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Review

Optimizing sleep in older adults: Treating insomnia

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ABSTRACT

As the world's population ages, the elevated prevalence of insomnia in older adults is a growing concern. Insomnia is characterized by difficulty falling or remaining asleep, or by non-restorative sleep, and resultant daytime dysfunction. In addition to being at elevated risk for primary insomnia, older adults are at greater risk for comorbid insomnia, which results from, or occurs in conjunction with another medical or psychiatric condition. In this review, we discuss normal changes in sleep that accompany aging, circadian rhythm changes and other factors that can contribute to late-life insomnia, useful tools for the assessment of insomnia and related problems in older people, and both non-pharmacological and pharmacological strategies for the management of insomnia and optimization of sleep in later life.

Contents

1. Introduction

The world's population of older adults is growing. In the past 50 years, the number of older adults has tripled, and it will triple again by 2050 [1]. Sleep complaints are prevalent among older adults, with over 50% reporting difficulty initiating or maintaining sleep [2]. Findings suggest that sleep complaints in older adults are due to multiple factors, including changes in circadian rhythms [3], an age-related increase in the prevalence of chronic medical conditions [4,5], and psychosocial changes that commonly accompany aging [6,7]. In addition to having an elevated prevalence of sleep complaints, older adults are more likely to have clinical levels of these complaints, and to receive a diagnosis of insomnia [8]. As the population of older adults continues to grow, so too will the prevalence of insomnia and associated conditions, making assessment and treatment increasingly important. Here, we briefly review normal age-related changes in sleep, primary and comorbid insomnia, tools for the assessment of insomnia and related problems, and pharmacological and non-pharmacological treatments for optimizing sleep in later life.

1.1. Age-related changes in sleep and circadian rhythms

Sleep is currently classified into four stages, which are differentiated by their waveforms on the electroencephalogram (EEG) and by other physiological signals. The first three stages are non-rapid eye movement (NREM) 1, 2, and 3, and the fourth stage is rapid eye movement (REM) sleep [9]. NREM 1 is the lightest stage, and accounts for 18% of older adults' sleep time [10]. Sleep deepens in NREM 2, which accounts for 48% of sleep time, and deepens further in NREM 3, which accounts for 16% of an older individual's sleep

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time and is referred to as slow-wave sleep (SWS), due to its slow (0.05–2 Hz), high-amplitude EEG signal [10]. Finally, REM sleep is referred to as "paradoxical sleep" because brain wave activity is similar to that of a waking brain, but the body is paralyzed [11]. Most dreaming occurs during REM sleep, which accounts for 18% of sleep time among older adults [10]. Beginning in middle age, adults spend less time in SWS and REM sleep and exhibit decreases in total sleep time, but sleep efficiency (the proportion of time in bed spent sleeping) continues to decrease past age 60 [12].

In addition to changes in sleep, older adults commonly demonstrate changes in *circadian rhythms*, the physiological and behavioral processes that oscillate once approximately every 24 h and are controlled by the suprachiasmatic nucleus (SCN) in the hypothalamus [13]. The sleep–wake cycle is one of these rhythms. The SCN responds to external cues, known as *zeitgebers*, such as light, which are central to synchronizing circadian rhythms to the environment [14]. Several physiological age-related changes are thought to produce alterations in circadian rhythms. For example, older adults exhibit decreases in the number and density of melatonin-, vasoactive intestinal polypeptide-, and vasopressin-expressing neurons in the SCN that may interfere with its normal function [13]. Deterioration of the SCN is believed to result in a reduction in the amplitude of core body temperature, a marker of circadian rhythms [15,16]. In addition, it is common for older adults to go to bed and wake up at earlier times than younger people. This shift is known as a circadian phase advance, and may result from the physiological changes just described and from additional factors [3].

1.2. Insomnia: definition and contributing factors

While over half of older adults report at least one sleep complaint, a smaller percentage of older adults meet clinical criteria for diagnosis of insomnia [2,8]. Insomnia can be separated into two types: "primary" and "secondary" or "comorbid." As of 2005, however, the National Institute of Health (NIH) recommended that the term "comorbid insomnia" replace "secondary insomnia" to reflect the clinical challenge in defining whether insomnia is a symptom of a primary disorder, or a separate, comorbid disorder [8]. Insomnia that initially occurs secondary to a physical or psychiatric disorder may evolve into an independent problem that has a bidirectional relationship with the original primary disorder [17]. As insomnia symptoms persist, the likelihood that cognitive and behavioral adaptations play an increasing role in the perpetuations of symptoms is enhanced. Primary insomnia is characterized by trouble initiating or maintaining sleep, or by non-restful sleep that causes impaired daytime functioning, and is not attributable to a general medical condition, medication, another sleep disorder, or a mental disorder [18]. While the etiology of primary insomnia remains unclear, both physiological and psychological theories have been proposed, including: hyperarousal, heightened physiological stress responses, predisposing personality characteristics, attitudes towards and misconceptions about sleep, and maladaptive compensatory behaviors (e.g., extending time in bed) [8].

Comorbid insomnia is similar in clinical presentation to primary insomnia but results from primary sleep disorders, medical conditions, psychiatric disturbances, medication use, and psychosocial factors associated with aging (e.g., retirement, inactivity, or caregiving) [6,7,19]. Compared to younger people, older adults have higher rates of primary sleep disorders other than insomnia, such as obstructive sleep apnea (OSA), restless legs syndrome (RLS), and periodic limb movements (PLMs), which are risk factors for insomnia symptoms [20]. OSA affects 19–57% of older adults and is characterized by repeated cessation or attenuation of breathing ("apneas" and "hypopneas", respectively) during sleep [21]. The most common OSA symptoms are loud snoring or gasping during sleep and daytime sleepiness [22]. RLS is characterized by uncomfortable sensations in the legs, marked by the urge to move one's legs, which are relieved by movement of the legs; these symptoms have a distinct circadian pattern (minimal in the morning and worse at night) making it difficult to sleep [20]. PLMs are involuntary limb jerks experienced by up to 45% of older adults, are frequently observed among individuals with RLS, and can disrupt the sleep of both affected individuals and their bed partners [23].

The bulk of insomnia symptoms in older adults may be attributable to the increased prevalence of chronic conditions in this population; only 1–7% of insomnia in later life occurs independently of chronic conditions [4,5]. A study of older adults found that approximately 25% of respondents had four or more chronic conditions, and two-thirds of these reported sleep problems [5]. Chronic pain is frequently accompanied by insomnia in older adults [5]. Pain and sleep are interrelated; pain has been shown to disrupt sleep, but sleep deprivation can also result in a decreased pain threshold [24]. Osteoarthritis (OA) is often a source of pain in older adults, affecting more than 50% of those aged 65 and older [25]. The chronic course of OA is associated with chronic comorbid insomnia in older adults [26].

Chronic insomnia is also prevalent in older adults with mood disorders, and insomnia is among the diagnostic criteria for several psychiatric disorders [18]. Insomnia is particularly associated with depression and generalized anxiety disorder (GAD)- between 40% and 60% of insomnia patients have depressive or GAD symptomatology [27]. Though insomnia can be a symptom of these disorders, it can also contribute to or exacerbate psychiatric disorders, and should be targeted for treatment when present [17].

Lifestyle changes common in old age, such as retirement, reduced mobility, and reduced social interaction are additional sources of sleep disruption [28–30]. Structured daily activities, such as work and scheduled social interactions, are thought to serve as zeitgebers [28]. It is common for older adults to become a caregiver for a family member with functional impairment—often a spouse. Caregivers, particularly those caring for a family member with dementia, have sleep patterns similar to individuals with depression or insomnia [31]. Sleep problems are more prevalent in women who are caregivers, and those experiencing greater caregiving-related distress, suggesting that caregiving may cause ruminations and anxiety while lying in bed [7,32]. Among family caregivers of older adults with dementia, disturbed care recipient sleep and related nocturnal behavior problems are burdensome and commonly cited as the reason for nursing home placement [33,34]. Recently, greater objectively measured sleep disturbance among a general population sample of older women was linked to a greater risk of functional decline and placement in a nursing or personal care home [35,36], but the mechanisms underlying this association remain unclear.

Circadian phase advance in older adults can lead to less total time in bed, greater daytime sleepiness, and more daytime napping, which can further contribute to complaints about nighttime sleep [14,15]. It has been hypothesized that age-related changes may occur downstream from the SCN (e.g., the age-related decline of behavioral and physiological rhythms may result in less effective periodic signals that are important for maintaining oscillation in peripheral tissues), and that, due to these downstream changes, circadian signals are no longer transmitted with the same strength to the pacemaker [37,38]. This results in a disrupted feedback loop, and leads to sleep complaints, and daytime sleepiness and napping [37,38]. Further, age-related yellowing of the eye's lens may restrict light input to the SCN, and reductions in input from this signal can disrupt the sleep–wake cycle [39].