

# REVIEW

# Diagnosis and management of geriatric insomnia: A guide for nurse practitioners

Preetha Krishnan, RN, BN, MN (Nurse Practitioner)<sup>1</sup> & Pamela Hawranik, RN, PhD (Associate Professor)<sup>2</sup>

1 Winnipeg Regional Health Authority, Long Term Care, Personal Care Home Program, Winnipeg, Manitoba, Canada 2 Faculty of Nursing, University of Manitoba, Winnipeg, Manitoba, Canada

#### Keywords

Geriatric insomnia; sleep disorder; pharmacological management; nonpharmacological management.

#### Correspondence

Preetha Krishnan, RN, BN, MN, Winnipeg Regional Health Authority, Nurse Practitioner, Lions Personal Care Centre, 320 Sherbrook Street, Winnipeg, Manitoba, Canada, R3B 2W6. Tel: 204-784-2710; Fax: 204-784-2723; E-mail: preethakri@gmail.com

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#### Abstract

**Purpose:** To discuss the assessment, diagnosis, and management of geriatric insomnia, a challenging clinical condition of older adults frequently seen by primary care providers.

**Data sources:** Extensive literature review of the published research articles and textbooks.

**Conclusions:** Complaints of insomnia among older adults are frequently ignored, considered a part of the normal aging process or viewed as a difficult to treat condition. Geriatric insomnia remains a challenge for primary care providers because of the lack of evidence-based clinical guidelines and limited treatment options available. Effective management of this condition is necessary for improved quality of life, which is a primary issue for the elderly and their families. Therefore, geriatric insomnia warrants thorough attention from the nurse practitioners (NPs) who provide care for older adults.

**Implications for practice:** Undiagnosed or under treated insomnia can cause increased risk for falls, motor vehicle accidents, depression, and shorter survival. Insomniacs double their risk for cardiovascular disease, stroke, cancer, and suicide compared to their counterparts. Insomnia is also associated with increased healthcare utilization and institutionalization. NPs could play a central role in reducing the negative consequences of insomnia through a systematic approach for diagnosis, evaluation, and management.

# Introduction

Sleep problems are common with aging and occur in over 50% of adults aged 65 and older (Ancoli-Israel, 2005). Sleep patterns change with advancing age, but sleep disorders are not an inevitable part of aging. The major sleep complaints among older adults include difficulty initiating and maintaining sleep, excessive daytime sleepiness, waking feeling unrested, waking too early, or frequent nocturnal waking. Disordered sleep in older adults may result in depression, falls, attention and memory impairment, slowed response time, lowered quality of life and performance, misdiagnosis as dementia, and possible institutionalization (Ancoli-Israel & Cooke, 2005; Cooke & Ancoli-Israel, 2006; Hidalgo et al., 2007). The causes of

geriatric insomnia are multifactorial. In order to effectively assess, diagnose, and intervene, the nurse practitioner (NP) should understand the sleep changes that occur with aging, the potential causes, the consequences of disordered sleep, and the types of management strategies and their effectiveness. The assessment and treatment options for older adults who are 65 years and older and who reside in the community will be discussed.

# Epidemiology

Sleep complaints are common in all ages, but their prevalence increases with age (Ancoli-Israel & Ayalon, 2006). In a survey of over 9000 community-dwelling adults conducted by the National Institute of Aging,

28% of the participants reported difficulty in initiating sleep, while 42% had difficulty both initiating and maintaining sleep (Ancoli-Israel, 2000). In the United States, 8 million older adults have insomnia at any given day, with approximately 1 million new cases occurring per year and nearly 1.3 million individuals experiencing a resolution of symptoms annually (Doghramji, 2006). The annual incidence of insomnia in older adults is estimated at 5% (Kamel & Gammack, 2006). The prevalence of insomnia is higher in older adults who have poor health and require medications (Ancoli-Israel & Cooke, 2005). Prevalence is higher in elderly women, who are separated, divorced, or widowed (Ohayon, 2002), but the prevalence increases in elderly men 85 years and older (Doghramji).

# Sleep and aging

Sleep architecture, the progression of sleep across a night, changes with age. Sleep can be divided into two stages, the nonrapid eye movement (NREM) and rapid eye movement (REM) stages (Ancoli-Israel, 2000; Ancoli-Israel & Cooke, 2005). As sleep progresses, NREM and REM stages cycle with a periodicity of 90–120 min. The NREM sleep is further divided into four stages, stages 1 through 4; stage 1 is the lightest level and stage 4 is the deepest level of sleep. Stages 3 and 4 are also called delta sleep or slow wave sleep (SWS). SWS is considered the most restorative part of sleep. Most of SWS occurs in the early part of the night, and most REM sleep occurs in the early morning hours (Kamel & Gammack, 2006).

Studies indicate that older adults have less SWS and REM sleep. They spend most of their night in stages 1 and 2, the lighter sleep (Ancoli-Israel, 2000; Ancoli-Israel & Ayalon, 2006; Ancoli-Israel & Cooke, 2005). These changes in the sleep architecture result in decreased quality and efficiency of sleep, reduced total sleep time in combination with an increased fragmentation of sleep, and frequent and early morning awakening.

The circadian rhythm (24-h physiological rhythm) is controlled by an endogenous pacemaker located in the suprachiasmatic nucleus of the anterior hypothalamus (Mathews, Kumar, Mathews, & Ramachandaran, 2004). Sleep-wake cycles, hormone secretions, core body temperature, and blood pressure observe a circadian rhythm. External cues such as light synchronize circadian rhythms to the 24-h day (Ancoli-Israel & Cooke, 2005). Loss of neurons that occurs with advancing age results in circadian rhythm disturbances and development of desynchronization between the circadian pacemaker and the environmental cues. In older adults, the sleep-wake circadian rhythm shifts and this condition is called advanced sleep phase syndrome (ASPS). This change in sleep-wake cycle is believed to be because of changes in the core body temperature, diminished light exposure, and according to recent research involvement of genetics (Ancoli-Israel & Cooke). Older adults with ASPS experience early evening sleepiness and early morning wakefulness because of early evening dropping of core body temperature and rising of body temperature at dawn. ASPS is not a medical disorder. It is an expected circadian rhythm shift with older age. No treatment is necessary for ASPS unless it affects the individual's day-to-day life (Ancoli-Israel & Cooke).

Older adults have more difficulty initiating sleep and spend more time in bed awake after retiring. These alterations in sleep patterns most often cause fatigue, daytime sleepiness, and frequent napping during the day, which eventually lead to reversal of the sleep-wake cycle (Kamel & Gammack, 2006). Older adults are more prone to sleep deprivation because of the alteration in normal sleep patterns along with other physical, social, and psychological factors.

#### **Definition and classification**

Insomnia is a subjective clinical diagnosis. The fourth edition of the Diagnostic and Statistical Manual of Mental Disorders defines insomnia as (a) difficulty initiating sleep (sleep onset), maintaining sleep (sleep maintenance), and/ or poor quality of sleep (nonrestorative sleep) for at least 1 month and (b) causing clinically significant distress or impairment in social, occupational, or other areas of functioning (American Psychiatric Association, 1994). Insomnia can be divided into acute (less than or equal to 4 weeks) and chronic (more than 4 weeks) depending upon the duration. Acute insomnia tends to be associated with acute illness, hospitalization, changes in sleeping environment, jet lag, or acute or recurring psychosocial stressors (Kamel & Gammack, 2006). Chronic insomnia is divided into primary and secondary insomnia. Primary insomnia includes sleep-disordered breathing (SDB)/sleep apnea, restless leg syndrome (RLS)/periodic limb movement disorder, and circadian dysfunction (Ancoli-Israel, 2004). Secondary insomnia arises out of underlying medical or psychiatric disorders or medication effects. Older adults tend to suffer from both primary and secondary insomnia (Schneider, 2002).

# **Causes of insomnia in older adults**

Insomnia is a symptom, not a diagnosis. Geriatric insomnia has multifactorial causes. The common causes of insomnia in older adults include primary sleep disorders, medical and psychiatric illness, adverse effects of drugs/ medications, psychosocial factors, and behavioral and environmental factors (Figure 1).



#### Figure 1 Causes of insomnia.

*Note.* BPH, benign prostatic hyperplasia; CHF, congestive heart failure; COPD, chronic obstructive pulmonary disease; CNS, central nervous system; CRD, circadian rhythm dysfunction; CV, cardiovascular; GERD, gastroesophageal reflux disease; GI, gastrointestinal; GU, genitourinary. Ancoli-Israel and Ayalon (2006); Ancoli-Israel (2004); Bain (2006); Kamel and Gammack (2006); Mathews et al. (2004); Zee (2006).

SDB, periodic leg movement disorder or syndrome, RLS, and rapid eye movement behavior disorder (RBD) are the most common primary sleep disorders in older adults. SDB is characterized by partial (hypopneas) and complete cessation of respiration (apneas) during sleep, lasting 10 or more seconds (Mathews et al., 2004). The prevalence of SDB in older adults is approximately 45%-60% compared to 4%–9% in middle-aged adults. SDB is more common in older men than older women (Ancoli-Israel & Ayalon, 2006). The major symptoms of SDB are snoring and excessive daytime sleepiness. Snoring is associated with airway collapse, and daytime sleeping is because of repeated nighttime awakenings (Mathews et al.). The daytime sleepiness negatively impacts on social and occupational functions. It also results in cognitive deficits, including decreased concentration, slowed response time, and memory and attention difficulties (Ancoli-Israel & Cooke,

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2005). SDB may actually exacerbate cognitive deficits experienced by older adults. SDB is a risk factor for hypertension and cardiac and pulmonary problems, all of which may shorten survival in this population (Ancoli-Israel & Cooke).

Periodic leg movement syndrome (PLMS) is characterized by repeated leg jerks every 20–40 s, resulting in brief awakenings with each jerk throughout the night. PLMS is associated with difficulty falling and staying asleep and increased daytime sleepiness (Ancoli-Israel & Ayalon, 2006). PLMS is diagnosed when more than five leg jerks per hour are detected (Mathews et al., 2004). The estimated PLMS prevalence in older adults is 45% compared to 5%–6% in younger adults (Ancoli-Israel & Ayalon). Often, RLS coexists with PLMS. RLS is described by the sufferers as "a creeping, crawling sensation or as pins and needles" (p 99), which usually occur in a restful relaxed state and can be relieved only by movement (Ancoli-Israel & Cooke, 2005). RLS incidence increases with age, resulting in 5%–15% of older adults being affected. RLS affects women twice as frequently as men. Approximately 80% of elderly who have RLS have PLMS, but only 30% of PLMSaffected older adults have RLS (Ancoli-Israel & Cooke, 2005). RLS can be associated with anemia, uremia, and peripheral neuropathy (Mathews et al.).

RBD is characterized by complex motor behavior that occurs in the second half of sleep when REM is more predominant (Mathews et al., 2004). The complex motoric behaviors include vigorous body movements and activities such as walking, talking, and kicking, which may harm the individual or the bed partner. Most often, the person is unable to recollect the events in the morning. RBD is more common in men, and the typical age of onset is the sixth or seventh decade of life (Ancoli-Israel & Ayalon, 2006). Studies indicate that acute RBD is associated with the use of tricyclic antidepressants, fluoxetine, monoamine oxidase inhibitors, and alcohol and sedative withdrawal (Ancoli-Israel & Ayalon; Mathews et al.). The etiology of chronic RBD is unknown, but it is associated with certain neurodegenerative disorders such as Parkinson's disease and Lewy body dementia (Ancoli-Israel & Cooke, 2005).

#### **Consequences of insomnia**

Sleep complaints in older adults are often considered a normal part of aging. However, geriatric insomnia is associated with reduced quality of life, increased healthcare utilization, and institutionalization (Ancoli-Israel & Cooke, 2005). Impaired sleep in older adults and the subsequent disruption of their caregivers' sleep can be a primary factor in the decision for institutionalization of an elder. More than 50% of long-term care admissions have been directly attributed to sleep disturbances (McCall, 2004). Chronic insomnia can result in psychological distress in older adults and is associated with difficulty in sustaining attention, slowed responses, diminished cognitive ability, daytime sleepiness, and decreased memory (Hidalgo et al., 2007). The slowed responses and decreased attention may increase the likelihood of falls, fractures, and automobile accidents (Ancoli-Israel, 2000). These insomnia-induced cognitive impairments can lead to the misdiagnosis of dementia. Sleep disturbances are a risk factor for falls, which is a strong predictor of long-term care placement (Latimer Hill, Cumming, Lewis, Carrington, & Le Couteur, 2007). Falls and related injuries significantly increase morbidity and mortality in older adults.

The most common consequences of insomnia are psychiatric disorders such as depression and anxiety (Cooke & Ancoli-Israel, 2006). Studies indicate older adults with unresolved insomnia are more likely to develop depression and vice versa (Ancoli-Israel, 2004; Kryger, Bliwise, & Ancoli-Israel, 2004). Impaired sleep is also associated with reduced pain threshold and insulin resistance (Roth, 2004). Older insomniacs have shorter survival and they are two times more likely to die from heart disease, stroke, cancer, and suicide compared to their counterparts who sleep well (Ancoli-Israel & Cooke, 2005). Inability to enjoy family and social relationships, increased incidence of pain, sense of poor health, and decreased ability to accomplish daily tasks are some other impairments associated with insomnia. Insomnia is an important clinical entity with a potentially significant impact on older adults and society (Walsh, 2004).

## Assessment

Evidence supporting the association between good sleep and good health has long been established; therefore, sleep should be considered a vital sign and sleeping habits should be reviewed with older adults during the office visit. Most often, older adults do not voluntarily discuss their sleep problems with the clinicians, so clinicians should initiate the discussion and assess insomnia during the annual physical examination, the review of chronic medical conditions and treatment options, and their primary care visit for other concerns (Culpepper, 2005). Because of the complex multidimensionality of geriatric insomnia, a thorough evaluation including a comprehensive history and physical examination, along with a sleep log/diary and structured questionnaire are necessary for the accurate diagnosis (Table 1) (Holcomb, 2006).

The history will allow the NP to discover concomitant medical and psychiatric disorders, polypharmacy, or other behavioral/environmental conditions that contribute to geriatric insomnia. Information from the bed partner is also useful and can shed light on diagnosis (McCall, 2004). Global sleep and insomnia symptoms can be assessed with the Pittsburg Sleep Quality index, the Epworth Sleepiness Scale, or the American Academy of Sleep Medicine's Sleep quiz. The Beck Depression Inventory is useful in identifying older adults who have an underlying depressive disorder that contributes to insomnia (McCall, 2005). While the sleep history provides an essential retrospective overview, the sleep diary yields night-by-night information on perceptions of sleep patterns and quality. Sleep diaries have become the staple of sleep assessment and are valuable in the assessment of insomnia as well as its treatment outcomes (Lichstein & Morin, 2000).

The initial workup for insomnia does not routinely include diagnostic tests, unless the clinicians suspect sleep disorders like SDB, PLMS, or circadian rhythm dysfunction. Polysomnography is a valuable test when the history

Insomnia Assessment						
Sleep History						
Bedtime Lights off time and is it dark? Bedroom environment (temperature, quiet, etc.) Leg symptoms Pain Partner snoring Other problems Minutes to sleep onset		<ul> <li>Sleep</li> <li>Number of awakenings</li> <li>Why awakened</li> <li>How long to get back to sleep</li> <li>Wake up too early</li> <li>Nocturia</li> <li>Pain, palpitations, difficulty breathing, heartburn, etc.</li> <li>Nightmares</li> <li>Movement</li> <li>Restless leg</li> <li>Do you snore</li> </ul>		<ul> <li>Daytime function</li> <li>Rested upon arising</li> <li>Driving ability</li> <li>Attention/concentration</li> <li>Cataplexy</li> <li>Naps</li> <li>Fall asleep watching TV or reading after dinner</li> </ul>		
Sleep Log or Diary Keeping a daily record of time to bed, time to sleep, number of awakenings, why, when back to sleep, time to fully awake, daytime functioning, naps, social factors, etc. Kept for 1 to 2 weeks. Should be completed in the morning for the previous day and not during the day or night if awakenings occur.						
R = restorative sleep	E = exces	sive daytime	S = snoring		T = total nightly sleep time	
Sleep/Psychological Assessment Scales         Pittsburg Sleep Quality Index       Epworth Sleepiness Scale         Beck Depression Inventory						
Social Factors						
Occupational Shift work Rotating Weekends Travel	Exercise Exercise Relaxa Yoga Medita	se Caffeine tion Tobacco Alcohol ation Cocaine				
Medications						
<ul> <li>Adrenergic agonists</li> <li>Amphetamines</li> <li>Some antibiotics, especially quinolones</li> <li>Anticonvulsants</li> </ul>	<ul> <li>Antidepressants</li> <li>Antihypertensives</li> <li>Cancer agents</li> <li>Decongestants</li> <li>Diuretics</li> </ul>		<ul> <li>Glucocorticoids</li> <li>Hypnotics</li> <li>Levodopa</li> <li>Niacin</li> <li>Oral contraceptivity</li> </ul>	cocorticoidsImage: StimulantsnoticsImage: SympathomimeticsodopaImage: TheophyllinecinImage: Thyroid replacemeI contraceptivesImage: Sympathomimetics		
Medical/Psychological Factors	6					
<ul> <li>Asthma</li> <li>Rheumatoid diseases</li> <li>Angina – coronary artery disease</li> <li>Cystic fibrosis</li> </ul>		<ul> <li>Menopausal symptoms</li> <li>Thyroid problems</li> <li>Gastroesophogeal reflux disease</li> <li>Irritable bowel syndrome</li> </ul>		<ul> <li>Chronic renal or liver failure</li> <li>Anxiety</li> <li>Depression</li> </ul>		
General						
<ul> <li>When did insomnia start?</li> <li>How many nights per week, month, etc., do you have insomnia?</li> <li>Any factors that influence insomnia such as weather, season, menstrual, etc.?</li> <li>How long between dinner and sleep?</li> <li>Do you get anxious about being able to sleep as bedtime gets closer?</li> </ul>		<ul> <li>Are you sleepy when you go to bed?</li> <li>What do you do if you cannot sleep?</li> <li>Do you often look at the clock?</li> <li>If you did not sleep well, and can sleep longer, do you stay in bed longer the next day?</li> <li>Does your sleep/rise routine change on days off, the weekend, holidays, etc.?</li> </ul>		<ul> <li>Previous history of insomnia?</li> <li>Have you previously seen someone about insomnia?</li> <li>What have you tried (OTC, prescribed medications, non- pharmacologic treatments, etc.)?</li> <li>Has anything worked?</li> </ul>		

Note. Holcomb, 2006, reprinted with permission.

suggests SDB or PLMS (Avidan, 2005). The Multiple Sleep Latency Test objectively measures daytime napping, a consequence of insomnia. The wrist actigraphy can be used to record body movement, which provides objective measures of sleep-wake cycles and can be used in the diagnosis of circadian rhythm disorders (Avidan). Blood tests have limited use in the diagnosis of insomnia except serum iron and renal function tests, which will rule out whether RLS is secondary to iron deficiency or renal failure (McCall, 2005; Wolkove, Elkholy, Baltzan, & Palayew, 2007).

## **Differential diagnosis**

The National Institutes of Health (NIH) highlighted the importance of distinguishing insomnia from normal aging and a number of sleep disorders. Conditions such as SDB, PLMS/RLS, circadian rhythm dysfunctions, insomnia associated with medical/psychiatric disorders, extrinsic sleep disorders because of exogenous causes like medication effect, and behavioral/environmental conditions must be considered and differentiated from insomnia (NIH State-ofthe-Science Conference Statement on manifestations and management of chronic insomnia in adults, 2005).

#### Management of insomnia

A National Sleep Foundation poll conducted in 2003 found that about 20% of older adults took something to help them sleep at least a few nights per week. Eleven percent reported taking prescription medication, 6% used over-the-counter (OTC) medications, such as diphenhydramine and other antihistamines, and about 6% used alcohol (Ancoli-Israel, 2005). Insomnia in older adults may have more than one underlying cause or contributing factor. Because of the high prevalence of comorbidities and use of concomitant prescription medication, it is imperative that the primary treatment goal of insomnia should identify and address any underlying condition as well as polypharmacy and potential drug-drug interactions within the existing medication regimen (McCall, 2004). Setting realistic expectations and reinforcing good sleep habits along with treatment of any underlying condition should be the initial intervention for insomnia in older adults. The goal of treatment includes at least improvement on nocturnal complaints and maintenance, if not enhancement of daytime functioning. Attainment of a long uninterrupted period of sleep may not be a satisfactory outcome if the intervention causes excessive daytime sleepiness (McCall, 2005).

# Nonpharmacologic treatments

The primary focus of nonpharmacologic strategies in the treatment of insomnia is aimed at correcting behaviors that

are not conducive to healthy sleeping (Bain, 2006). In an attempt to develop nonpharmacologic practice guidelines for managing insomnia, the American Academy of Sleep Medicine revealed that nonpharmacologic therapies produce reliable and long-lasting positive outcomes in chronic insomniacs (Benca, 2005). Holcomb (2006) suggests nonpharmacologic interventions can improve sleep and decrease insomnia in 70%-80% of the sufferers. Evidence also suggests that nonpharmacologic therapies, especially behavioral therapies, are as effective as pharmacotherapy and should be considered as the first-line intervention for chronic insomnia, especially in older adults who are at high risk for adverse drug reactions (Ancoli-Israel, 2000; Kamel & Gammack, 2006). Bain points out that nonpharmacological interventions are most often underused because of health providers' lack of awareness and training.

Cognitive behavioral therapy (CBT) tends to be the most widely used psychological nonpharmacological intervention for insomnia (Bain, 2006). CBT is a combination of stimulus control and/or sleep restriction plus cognitive restructuring, relaxation, and good sleep hygiene (Table 2) (Ancoli-Israel & Ayalon, 2006). Stimulus control therapy and sleep restriction are considered to be the most effective single therapies, whereas sleep hygiene education is not effective as a single therapy (Bain). The advantages of CBT include durability and lack of dependency or rebound insomnia (Ancoli-Israel & Ayalon). The disadvantages include noncompliance with adherence, take along time (at least 2 weeks) for effectiveness, and difficulty in obtaining reimbursement (Bain). Considering the safety profile and less expensive nature, the behavioral therapies should be given high priority and implemented as first line in the treatment of geriatric insomnia and be continued even when pharmacotherapy is needed (Kamel & Gammack, 2006)

# **Pharmacological treatments**

Pharmacological treatments should only be considered for chronic insomnia, when the older adult cannot or will not comply with nonpharmacological interventions or when treatment of the primary cause is insufficient to alleviate the insomnia (McCall, 2005). The pharmacokinetic and pharmacodynamic changes in drug metabolism that occur with the aging process should also be considered when choosing safe and effective pharmacotherapy for the elderly (McCall, 2004). The decision to initiate pharmacotherapy should be based on the presence and severity of daytime symptoms, particularly their impact on the older adult's quality of life (Ancoli-Israel, 2000; Kamel & Gammack, 2006). The basic principles of rational pharmacotherapy for geriatric insomnia include: use of lowest effective dose, use of intermittent dosing (two to four times

Table 2	Nonpharmacol	logical	interventions
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Therapy	Objective	Description
Stimulus control	To improve healthy sleep habits through reassociation	<ol> <li>Use bedroom for sleep and sex only</li> <li>Go to bed only when sleepy</li> <li>Leave the bedroom when unable to sleep after 20 min, engage in relaxing activities until drowsy, and then return to bed</li> <li>Maintain regular morning rise time daytime</li> <li>Napping should be limited to 30 min in early afternoon</li> </ol>
Sleep hygiene education	To educate about the importance of healthy behaviors that promote enhanced sleep	<ol> <li>Regular exercise (not within 3–4 h of bedtime)</li> <li>Avoid tobacco, stimulants, caffeine, and alcohol 4–6 h before bed</li> <li>Avoid excessive liquids and heavy meals 2–3 h before bed</li> <li>Avoid napping late in the day</li> <li>Maintain routine sleep/wake schedule 7 days a week</li> <li>Make sure the bedroom is dark, comfortable temperature, and quiet</li> <li>Avoid unpleasant task right before bedtime, read or think about pleasant thoughts before trying to sleep, avoid stimulating movies before bedtime</li> <li>Avoid watching the clock</li> <li>Use of low-impact activities such as board game and gentle stretching to improve sleep quality</li> </ol>
Sleep restriction	To improve sleep efficiency through mild sleep deprivation (sleep debt)	<ol> <li>Instruct to maintain a sleep log and determine average total sleep time</li> <li>Allowable time in bed is decreased to the amount of time usually spend sleeping (but not less than 5 h)</li> <li>After 90% sleep efficiency attained, increase sleep time by 15 min</li> <li>If sleep efficiency is 80% or less, decrease sleep time by 15 min</li> <li>Wake time is kept constant and bedtime is adjusted</li> <li>Older adults are allowed to have a short afternoon nap</li> </ol>
Relaxation therapies (progressive muscle relaxation, meditation, abdominal breathing, imaginary training, electromyography/ electroencephalography biofeedback)	To decreases the occurrences of physiological, cognitive, or emotional arousals to allow for sleep initiation or to reduce sleep latency	<ol> <li>Progressive muscle relaxation focused to reduce physiological arousal by alternating contraction and relaxation of muscle groups throughout the body</li> <li>Intended to produce relaxation and inhibit anxiety-associated arousal that could inhibit sleep</li> <li>Meditation focused on psychological arousal</li> <li>In biofeedback, devices are used to amplify physiological processes (e.g., blood pressure, muscle activity)</li> </ol>
Paradoxical intention	To improve sleep through cognitive restructuring (reverse psychology)	<ol> <li>Advise to remain awake in order to decrease any performance anxiety that could interfere with the ability to fall asleep</li> <li>Encourage to engage in the feared behavior of staying awake</li> </ol>
Cognitive Behavioral Therapy	To provide a sense of control and self-efficacy over sleep	<ol> <li>Identify dysfunctional beliefs and attitudes about sleep</li> <li>Replace those with more adaptive substitutes (decatastrophizing, reappraisal, and attention shifting are some specific techniques used as adaptive substitutes)</li> </ol>

Note. Bain (2006); Holcomb (2006); McCall (2005); NIH State-of-the-Science Conference Statement on Manifestations and Management of Chronic Insomnia in Adults (2005); Petit, Azad, Byszewski, Sarazan, and Power (2003); University of Texas at Austin School of Nursing, Family Nurse Practitioner Program-Academic Institution (2005).

weekly), short-term use of drugs (no more than 3-4 weeks) that have shorter elimination half-lives and daytime sedation, and are amenable to gradual discontinuation without causing rebound insomnia (Kamel & Gammack; Mathews et al., 2004). Medications that are available without prescription (OTC) and with prescription can be used for insomnia.

# Nonprescription medications

Commonly used nonprescription medications include antihistamines, melatonin, and valerian (Holcomb, 2006; Kamel & Gammack, 2006; Turkoski, 2006). Many insomniacs self-medicate with OTC products before seeking medical advice because of their availability and inexpensiveness (Ancoli-Israel, 2005; Bain, 2006). A study

conducted in adults aged 60 years and older found that the self-prescribed therapies included alcohol (13%), antihistamines (36%), and dietary supplements (11%) (Bain). The most commonly used OTC antihistamines for insomnia are diphenhydramine or doxylamine (Bain; Kamel & Gammack). Studies have indicated those antihistamines are generally inappropriate for use in elderly because of their anticholinergic properties such as urinary retention, mental confusion, dry mouth, and constipation (Ancoli-Israel, 2005; Bain; Kamel & Gammack). The other adverse effects of antihistamines, which can appear after 3 days include residual sedation, impaired psychomotor and cognitive function, blurred vision, orthostatic hypotension, dizziness, palpitations, and medication tolerance (Ancoli-Israel, 2005). According to the NIH State of Science Conference on Insomnia in Adults, there is no scientific evidence on the safety and efficacy of antihistamines as hypnotics in the elderly, but there are significant concerns about their adverse effects. They are not recommended for treating insomnia (Ancoli-Israel, 2005; McCall, 2004).

Melatonin, a neurohormone secreted primarily at night by the pineal gland is important in the sleep-wake cycle (Holcomb, 2006). Melatonin secretion decreases with age and may be partly responsible for insomnia in older adults (Ringdahl, Pereira, & Delzell, 2004). Synthetic versions of the naturally occurring melatonin are available OTC and have been shown to improve sleep efficiency and reduce wake time after sleep onset (McCall, 2005). However, quality control data on the purity, appropriate dosage, and administration time of this product are lacking; furthermore, OTC melatonin is not approved by the Food and Drug Administration (FDA) for treatment of geriatric insomnia (Kamel & Gammack, 2006).

Valerian, also known as nature's valium, is a herbal preparation derived from the root of a plant, Valeriana. Its sedative effect is attributed to the possible gamma amino butyric acid (GABA) like properties (Holcomb, 2006; Ringdahl et al., 2004). It is available OTC and generally recognized as safe (GRAS) in the United States for food use and is officially in the European pharmacopoeia (Ringdahl et al.). Scientific data regarding its safety is lacking and valerian is not approved by the FDA for the treatment of insomnia (Turkoski, 2006).

#### **Prescription medications**

FDA-approved prescription treatment for insomnia includes five benzodiazepines (BDZs), four non-BDZs, and Ramelteon, a melatonin receptor agonist as sedative hypnotics (Table 3) (Bain, 2006; Turkoski, 2006).

BDZs are shown to improve insomnia by shortening REM sleep, decreasing sleep latency, decreasing nocturnal awakenings, and increasing total sleep time (Kamel & Gammack, 2006; Ringdahl et al., 2004). Their

Tab	le	3	FD/	A-approved	medications	tor	geriatric	insomnia
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Mechanism Half-life (h) Geriatric name Geriatric Onset of Active Duration Special metabolites (Brand name) of action dose in mg action (h) (range) of action (h) considerations BD7s BDZ receptor agnosit Estazolam (Prosom) 15(10-24)05 - 11 - 26 - 10No 8.8 (3.5-18.4) 6-10 Temazepam (Restoril) 7.5-15 1 - 2Rule out sleep apnea No before prescribing Triazolam (Halcion) 0.125 2.5 (1.5-5.5) 1 - 2No 2 - 5Non-BDZs BDZ receptor agonist Eszolpiclone (Lunesta) 1 - 20.5 - 16 (5-7) 5-8 The only approved No medication for long-term use Zaleplon (Sonata) 5 Administered on waking 0.25 - 0.5No 1 (0.9-1.1) 2-4 during the night Zolpidem (Ambien) 5 0.25-0.5 No 2.5 (1.4-4.5) 3-8 Zopiclone (Imovane) 3.5-7.5 0.25-0.5 yes 5 (3.5-6.5) 3-8 The only non-BDZ available in Canada, not available in the United States Ramelteon (Rozerem) 8 0.25-0.5 Caution should be taken Melatonin receptor Yes 2(1-2.6)6-8 agonist with hepatic/renal diseases

Note. Ancoli-Israel (2005, 2004); Bain (2006); Holcomb (2006); Silber (2005); Wolkove et al. (2007). BDZ, benzodiazepine.

nonselective binding to BDZ2 and BDZ3 receptor subtypes results in anxiolytic, muscle relaxant, and anticonvulsant properties (Dolder, Nelson, & McKinsey, 2007; Ringdahl et al.). The ideal BDZs that can be used in older adults should have a rapid onset of action with no active metabolites, a short half-life, and minimal drug–drug interaction (Bain, 2006). Flurazepam and quazepam have active metabolites and longer half-lives and therefore should be avoided for the treatment of geriatric insomnia (Bain; Turkoski, 2006).

The key in BDZ selection is to consider the pharmacokinetic profile that matches the sleeping complaints. The choice of BDZ for difficulty initiating sleep is a medication with rapid onset of action, such as triazolam. An intermediate acting BDZ such as temazepam or estazolam is the choice of treatment for nocturnal or early morning awakenings (Bain, 2006). BDZs are not associated with an increased risk of mortality in the elderly. However, their increased sensitivity to BDZ can result in adverse effects, especially with chronic use, higher dosage, and long-acting agents (McCall, 2005). The most common adverse effects of BDZ treatment include daytime sedation, dizziness, falls, orthostatic hypotension, anterograde amnesia, and accidents (Kamel & Gammack, 2006; Silber, 2005). Longterm use of BDZs is associated with rebound insomnia, dependency, and withdrawal; therefore, their chronic use is not recommended in older adults with chronic insomnia (Holcomb, 2006).

#### Nonbenzodiazepine sedative hypnotics

Currently, there are four non-BDZs approved by the FDA for treatment of insomnia (Bain, 2006). Compared to BDZs, non-BDZ agents have selective binding affinity for receptor subtypes and therefore their anticonvulsant, muscle relaxant, and anxiolytic properties are lacking at sedative hypnotic dosages (Bain). The primary efficacy of non-BDZs is sleep onset rather than sleep maintenance (Benca, 2005). They increase sleep latency and total sleep time, but they do not decrease REM sleep, especially zolpidem (Ringdahl et al., 2004). These sedative hypnotics have lower risk for tolerance, rebound insomnia, daytime sleepiness, and drug-drug interaction (McCall, 2004). Common side effects of non-BDZs include headache, dizziness, nausea, and somnolence. The risk of falls associated with non-BDZ use in older adults is low compared to BDZs but they have been known to produce negative effects on balance, gait, and equilibrium (Dolder et al., 2007).

Ramelteon is a selective melatonin agonist that works on receptors in the suprachiasmatic nucleus, which regulates the circadian rhythms including the wake-sleep cycle. The MT-1 receptor induces sleep and the MT-2 receptor regulates circadian rhythm between day, wake, and night sleep. Ramelteon, a sedative hypnotic approved by the FDA for long-term use does not contain any controlled substances (Holcomb, 2006). It improves sleep latency and sleep maintenance. It also has adverse effects such as headache, nausea, and fatigue (Bain, 2006). Daytime grogginess, rebound insomnia, and withdrawal are non-extant with this agent (Kamel & Gammack, 2006). Drug-drug interactions are also minimal (Dolder et al., 2007).

Indiplon is a non-BDZ pyrazolopyrimidine sedative hypnotic agent not yet approved by the FDA but has completed all phase I–III clinical trials. It is considered to be useful for treating difficulties associated with the onset and maintenance of sleep (Bain, 2006). Gaboxadol, a GABA receptor agonist, is under investigation and is claimed to improve sleep duration and quality and to decrease sleep awakenings (Holcomb, 2006).

#### Sedative antidepressants

Off-label use of some sedative antidepressants is also prevalent for insomnia treatment (Turkoski, 2006). Trazodone is a serotonin reuptake inhibitor and is the most commonly prescribed agent for insomnia other than zolpidem (Benca, 2005). Even though scientific evidence about its efficacy and safety and FDA approval as a hypnotic is lacking, it is reported to be one of the most sedating antidepressants that increases SWS. At low doses, trazodone may be beneficial for older adults with psychotropic-induced insomnia, monoamino oxidase inhibitor-induced insomnia, or contraindications for BDZ use (Kamel & Gammack, 2006). The common side effects include drowsiness, tiredness, gastrointestinal disorders, headache, orthostatic hypertension, agitation, tachycardia, and falls (Bain, 2006; Kamel & Gammack). Priapism is a rare but serious side effect of trazodone and is considered a urological emergency (Bain).

# Monitoring and follow-up of pharmacologic treatment

Follow-up with the older adult within the first week of treatment is essential to assess the effectiveness as well as adverse effects of medications (Zee, 2006). Older adults should be seen approximately every 2 weeks while changes are being made. Encourage them to keep sleep diaries and report the results of therapy in every visit (Ancoli-Israel & Ayalon, 2006). If insomnia persists despite the treatments, reevaluate for secondary causes. A referral may be needed for assessment of severe depression, anxiety, or sleep-related disorders (Zee).

# Conclusions

In conclusion, recognizing that multifactorial etiologies contribute to sleep complaints in older adults improves our ability to treat insomnia appropriately in order to improve both the night sleep and the daytime performance. The key for NPs is to find the appropriate treatment or treatment combinations, including both nonpharmacological and pharmacological therapies. Proper management of insomnia has the potential to reverse insomnia-related morbidities, including depression, disability, and impaired quality of life (Kamel & Gammack, 2006). Timely identification and management of geriatric insomnia can increase productivity and cognition and decrease healthcare use, institutionalization, and risk of falls and accidents.

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# **Conflict of interest disclosure**

The authors report no conflicts of interest related to the contents of this article.

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