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REVIEW

Tackling sleeplessness: psychological treatment options for insomnia in older adults

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Abstract: This paper provides a broad review of the extant literature involving the treatment of sleeplessness in older adults with insomnia. First, background information (including information regarding key issues in late-life insomnia and epidemiology of late-life insomnia) pertinent to achieving a general understanding of insomnia in the elderly is presented. Next, theories of insomnia in older adults are examined and discussed in relation to treatment of insomnia in late-life. With a general knowledge base provided, empirical evidence for both pharmacological (briefly) and psychological treatment options for insomnia in late-life are summarized. Recent advances in the psychological treatment of insomnia are provided and future directions are suggested. This review is not meant to be all-inclusive; however, it is meant to provide professionals across multiple disciplines (physicians; psychologists; applied and basic researchers) with a mix of breadth and depth of knowledge related to insomnia in late-life. It is our hope that readers will see the evidence in support of psychological treatments for late-life insomnia, and the utility in continuing to investigate this treatment modality.

Keywords: insomnia, elderly, older adults, geriatric, sleep

Introduction

With prevalence rates as high as 60%, insomnia is the most common sleep disturbance in older adults.[1] Insomnia in the elderly is typically more chronic and comorbid in nature than insomnia in younger adults.[2] Further, insomnia in late-life is associated with a myriad of quality of life and societal consequences.[3–6] Thus, a comprehensive understanding of the nature, consequences, conceptualization, and treatment options for this disorder in older adults is urgently needed. This review will provide information about the epidemiology, presentation, conceptualization, and treatment of late-life insomnia. Both psychological and pharmacological treatment options will be reviewed; however, psychological treatments will be described and discussed in greater detail. Lastly, future directions and areas needing additional investigation will be suggested.

Key issues in late-life insomnia

While definitions vary across different nosological systems,[7–9] insomnia is generally defined as a subjective difficulty initiating or maintaining sleep, or sleep that is non-restorative in quality, that is associated with daytime impairment and occurs at least 3 nights per week for a period of one month or longer. This definition highlights the subjective nature of the sleep complaint, its interference with normal functioning,

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the frequency of the sleep difficulty, and the persistence of the problem. Understanding sleep complaints among older adults within the context of normal age-related changes in sleep architecture is important. As people age, there are normative decreases in rapid eye movement (REM) sleep and delta "deep sleep," or slow-wave sleep (SWS), which are accompanied by an increase in lighter, less restorative, Stage 1 sleep.[10,11] Additionally, due to changes in circadian rhythms and homeostatic mechanisms, many older adults experience an earlier shift in their sleep–wake preferences.[10,12,13] In other words, they tend to feel sleepy earlier in the evening and wake earlier in the mornings compared to during their younger- and middle-adult years. Behavioral and environmental factors, such as irregular schedules, reduced exposure to light, decreased exercise, and increased daytime napping, may further increase the risk for disturbed sleep among older adults. Thus, many factors may be contributing to the complaint of poor sleep in late-life. These factors will be discussed in greater detail below.

Within older adults, the clinical picture of insomnia is often complex. Careful assessment of the insomnia complaint is needed to obtain a detailed understanding of the sleep problem, as well as to identify or rule out potential comorbid physical and mental conditions, including the possibility of other undetected sleep disorders. The presence of several physical conditions, such as pain, cardiovascular conditions, menopause, and prostate problems can increase the risk for developing insomnia.[14] Therefore, asking patients about physical health problems is an important component of an evaluation for insomnia. Additionally, it is essential to obtain a complete list of medications to identify possible pharmacological contributors to insomnia symptoms, such as beta-blockers, central nervous system stimulants, corticosteroids, antidepressants, bronchodilators, calcium channel blockers, decongestants, stimulating antihistamines, and thyroid hormones, all of which are known to produce sleep complaints in the elderly.[10]

A thorough clinical interview will likely provide information about the specific insomnia complaint and can identify symptoms that suggest underlying sleep, mental, and physical disorders that may be contributing to said complaint. As the comorbidity between insomnia and mental/psychological disorders, such as depression and anxiety are high,[1,15] it is important to thoroughly assess for the presence of these conditions within routine insomnia evaluations in elderly patients. Use of validated self-report measures, such as the Geriatric Depression Scale (GDS),[16] State-Trait Anxiety Inventory (STAI),[17] and Epworth Sleepiness Scale (ESS),[18] are useful to assess for symptoms of depression, anxiety, and excessive daytime somnolence.[19,20] Of note, research has shown that the prevalence rate of insomnia among older adults drops significantly when comorbid mental and physical conditions are controlled.[15,21] However, the practice of calculating prevalence rates after controlling for potential comorbid conditions should be viewed with caution as insomnia should not typically be conceptualized as 'secondary' to another disorder.[22] In fact, it is unlikely that insomnia symptoms will completely dissipate following the treatment of the 'primary' disorder.[23] Insomnia has been found to persist in a large proportion of individuals (44%–88%), following standard treatments addressing the 'primary' medical or psychological condition.[24,25]

While not routinely recommended as part of the assessment for insomnia, polysomnography (PSG) can be used to diagnose the presence of additional sleep disorders, such as obstructive sleep apnea (OSA) or periodic limb movement disorder (PLMD). Furthermore, PSG may have increased utility in the elderly due to the finding of reduced usefulness of symptom-based questions for detection of occult sleep disorders, as well as the increased comorbidity of such disorders with insomnia among older adults.[26–28] Sleep diaries are a key assessment instrument, providing a daily record of individuals' subjective sleep patterns over a recommended 2-week period. They can also be used throughout treatment to document treatment effects.[29]

Epidemiology of late-life insomnia

Epidemiological studies describing prevalence rates for insomnia in older adults have varied widely, depending upon how the construct of insomnia was defined. For example, in studies defining insomnia in terms of a subjective sleep complaint, without reference to specific frequency or duration criteria or the presence of daytime dysfunction, estimates have been reported as high as 30%–60%.[1,15] However, in studies using more restrictive criteria, including both a requirement of daytime dysfunction and a specific frequency/duration threshold for the subjective sleep complaint, estimates have been lower and along the lines of 12%–25%.[7,30] One consistent finding across the literature is that insomnia tends to increase with age,[15,31] and late-life insomnia also tends to be more chronic and severe than insomnia in younger adults.[2] For individuals aged 65 or older, the one year incidence rate for development of insomnia has been reported as 3.1%–7.3%.[2] Rates of insomnia are also known to be more frequent among women in the general population,[32,33] and this remains true among populations of older adults.[33,34]

Unfortunately, many older adults hold the inaccurate belief that poor sleep is a normal part of the aging process.[1] While normative age-related changes in sleep architecture and homeostatic processes do occur, insomnia is not normative at any age, and randomized-controlled trials (RCTs) and meta-analyses have concluded that it can be successfully treated in older adults.[35,36] Oftentimes, older adults attempt to self-treat their insomnia condition with alcohol or over-the-counter (OTC) sleep aids. The National Sleep Foundation's 2003 poll reported that 6% of older adults use OTC sleep aids at least a few nights per week and 6% of older adults use alcohol at least a few nights per week to self-treat sleep problems. Unfortunately, these strategies often serve to exacerbate the sleep problem over time.

When older adults seek professional treatment for sleep problems, they are more likely to present to a primary care setting rather than a specialty sleep clinic,[29,37] with elderly women being more likely than elderly men to present with insomnia complaints in medical settings.[2,30,32,38] As insomnia becomes more severe and chronic, the likelihood increases that individuals will bring up this concern with their health care provider.[2] Late-life insomnia is associated with increased severity, chronicity, and comorbidities, which raises the likelihood that individuals will eventually discuss this problem with their medical provider.[2] Currently, medications are typically the first-line treatment provided to address insomnia in late-life.[37,39,40] This is an unfortunate situation, given that psychological approaches may be preferable to pharmacological ones as a frontline approach to treating chronic, late-life insomnia, as we will detail below.

Sedative/hypnotic medications are the most common treatment prescribed for older adults presenting with insomnia complaints. Large cross-sectional studies and reviews have reported that older adults are more than twice as likely to receive a sedative/hypnotic medication as are younger adults.[33,41] Evidence from meta-analyses has demonstrated the utility of benzodiazepine receptor agonists (BZRA) for the treatment of acute insomnia,[42–45] however the data evaluating use of these medications beyond the acute phase of treatment in older adults is limited. Further, evidence from meta-analyses suggests that these medications are associated with an increased risk of side effects[43,44] and potential drug interactions[46] in older adults, as well as the likelihood of developing tolerance and/or dependence to these medications with chronic use.[47–53] These concerns are particularly salient to populations of older adults due to the disproportionately greater use of sedative and hypnotic medications among this group, often for long periods of time and at increased risk of adverse effects.[1,33,54–56] As late-life insomnia tends to be chronic in nature, the lack of evidence supporting the efficacy of BZRA medications in the elderly suggests that these medications may not be an optimal treatment choice for this population. Studies have indicated that between one-third and one-half of elderly adults with significant sleep problems report using sedative or hypnotic medications,[1,37] and elderly women are prescribed these medications more frequently than elderly men.

Epidemiological studies have demonstrated substantial morbidity, as well as economic and social costs associated with insomnia.[3–5] Research has shown insomnia in older adults is associated with increased healthcare costs and utilization,[57,58] physical and psychological difficulties,[59–61] risk of accidents and falls,[15] poorer quality of life, and reduced functioning.[6,15,60] Additionally, insomnia has been identified as a risk factor for the development or recurrence of psychiatric and medical conditions including depression[62–64] and heart disease,[65,66] and is a risk factor for nursing home placement in elderly individuals.[67] Furthermore, a study by Dew and colleagues reported that specific electroencephalographic (EEG) characteristics of older adults' sleep (ie, sleep onset latency (SOL) $>$ 30 min, SE $<$ 80%, REM % in highest or lowest 15% of distribution) are associated with an increased risk of death, after controlling for age, gender, and medical conditions.[68]

Models of late-life insomnia

Understanding the pathophysiological mechanisms of insomnia is essential for the meaningful advancement of its treatment, and the development of testable models of insomnia may aid in uncovering these mechanisms.[69] Most current models of insomnia focus predominantly on sources of hyperarousal, primary insomnia, and younger adult populations. While evidence for hyperarousal in the etiology of primary insomnia in younger adults exists,[70,71] research suggesting a critical role of hyperarousal in late-life insomnia is sparse and less convincing.[69,72] This may be explained by two observations: 1) late-life insomnia is commonly associated with comorbid mental or physical health conditions and 2) the pathophysiology of comorbid insomnia likely differs from primary insomnia in hyperarousal's etiological role.[69,73,74] Regrettably, few models of late-life insomnia have been developed[74] and no current models focus on the pathophysiological mechanism of late-life insomnia. Since little is known about the pathophysiology of insomnia in general, and less about comorbid insomnia, models of late-life insomnia will require considerable conceptual and scientific work.

In addition to arousal, mechanisms such as those related to the sleep systems[69] and cognitive factors,[75] may have direct roles in the etiology of late-life insomnia. Indeed, changes in arousal systems, sleep processes, and cognitive factors are common in later life. In this review, these three factors will be discussed in relation to the treatment of late-life insomnia. Ultimately, late-life insomnia likely has a multifactorial etiology and arousal models may be insufficient for understanding all the mechanisms underlying this problem.

Arousal systems

Current physiological, behavioral, and cognitive perspectives consider hyperarousal the principal mechanism underling insomnia.[76] Sources of arousal believed to lead to and maintain insomnia are well described in these models. However, pathophysiological mechanisms underlying this relationship remain unclear. Hyperarousal, nevertheless, is likely involved in the etiology of late-life insomnia. Potential mechanisms of arousal in the etiology of insomnia include increased or dysregulated expression of the ascending arousal systems in the brain, including projections from the orexin/hypocretin neurons in the laterodorsal hypothalamus, histaminergic tubermammillary nuclei of the hypothalamus, cholinergic pedunculopontine tegmentum nucleus, noradrenergic locus coeruleus, and seratonergic raphé nuclei.[69] Age-related changes in several of these systems have been identified. For example, with age, orexin-A concentrations appear to increase,[77] serotonergic innervations of the neocortex are reduced, and there is a loss of noradrenergic cell bodies in the locus coeruleus.[78] Research is needed to determine if age-related changes to the arousal systems of the brain originate from endogenous or exogenous factors related to aging and whether these changes contribute to late-life insomnia.

Sleep processes

Several sleep-promoting brain systems and mechanisms have been investigated including homeostatic, circadian, global sleep state mechanisms,[69] and humoral or local sleep mechanisms.[79,80] In this review, two of these mechanisms (ie, articulated in the two-process model of sleep) will be discussed in relationship to late-life insomnia, specifically homeostatic and circadian sleep processes.[81] Age-related changes in these processes may contribute to the onset and maintenance of late-life insomnia. Likewise, behavioral, environmental, and pharmacokinetic changes associated with aging may also contribute to sleep process changes in late-life.

First, age-related changes in circadian processes have been indentified in several studies.[82,83] These changes may, in part, contribute to late-life insomnia. For example, there is evidence, though limited, that diurnal melatonin levels are indeed disrupted in older adults with insomnia.[84] Exogenous (eg, bright light, exercise, and social activity) and endogenous (eg, melatonin excretion, body temperature) factors that contribute to the circadian sleep drive are commonly altered in late-life. Exogenous changes in the circadian sleep process may result from lifestyle and/or illness. For example, many older adults receive less frequent, more sporadic, and lower intensity bright light exposure than younger populations.[85] This may alter the circadian sleep system and other circadian processes involved in sleep such as body temperature. Lifestyle or illness can also lead to reduced physical activity in late-life which may disrupt circadian thermoregulation which is important to sleep onset and maintenance. Second, although not considered the main etiological factor in most theories of primary insomnia (see[86] for a notable exception), the sleep homeostatic process may play a more central role in late-life insomnia as indicated by the pronounced reduction in SWS quantity and amplitude among older adults.[74,87]

Sleep architecture changes, described above, are seen in older adults with and without sleep complaints. Therefore these changes are viewed as contributing factors to late-life insomnia and not stand-alone causes, per se. Sleep deterioration seen in late-life is likely the result of age-related changes in the interactions of circadian and homeostatic sleep processes[12,88] which may predispose older adults to developing insomnia.[74]

Cognitive factors

As a subjective complaint, cognitive factors have a direct etiological relationship to insomnia. Specifically, the perception of sleeplessness as a problem is a cognitive factor explicit in the definition of insomnia.[75] There may be cognitive changes in late-life that contribute to insomnia complaints. Indeed, attention,[89] information processing, sensory processing, and long-term memory have been posited as direct etiological mechanisms in insomnia,[76] and reduced working memory, processing speed, reaction time, and controlled attention are considered part of normal aging.[90,91] Late-life insomnia may relate to these specific changes in cognitive functions via a sleep-interpreting process.[75] For example, decreased controlled attention may disrupt the required deactivation of sensory processing and consciousness centers required for the perception of sleep onset. As with the other two factors discussed above, cognitive factors alone are insufficient for explaining the etiology of insomnia (eg, malingering or hypochondria should be considered in this case).

Mechanistic interactions of etiological factors of late-life insomnia

The three etiological factors of late-life insomnia discussed above may not only directly contribute to insomnia but may also interact with each other, thus they may have an indirect etiological role as well. Indeed, each factor alone is insufficient for explaining the etiology of late-life insomnia. Presently, it is believed that the sleep systems and arousal systems of the brain function relatively independently,[92] but they likely interact with cognitive factors such that the presence of either arousal and cognitive factors or sleep system dysregulation and cognitive factors may be necessary for the manifestation of late-life insomnia. Treatment options which enhance sleep propensity (via consolidation of the sleep systems), decrease arousal during the desired sleep period, and target relevant cognitive factors are likely to be effective at treating most cases of late-life insomnia.

Targets of psychological treatment of late-life insomnia

A common assumption is that health problems with physiological mechanisms, such as late-life insomnia, require medical and/or pharmacological intervention. However, there is growing recognition that this view overlooks the importance of the mind–body connection and the demonstrated ability of nonmedical interventions, such as cognitive–behavioral techniques, to therapeutically alter the physiological mechanisms underlying health problems. Specifically, in the case of late-life insomnia, the effectiveness of cognitive–behavioral techniques in treating insomnia may be, in part, due to their ability to enhance physiological homeostatic and circadian sleep propensity and reduce physiological arousal during the desired sleep period.

Each component of cognitive–behavioral therapy for insomnia (CBTi), described in detail below, may target one or all of the three factors posited to contribute to late-life insomnia. Concerning arousal, reconditioning the bed such that it no longer induces arousal is a major goal of this treatment. Relaxation techniques are believed to reduce cognitive and physiological arousal, while several sleep hygiene suggestions are aimed at reducing behaviors and eliminating substances (eg, tobacco) believed to increase arousal before bedtime. With respect to the sleep systems, the treatment techniques of sleep restriction and stimulus control seek to consolidate sleep into a desired sleep period.[86] Sleep hygiene recommendations to avoid caffeine (an adenosine antagonist which reduces this homeostatic substance), and include daily exercise (to enhance adenosine), may help enhance the efficacy of the homeostatic sleep drive and improve sleep in older adults with insomnia. Indeed, at least one study conducted in older adults with insomnia showed that sleep restriction significantly increased SWS and boosted slow-wave activity by about 30%.[93] Treatments aimed at regulating and maximizing the circadian sleep drive include regular in and out of bed times, bright light exposure, and exercise.

Relating to cognitive factors, treatment components include psychoeducation about the individual's unique sleep requirements, which may be significantly less than the ideal 8 hours he or she received as a young adult, and cognitive therapy for challenging thoughts about sleep, such as attributing all daytime impairment to sleep difficulties (ie, determining whether daytime impairments are due to sleep loss or have some other explanation). Overall, the development of new cognitive-behavioral techniques may be guided by the cumulative goal of enhancing homeostatic and circadian sleep propensity during the desired sleep period, mitigating arousal, and providing a cognitive state conducive to sleep.

Treatment of late-life insomnia

Pharmacological treatments

Sedative/hypnotic medications are frequently prescribed as a treatment for insomnia in older adults. As described above, there is considerable evidence[42–45] supporting use of BZRA medications for treatment of acute insomnia. While the majority of these investigations have included few or no older adults as part of the study population, a meta-analysis by Glass and colleagues[43] supported use of BZRA medications for acute insomnia treatment in older adults. A consideration discussed in the meta-analytic review by Dundar and colleagues is that funding by the pharmaceutical industry for many of the clinical trials examining the efficacy of BZRA medications may introduce bias into the findings.[42]

There has been less investigation done on these medications for long-term treatment, with almost no data on long-term use of BZRA medications in older adults. Double-blind, placebo-controlled, multicenter studies have supported long-term use (3–6 months) of eszopiclone (N = 791),[94] zolpidem (N = 199),[95] ramelteon (N = 451),[96] and zaleplon (N = 576; open-label trial).[97] Of these medications, however, only zaleplon was tested in an older adult population.[97] Zaleplon produced improved sleep in older adults relative to placebo during a 6–12-month open label trial.[97] As the other medications were examined in adult populations, but not specifically examined for use in older adults, it is dif-

ficult to determine whether these results may be generalized to elderly individuals given the unique circumstances of increased likelihood of comorbidities, changes in physiological sleep parameters, and altered drug metabolism in this group.

An RCT in older adults with primary insomnia[98] reported no difference between zopiclone and placebo for most outcomes, and found that cognitive behavioral therapy outperformed zopiclone on 3 of 4 outcome measures (sleep efficiency, total awake time, minutes of slow wave sleep) at both short-term (6 weeks) and long-term (6 months) evaluation. Additionally, a recent review[99] synthesizing results from meta-analyses and RCTs comparing BZRA medications and behavioral treatments for insomnia concluded that both treatments are efficacious in the short-term. However, they noted that behavioral treatments produced significantly more durable effects after treatment discontinuation compared to BZRA medications, and there was very limited evidence that BZRA medications retain long-term efficacy after treatment discontinuation.

These findings have important implications for treatment decision-making. The newer BRZA medications, such as zolpidem, zaleplon, and eszopiclone, were created to address the negative side effect profile of the older benzodiazepine medications. The BZRA medications, including both the older benzodiazepine medications and the newer "z-drugs" (eg, zolpidem, zaleplon, zopiclone, eszopilone), can result in a number of adverse effects, including impaired cognitive functioning, daytime sedation, confusion, lack of motor coordination, and increased risk of falls.[22,51,100] Some initial evidence suggested that these newer medications had fewer side effects than the older BZRA medications,[101–103] although additional meta-analyses have reported similar adverse event profiles for older benzodiazepine medications and newer z-drugs in older adults.[43] However, these adverse effects tend to be less severe and less frequent with the newer z-drugs, likely due to the shorter half-lives of these newer agents.[22] Elderly individuals appear to be particularly susceptible to adverse effects due to age-related changes in pharmacodynamics, pharmacokinetics, and drug interactions that affect how medications are metabolized.[103] These medications also change the sleep architecture in older adults.[104] Additionally, some of the more unusual, but rare, side effects of the newer BRZA medications, such as sleep-walking and sleep-eating,[105–108] could be particularly problematic for elderly individuals, due to risks of falls or comorbid medical conditions such as diabetes.

While not approved for this use, antidepressant medications are often prescribed off-label as treatment for insomnia in older adult populations. There is little evidence to support this practice, and a recent study by Reynolds and colleagues found that an antidepressant medication (paroxetine) was not effective for treating insomnia in an elderly sample.[109] Use of antidepressants for treatment of insomnia among older adults also carries the risk of serious adverse events, such as falls, confusion, cognitive difficulties, and exacerbation of occult sleep disorders.[110] The possibility for interactions with medications taken for other conditions further increases the risk of negative side effects.

In summary, there is a large body of evidence from RCTs and meta-analyses to support the use of BZRA medications as treatment options for acute insomnia.[42–45] Additionally, while there are data to support long-term use of eszopiclone, zaleplon, ramelteon, and zolpidem, only zaleplon has been examined within an older adult population. This limits the extent to which support for other medications can be applied to older adults. Due to the chronicity, increased likelihood of comorbid conditions, and the possibility of interactions with other medications, as well as the altered pharmacokinetics of these medications in older adults, these medications should be used with caution and monitored closely for adverse effects and potential drug interactions. Patients using these medications for an extended period of time should also be monitored for the potential development of dependence and tolerance. Evidence is lacking for the use of antidepressant medication to treat insomnia among elderly individuals, either as an acute intervention or on a more chronic basis.

Psychological treatment approaches

Psychologists trained in behavioral sleep medicine, a subspecialty of behavioral medicine and clinical/health psychology, use a variety of different techniques all of which are aimed at improving and/or alleviating the sleep complaints of older adults with insomnia. The most commonly employed techniques include: sleep education, sleep hygiene, relaxation training, cognitive therapy, stimulus control, and sleep restriction. For the most part, these various techniques have been more thoroughly researched than the previously described sedative/hypnotic medications. Each technique is individually described below, including empirical evidence for or against their use. This section is followed by a subsection which describes commonly applied combination packages of psychological interventions. This is typically referred to as cognitive–behavioral therapy for insomnia, or CBTi.

Sleep education

Sleep education is comprised of several basic facts regarding late-life changes in sleep (ie, increased awake time during the night) and sleep need (ie, potential reduction in total sleep requirements). To our knowledge, sleep education has not been evaluated as a stand-alone treatment modality for insomnia experienced by older adults and is used only as an adjunct to other psychological treatment techniques. Furthermore, we do not believe the basic edification of patients to be sufficient to engender change independent of other therapeutic techniques. The basic concepts taught during sleep education with the elderly are described in Table 1.

Table 1 Psychological techniques used in the treatment of late-life insomnia

Common sleep education components for older adults*

- Increased prevalence of sleep disturbance
- Increase in sleep onset latency (SOL)
- Increase in wake time after sleep onset (WASO)
- Increase in number of nighttime awakenings (NWAK)
- Increase in hypnotic use
- Increase in napping
- Decreased total sleep time (TST)
- Good sleep can be re-learned

Common sleep hygiene components

- Avoid caffeine after noon
- Avoid exercise within 2 hours of bedtime
- Avoid nicotine within 2 hours of bedtime
- Avoid alcohol within 2 hours of bedtime
- Avoid heavy meals within 2 hours of bedtime

Common relaxation practices**

- Progressive muscle relaxation
- Passive muscle relaxation
- Autogenic phrases
- Diaphragmatic/deep breathing
- Mental imagery
- Meditation
- Biofeedback

Stimulus control instructions***

- Go to bed only when tired
- Do not use the bed/bedroom for anything but sleep and sex
- If sleep is not obtained in 15–20 minutes, leave the bed/bedroom
- Only return to bed upon tiredness
- Repeat bullet #3 as necessary
- Wake at the same time every morning
- Avoid daytime napping

(*Continued*)

Table 1 (*Continued*)

Sleep restriction instructions

- Calculate average time in bed (TIB) and TST for the previous 1–2 Weeks
- If average sleep efficiency (SE) $>$ 90%, Increase TIB by 30 minutes****
- If average SE $<$ 85%, Decrease TIB by 30 minutes****
- Retire at same time every night. wake at the same time every morning
- Avoid daytime napping

Cognitive therapy guidelines/instructions

- Maladaptive thoughts, beliefs, and attitudes can precipitate and/or perpetuate insomnia
- Maladaptive thoughts, beliefs, and attitudes can cause negative emotional responses that may disrupt sleep
- Maladaptive thoughts, beliefs, and attitudes can be changed
- Cognitive restructuring or thought challenging is a technique to examine the evidence for/against thoughts, beliefs, and attitudes about sleep and aims to replace them with more adaptive/realistic thoughts, beliefs, and attitudes

Notes: *Components are commonly observed age-related changes in sleep. **All the above forms of relaxation aim to engender reductions in physiological and cognitive arousal. ***Sleep may initially worsen. This should be expected, but may result in a sleep debt that may facilitate later positive changes. ****If SE is between 85% and 90% do not adjust TIB. The above techniques can be effectively combined to created multicomponent psychological treatments for older adults with insomnia.
Abbreviations: SOL, sleep onset latency; WASO, wake after sleep onset; NWAK, number of nocturnal awakenings; TST, total sleep time.

Sleep hygiene

Many older adults adopt behaviors that are disruptive to achieving optimal sleep. Sleep hygiene is a composite of instructions that targets these sleep disruptive behaviors and aims at eliminating them from the patient's behavioral repertoire. The most common sleep hygiene recommendations are listed in Table 1. Currently, there is a limited number of empirical investigations of the utility of sleep hygiene as a stand-alone treatment for late-life insomnia. To this end, several researchers have employed sleep hygiene as part of a control/placebo condition,[109,111,112] supporting the broadly subscribed to idea that sleep hygiene, in isolation, is unlikely to generate clinically meaningful change in the sleep of older patients. Not surprisingly, a recent review conducted by McCurry and colleagues revealed that sleep hygiene alone does not meet the needed criteria to be considered an evidence-based treatment for late-life insomnia. Criteria used to determine if treatments were evidence-based included: 50% of the target problem post-treatment outcome measures must have demonstrated statistically significant between-group treatment effects; and between group effect sizes must have been at

least 0.20.[113] However, it is our experience that when non-sleep specialists (medical doctors and psychologists alike) employ non-pharmacological treatments for late-life insomnia, sleep hygiene is the most common treatment employed.

Relaxation training

Given the theorized connection between hyperarousal and sleep disturbances in late-life, psychological treatments aimed at reducing arousal in order to improve sleep are common. Relaxation training broadly includes a variety of techniques each aimed at minimizing patient physiological and/or cognitive arousal with the hope of generating positive changes in the individual's ability to commence and/or sustain sleep. As an aside, the elderly have a much greater likelihood of experiencing a concomitant pain disorder. The presence of a pain disorder may make progressive muscle relaxation (PMR) ineffective at inducing a sense of relaxation, because tensing and releasing muscles in the areas affected by pain may exacerbate the pain condition; thus, we recommend the use of a passive relaxation procedure for such patients, such as the technique outlined by Lichstein.[114]

The efficacy of relaxation training to engender desired changes, when used as a stand-alone psychological treatment, in older adults' sleep is uncertain. In fact, empirical investigations comparing relaxation training to other forms of psychological treatment for late-life insomnia have unanimously found minimal positive results that were typically inferior to other psychological treatment modalities to which relaxation was being compared.[114] This conclusion is underscored by McCurry and colleagues' verdict that relaxation training does not have sufficient empirical backing to be considered an evidence-based treatment for late-life insomnia.[113] Various relaxation strategies are listed in Table 1.

Cognitive therapy

Theories of cognitive psychology posit that cognitive distortions (ie, unrealistic beliefs, attitudes, and thoughts) contribute to, engender, and sustain unhealthy emotions and behaviors. Cognitive therapy aims to identify and change sleep-incompatible thoughts, attitudes, beliefs, and expectations in order to reduce negative sleep-associated emotions and promote sleep-compatible behaviors. This is classically achieved through the application of psychological techniques, such as cognitive restructuring and thought-challenging. There are, to the best of our knowledge, no published empirical investigations examining the effects of cognitive therapy, unaided, on the sleep complaints of elderly patients. In this vein, McCurry and associates' review of the extant literature did not reveal adequate support to propose cognitive therapy be considered an evidence-based treatment for late-life insomnia.[113] The general practices and principles of cognitive therapy for late-life insomnia are listed in Table 1.

Stimulus control

Older adults with insomnia commonly develop learned associations between the bed or bedroom and being awake. Stimulus control[115] is a behavioral technique that attempts to reduce patients' learned behavioral associations between the bed, bedroom, and wakefulness. These instructions are exclusively intended to amplify the patient's connection of the bedroom and bed to somnolence and sleeping. The specifics of stimulus control (Table 1) are intended to minimize patient bedroom/bed behavior to sleep and sex only, hence increasing the bedroom and bed as a cue for sleep. Most sleep psychologists view stimulus control as a very powerful tool to improve disturbed sleep. It has even been suggested that stimulus control is "one of the most effective single-component treatments" for late-life insomnia.[116] This argument is supported by numerous empirical studies that have found moderate to strong effects of stimulus control on the self-reported sleep of older adults.[117–119] For example, a trial comparing sleep hygiene plus stimulus control to a waitlist condition in older adults found improvements in sleep onset latency of over 30 minutes and improvements in wake time after sleep onset of approximately 13 minutes while the waitlist condition did not significantly change.[119] Yet, stimulus control has not yet met the necessary criteria to be considered an evidence-based treatment for late-life insomnia. This appears to primarily be the result of a lack of research investigating the effects of this psychological treatment option in isolation from other techniques often included under the rubric of CBTi.[113]

Sleep restriction

Sleep restriction is a behavioral technique aimed at reducing the quantity of superfluous awake time in bed that the patient experiences during the course of the night. (Note, sleep compression is a very similar psychological technique with the same goals as sleep restriction. However, sleep compression involves a more gradual reduction of time in bed.) This is accomplished by prescribing sleep time (or better described as amount of time to be spent in bed) that is close in quantity to actual time spent asleep by that patient (Table 1). The main goals of restriction practices are to provide the patient with one long, continuous block of sleep that is comparatively continuous and of decent quality. Like stimulus control, sleep restriction is believed to engender change through a relative reduction of the association between the bed/bedroom

and being awake. Sleep restriction may also exert change by building a sleep debt which may consequently assist in improving disrupted sleep. Sleep restriction is often regarded as an efficacious psychological treatment for insomnia in late-life. Improvements are traditionally seen in the subjectively-reported sleep onset latency (SOL) and wake time after sleep onset (WASO) of elderly patients.[120–123] For example, a study comparing the treatment of insomnia in older adults using sleep hygiene and sleep restriction reported improvements in the sleep restriction group of 25–30 minutes for both SOL and WASO, while the sleep hygiene group experienced much less change (ie, 2 minute reduction in SOL and 17 minute reduction in WASO).[122] Sleep restriction is the only psychological treatment option in isolation that fulfills the American Psychological Association's requirements to be considered an evidence-based treatment for late-life insomnia.[113]

Combination treatments

As was just shown, only one psychological technique currently meets the criteria to be considered a stand-alone efficacious treatment for late-life insomnia. However, using various combinations of the previously described techniques, psychologists have developed and employed multicomponent treatment approaches to the psychological treatment of late-life insomnia. As previously stated, these multicomponent treatments are commonly referred to as CBTi. To be considered CBTi, treatment packages must include at least 2 or more of the previously described treatment options. It is our experience that a very common multicomponent CBTi package typically includes: sleep education, sleep hygiene, relaxation training, stimulus control, sleep restriction, and possibly cognitive therapy. These multicomponent psychological treatment packages have been empirically revealed to engender significant improvements (both statistically and clinically) in the subjective report of disruptive sleep (improvements in SOL, WASO, and sleep quality rating) in older adults with insomnia.[111,118,124] Furthermore, multicomponent treatment packages fulfill all the necessary requirements to be considered an evidence-based treatment for late-life insomnia.[113]

Pharmacological versus psychological treatment approaches

The previous reviews of BRZA medications and psychological treatments for late-life insomnia point at dramatically different outcomes. Head-to-head comparisons of these two distinct treatment modalities (pharmacotherapy vs psychotherapy) have yet to be thoroughly investigated in older adults. In one notable exception, a direct comparision of multi-component CBTi, zopiclone, and a placebo control group was made. Results revealed that the CBTi condition produced objective and subjective improvements in sleep (ie, PSG and sleep diary total wake time improved approximately 50 minutes, sleep efficiency improved approximately 10%, and time spent in slow wave sleep improved by 17 minutes) while the placebo and zopiclone conditions did not significantly differ from each other.[98] In the only other comparison trial, it was found that multi-component CBTi, temazepam, and a combined CBTi and temazepam group were roughly equivalent in producing positive change in sleep patterns at 8 weeks. However, multi-component CBTi was rated as the most favorable treatment condition by patients and produced the most sustainable long-term changes, as measured at 24-month follow-up.[36] Thus, based on the admittedly limited direct empirical evidence, it appears appropriate to conclude that psychological treatment approaches to the treatment of insomnia in older adults are preferable to treatment with sedative/hypnotic medications.

Despite that fact that the efficacy of CBTi has been established through numerous studies, these interventions remain underutilized.[125] This is somewhat surprising when, in contrast to medications, cognitive-behavioral approaches to insomnia treatment have proven effective in the long-term treatment of insomnia among older adults,[35,36] without inviting such concerns as tolerance, adverse side effects, and dependence. Moreover, research suggests that older adult patients prefer these treatments to medications.[126] Psychological treatments of insomnia produce medium-large effect sizes (ranging from 0.65–0.94),[127,128] with 70%–80% of individuals showing improvements following treatment.[127] Of particular note, the improvements obtained from cognitive–behavioral approaches to insomnia treatment are generally well-maintained following discontinuation of treatment.[36,129,130] This finding is a notable divergence from the oft-reported finding of the loss of benefit in pharmacological treatments when the medication is discontinued. Additionally, there are few side effects to CBTi and no concerns about adverse drug effects. Notwithstanding these many benefits and the demonstrated superiority of cognitive–behavioral treatments for insomnia compared to pharmacological approaches, this type of treatment is underemployed.[125] This appears to be the result of both an insufficient number of clinicians adequately trained in the proper provision of psychological treatments, and medical providers' unfamiliarity with psychological treatment options for late-life insomnia.

Innovative psychological approaches with older adults

The psychology of sleep is an ever-evolving field with new treatment approaches consistently being introduced. Several

of these novel approaches are particulary promising when used with older adults due to increased applicability and their potential to target age-specific aspects of insomnia, as described in brief below.

Short-term and group treatment options

Multicomponent CBTi is often very time intensive and is typically delivered in 6–10 hour-long individually administered sessions spaced approximately 1-week apart. In an attempt to create more primary-care-friendly versions of multi-component CBTi, several recent empirical investigations have examined the efficacy of an altered multi-component CBTi that can be effectively delivered in a much reduced amount of time. In this vein, researchers have documented the successful administration of multicomponent CBTi for older adults in the following abbreviated formats: four 30-minute sessions;[118] one 45-minute session (with one 30-minute booster session);[124] two 25-minute sessions;[111] and two 50-minute sessions (with two 30-minute phone sessions).[112] Such trials have typically produced improvements in sleep that are equivalent in size to treatments of longer duration (ie, approximately 30-minute improvements in SOL and WASO). A recent review of short-term treatment approaches supports their utility for delivering effective multi-component CBTi to older individuals with insomnia.[131]

An alternative to the time intensive practice of administering multicomponent CBTi in individual sessions may be the use of a group therapy format. Multiple empirical investigations have successfully implemented multicomponent CBTi in small groups. However, this has been typically done in a mixed age range, not specific to older adults. In an exception, at least one empirical investigation has succesfully implemented multi-CBTi for elderly patients in groups of 4–6 individuals.[132] Similar to abbreviated treatment formats, a review of group treatment approaches suggests it, too, holds promise for delivering effective multicomponent CBTi.[131] However, it appears no known multicomponent CBTi group treatment study has attempted to capitalize on traditional group factors. McCrae and colleagues have suggested a need to investigate the additive benefit of potentially capitalizing on these group factors rather than simply applying individual multi-component CBTi to multiple individuals at once.[131]

Bright light therapy

The aim of bright light therapy (BLT) is to regulate the circadian concentrations of melatonin to improve sleep. This treatment has been studied as an intervention for late-life insomnia with mixed results. In general, these studies did not support its use as a stand-alone treatment.[133–135] Importantly, BLT is not a standardized treatment and there is a lack of consensus on optimum administration of bright light (ie, intensity, duration, and timing of exposure). As a result, BLT does not currently meet the scientific requirement to be considered an evidence-based treatment for late-life insomnia.[136] Nevertheless, preliminary research suggests that BLT may regulate circadian body temperature, reduce nighttime wakening, and daytime napping in older adults. It is possible that when combined with other interventions, such as exercise or multicomponent CBTi, this treatment may improve sleeplessness, particularly for older individuals with limited light exposure, such as institutionalized older adults. Additional research is needed to determine if BLT may enhance the effectiveness of CBTi in elderly populations. It should be noted that negative side effects have been reported with this treatment including: irritation, anxiety, agitation, headaches, nausea, dry skin, as well as, eye dryness, sensitivity, and vascular injury to eye tissue.[85]

Exercise

Exercise is often included as a sleep hygiene suggestion but recent research suggests that spending more time specifically working with patients to develop an exercise plan may be beneficial to their sleep. Although research on exercise in older adults with insomnia is sparse, the potential of this behavioral treatment to improve sleep in late-life is both promising and exciting. In three randomized control trials, exercise significantly improved PSG markers of sleep depth and continuity, self-reported sleep quality, sleep onset latency, sleep duration, sleep restoration, and sleep efficiency among older adults with moderate sleep complaints.[137–139]

Several potential mechanisms have been proposed to explain the relationship between exercise and sleep improvement.[140] Thermoregulation theorists posit that daily increases in core body temperature, through exercise, activates the production and release of melatonin, thus enhancing the circadian sleep drive and sleep promotion.[141,142] In contrast, proponents of the restoration theory argue that increased daytime catabolic activity (via exercise) leads to increased anabolic activity during sleep, resulting in improved sleep.[143,144] Another possibility, articulated in the biochemical regulation theories of sleep, is that exercise increases sleep promoting substances (ie, adenosine,[145] IL-1, IL-6, and TNF-α[146]) in the brain which are related to enhanced sleep homeostasis. Other proposed mechanisms include potential anxiolytic and antidepressant effects, health promoting effects, and

circadian phase shift effects of exercise that may indirectly improve sleep.[147] Regrettably, the number of studies to suggest that exercise is a stand-alone, evidence-based treatment for late-life is insufficient.[147] There is an urgent need for randomized control trials of treatments that include an exercise component for late-life insomnia.[140]

Psychological treatment of insomnia in special populations of older adults

The elderly are at an increased risk for many health-related disorders, making the diagnosis and treatment of primary or solitary insomia increasingly unlikely within this patient population. The sections below provide a brief review of the literature which has examined the psychological treatment of insomnia in older dementia patients and caregivers, comorbid insomnia in late-life, and insomnia in older adults who are dependent on sedatives/hypnotics.

Dementia patients and dementia caregivers

Older adults with dementia frequently have an accompanying sleep disturbance and pharmacotherapy appears to be the treatment of choice among physicians. However, the efficacy of sedatives/hypnotics in this patient population has been questioned.[148] Adapting commonly employed multicomponent CBTi practices for use with dementia patients may be more appropriate. One such adaptation has been to train caregivers of dementia patients to implement multicomponent CBTi with their care recipients.[149] Attention must also be paid to the sleep of the caregiver. Caregivers commonly complain of poor sleep and this has shown to be predictive of caregivers' decision to institutionalize their care recipients. Thus, multicomponent CBTi, including exercise, has been suggested as a frontline treatment option for caregivers.[150] Additional research is still needed in this arena to determine the efficacy of multi-component CBTi when applied to dementia patients and their caregivers.

Comorbid insomnia

Comorbid insomnia is very common in late-life and can occur in conjunction with medical (eg, arthritis, cancer, dementia, etc) and/or psychological (eg, anxiety, bereavement, depression/dysthymia, adjustment disorders, etc) conditions. Late-life insomnia comorbid with medical conditions has been found to be responsive to multicomponent CBTi.[151] A study comparing the responsiveness of older adults with either comorbid medical or psychological disturbances to multi-component CBTi found no distinctions between the groups; both responded equally well.[152] Furthermore, some researchers have not employed the typical medical and psychological exclusionary criteria used in the majority of psychological treatment studies and have still reported multi-component CBTi to be an effective treatment of late-life sleep disturbances.[118,124] Thus, in the context of normal age-related medical and psychological comorbidities, CBTi appears efficacious in treating insomnia.

Hypnotic-dependent insomnia

High reliance on pharmacotherapies coupled with the lack of long-term improvements associated with hypnotic medication have resulted in hypnotically dependent insomnia in late-life being a quite common experience. Hypnotic-dependent insomnia is a condition characterized by the experience of insomnia symptoms concomitant with sedative/hypnotic medication use. Importantly, sleep may actually worsen upon medication termination. This is referred to as rebound insomnia whereby individuals find themselves unable to stop taking their prescription medication, but they continue to report disrupted sleep. Multicomponent CBTi has been shown to be effective in improving the sleep of hypnotically dependent elders with insomnia without requiring cessation of their sedative/hypnotic medications.[153] Multicomponent CBTi has also been employed as an adjunct to traditional medication tapering procedures. When used in addition to medication tapering, CBTi + tapering produces much higher rates of hypnotic abstinence at 12-month follow-up[154] and reductions in insomnia symptoms than tapering alone.[155] In summary, it appears multicomponent CBTi should be an integral element of sedative/hypnotic withdrawal programs for older adults with insomnia.

Conclusion

A plethora of factors appear to be related to the occurrence of late-life insomnia. The myriad of quality of life and societal consequences of this disorder necessitate effective treatment. Late-life insomnia's chronic and comorbid nature makes psychological treatment techniques a preferable treatment approach; however, they remain vastly underutilized while sedative/hypnotic medications remain the most commonly prescribed treatment. This may be due to a lack of professionals trained in the psychological treatment of late-life insomnia or to a deficiency of knowledge regarding these treatment options by primary care physicians, to whom the majority of older adults with insomnia present. Whatever the cause, active steps to remedy the problem appear warranted.

A major research initiative to develop methods to increase referrals for psychological treatment of late-life insomnia and

explore factors associated with physician hesitation to refer patients for such treatment seems appropriate. Future research should also continue to investigate ways to further adapt psychological treatments of insomnia to reach a maximal number of patients (ie, short-term and group approaches). Additional empirical investigations into the effectiveness, as opposed to the efficacy, of psychological treatments for late-life insomnia are needed. This will likely require researchers to continue to develop adequate placebo options for CBTi trials. Thus far, psychological treatments for insomnia in late-life have typically been compared to a waitlist control condition. However, several studies have employed a quasi-desensitization placebo condition which employs hierarchical imaginary exposure to sleep-related material.[123,156] These studies largely resulted in similar patterns of outcomes to those found in waitlist-controlled trials. Demonstration of the real-world applicability and translation of laboratory-based science into the public health sector must be the ultimate goal of applied psychological inquiry. Thus, development of adequate placebo conditions that can demonstrate the effectiveness of CBTi over "common factors" is of critical importance.

Future empirical investigators would be wise to examine the experience level of the therapist involved in clinical trials, as the vast majority of trials employ graduate student level trainees as therapists. Lastly, special attention to the recruitment, screening, and retainment of research participants is necessary. There is great heterogeneity between research trials, and that makes recommendations and comparisons difficult. Additionally, many clinical trials are plagued by small sample sizes, potentially introducing issues related to the generalizability of findings.

Disclosures

The authors report no conflicts of interest in this work.

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