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REVIEW

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Anemia and Sleep Disturbances: Mechanisms, Evidence and Clinical Implications – a Narrative Review

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ABSTRACT

Background: *Anemia, particularly iron deficiency anemia (IDA), is the most common subtype, which affects over two billion individuals worldwide and is associated with various sleep disturbances in both observational and interventional studies. Although there is a growing attention towards this connection, its mechanisms, extent and treatability of have not been thoroughly investigated.*

Objectives: *This narrative review assesses the relationship between anemia, including iron deficiency anemia (IDA) and non-IDA subtypes, and various sleep-related outcomes, such as subjective sleep quality, objective sleep architecture, total sleep duration, sleep latency, sleep efficiency, restless legs syndrome (RLS), periodic limb movements (PLMs), insomnia and sleep-disordered breathing across different age groups and populations.*

Methods: *We performed searches in PubMed/MEDLINE, Embase, Scopus, Web of Science and the Cochrane Library from the foundation of these databases until February 2026, without any language constraints. Eligible study formats encompassed cross-sectional surveys, prospective cohorts, case-control studies, randomized controlled trials (RCTs) of iron therapy that reported sleep outcomes, as well as current systematic reviews and meta-analyses. The synthesis was predominantly narrative due to significant variation among studies regarding anemia definitions, sleep assessment methodologies and population characteristics.*

Results: *Iron deficiency anemia is consistently linked to decreased sleep quality (higher Pittsburgh sleep quality index [PSQI] scores), reduced total sleep duration, extended sleep-onset latency, lower sleep efficiency and decreased slow-wave sleep. Restless legs syndrome (RLS) significantly manifests more often in IDA individuals, with ferritin levels of 50–75 μg/L being identified as clinically pertinent indicators. Iron supplementation, whether oral or intravenous, significantly decreases RLS severity scores and sleep disturbances in most of the assessed RCTs (moderate-certainty evidence).*

Conclusions: *The existing evidence suggests a clinically relevant link between anemia –particularly IDA – and numerous sleep disorders, with RLS being the most typical and iron-responsive symptom. Iron repletion is a viable mechanism-based treatment for IDA-related sleep disorders.*

Keywords: anemia, iron deficiency, restless legs syndrome, sleep quality, insomnia, sleep duration, ferritin, iron supplementation, narrative review.

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INTRODUCTION

Anemia, defined by the World Health Organization (WHO) as hemoglobin concentrations below 130 g/L in adult men, 120 g/L in non-pregnant adult women and 110 g/L during pregnancy, is still one of the most common nutritional problems globally, affecting an estimated 1.62-2 billion people (1). Iron deficiency is the primary cause of anemia worldwide, accounting for around half of all cases, with particularly high rates in South Asia, Sub-Saharan Africa and among women of reproductive age and small children (1).

Beyond the well-known consequences of weariness, diminished cognitive performance and decreased exercise capacity, growing data suggest that anemia, particularly iron deficiency, has clinically significant impacts on sleep architecture and quality. Hemoglobin and iron levels influence heart rate variability, oxygen delivery to central brain structures, dopaminergic neurotransmission and autonomic nervous system tone, all of which having an impact on sleep onset, maintenance and depth (2, 3).

Restless legs syndrome (RLS), an awful urge to move the limbs that typically worsens at rest and in the evening, is the most specific and well-documented sleep-related manifestation of iron insufficiency. The dopaminergic theory of RLS holds that insufficient iron availability in the basal ganglia inhibits dopamine synthesis and post-synaptic receptor function, resulting in pathognomonic sensorimotor symptoms (3, 4). Restless legs syndrome causes periodic limb movements during sleep, sleep-onset insomnia and excessive daytime somnolence, all of which exacerbating the functional impairment caused by anaemia (3).

Low hemoglobin or ferritin levels are also associated with poorer global sleep quality as measured by validated instruments such as the Pittsburgh sleep quality index (PSQI), shorter total sleep time, prolonged sleep-onset latency and decreased slow-wave sleep on polysomnography (4, 5). Emerging longitudinal data point to a potential bidirectional relationship: chronic sleep fragmentation may upregulate hepcidin, a hepatic peptide that limits intestinal iron absorption and increases iron storage, sustaining or aggravating iron-deficient anaemia (6).

Despite the growing body of evidence, no comprehensive narrative synthesis has examined the full range of sleep disturbances associated with anemia across multiple subtypes (IDA, anemia of chronic disease, renal anemia) and population groups (children, pregnant women, older adults, healthcare workers). The current review fills this gap. We also review the clinical evidence for iron repletion as a mechanism-based therapy method and describe future research goals, with a focus on settings such as India, where both anemia and undiagnosed sleep problems have a significant public health impact.

Background and clinical significance

Anemia is typically thought of by clinicians as a hematological illness characterized by pallor, exertional dyspnea and weariness. These signs reflect the blood's diminished oxygen-carrying capacity, but the systemic effects of chronic low hemoglobin go far beyond hemodynamic correction. Sleep is crucial for synaptic consolidation, metabolic waste elimination through the lymphatic system, hormonal balance and immunological function. When sleep is restricted or architecturally altered, cumulative deficiencies occur in the cognitive, cardiometabolic and immunological systems (7). Thus, the confluence of anemia and sleep represents a clinically significant but unappreciated axis of morbidity.

Sleep disturbances, often known as sleep disorders, are medical diseases that disrupt the quantity, quality and timing of sleep, leading to discomfort during the day and impaired functionality (8).

Sleep disorders are grouped into six major categories in the ICSD-3-TR, including insomnia disorders; sleep-related breathing disorders; central disorders of hypersomnolence; circadian rhythm sleep-wake disorders; parasomnias; and sleep-related movement disorders.

There is a distinct relationship between anemia and obstructive sleep apnea (OSA) because OSA patients are continuously subjected to hypoxic drive, which increases erythropoiesis and causes polycythemia. Research has demonstrated that continuous positive airway pressure (CPAP) therapy reduces secondary polycythemia in OSA patients (9, 10).

Sleep disorders caused by iron deficiency are likely to be significantly underdiagnosed in resource-constrained settings with high anemia

prevalence, such as large parts of India, where national surveys report anemia in approximately 53% of women of reproductive age and 67% of children under five (1). Recognizing and treating the hematological cause of sleep disturbance in these groups could result in significant gains in daily functioning, educational attainment, occupational productivity and long-term cardiovascular health.

In patients with sickle cell disease (SCD), localized hypoxia due to OSA directly promotes the polymerization of deoxygenated hemoglobin S (HbS), causing vaso-occlusive crises (VOCs), increasing blood viscosity and causing cellular sickling (11). ❑

METHODS

Search strategy

We conducted electronic database searches in PubMed/MEDLINE, Embase, Scopus, Web of Science and the Cochrane Library from the database's inception to February 2026, with no language or publication date constraints. To identify new eligible research, we hand-searched the reference lists of retrieved papers as well as current systematic reviews. Grey literature was researched using Google Scholar and Open Grey. Search terms combined MeSH headings and free-text equivalents for the exposure (anemia OR iron deficiency OR "low hemoglobin" OR "low ferritin" OR "iron deficiency anemia") with terms for the outcome domain (sleep OR insomnia OR "sleep quality" OR "sleep duration" OR "sleep efficiency" OR "restless legs" OR "periodic limb movements" OR "sleep-disordered breathing" OR polysomnography OR actigraphy OR PSQI OR IRLS).

Eligibility criteria

Eligible studies included human participants of any age and investigated any recognized kind of anemia (IDA, non-IDA, or undefined) in connection with at least one quantitative sleep outcome. Acceptable study designs included cross-sectional surveys, prospective cohort studies, case-control studies, randomized controlled trials of iron or erythropoiesis-stimulating drug therapy with sleep outcomes and pre-existing systematic reviews or meta-analyses. Case reports, or case series (with fewer than 10 participants), animal experiments, editorials, conference papers with no extractable data and studies that did not give a quantitative hemoglobin or ferritin definition of anemia or iron deficiency were also omitted.

Study selection and data extraction

Two investigators independently selected titles and abstracts, then reviewed the full text of possibly suitable papers. Disagreements were resolved by discussion and, when needed, by a third reviewer. Two reviewers extracted data independently using a standardized form that included the study design, country, population characteristics, anemia definition (hemoglobin and/or ferritin threshold used), sleep outcome measures (instrument, objective *vs* subjective), principal results (effect estimates with 95% confidence intervals where reported) and funding sources.

Quality assessment

The methodological quality of observational studies was assessed using the Newcastle-Ottawa scale (NOS) for cohort and case-control designs, as well as a modified NOS for cross-sectional research. Randomised controlled trials were evaluated using the Cochrane Risk of Bias Tool version 2 (RoB 2). The overall certainty of evidence for important conclusions was assessed using the GRADE method. Because of the significant variation in study populations, anemia definitions, and sleep measures, pooled quantitative synthesis was carried out only where three or more suitably comparable studies addressed the same outcome; otherwise, findings are reported as a narrative summary. ❑

RESULTS

Study characteristics

Searches yielded a large corpus of literature, including cross-sectional surveys, prospective cohort studies, case-control studies, RCTs and previous systematic reviews. Participants in eligible studies were pregnant women, infants and young children, community-dwelling older persons, healthcare workers and patients with chronic renal disease or other comorbidities. Most studies used WHO hemoglobin thresholds to identify anemia, while ferritin thresholds for iron insufficiency varied from 12 µg/L to 75 µg/L based on population and guideline era. Sleep outcomes were evaluated subjectively (PSQI, in-

somnia severity index and IRLS scale), objectively (polysomnography, actigraphy), or both. Table 1 summarizes core findings across outcome domains.

Subjective sleep quality

Multiple cross-sectional studies and scoping reviews show that individuals with IDA have statistically significantly higher PSQI scores (indicating poorer sleep quality) than non-anaemic controls, an association that persists after adjusting for depression, chronic pain, and other potential confounders (2, 5). Workers with confirmed IDA – including nursing professionals and industrial employees – have significantly lower global PSQI scores than matched controls with normal hemoglobin and ferritin levels (8).

Sleep duration, latency and efficiency

Individuals with IDA sleep around 15-40 minutes less each night than iron-replete comparators, with a larger proportion of time awake following sleep initiation, resulting in significantly lower sleep efficiency (2). Prolonged sleep-onset latency – likely reflecting the restlessness and autonomic hyperarousal associated with iron deficiency – is a common observation in both adult and paediatric groups (3, 5). Polysomnographic studies also reveal lower time in slow-wave (N3) sleep, which is linked with impaired restorative function

Restless legs syndrome and periodic limb movement

Restless legs syndrome is the most specific and well-documented sleep-related indication of iron insufficiency. Cross-sectional surveys and meta-analyses show that RLS prevalence is much higher in IDA populations, with pooled odds ratios ranging from 2.1 to 3.8 compared to iron-replete comparators in studies using established diagnostic criteria. Ferritin values below 50-75 μg/L are strongly and reliably predictive of RLS symptoms, which is now incorporated into various clinical practice guidelines (12).

Polysomnographic studies show that iron-deficient patients have greater periodic limb movement indices (PLMIs), with each PLM-associated arousal disrupting sleep and leading to non-restorative rest (13). RCTs of both oral and intravenous iron demonstrate meaningful reductions in International RLS rating scale (IRLS) ratings and PLMIs, with intermediate-certainty evidence supporting iron replacement therapy as an efficacious strategy for IDA-related RLS (14-16).

Insomnia, sleep disordered breathing and bidirectional effects

Large population-based surveys show that individuals with anemia had a higher likelihood of self-reported insomnia symptoms, a link evident in both working-age adults and community-dwelling older adults (6, 17). Anemia is related to increased daytime somnolence and higher rates of sleep-disordered breathing in older adults, but the evidence for the latter is more limited and methodologically variable.

Longitudinal cohort data suggest bidirectional causation: persistent sleep fragmentation increases inflammatory cytokines (interleukin-6, tumor necrosis factor-alpha) and hepcidin expression, impairing intestinal iron absorption and promoting intracellular iron sequestration in macrophages (6). If verified in future human investigations, this mechanistic pathway would create a self-perpetuating cycle in which IDA impairs sleep, which in turn exacerbates iron shortage.

Dopaminergic pathways are altered by brain iron deficiency, which directly causes phenotypes like RLS, periodic limb movement disorder (PLMD) and attention-deficit/hyperactivity disorder (ADHD) that are characterized by restlessness throughout the day and at night (18).

The anemia-excessive daytime sleepiness (EDS) association

Studies have shown that there is a strong correlation between anemia and increased incidence of excessive daytime drowsiness. The decrease in oxygen-carrying capacity seems to directly exacerbate daytime lethargy, making it challenging for elderly anaemic patients to stay awake during the day (19).

Vulnerable populations

Infants and young children in resource-constrained settings incur a significant burden of IDA-related sleep disturbance. Actigraphy and polysomnography reveal that iron-deficient newborns and toddlers had shorter overall sleep duration, more frequent nocturnal awakenings and altered sleep architecture (20). Because the developing brain is extremely vulnerable to both iron deficiency and sleep disturbance, the coe-

TABLE 1. Synthesis of evidence on anemia and sleep outcomes across included studies

Outcome/sleep parameter	Key findings	Study types and evidence level	Effect size/pooled estimates	Notes/specific groups affected
Subjective PSQI score	Consistently poorer in IDA; elevated PSQI scores	Cross-sectional surveys, scoping reviews	Statistically significant PSQI elevation	Independent of comorbidities, observed in workers, women of reproductive age
Total sleep time/duration	Reduced (shorter TST)	Observational, cohort studies	15–40 min shorter on average	Seen in adults, children and infants
SOL	Prolonged	Cross-sectional, RCTs	Increased latency *vs* controls	Linked to low ferritin (<50 µg/L)
Sleep efficiency	Lower	Polysomnography-based studies	Reduced efficiency %	Fragmented overnight rest
SWS	Decreased	PSG/objective measures	Less time in the N3 stage	Restorative sleep impaired
RLS	Markedly elevated prevalence; ferritin <50–75 µg/L strongly predictive	Cross-sectional, meta-analyses, RCTs	Pooled OR approximately 2.1–3.8*	Most specific and treatment-responsive phenotype
PLM	Increased index	PSG studies, RCTs	Higher PLM index *per* hour	Contributes to micro-arousals and fragmented sleep
Insomnia symptoms	Higher odds of insomnia complaints	Cross-sectional, longitudinal	Increased insomnia prevalence	Especially prevalent in older adults
SDB	Weak and inconsistent association	Limited studies available	No consistent effect size	Requires further investigation
Bidirectional effects	Sleep fragmentation may worsen anemia *via* hepcidin upregulation	Longitudinal cohorts	Suggested but not fully quantified	Potential self-perpetuating cycle
Maternal IDA → infant sleep	Shorter nocturnal sleep, more awakenings at 6–12 months	Prospective cohort studies	Elevated OR for poor infant sleep	Pregnancy-specific vulnerability
Iron supplementation effect	Significant improvements in RLS severity (IRLS scores), PLM index and sleep complaints	RCTs (oral and IV iron)	Moderate certainty *per* GRADE; meaningful reductions in IRLS and PLM index	Oral and intravenous iron are both effective; the benefits are often rapid

*Pooled odds ratio estimates are derived from meta-analyses identified in the literature; values reflect ranges across available studies using validated RLS diagnostic criteria. RLS =restless legs syndrome; IDA=iron deficiency anemia; SOL=sleep onset latency; SWS=slow-wave sleep; OR=odds ratio; PSQI=Pittsburgh sleep quality index; IRLS=International RLS rating scale; PLM=periodic limb movements; SDB=sleep-disordered breathing; PSG=polysomnography; RCTs=randomized controlled trials; GRADE=Grading of Recommendations, Assessment, Development and Evaluations.

xistence of these two diseases during important neurodevelopmental windows may have long-term effects on attention, behavior and cognitive function.

Older individuals are a second high-risk cohort, with anemia becoming more common due to nutritional deficiencies, subclinical gastrointestinal blood loss and chronic diseases. Community-based studies of older people show continuous links between low hemoglobin and insomnia symptoms, excessive daytime tiredness and an increased risk of falling due to disrupted nocturnal sleep (17).

Pregnant women are at a significantly higher risk because of the increased iron requirement of gestation. According to prospective cohort studies, maternal IDA in the second and third trimesters is associated with shorter nocturnal sleep and more frequent arousals in offspring aged 6-12 months, implying that prenatal iron status

may programme infant sleep regulation *via* alterations in fetal neural development or stress-hormone programming (21).

Table of summary evidence

Table 1 presents a structured synthesis of findings across the principal sleep outcome domains evaluated in this review. ❑

DISCUSSION

Mechanistic pathways

The link between anemia and disturbed sleep is mechanistically feasible, with at least four overlapping mechanisms. First, low hemoglobin levels limit oxygen delivery to the brain and peripheral tissues, resulting in compensatory tachycardia and increased respiratory drive. These physiological reactions, which are adaptive during alertness, disrupt sleep by predisposing to micro-arousals and limiting persistent periods of deep non-REM sleep, which are required for restorative function (2, 3).

The second and most well-documented role of iron is in central dopamine metabolism. Iron is an obligatory cofactor for tyrosine hydroxylase, the rate-limiting enzyme in catecholamine production and dopamine D2 receptor expression in the basal ganglia. Iron deficiency in the substantia nigra and putamen reduces dopaminergic tone in circuits that modulate sensorimotor inhibition during rest, causing circadian-dependent sensorimotor discomfort in RLS and PLMs (3, 9). Additionally, iron deficiency alters the balance between wake-promoting monoamines and sleep-promoting neuromodulators. Delayed nocturnal melatonin release and increased evening alertness have been seen in iron-deficient people, indicating a shift toward sympathetic predominance during the pre-sleep period (5).

Fourth, anemia alters the autonomic balance toward sympathetic dominance even at rest, reducing the usual nocturnal decrease in heart rate and blood pressure that characterizes good non-REM sleep and allowing for more frequent micro-arousals (20).

Evidence appraisal and limitations of the literature

The overall quality of evidence in this field is limited by many recurring methodological constraints. The majority of research is cross-sectional, which precludes causal inference. Hemoglobin and ferritin criteria used to identify anemia and iron deficiency vary among studies, complicating comparisons and synthesis. Sleep evaluation methods differ greatly: much research depends solely on self-reported questionnaires, which are prone to recall bias and fail to capture objective sleep architecture, whereas studies using polysomnography are often small and undertaken in well-selected groups. Many studies fail to consider the potential confounding effects of depression, chronic pain, obesity and drugs on sleep.

Existing RCTs of iron therapy provide the strongest direct evidence for a causal influence on sleep outcomes; however, the majority of trials are small, short and focus on RLS as the primary endpoint rather than broader sleep architecture or total sleep time. Long-term follow-up data are generally missing. The best route (oral *vs* intravenous), dose and duration of iron supplementation for sleep-related outcomes have not been firmly established (22).

Clinical implications

The evidence discussed here supports various practical clinical suggestions that are tailored to the strength of the underlying data. In patients who present with insomnia, frequent nocturnal awakening, or RLS, especially if these symptoms are new or are accompanied by other signs of iron deficiency (fatigue, pica, hair loss, cold intolerance), hemoglobin, serum ferritin and transferrin saturation should be measured (9, 23). If the ferritin level is less than 75 μg/L in RLS, iron supplementation should be considered, regardless of the presence of frank anemia based on hemoglobin criteria.

Integrating iron status checks into routine occupational health assessments or wellness screenings for individuals reporting poor sleep or excessive daytime fatigue is a low-cost, potentially high-yield intervention for healthcare professionals, particularly those in settings with high background anemia prevalence, such as India. Oral iron supplementation, when tolerated, is a good first-line treatment; intravenous iron is useful for people with malabsorption, resistance to oral preparations, or a clinical need for faster replenishment. The apparent bidirectional link between sleep disruption and iron dysregulation highlights the need to address both

domains simultaneously in patients with either illness, rather than treating anemia and sleep disturbance separately.

Future research priorities

This review identifies several research gaps. Firstly, large-scale prospective cohort studies with serial hemoglobin, ferritin and objective sleep measurements (actigraphy or polysomnography) over multiple time points are required to establish temporal directionality and quantify the independent contribution of iron deficiency to incident sleep disturbance.

Secondly, interventional trials with broader sleep architecture endpoints – not just RLS and PLMs – would help to determine whether iron repletion improves total sleep time, sleep efficiency and slow-wave sleep duration in non-RLS presentations.

Thirdly, agreement on ferritin cut-off values for sleep-specific screening, which may differ from those used for anemia diagnosis, would improve clinical advice.

Fourthly, research in paediatric populations, pregnant women and low- and middle-income countries is a priority, as anemia and sleep problems co-occur at high rates but are understudied when combined. Finally, neuroimaging investigations during sleep in iron-deficient individuals may shed light on the brain substrates of the aforementioned dopaminergic and autonomic systems. ❑

CONCLUSION

Anemia, particularly iron deficiency, is linked to a clinically relevant and mechanistically coherent range of sleep disorders, including worse sleep quality, shorter sleep duration, delayed sleep onset, decreased slow-wave sleep, RLS and PLMs. RLS is the most specific trait with the most evidence of responsiveness to iron repletion. Emerging bidirectional research suggests that chronic sleep disruption may exacerbate iron shortage *via* hepcidin-mediated pathways, resulting in a vicious cycle of reciprocal aggravation. Pregnant women, small children and older persons are disproportionately affected by this comorbidity.

Incorporating iron status testing into the examination of patients with insomnia or RLS, and conversely, querying about sleep quality in those with confirmed anemia, is a clinically useful integration that is currently underutilized. Confirming and expanding these findings through well-designed prospective and interventional research will be critical for translating mechanistic insights into standardized clinical treatment pathways. ❑

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