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Review

The Sleep–Skin Axis: Clinical Insights and Therapeutic Approaches for Inflammatory Dermatologic Conditions

Alana Sadur [1,*], Lucie Joerg [2], Amelia Stapleton Van Doren [2], Ellen T. Lee [1], Dia Shah [2], Aniket K. Asees [1] and Sonal Choudhary [1]

[1] Department of Dermatology, University of Pittsburgh Medical Center, Pittsburgh, PA 15206, USA; leee9@upmc.edu (E.T.L.); dhariwala@upmc.edu (A.K.A.); choudharys@upmc.edu (S.C.)
[2] Albany Medical College, Albany, NY 12208, USA; joergl@amc.edu (L.J.); staplea@amc.edu (A.S.V.D.); shahd2@amc.edu (D.S.)
* Correspondence: sadurak@upmc.edu

Abstract

Sleep is crucial to overall health and plays a significant role in skin function. While the circadian rhythm has been extensively researched for its impact on the body's optimal functioning, the skin also possesses an independent circadian system that serves many important functions. Sleep disruptions or deprivation can significantly affect skin conditions, by compromising the skin barrier and impairing processes such as collagen production, cellular repair, and wound healing. Given the commonality of sleep disturbances, it is crucial to understand the connection between sleep, circadian regulation, and skin health. This is particularly important in understudied populations, such as those with occupational sleep disruption and individuals with hormone-related conditions like PCOS and menopause. Bidirectional relationships have been established between sleep and several inflammatory skin conditions, including atopic dermatitis, psoriasis, rosacea, and hidradenitis suppurativa. While acne is influenced by sleep, the reverse relationship, how acne affects sleep quality, has not been well established. Chronic sleep disruption can increase cortisol levels and oxidative stress, both of which contribute to skin aging and the progression of autoimmune skin conditions, including systemic lupus erythematosus. As sleep is a modifiable risk factor, it is crucial to consider therapeutic options and interventions to prevent or alleviate skin conditions. This review discusses various therapeutic approaches, including melatonin, L-Theanine, Magnesium-L-threonate, Inositol, Cinnamomi cortex, nervous system regulation, and proper sleep hygiene. These therapeutic options have been studied for their impact on sleep, and importantly, several have been evaluated for their utility as adjuncts for treating skin conditions. Overall, the relationship between sleep and skin health is clear, and incorporating sleep-focused therapeutic interventions offers potential to improve both sleep quality and skin health in individuals with a variety of skin conditions.

Keywords: sleep; skin; circadian rhythm; inflammatory skin conditions; sleep deprivation; atopic dermatitis; psoriasis; rosacea; hidradenitis suppurativa; acne

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1. Introduction

Sleep is an integral part of life, essential to overall health, and the skin. While circadian timing is well-known, there is limited literature on the skin's circadian rhythm. All bodily functions are adapted to a 24 h cycle, and alignment of the circadian clocks in every organ is required for optimal functioning of the body [1]. The central circadian clock, which

lies within the suprachiasmatic nucleus (SCN) in the brain, was believed to control the peripheral clocks throughout the body [2]. This theory was later amended after studies demonstrated that peripheral circadian clock functioning remains intact after ablation or scarring of the SCN [2]. In alignment with the updated model, the skin has been shown to possess an independent circadian system [3–5], regulated by many independent clocks, reflecting the complexity of the organ [6]. These independent circadian clocks in the skin serve many functions and regulate multiple local processes [7], including transepidermal water loss, keratinocyte proliferation, and temperature regulation [8].

At the molecular level, transcription factors including Clock and Bmal1 induce expression of their inhibitors, Period and Cryptochrome, producing self-regulating 24 h rhythms in gene expression [3,6]. Then nuclear receptors, such as Ror and REVErb, create a transcriptional loop responsible for modulating the expression of Bmal1. These circadian clock transcription factors, which act at their respective regulatory sequences, result in rhythmic oscillations in the expression of many output genes [6,9]. Importantly, the immune and circadian systems are also closely linked. As such, inflammation in the body can disrupt not only local circadian clocks, but also the central, master clock [2]. This disruption may be due to inflammatory pathways and cytokines including nuclear factor-kappa B (NF-kB), tumor necrosis factor-alpha (TNF-α), interferon gamma (IFN-γ), interleukin-1 (IL-1), and lipopolysaccharide [2].

Sleep deeply affects every aspect of health, including the integrity and proper functioning of our skin. The purpose of skin is to establish and maintain a healthy skin barrier and even a single night of sleep deprivation can compromise its stability [10,11]. Sleep deprivation affects skin regeneration by impairing the body's activation of collagen production, cellular repair, growth factor release, keratinocyte proliferation, wound healing and skin permeability during sleep [8,12]. After sleep restriction of 25–50% of the typical 8 h sleep time, markers of inflammation, including IL-6 and TNF-α, are elevated [10,13]. One study found that individuals with lower self-reported sleep quality exhibited significantly greater levels of transepidermal water loss and reduced skin recovery compared to individuals with higher quality sleep, resulting in decreased skin integrity and slower recovery [14]. Sleep deprivation increases cortisol release through the hypothalamic–pituitary–adrenal axis and exacerbates conditions including psoriasis and eczema [12]. Women may be more sensitive to the inflammatory effects of sleep deprivation [13].

The role of sleep in aging and autoimmunity has been elucidated in many prior studies. Chronic sleep disruption refers to impaired sleep quantity, quality, or continuity, commonly observed in conditions such as insomnia and obstructive sleep apnea [15]. This form of sleep deprivation results in oxidative stress [16], which has been shown to accelerate cutaneous senescence, decreasing hydration and skin elasticity [17,18]. Preventing oxidative stress is key in delaying cutaneous aging [19]. Systemic lupus erythematosus, an autoimmune disease with involvement of the skin, has been associated with poorer sleep quality measured by the Pittsburgh Sleep Quality Index (PSQI) [20,21] and increased prevalence of sleep disorders, ranging from 55% to 85% [22].

Given the robust data demonstrating the critical role of sleep in skin homeostasis, the increasing prevalence of sleep deprivation is a concerning phenomenon. Understanding the connection between sleep, circadian regulation, and skin health is vital, as more than one-third of adults are not meeting the recommended seven hours of sleep per night [23,24].

In this paper, we discuss the mechanistic pathways linking circadian disruption to the following inflammatory skin conditions: atopic dermatitis (AD), acne, psoriasis, rosacea, and hidradenitis suppurativa (HS). These conditions were prioritized due to their significant impact on quality of life, clinical relevance, and more robust research surrounding these conditions and their potential link to circadian rhythm disruptions compared to other

inflammatory skin conditions. We also outline novel, targeted interventions that have been proven to address this skin health through sleep-related modifications.

2. Skin Conditions

2.1. Atopic Dermatitis

AD is the most common chronic inflammatory skin condition, affecting up to 30% of children and 10% of adults in the United States alone [25]. Through aberrations including increased immune system activity, skin barrier compromise, and psychosocial stress, dry, inflamed, and itchy skin are typical manifestations of this disease [26–29].

A multidirectional relationship between AD, psychological stress, and sleep disruption has been demonstrated, with sleep disorders being observed in up to 60% of patients with AD [30,31]. Intense pruritus in patients with AD disrupts sleep [32], and this disruption likely amplifies neuronal and inflammatory pathways [33]. These pathways involve communication between keratinocytes and immune cells, which release itch-causing substances and inflammatory mediators such as IL-4, IL-13, IL-31, TSLP (thymic stromal lymphopoietin), IL-33, and substance P. Histamine-independent C fibers express receptors for these pruritogens, which intensify the pruritus. Notably, TSLP and substance P activate Th2 immune cells, such as mast cells and eosinophils, which further contribute to the sensation of itch. Together, these amplified pathways drive the itch–scratch cycle seen in AD [33]. A large cohort study found impaired sleep quality in pediatric AD patients regardless of symptom severity [34]. Each participant received a sleep quality score (0–4), with one point assigned for each of the following reported in the past year: $\geq$1 nighttime awakening per night, morning awakenings, delayed sleep latency, and nightmares [34]. Additional research based on actigraphy measurements and polysomnography supports these conclusions, finding sleep alterations in approximately 60% of children with AD, and that children with AD have significantly longer sleep onset latency, increased sleep disruptions, and decreased sleep efficiency [35,36]. Supporting these findings, decreased melatonin levels have been measured in AD patients [30,36].

Many studies have investigated the role of cortisol in AD to further characterize the relationship between stress and AD. The circadian rhythm regulates cortisol levels, which are increased in the morning and decrease throughout the day [10]. This pattern, in addition to cortisol's proposed anti-inflammatory properties, may explain why many individuals with pruritic skin conditions experience increased pruritus at night [10,37]. Cortisol may be negatively correlated with disease activity and severity, with lower salivary cortisol levels in patients with severe AD compared to those with mild and moderate AD [29,38,39]. These studies illustrate the intricate relationship between sleep, cortisol, and its effects on AD.

2.2. Psoriasis

Psoriasis vulgaris is a chronic, inflammatory and autoimmune condition affecting approximately 2% of individuals globally [40,41]. Characterized by pruritic or painful erythematous plaques with silver scaling, this condition commonly affects the symmetrical extensor surfaces, scalp, and lumbosacral region [42]. The disease is driven by keratinocyte hyperproliferation, acanthosis, and an immune response involving Th-1 signaling and pro-inflammatory cytokines such as IL-2, IL-17, IL-23, IFN-γ, and TNF-α [42–44]. Its pathogenesis is multifactorial, influenced by genetic susceptibility, immune dysregulation, psychological factors, BMI, and environmental exposures [45,46].

Several human and animal studies [47–49] suggest that aberrant circadian rhythms contribute to psoriasis pathophysiology. Consistent with this, a large cohort study identified an increased risk of psoriasis among night-shift workers [50–52]. These individuals

are known to have chronic circadian misalignment, representing a mismatch between the endogenous circadian rhythm and external cues, as well as suppressed melatonin secretion [50–52]. Furthermore, psoriatic flares, particularly pruritus, follow a diurnal pattern that worsens in the evening and peaks at night, often leading to sleep disturbances in patients with psoriasis [53–55]. Pruritus is a key predictor of sleep disturbance, as demonstrated by a large cross-sectional study that found individuals with psoriasis slept one hour less than controls [56]. Other sleep disorders, such as obstructive sleep apnea (OSA), have been associated with nearly double the risk of psoriasis [57]. These findings underscore the critical role of sleep disturbances in the pathogenesis and exacerbation of psoriasis.

2.3. Acne

Acne is the most common dermatological condition in the United States, affecting 85% of adolescents and ranking as the eighth-most prevalent disease globally [58,59]. Acne is a stress-reactive condition typically presenting with the onset of pubertal hormonal changes as non-inflammatory open or closed comedones or inflammatory lesions, including pustules, papules, nodules, or cysts [60]. The multifactorial nature of acne's pathogenesis is highlighted by its extensive exposome. Extensive research has identified precipitating factors, including genetic predisposition, environmental exposures, local immune response, hormonal fluctuations, diet, stress, and sleep disruption [61–63].

Previous literature has established a correlation between the increased prevalence of acne and poor sleep quality according to the PSQI, characterized by patterns such as reduced sleep duration, prolonged sleep latency, and insomnia [64,65]. A 2015 survey study found that stressed individuals were more likely to experience fatigue upon waking ($p < 0.0001$) and had a higher likelihood of having acne (OR = 1.975; $p < 0.0001$) as either a cause or consequence of stress [66]. Impaired skin barriers due to poor sleep can weaken defenses against external stimuli and can induce a state of disrupted immune function, increasing susceptibility to conditions like acne [67].

Furthermore, disruption of the circadian rhythm has been recognized as a potential contributor to acne pathogenesis. A study on night-shift workers with circadian disruption found that poor sleep quality based on the PSQI was associated with a higher prevalence and severity of skin diseases, including acne [68]. Given the central role of sebaceous gland activity in acne development, circadian alterations may contribute to its pathogenesis. Sebaceous gland activity reaches its lowest levels overnight and peaks around midday [8,69,70], which suggests a crucial relationship between acne and the circadian rhythm. Although these findings demonstrate the impact of sleep on acne pathogenesis, there is mixed evidence on whether a bidirectional relationship exists between the two. The ability to elucidate this connection may be impacted by factors such as hormonal fluctuations and diet, which impact both circadian rhythm and acne pathogenesis, therefore supporting the need for further research in this area.

2.4. Rosacea

Rosacea is a chronic inflammatory skin disorder characterized by burning, erythema, and flushing [71], primarily affecting the central face [72]. Several studies have reported associations between sleep disorders and rosacea, with results suggesting an association between poor sleep quality, based on the PSQI, and the severity of rosacea [73]. A large retrospective cohort study of individuals with rosacea found a significantly higher prevalence of sleep disorders in patients with rosacea compared to controls [72]. Experimental mouse models have also shown that sleep deprivation aggravates the rosacea-like phenotype in mice, evidenced by increased pro-inflammatory substrate expression [73]. Another animal model showed that sleep disturbances can lead to immunological compromise [74], with

an increased release of inflammatory cytokines, including TNF-α, IL-1, and IL-6 [75], which may play a role in both the development and worsening of rosacea [71].

Interestingly, the classic symptom of a burning sensation in patients with rosacea may be exacerbated by poor sleep, as sleep plays a key role in temperature regulation and catecholamine release [74,75]. As such, poor sleep may result in temperature dysregulation and vasodilation of the face, further aggravating rosacea [71]. Similar to the other dermatologic conditions discussed in this paper, the aforementioned symptoms of rosacea also have the potential to negatively affect sleep, worsening this negative cycle between sleep and rosacea [71]. Nevertheless, additional research is needed to further characterize the relationship between sleep and rosacea.

2.5. Hidradenitis Suppurativa

HS, a painful, chronic, relapsing condition characterized by follicular occlusion in intertriginous areas of the body [76], has also been found to have a relationship with sleep quality. Similar to other inflammatory conditions, several studies have shown patients with HS experience worse sleep quality, based on the Athens Insomnia Scale and PSQI, as a consequence of disease-associated pruritus and pain [77–80]. In a claims data analysis study assessing the comorbidity burden of HS patients in the US, Kimball et al. found sleep–wake disorders including insomnia and hypersomnia, to be 1.5 times more prevalent among patients with HS compared to patients without the disease [81]. The comorbidity between OSA and HS further highlights the strong connection between sleep and the development of HS [77] (Figure 1, Table 1). While circadian targeted therapies hold promise, and evidence supports the efficacy of these therapies, such as melatonin, in AD and psoriasis, its role in HS warrants further investigation. While the body of HS research is increasing, further studies are necessary to solidify the link between HS symptomatology and circadian disruption.

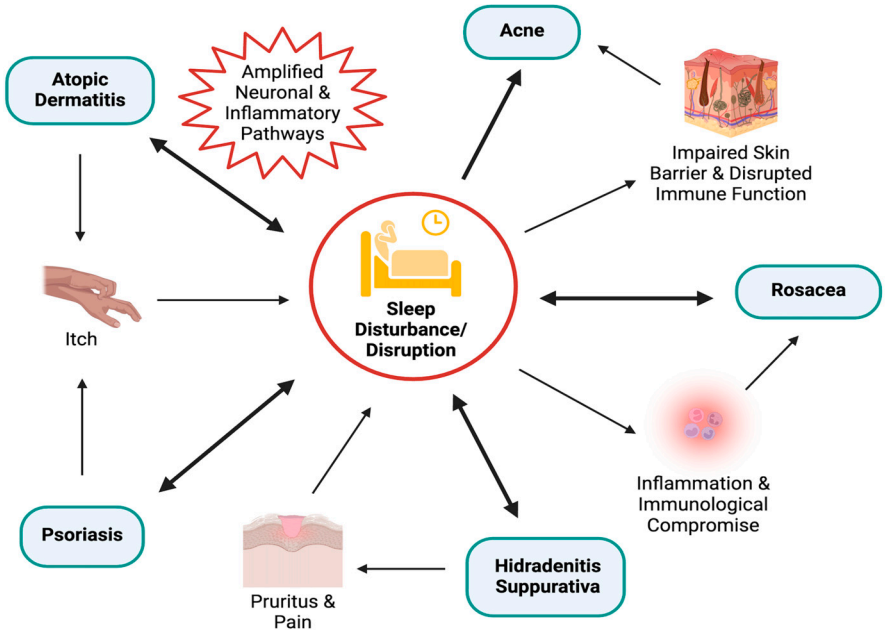


Figure 1. The relationship between sleep aberrations and inflammatory skin conditions. Graphical abstract summarizing key findings of the review. Data adapted from [29–31,33,42,47–49,53–55,64,65,67,71–75,77–81].

Table 1. Key findings on the relationship between circadian disruption and skin conditions.

Skin Condition	Key Findings	References
Atopic Dermatitis (AD)	Multidirectional relationship between AD, psychological stress, and sleep disruption, with sleep disorders observed in up to 60% of patients with AD.	[30,31]
	Intense pruritus in patients with AD disrupts sleep, this sleep disruption likely amplifies neuronal and inflammatory pathways.	[32,33]
	Impaired sleep quality in children with AD independent of symptom severity.	[34]
	Sleep alterations were identified in approximately 60% of children with AD.	[35]
	Children with AD have significantly longer sleep latency, increased sleep disruptions, and decreased sleep efficiency.	[36]
	Nocturnal pruritus may be mediated by circadian rhythm regulation of cortisol.	[37]
Psoriasis	Circadian dysregulation contributes to psoriasis pathophysiology	[47–49]
	An increased risk of psoriasis has been observed among night-shift workers, suggesting chronic circadian misalignment may be implicated in pathogenesis.	[50–52]
	Psoriatic flares, particularly pruritus, follow a diurnal pattern, often leading to sleep disturbances in patients with psoriasis.	[53–55]
	Pruritus is a key predictor of sleep disturbance, individuals with psoriasis slept one hour less than controls.	[56]
	Comorbid sleep disorders, such as obstructive sleep apnea (OSA) have been associated with nearly double the risk of psoriasis.	[57]
Acne	There is a correlation between the prevalence of acne and poor sleep quality.	[64,65]
	Psychosocial stress is associated with morning fatigue and occurrence of acne	[66]
	Impaired skin barriers due to poor sleep can weaken defenses against external stimuli, and induce a state of disrupted immune function, increasing susceptibility to conditions like acne.	[67]
	Poor sleep quality in night-shift workers with circadian disruption show a higher prevalence and severity of skin diseases, including acne.	[68]
	Sebaceous gland activity follows a circadian rhythm, peaking midday and declining overnight, suggesting rhythmic endocrine regulation may contribute to acne pathogenesis.	[68–70]
Rosacea	Individuals with rosacea were found to have a significantly higher prevalence of sleep disorders compared to controls.	[72]
	Sleep deprivation aggravates the rosacea-like phenotype in mice, evidenced by increased pro-inflammatory substrate expression.	[73]
	Sleep disturbances disrupt immune regulation, leading to increased release of inflammatory cytokines (TNF-α, IL-1, and IL-6) potentially playing a role in the development and worsening of rosacea.	[71,74,75]
	The classic symptom of a burning sensation in patients with rosacea may be exacerbated by poor sleep, as sleep plays a key role in temperature regulation and catecholamine release.	[74,75]
	Poor sleep may result in temperature dysregulation and vasodilation of the face, further aggravating rosacea.	[71]

Table 1. *Cont.*

Skin Condition	Key Findings	References
Hidradenitis Suppurativa (HS)	Patients with HS experience worse sleep quality as a consequence of disease-associated pruritus and pain.	[77–80]
	Sleep–wake disorders including insomnia and hypersomnia were found to be 1.5 times more prevalent among patients with HS.	[81]
	The comorbidity between OSA and HS highlights the strong connection between sleep and the development of HS.	[77]

3. Therapeutic Interventions

In the cases where sleep is a modifiable risk factor for patients, these therapeutic options and interventions can help reduce the development or exacerbation of skin conditions (Figure 2):

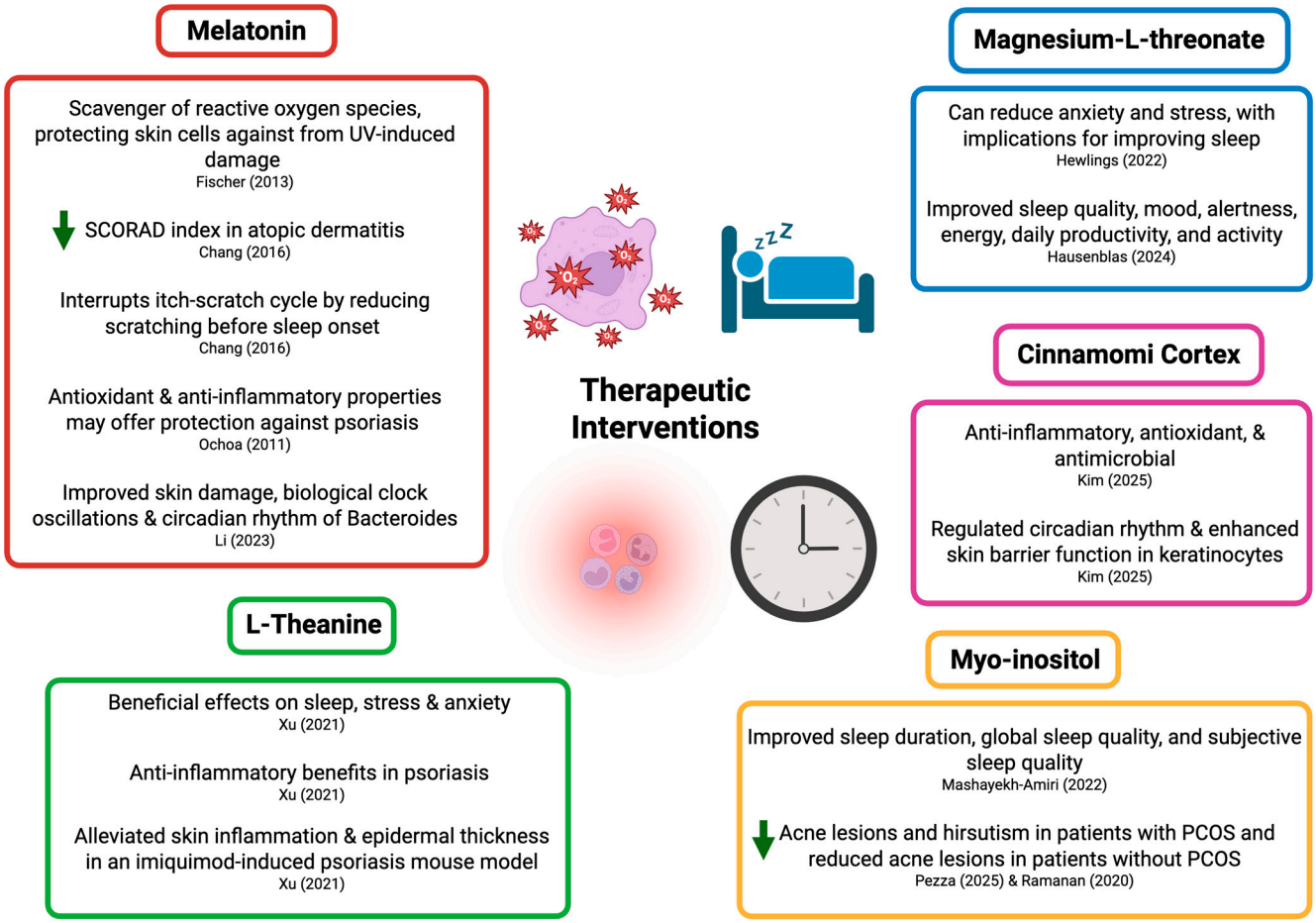


Figure 2. Therapeutic interventions for skin conditions affected by sleep [82–92].

3.1. Melatonin

Melatonin is a naturally produced antioxidant that impacts circadian rhythm and sleep, DNA damage repair, and immunomodulation. Acting as a scavenger of free radicals, melatonin neutralizes reactive oxygen species (ROS) that could otherwise damage skin cells, protecting against damage from ultraviolet (UV) light [82]. Melatonin as a potential treatment has been studied in allergic skin diseases, most extensively in patients with AD. A randomized controlled trial (RCT) found that melatonin supplementation in pediatric patients with AD resulted in reduction in SCORAD index by 9.1 compared to placebo (95%

CI, −13.7 to −4.6; $p < 0.001$), supporting its potential role in interrupting the itch–scratch cycle and improving skin inflammation and sleep latency [83]. Melatonin has also been investigated in other dermatologic conditions including vitiligo, chronic urticaria, melasma, and psoriasis [93]. Given the antioxidant and anti-inflammatory properties of melatonin and the reduced levels of melatonin found in psoriatic patients, melatonin supplementation may offer protection against psoriasis [48,84]. Loss of melatonin can also contribute to skin aging, as serum melatonin has been shown to decrease with age [94]. A sleep-restriction mouse model study found that supplementation improved skin damage, biological clock oscillations, and the circadian rhythm of Bacteroides, which had been dysregulated due to sleep restriction [85]. Melatonin demonstrates strong potential as an adjuvant given its benign safety profile, affordability, and accessibility.

3.2. L-Theanine

L-theanine (L-THE), an amino acid derived from green tea (Camellia sinensis) has been known to have beneficial effects on sleep, stress, and anxiety, which are recognized health benefits [86]. Importantly, L-THE has also been reported to have anti-inflammatory properties, which have been evaluated in psoriasis, given its inflammatory nature [86]. Despite promising immunomodulatory effects in preclinical models, its utility in dermatology remains underexplored. In an imiquimod-induced psoriasis mouse model, topical application of L-THE significantly alleviated skin inflammation and epidermal thickness through regulation and inhibition of genes involved in the IL-17A and NF-kB signaling pathways, such as CXCL2, CCL3, and CCL4 [86].—Overall this study demonstrated that L-THE inhibits IL-23 expression in dendritic cells and IL-17A expression in keratinocytes, two cytokines critical to the development of psoriasis [86]. This topical treatment offers strong potential to improve inflammatory skin conditions associated with sleep.

3.3. Magnesium-L-Threonate

While observational studies have suggested that magnesium improves sleep quality based on the PSQI, there are limited RCTs demonstrating this [95]. Magnesium-L-threonate (MgT) has higher brain bioavailability due to the inclusion of L-threonate. MgT can reduce anxiety and stress, which has implications for improving sleep [87]. In an RCT of 80 individuals with self-assessed sleep problems, supplementation with 1 g/day of MgT resulted in improved sleep quality, mood, alertness, energy, and daily productivity, and activity [88]. These outcomes were assessed based on standardized measures including the Leeds Sleep Evaluation Questionnaire, Insomnia Severity Index, Restorative Sleep Questionnaire, and Oura ring outputs. This holds promise for patients with comorbid sleep and skin conditions.

3.4. Inositol

Myo-inositol, a sugar alcohol responsible for regulating hormones including insulin, has also been considered as a supplement to improve sleep quality [96]. In an RCT of 60 pregnant women, supplementation with 2000 mg of myo-inositol improved sleep duration, global sleep quality, and subjective sleep quality, based on the PSQI, compared to placebo [89]. While studies have not specifically investigated the combined benefits of myo-inositol supplementation on both sleep and acne, research has consistently shown that inositol supplementation may reduce acne lesions in patients with polycystic ovary syndrome (PCOS) [90]. These findings suggest that myo-inositol may represent a promising dual-target therapy for hormonal acne and sleep dysregulation in this understudied group. A study in patients with PCOS found that supplementation with combined myo-inositol, microlipodispersed magnesium, and folic acid resulted in decreased acne and hirsutism, with no side effects [90]. Myo-inositol can act as a second messenger in insulin

signal transduction, improving insulin sensitivity and reducing hyperinsulinemia, a key driver of ovarian androgen overproduction in PCOS. Reduced circulating androgens subsequently diminish sebaceous gland activity and keratinocyte proliferation, both implicated in acne pathogenesis. This may potentially explain inositol's clinical utility in treating PCOS-associated acne [97].

While most research on inositol supplementation has been focused on patients with PCOS, another study investigated the efficacy of Tracnil™, a myo-inositol, folic acid, and vitamin D3 combination supplement, in menstruation-age, overweight women with normal hormone levels [91]. This single-arm study found a reduction in acne-related lesions in 8 weeks when women took Tracnil™ [91]. With little to no side effects reported in these studies, inositol supplementation appears to be a promising supplement to improve both sleep quality and dermatologic conditions like acne.

3.5. Cinnamomi Cortex

Novel therapeutic options are consistently being investigated as methods to improve sleep with potential to improve skin conditions. Cinnamomi cortex, an anti-inflammatory, antioxidant, and antimicrobial agent, has been implicated in regulating circadian rhythms, with recent research finding that its specific components epicatechin and linalool regulated circadian rhythm and enhanced skin barrier function in keratinocytes [92]. As a novel botanical therapeutic, it warrants further study for its role in enhancing sleep quality and barrier function.

3.6. Nervous System Regulation

Through regulation of the nervous system, specifically, reduction in the sympathetic "fight-or-flight" response, there is potential to improve sleep. Studies have suggested an inverse relationship between cortisol, a "stress hormone", and melatonin, further supporting the utility of stress-reduction in improving sleep [98]. Mind–body therapies (MBTs), including biofeedback, habit reversal, cognitive behavioral therapy, meditation, hypnotherapy, guided imagery, and progressive muscle relaxation have been studied in stress-related skin diseases; however, they have not been extensively investigated for their efficacy in skin conditions affected by sleep [99]. In a review of MBTs as adjunctive treatments in skin conditions, Graubard et al. reported in patients with AD, hypnotherapy and muscle relaxation techniques resulted in statistically significant symptomatic relief of pruritus and sleep disturbances [99,100]. Overall, the state of the nervous system undoubtedly impacts the skin; however, further research is needed to elucidate the benefits of nervous system regulation on sleep and skin health.

3.7. Proper Sleep Hygiene

Sleep and skin health are intricately related as described in this paper. As such, targeting sleep hygiene as an intervention is pertinent to break the inflammatory skin disease and insomnia cycle. Evidence-based recommendations for improving sleep include having a regular bedtime routine, maintaining a consistent sleep/wake schedule, obtaining 7 to 9 h of sleep [101], exercising regularly [102], avoiding late afternoon/evening naps [103], light exposure near bedtime [104], caffeine intake beyond midday [105], alcohol consumption [106], unhealthy foods, and large and mixed-macronutrient meals near bedtime [107,108], practicing mindfulness techniques [109], and creating a cool [110], dark, and quiet sleeping environment [103].

4. Conclusions

The link between sleep and skin is complex and robust evidence highlights how sleep quality, circadian rhythms, and skin health are intricately intertwined. As the skin is

hypothesized to follow independent circadian rhythms, misalignment of these rhythms may impair essential processes, including maintaining the skin barrier, collagen production, and cellular repair.

Given the heterogeneity in research methods utilized by the studies represented in this paper, some papers included were based on self-reported sleep data. While a variety of validated instruments were used in these studies, there may be limitations in the associations observed between sleep quality and inflammatory skin diseases. With the complex, highly individualized nature of both sleep and inflammatory skin diseases, patients may perceive their conditions in different ways, thus leading to the potential introduction of subjective variability. Future research should aim to conduct large-scale, longitudinal studies to elucidate causal pathways linking circadian disruption, sleep, and skin disease. There is also a need for clinical trials investigating dual-targeted interventions that concurrently address dermatologic inflammation and sleep dysfunction.

Additionally, the scope of this review, resource constraints, and the need for consistent data interpretation necessitated the exclusion of non-English language studies. This may have introduced selection or regional bias that underrepresents findings from non-English-speaking populations where sleep and dermatologic conditions may be studied differently or more extensively. Recognizing this, future reviews should aim to include studies across multiple languages to ensure a more comprehensive and globally representative analysis.

While this review highlights promising sleep-targeted therapies for improving inflammatory skin conditions, it is important to consider the limitations of the underlying evidence. Some RCTs referenced, such as those investigating melatonin, magnesium-L-threonate, and myo-inositol, demonstrated positive outcomes but were limited by relatively small sample sizes, short intervention durations, and limited generalizability. Observational studies contributed valuable insights, particularly regarding associations between sleep disturbance and skin disease severity; however, they are inherently subject to bias, including self-reporting inaccuracies, misclassification, attrition, selection bias, and residual confounding. These factors should be considered when interpreting findings and underscore the need for future large-scale, longitudinal trials with standardized outcome measures.

Since sleep has been shown to have a bidirectional relationship with several inflammatory skin conditions including AD, psoriasis, rosacea, and HS, it is essential for providers to consider sleep as a potential contributing factor. It is important to note that the strength of evidence supporting this bidirectional relationship as well as the interventions discussed throughout this review vary. For example, there is robust data supporting the bidirectional relationship between AD and psoriasis with sleep; however, there is only preliminary evidence for acne, rosacea, and HS. Management and treatment of these skin conditions may be improved through evidence-based techniques to improve sleep quality including nervous system regulation and proper sleep hygiene. Therapeutic interventions such as melatonin, L-THE, MgT, and inositol supplementation also have the potential to enhance skin health by improving sleep quality (Table 2). While there is strong evidence supporting melatonin's ability to improve sleep in AD, there is only preliminary evidence supporting interventions such as L-THE, MgT, and inositol, highlighting the need for validation and further research to investigate their potential to improve sleep in specific inflammatory skin conditions.

Given the increasing prevalence of sleep aberrations and their effects on the skin, further research is critical to develop novel strategies and treatments to address sleep-associated skin conditions. By incorporating sleep-focused interventions in addition to traditional dermatological treatments, there is potential to improve both the quality of sleep and skin health of individuals with a variety of skin conditions.

Table 2. Therapeutic interventions for improving sleep quality with recommended dosages.

Intervention	Recommendations	Clinical Context
Melatonin	0.5–5 milligrams (mg) melatonin to improve sleep [111] 4 mg of melatonin taken 3 hours before sleep to improve total sleep time and reduce sleep onset latency in adults [112] 3 mg of melatonin to shorten sleep latency and improve SCORAD index in pediatric patients with AD [83]	General adult population with subjective sleep disturbances Adults with insomnia Pediatric patients with AD
L-Theanine (L-THE)	200 mg to reduce sleep latency and disturbance [113] and improve sleep quality [114]	Healthy adults, demonstrated efficacy in preclinical models of psoriasis
Magnesium-L-threonate (MgT)	1 g/day of MgT to improve sleep quality, particularly deep/REM sleep stages [88]	Adults with subjective sleep disturbances and mood symptoms
Myo-inositol	2000 mg of myo-inositol to improve global sleep quality, subjective sleep quality, and sleep duration [89].	Pregnant women, potential benefit in PCOS-associated acne and sleep disruption
Cinnamomi Cortex	Further research is necessary to understand the potential sleep-related benefits of cinnamomi cortex and appropriate dosage.	Theoretical relevance to circadian regulation and skin barrier function

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Abbreviations

The following abbreviations are used in this manuscript: SCN, suprachiasmatic nucleus; NF-kB, nuclear factor-kappa B; TNF-α, tumor necrosis factor-alpha; IFN-γ, interferon gamma; IL, interleukin; AD, atopic dermatitis; HS, hidradenitis suppurativa; OSA, obstructive sleep apnea; ROS, reactive oxygen species; RCT, randomized controlled trial; L-THE, L-theanine; MgT, Magnesium-L-threonate; PCOS, polycystic ovarian syndrome; MBTs, mind–body therapies.

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