

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

Gaity Wahab, BS¹, Chelsea Barrows, BS², Kailey Bae, BA³, Danny Lee, MD⁴, Carlene Radix, MD, MPH¹

1. Ross University School of Medicine, Barbados, 2. Stritch School of Medicine at Loyola University of Chicago, 3. California Health Sciences University College of Osteopathic Medicine, 4. University of California, San Francisco,



Introduction

Sleep is a critical yet under-recognized factor in skin health. Disruptions such as insomnia, shift work, and circadian misalignment drive inflammation, barrier dysfunction, delayed healing, and premature aging, largely via cortisol elevation, melatonin reduction, immune shifts, and microbiome imbalance.¹⁻³ Despite mounting evidence, sleep is rarely assessed in dermatologic care.⁴

Objective

To synthesize findings from 2015–2025 on how sleep quality impacts skin health, highlighting molecular mechanisms like inflammation, oxidative stress, barrier dysfunction, and circadian disruption and to explore potential clinical interventions.

Methodology

A narrative review of 30 peer-reviewed studies (2015–2025) was conducted via PubMed, Scopus, and Google Scholar using terms like “sleep,” “skin,” “melatonin,” and “circadian disruption.”

- Inclusion: human or animal studies offering clinical or mechanistic insight into sleep-related dermatologic outcomes.
- Exclusion: non-dermatologic, non-English, non-peer-reviewed, or editorial sources.
- Thematic analysis revealed four domains: inflammation, oxidative stress, barrier dysfunction, and environmental amplification, with attention to the gut–skin axis and circadian misalignment.

Scan for reference



Results

1. Inflammation:

- Sleep loss increases IL-6 and TNF- α , triggering psoriasis and eczema.
- Atopic dermatitis worsens via \uparrow IgE, IL-1 β , and eosinophils.^{9 10}

2. Oxidative Stress:

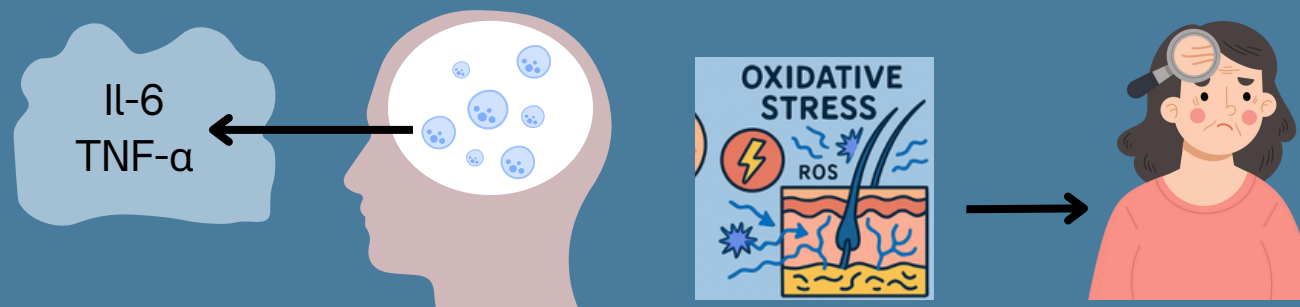
- Melatonin loss \rightarrow \uparrow ROS, \uparrow MMP-9 (matrix metalloproteinase-9) \rightarrow Collagen degradation, increased TEWL (transepidermal water loss), and wrinkles.^{11 12 13}

3. Barrier & Microbiome Disruption:

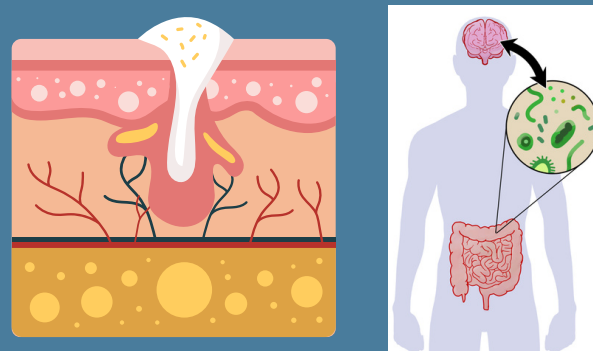
- \downarrow Ceramides, \uparrow TEWL; gut-skin axis shifts via reduced SCFAs (short-chain fatty acids).^{14 15 16}

4. Environmental Amplification:

- Sleep-deprived skin shows 4 \times UV erythema; blue light alters repair genes.^{17 18 19}

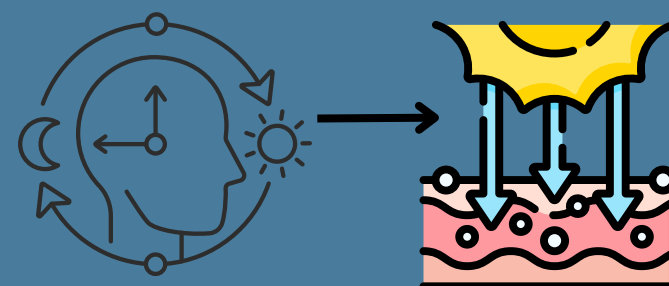


“Poor sleep raises cytokines in just two nights.”



“Sleep loss depletes ceramides and disrupts flora.”

“Melatonin loss increases ROS by 80%.”



“Circadian disruption amplifies UV & digital light damage.”

Future Directions: Innovations in Sleep & Skin

- **Wearables:** Smart sleep tracking in dermatology care
- **AI Analytics:** Predict flares via sleep–skin pattern learning
- **Melatonin Topicals:** Timed-release skincare for circadian recovery
- **Chronotherapy:** Align phototherapy and Rx timing with biological rhythms
- **Precision Models:** Customize care with PSQI + microbiome data



Melatonin restores antioxidant rhythm



PSQI detects chronic circadian disruption

Conclusion

Sleep loss is a modifiable risk factor in dermatology. Incorporating tools like the PSQI and wearables into routine care may reduce flares, enhance skin repair, and delay aging. Melatonin use and reducing screen time are simple, evidence-based steps. Future trials should explore sleep-optimized interventions and biomarkers for personalized treatment.