

Sleep and Subjective Age: A Narrative Review of Epidemiological Evidence, Underlying Mechanisms, and Population Differences

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Abstract: Subjective age—how old an individual feels—is a robust predictor of health outcomes and mortality independent of chronological age. Sleep, as a fundamental and modifiable health behavior, has been increasingly implicated as a determinant of subjective aging, yet the evidence remains fragmented across disciplines. This narrative review synthesizes current knowledge on the association between sleep problems and subjective age by critically appraising epidemiological evidence, delineating potential mechanistic pathways, and examining population-level moderators. Converging evidence from cross-sectional surveys, longitudinal cohorts, and one randomized crossover trial consistently demonstrates that poor sleep quality and insufficient sleep are associated with older subjective age. Daytime sleepiness is the only mediator with preliminary empirical support; additional candidate pathways—including accelerated brain aging, cortisol rhythm dysregulation, impaired emotional regulation, and appearance-related social feedback—remain theoretically plausible but empirically untested. The association appears stronger in women, middle-aged and older adults, and morning chronotypes, although moderator evidence is limited. Critical gaps persist, including the absence of chronic sleep intervention trials in older populations, near-exclusive reliance on self-reported sleep measures, and insufficient mechanistic research. Future priorities include prospective studies integrating objective sleep assessment, randomized trials of evidence-based sleep interventions targeting subjective age in midlife and older populations, and direct testing of the proposed multi-level mechanistic framework. These findings position sleep health as a promising yet underexplored leverage point for promoting psychological healthy aging.

Keywords: subjective age, sleep quality, insufficient sleep, daytime sleepiness

Introduction

The Concept and Significance of Subjective Age in the Context of Global Aging

Global population aging has emerged as one of the most consequential social trends of the 21st century. According to United Nations projections, the worldwide population aged 65 years and older is expected to nearly double from 771 million in 2022 to approximately 1.6 billion by 2050.¹ Against this backdrop, achieving “healthy aging”—that is, maintaining functional capacity and subjective well-being in later life—has become a central concern in public health. Traditionally, chronological age has served as the primary indicator of the aging process; however, this metric is increasingly recognized as fundamentally limited, given that individuals of the same chronological age may vary dramatically in physiological function, psychological status, and social engagement.² Subjective age refers to the age that a person feels they have. It is a biomarker of aging at the biopsychosocial level and is highly sensitive to various aging-related cues.³ Due to its unique predictive value, it is receiving increasing attention.

Subjective age has now become an important research construct across geriatric medicine, psychology, and public health. It is typically assessed by asking respondents, “How old do you feel?” and quantified as the discrepancy between felt age and chronological age (ie., subjective age discrepancy) to capture both the direction and magnitude of perceived

aging. A large body of research indicates that the majority of adults tend to feel younger than their chronological age—a phenomenon especially prevalent in Western cultures—and that this discrepancy widens with advancing age.⁴

The primary reason subjective age has attracted such broad interest is that it predicts health outcomes and survival more accurately than chronological age alone. In a meta-analysis synthesizing data from 22 longitudinal studies encompassing over one million participants, Westerhof et al found that individuals who felt older than their chronological age had significantly elevated all-cause mortality risk, and this association remained robust after adjusting for chronological age, sex, educational attainment, and baseline health status.⁵ With respect to specific health domains, older subjective age (ie., feeling older than one's chronological age) has been significantly associated with accelerated cognitive decline, increased cardiovascular disease risk, greater depressive symptomatology, and impaired daily functioning.^{6–9} Collectively, this evidence suggests that subjective age is not merely a psychological impression but rather a sensitive indicator reflecting an individual's integrated physiological, psychological, and social functional status.

Critically, subjective age is dynamically malleable—unlike irreversible chronological age, it can be modified by lifestyle, psychological states, and health behaviors.¹⁰ This plasticity endows subjective age with unique intervention potential: if modifiable factors contributing to older subjective age can be identified and addressed, it may be possible to attenuate perceived aging, which could in turn generate positive cascading effects through improved health behaviors and psychological well-being. Identifying key modifiable determinants of subjective age has therefore become an important research priority for promoting healthy aging.

Sleep: A Key Modifiable Factor Influencing Subjective Age

Among the various lifestyle factors that may influence subjective age, sleep warrants particular attention owing to its universality, modifiability, and profound impact on the aging process. Sleep is a fundamental physiological process essential for the normal functioning of the nervous, endocrine, and immune systems. However, with advancing age, sleep architecture undergoes progressive deterioration, characterized by reductions in slow-wave sleep, increased sleep fragmentation, prolonged sleep onset latency, and diminished circadian rhythm amplitude.¹¹ These age-related sleep changes render older adults a high-risk population for sleep disturbances. Epidemiological surveys indicate that approximately 50% of adults aged 65 years and older worldwide experience chronic insufficient sleep or poor sleep quality, a proportion that is even higher among those with comorbid chronic diseases.¹²

Substantial evidence indicates that sleep problems exert multidimensional adverse effects on the healthy aging process. At the cognitive level, acute sleep deprivation is closely linked to impaired executive function, episodic memory deficits, and attentional lapses.¹³ At the somatic level, sleep problems increase the risk of cardiovascular disease, metabolic syndrome, and immune dysregulation.¹⁴ At the psychological level, poor sleep quality is a well-established risk factor for depressive and anxiety disorders.¹⁵ Notably, these very domains—cognitive decline, physical deterioration, and emotional disturbance—also constitute core experiential sources through which individuals perceive themselves as “growing older”, thereby providing a theoretical rationale for the link between sleep problems and older subjective age.

Over the past decade, the direct association between sleep and subjective age has emerged as an active area of investigation. Preliminary evidence from cross-sectional studies, longitudinal cohort studies, and randomized controlled trials consistently suggests that sleep problems may represent an important risk factor for older subjective age.^{16–18} For example, a randomized crossover trial provided the first experimental demonstration that acute sleep restriction significantly increases subjective age in young adults;¹⁸ whereas a longitudinal study showed that an older subjective age, in turn, can predict subsequent deterioration in sleep quality.¹⁶ This suggests a potential bidirectional association between the two.

Despite growing research interest, the existing evidence base has several critical limitations. Firstly, relevant studies are scattered across geriatric medicine, sleep medicine, and psychology, and no review has yet systematically synthesized this evidence. Secondly, the specific mechanistic pathways through which sleep affects subjective age lack a coherent theoretical framework. Thirdly, existing studies vary considerably in sample characteristics (age, sex) and sleep assessment methods, necessitating critical appraisal to clarify the consistency and limitations of the evidence.

Aims and Scope of the Review

Building on the research background and gaps identified above, this narrative review aims to: (1) synthesize and critically appraise the epidemiological evidence on the association between sleep problems and subjective age; (2) delineate the potential mechanistic pathways through which sleep influences subjective age; (3) examine how this association varies across populations; and (4) propose future research directions and clinical implications.

The literature search was conducted using PubMed and Web of Science databases, covering the period from inception through September 2025. The English-language search strategy combined the following subject headings and free-text terms: “subjective age” OR “felt age” OR “perceived age” OR “age identity” OR “awareness of age-related change”, combined with “sleep” OR “sleep quality” OR “sleep deprivation” OR “sleep restriction” OR “sleep duration” OR “sleep fragmentation” OR “circadian rhythm” OR “daytime sleepiness” using the AND operator. No restrictions were placed on study design; cross-sectional studies, longitudinal cohort studies, randomized controlled trials, and relevant mechanistic studies were all included. In addition, reference lists of included articles were hand-searched to ensure comprehensive literature coverage.

Definition of Key Concepts

To ensure clarity in the discussion that follows, this section defines the core terms used throughout the review. These concepts can be grouped into two categories: sleep-related concepts and subjective age-related concepts.

Sleep-Related Concepts

The sleep-related terms used in this review span multiple dimensions, from objective measurement to subjective perception, and are defined as follows:

Sleep quality is a complex, multidimensional construct that encompasses both subjective perceptions of sleep and objective sleep characteristics, typically including sleep onset latency, sleep duration, sleep efficiency, and sleep disturbances.¹⁹ In research, sleep quality is most commonly assessed using the Pittsburgh Sleep Quality Index (PSQI).

Insufficient sleep refers to a condition in which an individual does not obtain adequate sleep duration to meet their physiological needs. In research, insufficient sleep may be assessed through self-report (e.g., “On how many of the past 30 days did you not get enough sleep?”) or objective monitoring. It is a descriptive concept referring to naturally occurring sleep duration that falls below recommended thresholds.

Sleep restriction refers to the experimental manipulation of reducing participants’ sleep duration below normal levels under controlled laboratory conditions. Unlike naturally occurring insufficient sleep, sleep restriction is a researcher-imposed intervention and represents a key experimental paradigm used in randomized controlled trials to establish causal relationships.

Subjective sleep refers to an individual’s self-evaluation of their own sleep, which does not necessarily correspond to information obtained through objective sleep measures.¹¹ Subjective sleep captures experiential dimensions of sleep that cannot be assessed through objective measurement alone.²⁰

Subjective Age-Related Concepts

Subjective age refers to the age an individual subjectively perceives themselves to be, as distinct from their objective chronological age; it represents a core cognitive appraisal of one’s own aging state.¹⁶

Subjective age discrepancy is the difference between subjective age and chronological age: positive values indicate older subjective age (feeling older than one’s actual age), while negative values indicate younger subjective age (feeling younger than one’s actual age).²¹

Awareness of Age-Related Change (AARC) captures an individual’s subjective awareness that their behaviors, functional performance, or life experiences have changed as a consequence of aging.²² AARC comprises both positive (gains) and negative (losses) dimensions, and compared with a single-item subjective age measure, AARC provides a more nuanced and multidimensional assessment of the aging experience.

Epidemiological Evidence on the Association Between Sleep and Subjective Age

Existing research on the association between sleep and subjective age encompasses cross-sectional surveys, longitudinal cohort studies, and a small number of randomized controlled trials (summarized in Figure 1), involving different sleep exposure measures and different directions of association. This section organizes the available evidence by content area (rather than strictly by study design) to provide a clearer map of the current knowledge base.

The Impact of Sleep Problems on Subjective Age: Observational Evidence

Subjective Sleep Quality and Subjective Age

Two important studies have provided evidence on the impact of subjective sleep quality on subjective age from complementary perspectives.

Yoon et al conducted a nationwide cross-sectional study in South Korea involving 2,349 community-dwelling residents aged 19–92 years, assessing subjective sleep quality with the PSQI.¹⁷ The PSQI evaluates 19 individual items across several domains, including subjective sleep quality, sleep onset latency, sleep duration, and habitual sleep efficiency, yielding a composite score reflecting overall subjective sleep quality over the preceding four weeks. Results indicated that, compared with the good sleep quality group (PSQI ≤ 5), the poor sleep quality group (PSQI > 5) exhibited a significantly higher subjective age discrepancy ($\beta = 1.05$, 95% CI: 0.26–1.83, $p < 0.001$), and this association remained stable after adjustment for sex, age, educational attainment, and depressive symptoms. Further analysis revealed that the association was more pronounced among women and adults aged 50–79 years, suggesting potential moderating roles of sex and age.

Sabatini et al, drawing on one-year follow-up data from the UK PROTECT cohort, explored the influence of subjective sleep difficulties on aging perceptions at a more granular level.²¹ This study enrolled 4482 participants at baseline (2776 completed follow-up) and examined the predictive role of nine subjective sleep indicators—including sleep quality, post-awakening alertness, sleep satisfaction, and frequency of early morning awakening—on subjective age discrepancy and AARC. After adjustment for demographic variables, mood, and daily functioning, several baseline sleep

SLEEP & SUBJECTIVE AGE: SCIENTIFIC EVIDENCE MAP

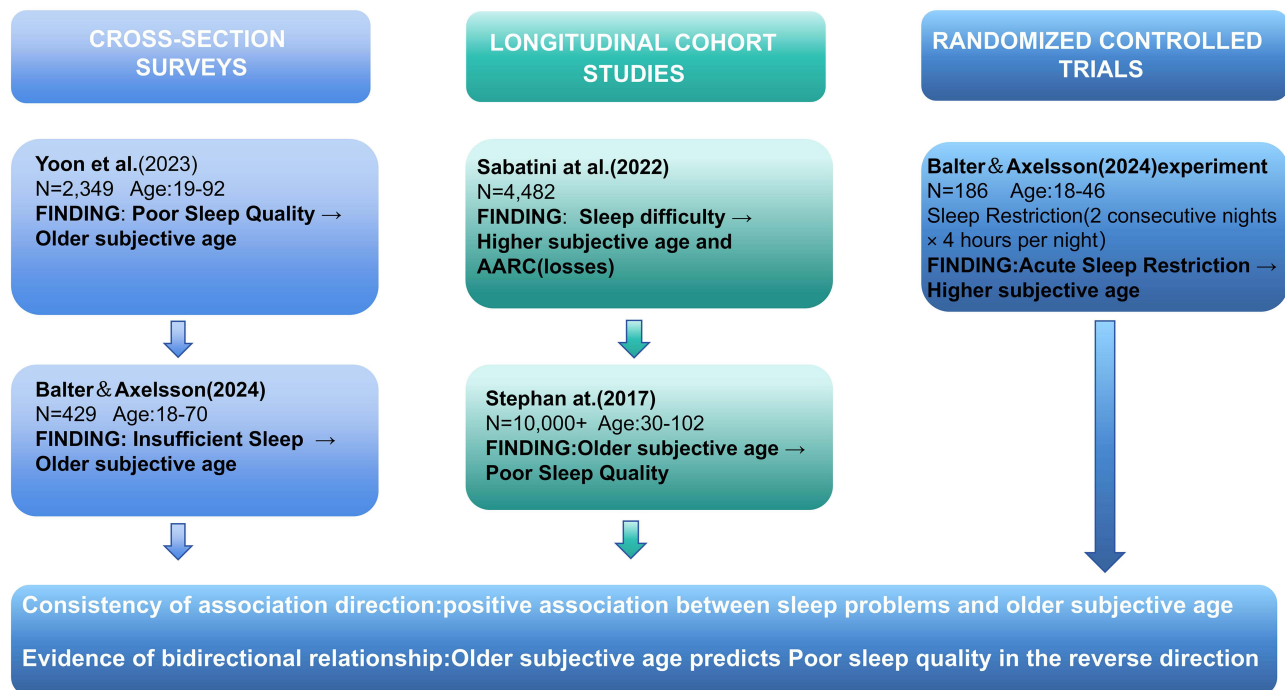


Figure 1 Epidemiological Evidence on Sleep and Subjective Age.

difficulty indicators significantly predicted one-year changes in AARC, including poor sleep quality ($\beta = -0.09$ to -0.15 , $p < 0.001$) and low post-awakening alertness ($\beta = -0.16$ to -0.31 , $p < 0.001$). Notably, however, the effect sizes were very small (explaining less than 0.5% of the variance), with statistical significance largely attributable to the high statistical power afforded by the large sample. Accordingly, although this study provides evidence for a longitudinal association between subjective sleep difficulties and perceived aging, its practical clinical significance should be interpreted with caution. Small effect sizes do not render the association meaningless; the influence of sleep on older subjective age is more likely to manifest through cumulative and interactive effects with other factors, such as mood, physical health, and social engagement. The value of this study lies primarily in confirming the directionality of the longitudinal association between sleep and older subjective age, rather than establishing sleep as a strong independent predictor of perceived aging. Future research should further investigate the cumulative effects of multiple sleep problems and their combined influence with other modifiable factors to more accurately gauge intervention potential.

Insufficient Sleep and Subjective Age

Balter and Axelsson conducted a cross-sectional survey in the United Kingdom enrolling 429 adults aged 18–70 years, assessing the number of days of insufficient sleep over the past 30 days (defined as the number of days on which respondents “did not get enough sleep”) and current subjective age via self-report.¹⁸ Results showed that each additional day of insufficient sleep over the past 30 days was associated with a significant increase of 0.23 years in subjective age. Individuals who reported no days of insufficient sleep over the 30-day period felt, on average, 5.81 years younger than their chronological age, whereas those with frequent insufficient sleep largely lost this youthful age bias.

Evidence for Bidirectionality: The Reverse Prediction of Sleep by Subjective Age

The studies reviewed above have primarily focused on the “sleep to subjective age” direction; however, a growing body of evidence suggests that the relationship may be bidirectional. Stephan et al, drawing on data from three large US longitudinal cohorts (the US Midlife in the United States, the Health and Retirement Study, the National Health and Aging Trends Study, total sample size exceeding 10,000; follow-up 3–10 years; age range 30–102 years), found that individuals with older subjective age at baseline were at significantly elevated risk for subsequent deterioration in sleep quality, and this association was independent of chronological age, depressive symptoms, anxiety, and chronic disease burden.¹⁶ This finding reveals a potential feedback loop between subjective age and sleep: sleep problems exacerbate perceived aging, which in turn may further impair sleep quality through mechanisms such as reduced motivation for health-promoting behaviors and increased psychological distress.

Experimental Evidence: The Causal Impact of Sleep Restriction on Subjective Age

Although observational studies have revealed significant associations between sleep problems and subjective age, their designs preclude the establishment of causal direction. Randomized controlled trials, by experimentally manipulating sleep conditions, provide more direct evidence for causal inference.

To date, the randomized crossover trial conducted by Balter and Axelsson represents the only experimental study in this field.¹⁸ This trial enrolled 186 healthy young adults aged 18–46 years and assessed subjective age under two experimental conditions: sleep restriction (two consecutive nights with only 4 hours of time in bed) and adequate sleep (two consecutive nights with 9 hours of time in bed). Results showed that, compared with the adequate sleep condition, acute sleep restriction increased participants’ subjective age by an average of 4.44 years (95% CI: 2.99–5.90, $p < 0.001$). This represents the first experimental demonstration of a causal effect of insufficient sleep on increased subjective age.

However, several limitations should be considered when extrapolating these findings. First of all, the sample comprised exclusively healthy young adults (aged 18–46 years), and whether the results generalize to middle-aged and older adults—the population in which the association between sleep problems and older subjective age appears most pronounced—remains to be determined. Additionally, the experimental paradigm involved acute sleep restriction (two consecutive nights), which differs fundamentally from the chronic insufficient sleep patterns commonly observed in the general population; the cumulative effects of the latter may be considerably more complex. At last, differences between the laboratory setting and real-world conditions may also limit the ecological validity of the observed effects.

Synthesis and Critical Appraisal of the Evidence

Synthesizing the observational and experimental evidence reviewed above, several preliminary conclusions can be drawn:

First and foremost, the association is consistent in direction. Regardless of the sleep assessment method employed (PSQI, multidimensional sleep difficulty questionnaire) or the study design (cross-sectional, longitudinal, experimental), a positive association between sleep problems and older subjective age is consistently observed, indicating a high degree of directional consistency across the evidence base.

Secondly, effect sizes are heterogeneous. Effect sizes vary substantially across studies—ranging from the very small effects reported by Sabatini et al (explained variance < 0.5%) to the sizeable effect observed in the Balter and Axelsson experiment (mean increase of 4.44 years). This heterogeneity likely reflects differences in sleep exposure type (acute vs. chronic), assessment instruments, and sample characteristics.

Thirdly, the evidence supports a bidirectional relationship. The available evidence supports a bidirectional association between sleep problems and subjective age, rather than a unidirectional causal effect. The longitudinal data from Stephan et al demonstrating that older subjective age predicts subsequent sleep quality deterioration suggest that future theoretical models and intervention strategies should account for this feedback loop.

However, common methodological limitations persist. Existing studies share several notable shortcomings: (1) sleep assessment relies predominantly on self-report measures, with no cross-validation using objective instruments such as polysomnography or actigraphy; (2) confounding factors are inadequately controlled, with sleep-related disorders (eg., obstructive sleep apnea), chronic disease severity, and medication use insufficiently accounted for; and (3) samples are drawn primarily from Western countries and South Korea, resulting in limited cultural diversity. These limitations indicate that current conclusions require further verification through more rigorously designed studies with more diverse samples.

Despite these methodological limitations, the available epidemiological evidence consistently indicates a significant association between sleep problems and older subjective age. However, merely establishing that an association exists is insufficient—to understand the nature of this association and to determine whether it is amenable to effective intervention, a deeper question must be addressed: Through what pathways do sleep problems influence an individual's perception of aging? In other words, what physiological, neural, and psychological processes intervene between a night of poor sleep and the next-day experience of “feeling years older”?

Potential Mechanisms Linking Sleep to Subjective Age

The available evidence indicates that the association between sleep problems and older subjective age is consistent across study designs and sleep metrics, and that experimental evidence provides preliminary support for a causal direction. The robustness of this association naturally prompts inquiry into its underlying mechanisms: How does insufficient sleep translate into the subjective experience of “feeling older”?

The answer to this question is unlikely to reside in any single pathway. Insufficient sleep simultaneously affects brain function, endocrine regulation, emotional states, and daytime behavioral performance, and changes in each of these domains may independently or synergistically shape an individual's perception of aging. This section organizes the available evidence into two tiers according to the directness of their empirical link to subjective age: mediating pathways with preliminary empirical support, and candidate mechanisms with a strong theoretical basis but awaiting direct testing. It should be noted at the outset that, because mechanistic research in this field remains in its early stages, the majority of pathways discussed in this section currently represent theory-driven hypotheses based on indirect evidence, rather than fully validated causal chains.

Mediating Pathway with Preliminary Empirical Support: Daytime Sleepiness

Among the limited mechanistic research conducted to date, daytime sleepiness is the only mediator with preliminary empirical support. Daytime sleepiness refers to excessive drowsiness occurring during periods when wakefulness should be maintained and represents the most immediate and common functional consequence of insufficient sleep.²³

The study by Balter and Axelsson provided key mediation analysis evidence: in their randomized crossover trial, when Karolinska Sleepiness Scale (KSS) scores were entered as a mediator in the statistical model, the direct effect of

sleep restriction on subjective age was substantially attenuated.¹⁸ This finding indicates that the effect of insufficient sleep on subjective age is largely transmitted through daytime sleepiness as a mediating pathway.

The plausibility of daytime sleepiness as a mediator can be understood from several perspectives. First, the physiological decline in vitality and reduced cognitive efficiency directly caused by sleepiness—such as slowed reaction times, impaired concentration, and difficulty with memory retrieval—constitute everyday experiences that individuals can readily perceive and attribute to “functional aging”.²⁴ Secondly, the reduction in social participation and physical activity caused by sleepiness deprives individuals of important external pathways for maintaining a youthful self-image.²⁵ Research has shown that both active social participation and regular exercise are associated with a younger subjective age.²⁶

It should be noted, however, that this mediation effect has thus far been demonstrated in only one acute sleep restriction experiment involving healthy young adults. Whether the same mediation pattern holds in the context of chronic insufficient sleep and among middle-aged and older populations requires confirmation in future studies. Furthermore, as a highly modifiable factor, daytime sleepiness—should its mediating role be corroborated by additional studies—would carry substantial clinical translation potential.

Candidate Mechanisms with Theoretical Support Awaiting Direct Testing

The following mechanistic pathways, while supported by robust evidence within their respective research domains, have not yet been directly linked to the specific outcome variable of subjective age in empirical studies. They remain plausible theoretical hypotheses.

Neurocognitive Pathway: Accelerated Brain Aging

Sleep serves as a critical period for brain repair, metabolic waste clearance, and memory consolidation. Sleep problems can induce aging-like changes in the nervous system across three levels: brain structure, brain function, and brain metabolism.

At the structural level, sleep restriction has been associated with reduced gray matter volume in multiple brain regions, particularly the postcentral gyrus.²⁷ Notably, independent lines of research have shown that individuals who feel older than their chronological age also exhibit smaller gray matter volumes in specific regions, including the inferior frontal gyrus and left postcentral gyrus.²⁸ Although no study has yet examined the complete chain from sleep problems to structural brain changes to increased subjective age within a single sample, the convergence of these two independent evidence streams at the common node of structural brain aging provides indirect support for this hypothesis.

At the level of metabolic waste clearance, the brain’s glymphatic system is highly active during sleep—particularly slow-wave sleep—facilitating the removal of metabolic byproducts such as β -amyloid.²⁹ Insufficient sleep significantly impairs glymphatic clearance efficiency, leading to the accumulation of neurotoxic proteins and, over the long term, potentially accelerating neurodegenerative processes.³⁰ It can be hypothesized that when this accumulation reaches a level that affects daily cognitive function, the individual’s experiences of mental slowness and memory difficulties may be attributed to “aging”, thereby increasing subjective age.

At the level of brain functional activity, sleep deprivation also leads to reduced activity in the prefrontal cortex, which is responsible for higher-order cognitive control, along with heightened activity in the amygdala, which processes emotions.³¹ Individuals may interpret this diminished sense of control as age-related functional decline.

Neuroendocrine Pathway: Cortisol Rhythm Dysregulation

Cortisol, the primary effector hormone of the hypothalamic–pituitary–adrenal (HPA) axis, normally follows a pronounced circadian rhythm, peaking in the early morning to promote arousal and declining to its nadir during the night.³² Because cortisol secretion is highly circadian in nature and particularly sensitive to time-of-day effects—especially during the evening—only studies employing 24-hour continuous sampling are discussed here. Research has demonstrated that even short-term restriction of sleep to 5.5 hours or less disrupts the normal cortisol secretory rhythm. Specifically, abnormal cortisol elevations emerge during the evening hours, while overall 24-hour cortisol levels remain unchanged. For instance, Broussard et al found that after restricting sleep to 4.5 hours (01:00–05:30), with blood samples collected every 15–30 minutes over a full 24-hour period, participants exhibited elevated cortisol levels between 17:00

and 21:00, with no significant elevations at other time points.³³ Nedeltcheva et al reported similar findings: after sleep restriction to 5.5 hours (00:30–06:00), with blood sampled every 15–30 minutes, cortisol levels were elevated between 20:00 and 22:00, with no significant changes at other times.³⁴

From a pathophysiological standpoint, sustained cortisol rhythm dysregulation—particularly abnormal evening and nighttime elevations—exerts multiple aging-promoting effects: neurotoxic actions on hippocampal neurons can impair learning and memory, immunosuppressive effects can increase inflammation and infection susceptibility, and metabolic disruption can promote central adiposity and insulin resistance.^{35,36} It is therefore plausible that HPA axis dysregulation induced by insufficient sleep may, through these physiological cascading effects, cause individuals to experience a constellation of bodily changes resembling those of aging, thereby indirectly influencing subjective age perception. However, it must be emphasized that this hypothesis has not yet been directly tested empirically.

Psychological and Behavioral Pathways

Insufficient sleep may also indirectly influence subjective age perception through multiple psychological and behavioral channels.

Impaired Emotional Regulation and Heightened Anxiety

Insufficient sleep can trigger or exacerbate anxiety.³⁷ Although the precise mechanisms linking sleep and anxiety remain incompletely understood, accumulating evidence indicates considerable overlap between the neural networks subserving sleep and emotion regulation. Neuroimaging studies have shown that, following sleep restriction, anxious individuals exhibit significantly enhanced amygdala and insular cortex reactivity to negative stimuli.³⁸ In an anxious state, individuals display heightened somatic vigilance and a tendency toward negative interpretation of bodily symptoms (eg., fatigue, muscle soreness), potentially leading them to attribute discomfort that is actually caused by sleep deprivation to “the body aging”. Additionally, elevated anxiety symptoms are closely associated with poor objective sleep quality.³⁹ A known bidirectional relationship exists between anxiety and sleep problems, which may create a vicious cycle in which anxiety leads to sleep deterioration, which in turn leads to an older subjective age, further exacerbating anxiety.

Cascading Degradation of Health Behaviors

Sleep-deprived individuals tend to exhibit a constellation of deteriorating health behaviors during the daytime, including reduced physical activity, increased sedentary time, and worsened dietary patterns (eg., increased intake of high-calorie foods).^{40,41} These behavioral changes not only objectively accelerate physiological aging but also enhance perceived aging through perceptible bodily changes such as declining physical fitness and weight gain. Given that regular physical activity is significantly associated with younger subjective age, the theoretical pathway whereby insufficient sleep contributes to older subjective age through reduced exercise participation is plausible.²⁶

Appearance Changes and Social Feedback

The impact of insufficient sleep on facial appearance has been experimentally documented: facial photographs taken after sleep deprivation are rated by independent observers as appearing more fatigued, less healthy, less attractive, and older-looking.^{42,43} These appearance changes may influence subjective age through two pathways: direct self-perception when individuals look in a mirror, and social feedback from others (eg., comments such as “you look tired” or “you look worn out”), both of which may reinforce the self-cognition that “I am getting older”.

Mechanistic Integration and Summary

Synthesizing the analyses above, sleep problems may influence subjective age through the multi-level, multi-pathway framework depicted in [Figure 2](#). Within this framework, daytime sleepiness—as the most immediate functional consequence of insufficient sleep—is currently the only pathway with preliminary mediation analysis support and constitutes the core element of the mechanistic model. Surrounding this core, accelerated brain aging at the neurocognitive level, cortisol rhythm dysregulation at the neuroendocrine level, and emotional dysregulation and health behavior deterioration at the psychological–behavioral level collectively form a broader network of candidate mechanisms.

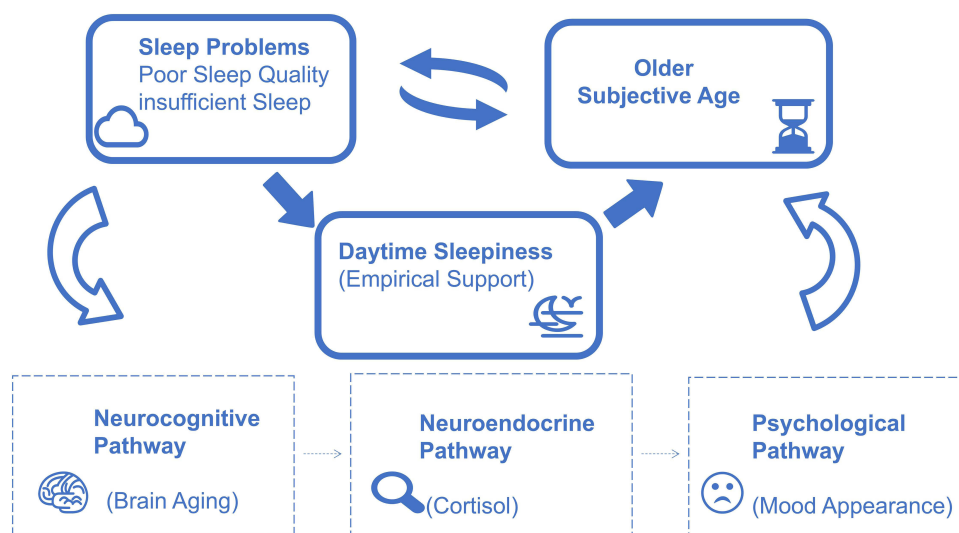


Figure 2 A Multi-Level Framework of Sleep Problems on Subjective Age.

Critically, these pathways are not mutually independent but may interact synergistically and in cascading fashion—for example, cortisol dysregulation may exacerbate cognitive decline, which may in turn impair emotional regulation, creating a mutually reinforcing network.

Key tasks for future research include: (1) simultaneously measuring sleep variables, candidate mediators, and subjective age within a single study to directly test the proposed theoretical pathways; (2) distinguishing the relative contributions of different mechanistic pathways; and (3) exploring how these mechanisms vary across populations.

However, the applicability of the mechanistic framework described above may be subject to important individual differences. On the one hand, sleep architecture and sleep needs vary considerably across populations—for instance, women face unique sleep challenges during the perimenopausal transition, older adults already have reduced slow-wave sleep, and individuals with different chronotypes vary in their tolerance to circadian disruption. On the other hand, the same degree of insufficient sleep may elicit markedly different physiological responses (eg., the magnitude of cortisol changes), cognitive effects (eg., the extent of attentional impairment), and psychological attributions (eg., whether discomfort is interpreted as “aging”) across individuals. This implies that the impact of sleep problems on subjective age is unlikely to be a “one-size-fits-all” effect but rather varies from person to person—understanding the patterns and sources of these individual differences is essential for developing precision-targeted intervention strategies.

Population Differences in the Association Between Sleep and Subjective Age

The preceding two sections established the link between sleep problems and older subjective age from both epidemiological and mechanistic perspectives. However, as noted at the end of [Potential Mechanisms Linking Sleep to Subjective Age](#), the strength and manifestation of this association are not uniform across all individuals—differences in physiological constitution, psychological traits, and sociocultural context may systematically moderate the extent to which sleep problems translate into perceived aging. Identifying these moderating factors not only deepens theoretical understanding of the sleep–subjective age relationship but is also a necessary prerequisite for translating research findings into personalized intervention strategies. To date, studies have examined three moderating factors: sex, age, and chronotype. The evidence for each is reviewed below, followed by a discussion of other potential moderators that remain to be explored.

Sex Differences

Available evidence suggests that sex may serve as an important moderator of the sleep–subjective age association. Yoon et al found in their cross-sectional study that the association between poor sleep quality and older subjective age was significantly stronger in women than in men.¹⁷ This sex difference may arise from multiple sources. From a physiological

standpoint, women experience more pronounced hormonal fluctuations across the lifespan (eg., perimenopausal estrogen variability), which directly affect sleep quality and result in more sustained exposure to sleep disturbances.^{44,45} From a psychological–cognitive standpoint, research has shown that women exhibit greater interoceptive awareness of their own health status, a trait that may predispose them to notice and amplify functional changes resulting from insufficient sleep.^{46,47} However, studies directly testing the moderating role of sex in the pathway from sleep to subjective age remain very limited, and the explanations offered above are largely theoretical and require verification through specifically designed future studies.

Age Differences

Age represents another important moderator. It is worth noting that older adults' responses to sleep deprivation are not uniformly more negative than those of younger adults across all domains. Research has shown that in terms of emotion regulation, older adults actually perform better than younger adults following sleep deprivation.⁴⁸ This phenomenon is partly attributed to an age-related increase in positivity bias, whereby older adults tend to prioritize the processing of positive information and avoid negative information.⁴⁹ Nevertheless, with respect to the association between insufficient sleep and older subjective age, middle-aged and older adults (typically those aged 50 years and above) show significantly greater sensitivity than younger adults. Yoon et al found that the association between poor sleep quality and older subjective age was significantly stronger in the 50–79 age group than in the 19–39 age group.¹⁷ This age-related difference may stem from several mechanisms. First, the sleep architecture of middle-aged and older adults has already undergone age-related deterioration (eg., reduced slow-wave sleep, increased sleep fragmentation), and poor sleep quality further exacerbates these changes, producing an “additive effect”.⁵⁰ Second, with advancing age, cognitive reserve and neural plasticity progressively decline, rendering middle-aged and older individuals more susceptible to the cognitive impairment caused by insufficient sleep.⁵¹ Third, middle-aged and older adults are more likely to experience age-related functional decline (eg., reduced physical stamina, cognitive deterioration), and poor sleep quality amplifies these declines, making individuals more likely to perceive themselves as “aging”.⁵² Although the positivity bias may partially buffer the negative emotional impact of sleep problems in older adults, on the dimension of subjective age—which involves somatic perception and functional self-assessment—the adverse effects of sleep problems remain more pronounced in middle-aged and older populations.

Chronotype Differences

Chronotype, which reflects the alignment between an individual's intrinsic biological clock and external time schedules, has recently been identified as a novel moderating factor in this field. Balter and Axelsson found significant differences in subjective age responses to sleep deprivation across chronotypes: under adequate sleep conditions, morning-type individuals reported subjective ages that were, on average, 3.35 years younger than those of evening-type individuals; however, under sleep restriction, morning-type individuals experienced a significantly greater increase in subjective age than their evening-type counterparts.¹⁸ This seemingly paradoxical finding may be explained as follows. Morning-type individuals maintain daily routines that are highly aligned with conventional social schedules, keeping their circadian rhythms in a relatively stable equilibrium. Consequently, when this equilibrium is disrupted by sleep restriction, the subjective impact is proportionally greater. In contrast, evening-type individuals are chronically adapted to the misalignment between their endogenous rhythm and social demands and may therefore exhibit greater tolerance to the circadian perturbation caused by sleep deprivation.⁵³ However, these explanations remain speculative and are based on findings from a single study; their replicability and applicability to other populations require further investigation.

Other Potential Moderating Factors

It is important to note that research on moderators of the sleep–subjective age association remains quite limited. The following factors may theoretically play a moderating role but have not yet been directly tested in existing studies:

Cultural Background

Cultures differ markedly in their attitudes toward and valuation of aging, which may influence how individuals interpret the functional changes resulting from insufficient sleep.⁵⁴ In cultures that venerate aging, the negative connotations of perceived aging may be attenuated, thereby buffering the impact of sleep problems on subjective age.

Socioeconomic Status

Individuals of lower socioeconomic status face both a higher risk of sleep problems and more limited access to health resources, which may amplify the effect of insufficient sleep on subjective age.⁵⁵

Limitations, Future Directions, and Practical Implications

Key Limitations of Existing Research

First, limitations in study design constrain causal inference. The vast majority of existing studies employ cross-sectional designs. The few longitudinal studies available primarily examined the reverse prediction of sleep by subjective age, rather than the prospective influence of sleep on subjective age.¹⁶ The only randomized controlled trial provides causal evidence but is limited to acute sleep restriction in healthy young adults, restricting the generalizability of its conclusions.¹⁸ There is currently a lack of studies that use chronic sleep problems as the exposure variable and prospectively track changes in subjective age in general population samples.

Moreover, sleep assessment relies predominantly on subjective report. Nearly all existing studies assess sleep status using self-report instruments, which are susceptible to the influence of mood states, cognitive biases, and social desirability, potentially introducing systematic measurement error. No study to date has employed polysomnography or other objective methods to cross-validate the accuracy of sleep assessments.

Besides, sample representativeness is insufficient. Existing samples are concentrated primarily in Western countries and South Korea, with a notable absence of data from Africa, South America, South Asia, and Southeast Asia. Given the profound influence of cultural context on aging attitudes and sleep habits, the cross-cultural applicability of current conclusions remains unknown. Additionally, some studies suffer from imbalanced sex ratios or narrow age ranges, limiting the generalizability of their findings.

Fourth, mechanistic research is severely lacking. To date, only daytime sleepiness has received preliminary support as a mediator, from a single study. All other candidate mechanisms (eg., brain aging, cortisol dysregulation, psychological-behavioral pathways) remain at the level of theoretical speculation without direct empirical testing.

Future Research Directions

Conduct Prospective Longitudinal Studies to Clarify Causal Direction

Future research should employ prospective cohort designs, assessing sleep status at baseline (incorporating both subjective and objective measures) and tracking changes in subjective age at multiple time points. Cross-lagged panel models can be used to simultaneously test causal pathways in both directions. A follow-up period of at least 2–3 years with repeated measurements at multiple time points is recommended to capture dynamic changes.

Conduct Randomized Controlled Trials of Chronic Sleep Interventions in Diverse Populations

The existing experimental evidence derives solely from an acute sleep restriction paradigm in young adults. Future studies should conduct randomized controlled trials of evidence-based sleep interventions—such as cognitive behavioral therapy for insomnia—in middle-aged and older populations to determine whether improving chronic sleep problems can effectively reduce subjective age. Such studies would not only provide stronger causal evidence but also directly assess the clinical feasibility and effectiveness of the intervention.

Integrate Subjective and Objective Sleep Assessment

Future research should combine self-report questionnaires with objective sleep monitoring tools (eg., actigraphy for long-term home-based monitoring) to clarify whether subjective sleep experience and objective sleep parameters differ in their ability to predict subjective age and how their interaction shapes aging perceptions.

Directly Test Candidate Mediating Mechanisms

Future studies should simultaneously measure sleep variables, candidate mediators (eg., daytime functioning, cognitive performance, emotional states, cortisol rhythms, neuroimaging markers), and subjective age within a single sample, using structural equation modeling or causal mediation analysis to directly test the multi-level mechanistic framework proposed in this review.

Expand Population Coverage and Cultural Diversity

Priority should be given to recruiting samples from diverse cultural backgrounds (particularly collectivist cultures and developing countries), different health conditions (eg., patients with sleep disorders, individuals with mild cognitive impairment, shift workers), and various socioeconomic strata to enhance the external validity of research conclusions.

Clinical and Public Health Implications

Although this field remains in its early stages of development, the available evidence already offers several preliminary implications for clinical practice and public health policy.

First, sleep assessment should be incorporated into clinical evaluation frameworks for subjective age and aging perceptions. In geriatric medicine and mental health settings, when patients report a marked increase in perceived aging, evaluating their sleep status (including sleep quality and daytime sleepiness levels) may help identify modifiable contributing factors. Second, sleep interventions may represent a promising strategy for promoting psychological healthy aging. Given the longitudinal association between older subjective age and multiple adverse health outcomes and the potential causal impact of sleep on subjective age, improving sleep quality may generate positive cascading effects by reducing subjective age.^{13,18} This provides a theoretical rationale for incorporating sleep health education and interventions into health promotion programs for older adults. Third, public health messaging should highlight the impact of sleep on aging perceptions. Helping the public recognize that adequate sleep not only benefits physical health but also helps people “feel younger” may prove more motivating than traditional health education approaches, thereby increasing the population’s commitment to sleep health.

Conclusion

Sleep problems represent a promising modifiable risk factor for older subjective age. The consistent directional evidence across diverse study designs supports a meaningful link between sleep and subjective aging, although considerable gaps remain in causal evidence, mechanistic understanding, and population generalizability. Future research should prioritize prospective longitudinal studies with objective sleep measures, randomized trials of chronic sleep interventions in middle-aged and older populations, and direct empirical testing of the proposed multi-level mechanistic framework. Clinically, incorporating sleep assessment into the evaluation of subjective aging and integrating sleep health promotion into healthy aging programs may offer a novel, actionable strategy for reducing perceived aging and its downstream health consequences.

Abbreviations

AARC, Awareness of Age-Related Change; PSQI, Pittsburgh sleep quality index; KSS, Karolinska Sleepiness Scale; HPA, hypothalamic-pituitary-adrenal.

Data Sharing Statement

No datasets were generated or analysed during the current study.

Author Contributions

Mingkun Dai: Conceptualization, Investigation, Writing – original draft, Writing – review & editing, Supervision. Xiao Kuang: Conceptualization, Validation, Writing – review & editing. Yifei Wang: Investigation, Writing – original draft, Writing – review & editing, Data curation. Wenlin Tai: Conceptualization, Resources, Writing – review & editing,

Supervision, Project administration, Funding acquisition. All authors gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work.

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Disclosure

The authors declare no competing interests.

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