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# Insomnia in the Elderly: A Review

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**Background:** Insomnia remains one of the most common sleep disorders encountered in the geriatric clinic population, frequently characterized by the subjective complaint of difficulty falling or maintaining sleep, or nonrestorative sleep, producing significant daytime symptoms including difficulty concentrating and mood disturbances.

**Methods:** A search of the literature was conducted to review the epidemiology, definition, and age-related changes in sleep, as well as factors contributing to late-life insomnia and scales utilized for the assessment of insomnia in older people. The aim is to summarize recent diagnostic guidelines and both nonpharmacological and pharmacological strategies for the management of insomnia in the older population.

**Results:** Insomnia remains a clinical diagnosis. There are several demographic, psychosocial, biologic, and behavioral factors that can contribute to late-life insomnia. Older adults are at higher risk for the medical and psychiatric effects of insomnia.

**Conclusions:** The most important aspect in evaluation of insomnia is detailed history taking and thorough physical examination. Nonpharmacological treatment options have favorable and enduring benefits compared to pharmacological therapy.

**Keywords:** cognitive behavioral therapy, elderly, insomnia, pharmacological treatment

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## SCOPE OF THE PROBLEM

The population of older adults continues to expand rapidly from the current 205 million persons aged 60 years or older, to a projected 2 billion by 2050.[1] One of the most common sleep disturbances in the older population is insomnia.[2–4] As many as 50% of older adults complain about difficulty initiating or maintaining sleep.[5] Prevalence of insomnia is higher in older individuals than in the younger population.[6] The overall prevalence of insomnia symptoms ranges from 30% to 48% in the elderly,[5,7,8] whereas the prevalence of insomnia disorder ranges from 12% to 20%.[9] Insomnia is often classified by the predominant symptom of either difficulty in sleep onset or sleep maintenance. Sleep maintenance symptoms are most prevalent among individuals with insomnia (50% to 70%), followed by difficulty in initiating sleep (35% to 60%) and nonrestorative sleep (20% to 25%).[10] A study of 6,800 older adults (age 65 years or older) observed an incidence rate for insomnia symptoms of 5% per year,[11] with a yearly incidence of 7.97% at 1-year follow-up.[12] Approximately 50% of the patients with symptoms of insomnia will have a remission during the follow-up period, with higher remission rates among older males relative to females.[12,13]

## DEFINITION

Insomnia is broadly defined as dissatisfaction with sleep either qualitatively or quantitatively. This is usually associated with one or more of the following: (1) difficulty initiating sleep, (2) difficulty maintaining sleep, characterized by frequent awakenings or problems returning to sleep after awakenings, and (3) early-morning awakening with inability to return to sleep.[14] The fifth edition of the Diagnostic and Statistical Manual for Mental Disorders (DSM-5) emphasizes that a sleep disturbance causes clinically significant distress or functional impairment, and occurs at least 3 nights a week for at least 3 months despite adequate opportunity to sleep, whereas the International Statistical Classification of Diseases and Related Health Problems, 10th revision (ICD-10) requires at least 1 month of symptoms not explained by another sleep-wake disorder, illicit substance use, or coexisting medical and psychiatric disorders. The term "nonrestorative sleep" is no longer an accepted diagnostic symptom for the DSM-5; however, it still remains in the ICD-10 criteria. The pathophysiology of insomnia disorder induces a state of hyperarousal during sleep and wakefulness. Hyperarousal is manifested as an elevated whole-body metabolic rate during sleep and wakefulness, elevated cortisol and adrenocorticotropic hormone during the early sleep period, and reduced parasympathetic tone in heart rate variability.[15] An important change with respect to diagnostic classifications was defined in the DSM-5 and the third edition of the International Classification of Sleep Disorders (ICSD-3). Insomnia in the ICSD-3 is defined as a complaint of trouble initiating or maintaining sleep that is associated with daytime consequences and is not attributable to environmental circumstances or inadequate opportunity to sleep. This replaces earlier categories of primary and secondary forms of insomnia in favor of a broad category for insomnia disorder when insomnia is comorbid with medical or psychiatric conditions.[16] In a study of 6,800 elderly patients (older than 65 years), Foley et al. demonstrated that 93% have one or more comorbid

conditions and other factors, most commonly depression, chronic pain, cancer, chronic obstructive pulmonary disease, cardiovascular diseases, medication use, and factors associated with aging (retirement, inactivity, or caregiving).[11,17–20] The increased prevalence of chronic conditions in later life may explain most insomnia symptoms in the older population; 1% to 7% of insomnia in later life occurs independently of chronic conditions.[20,21] Reduced mobility, retirement, and reduced social interaction are sources of sleep disturbances.[22–24] Caregiving may be responsible for ruminations and anxiety while trying to sleep. Women who are caregivers are found to have increased prevalence of sleep complaints.[18,25] Women are more often the primary caregivers for their children, parents, or partner, in addition to working outside of the home, affecting their total sleep time. Women are also more likely than men to complain of sleep problems and see a general practitioner for those complains.

## FACTORS CAUSING INSOMNIA

Spielman and colleagues demonstrated a three-factor model for understanding the etiology and persistence of insomnia. This model identifies predisposing, precipitating, and perpetuating factors that combine to raise the likelihood of insomnia above the insomnia threshold.[26]

### Predisposing Factors

These include demographic, biologic, psychological, and social characteristics. Women older than 45 years are 1.7 times more likely to have insomnia than men.[7] Those divorced, separated, or widowed are also more likely to have insomnia than married individuals.[7] Lower levels of education or income may contribute to insomnia in some cases.[7,27] Smoking, alcohol use, and reduced physical activity are other factors associated with higher rates of insomnia in older adults.[7,18]

### Precipitating Factors

These factors generally include stressful life events or medical conditions that may disrupt sleep. Older adults with respiratory symptoms, physical disability, and fair to poor perceived health are at increased risk of insomnia.[11] Medications such as beta blockers, glucocorticoids, nonsteroidal anti-inflammatory drugs, decongestants, and antiandrogens may be one of the factors contributing to insomnia. Several studies have demonstrated that patients with depression and generalized anxiety disorder have higher rates of insomnia.[11,13,28]

### Perpetuating Factors

These factors often consist of behavioral or cognitive changes that arise as a result of acute insomnia. An acute episode of insomnia will not necessarily develop into chronic insomnia without these provoking behavioral and cognitive events. Examples include spending excessive time in bed, frequent naps, and conditioning (increased anxiety before sleep onset due to fear of spending another sleepless night). Nonpharmacological treatment options often target these perpetuating factors.

## CHANGES IN SLEEP WITH AGING

Along with many physiologic changes seen with aging, significant changes also occur in sleep and circadian rhythm across the lifespan. Differentiated by waveforms on electroencephalogram and other physiologic signals, sleep is currently classified into four stages. The first three are non-rapid eye movement (NREM) stages: stage N1, N2, and N3 sleep. Rapid eye movement (REM) sleep occurs in the fourth stage, stage R sleep.[29] Stage N1 sleep is the lightest stage and it accounts for 18% of older adults' sleep time.[30] In stage N2 sleep, brain waves slow, body temperature begins to drop, and heart rate slows as sleep deepens, accounting for 48% of sleep time. Sleep further deepens in stage N3 sleep, characterized by very slow brain waves referred to as delta or slow wave sleep. This stage accounts for 16% of sleep.[30] Stage R sleep is "paradoxical sleep" because brain activity is similar to that in awake state with increased sympathetic tone characterized by rise in blood pressure and heart rate but with muscle atonia.[31] Dreaming occurs in this stage of sleep and accounts for 18% of sleep time in older adults.[30] Total sleep time decreases considerably from 10 to 14 hours a night in the pediatric age range, to 6.5 to 8.5 hours a night as a young adult, then decreases at a slower rate in the older age range to 5 to 7 hours a night, and plateaus at about 60 years of age.[32] The natural shortening of their total sleep time in some older adults may generate unrealistic expectations about sleep duration, producing anxiety that could cause or worsen insomnia.

Beginning in middle age, adults spend less time in slow wave sleep and REM sleep. Sleep efficiency continues to decrease past age 60 years. There is a prominent increase in wakefulness after sleep onset, but no change is observed in sleep latency.[32] It is common for healthy older adults to exhibit a temporally advanced sleep phase (falling asleep early and waking up early).[5] However, this may not be true for older adults with insomnia symptoms, who have a delayed circadian phase.[33] These individuals tend to have circadian dispersion and lack of synchronization compared to healthy subjects.[33] Early awakenings may result in frequent daytime naps, which further accentuates the problem of insomnia during the night.[34] Important time cues (zeitgebers) for circadian rhythm may erode as one ages; for example, elderly individuals may lack fixed work schedules or meal times due to retirement, which further contributes to insomnia. Healthy elderly individuals sleep as well as younger subjects according to an epidemiological study done by Ohayon.[7] Research shows that older individuals may be more tolerant of sleep deprivation than younger ones. A study on psychomotor vigilance task performance after several nights of sleep deprivation in women aged 20 to 30 years compared to older women aged 55 to 65 years found younger women had more prominent impairments with sleep deprivation compared to an older age group.[35] The American Insomnia Survey of 10,094 individuals 18 years and older noted self-reported complaints of insomnia rates were lower in older adults (older than 65 years) compared to the younger group (18 to 64 years). This highlights the importance of approaching any complaint of insomnia in the older population with more vigilance.[9]

## MORBIDITY ASSOCIATED WITH INSOMNIA

Insomnia is associated with significant morbidity if left untreated. The strongest level of evidence is for mental illness. Older individuals with insomnia have a 23% increase in risk of development of depression symptoms.[36] Several studies have documented an increased risk of depression in older patients with persistent insomnia.[37–39] A recent study noted 44% of older patients with persistent insomnia continued to have depression 6 months later as compared to only 16% of those without insomnia.[39] Insomnia and mental disorders such as depression and anxiety have a bidirectional relationship.[40] Additionally, insomnia also confers an increased risk of suicidal tedencies.[41] A meta-analysis of insomnia symptoms and its association with heart disease, after adjusting for age and other cardiovascular risk factors, identified that risk ratios for heart disease from insomnia symptoms ranged from 1.47 to 3.90.[42] Sleep loss and insomnia are associated with hypertension, myocardial infarction, and perhaps stroke.[43–46] In the Sleep Heart Health Study, a community-based cohort, adults (middle-aged and older) who reported 5 hours of sleep or less were 2.5 times more likely to have diabetes, compared with those who slept 7 to 8 hours per night.[47] Another study has also demonstrated that people with insomnia are at greater risk for metabolic syndrome.[48] Recent research also demonstrates that insomnia symptoms may lead to increased rates of cancer such as prostate cancer.[49] Long-term insomnia symptoms are also associated with greater risk of developing cognitive impairment.[50,51] A cross-sectional correlation between poor sleep quality and cortical atrophy has been shown in community-dwelling older adults.[52] Insomnia is regarded as an independent risk factor for work disability, sick leave, and reduced work performance.[53] Economically driven analyses conclude that insomnia is associated with high direct and indirect costs for the health care system and society.[54]

## DIAGNOSIS

The evaluation and diagnosis of insomnia is a clinical one, based on a thorough clinical history of the sleep problems and relevant comorbidities obtained from the patients, their partners, and/or caregivers. Evaluation of insomnia symptoms presents challenges as they may occur as a primary disorder or result from other comorbid conditions. The clinician should evaluate the nature, frequency, evolution, and duration of symptoms, as well as the response to treatment. Using various sleep diaries and questionnaires, a thorough assessment of insomnia can be achieved. A Consensus Sleep Diary that includes detailed questions can assist in obtaining additional sleep history.[55] The temporal aspects of sleep (time at which a patient goes to bed, attempts at sleep, wake-up time, and final time out of bed), quantitative aspects (sleep onset latency, number and duration of awakenings, wakefulness after sleep onset, total sleep time), and qualitative aspects (subjective sleep quality, satisfaction) should be noted. Behavioral factors, such as the use of electronic devices before going to bed, should also be addressed because these can suppress bedtime melatonin production, adversely affecting circadian rhythm.[56] Environmental factors including bedroom temperature, light intensity, sound level, and sleep patterns of the partner should also be assessed. The clinician should also inquire about symptoms generated by other sleep disorders including obstructive sleep apnea (snoring, breathing pauses), restless leg syndrome (urge to move the extremities), parasomnias (unusual sleep behaviors), and circadian rhythm disorders (unusual sleep timings). Determining the use of alcohol, caffeinated drinks, cigarettes, and any other substance that can adversely affect the quality of sleep is also very important. An insomnia evaluation should also include a history and physical examination related to medical and psychiatric disorders that can exacerbate insomnia. Neurological disorders (stroke, migraine), chronic pain, endocrine disorders (hypothyroidism/hyperthyroidism), chronic obstructive pulmonary disease, asthma, gastroesophageal reflux, and congestive heart failure can lead to or worsen insomnia. The clinician should also ask about depression, bipolar disorder, and anxiety disorders. Medication use should be reviewed, as sedatives, antidepressants, antihypertensives, steroids, and antihistamines can interfere with sleep.

### Modalities That Assist the Clinician in the Evaluation of Insomnia

#### *Wrist Actigraphy*

Wrist actigraphy, which monitors and stores movement data for up to 28 days, can be used to monitor treatment response and to screen for other circadian disorders.[57–59]

#### *Polysomnography*

Polysomnography is not recommended for the evaluation for insomnia, but contributes to the evaluation of sleep apnea or parasomnias.[60]

#### *Insomnia Rating Scales*

Numerous insomnia rating scales record symptoms and monitor the response to treatment.

The Insomnia Severity Index measures the subjective symptoms and negative outcomes of insomnia over the previous 2 weeks. On this scale, scores higher than 14 suggest "clinical insomnia."[61] The Pittsburgh Sleep Quality Index, a 19-item questionnaire, measures 7 domains of sleep over the prior month. Global scores higher than 5 indicate clinically significant sleep disturbances.[62]

#### *Imaging Studies*

Daytime imaging studies are not needed for diagnosis of insomnia; however, if performed, MRI studies detect gray matter reduction in the frontal lobes of the brain[63–65] and reduced hippocampal volume.[66–68]

## TREATMENT

If left untreated, insomnia can have multiple medical and psychological consequences, emphasizing the importance of insomnia at any age. Treatment can be divided into nonpharmacological and pharmacological options.[16,24,25,69] Aging

increases body fat, and reduces total body water and plasma proteins, resulting in increased drug elimination half-life and the potential risk of adverse effects. Older adults should therefore be treated with nonpharmacological options prior to pharmacological options.[16,70]

## Nonpharmacological

There are several nonpharmacological options for the treatment of insomnia, including relaxation techniques, improving sleep hygiene, and cognitive behavioral therapy. These options are effective in managing insomnia for extended periods of time, even in patients with cognitive impairment.[71]

### *Sleep Hygiene Education*

Education regarding sleep hygiene consists of several interventions that promote healthy stable sleep and a nondisruptive sleep environment. These include avoiding daytime naps, maintaining a regular sleep schedule, limiting substances such as caffeinated beverages, nicotine, and alcohol that adversely affect sleep, and exercising at least 6 hours before bedtime.[72,73]

### *Cognitive Behavioral Therapy for Insomnia*

When sleep hygiene is not effective, Cognitive Behavioral Therapy for Insomnia (CBT-I), effective in older adults, should be attempted.[73–75] The American College of Physicians recommends CBT-I as first-line management for insomnia in adults.[76] It consists of 6 to 10 sessions with a trained therapist that focus on cognitive beliefs and counterproductive behaviors that interfere with sleep.

### *Sleep Restriction Therapy*

This therapy involves restricting time in bed to the number of hours of actual sleep, until sleep efficiency improves. If after 10 days sleep efficiency remains lower than 85%, sleep time in bed should be restricted by 15 to 30 minutes until sleep efficiency improves. Time in bed is gradually advanced by 15 to 30 minutes when the time spent asleep exceeds 85% of total time in bed.[77]

### *Stimulus Control Therapy*

This therapy attempts to reassociate use of the bed and the desired bedtime to sleep only. This includes going to bed only when one feels tired, not using bed for reading, working, or lounging, leaving the bed if unable to sleep in 15 to 20 minutes, and maintaining a constant wake-up time each morning.[78]

### *Relaxation Techniques*

These include progressive muscle tensing and relaxing, guided imagery, paced diaphragmatic breathing, or meditation.[25]

### *Brief Behavioral Therapy for Insomnia*

Due to financial constraints and lack of psychological resources needed for CBT-I, a shorter form of therapy known as brief behavioral therapy for insomnia is also available and involves core techniques from CBT-I, directed at improving circadian regulation of sleep in more than two sessions. It has been found to be effective in the geriatric population, with benefits persisting for 6 months and beyond.[79] Internet-based behavioral therapies have also been found to be effective in older populations.[80] Multicomponent cognitive behavioral therapy that involves sleep hygiene measures, relaxation techniques, sleep restriction, and stimulus control is also as effective in older adults as a stand-alone treatment.[81,82]

## Pharmacological

There are several pharmacological options available for use in older patients with insomnia. Pharmacological treatments are primarily classified as benzodiazepine sedatives, nonbenzodiazepine sedatives, melatonin receptor agonists, antidepressants, and orexin receptor antagonists. Recently published clinical practice guidelines of the American Academy of Sleep Medicine for the pharmacological treatment of chronic insomnia represent an evidence-based review of each class of drug commonly used in the treatment of insomnia.[83]

### *Benzodiazepines and Nonbenzodiazepine Sedatives*

Both benzodiazepines and nonbenzodiazepine receptor agonists have a common mechanism of action. They work by binding to a specific receptor site on gamma-aminobutyric acid type A receptors, with the difference being nonbenzodiazepines are more selective for the alpha-1 subclass of receptors, which while causing sedation has minimal anxiolytic, amnesic, and anticonvulsant effects compared to that of benzodiazepines.[84] Both classes of drugs effectively treat insomnia-related parameters such as sleep onset latency, number of nighttime awakenings, total sleep time, and sleep quality in the short term, but not with chronic use.[85–87]

Prolonged use of these drugs can lead to tolerance, dependence, rebound insomnia, residual daytime sedation, motor incoordination, cognitive impairment, and increased risk of falls in institutionalized older individuals.[88] These drugs can have additive effects if taken together. Because of these adverse effects, and the equivalent or superior response seen with CBT-I for longer duration therapy, use of these drugs should be avoided in older individuals. The recent 2015 Beers criteria strongly advise avoiding these drugs in the elderly.[89]

Pharmacokinetic properties of these drugs dictate the differences between the drug effects on sleep parameters. Zolpidem has a shorter half-life (2 to 3 hours) and so may have less potential for residual daytime adverse events than zopiclone, which has a longer half-life (5 to 6 hours). However, the shorter half-life of zolpidem renders it less useful in treating sleep maintenance insomnia. These drugs have a faster onset of action and therefore can be used in treating sleep onset insomnia (reduce sleep onset latency).[84]

Although the benefits outweigh the harms, there have been reports of impairment in daytime concentration tasks, such as driving while on zopiclone.[90,91] In women, in whom zolpidem clearance occurs more slowly than in men, morning blood levels following the recommended previous bedtime dose could be considerably higher, affecting psychomotor performance.[92] In 2013, this led the United States Food and Drug Administration (FDA) to require the manufacturers of zolpidem to lower the recommended dose, particularly for women, from 10 mg to 5 mg for immediate-release preparations, and 12.5 mg to 6.5 mg for extended-release forms. It also required manufacturers to lower the recommended doses for men.

### Antidepressants

Various antidepressants, including phenylpiperazine compounds (trazodone), tricyclic antidepressants (doxepin), and serotonergic antidepressant (mirtazapine), have sedating properties and are often used for the treatment of insomnia.

Trazodone: It is widely prescribed for insomnia in doses of 25 to 100 mg. A study on trazodone comparing its effect with zolpidem in 21 to 65 year olds showed it has similar efficacy for sleep latency and sleep efficiency, with these effects dissipating after the first week.[93] Adverse events such as dizziness, cardiac arrhythmias, orthostatic hypotension, and potential priapism can be significant in the elderly population.[94] Clinical practice guidelines from the American Academy of Sleep Medicine suggest that clinicians not use trazodone for sleep onset or maintenance insomnia because its harms outweigh benefits.[83]

**Doxepin:** Of all the antidepressants, only doxepin is FDA approved for insomnia at doses of 3 to 6 mg. It is selective for histamine 1 receptors. Studies of men and women age 65 years and older with doses of 1 mg and 3 mg have shown that doxepin 1 mg and 3 mg significantly improved measures of sleep onset (patient reported), sleep duration, sleep quality, and global treatment outcomes over the 12-week study period.[95] Higher doses of doxepin 3 mg and 6 mg to adults (18 to 64 years) with chronic primary insomnia has also been reported to lead to significant and sustained improvements in sleep maintenance and early morning awakenings.[96]

**Mirtazapine:** This antidepressant with strong 5-HT2 antagonism may also improve insomnia. In a study of adults age 18 to 75 years old with mean age of 40.9 years, the mirtazapine group had significant improvement in sleep latency, sleep efficiency, and awakenings after sleep onset after only 2 weeks of treatment.[97] It may be preferred over other drugs as it produces sedative effects solely through histamine receptor antagonism.[97] Because of conflicting evidence and habituation to its sedative effects, it should not be used to treat insomnia in the absence of depression.

### Melatonin Receptor Agonists

**Ramelteon:** It is also FDA approved for treatment of insomnia. In a study of older adults (age 65 years or older), treatment with ramelteon significantly reduced patient reports of sleep latency over 5 weeks of treatment with no significant rebound insomnia or withdrawal effects.[98] It is not associated with dependence, memory disturbances, and nocturnal gait instability in older individuals.[99,100]

### Herbal Supplements

**Valerian:** As a dietary supplement it lacks FDA approval and monitoring. Its mechanism of action is believed to occur through interaction with the neurotransmitter gamma aminobutyric acid and its receptors. There are limited studies of valerian in elderly individuals and data are lacking in terms of its efficiency in treating insomnia.

**Melatonin:** Melatonin at a dose of 2 mg has been approved in Europe for the short-term treatment of insomnia in patients age 55 years and older based on decline in melatonin production seen with age.[101] Treatment has been shown to be effective for primary insomnia in some studies; however, formal recommendations for the use of melatonin in the treatment of insomnia requires further research.[83,101,102]

### Orexin Receptor Antagonists

**Suvorexant:** It is the first FDA-approved dual orexin receptor antagonist and may be prescribed up to a 20-mg dose. It targets wakefulness-promoting neuropeptides that regulate the sleep-wake cycle, demonstrating its efficacy in decreasing sleep latency and in increasing total sleep time. Suvorexant has been studied in both elderly (age 65 years or older) and non-elderly (age 18 to 64 years) patients, identifying no significant efficacy or safety differences between these two groups.[103] Although it is well tolerated by older adults, long-term data are still lacking.[103–105]

## CONCLUSIONS

Insomnia is very prevalent in older adults. Using the history and physical examination along with insomnia scales, clinicians can evaluate and treat insomnia in our rapidly aging population. Behavioral and cognitive behavioral therapies offer very effective longer duration treatment and are recommended as first-line treatment options for insomnia compared to hypnotic medications in older adults.

## ABBREVIATIONS

CBT-I, cognitive behavioral therapy for insomnia
DSM, Diagnostic and Statistical Manual for Mental Disorders
FDA, United States Food and Drug Administration
ICD, International Statistical Classification of Diseases and Related Health Problems
ICSD, International Classification of Sleep Disorders
NREM, non-rapid eye movement
REM, rapid eye movement

## REFERENCES

1. United Nations Department of Economics and Social Affairs: Population Division. World population ageing: 1950–2050. http://www.un.org/esa/population/publications/worldageing19502050/. Published 2001. Accessed May 23, 2017.
2. Morin CM, Benca R. Chronic insomnia. *Lancet.* 2012; 379(9821):1129–1141.
3. Shochat T, Loredo J, Ancoli-Israel S. Sleep disorders in the elderly. *Curr Treat Options Neurol.* 2001;3(1):19–36.
4. Foley DJ, Monjan AA, Brown SL, Simonsick EM, Wallace RB, Blazer DG. Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep.* 1995;18(6):425–432.
5. Crowley K. Sleep and sleep disorders in older adults. *Neuropsychol Rev.* 2011;21(1):41–53.
6. Klink ME, Quan SF, Kaltenborn WT, Lebowitz MD. Risk factors associated with complaints of insomnia in a general adult population. Influence of previous complaints of insomnia. *Arch Intern Med.* 1992;152(8):1634–1637.

7. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev.* 2002;6(2):97–111.
8. Hillman DR, Murphy AS, Pezzullo L. The economic cost of sleep disorders. *Sleep.* 2006;29(3):299–305.
9. Roth T, Coulouvrat C, Hajak G, et al. Prevalence and perceived health associated with insomnia based on DSM-IV-TR; International Statistical Classification of Diseases and Related Health Problems, Tenth Revision; and Research Diagnostic Criteria/International Classification of Sleep Disorders, Second Edition criteria: results from the America Insomnia Survey. *Biol Psychiatry.* 2011;69(6):592–600.
10. Buysse DJ. Insomnia. *JAMA.* 2013,309(7):706–716.
11. Foley, DJ, Monjan A, Simonsick EM, Wallace RB, Blazer DG. Incidence and remission of insomnia among elderly adults: an epidemiologic study of 6,800 persons over three years. *Sleep.* 1999;22(Suppl 2):S366–S372.
12. Gureje O, Oladeji BD, Abiona T, Lebowitz MD. The natural history of insomnia in the Ibadan study of ageing. *Sleep.* 2011;34(7):965–973.
13. Foley DJ, Monjan AA, Izmirlian G, Hays JC, Blazer DG. Incidence and remission of insomnia among elderly adults in a biracial cohort. *Sleep.* 1999;22(Suppl 2):S373–S378.
14. American Psychiatric Association. *Diagnostic and Statistical Manual of Mental Disorders.* 5th ed. Arlington, VA: American Psychiatric Publishing; 2013.
15. Riemann D, Spiegelhalder K, Feige B, et al. The hyperarousal model of insomnia: a review of the concept and its evidence. *Sleep Med Rev.* 2010;14(1):19–31.
16. NIH State-of-the-Science Conference Statement on manifestations and management of chronic insomnia in adults. *NIH Consensus State Sci Statements.* 2005;22(2):1–30.
17. Castro CM, Lee K, Bliwise DL, Urizar GG, Woodward SH, King AC. Sleep patterns and sleep-related factors between caregiving and non-caregiving women. *Behav Sleep Med.* 2009;7(3):164–179.
18. Morgan K. Daytime activity and risk factors for late-life insomnia. *J Sleep Res.* 2003;12(3):231–238.
19. American Academy of Sleep Medicine. *International Classification of Sleep Disorders.* Darien, IL: American Academy of Sleep Medicine; 2014.
20. Foley D, Ancoli-Israel S, Britz P, Walsh J. Sleep disturbances and chronic disease in older adults: results of the 2003 National Sleep Foundation Sleep in America Survey. *J Psychosom Res.* 2004;56(5):497–502.
21. Vitiello MV, Moe KE, Prinz PN. Sleep complaints cosegregate with illness in older adults: clinical research informed by and informing epidemiological studies of sleep. *J Psychosom Res.* 2002;53(1):555–559.
22. Aschoff J, Fatranska M, Giedke H, Doerr P, Stamm D, Wisser H. Human circadian rhythms in continuous darkness: entrainment by social cues. *Science.* 1971;171(3967):213–215.
23. Benloucif S, Orbeta L, Ortiz R, et al. Morning or evening activity improves neuropsychological performance and subjective sleep quality in older adults. *Sleep.* 2004;27(8):1542–1551.
24. Naylor E, Penev PD, Orbet L, et al. Daily social and physical activity increases slow wave sleep and daytime neuropsychological performance in the elderly. *Sleep.* 2000;23(1):87–95.
25. McCurry SM, Logsdon RG, Teri L, Vitiello MV. Evidence-based psychological treatments for insomnia in older adults. *Psychol Aging.* 2007;22(1):18–27.
26. Spielman AJ, Caruso LS, Glovinsky PB. A behavioral perspective on insomnia treatment. *Psychiatr Clin North Am.* 1987;10(4):541–553.
27. Patel NP, Grandner MA, Xie D, Branas CC, Gooneratne N. "Sleep disparity" in the population: poor sleep quality is strongly associated with poverty and ethnicity. *BMC Public Health.* 2010;10:475.
28. Brenes GA, Miller ME, Stanley MA, Williamson JD, Knudson M, McCall WV. Insomnia in older adults with generalized anxiety disorder. *Am J Geriatr Psychiatry.* 2009;17(6):465–472.
29. Iber C, Ancoli-Israel S, Chesson AL Jr, Quan SF; for the American Academy of Sleep Medicine. *The AASM Manual for the Scoring of Sleep and Associated Events: Rules, Terminology, and Technical Specifications.* 1st ed. Westchester, IL: American Academy of Sleep Medicine; 2007.
30. Moser D, Anderer P, Gruber G, et al. Sleep classification according to AASM and Rechtschaffen & Kales: effects on sleep scoring parameters. *Sleep.* 2009;32(2):139–149.
31. Siegel JM. REM Sleep. In: Kryger MH, Roth T, Dement WC, eds. *Principles and Practices of Sleep Medicine.* 5th ed. St. Louis, MO: Elsevier; 2010.
32. Ohayon, MM, Carskadon MA, Guilleminault C, Vitiello MV. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep.* 2004;27(7):1255–1273.
33. Youngstedt SD, Kripke DF, Elliott JA, Klauber MR. Circadian abnormalities in older adults. *J Pineal Res.* 2001;31(3):264–272.
34. Ancoli-Israel S, Ayalon L, Salzman C. Sleep in the elderly: normal variations and common sleep disorders. *Harv Rev Psychiatry.* 2008;16(5):279–286.
35. Stenuit P, Kerkhofs M. Age modulates the effects of sleep restriction in women. *Sleep.* 2005;28(10):1283–1288.
36. Jaussent I, Bouyer J, Ancelin ML, et al. Insomnia and daytime sleepiness are risk factors for depressive symptoms in the elderly. *Sleep.* 2011;34(8):1103–1110.
37. Perlis ML, Smith LJ, Lyness JM, et al. Insomnia as a risk factor for onset of depression in the elderly. *Behav Sleep Med.* 2006;4(2):104–113.
38. Cole MG, Dendukuri N. Risk factors for depression among elderly community subjects: a systematic review and meta-analysis. *Am J Psychiatry.* 2003;160(6):1147–1156.
39. Pigeon WR, Hegel M, Unutzer J, et al. Is insomnia a perpetuating factor for late-life depression in the IMPACT cohort? *Sleep.* 2008;31(4):481–488.
40. Jansson-Frojmark M, Lindblom K. A bidirectional relationship between anxiety and depression, and insomnia? A prospective study in the general population. *J Psychosom Res.* 2008;64(4):443–449.
41. Pigeon WR, Pinquart M, Conner K. Meta-analysis of sleep disturbance and suicidal thoughts and behaviors. *J Clin Psychiatry.* 2012;73(9):e1160–e1167.
42. Schwartz S, McDowell Anderson W, et al. Insomnia and heart disease: a review of epidemiologic studies. *J Psychosom Res.* 1999;47(4):313–333.
43. Palagini L, Bruno RM, Gemignani A, Baglioni C, Ghiadoni L, Riemann D. Sleep loss and hypertension: a systematic review. *Curr Pharm Des.* 2013;19(13):2409–2419.
44. Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hypertension. *Sleep.* 2009;32(4):491–497.
45. Bradley TD, Logan AG, Kimoff RJ, et al. Continuous positive airway pressure for central sleep apnea and heart failure. *N Engl J Med.* 2005;353(19):2025–2033.
46. Laugsand LE, Vatten LJ, Platou C, Janszky I., Insomnia and the risk of acute myocardial infarction: a population study. *Circulation.* 2011;124(19):2073–2081.
47. Gottlieb DJ, Punjabi NM, Newman AB, et al. Association of sleep time with diabetes mellitus and impaired glucose tolerance. *Arch Intern Med.* 2005;165(8):863–867.
48. Troxel WM, Buysse DJ, Matthews KA, et al. Sleep symptoms predict the development of the metabolic syndrome. *Sleep.* 2010;33(12):1633–1640.
49. Sigurdardottir LG, Valdimarsdottir UA, Mucci LA, et al. Sleep disruption among older men and risk of prostate cancer. *Cancer Epidemiol Biomarkers Prev.* 2013;22(5):872–879.
50. Cricco M, Simonsick EM, Foley DJ. The impact of insomnia on cognitive functioning in older adults. *J Am Geriatr Soc.* 2001;49(9):1185–1189.
51. Yaffe K, Falvey CM, Hoang T. Connections between sleep and cognition in older adults. *Lancet Neurol.* 2014;13(10):1017–1028.
52. Sexton CE, Storsve AB, Walhovd KB, Johansen-Berg H, Fjell AM. Poor sleep quality is associated with increased cortical atrophy in community-dwelling adults. *Neurology.* 2014;83(11):967–973.
53. Kucharczyk ER, Morgan K, Hall AP. The occupational impact of sleep quality and insomnia symptoms. *Sleep Med Rev.* 2012;16(6):547–559.
54. Leger D, Bayon V. Societal costs of insomnia. *Sleep Med Rev.* 2010;14(6):379–389.
55. Carney CE, Buysse DJ, Ancoli-Israel S, et al. The consensus sleep diary: standardizing prospective sleep self-monitoring. *Sleep.* 2012;35(2):287–302.
56. Wood B, Rea MS, Plitnick B, Figueiro MG. Light level and duration of exposure determine the impact of self-luminous tablets on melatonin suppression. *Appl Ergon.* 2013;44(2):237–240.

57. Bloom HG, Ahmed I, Alessi CA, et al. Evidence-based recommendations for the assessment and management of sleep disorders in older persons. *J Am Geriatr Soc*. 2009;57(5):761–789.
58. Buysse DJ, Ancoli-Israel S, Edinger JD, Lichstein KL, Morin CM. Recommendations for a standard research assessment of insomnia. *Sleep*. 2006;29(9):1155–1173.
59. Morgenthaler T, Alessi C, Friedman L, et al. Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: an update for 2007. *Sleep*. 2007;30(4):519–529.
60. Littner M, Hirshkowitz M, Kramer M, et al. Practice parameters for using polysomnography to evaluate insomnia: an update. *Sleep*. 2003;26(6):754–760.
61. Bastien CH, Vallieres A, Morin CM. Validation of the Insomnia Severity Index as an outcome measure for insomnia research. *Sleep Med*. 2001;2(4):297–307.
62. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res*. 1989;28(2):193–213.
63. Altena E, Vrenken H, Van Der Werf YD, Van den Heuvel OA, Van Someren EJ. Reduced orbitofrontal and parietal gray matter in chronic insomnia: a voxel-based morphometric study. *Biol Psychiatry*. 2010;67(2):182–185.
64. Stoffers D, Moens S, Benjamins J, et al. Orbitofrontal gray matter relates to early morning awakening: a neural correlate of insomnia complaints? *Front Neurol*. 2012;3:105.
65. Joo EY, Noh HJ, Kim JS, et al. Brain gray matter deficits in patients with chronic primary insomnia. *Sleep*. 2013;36(7):999–1007.
66. Joo EY, Kim H, Suh S, Hong SB. Hippocampal substructural vulnerability to sleep disturbance and cognitive impairment in patients with chronic primary insomnia: magnetic resonance imaging morphometry. *Sleep*. 2014;37(7):1189–1198.
67. Neylan TC, Mueller SG, Wang Z, et al. Insomnia severity is associated with a decreased volume of the CA3/dentate gyrus hippocampal subfield. *Biol Psychiatry*. 2010;68(5):494–496.
68. Riemann D, Voderholzer U, Spiegelhalder K, et al. Chronic insomnia and MRI measured hippocampal volumes: a pilot study. *Sleep*. 2007;30(8):955–958.
69. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. *Am J Psychiatry*. 2002;159(1):5–11.
70. Rieman D, Perlis ML. The treatments of chronic insomnia: a review of benzodiazepine receptor agonists and psychological and behavioral therapies. *Sleep Med Rev*. 2009;13(3):205–214.
71. Shub D, Darvishi R, Kunik ME. Non-pharmacologic treatment of insomnia in persons with dementia. *Geriatrics*. 2009;64(2):22–26.
72. Montgomery P, Dennis J. Cognitive behavioural interventions for sleep problems in adults aged 60+. *Cochrane Database Syst Rev*. 2002(2):CD003161.
73. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia: a randomized controlled trial. *JAMA*. 1999;281(11):991–999.
74. Alessi C, Vitiello MV. Insomnia (primary) in older people. *BMJ Clin Evid*. 2011;2011:2302.
75. Montgomery P, Dennis J. A systematic review of non-pharmacological therapies for sleep problems in later life. *Sleep Med Rev*. 2004;8(1):47–62.
76. Qaseem A, Kansagara D, Forciea MA, Cooke M, Denberg TD; Clinical Guidelines Committee of the American College of Physicians. Management of chronic insomnia disorder in adults: a clinical practice guideline from the American College of Physicians. *Ann Intern Med*. 2016;165(2):125–133.
77. Spielman AJ, Saskin P, Thorpy MP. Treatment of chronic insomnia by restriction of time in bed. *Sleep*. 1987;10(1):45–56.
78. Bootzin RR, Epstein D, Wood JM. *Case Studies in Insomnia: Stimulus Control Instruction*. New York, NY: Plenum Medical; 1991.
79. Buysse DJ, Germain A, Moul DE, et al. Efficacy of brief behavioral treatment for chronic insomnia in older adults. *Arch Intern Med*. 2011;171(10):887–895.
80. Cheng SK, Dizon J. Computerised cognitive behavioural therapy for insomnia: a systematic review and meta-analysis. *Psychother Psychosom*. 2012;81(4):206–216.
81. Epstein DR, Sidani S, Bootzin RR, Belyea MJ. Dismantling multicomponent behavioral treatment for insomnia in older adults: a randomized controlled trial. *Sleep*. 2012;35(6):797–805.
82. Irwin MR, Cole JC, Nicassio PM. Comparative meta-analysis of behavioral interventions for insomnia and their efficacy in middle-aged adults and in older adults 55+ years of age. *Health Psychol*. 2006;25(1):3–14.
83. Sateia MJ, Buysse DJ, Krystal AD, Neubauer DN, Heald JL. Clinical practice guideline for the pharmacologic treatment of chronic insomnia in adults: an American Academy of Sleep Medicine clinical practice guideline. *J Clin Sleep Med*. 2017;13(2):307–349.
84. Jacobson SA. *Clinical Manual of Geriatric Psychopharmacology*. 2nd ed. Washington, DC: American Psychiatric Publishing; 2014.
85. Morin CM, Vallieres A, Guay B, et al. Cognitive behavioral therapy, singly and combined with medication, for persistent insomnia: a randomized controlled trial. *JAMA*. 2009;301(19):2005–2015.
86. Wu R, Bao J, Zhang C, Deng J, Long C. Comparison of sleep condition and sleep-related psychological activity after cognitive-behavior and pharmacological therapy for chronic insomnia. *Psychother Psychosom*. 2006;75(4):220–228.
87. Jacobs GD, Pace-Schott EF, Stickgold R, Otto MW. Cognitive behavior therapy and pharmacotherapy for insomnia: a randomized controlled trial and direct comparison. *Arch Intern Med*. 2004;164(17):1888–1896.
88. Berry SD, Lee Y, Cai S, Dore DD. Nonbenzodiazepine sleep medication use and hip fractures in nursing home residents. *JAMA Intern Med*. 2013;173(9):754–761.
89. American Geriatrics Society Beers Criteria Update Expert Panel. American Geriatrics Society 2015 updated Beers Criteria for potentially inappropriate medication use in older adults. *J Am Geriatr Soc*. 2015;63(11):2227–2246.
90. Leufkens TR, Ramaekers JG, De Weerd AW, Riedel WJ, Vermeeren A. Residual effects of zopiclone 7.5 mg on highway driving performance in insomnia patients and healthy controls: a placebo controlled crossover study. *Psychopharmacology*. 2014;231(14):2785–2798.
91. Leufkens TR, Vermeeren A. Highway driving in the elderly the morning after bedtime use of hypnotics: a comparison between temazepam 20 mg, zopiclone 7.5 mg, and placebo. *J Clin Psychopharmacol*. 2009;29(5):432–438.
92. Farkas RH, Unger EF, Temple R. Zolpidem and driving impairment--identifying persons at risk. *N Engl J Med*. 2013;369(8):689–691.
93. Walsh JK, Erman M, Erwin CW, et al. Subjective hypnotic efficacy of trazodone and zolpidem in DSMIII-R primary insomnia. *Human Psychopharmacology*. 1998;13(3):191–198.
94. Mendelson WB. A review of the evidence for the efficacy and safety of trazodone in insomnia. *J Clin Psychiatry*. 2005;66(4):469–476.
95. Krystal AD, Durrence HH, Scharf M, et al. Efficacy and safety of doxepin 1 mg and 3 mg in a 12-week sleep laboratory and outpatient trial of elderly subjects with chronic primary insomnia. *Sleep*. 2010;33(11):1553–1561.
96. Krystal AD, Lankford A, Durrence HH, et al. Efficacy and safety of doxepin 3 and 6 mg in a 35-day sleep laboratory trial in adults with chronic primary insomnia. *Sleep*. 2011;34(10):1433–1442.
97. Winokur A, DeMartinis NA 3rd, McNally DP, Zary EM, Cormier JL, Gary KA. Comparative effects of mirtazapine and fluoxetine on sleep physiology measures in patients with major depression and insomnia. *J Clin Psychiatry*. 2003;64(10):1224–1229.
98. Roth T, Seiden D, Sainati S, Wang-Weigand S, Zhang J, Zee P. Effects of ramelteon on patient-reported sleep latency in older adults with chronic insomnia. *Sleep Med*. 2006;7(4):312–318.
99. Zammit G, Wang-Weigand S, Rosenthal M, Peng X. Effect of ramelteon on middle of the night balance in older adults with chronic insomnia. *J Clin Sleep Med*. 2009;5(1):34–40.
100. Mets MA, de Vries JM, de Senerpont Domis LM, Volkerts ER, Olivier B, Verster JC. Next-day effects of ramelteon (8 mg), zopiclone (7.5 mg), and placebo on highway driving performance, memory functioning, psychomotor performance, and mood in healthy adult subjects. *Sleep*. 2011;34(10):1327–1334.
101. Wade AG, Crawford G, Ford I, et al. Prolonged release melatonin in the treatment of primary insomnia: evaluation of the age cut-off for short- and long-term response. *Curr Med Res Opin*. 2011;7(1):87–98.

102. Riemersma-van der Lek RF, Swaab DF, Twisk J, Hol EM, Hoogendijk WJ, Van Someren EJ. Effect of bright light and melatonin on cognitive and noncognitive function in elderly residents of group care facilities: a randomized controlled trial. *JAMA*. 2008;299(22):2642–2655.

103. Rhyne DN, Anderson SL. Suvorexant in insomnia: efficacy, safety and place in therapy. *Ther Adv Drug Saf*. 2015;6(5):189–195.

104. Owen RT. Suvorexant: efficacy and safety profile of a dual orexin receptor antagonist in treating insomnia. *Drugs Today*. 2016;52(1):29–40.

105. Norman JL, Anderson SL. Novel class of medications, orexin receptor antagonists, in the treatment of insomnia - critical appraisal of suvorexant. *Nat Sci Sleep*. 2016;8:239–247.

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## DISCLOSURE STATEMENT

All authors have seen and approved the manuscript. The authors report no conflicts of interest.