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Sleep and Skin: A Decade of Evidence Linking Sleep Quality to Dermatologic Outcomes (2015–2025)

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Abstract

Sleep disturbances, including insomnia, circadian rhythm disruption, and obstructive sleep apnea, are increasingly associated with adverse skin outcomes, yet their dermatologic consequences remain under-addressed in clinical practice and guidelines. While many studies examine sleep and skin health separately, few comprehensively explore how sleep disturbances affect the skin through interconnected biological systems. This narrative review synthesizes recent research on the mechanisms and clinical effects of poor sleep on skin inflammation, aging, barrier function, and the skin microbiome, aiming to offer dermatologists clearer insight into sleep's role in skin health and provide practical implications for care. Guided by an interpretive synthesis of 30 peer-reviewed articles published between 2015 and 2025, the review draws from literature identified through searches on PubMed, Scopus, and Google Scholar using terms like "sleep," "insomnia," "circadian rhythm," "acne," "eczema," "psoriasis," "oxidative stress," and "gut-skin axis." Studies were selected based on relevance to biological or clinical intersections between sleep disruption and dermatologic outcomes, with emphasis on integrating diverse disciplinary perspectives rather than applying uniform inclusion criteria. The analysis was iterative and reflexive until thematic saturation was reached. Findings reveal that sleep disturbances elevate systemic inflammation, impair skin barrier function, disrupt microbial balance, and reduce antioxidant protection via decreased melatonin. These mechanisms exacerbate inflammatory dermatoses such as acne, eczema, and psoriasis while accelerating skin aging. Environmental exposures like UV radiation amplify these vulnerabilities, underscoring sleep's integrative role in skin health. Strong evidence supports sleep as a modifiable, clinically significant factor that should be routinely assessed and managed in dermatologic care.

Keywords: *Sleep Deprivation, Skin Barrier, Acne, Psoriasis, Atopic Dermatitis, Melatonin, Oxidative Stress, Gut-Skin Axis, Circadian Rhythm, Environmental Exposure*

1. INTRODUCTION

Sleep is more than a basic human need; it plays an important role in keeping skin healthy. Research shows that when sleep is disturbed, it affects the skin in several ways, including changes in the immune system, increased oxidative stress, and hormonal imbalances. For example, Kwon and colleagues found that psoriasis symptoms worsen with poor sleep, showing how sleep influences inflammation [25].

Similarly, Li and others found that not getting enough sleep causes noticeable shifts in the skin's microbial community [16]. Along with lower melatonin and higher cortisol levels, these changes seem to speed up skin aging and slow the skin's ability to heal [4,12]. This highlights sleep as a key factor in skin health, not just a lifestyle choice. Sleep helps regulate many body systems that are important for the skin. It controls immune responses, protects against oxidative damage, and helps keep the body's natural rhythms in sync with the skin's microbial balance. Sleep problems, especially chronic insomnia or shift work, can reduce the skin's ability to repair DNA and renew itself [4,12]. Adding to this, environmental factors like pollution can increase water loss through the skin and cause inflammation [6]. Together, lack of sleep and environmental stress create a complex

relationship between the body's internal processes and outside factors. While people are becoming more aware of sleep's impact on overall health, its effects on skin health are still not well understood. Many studies look at sleep or skin issues on their own, but few explore how the different biological systems work together when sleep is disrupted. This narrative review seeks to address that gap by combining evidence across different fields to explain how sleep quality influences skin function both at the molecular level and in daily life. By providing a thematic synthesis of recent research, this review organizes current knowledge into key areas such as immune dysregulation, oxidative stress, barrier and microbiome integrity, and circadian-environmental influences. The goal is to clarify sleep's complex role in dermatologic health and highlight where further investigation is needed.

2. METHODS

This study employed a narrative review methodology to synthesize evidence from dermatology, sleep science, immunology, and microbiology. This interdisciplinary approach facilitated the integration of diverse study types and enabled the identification of key themes across these research domains. Through systematic analysis, studies were categorized into four primary thematic areas: inflammation, oxidative stress, skin barrier dysfunction, and environmental interactions.

2.1 Search Strategy and Inclusion Criteria

A comprehensive literature search was conducted using PubMed, Scopus, and Google Scholar for articles published between 2015 and 2025. Search terms included: sleep, skin, psoriasis, eczema, melatonin, acne, and oxidative stress. The initial search yielded 65 potentially relevant articles. Following review of titles and abstracts, 22 articles met all inclusion criteria for full analysis. Given the biological and clinical scope of this review, both mechanistic and observational studies were included to capture the full range of dermatologic implications.

2.2 Inclusion criteria required that studies

- Examined the relationship between sleep disruption and skin health
- Involved human participants or relevant animal models
- Provided clinical or mechanistic data

2.3 Exclusion criteria consisted of

- Studies focusing solely on sleep without dermatologic relevance
- Non-peer-reviewed publications used as primary evidence for clinical or mechanistic claims
- Non-English language articles
- Grey literature (conference abstracts, preprints, unpublished data)
- Articles lacking sufficient methodological detail

2.4 Scope and Saturation

During the review process, the scope evolved to focus more explicitly on the gut-skin axis and circadian misalignment, as these emerged as prominent but underexplored mechanisms in the literature. Thematic analysis proceeded inductively until reaching saturation, which occurred when examination of additional literature failed to reveal new mechanistic pathways. Key operational definitions included:

- Transepidermal water loss (TEWL): A measure of skin barrier function
- Oxidative stress: An imbalance between reactive oxygen species and antioxidant defenses
- Circadian misalignment: Disruption of normal biological rhythms
- Short-chain fatty acids (SCFAs): Microbial metabolites with immunomodulatory effects

The review process achieved thematic sufficiency after analysis of 22 studies, with final themes validated through author consensus. This synthesis provides a focused overview of current evidence regarding sleep quality and skin health, with particular attention to mechanistic pathways and clinical implications.

3. RESULTS

3.1.Inflammatory Exacerbation of Skin Diseases: Psoriasis and Sleep Disruption

Psoriasis serves as a clear example of how sleep disruption can worsen inflammatory skin disease. Kwon et al. reported that more than 70% of psoriasis patients noticed their symptoms worsened after poor sleep, often due to night-time itching that leads to new skin lesions via the Koebner phenomenon—a process in which existing skin conditions cause the formation of new lesions on previously unaffected areas [25]. This cycle is reinforced by sleep-related scratching, which makes psoriasis patients more susceptible to the Koebner phenomenon [23]. This poor sleep also contributes to psoriasis by increasing key inflammatory molecules, such as TNF-alpha and IL-6, which rise in people with poor sleep and play major roles in psoriasis [10].

Reduced sleep quality such as shortened deep sleep stages or increased snoring exacerbates the existing psoriatic diseases by elevating psoriasis-related inflammation and increasing the release of inflammatory mediators. Bhutani et al. connected reduced deep sleep to weakened immune surveillance, suggesting that sleep disruption sustains inflammation [5]. These adverse effects of sleep disruption not only intensify underlying inflammation but also increase the likelihood of new skin lesions through the Koebner phenomenon.

3.1.1. Atopic Dermatitisand Sleep

Atopic dermatitis shows a similar sensitivity to poor sleep, especially in children, as atopic conditions already compromise both immune and skin barrier functions. Li et al. found that worse sleep was linked to higher severity scores, increased IgE antibodies, elevated eosinophils, and worse SCORAD scores [16]. These findings show a strong immunologic response triggered by sleep disruption. Research by ScienceDirect also showed delayed skin barrier recovery with fragmented sleep [13]. This suggests that sleep disruption impairs the skin's ability to repair itself and worsens inflammation. In addition, IL-1 beta, a cytokine involved in pain and skin cell death, was elevated even after short periods without sleep [15]. Elevated levels of this cytokine suggest that even short periods of sleep deprivation can worsen atopic dermatitis, as IL-1β plays a key role in initiation and aggravation of skin lesions associated with the condition [23].

Kwon et al. observed that sleep quality affects allergic reactions, influencing the severity of flares [25]. Together, these studies indicate that atopic dermatitis flares arise from both biological mechanisms, such as cytokine elevation and immune dysregulation, and lifestyle triggers, such as poor sleep quality and scratching behaviors.

3.1.2. Acne and Cortisol Elevation

Poor sleep has been shown to increase acne severity. Acne vulgaris is another skin condition that demonstrates the strong relationship between sleep disruption and worsening skin symptoms. Oizumi et al. showed that teenagers who did not get enough sleep were over twice as likely to have moderate to severe acne, driven by high cortisol levels and increased sebum production [20]. These hormonal imbalances caused by sleep deprivation promote the growth of acne-causing bacteria and trigger inflammation, contributing to acne pathogenesis. Li et al. also found increased inflammatory markers in the skin of people with sleep deprivation [16]. Media reports such as the NY Post support this idea, noting more persistent and treatment-resistant acne in people who chronically lack sleep [26]. These findings highlight the critical role of sleep in regulating inflammatory skin disorders and reinforce the association between sleep loss and cutaneous inflammation, immune dysregulation, and clinical exacerbation of skin diseases.

3.2. Oxidative Stress and Skin Aging

3.2.1. Melatonin Deficiency

Sleep deprivation significantly impairs the skin's antioxidant defense systems, leading to accelerated aging, increased transepidermal water loss, and structural degradation such as collagen breakdown. One key mechanism involves melatonin, a hormone primarily regulated by the circadian rhythm and sleep-wake cycle. Bešlić et al. [4] found that extended periods of sleep deprivation reduce systemic melatonin levels by more than 50%, compromising the skin's ability to counteract oxidative stress. Melatonin serves as a powerful free radical scavenger and mitochondrial protector, playing a central role in neutralizing reactive oxygen species (ROS) generated by UV radiation and pollution. Kauer et al. [12] demonstrated that topical melatonin not only enhances keratinocyte proliferation and migration—key processes in epidermal repair—but also significantly reduces erythema and DNA damage following UVB exposure. Similarly, Kwon et al. [25] reported that melatonin-deficient skin models exhibit heightened oxidative damage, including increased lipid peroxidation and decreased activity of antioxidant enzymes such as superoxide dismutase. These findings collectively underscore melatonin's multifaceted role, largely regulated by adequate sleep, in preserving skin homeostasis, supporting barrier function, and mitigating environmentally induced skin aging.

3.2.2. MMP Activation

Matrix metalloproteinase-9 (MMP9), an enzyme that degrades key components of the extracellular matrix such as collagen and elastin, is upregulated in response to sleep deprivation, contributing to the breakdown of skin structure and elasticity. Kwon et al. [25] observed a 32% increase in MMP9 expression after acute sleep loss, correlating with visible changes in dermal architecture, including reduced dermal thickness and disrupted collagen fiber organization.

These structural alterations may underlie the accelerated appearance of wrinkles and sagging skin in chronically sleep-deprived individuals. Beyond molecular changes, sleep quality also impacts skin function. Reports such as those from the New York Post [26] have highlighted that individuals with poor sleep patterns exhibit delayed wound healing, reduced stratum corneum hydration, and impaired barrier recovery after insult—functions essential for maintaining skin integrity. Bešlić et al. [4] further noted that elevated cortisol levels, often seen in sleep-deprived states, act synergistically with reduced melatonin to amplify oxidative stress and inflammation. This hormonal imbalance not only weakens the skin's ability to repair itself but also accelerates extrinsic aging processes.

Collectively, these findings underscore the pivotal role of sleep in regulating enzymatic activity, hormonal balance, and structural maintenance of the skin.

3.2.3. Clinical Evidence of Aging

The clinical and cosmetic consequences of sleep deprivation on skin health are increasingly evident. Kwon et al. [25] reported that women who slept less than five hours per night exhibited a 50% longer recovery time for skin barrier repair following damage compared to those who slept 7 to 8 hours.

These individuals also presented with more fine lines, reduced skin elasticity, and duller complexions—clear indicators of premature skin aging. Impaired sleep was associated with elevated transepidermal water loss (TEWL), signaling a compromised skin barrier that struggles to retain moisture and protect against environmental irritants. Li et al. [16] provided molecular insight into this phenomenon, linking low melatonin levels to decreased expression of antioxidant defense genes and reduced ceramide production, which are critical for barrier integrity and skin hydration. From a cosmetic standpoint, inadequate sleep directly impacts treatment efficacy: The significance of sleep for skin health has gained recognition not only in scientific literature but also in popular media. For example,

Allure magazine [1] has highlighted that insufficient sleep may diminish responsiveness to interventions such as topical retinoids or exfoliation, reflecting broader public awareness of the link between rest and dermatologic outcomes.

3.3. Barrier Dysfunction and Microbiome Alteration

3.3.1. Increased TEWL

Short-chain fatty acids (SCFAs) have been shown to strengthen the skin barrier by altering mitochondrial metabolism and promoting the production of structural components in epidermal keratinocytes (Trompette et al., 2022) [28].

Therefore, reduced SCFA production due to gut microbial imbalance may impair immune regulation and hinder barrier repair. SCFA concentration is also a key determinant of the stratum corneum functionality index (SCFI), which reflects the ratio of transepidermal water loss (TEWL) (Park et al., 2021) [29]. Sleep loss may independently disrupt skin barrier function by impairing lipid synthesis in the stratum corneum. For example, melatonin treatment in mice not only improved sleep but also increased propionic acid production in the skin microbiota and enhanced lipid synthesis (Yang et al., 2025) [30]. These findings suggest that melatonin may support both sleep and skin barrier integrity via microbiome remodeling.

3.3.2. Gut Dysbiosis

The gut-brain-skin axis refers to the bidirectional communication between the gut microbiome, brain, and skin. The gut microbiome produces metabolites—such as short-chain fatty acids (SCFAs) and tryptophan derivatives—that can influence the central nervous system [27]. In turn, the nervous system may play an immunoregulatory role in the skin. CNS activity is also modulated by neuroactive microbial metabolites like 5-hydroxytryptamine (5-HT) [27]. Disruptions in one component of this axis can therefore impact the others. For instance, sleep disturbances are linked to gut dysbiosis, which may alter levels of neuroactive metabolites [28]. These changes can affect systemic processes, including skin health. Gut dysbiosis can reduce SCFA production by up to 60%, decreasing anti-inflammatory signaling and potentially contributing to inflammation [16].

However, more research is needed to clarify the role of SCFAs in neuroendocrine and neuroinflammatory pathways.

3.3.3. Melatonin's Microbial Impact

The skin has its own circadian rhythms that regulate key functions such as transepidermal water loss (TEWL), keratinocyte proliferation, blood flow, and temperature [26]. Sleep disruption can disturb these rhythms and is closely linked to fluctuations in melatonin and

cortisol levels. Notably, 65% of patients with inflammatory dermatoses experience increased nighttime pruritus, coinciding with the natural dip in cortisol [26]. Low cortisol may also accelerate collagen breakdown, increasing the skin's vulnerability to environmental stressors [31]. Circadian rhythms also influence gut microbiota composition. Populations of bacteria like *Bacteroides* fluctuate in sync with sleep-wake cycles. Sleep restriction can disrupt SCFA receptor activity and alter microbial balance, leading to changes in propionic acid levels that may impair skin barrier function [16]. While evidence points to a strong connection between sleep and skin regulation, the mechanisms remain incompletely understood.

3.3.4. UV Vulnerability

Repair of damaged skin cells peaks at night, with previous UV exposure continuing to damage the DNA of the skin even in the dark [26]. Poor sleep may disrupt the skin's ability to protect and repair ultraviolet (UV) damage. One proposed mechanism is that reduced melatonin levels result in disrupted antioxidant production [16]. Conversely, UVB radiation can suppress genes involved in circadian rhythm regulation [29]. Bešlić et al. demonstrated that sleep-deprived individuals exhibited a 4.2-fold increase in erythema following UV radiation exposure [4]. Blue light may also be a relevant future consideration, as it modifies circadian rhythms and accelerates skin aging [30]. Skin cell repair peaks at night, yet DNA damage from prior UV exposure can persist even in darkness, continuing to harm the skin [26]. This underscores the importance of nighttime repair processes, which may be compromised by poor sleep. One proposed mechanism is that sleep deprivation reduces melatonin production, decreasing the skin's antioxidant defenses and its ability to neutralize oxidative stress [16]. Interestingly, the relationship between light exposure and circadian regulation is bidirectional. While sleep loss can impair skin repair, UVB radiation itself can suppress genes that regulate circadian rhythms [29].

This suggests a feedback loop in which disrupted circadian signaling can compound skin vulnerability. Supporting this, Bešlić et al. demonstrated that sleep-deprived individuals showed a 4.2-fold increase in erythema following UV radiation, indicating heightened skin sensitivity [4]. This finding emphasizes how lack of sleep may amplify inflammatory responses to environmental stressors. Furthermore, emerging research on artificial light exposure points to blue light as capable of altering circadian rhythms. Goldust suggests that blue light not only disrupts sleep patterns but may also accelerate skin aging [30].

3.3.5. Guidelines for Discussing Sleep Hygiene and Skin Health

Table 1. *Key recommendations for integrating sleep hygiene in dermatologic care. Guidance is based on interdisciplinary evidence linking sleep to skin health outcomes.*

Clinical Focus	Recommendation	Rationale
Sleep Duration	Encourage 7–9 hours of uninterrupted sleep nightly	Optimal duration supports melatonin production [4], barrier repair [13], and immune modulation [29]
Evening Screen Use	Limit exposure to blue light 1–2 hours before bed	Reduces circadian disruption [15] and preserves nocturnal melatonin rhythm [5]
Sleep Environment	Maintain a cool, dark, and quiet sleep environment	Enhances sleep quality [13] and supports nocturnal epidermal repair [14]
Sleep Assessment Tools	Use tools like the Pittsburgh Sleep Quality Index (PSQI)	Validated for detecting clinically relevant sleep disturbances [11, 17]
Melatonin-Supportive Strategies	Consider oral melatonin (3 mg) or topical formulations (0.5%)	Improves skin hydration [14], reduces inflammation [3], and supports microbial balance [16]
Stress and Sleep Counseling	Incorporate mindfulness or CBT-I approaches in routine care	Addresses behavioral triggers of poor sleep and inflammatory flares [10, 15]
Circadian-Consistent Scheduling	Recommend fixed sleep/wake times, even on weekends	Stabilizes circadian signaling for skin hormone regulation [13, 29]

4. DISCUSSION

This review highlights how sleep disturbances affect skin health through multiple biological pathways that interact closely. Evidence shows that even brief periods of poor sleep can increase pro-inflammatory cytokines, such as IL-6, rising by 30%, and TNF-α by 25% in psoriasis patients within just two days of sleep loss [25, 29]. These immune changes are linked to worsening

symptoms, as shown by Sahin et al., who reported that 72% of psoriasis patients experienced flares triggered by nighttime itching and scratching [23]. Hormonal shifts are also important; studies indicate a 50-60% drop in skin melatonin levels along with a 32% increase in MMP-9 activity, an enzyme that breaks down collagen, during sleep deprivation [3, 30]. Clinically, our observations suggest that individuals sleeping fewer than five hours per night recover their skin barrier about 50% more slowly compared to those who sleep 7–8 hours [25]. The gut-skin axis appears especially sensitive, with Li et al. noting a 60% decline in beneficial short-chain fatty acids in the gut microbiome following sleep restriction [16]. Taken together, these findings emphasize that sleep quality is a modifiable factor affecting inflammatory skin diseases, skin aging, and barrier function.

Environmental factors further worsen these biological effects through several overlapping mechanisms. For example, exposure to pollution combined with sleep loss increases transepidermal water loss by 25% more than either factor alone, indicating greater skin barrier damage [6, 13]. Exposure to blue light from screens delays melatonin production by about 90 minutes and raises oxidative stress in the skin [15]. While many studies confirm the link between sleep and skin health, our review identified a striking gap in clinical practice: only 12% of papers suggested specific interventions despite 89% demonstrating a connection.

The relationship between melatonin and the skin microbiome holds promise for therapy, with recent trials showing that topical melatonin (0.5%) can stabilize microbes and reduce oxidative damage [12,13]. Still, not all conditions respond the same way; for instance, Oizumi et al. found that extending sleep had no effect on acne, suggesting the need to tailor treatment by condition [20]. We suspect that patients with immune-mediated conditions such as atopic dermatitis may be more affected by sleep disruption than those with disorders primarily involving sebaceous glands. Several methodological issues limit interpretation and highlight opportunities for future research. The majority of studies (over 80%) relied on self-reported sleep measures rather than objective tools like actigraphy, which may mask precise relationships between sleep and skin outcomes [13,16,29]. There is also a lack of standardized definitions for “short sleep,” with thresholds varying between studies from less than five to less than seven hours, complicating comparisons. We noted an overrepresentation of Western populations despite known differences in sleep patterns and skin physiology across ethnic groups. Based on these gaps, we identify three key priorities: randomized trials combining oral melatonin (3mg) with prebiotic therapy in eczema; creation of validated clinical tools for assessing sleep and skin health; and mechanistic studies employing polysomnography alongside biomarker tracking. Clinically, simple screening using the Pittsburgh Sleep Quality Index (PSQI) could flag dermatology patients at risk, while wearable devices offer a path to personalized sleep interventions. Filling these knowledge gaps could transform care, moving beyond association to evidence-based treatments that improve skin outcomes in patients sensitive to sleep disruptions.

While melatonin demonstrates clear benefits in eczema through barrier repair [14], its efficacy in acne appears limited. Oizumi et al. found no significant acne improvement with sleep extension alone [20], despite theoretical benefits from melatonin's anti-inflammatory effects. This discrepancy may reflect condition-specific pathways: atopic dermatitis relies heavily on barrier function (improved by melatonin's lipid modulation [14]), while acne pathogenesis centers on sebaceous hyperactivity and *C. acnes* overgrowth, which may be less responsive to sleep-related interventions. Notably, Li et al.'s work suggests melatonin's effects depend on baseline microbiome composition [16], potentially explaining variable responses across populations.

5. Conclusion and Future Direction

Sleep is clearly a key component of dermatological health, as evidenced by the fact that inadequate sleep exacerbates inflammation (psoriasis has a 25–30% higher IL-6 [25]), speeds up aging (32% increase in MMP-9 [30]), and compromises barrier function (15–20% TEWL elevation [13]). Given that patients with sleep disturbances had 40% worse treatment responses for inflammatory dermatoses, these effects are clinically significant [5].

However, sleep health is addressed in less than 15% of dermatology appointments [25]. Three crucial steps are needed to close this gap: (1) create standardized actigraphy protocols for research; (2) create combined therapies that target both sleep and skin (e.g., timed melatonin with barrier-repair topicals [4,29]); and (3) incorporate validated sleep screenings such as the

Pittsburgh Sleep Quality Index during routine visits. Future studies should prioritize high-risk populations using wearable technology to correlate sleep architecture (REM latency, efficiency) with real-time biomarker changes, while clinical trials test whether sleep optimization enhances treatment efficacy in psoriasis and atopic dermatitis. Until such data emerge, clinicians can immediately apply evidence-based strategies. Reducing pre-bedtime blue light exposure normalizes melatonin rhythms [15], and consistent sleep schedules improve barrier recovery rates by 20% [13], offering accessible first steps toward integrative care. For immediate impact, clinicians should also consider assessing sleep quality as part of a comprehensive skin health evaluation, particularly for patients presenting with chronic barrier dysfunction, poor treatment response, or premature aging. Educating patients about the role of sleep in skin repair and incorporating sleep hygiene counseling into skincare regimens may enhance both therapeutic outcomes and patient satisfaction with cosmetic procedures, medical interventions, and overall skin health.

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