep. Concurrently, IL-1 also acts on the wake-promoting neurons, which are inhibited, thereby further tipping the balance in favor of sleep. The diagram also shows that IL-1 can stimulate the HPA axis, as it stimulates the release of CRH from the PVN, activating the secretion of ACTH and subsequently corticosteroids from the adrenal gland. This forms a feedback loop in the sense that the corticosteroids can, in turn, affect the immunological activity and possibly modulate IL-1. Although CRP is not drawn, it acts as a systemic indicator of this underlying inflammation. In contrast, IL-6 and TNF-alpha are known to be upstream regulators of both IL-1 production and HPA axis activation, indicating that they initiate or amplify the cascade illustrated. Thus, the image conceptualizes how an inflammatory state, signalled by cytokines, directly changes brain circuits involved in the sleep-wake cycle and recruits the stress response systems, such that disrupted, often fragmented, sleep patterns are observed, as is usually the case during illness or chronic inflammatory conditions.

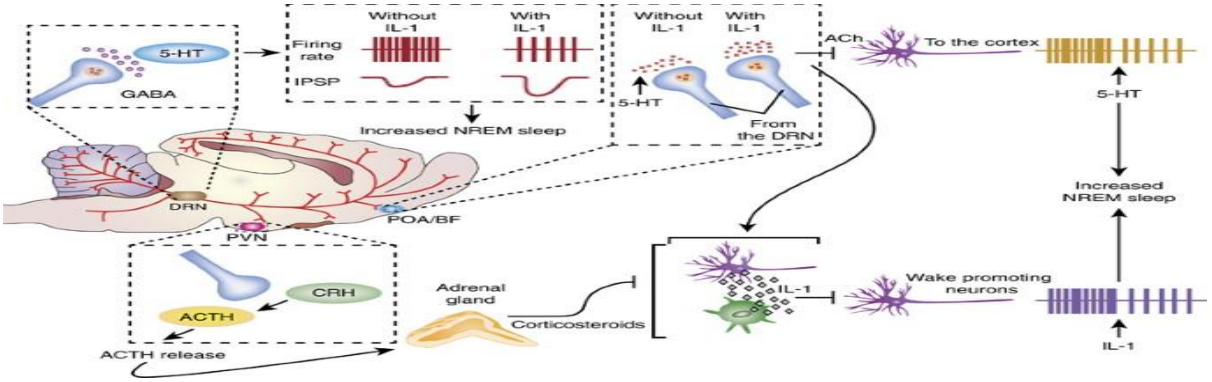

Figure 3. Interplay Between Serotonin, Acetylcholine, and Interleukin-1 Signaling in Regulating NREM Sleep: Modulation by the Dorsal Raphe Nucleus, Hypothalamus, and HPA Axis. DRN - dorsal raphe nucleus; HPA-hypothalamic-pituitary-adrenal; CRH - corticotropin-releasing hormone; PVN - paraventricular nucleus; ACTH - adrenocorticotropic hormone. (Irwin and Opp, 2017)

Challenges and Research Gaps

Despite the increasing awareness of the effects of sleep problems on chronic illnesses, challenges persist in establishing strong, generalizable evidence and translating it into clinical practice (St-Onge et al., 2025). The current literature has shown there are major methodological, demographic, and translational weaknesses that exist to achieving a complete understanding of how sleep interventions can be optimized within disease populations (Jean-Louis et al., 2021). This section describes the significant gaps and areas where research needs to be focused.

While numerous studies have been conducted to investigate the link between sleep disturbance and chronic illnesses, most of these are small-scale or cross-sectional in nature, and many are observational. Randomized Controlled Trials (RCTs) focused on specific chronic diseases, as well as extensive multicenter studies assessing the effects of targeted sleep interventions on these diseases, are scarce (Frank et al., 2017). Most of the existing RCTs are focused on primary insomnia or general populations and do not have external validity for patients suffering from comorbid conditions (e.g., diabetes, cancer, autoimmune disorders). For instance, there is limited evidence on the efficacy of CBT-I in treating insomnia in oncology or cardiovascular cohorts, primarily based on small sample sizes or pilot studies (Mijnster et al., 2022). Similarly, pharmacological studies examining agents such as melatonin agonists, orexin receptor antagonists, or sedating antidepressants rarely stratify outcomes by type of disease, disease stage, or medication interactions (Álamo et al., 2024).

This absence of disease-specific, large-scale trials is a barrier to developing standardized treatment protocols, which are sought due to the unique physiological and psychological demands of different chronic conditions (Pasanen et al., 2017). Future research efforts must prioritize providing adequate power and rigorously designed RCTs that include both objective measurements of sleep (such as polysomnography or actigraphy) and the evaluation of biomarkers of disease, to assess both symptomatic relief and long-term health outcomes (Whale et al., 2022).

Underrepresentation of Elderly and Multimorbid Populations

A significant shortcoming in the current literature on sleep is the understudying of older adults and patients with multiple chronic diseases. This demographic, though, makes up the majority of the people suffering from both chronic diseases and sleep disturbances (Calderón-Larrañaga et al., 2021). Aging is associated with changes in circadian rhythm, melatonin secretion, and sleep structure, leading to a high incidence of insomnia, fragmented sleep, and early morning awakening. Moreover, multimorbidity has also added to the complex interactions between disease pathophysiology, polypharmacy, and psychosocial factors that can aggravate sleep problems (Gorgoni & Gennaro, 2024).

Most clinical trials only include people up to a certain age or those with fewer diseases, so the results are not generalizable to the broader population. For example, an old, diabetic, hypertensive, and osteoarthritic patient, there are multiple concomitant factors contributing to inadequate sleep: from pain, neuropathy, side effects of medications, among others (Bloom et al., 2009). Interaction is currently poorly investigated. Furthermore, gender differences and socioeconomic variables are frequently overlooked, even though there is evidence that the sleep-related aspects of their disease disproportionately burden women and low-income populations in chronic illness (Ramos et al., 2023).

To overcome this gap, future research should use trial designs that are inclusive and representative of the actual heterogeneity of patient populations. Stratified analyses by age, comorbidity burden and medication profiles would be helpful for better understanding differential response to treatment and help identify personalized treatment strategies.

Limited Longitudinal Data on Safety, Efficacy, and Adherence

Another major challenge is the paucity of longitudinal studies of long-term safety, adherence and effectiveness of both pharmacological and psychological approaches to treating sleep disturbances in chronic illnesses (Harvey et al., 2014). Most current research is short-term, typically lasting four to twelve weeks, and provides limited information about sustained therapeutic outcomes or long-term adverse effects.

Pharmacological agents (Benzodiazepines and non-benzodiazepine hypnotics), though effective in the short term, create a concern about tolerance and misuse, dependence, and cognitive impairment, especially for the elderly and medically fragile population (Bowen & Larson, 1993). Similarly, long-term adherence to non-pharmacological management such as CBT-I, mindfulness, or relaxation therapies is often less than ideal because of motivational barriers, poor access to trained therapists, and competing demands of disease management (Hughes, 2023).

Moreover, there is scanty integration of digital sleep interventions (i.e. telehealth-based CBT-I or mobile sleep tracking tools) into chronic care models, resulting in a gap in knowledge about their feasibility and long-term compliance. Future research should extend the follow-up period, systematically evaluate the relative importance of adherence determinants, and integrate safety monitoring to ensure that the benefits of sleep interventions outweigh the potential risks associated with the techniques in complex patient populations (Matthews et al., 2013).

Integration of Sleep Therapy within Chronic Care Models

Despite increasing evidence of the bidirectional link between sleep and the progression of chronic disease, sleep management remains an underprioritized aspect of chronic disease management. Current disease management programs, such as those for diabetes, cancer survivorship, or cardiovascular rehabilitation, rarely include the structured identification, evaluation, and treatment of sleep as standard practice (Gupta et al., 2022). This fragmentation stems from a lack of interdisciplinary work on sleep medicine between sleep researchers, psychologists, and primary care providers.

Furthermore, healthcare systems are often not standardized in terms of screening tools and referral algorithms for identifying and administering sleep problems within the chronic disease clinics. For example, when managing diabetes, many parameters are routinely checked, such as blood sugar levels and lipid profiles, but sleep quality or duration is rarely assessed (Chaput & Shiau, 2019). Similarly, the care pathways in oncology have a significant focus on tumor control and pain management. Still, sleep-related issues are often ignored or put down to the psychological stress the person is under.

To address this gap, integrative models of care are necessary, incorporating sleep evaluation and management into the routine assessment of chronic disease. Collaborative care models that integrate behavioral health professionals, sleep experts and disease management can experience early identification and holistic management (Pittman et al., 2024). Moreover, digital health diagnosis and teleconsultation innovations such as apps can be used to expand the integration of sleep therapy into the care of comorbid illnesses on a large scale.

Need for Mechanistic Studies Linking Psychological and Biological Pathways

Although the link between sleeping problems and chronic illness outcomes is well established, the specific mechanisms for this relationship are still poorly understood. Most current studies provide descriptions of correlations but not causal pathways, leaving significant mechanistic issues unresolved (Engert & Besedovsky, 2025). The complex interplay between psychological stress, neuroendocrine regulation, and immune system activation requires further investigation.

The disruption of sleep due to chronic sleep deprivation has emerged as a mounting body of evidence that can affect our immune system by increasing the body's level of pro-inflammatory cytokines like interleukin-6 (IL-6) and tumor necrosis factor-alpha (TNF-alpha), which in turn promote and worsen disease processes in diseases such as diabetes, atherosclerosis, and autoimmune diseases (Zhang & Dhalla, 2024). Simultaneously, psychological factors, including anxiety, depression, and stress, related to illness, play a role in raising the hypothalamic-pituitary-adrenal (HPA) axis and affecting cortisol rhythms, which affects the health of metabolism and cardiovascular function.

Mechanistic studies incorporating neuroimaging, molecular biomarkers, and behavioural evaluation are required to understand the convergence of psychological and biological pathways in the mediating role of sleep regarding disease. Such insights make it easier to design interventions to fit precision, i.e. individual neurobiological and psychosocial profiles, thereby ensuring optimal therapeutic efficacy.

Future Perspectives

The way sleep disturbance is managed in chronic illnesses is changing rapidly, shifting from generalized, symptom-based interventions to more tailored, data-driven, and interdisciplinary paradigms. As evidence accumulates on the biological and psychosocial bases of sleep dysregulation, the potential for the future of clinical management lies in combining precision medicine, digital therapeutic options, artificial intelligence (AI)-based monitoring, and cross-disciplinary collaboration to achieve long-term improvement and enhanced quality of life (Garbarino & Bragazzi, 2024).

Precision Medicine Approaches

Emerging trends in sleep medicine have become increasingly grounded in the concept of precision medicine, the goal of which is to personalize the therapeutic interventions according to an individual's genetic, epigenetic, and biomarker profile. Sleep disturbances are not a homogeneous phenomenon as in chronic diseases such as diabetes, cardiovascular disease and neurodegenerative diseases (Cortese, 2021). Sleep disturbances occur differently depending on genetic susceptibilities, inflammatory pathways and pharmacogenetic interactions. For example, polymorphisms in genes involved in the circadian rhythm (e.g. CLOCK, PER3, BMAL1) or in neurotransmitter activity (e.g. GABRA1, HTR2A) have been identified as differential risk factors for insomnia vulnerability and differential responses to hypnotic agents (Lind & Gehrman, 2016). Similarly, genetic differences that influence drug metabolism (e.g., CYP450 isoenzymes) can impact the efficacy and safety of drugs commonly used to treat sleep disorders, such as benzodiazepines and melatonin receptor agonists (Krystal & Prather, 2019).

Incorporation of biomarker-based diagnostics can further aid in refining therapeutic decisions. Example: In the case of autoimmune and metabolic diseases, markers of inflammation such as C-reactive protein (CRP), interleukin-6 (IL-6), and tumor necrosis factor-alpha (TNF-α) have been found to have strong links with sleep fragmentation and fatigue (Engert & Besedovsky, 2025). Biomarkers of circadian alteration in neurodegenerative diseases, neuroimaging biomarkers, particularly alterations in the connectivity of the thalamus or hypothalamus. In the future, these parameters may help guide both pharmacological and behavioral interventions, enabling clinicians to predict which patients respond best to melatonin supplementation, CBT-I, or a combination of both (Moderie & Boivin, 2024). Thus, precision sleep medicine holds the potential for a paradigm shift in sleep medicine, transitioning from a reactive to a proactive model of care focused on prevention and individual treatment optimization.

Digital Therapeutics and AI-Based Sleep Monitoring

Digital health technologies are revolutionizing sleep medicine by introducing non-invasive, scalable tools for diagnosing, intervening in, and longitudinally monitoring sleep disorders. Wearable devices, sleep trackers (such as smartphones), and home polysomnography can be used to continuously record physiological variables, including heart rate variability, actigraphy, body temperature, and respiratory patterns (Zambotti et al., 2023). These data streams, when parsed through AI algorithms to identify inanimate biases in an individual's sleep structure, can detect deviations from normal before clinical symptoms appear in the patient. Such proactive monitoring is particularly relevant in chronic diseases, such as diabetes or Parkinson's disease, in which sleep disturbances develop gradually, allowing for early behavioral or pharmacological adjustments (Boe et al., 2019).

AI-powered platforms are also used for personalizing behavioral therapies. For instance, machine learning models can assess how patients are following their treatment, how they may be feeling, or even environmental factors that could inform the real-time adaptation of CBT-I modules (Nelson et al., 2025). Several digital therapeutics, such as Somryst or Sleepio, have already proven effective in treating chronic insomnia. Future development of these tools could integrate the use of information from biosensors along with predictive analytics in order to personalize the delivery of therapy (Gkintoni et al., 2025). In addition, telemedicine integration helps to ensure continuity of care for patients in remote or underserved areas, thereby minimizing barriers to specialized sleep management.

In research contexts, AI is likely to contribute to advances in understanding the relationships between sleep and disease by mining massive datasets that contain a combination of clinical, genetic, and behavioral variables (Gamel & Talaat, 2023). The new therapeutic targets in the context of sleep disruption are thus deep learning models that can reveal new patterns of association between sleep parameters and either metabolic or neuroinflammatory parameters. Nonetheless, the ethical provision of the data of patients, transparency of algorithms, and equal access to those technologies remain pressing issues that need to be addressed through regulatory bodies and clinician education (Bandyopadhyay & Goldstein, 2022). In the next decade, the interaction between AI and the expertise of medical doctors could change the essence of sleep medicine from a static, episodic, one-time attack to a continuous, adaptive process.

Cross-Disciplinary Collaboration

Sleep disturbances in chronic illnesses are inherently multifactorial, interwoven with neurobiology, endocrinology, immunology, and psychosocial factors. Consequently, effective management requires the collaboration of clinicians, psychologists, pharmacologists, and data scientists within an everyday decision-making context (Shehab et al., 2025). Such an integrative interdisciplinary approach is the way to ensure that therapeutic planning aims to address both the biological substrates and the behavioral determinants of disordered sleep.

Clinicians play a pivotal role in identifying comorbidities and adjusting pharmacotherapy to reduce the number of iatrogenic factors provoking sleep disturbance such as corticosteroids or beta-blockers (Stoppe, 2022). The contribution of the psychologists involves, to some degree, evidence-based behavioral interventions such as CBT-I, relaxation training and mindfulness-based stress reduction (MBSR) which targets cognitive distortions and maladaptive sleep habits (Walker et al., 2022). Pharmacologists as well as neuroscientists, in turn, are essential for designing new drug agents, such as orexin antagonists. These agents modulate the function of the neurotransmitter GABA or anti-inflammatory agents with fewer side effects, which selectively modulate sleep architecture (Kron et al., 2023).

Furthermore, collaboration through research programs between clinicians specialized in sleep and bioinformaticians can enable multi-omic analyses, which will allow systems-level insights into how sleep is related to metabolic and immune networks (Wei et al., 2022). Interdisciplinary sleep clinics, which combine expertise in neurology, psychiatry, endocrinology, and behavioral science, are an ideal model for patient management. These centers can also serve as centers for patient education and lifestyle modification, focusing on diet, physical activity, circadian hygiene, and pharmacological care (Falkai et al., 2022). Ultimately, such cross-disciplinary cooperation leads to a more integrated patient-centered approach in the management of sleep medicine in chronic illness.

Emphasis on Long-Term Outcomes and Quality-of-Life Measures

Historically, attention in sleep research has focused on short-term treatment improvements, such as reduced sleep latency and nocturnal awakenings. However, as chronic illnesses are typically long-duration and span years or decades, the focus will need to be on understanding the effects of chronic sleep improvement on long-term health and quality of life (Zheng et al., 2024). Future studies should incorporate multidimensional endpoints that assess not only sleep metrics but also cognitive performance, emotional resilience, social functioning, and overall life satisfaction (Chung et al., 2021).

Longitudinal cohort studies are essential to understand how long-term adherence to behavioral interventions/optimum pharmacotherapy affects the course of disease. For example, sleep enhancement in patients with diabetes may improve glycemic control while reducing cardiovascular risk (Martyn-Nemeth et al., 2022). In patients with cancer, sleep enhancement may help them deal with fatigue and help them recover their immunity when they are undergoing chemotherapy. Moreover, patient-reported outcome measures (PROMs) should become an integral part of clinical trials in order to capture the subjective improvements which are often neglected by objective measures (Mentink et al., 2023). The inclusion of wearable-based digital phenotyping could produce continuous and real-world data on sleep-wake behavior, providing a more comprehensive picture of how people function daily.

Quality of life should also be considered from the perspective of socioeconomic and cultural factors that affect sleep health, particularly in resource-limited settings. Public health strategies focused on sleep hygiene education, stress management, and early screening for sleep disorders may serve an essential transformative role in the management of chronic diseases on a population level (Ramos et al., 2023). Ultimately, the purpose is not simply to normalize sleep patterns; rather, it is to advocate for lasting good health, aggression, and functional recovery through the accurate and helpful treatment of well-informed interventions.

CONCLUSION

The complex bi-directional link between chronic illness and disturbance in sleep requires a paradigm shift in the management of

these conditions. There is evidence to suggest that a poor sleep pattern is not merely a side effect, but a significant driving force in the progression of disease through neuroendocrine dysregulation and increased inflammatory processes. Although pharmacological agents provide short-term relief, long-term improvement requires the integration of non-pharmacological approaches, such as CBT-I, into routine care pathways. There are still significant gaps in knowledge, especially concerning long-term outcomes among complex, multimorbid populations and the mechanistic pathways between psychological stress, biological pathways, and their interactions. What will make future success meaningful is the adoption of precision medicine approaches using biomarkers and genetics, the use of AI-based digital tools for continuous patient monitoring and personalized therapy, and the establishment of real interdisciplinary collaboration between clinicians, psychologists, and data scientists. Ultimately, integrating full-spectrum sleep measurement and care into models of chronic disease nursing interventions is essential to maximize positive outcomes, not only in physiological measures, but also in patients' long-term ability to function, emotional strength, and overall quality of life.

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