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Sleep Disturbance as a Mediator Between Chronic Skin Diseases and Mental Health Disorders

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Abstract

Introduction: Chronic skin diseases impose a multidimensional burden that extends far beyond physical symptoms. Epidemiological evidence consistently reveals a disproportionately high prevalence of psychiatric comorbidities in dermatology patients, particularly anxiety and depression. Historically, the psychological distress observed in this population has been attributed primarily to the psychosocial impact of body image dissatisfaction. However, this perspective is increasingly viewed as incomplete. Emerging research suggests that persistent sleep disturbance acts as a physiological pathway linking cutaneous inflammation to mental health decline, yet its specific role as a functional mediator remains underappreciated.

Aim of the study: This review aims to evaluate the evidence supporting sleep disturbance as a key mediator linking cutaneous symptoms to mental health disorders.

Materials and method: A narrative literature review was conducted in the PubMed database up to the year 2025.

Conclusions: Evidence suggests that nocturnal pruritus cause severe sleep fragmentation and alter sleep architecture, particularly reducing rapid eye movement sleep. This sleep deprivation acts as a primary biological stressor, activating the HPA axis and upregulating pro-inflammatory cytokines, which are shared biomarkers for both cutaneous inflammation and major depressive disorder. Furthermore, sleep loss impairs emotional regulation and lowers the threshold for stress coping, creating a cycle where psychological distress exacerbates skin symptoms. Sleep disturbance is not merely a secondary symptom of skin disease but a critical mediator of psychiatric morbidity. Effective management of chronic skin diseases must include the assessment and restoration of sleep quality. Targeting sleep disruption - through itch control or behavioral interventions - may serve as a potent strategy to prevent the development of secondary mental health problems in dermatological patients.

Keywords: psychodermatology; sleep disturbance; brain-skin axis; pruritus; atopic dermatitis; psoriasis; chronic urticaria; mental health.

1. Introduction

Chronic skin diseases (CSD), such as atopic dermatitis, psoriasis and chronic spontaneuous urticaria, represent a complex group of inflammatory disorders characterized by immune dysregulation and cutaneous barrier defects. They impose a considerable problem that extends

far beyond physical disfigurement, affecting millions of individuals. While often perceived by the general public as cosmetic inconveniences, they are associated with significant impairment in quality of life. A substantial body of epidemiological evidence has established a robust link between dermatological conditions and psychiatric comorbidities. Large-scale cross-sectional studies indicate that patients with skin diseases exhibit a disproportionately high prevalence of anxiety, depression, and suicidal ideation compared to the general population [1, 2]. Consequently, the global burden of skin disease is immense. According to the Global Burden of Disease Study, skin conditions rank as the fourth leading cause of non-fatal disability worldwide, affecting nearly one-third of the global population at any given time [3]. Traditionally, this psychological morbidity has been attributed to the psychosocial stigma of visible disfigurement and social isolation of skin conditions. While valid, this explanation fails to fully account for the physiological mechanisms driving such severe mental health outcomes. From a pathophysiological perspective, chronic skin diseases arise from a complex interaction between genetic predisposition, environmental triggers, and immune system dysfunction. That factors lead to the upregulation of pro-imflammatory cytokines such as interleukins (IL) and tumor necrosis factor-alpha (TNF-α) [4]. Clinically, this inflammation manifests as persistent erythema, scaling, and, most notably, intractable pruritus or pain. These symptoms are not static; they fluctuate in intensity, often governed by circadian rhythms that exacerbate cutaneous inflammation during the nocturnal period [5, 6].

This highlights the critical, yet often underappreciated, role of sleep disturbance. Sleep is not merely a passive state of rest but a vital active process required for neuroendocrine regulation, memory consolidation, and emotional processing [7]. In patients with chronic skin diseases, the relentless "itch-scratch cycle" and nocturnal pain lead to severe sleep fragmentation and a reduction in rapid eye movement (REM) sleep [8]. This chronic sleep deprivation acts as a potent biological stressor, dysregulating the hypothalamic-pituitary-adrenal (HPA) axis and impairing the brain’s ability to regulate emotion [9].

The objective of this narrative review is to synthesize current literature regarding the role of sleep disturbance as a functional mediator between chronic skin disease and mental health problems. We will examine the pathophysiological mechanisms connecting skin symptoms to sleep architecture disruption and subsequent psychiatric decline, arguing that effective management of sleep is an indispensable strategy in modern psychodermatology.

2. The Dermatological Trigger: Why Skin Disrupts Sleep

The etiology of sleep disturbance in chronic skin diseases is multifactorial, transcending simple symptom perception. It stems from a convergence of dysregulated nociceptive pathways,

altered circadian rhythms, and impaired thermoregulation. Understanding these mechanisms is requisite for appreciating how cutaneous pathology translates into systemic sleep fragmentation.

2.1 The Neurobiology of Pruritus and Nocturnal Exacerbation

While acute itch is largely histamine-mediated, the chronic pruritus characterizing CSDs involves complex, histamine-independent pathways that render traditional antihistamines largely ineffective for sedation-independent sleep improvement. The transmission of pruritic stimuli is mediated by specific unmyelinated C-nerve fibers, which are functionally distinct from pain-transmitting fibers but interact intimately within the dorsal horn of the spinal cord [10]. In chronic inflammatory states, these nerve fibers undergo peripheral sensitization (sprouting) and central sensitization, lowering the threshold for stimuli perception. This reduction of competing sensory input is a process best elucidated by the gate control theory of pain originally proposed by Melzack and Wall [11]. During waking hours, the central nervous system is inundated with visual, auditory, and tactile stimuli that activate inhibitory interneurons in the dorsal horn of the spinal cord, effectively dampening pruritic signaling. As these external distractions recede during the pre-sleep period, this "sensory shielding" dissolves, allowing afferent signals from unmyelinated C-fibers to be transmitted to the somatosensory cortex with unchecked intensity [12]. Consequently, innocuous stimuli such as heat or tactile pressure from bedding can trigger intense pruritic episodes at night (alloknesis).

The phenomenon of “nocturnal pruritus" - the intensification of itch during the sleep phase - is also driven by temporal fluctuations in neuroendocrine and immune mediators. The nocturnal physiological nadir of plasma cortisol levels diminishes the body's endogenous anti-inflammatory protection precisely when pro-inflammatory cytokines, including IL-2, IL-8, and the pruritogenic "master cytokine" IL-31, reach their circadian peak [8]. This temporal mismatch creates a window of heightened vulnerability to inflammation. Furthermore, the nocturnal upregulation of substance P and calcitonin gene-related peptide (CGRP) amplifies neurogenic inflammation, perpetuating a cycle where scratching damages the epidermal barrier, induces further cytokine release, and fragments sleep architecture via repeated cortical arousals [13].

Concurrently, the circadian regulation of body temperature plays a pivotal role; the physiological necessity to lower core body temperature for sleep onset triggers cutaneous vasodilation. This increase in skin blood flow and temperature lowers the activation threshold of peripheral itch receptors and enhances the enzymatic activity of cutaneous pruritogens, creating a "perfect storm" of neuro-inflammatory activity that directly opposes the initiation of sleep [14].

2.2 Circadian Dysregulation

Beyond the immediate sensation of itch, the pathophysiology of chronic skin disease is linked to the dysregulation of the circadian system, regulated by "clock genes" (*CLOCK*, *BMAL1*, *PER1-3*) expressed in keratinocytes and fibroblasts [15]. These genes orchestrate critical nocturnal functions, including DNA repair, cell proliferation, and barrier permeability. In patients with skin diseases, cutaneous inflammation disrupts the expression of these clock genes, leading to a decoupling of the skin's circadian rhythm.

A critical consequence of this dysregulation involves Transepidermal Water Loss (TEWL) and thermoregulation. Physiologically, TEWL peaks at night to facilitate core body temperature cooling, a prerequisite for the initiation of slow-wave sleep. However, in atopic dermatitis and psoriatic skin, barrier defects cause excessive, unregulated nocturnal water loss, leading to severe xerosis (dryness) that exacerbates pruritus and fragments REM sleep [15, 16].

Healthy sleep architecture also relies on a precise hormonal interplay: cortisol levels drop in the evening to facilitate sleep onset, while melatonin rises to maintain sleep continuity [17]. In patients with inflammatory dermatoses, this rhythm is frequently inverted or blunted. Research indicates that the chronic stress of the condition leads to the release of corticotropin-releasing hormone (CRH) that directly stimulates mast cell degranulation, further linking psychological stress, circadian misalignment, and nocturnal exacerbation of skin disease [4, 18].

Furthermore, the immune system follows its own circadian rhythm, with pro-inflammatory cytokines such as IL-6 and tumor necrosis factor-alpha (TNF-α) naturally peaking during the nocturnal phase [12, 19]. In healthy individuals, this peak is sub-clinical; however, in patients with pre-existing cutaneous inflammation, this nocturnal surge acts as a biological amplifier, exacerbating symptoms precisely when the body is attempting to rest.

3. Epidemiology of Sleep Disturbance in Dermatology

The prevalence of sleep disturbance in chronic skin diseases is pervasive, yet its phenotypic expression varies significantly across dermatoses. While the overarching symptom of "poor sleep" is ubiquitous, the specific architecture of sleep loss, driven by the distinct immunobiological profiles of atopic dermatitis (AD), psoriasis, and chronic spontaneous urticaria (CSU), requires differential analysis.

3.1 Atopic Dermatitis

In AD, sleep impairment is the single most burdensome symptom after pruritus. Large-scale epidemiological evidence establishes a linear dose-response relationship between disease severity and sleep fragmentation. In a cross-sectional analysis of 2,893 U.S. adults, Silverberg et al. demonstrated that 40.7% reported 1 or more, 11.1% reported 3 to 4, and 9.5% patients with AD reported 5 to 7 nights of sleep disturbance [20]. The odds of sleep disruption increased concomitantly with disease severity; patients with severe AD (assessed via Patient-Oriented Eczema Measure scores) exhibited a markedly elevated risk of insomnia compared to those with mild disease. A case-control study by Fishbein et al. assessed sleep disturbances in children with moderate-to-severe AD using actigraphy and questionnaires, comparing them to healthy, age-matched controls [21]. The findings reveal that while total sleep duration is similar between groups, children with AD experience significantly more fragmented sleep, restless sleep, daytime sleepiness, difficulty falling back to sleep at night, and teacher-reported daytime sleepiness, which correlates strongly with disease severity.

3.2 Psoriasis

While AD-associated sleep loss is primarily itch-driven, psoriasis introduces a dual burden of pruritus and physical discomfort. Nowowiejska et al. conducted a questionnaire-based cross-sectional analysis revealing that mean PSQI (Pittsburgh Sleep Quality Index), risk of obstructive sleep apnea syndrome, and Restless Leg Syndrome severity of psoriatics were significantly higher than in controls Approximately 80% of psoriatic patients in this cohort were classified as poor sleepers. Crucially, sleep disruption in psoriasis is often exacerbated by comorbidities, specifically Psoriatic Arthritis (PsA). Joint pain creates a "positional insomnia," where physical immobility and pain upon movement prevent the settling necessary for sleep initiation, distinct from the pure pruritic arousal seen in AD.

3.3 Chronic Spontaneous Urticaria

Chronic spontaneous urticaria (CSU) represents a unique chronobiological challenge. Unlike the persistent plaques of psoriasis, CSU is characterized by fleeting, unpredictable wheals and angioedema that often surge nocturnally due to the circadian release of histamine and pro-inflammatory mediators [23]. Although visible skin damage may be temporary, the subjective burden of sleep loss in CSU often rivals or exceeds that of other dermatoses. The intense, burning sensation of urticarial itch, combined with the anxiety of potential angioedema (e.g.,

laryngeal swelling), induces a state of hyperarousal that prolongs sleep latency [24, 25]. Lee et al., in a study of patients with chronic pruritus (a category encompassing CSU phenotypes), found that patients with insomnia symptoms reported significantly higher median pruritus intensity (intensity of pruritus measured using the visual analog scale - VAS 7.0 vs. 5.0) than those without, confirming that the intensity of the sensory signal in urticarial states is directly proportional to sleep destruction [26].

3.4 Objective vs. Subjective Architecture

A critical inconsistence exists between patient perception and physiological reality. While subjective scales (PSQI, ISI) capture the psychological distress of insomnia, objective metrics reveal profound structural deficits. Mann et al. utilized polysomnography to demonstrate that patients with active skin disease exhibit not only prolonged sleep latency but also a significant reduction in Sleep Efficiency (SE) and an increase in Wake After Sleep Onset (WASO) [27]. Specifically, these patients show a deficit in Rapid Eye Movement (REM) sleep and N3 (deep sleep). This objective fragmentation suggests that even when patients report "sleeping," the quality of that sleep is non-restorative, lacking the deep-phase continuity required for neuroplasticity and emotional regulation [28].

4. The Mediation Hypothesis

The established comorbidity between chronic skin diseases and psychiatric disorders has traditionally been interpreted through a psychosocial lens, attributing depression and anxiety solely to the stigma of visible disfigurement. However, recent pathomechanical models challenge this direct causality, positing that sleep disturbance is not merely a concurrent symptom but the primary biological mediator linking cutaneous inflammation to mental health decline. This "mediation hypothesis" suggests that the physiological stress of sleep deprivation, rather than the skin disease itself, acts as the proximate driver of psychiatric morbidity.

4.1 Statistical and Genetic Evidence of Causality

Empirical validation of this hypothesis is provided by multivariate regression analyses that isolate sleep as a variable. Lee et al. conducted a cross-sectional analysis of patients with chronic pruritus, revealing a profound correlation: patients with moderate-to-severe pruritus demonstrated a 10.95-fold increased odds (OR 10.95; 95% CI 2.24–53.06) of developing depression [26]. Crucially, mediation analysis indicates that when sleep quality is controlled for, the direct statistical strength of the relationship between itch and depression is significantly attenuated, implying that pruritus precipitates depression primarily *via* sleep fragmentation.

To transcend observational correlations, recent genetic studies have employed Mendelian Randomization (MR) to infer causality. Budu-Aggrey et al., in a large-scale bidirectional MR study, confirmed a causal effect of atopic dermatitis on the risk of depression and anxiety, suggesting that this association is not confounded by environmental factors but rooted in shared biological susceptibility [29]. These findings reinforce the concept that the pathway from skin to mind is biologically hardwired, with sleep acting as a critical modifiable gatekeeper.

4.2 The "Common Inflammatory Soil" Hypothesis

The biological plausibility of sleep as a mediator lies in the concept of a shared inflammatory milieu. Irwin et al. have extensively documented that sleep disturbance functions as a potent physiological stressor, triggering the release of pro-inflammatory cytokines, specifically IL-6 and TNF-α, which are well-established biomarkers of both depression and systemic inflammation [30]. In dermatological patients, especially with AD or psoriasis, this creates a "double hit" phenomenon: the systemic inflammation driven by T-helper cells pathways is compounded by the sleep-loss-induced cytokine storm [31, 32]. This cumulative inflammatory burden crosses the blood-brain barrier, altering neurotransmitter metabolism (e.g., serotonin depletion) and impairing neuroplasticity. Thus, sleep loss converts a localized skin inflammation into a systemic neuro-immunological disorder.

4.3 The "Vicious Cycle" of Stress and Itch

This relationship forms a self-perpetuating "vicious cycle" mediated by the HPA axis. Arck et al. elucidated the brain-skin axis, demonstrating that stress - whether psychological or induced by sleep loss - stimulates the release of neuropeptides such as Substance P (SP) from cutaneous nerve endings [4]. These mediators directly induce mast cell degranulation, releasing histamine and tryptase, which intensifies pruritus and promotes neurogenic inflammation. Consequently, sleep loss induced by itch generates systemic stress, which in turn lowers the itch threshold via the brain-skin axis, leading to further sleep loss. Breaking this cycle requires therapeutic interventions that target this central feedback loop rather than cutaneous symptoms alone.

5. Therapeutic Implications and Management

Accepting sleep disturbance as a pivotal mediator between cutaneous inflammation and psychiatric morbidity requires a paradigm shift in management. Therapeutic strategies must move beyond surface-level symptom control to target the neuro-immune drivers of nocturnal

arousal. Evidence suggests that restoring sleep architecture through targeted biologic therapy and specific psychopharmacological interventions can disrupt the trajectory towards depression.

5.1 Biologic Therapy as Sleep Rescue

The advent of biologic agents targeting the Th2 pathway has provided compelling evidence that sleep restoration is achievable through cytokine blockade. In the pivotal phase 3 SOLO 1 and SOLO 2 trials, Simpson et al. evaluated the efficacy of dupilumab (a monoclonal antibody inhibiting IL-4 and IL-13 signaling) in moderate-to-severe atopic dermatitis [33]. Beyond cutaneous clearance, dupilumab demonstrated a rapid and profound reduction in sleep disruption. Patients treated with dupilumab reported significantly greater improvement in the Peak Pruritus Numerical Rating Scale (NRS) and sleep loss scores compared to placebo as early as week 2 ($p<0.001$). This rapid onset suggests that inhibiting the proximal mediators of itch directly attenuates the nocturnal sensory bombardment that fragments sleep.

Long-term data further validate the durability of this effect. The LIBERTY AD CHRONOS trial assessed the administration of dupilumab with concomitant topical corticosteroids over 52 weeks [34]. The study confirmed that the improvement of sleep disturbance was sustained throughout the one-year period, compared to placebo. These findings indicate that effectively dampening the inflammatory drive not only resolves skin lesions but functionally restores the quality of sleep, potentially lowering the risk of secondary psychiatric comorbidities.

5.2 Psychopharmacological Interventions: Beyond Antihistamines

For patients with nocturnal pruritus unresponsive to standard dermatologic therapy, psychoactive agents offer a strategic "off-label" utility. While first-generation antihistamines are frequently prescribed for sedation, their efficacy is limited by tachyphylaxis and a lack of direct antipruritic effect in non-histaminergic itch [35]. In contrast, Hundley and Yosipovitch identified mirtazapine, a tetracyclic antidepressant, as a potent therapeutic option [36]. Through its unique mechanism as a noradrenergic and specific serotonergic antidepressant (NaSSA), mirtazapine exerts high-affinity antagonism at H1 (histamine) and 5-HT2 (serotonin) receptors. In a pilot study, low-dose mirtazapine (15 mg at night) significantly reduced nocturnal itch and sleep latency in patients with chronic pruritus. This dual action - sedative/antipruritic and antidepressant - makes it an ideal agent to address the bidirectional mediation identified in this review, simultaneously targeting the symptom (itch) and the consequence (depression). Additionally, melatonin supplementation has been proposed not only for its chronobiological regulation but also for its anti-inflammatory and antioxidant properties, which may mitigate the nocturnal oxidative stress in the skin [37-39].

6. Conclusions

The compendium of evidence presented in this review necessitates a fundamental recalibration of how chronic skin diseases (CSD) are conceptualized in clinical practice. The traditional dermatological model, which views psychiatric comorbidities as a direct emotional reaction to visible disfigurement, is demonstrably incomplete. Instead, the data support a mediation model suggesting that sleep disturbance acts as a critical biological bridge connecting cutaneous inflammation to mental health decline.

We have elucidated that the "itch-scratch-insomnia" cycle is not merely a behavioral phenomenon but a complex neuro-immuno-endocrine cascade. The nocturnal surge in pro-inflammatory cytokines (IL-31, IL-6), coinciding with circadian cortisol nadirs and thermoregulatory dysfunction, dismantles sleep architecture. This chronic fragmentation establishes a shared physiological vulnerability, often termed a "common inflammatory soil." In this state, sleep loss drives systemic inflammation and HPA axis dysregulation, directly triggering anxiety and depression. Statistical modeling, reinforced by Mendelian Randomization studies, confirms that when sleep quality is restored, the direct link between skin severity and psychiatric morbidity is significantly attenuated.

Consequently, the management of sleep in dermatology is no longer elective; it is a therapeutic imperative. The current clinical inertia, where sleep is often relegated to a secondary quality-of-life metric, must be overcome. Routine interrogation of sleep quality using validated tools such as the PSQI or ISI should be as standard as the assessment of BSA (Body Surface Area). Furthermore, the proven efficacy of biologic agents and targeted psychopharmacology in restoring sleep architecture demonstrates that the pathway to psychiatric remission in dermatology often runs through the night. Future longitudinal research must now focus on whether early, aggressive sleep intervention can prevent the onset of the "dermatologic depression" phenotype entirely.

Disclosure

Author’s contribution

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Declaration of the use of generative AI and AI-assisted technologies in the writing process

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