REVIEW ARTICLE

The Vicious Cycle of Menopause: A Narrative Review of the Interplay Between Urinary Incontinence, Mood Disturbances, and Sleep

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ABSTRACT

Background: The menopausal transition is a period marked by the significant co-occurrence of urinary incontinence (UI) and mood disturbances (MD), such as depression and anxiety, which substantially impair quality of life. While often attributed separately to hormonal decline, the underlying mechanisms for their comorbidity are not fully explained by oestrogen deficiency alone. A growing body of evidence suggests a complex interplay, potentially mediated by other factors, with sleep disturbance emerging as a critical candidate. **Methods:** This narrative review synthesized literature identified through a comprehensive search of PubMed, Scopus, and PsycINFO databases. The search strategy combined keywords related to menopause, urinary incontinence, mood disorders, and sleep. We included observational studies, clinical trials, and relevant reviews published in English, focusing on peri- and postmenopausal women. The analysis was structured to evaluate the bidirectional relationships between UI, MD, and sleep, and to assess the evidence for sleep's role as a mediating factor. **Results:** The synthesis of evidence reveals strong, bidirectional relationships between all elements of the proposed "menopausal triad." UI, particularly through nocturia, directly fragments sleep architecture, leading to decreased sleep efficiency and reduced restorative slow-wave sleep. This sleep disruption, in turn, provokes neurobiological changes (e.g., amygdala hyperactivity, prefrontal cortex dysfunction) and HPA-axis dysregulation that predispose to and exacerbate MD. Concurrently, MD heightens the perception of UI severity through somatic awareness and catastrophizing, while also reducing adherence to self-management strategies. Critically, sleep disturbance functions as a central mediator, creating a self-perpetuating cycle where UI worsens sleep, which in turn worsens mood, ultimately increasing the burden and perception of UI. **Conclusion:** This review establishes that UI and MD in midlife women are intricately linked through the mediating pathway of sleep disturbance. The "menopausal triad" model provides a transformative framework for clinical practice, moving beyond a siloed approach. It mandates integrated assessment, where screening for all three components is essential, and promotes treatment strategies that target sleep improvement as a powerful leverage point to break the vicious cycle. This holistic approach is vital for allied health professionals to improve the overall quality of life for women navigating midlife.

INTRODUCTION

The menopausal transition is a pivotal life stage characterised by profound endocrinological changes that precipitate a wide array of symptoms, significantly impacting a woman's physical and psychological well-being (Monteleone et al., 2018). Among the most prevalent and distressing symptoms are urinary incontinence (UI) and mood disturbances (MD), including major depressive disorder and generalised anxiety disorder (Gibson et al., 2019). Epidemiological data consistently reveal that UI and MD co-occur at rates significantly higher than chance, suggesting a shared pathophysiology beyond common hormonal origins (Huang et al., 2020).

The conventional clinical narrative often attributes the parallel emergence of UI and MD to declining oestrogen levels. Oestrogen deficiency contributes to urogenital atrophy, decreased urethral closure pressure, and altered bladder function (Kim et al., 2021). It also simultaneously modulates central nervous system neurotransmitters like serotonin and norepinephrine, which are critical for mood regulation (Soares, 2017). However, this reductionist hormonal model is insufficient. Many women experience severe UI without MD, and vice versa, indicating the involvement of other critical, modifiable mediators (Avis et al., 2018).

This narrative review proposes that sleep disturbance is a crucial and underappreciated mediator that fuels a powerful bidirectional cycle between UI and MD. We synthesise evidence demonstrating that UI is a direct cause of sleep fragmentation (Perez-Lopez et al., 2021) and that poor sleep is, in itself, a robust and independent predictor for the onset and exacerbation of MD (Joffe et al., 2019;

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Palagini et al., 2022). Conversely, MD can exacerbate the subjective burden of UI and perpetuate sleep problems through cognitive and behavioural pathways (Narrow et al., 2022). The primary objective of this review is to integrate findings from urology, psychiatry, and sleep medicine to build a compelling case for this "menopausal triad." By presenting this conceptual model, we aim to foster a holistic clinical approach and stimulate research into multimodal interventions that target the entire cycle.

FROM URINARY INCONTINENCE TO SLEEP DISRUPTION

The pathway from UI to poor sleep is both direct and multifactorial. Nocturia, or the need to wake at night to void, is a primary mechanism. Each awakening fragments the natural sleep cycle, significantly reducing time spent in deep, restorative (N3) sleep and rapid eye movement (REM) sleep, which are critical for cognitive function and emotional homeostasis (Tikkinen et al., 2010; Bliwise et al., 2021).

The burden of nocturia extends beyond frequency; its bother and the associated worry significantly correlate with poorer sleep quality independent of the number of voids (Perez-Lopez et al., 2021). Beyond the physiological trigger of nocturia, the constant fear of leakage can lead to a state of heightened vigilance and pre-emptive waking, even in the absence of a full bladder. This state of cognitive hyperarousal, characterized by anxiety about incontinence, is a core feature of insomnia and actively prevents sleep initiation and maintenance (Roth, 2020).

FROM SLEEP DISRUPTION TO MOOD DISTURBANCES

The neurobiological consequences of chronic sleep deprivation create a fertile ground for MD. Sleep is fundamental for effective emotional regulation; sleep loss is associated with increased amygdala reactivity (the brain's fear centre) and decreased functional connectivity with the prefrontal cortex, which governs executive function and emotional control (Goldstein & Walker, 2014). This neural dysregulation manifests behaviourally as increased irritability, emotional lability, and a pronounced negative cognitive bias, all of which are core features of depression and anxiety disorders (Palagini et al., 2022). Furthermore, sleep disruption reliably dysregulates the hypothalamic-pituitary-adrenal (HPA) axis, leading to elevated cortisol levels and a blunted circadian rhythm, which are established physiological correlates of depressive disorders (Kalmbach et al., 2018). In menopausal women, sleep disruption arising from vasomotor instability may act synergistically with UI-related nocturnal awakenings, producing an amplified adverse cascade on mood and affective stability (Joffe et al., 2019).

THE INFLUENCE OF MOOD ON URINARY INCONTINENCE AND SLEEP

It is critical to recognize that the relationship within the triad is not unidirectional. Mood disorders can significantly influence the perception, severity, and management of UI and are, themselves, primary drivers of sleep disturbance. Depression and anxiety are strongly associated with heightened somatic awareness and catastrophizing, cognitive processes that can amplify the perceived severity and distress associated with UI symptoms (Narrow et al., 2022). Behaviourally, low mood, anhedonia, and fatigue can profoundly reduce motivation and adherence to effective self-management strategies, such as consistent pelvic floor muscle training (Cody et al., 2012). From a physiological perspective, chronic anxiety can increase overall muscular tension, including dysfunctional tension in the pelvic floor muscles, potentially worsening symptoms of specific UI subtypes (Lone et al., 2020). Finally, MD are established primary causes of sleep-onset and maintenance insomnia, thereby directly entrenching the cycle of poor sleep and ensuring its self-perpetuating nature (Roth, 2020).

METHODS

Review Design and Aim

This study employed a narrative review methodology to synthesize and critically appraise the existing literature on the interplay between urinary incontinence (UI), mood disturbances (MD), and sleep in midlife women. The primary aim was to develop a novel conceptual model—the "menopausal triad"—that positions sleep as a central mediator. Unlike a systematic review, which aims for a comprehensive, protocol-driven aggregation of all empirical evidence on a specific, narrow question, a narrative review allows for a broader, more exploratory synthesis of a complex and interdisciplinary topic, enabling the identification of themes, gaps, and the development of new theoretical frameworks (Ferrari, 2015; Green et al., 2006).

Search Strategy and Data Sources

To ensure a rigorous and comprehensive literature search, we queried three major electronic databases: PubMed/MEDLINE, Scopus, and PsycINFO. The search strategy was designed to capture the core concepts of the population and the key variables of interest. A combination of Medical Subject Headings (MeSH) terms

and free-text keywords was used, including: ("menopause" OR "perimenopause" OR "postmenopause" OR "midlife women") AND ("urinary incontinence" OR "nocturia" OR "overactive bladder") AND ("mood disorders" OR "depression" OR "anxiety" OR "affective symptoms") AND ("sleep" OR "sleep wake disorders" OR "sleep quality" OR "insomnia"). The search was limited to articles published in English from January 2000 to December 2023. Reference lists of key articles were also hand-searched to identify additional relevant publications.

Eligibility Criteria

Studies were selected based on the following criteria:

- Population: Peri- or postmenopausal women (typically aged 40-65).
- Concepts: Studies had to investigate a relationship between at least two of the three core elements of the triad (UI, MD, and sleep).
- Study Types: Original research articles (cross-sectional, case-control, longitudinal cohort studies, and clinical trials), systematic reviews, and meta-analyses were included to provide a comprehensive overview of evidence and established consensus.

The exclusion criteria were: (1) studies focusing on populations with specific comorbid neurological diseases (e.g., Parkinson's, multiple sclerosis) or active cancer treatment, as these conditions could independently and severely affect all three triad components; (2) studies not published in English; and (3) editorials, opinion pieces, and single case reports.

Selection Process and Data Synthesis

The literature selection and synthesis process followed the phases outlined in the established guidance for narrative reviews by Green et al. (2006) and Ferrari (2015), as depicted in Figure 1. The process began with the initial database search, which yielded a large volume of records. After removing duplicates, the titles and abstracts of the remaining records were screened for relevance against the eligibility criteria. The full texts of potentially relevant articles were then retrieved and thoroughly assessed to make the final inclusion decision.

Given the narrative synthesis approach, data was not extracted for meta-analysis. Instead, the included literature was analysed thematically to map the key pathways and interactions between UI, MD, and sleep. The synthesis was structured to first examine the direct relationships between each pair of variables (UI-Sleep, Sleep-MD, MD-UI) before integrating these findings to build the case for sleep's role as a central mediator in the proposed triad model. The quality of included studies was considered during the interpretation and discussion of the findings, with greater weight given to findings from longitudinal studies and high-quality systematic reviews.

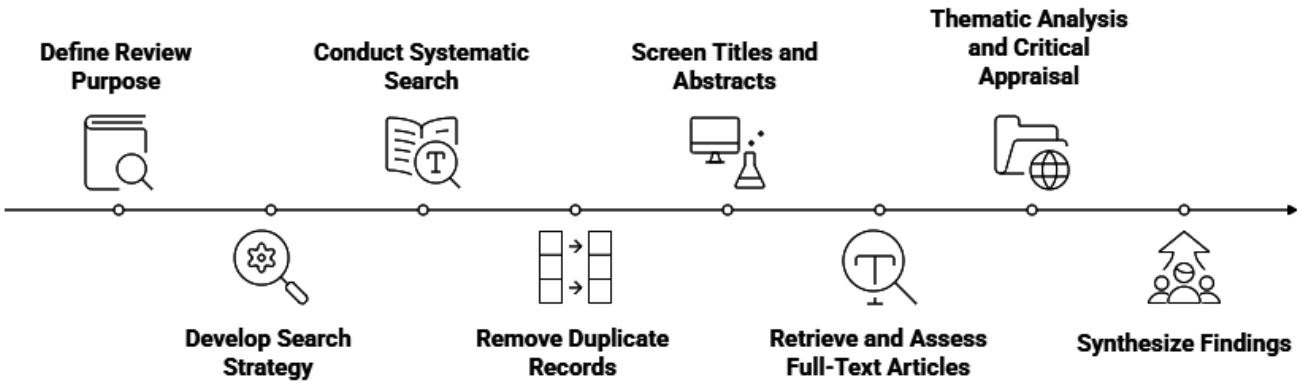

Figure 1: The literature selection and synthesis process.

SYNTHESIZING THE EVIDENCE: SLEEP AS THE CENTRAL MEDIATOR

When these individual pathways are considered together, a compelling and clinically useful model emerges. We propose that sleep is not merely a secondary symptom but a central mechanistic link that binds UI and MD in a self-perpetuating cycle. Consider a typical sequence: a woman experiences nocturia due to UI, leading to fragmented and non-restorative sleep. This sleep deprivation impairs her prefrontal cortex function and emotional resilience the following day, increasing her susceptibility to low mood, irritability, and anxiety (Baglioni et al., 2011). This

worsened mood, in turn, increases catastrophic thinking regarding her urinary symptoms and reduces her energy for pelvic floor exercises. These effects potentially worsen UI severity, leading to another night of poor sleep and further mood deterioration. This vicious cycle, illustrated in Table 1, powerfully demonstrates how sleep disturbance can act as the engine that drives the comorbidity of UI and MD, moving beyond a simple correlation to a dynamic pathological process.

Table 1: *The Vicious Cycle of the Menopausal Triad: Exemplar Pathways*

Stage	Symptom Manifestation	Underlying Mechanism	Consequence
1	Increased urinary incontinence, particularly nocturia.	Urogenital atrophy, decreased bladder capacity, detrusor overactivity.	Frequent nighttime awakenings, sleep fragmentation, reduced slow-wave sleep.
2	Sleep disruption and deprivation.	Reduced slow-wave and REM sleep; HPA axis dysregulation; increased inflammatory markers.	Impaired emotional regulation, increased negative affect, fatigue, hyperalgesia.
3	Worsening mood (depression, anxiety).	Amygdala hyperactivity, prefrontal cortex dysfunction, noradrenergic/serotonergic imbalance.	Heightened somatic awareness, catastrophizing about UI, reduced self-efficacy, social withdrawal.
4	Perceived worsening of UI burden.	Behavioural withdrawal from self-care (e.g., pelvic floor exercises); anxiety-induced pelvic floor tension.	Potential physiological worsening of UI; increased focus on symptoms.
5	Cycle Repeats	Return to Stage 1 with heightened severity across all domains.	Consolidation of the vicious cycle leads to a significant decline in quality of life.

CLINICAL IMPLICATIONS AND A CALL FOR INTEGRATED CARE

The "menopausal triad" model has profound implications for clinical practice, mandating a shift away from symptom-specific silos toward a more holistic, patient-centred approach. A fundamental implication is the necessity for systematic, holistic assessment. A woman presenting with one element of the triad should be automatically screened for the other two. For example, a gynecologist or primary care physician managing a patient's UI should use brief, validated tools like the Patient Health Questionnaire-9 (PHQ-9) for depression and the Pittsburgh Sleep Quality Index (PSQI) for sleep (Kroenke et al., 2001; Buysse et al., 1989). Similarly, a psychiatrist or therapist treating a perimenopausal woman for new-onset anxiety should proactively inquire about UI and sleep quality. This cross-disciplinary screening is the first step in identifying the presence of the full cycle.

Following assessment, treatment plans must be integrated and designed to target multiple components of the triad simultaneously for maximum effect. Effectively managing UI through evidence-based interventions such as pelvic floor physical therapy, pharmacotherapy (e.g., anticholinergics, beta-3 agonists), or neuromodulation can directly reduce nocturia, thereby directly improving sleep quality and indirectly alleviating mood symptoms (Kim et al., 2021). Conversely, treating MD with cognitive-behavioural therapy (CBT) or appropriate medication can improve sleep quality and enhance the patient's psychological capacity and motivation to manage her UI (Soares, 2017) proactively.

Perhaps most promisingly, directly addressing sleep disturbance with targeted interventions like Cognitive Behavioural Therapy for Insomnia (CBT-I) could serve as a powerful primary intervention to disrupt the entire cycle, potentially leading to concurrent improvements in both

mood and the perceived burden of UI (Kalmbach et al., 2018). Table 2 summarizes these multidimensional strategies, underscoring the need for future clinical frameworks that operationalize integrated care for menopausal health.

Table 2: *Clinical Considerations for Managing the Menopausal Triad*

Clinical Element	Key Considerations	Suggested Tools & Interventions
Assessment & Screening	Screen for all three components regardless of presenting complaint. Implement cross-disciplinary screening protocols.	**UI:** ICIQ-UI SF, 3-day bladder diary. **Mood:** PHQ-9, GAD-7. **Sleep:** PSQI, ISI.
Integrated Treatment Planning	Develop a collaborative, multi-component treatment plan. Treating one component can positively impact the others.	**UI:** Pelvic floor physical therapy, anticholinergics/beta-3 agonists. **Mood:** CBT, SSRIs/SNRIs. **Sleep:** Sleep hygiene, CBT-I.
Patient Education & Empowerment	Validate the patient's experience by explaining the interconnected nature of her symptoms. Frame treatment as breaking a cycle.	Use the triad model to illustrate the vicious cycle. Set realistic goals for incremental improvement across domains. Emphasize that these symptoms are a common, treatable part of midlife health.

Note: ICIQ-UI SF = International Consultation on Incontinence Questionnaire-Urinary Incontinence Short Form; PHQ-9 = Patient Health Questionnaire-9; GAD-7 = Generalised Anxiety Disorder-7; PSQI = Pittsburgh Sleep Quality Index; ISI = Insomnia Severity Index; CBT = Cognitive Behavioural Therapy; CBT-I = Cognitive Behavioural Therapy for Insomnia; SSRIs/SNRIs = Selective Serotonin Reuptake Inhibitors/Serotonin-Norepinephrine Reuptake Inhibitors.

CONCLUSION AND FUTURE DIRECTIONS

In conclusion, the interplay between urinary incontinence and mood disturbances in midlife women is best understood through a triad model that centrally incorporates sleep disturbance. Viewing these conditions as interconnected parts of a pathological cycle, rather than as isolated sequelae of oestrogen decline, provides a more accurate, comprehensive, and compassionate framework for patient care. Recognizing sleep as a key mediating factor offers a tangible and modifiable target for clinical intervention, providing a new avenue for improving outcomes.

Future research should prioritize longitudinal studies to confirm the temporal relationships and causal pathways implied in this model. Employing sophisticated statistical methods, such as cross-lagged panel analysis and formal mediation analysis, will be crucial to quantify the relative contribution of sleep within the triad. Furthermore, interventional trials are critically needed to test the efficacy of combined treatment approaches. For instance, a randomized controlled trial comparing the outcomes of pelvic floor therapy combined with CBT-I against standard, single-symptom care would provide robust evidence for the integrated model. Investigating the role of novel therapies, such as mindfulness-based stress reduction, which may simultaneously target mood, sleep, and the cognitive perception of UI, is another promising direction. By embracing the complexity of the menopausal experience, clinicians and researchers can forge person-centred strategies that not only break this vicious cycle but also promote enduring well-being in midlife and beyond.

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