

Sleep, Gut Microbiota and the Hair Follicle: A Narrative Review and Integrative Model of the Sleep Gut Follicle Axis

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Abstract

Sleep is an active physiological process that organizes immunity, metabolism and tissue regeneration, and is now recognized as a regulator of the gut microbiota. This narrative review examines the evidence that connects sleep, the intestinal microbiota and the health of the hair follicle, and proposes an integrative model of a sleep gut follicle axis. Short, fragmented or misaligned sleep is associated with changes in microbial composition and diurnal rhythmicity, impaired intestinal barrier function and low grade systemic inflammation, and the microbiota, in turn, influences sleep, the circadian clock and the stress response. Each of these intermediate signals has a documented target in the hair follicle: the human follicle senses bacterial products through toll like receptor 4, its growth is inhibited by interleukin 1, tumour necrosis factor alpha and interleukin 6, its stem cells depend on regulatory T cells that are shaped by microbial metabolites, it synthesizes and responds to melatonin, it operates a local equivalent of the hypothalamic pituitary adrenal axis, and it runs its own circadian clock, which is disturbed by night shift work. Clinical studies associate sleep disorders with alopecia areata and with the severity of androgenetic alopecia, and Mendelian randomization suggests that specific gut taxa influence the risk of both conditions. No study has yet measured sleep, microbiota and hair outcomes in the same individuals; the axis is therefore presented as a mechanistic model with explicit levels of evidence and testable predictions. Sleep deserves to be assessed in trichological practice as a modifiable determinant of follicular health.

Keywords: Sleep; Gastrointestinal Microbiome; Hair Follicle; Circadian Rhythm; Alopecia

Introduction

Sleep represents much more than a period of passive rest. It is an active physiological state, essential for systemic homeostasis, memory consolidation, metabolic regulation and immune balance, and recent research has added a new dimension to this list: the preservation of a stable intestinal microbial ecosystem^{1,2}. The relationship between sleep and the gut microbiota is bidirectional and dynamic. Sleep, through its duration, quality and regularity, modulates the composition and function of the microbiota, while the microbiota, through metabolites, neurotransmitter precursors and immune signals, influences sleep architecture and the circadian system¹⁻⁴.

For the trichologist, this dialogue matters for a simple reason. The hair follicle is one of the most metabolically active and most finely regulated mini organs of the human body, and it is exquisitely sensitive to inflammation, to stress hormones and to timing signals^{5,6}. If poor sleep disturbs the gut, and if the gut communicates with the rest of the body through inflammatory, immune, endocrine and circadian signals, then the follicle sits at the end of a chain that begins in the bedroom. Every clinician who has observed a patient

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whose shedding worsened during months of night shifts, insomnia or caregiving recognizes this chain intuitively; what has been missing is its biological description.

The aim of this review is to integrate the evidence that connects sleep, the intestinal microbiota and hair follicle biology, to organize it into a coherent model with explicit levels of evidence, and to translate it into clinical reasoning and a research agenda.

Methodology

This narrative review was built from two earlier unpublished texts by the author on sleep and the microbiome, written for the book *Microbiome and Hair Health*, which were reorganized and fully updated. A targeted search was performed in PubMed, Scopus and Google Scholar, using combinations of the keywords "sleep", "sleep deprivation", "circadian rhythm", "gut microbiota", "intestinal permeability", "melatonin", "regulatory T cells", "hair follicle", "alopecia areata", "androgenetic alopecia" and "telogen effluvium". Studies were included when they (1) evaluated the effect of sleep or circadian disruption on the gut microbiota or intestinal barrier, (2) described a mechanism by which microbial, inflammatory, endocrine or circadian signals act on the hair follicle, or (3) reported clinical associations between sleep, microbiota and hair loss disorders. Human studies, *ex vivo* human hair follicle organ culture and mechanistic animal studies were considered and are identified as such in the text. Every reference was checked against the original publication, and each statement in this review was verified against the full text or the abstract of the article that supports it.

Findings

Sleep and the gut microbiota: a bidirectional relationship

The intestinal microbiota is not static over the twenty four hours of the day. In mice and in humans, its composition and metabolic activity oscillate diurnally, largely driven by the rhythm of feeding and fasting, and the ablation of host clock components or the induction of jet lag disrupts these oscillations and produces dysbiosis; more than that, microbiota from jet lagged mice and humans transferred glucose intolerance and obesity to germ free mice⁷. The oscillations themselves are functional: the daily rhythm of microbial localization and metabolite production programs the circadian epigenetic and transcriptional landscape of the host, in the intestine and at distant sites such as the liver⁸. Persistent jet lag, an obesogenic diet and clock gene deficiency dampen this rhythmicity, which can be rescued by time restricted feeding¹.

In humans, the first experimental evidence came from a randomized crossover study in nine normal weight young men, in whom two nights of partial sleep deprivation increased the Firmicutes to Bacteroidetes ratio, increased Coriobacteriaceae and Erysipelotrichaceae and reduced Tenericutes, together with a fall in insulin sensitivity, while beta diversity and faecal short chain fatty acids were unchanged⁹. The authors themselves described these changes as subtle, and it is worth remembering that short experiments capture only the first steps of a process that extends over years. Observational data point in the same direction: in 26 healthy men followed by actigraphy, total microbiome diversity correlated positively with sleep efficiency and total sleep time and negatively with wake after sleep onset, and positively with interleukin 6¹⁰. Studies of patients with insomnia, summarized in a recent review, describe reduced abundance of butyrate producers such as *Faecalibacterium* and *Roseburia*².

In sleep deprived mice, plasma norepinephrine increased and melatonin decreased, antioxidant capacity fell, proinflammatory cytokines rose, colonic goblet cells, MUC2 and tight junction proteins were reduced, and microbial diversity declined, with loss of

Akkermansia, *Bacteroides* and *Faecalibacterium* and expansion of the pathogen *Aeromonas*; melatonin supplementation reversed these changes¹¹. Severe sleep loss also causes accumulation of reactive oxygen species specifically in the gut of flies and mice, and, in flies, neutralizing these species in the intestine prevents the lethality of sleep deprivation¹². Sleep disruption reduces the expression of BMAL1, CRY1 and occludin in the intestinal epithelium, which compromises barrier integrity².

The relationship runs in both directions. Gut microbial metabolites influence central and hepatic clock gene expression and sleep duration, and sleep fragmentation and short sleep are associated with dysbiosis, possibly through activation of the hypothalamic pituitary adrenal axis¹. Specific genera such as *Lactobacillus* and *Bifidobacterium* contribute to the serotonergic and GABAergic signalling involved in sleep regulation, and probiotic, prebiotic and dietary interventions have shown effects on sleep quality, although results vary across strains, doses and populations¹⁻³.

From the gut to the circulation: barrier, endotoxemia and low grade inflammation

The intestinal barrier is the interface that converts a local dysbiosis into a systemic signal. Bacterial lipopolysaccharide that crosses a permeable epithelium reaches the circulation, and, in mice, a chronic two to threefold elevation of plasma lipopolysaccharide, a condition defined as metabolic endotoxemia, was sufficient to induce inflammation, weight gain and hepatic insulin resistance¹³. In 73 soldiers, a four day multistressor military exercise, in which sleep deprivation was likely although not quantified, increased intestinal permeability in parallel with changes in microbiota composition and metabolism¹⁴. The most direct human link between sleep, melatonin and the barrier comes from obstructive sleep apnoea: in 235 men evaluated by polysomnography, serum melatonin fell as apnoea severity increased while zonula occludens 1, lipopolysaccharide and C reactive protein increased, and melatonin statistically mediated about one third of the effect of the apnoea hypopnoea index on barrier and endotoxin markers¹⁵.

At the systemic level, a meta analysis of 72 studies showed that sleep disturbance is associated with higher circulating C reactive protein and interleukin 6, whereas experimental sleep deprivation did not consistently change these markers¹⁶; it is chronic poor sleep, more than a single bad night, that sustains this inflammatory tone. Such inflammatory signals do not remain in the bloodstream; they reach tissues, and the skin and its appendages are among the most responsive of them^{5,17}. Poor sleepers already show a measurable cutaneous phenotype: higher transepidermal water loss, slower recovery from ultraviolet erythema, and good sleepers showed 30% greater barrier recovery 72 hours after tape stripping¹⁸.

The hair follicle as a target of systemic inflammation

The human hair follicle is equipped to perceive this inflammatory environment. Its epithelium expresses toll like receptor 4, the receptor for lipopolysaccharide, together with toll like receptor 5 and inducible antimicrobial peptides, which means that the follicle can directly sense bacterial products¹⁹. The cytokines associated with poor sleep and endotoxemia are hostile to follicular growth: interleukin 1 alpha, interleukin 1 beta and tumour necrosis factor alpha inhibit the growth of isolated human hair follicles²⁰, and interleukin 6, which is secreted by balding dermal papilla cells in response to dihydrotestosterone, inhibits hair shaft elongation and matrix keratinocyte proliferation in organ cultured human follicles and induces premature catagen in mice²¹.

Systemic administration of lipopolysaccharide to mice produced a state resembling a cytokine storm, with increased interleukin 6 and neutrophilia, and accelerated the transition of hair follicles into telogen, establishing a model of telogen effluvium triggered

by acute systemic exposure to lipopolysaccharide²². In patients with alopecia areata, plasma claudin 3, a marker of intestinal barrier integrity, was higher than in controls and correlated with the severity of the disease measured by the SALT score²³.

The immune regulatory arm: microbial metabolites, regulatory T cells and follicular regeneration

Inflammation is only half of the immune story. The microbiota also produces signals that restrain the immune system, and these signals are directly relevant to hair regeneration. Butyrate produced by commensal bacteria induces the differentiation of colonic regulatory T cells²⁴, and butyrate and propionate promote the generation of regulatory T cells outside the intestine²⁵; short chain fatty acids, secondary bile acids and tryptophan metabolites are now recognized as regulators of every stage of the T cell lifecycle²⁶. In the skin, regulatory T cells localize preferentially to hair follicle stem cells and, through the Notch ligand Jagged 1, drive stem cell proliferation and differentiation; their depletion impairs the telogen to anagen transition and hair regeneration²⁷.

The gut and the follicle are connected through this regulatory arm. In a mouse model of atopic dermatitis like inflammation, a fermentable fibre rich diet increased gut derived short chain fatty acids, particularly butyrate, which strengthened the epidermal barrier by altering keratinocyte mitochondrial metabolism, a demonstration that intestinal metabolites reach the skin²⁸. Oral *Lactobacillus reuteri* shifted the follicles of aged mice into anagen and produced a thicker and more lustrous coat, an effect that depended on interleukin 10²⁹. Conversely, antibiotic induced dysbiosis with overgrowth of *Lactobacillus murinus* caused alopecia in mice fed a biotin deficient diet, because the overgrown bacterium consumed the available biotin³⁰; this is a causal demonstration that the composition of the gut microbiota can determine the fate of the hair coat. In humans, a randomized double blind study in 160 patients with alopecia areata found significant improvement of the SALT score with a topical product containing postbiotics and biomimetic peptides, without significant change in the placebo group³¹.

Tryptophan, serotonin and melatonin: a shared chemistry between gut, sleep and follicle

Tryptophan is the common substrate of serotonin and melatonin, and the gut is the main site where its fate is decided. Much of the body's serotonin is produced by enterochromaffin cells of the intestine, and indigenous spore forming bacteria promote its biosynthesis, which demonstrates that the microbiota regulates peripheral serotonin³². Serotonin is the precursor of melatonin, the hormone that signals darkness to the body, and, as shown above, sleep deprivation lowers plasma melatonin and melatonin restores the microbiota and the barrier in sleep deprived mice¹¹, while lower melatonin mediates barrier dysfunction in apnoea patients¹⁵.

The hair follicle speaks the same chemical language. Mouse and human scalp follicles synthesize melatonin at concentrations far above serum levels, production increases with norepinephrine, and, in murine skin, transcripts of the melatonin receptor MT2 and of the nuclear receptor ROR α fluctuate with the hair cycle, peaking in catagen³³. The follicle is therefore both an extrapineal source and a target of melatonin, which may regulate keratinocyte apoptosis and protect the bulb against oxidative stress³⁴. Clinically, a double blind randomized pilot trial in 40 women showed that topical melatonin 0.1% for six months increased the anagen hair rate in occipital hair of women with androgenetic alopecia and in frontal hair of women with diffuse alopecia, with plasma levels remaining within the physiological night peak³⁵.

Stress hormones: the HPA axis of the body and of the follicle

Sleep loss is a stressor for the endocrine system. After partial or total sleep deprivation, evening plasma cortisol increased by 37% and 45% respectively, and the onset of the quiescent period of cortisol secretion was delayed, suggesting impaired glucocorticoid feedback³⁶. The microbiota calibrates this response: germ free mice show an exaggerated adrenocorticotrophic hormone and corticosterone response to restraint stress, which is reversed by colonization with *Bifidobacterium infantis*³⁷.

The follicle responds to this endocrine signal at two levels. Systemically, corticosterone, the rodent equivalent of cortisol, acts on the dermal papilla to suppress the expression of growth arrest specific 6, which prolongs hair follicle stem cell quiescence and keeps follicles in an extended telogen under chronic stress³⁸. Locally, human scalp follicles display a functional equivalent of the hypothalamic pituitary adrenal axis: they respond to corticotropin releasing hormone by producing proopiomelanocortin derived peptides and secrete cortisol, while corticotropin releasing hormone itself modulates hair shaft elongation and catagen induction³⁹.

The circadian clock as the common conductor

If one element unifies sleep, microbiota and follicle, it is the circadian clock. Isolated human scalp follicles, removed from any central influence, maintain circadian expression of CLOCK, BMAL1 and Period1, and silencing BMAL1 or Period1 prolongs anagen, which establishes the peripheral clock as an integral component of the human hair cycle clock⁶. In mice, the local clock gates mitosis of matrix keratinocytes, so that follicular cell division follows a daily rhythm⁴⁰. The same clock that organizes the follicle also organizes the microbiota^{7,8}, and both depend on a regular alternation of light and darkness, feeding and fasting, sleep and wakefulness.

Human evidence shows that disturbed sleep timing reaches the follicular clock. In men working three or more consecutive night shifts, Period3 and Nr1d2 expression in beard hair follicle cells showed weaker 24 hour rhythmicity than in day workers or single night shift workers, and Period3 expression was lower than in day workers, although with substantial individual variation⁴¹. Long term deregulation of circadian rhythm in shift workers has also been reported to alter the regenerative properties of human skin and hair precursor cells⁴². It is worth remembering that reduced Period3 expression and later sleep onset have been described in androgenetic alopecia⁴³.

Clinical evidence: sleep, microbiota and hair loss disorders

The clinical literature closes the circle. A systematic review of 29 studies found that sleep disturbance is consistently elevated in alopecia areata, androgenetic alopecia, telogen effluvium and lichen planopilaris, although the overall quality of evidence is low to moderate and mostly cross sectional⁴³. In a Korean national cohort of 25,800 patients with sleep disorders and 129,000 controls, sleep disorders were an independent risk factor for alopecia areata, with an adjusted hazard ratio of 1.65, especially in individuals younger than 45 years⁴⁴, and a Taiwanese cohort described a bidirectional association between alopecia areata and sleep disorders⁴⁵. In 446 men, severe androgenetic alopecia was associated with total sleep time of six hours or less, a Pittsburgh Sleep Quality Index above 5 and a high risk of obstructive sleep apnoea⁴⁶.

On the microbial side, patients with alopecia universalis did not differ from controls in overall gut diversity, but a model based on *Parabacteroides distasonis* and the Clostridiales vadin BB60 group correctly classified 80% of participants⁴⁷. Mendelian randomization analyses, which reduce confounding and reverse causation, identified gut taxa with a causal association with alopecia areata⁴⁸ and with androgenetic alopecia⁴⁹. Increased

intestinal permeability and gut inflammation have been proposed as primers of the immune dysregulation of alopecia areata⁵⁰, a disease whose central event is the collapse of hair follicle immune privilege⁵¹.

Discussion and Conclusion

An integrative model of the sleep gut follicle axis

The evidence reviewed allows the construction of a model in which insufficient, fragmented or misaligned sleep acts on the follicle through four convergent routes (Figure 1, Table 1). The first is inflammatory: dysbiosis and barrier dysfunction release bacterial products and cytokines that the follicle recognizes and that shorten anagen. The second is immune regulatory: the loss of butyrate producers reduces the supply of metabolites that sustain regulatory T cells, which are themselves required for follicular stem cell activation. The third is neuroendocrine: sleep loss raises cortisol, a microbiota that is out of balance amplifies the stress response, and glucocorticoids hold follicular stem cells in quiescence, while the tryptophan, serotonin and melatonin pathway links intestinal metabolism to a hormone that the follicle both produces and uses. The fourth is chronobiological: the same clock that times sleep and microbial rhythms times the hair cycle, and night work demonstrably disturbs it in human follicles.

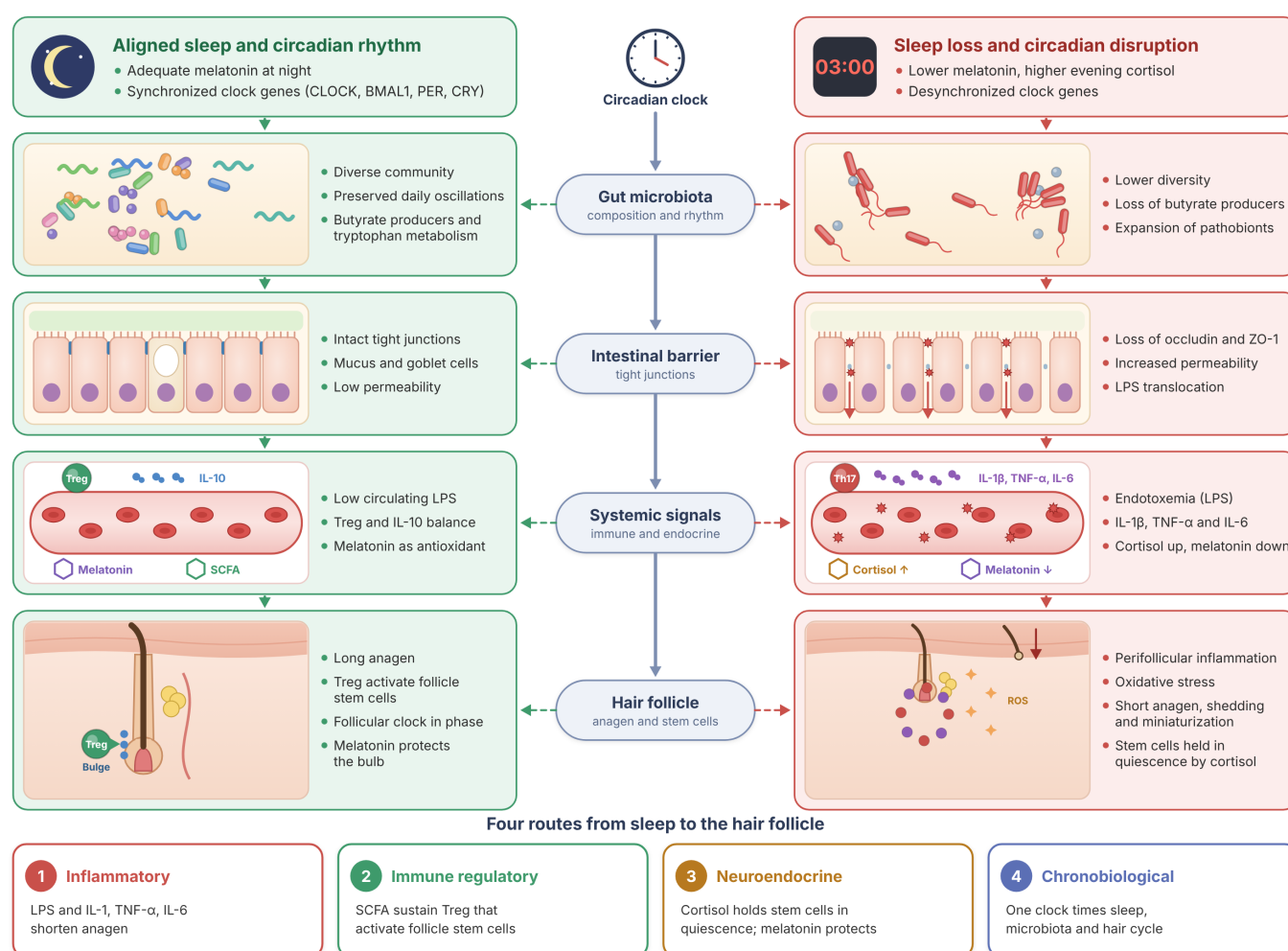


Figure 1. Integrative model of the sleep gut follicle axis. Left: aligned sleep and circadian rhythm sustain a diverse microbiota with preserved daily oscillations, intact tight junctions, low circulating lipopolysaccharide with balance of regulatory T cells and interleukin 10, and a follicle in long anagen. Right: sleep loss and circadian disruption are associated with dysbiosis, loss of tight junction proteins and translocation of lipopolysaccharide,

endotoxemia with proinflammatory cytokines, higher cortisol and lower melatonin and, at the follicle, perifollicular inflammation, oxidative stress, short anagen, shedding and miniaturization. Bottom: the four routes discussed in the text. Illustration based on the author's original drawing.

Table 1. Links of the sleep gut follicle axis, main supporting evidence, experimental model and strength of evidence.

Link in the axis	Key evidence	Model	Strength
Sleep and circadian disruption alter the gut microbiota	Two nights of partial sleep deprivation shift taxa ⁹ ; diversity tracks sleep efficiency ¹⁰ ; jet lag disrupts microbial oscillations ⁷	Human, animal	Moderate
Dysbiosis and barrier failure produce systemic inflammation	Loss of tight junctions reversed by melatonin ¹¹ ; melatonin mediates ZO-1 and LPS in apnoea ¹⁵ ; endotoxemia ¹³ ; poor sleep and CRP, IL-6 ¹⁶	Human observational, animal	Moderate
Inflammation acts on the follicle	TLR4 in human follicle ¹⁹ ; IL-1 and TNF- α ²⁰ ; IL-6 ²¹ ; LPS induced telogen effluvium ²²	Human <i>ex vivo</i> , animal	Strong mechanistic
Microbial metabolites sustain regulatory T cells and follicle stem cells	Butyrate induces Treg ^{24,25} ; Treg drive stem cell activation ²⁷ ; probiotic induced anagen ²⁹ ; dysbiosis induced alopecia ³⁰	Animal	Mechanistic
Tryptophan, serotonin and melatonin	Microbiota regulate serotonin ³² ; follicle synthesizes melatonin ³³ ; topical melatonin raises anagen rate ³⁵	Animal, human <i>ex vivo</i> , pilot RCT	Moderate
Stress hormones	Sleep loss raises evening cortisol ³⁶ ; microbiota calibrate HPA ³⁷ ; corticosterone holds stem cells in quiescence ³⁸ ; follicular HPA ³⁹	Human, animal, human <i>ex vivo</i>	Moderate
Circadian clock	Clock genes modulate the human hair cycle ⁶ ; night shifts disturb follicular clock genes ⁴¹	Human <i>ex vivo</i> , human observational	Moderate
Clinical associations	Sleep disorders and alopecia areata ^{44,45} ; sleep and severe AGA ⁴⁶ ; systematic review ⁴³ ; Mendelian randomization ^{48,49}	Human observational, genetic	Low to moderate

AGA, androgenetic alopecia; CRP, C reactive protein; HPA, hypothalamic pituitary adrenal axis; IL, interleukin; LPS, lipopolysaccharide; RCT, randomized controlled trial; TLR4, toll like receptor 4; TNF, tumour necrosis factor; Treg, regulatory T cells; ZO-1, zonula occludens 1.

In real patients these routes probably operate together, and this is why the axis helps to explain a clinical observation that is common in trichology and rarely written down: patients with poor sleep frequently present with a combination of shedding, scalp sensitivity, slower response to treatment and relapses that coincide with periods of night work, insomnia or emotional overload.

Clinical translation

For clinical practice, the model supports three practical attitudes. First, sleep should be assessed routinely in patients with telogen effluvium, androgenetic alopecia and alopecia areata, using validated instruments such as the Pittsburgh Sleep Quality Index, and patients with snoring or daytime sleepiness should be screened for obstructive sleep apnoea, given the association of a high STOP BANG score with severe androgenetic alopecia and of apnoea severity with barrier dysfunction^{15,46}. Second, sleep hygiene should be treated as part of the trichological prescription: regular sleep and wake times, including weekends, protection of darkness during the night, meals concentrated in the active phase of the day, a diet rich in fermentable fibre, regular physical activity and the management of stress are coherent with the mechanisms described here^{1,2,7,28}. Third, microbiota directed interventions, melatonin and topical cosmetic products formulated with melatonin or postbiotics are biologically plausible adjuvants that may act on these pathways, but their use for hair outcomes remains to be validated in controlled trials; the available clinical data are limited to a small pilot trial of topical melatonin³⁵ and one trial of a topical postbiotic product in alopecia areata³¹.

Limitations and research agenda

No study has yet measured sleep, gut microbiota and hair outcomes in the same individuals, and therefore the axis is a model built from links that were each demonstrated separately. Several links rest on animal experiments or on human follicles in organ culture, the human sleep deprivation studies are short and small⁹, and most clinical associations are cross sectional⁴³. The model nevertheless generates predictions that can be tested: (1) markers of intestinal permeability and endotoxemia should correlate with sleep quality and with trichogram parameters in patients with telogen effluvium; (2) the follicular clock gene profile measured in plucked hairs should differ between shift workers with and without hair loss; (3) interventions that improve sleep, such as cognitive behavioural therapy for insomnia or treatment of sleep apnoea, should improve both microbial markers and the anagen rate; and (4) microbiota targeted interventions should be tested with hair density and the trichogram as primary outcomes.

Conclusion

Sleep organizes biological time, and when it fails, the gut, the immune system, the stress axis and the hair follicle lose their synchrony. The evidence available today allows the trichologist to describe a sleep gut follicle axis with four biologically grounded routes and to treat sleep as a modifiable determinant of follicular health. Confirming this axis in prospective human studies is the next step, and it is a step that trichology is well positioned to take.

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Management of Conflicting Interests

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Declaration of Generative AI Use

Generative artificial intelligence (Claude, Anthropic) was used to assist in the preparation of this manuscript. The author reviewed all content and takes full responsibility for it.