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Treatment of Late-life Insomnia

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Introduction

Insomnia, defined as difficulty initiating and/or maintaining sleep at least 3 nights/week that is accompanied by complaints of sleep-related daytime impairment^{1,2}, is the most common sleep disturbance in later life. Although insomnia can occur as an acute disorder (7 days or less), older adults are often afflicted with chronic insomnia (12 months or more³).

Additionally, insomnia in older individuals is most frequently co-morbid in nature, occurring in the context of age-related medical/psychiatric conditions, increased medication usage, and/or polypharmacy. This chapter will focus on the conceptualization, assessment and treatment of late-life insomnia from a behavioral sleep medicine perspective. Evidence for both behavioral and pharmacological treatment approaches will be presented. However, as will be shown, late-insomnia's chronic and co-morbid nature make behavioral techniques the preferable treatment approach.

Rates of Late-Life Insomnia

The prevalence and incidence rates of late-life insomnia depend largely on the criteria used in the specific study in question. Epidemiological surveys in the United States generally do not reference the chronicity of the sleep complaint, do not require daytime impairment in the criteria of insomnia, or fail to screen out insomnia participants with comorbid somatic complaints. Likewise, these studies tend to yield larger prevalence rates (i.e., 30%–60%)^{4,5} than rates derived from studies that include these more stringent criteria (i.e., 12–25%)^{6,7}. Regardless of the criteria, prevalence and incidence rates of insomnia significantly increase with age^{4,8}. The one year incidence rate of insomnia in the 65+ population has been reported to range between 3.1%–7.3%^{9,10}. Importantly, the increased prevalence of late-life insomnia may be at least partially attributable to the finding that remission of insomnia is less common in older individuals than it is in younger individuals¹¹. Research suggests that the increased rates of insomnia from mid-life to late-life are seen most prominently among older women, as elderly women present in a medical setting with insomnia complaints more frequently than men^{6,9,12,13}. While aging is associated with increased rates of insomnia, normal aging does not necessitate the onset of insomnia^{12,14}. Indeed, when mental and physical co-morbidities are controlled, the prevalence of late-life insomnia may be as low as 1%–7.5%^{4,15}. Insomnia leads to and is precipitated by several health complaints¹⁶, and

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because insomnia is often co-morbid with age related health problems, determining if aging has a direct or indirect role in the increased prevalence of insomnia has been difficult.

Treatment Seeking

Older adults are more likely to present in a primary care setting (i.e., general practice) with sleep complaints than are younger adults^{17, 18}. This may be due in large part to the increased co-morbidity, chronicity, and severity of late-life insomnia⁹. Indeed, the likelihood of a complaint of insomnia increases when the sleep disturbance is more chronic and severe¹⁰. In the primary care setting, however, sleep complaints are often poorly assessed, trivialized, and/or attributed to other mental and/or physical co-morbidities¹⁹. The recommendation that any co-morbid mental and physical health complaint should be the primary aim of treatment in late-life insomnia has been long maintained. However, two erroneous assumptions stemming from this perspective must be avoided: (1) that insomnia will generally subside once the co-morbidities have been treated and (2) that the behavioral treatment of insomnia cannot be successful in the presence of serious co-morbidities.

First, insomnia generally persists long after co-morbidities have subsided. Even when insomnia is preceded by another health condition, cognitive and behavioral factors often emerge to precipitate and perpetuate insomnia. Late-life insomnia presents so commonly with other health complaints that it may more appropriately be thought of as a co-morbid condition rather than a secondary complaint (i.e., secondary insomnia). In fact, at a recent National Institute of Health (NIH) State-of-the Science Conference, it was recommended that the term co-morbid insomnia be used in place of secondary insomnia²⁰. Second, cognitive-behavioral treatment for insomnia was effective for late-life-insomnia in a study sample of older adults with representative rates of mental and physical co-morbidities, suggesting that CBTi need not be postponed until after co-morbid conditions have been treated²¹. Additional research is needed to validate and/or modify CBTi for specific co-morbid conditions. Ultimately, late-insomnia (co-morbid or in isolation) should be viewed as a disorder that can be effectively treated¹⁴. However, many older adults with insomnia do not receive evidence-based assessment or treatment for their sleep complaints²² which may contribute to a dramatic difference in the way in which older adults receive treatment for insomnia in the primary care setting compared to younger adults. For example, older adults are more than twice as likely to be prescribed a sedative/hypnotic medication for insomnia, as are younger adults.²³ Over the course of a year, roughly 32% of older adults with insomnia²⁴ and 14% of the total population of adults aged 65–79 report using some type of hypnotic drug as a sleep aid⁵. Older women are more likely to be prescribed hypnotic medications for sleep complaints than are older men. Many sleep medications involve risk of tolerance and dependence, and for older adults sleep aids may pose additional risks including: polypharmacy, increased side effects, and exacerbation of sleep apnea²⁵. In addition, although most sleep medications are not implicated for use beyond 4–8 weeks, adults over the age of 65 make-up 50 percent of people using hypnotic medication for months and even years⁵. Conversely, CBTi has been implicated for the treatment of chronic late-life insomnia and has no known adverse side effects. Thus, it appears that CBTi is an optimal candidate for the treatment of insomnia complaints in older adults. However, while older adults are increasingly seeking care, the availability of professionally delivered CBTi is still relatively sparse.

Models of Insomnia

Several models of insomnia have been developed. The focus of this section will be to discuss the major models of insomnia in relationship to late-life insomnia. No current model of insomnia adequately captures the myriad factors and variables that may be involved in late-life insomnia, but each has utility in understanding late-life insomnia.

Physiological Sleep Models of Insomnia—Physiological sleep models of insomnia consider dysfunction in the sleep system(s) to be the primary source of sleep disturbance. Borbély and colleagues have developed a theory of sleep that includes two major sleep systems²⁶: 1) the circadian and 2) the homeostatic processes. Advanced age is associated with changes in these sleep systems. Amplitude and phase changes are seen in the circadian system^{27, 28}, and reduced amounts of slow-wave activity, sleep fragmentation, and early morning awakening are seen in the homeostatic system^{29, 30}. Dysregulation of these systems may explain in part the increase rates of insomnia in late-life.

Hyperarousal Models of Insomnia

Physiological Arousal Models: Physiological models posit that the arousal and the sleep systems function independently and that insomnia is due to dysfunction in the sympathetic arousal system³¹ and not an internal dysfunction of the sleep systems themselves. Age-related changes in the arousal system or increased rates of hyperarousal related health conditions in late-life such as chronic pain, may play a central role in the increased rates of insomnia in late-life.

Behavioral models: Behavioral models of insomnia highlight behavioral/environmental interactions which prevent and promote healthy sleep. Lifestyle, living situations, and behavioral changes common in late-life are thought to drive the increased prevalence of late-life insomnia. Behavioral factors that may contribute to increased rates of late-life insomnia include decreased physical activity, bereavement, napping, spending more time in bed, and delayed/reduced light exposure²⁴. Behavioral changes in late-life are observed in both healthy and poor sleeping older adults; therefore, these changes should not be thought of as the cause of late-life insomnia³². Nonetheless, behavioral changes in late-life may contribute to the overall increased rates of insomnia in this population in conjunction with other factors (i.e., age-related health factors).

Cognitive models: Cognitive models focus on mental processes which activate arousal and disrupt sleep. Reduced working memory, processing speed, reaction time, and controlled attention are considered part of normal aging. Additional research is needed to parse out the potential role normal age-related changes in cognitive functioning may play in insomnia in late-life.

Assessment of late-life insomnia

Late-life insomnia is a complex disorder to assess because, as previously mentioned, it is often co-morbid with other mental, physical, and sleep disorders. This section is intended to review the evidenced-based tools and techniques available for clinicians to assess and diagnose late-life insomnia from the scientist-practitioner approach, which utilizes the scientific method to inform clinical practice. A three step process is applied to assessment: (1) clinical data about the patient is obtained, (2) a “diagnosis hypothesis” is formulated and tested, and finally, (3) these steps are repeated as needed to rule-in and rule-out differential diagnoses.

Because insomnia is highly co-morbid with myriad physical health problems, older adults are more likely to present in a primary care setting with complaints of physical ailments rather than to a sleep center with insomnia complaints^{33, 34}. Primary care physicians should be aware that cardiopulmonary disease, painful muscle conditions, depression, and prostrate problems commonly predispose individuals to the development of insomnia³⁵, and patients are generally not aware of the impact that these health problems may have on their sleep³⁴. It is, therefore, the responsibility of the primary care physician to identify potential markers of sleep disturbance and consider any sleep complaint as an issue for further evaluation³⁶.

Even partial, or subclinical, symptoms of insomnia can have a profound impact on the patient's health and often precede the onset of chronic insomnia. Therefore, early detection and intervention of insomnia symptoms may prevent or mitigate these negative consequences¹⁹. Negative consequences of late-life insomnia include increased risk for falls, poorer health, fatigue, and decreased quality of life. Regrettably, late-life insomnia often goes unnoticed by primary care physicians caring for older adults¹⁸. Bailes et al. (2008) suggested that primary care physicians may be aided in identifying those patients that require additional sleep assessment by giving a brief questionnaire such as the Sleep Symptom Checklist (SSC;³⁷) or the Pittsburgh Sleep Quality Index (PSQI;³⁸). Patients who endorse insomnia symptoms and who are amenable to further assessment are likely to benefit from referral and further sleep assessment by a sleep specialist. When insomnia and medical problems are combined, patients commonly present with a host of mental health concerns. Therefore, insomnia patients may best be served if referred to an integrative health care team that includes a clinical/health psychologist trained to handle the psychological side of the patients care. Indeed, with the relatively recent recognition of psychology as a health care profession, psychologists are better able to become integrated into the general health care arena to provide more comprehensive care for patients³⁹. This is nowhere more apparent than for clinical and health psychologists with training in behavioral sleep medicine. Ideally, in a collaborative and comprehensive health care system, older adults who present to their primary care doctor with insomnia complaints should routinely be referred to a behavioral sleep specialist to receive assessment and treatment. Currently, there are not enough behavioral sleep specialists to meet the potential demand for their services. However, recognition of the need for such services has prompted greater interest in the field of behavioral sleep medicine, and as a result, growing numbers of psychologists and other healthcare providers are choosing to specialize in this area.

Step 1: Gathering Data—The major goal in assessing late-life insomnia is first to determine if the disrupted sleep of the patient is related to clinically significant daytime impairment, and second to determine if the sleep disturbance meets criteria for insomnia and is not attributable to another sleep disorder. Doing this requires the clinician to gather data from multiple sources, because daytime dysfunction in older adults may be related to other mental or physical complications yet be wrongly attributed to sleep disturbance by the patient. Moreover, sleep disorders other than insomnia (i.e., sleep apnea) are more prevalent among older adults and must be carefully differentiated from insomnia. Finally, age-related changes in sleep can imitate the symptoms of insomnia. Many older adults may present with insomnia complaints but do not require treatment. Below are several sources of data that are useful in assessing and diagnosing late-life insomnia.

Available Medical Records: As clinical and health psychologists become more integrated in the larger healthcare system, access to medical records will enhance the clinicians' ability to assess and treatment late-life insomnia. Particular attention should be paid to previous and current medical or psychiatric conditions that cosegregate with late-life insomnia such as chronic pain, depression, cardiovascular complications and cancer. In addition, current prescriptions should be reviewed to determine what impact the patient's current medication regimen may have on sleep.

Intake Packet: Sending an intake packet to the patient's home before the first visit is recommended to save time and to allow the clinician to gather more detailed information during the initial assessment visit. The introductory packet may include questions concerning patient demographics such as educational attainment, socioeconomic status, work history, social relationships, medical status/history, sleep/wake patterns, and current medication use. Ideally, this information can be compared to available medical records.

Clinical Interview: The clinical interview is an essential element in the assessment of late-life insomnia. During this interview, the clinician is able to ask more in-depth questions than can be obtained from questionnaires and can clarify information obtained from the information packet. In addition, the clinical interview provides an opportunity for the clinician to make behavioral observations about the cognitive, motor, and interpersonal functioning of the patient. Most importantly, the clinical interview can assess specific information about patients' life style, daily stressors, behavioral patterns, and other factors that may impact sleep. In addition, the patient's perception of sleep and daytime functioning can be obtained.

Informant Report: Informant report is a good source of information about the patient's history, particularly information relevant to sleep behaviors that the patient may not be aware of, such as symptoms of restless leg syndrome, snoring, and OSA.

Psychological and Sleep Questionnaires: Higher levels of depression on Beck's Depression Inventory (BDI) and also anxiety on the State-Trait Anxiety Inventory (STAI) differentiate older adults with insomnia from older adults without insomnia⁴⁰. Assessing mood during the initial interview through these questionnaires provides information regarding whether older adults are experiencing daytime distress related to their sleep problems. Beck's Depression Inventory (BDI-II) and the Geriatric Depression Scale (GDS) have support for use in older adults and may appropriately be used in assessing late-life insomnia.

In addition, several questionnaires aimed at assessing sleep and daytime functions have been useful in identifying people with late-life insomnia including the Pittsburg Sleep Quality Index (PSQI)⁴¹, the Epworth Sleepiness Scale (ESS)⁴², and the Dysfunctional Beliefs and Attitudes about Sleep (DBAS)⁴³. Because these measures rely heavily on the patient's memory over the past weeks and months, these tools may be ineffective in older adults with memory problems.

Step 2: Formulating and Testing the Diagnosis Hypothesis—From the information obtained from the various sources described above, the clinician is prepared to formulate a hypothesis about the patient's sleep problem. Report of prolonged time to fall asleep, time spent awake in the middle of the night, or early morning awakenings are all indicative of insomnia. Further, a perceived low quality of sleep and the presence of daytime dysfunction (e.g., fatigue, concentration problems, etc.) may also suggest insomnia. When formulating the diagnosis hypothesis, it is imperative to consider alternative diagnoses (i.e., another sleep disturbance). The Diagnostic and Statistical Manual of Mental Disorder (4th edition text revision) is a valuable assessment tool that outlines the criteria against which the diagnosis hypothesis is evaluated. The criteria set for the diagnosis of insomnia are difficulty initiating and/or maintaining sleep for at least 3 nights/week that is accompanied by complaints of sleep-related daytime impairment⁷. With the data gathered from step one, a preliminary diagnosis may be established. The first step is to test the null hypothesis, that is, that the sleep disturbance is not due to insomnia. To reject the null hypothesis, differential diagnoses (i.e., other sleep disorders) must first be ruled-out by collecting additional data from sleep diaries, actigraphy, or PSG. See below for a discussion of the utility of these tools in assessing insomnia. Differential diagnosis is complicated by the fact that insomnia can and often does co-occur with other sleep disorders. Thus, as discussed in step 3, even when another sleep disorder is diagnosed, it is important to consider whether the insomnia is an independent disorder or is simply a symptom.

Sleep Diary: Sleep diaries supplement and confirm the insomnia diagnosis. They are an inexpensive way to obtain data about the daily patterns of the patient's sleep. It is

recommended that two weeks of sleep diary data be obtained^{14, 44}. Not only are sleep diaries used to help diagnosis late-life insomnia⁴¹, they can also be used during treatment as a scientific gauge of progress throughout therapy¹⁷.

Actigraphy: Actigraphy is a relatively recent objective measure used to assess sleep/wake continuity. The clinical utility and accuracy of actigraphy compared to polysomnography (PSG) in older adults has been validated in several studies^{45, 46}. However, the accuracy of actigraphy is particularly low in patients with poor sleep quality and highly impaired sleep and therefore, should be interpreted with caution in these patients. Because actigraphy poorly detects wakefulness, it may be more effectively utilized in the clinical setting in conjunction with other assessment methods⁴⁷. For example, when combined with sleep diaries, actigraphy may be a relatively inexpensive way to assess sleep state misperception, sleep disordered breathing, or respiratory disorders. Obtaining at least three days of actigraphy data is recommended when assessing insomnia⁴⁸. Obtaining several days and weeks of actigraphy may be helpful in assessing the effectiveness of treatment for late-life insomnia¹⁷.

Polysomnography (PSG): Polysomnography (PSG) is not indicated for the routine assessment of insomnia. This is due to the subjective nature of an insomnia complaint and the high expense and invasiveness of PSG⁴⁹. It is, however, recommended to obtain a differential diagnosis for the other sleep disorders highly co-morbid with late-life insomnia - specifically, obstructive sleep apnea (OSA), sleep-related breathing disorder (SBD), periodic leg movements in sleep (PLMS), and persistent circadian disorders (ASDA, 1995a, 1995b). In the differential diagnosis of OSA, in particular, self-report questions about snoring or gasping for breath are insufficient in making a differential diagnosis, and PSG is required. Because late-life insomnia is highly co-morbid with these other sleep disorders, the usefulness of PSG in assessing insomnia may increase with age⁵⁰.

Step 3: 'Ruling In/Out' Insomnia—In late-life, insomnia becomes increasingly co-morbid not only with other sleep problems but also with mental and physical conditions for which sleep disturbance is a symptom and with medications that induce sleep disturbance. This makes it more difficult to rule-out insomnia when another disorder has been ruled-in. Even when other sleep disorders are diagnosed, insomnia may still be present. For example, 50% of patients diagnosed with sleep disordered breathing have problematic insomnia symptoms that may be ruled-in as insomnia⁵¹. Thus, regardless of co-morbidities, insomnia should be considered as a potentially independent disorder requiring treatment.

Treatment

The treatment of late-life insomnia can be broadly classified into two distinct categories: pharmacological and behavioral (including cognitive-behavioral). The following sections summarize the empirical evidence for the use of treatments that fall within these domains.

Treatment as Usual

Historically, older adults' complaints of poor sleep were treated with benzodiazepine receptor agonists, which have been associated with side effects, such as increased risk of confusion and falls⁵². Thus, contemporary non-benzodiazepine receptor agonists (e.g., zolpidem, zaleplon, and eszopiclone) were developed to minimize such side effects. Initially, these agents appeared to carry fewer/less severe unwanted side effects (including, but not limited to: headache, somnolence, dizziness, bad taste, and decreased balance)⁵²⁻⁵⁴. However, no differences have been found between the sleep characteristics and number/

severity of adverse side effects of older adults treated with benzodiazepine receptor agonists or non-benzodiazepine receptor agonists⁵³—thereby, limiting their utility.

The majority of prescription hypnotic medication clinical trials are funded directly by the manufacturing company, contain few older patients, and have relatively short treatment/follow-up periods. With the exception of eszopiclone, hypnotic medications are not recommended for long-term usage. Given that the oldest patient in these clinical trials was 69 years of age, a recent review of eszopiclone for the treatment of late-life insomnia concluded that there was insufficient evidence to warrant extended use in the elderly⁵⁵. Given the known age-related changes in pharmacodynamics, pharmacokinetics, and drug interactions⁵⁴ close monitoring of the elderly patient beginning pharmacotherapy for insomnia is a necessity.

Antidepressant medication (both sedating and non-sedating), while not indicated or FDA approved for the treatment of sleep disturbances, is commonly prescribed in an attempt to improve sleep. Reports of improved sleep following administration of such medication is sparse—particularly in aged patients. Further, the potential of serious adverse events (e.g., cognitive impairment, falls, confusion, and exacerbation of occult sleep disorders) suggests antidepressants are a less than optimal alternative to other hypnotic medications and CBTi. Illustrative of this are the results of a randomized, double-blinded controlled trial of paroxetine (an antidepressant SSRI) combined with sleep hygiene (a behavioral technique empirically shown to be relatively inert as a stand alone treatment option) versus placebo in combination with sleep hygiene⁵⁶. Reynolds and colleagues aptly concluded that paroxetine is not effective in the treatment of late-life insomnia⁵⁶.

In summation, hypnotic medication use is very common in older adults. However, the questionable effectiveness of such medications and the potential for adverse events (including the potential for serious interactions) suggests alternative treatment modalities are needed. Thus, based on the evidence presented below, CBTi should always be considered a forerunner in the selection of potential treatment options for older adults with insomnia.

Behavioral Sleep Medicine Treatment Approaches

Behavioral Sleep Medicine specialists employ a vast array of techniques all aimed at producing improvements in the sleep of older adults with insomnia. Commonly used techniques include: sleep education, sleep hygiene, relaxation training, stimulus control, sleep restriction/compression. Generally, these techniques are better researched than the aforementioned pharmacotherapies. These techniques are individually detailed below and subsequently followed by a section describing commonly applied combination packages of techniques (commonly referred to as CBTi).

Sleep Education—Sleep education is comprised of several basic facts related to age-related changes in sleep and sleep need. To our knowledge, sleep education has never been evaluated as a stand alone treatment modality for late-life insomnia and is used only in adjunct with other commonly used techniques. Further, we do not believe these basic knowledge principles would be sufficient to engender change independent of other therapeutic techniques. Basic principles of sleep education with older adults are described in Table 1.

Sleep Hygiene—Sleep Hygiene is a set of instructions that aims at eliminating sleep disruptive behavior from the patient's behavioral repertoire. Common sleep hygiene recommendations are listed in Table 2. Limited empirical investigations have investigated the utility of sleep hygiene as a stand-alone treatment for late-life insomnia. In fact, several researchers have used sleep hygiene instruction as part of a control or placebo

condition^{56–58}— clearly indicating the widely held belief that sleep hygiene alone is unlikely to produce meaningful change in the sleep of elderly patients. Additionally, a recent review conducted by McCurry and associates indicated that sleep hygiene alone does not meet the necessary criteria to be considered an Evidence-Based Treatment for late-life insomnia⁵⁹.

Relaxation Training—Relaxation includes a variety of strategies all aimed at reducing patient levels of physiological or cognitive arousal in order to produce positive changes in the individual’s ability to initiate and maintain sleep. [Note: Given older adults greater likelihood to have a concomitant pain disorder, we recommend the use of the passive relaxation procedure outlined by Lichstein⁶⁰.] However, the efficacy of relaxation to produce desired changes, in isolation, in older adults’ sleep is questionable. In fact, studies comparing relaxation to other forms of CBTi have universally found minimally positive results, typically inferior to the comparison treatments⁶⁰. This conclusion is highlighted by McCurry and colleagues’ finding that relaxation training does not meet the necessary criteria to be considered an Evidence-Based Treatment for late-life insomnia⁵⁹.

Cognitive Therapy—Cognitive therapy aims to confront and address sleep incompatible thoughts and expectations. This is typically done through the employment of techniques, such as cognitive restructuring and thought challenging. To our knowledge, there is no published research examining the effects of cognitive therapy alone on the sleep complaints of older adults. Accordingly, McCurry and colleagues’ review of the literature did not reveal sufficient evidence to suggest cognitive therapy be considered an Evidence-Based Treatment for late-life insomnia, according to American Psychological Association criteria⁵⁹. However, cognitive therapy is commonly employed in combination with other CBTi techniques to treat insomnia in late-life.

Stimulus Control—Stimulus control⁶¹ is a set of techniques that target the patient’s learned behavioral association between the bed, bedroom, and being awake. The instructions are specifically designed to increase the patient’s association of the bedroom and the bed to sleeping. Therefore, the specific instructions (as outlined in Table 4) are intended to limit patient bedroom and bed behavior to sleep and sex only. The effects of stimulus control are generally regarded as positive. In fact, it has been suggested that stimulus control is “one of the most effect single-component treatments” for late-life insomnia⁶². This contention is supported by several investigations that report moderate to strong effects of stimulus control on the subjective sleep (sleep onset latency; SOL and wake after sleep onset; WASO) of elders^{63–65}. However, stimulus control does not meet the necessary criteria to be considered an Evidence-Based Treatment for late-life insomnia, primarily due to a lack of research examining the effect of this treatment modality in isolation from other forms of CBTi⁵⁹.

Sleep Restriction/Compression—Sleep restriction and sleep compression are similar techniques used to reduce the amount of unwanted awake time the patient experiences during the course of the night by matching the prescribed sleep time to actual time spent asleep (see Table 5 for detailed instructions). Thus, one of the main goals of restriction and compression practices is to provide the patient with a long, continuous block of sleep that is relatively uninterrupted and of good quality. The mechanisms by which these techniques are believed to work is through a reduction of the association between the bed, bedroom, and being awake and through building a sleep debt which may subsequently aid in improving sleep. The main distinction between the two alternative strategies is that sleep restriction sharply reduces the amount of time the patient spends in bed and then gradually increases this time, if indicated, while sleep compression is conducted by slow and gradual reduction of time spent in bed. Both sleep restriction and compression are regarded as highly efficacious treatments for late-life insomnia. Typical improvements are seen in the self-

reported SOL and WASO of older patients. This point is illustrated by the generally positive findings from several studies employing one of these two techniques^{66–69}. In fact, sleep restriction and sleep compression fulfill the American Psychological Association's requirements to be considered an Evidence-Based Treatment for late-life insomnia⁵⁹.

Combination Treatments—Using a combination of the above described techniques, clinicians have developed multi-component treatment approaches to the treatment of late-life insomnia. As previously mentioned, these multi-component treatment approaches are typically referred to as CBTi. Typical treatment packages include use of 2 or more of the previously described techniques. One of the most common combinations is: Sleep Education, Relaxation Training, Stimulus Control, and Sleep Restriction (sometimes also including Cognitive Therapy). Such multi-component treatment packages have been empirically shown to provide improvements in the subjective experience of sleep (SOL, WASO, and SQR) in older adults with insomnia^{57, 64, 70}. Multi-component treatment packages fulfill the American Psychological Association's requirements to be considered an Evidence-Based Treatment for late-life insomnia⁵⁹.

Treatment as Usual versus Behavioral Sleep Medicine Treatment Approaches

The previous reviews of pharmacotherapy and psychotherapy for the treatment of late-life insomnia suggest drastically different outcomes. However, how these two treatment modalities fair when in direct comparison with one another is a scantily investigated area. Direct comparison of multi-component CBTi, zopiclone, and a placebo resulted in CBTi producing objective and subjective improvements in sleep, while the placebo and zopiclone conditions did not differ from each other⁷¹. Comparison of multi-component CBTi, temazepam, combined CBTi and temazepam, and placebo revealed that CBTi, temazepam, and combined CBTi and temazepam were roughly equally capable of producing positive change at 8-weeks. However, CBTi was rated as the most favorable treatment condition and produced the most sustainable longterm changes, as measured at 24-month follow-up⁷². Thus, it appears warranted to conclude that Behavioral Sleep Medicine approaches to the treatment of insomnia in late-life are preferable to hypnotic medications.

Innovative Behavioral Sleep Medicine Approaches with Older Adults

Behavioral Sleep Medicine is an ever evolving field. New treatment approaches are consistently introduced in the field. Several of these newer approaches are particularly promising when used with older adults and are briefly described below:

Short-term Treatment—Multi-component CBTi is typically delivered in 6–10, hour long sessions spaced approximately 1-week apart. However, in an attempt to produce more primary care friendly versions of CBTi, several recent investigations have suggested that CBTi can be effectively delivered in a much reduced timeframe. Specifically, investigators have documented the successful implementation of CBTi for older adults in as little as: four 30-minute sessions⁶⁴; one 45-minute session (with one 30-minute booster session)⁷⁰; two 25-minute sessions⁵⁷; and two 50-minute sessions (with two 30-minute phone sessions)⁵⁸. A recent review of short-term treatment approaches supports their promise for delivering effective CBTi⁷³ to older individuals.

Group Treatment—Multi-component CBTi is also typically delivered in individual, hour long sessions. However, an alternative to the time consuming practice of individual sessions may be the employment of a group therapy format. Several investigations have successfully implemented CBTi in small group settings—although typically in a mixed age range. However, at least one investigation has successfully implemented CBTi for older adults in groups of 4–6 patients⁷⁴. A review of group treatment approaches suggests it too holds

promises for delivering effective CBTi (McCrae, Dautovich, & Dzierzewski, in press). However, McCrae and colleagues have suggested a need to investigate the additive benefit of capitalizing on group factors rather than simply applying individual CBTi to multiple individuals at once ⁷³.

Exercise as a Treatment—The use of exercise as a potential treatment for late-life insomnia is intriguing, especially given exercise's positive influence on mood (for review see ⁷⁵), cognitive functioning (for review see ⁷⁶), and independence (for review see ⁷⁷) in late-life. To date, several clinical trials have attempted to treat older adults with a moderate sleep complaint through exercise ^{78, 79}. All of these trials have produced impressive reductions in SOL and gains in TST. Thus, it appears that exercise may be a useful treatment modality for late-life insomnia. Further research is needed to confirm this conclusion.

Treatment of Special Populations of Older Adults

Older adults are at an increased risk for several health-related disorders, making the diagnosis and treatment of primary/solitary insomnia increasingly unlikely with this patient population. Specifically, the section below provides a review of the literature which examines the treatment of insomnia in dementia patients and caregivers, comorbid insomnia in late-life, and hypnotic dependent insomnia in older adults.

Dementia Patients and Dementia Caregivers—Older patients with dementia often have an accompanying sleep disturbance. This sleep disturbance is typically treated with pharmacotherapy; however, the efficacy of such practices has been questioned ⁸⁰. It appears more appropriate to adapt commonly employed CBTi practices to be used with dementia patients. This adaptation may take the shape of training caregivers to implement CBTi with dementia patients ⁸¹. However, attention should also be paid to the sleep of the caregiver. Caregivers frequently complain of poor sleep, and CBTi (including exercise) has been suggested as frontline treatment option ⁸². Much additional research is still needed in this arena.

Comorbid Insomnia—Comorbid insomnia includes any case of insomnia that does not occur in solitary. As such, comorbid insomnia is very common and can occur in conjunct with medical (e.g., pain, arthritis, cancer, etc.) and/or psychological (e.g., depression, anxiety, bereavement, etc.) conditions. Insomnia of older adults experiencing concomitant medical conditions has been shown to be responsive to CBTi practices ⁸³. Furthermore, some researchers have not employed the typical medical and psychological exclusion criteria used in treatment studies and have still reported CBTi to be an effective treatment of late-life sleep disturbances ^{64, 70}. Thus, in the context of normal age-related medical co-morbidities CBTi appears efficacious in treating insomnia. Lastly, a study comparing the responsiveness of older adults with either comorbid medical or psychological disturbances to CBTi found no distinctions between the groups. Both responded equally well ⁸⁴. In general, it appears that insomnia comorbid with another condition (either medical or psychological in nature) will respond well to CBTi.

Hypnotic Dependent Insomnia—Given physicians' propensity to prescribe hypnotic medication and the lack of long-term improvements associated with such medication, hypnotically dependent insomnia in late-life may be quite common. Hypnotic dependent insomnia is a condition in which an individual continues to experience insomnia symptoms during the course of hypnotic medication use. Sleep may actually worsen upon halting medication use; thus, individuals find themselves unable to stop taking their prescription medication but continuing to sleep poorly. CBTi has been shown effective in improving the sleep of hypnotically dependent older adults with insomnia, without medication

termination⁸⁵. Additionally, CBTi has been used as an adjunct to traditional medical tapering procedures. When used in this manner, CBTi + tapering produces much higher rates of hypnotic abstinence at 12-month follow-up⁸⁶ and reductions in insomnia symptoms⁸⁷. Thus, it appears CBTi should be an integral component of hypnotic withdrawal programs for older adults.

Conclusion

A variety of factors (sleep architecture changes, medications, comorbidities, chronicity) contribute to the high prevalence of insomnia in later life. Comorbidities appear to play a particularly important role, because when they are controlled for, the prevalence of late-life insomnia drops considerably. Understanding the role of late-life insomnia's multifactorial nature, particularly the role of comorbidities, is important for accurate and effective assessment, diagnosis, and treatment. Older adults are likely to seek treatment for insomnia in primary care settings, and several proven tools are available to help improve the detection of insomnia in such settings. Both pharmacological and behavioral treatment approaches have demonstrated utility. However, late-insomnia's chronic and co-morbid nature make behavioral techniques the preferable treatment approach.

References

1. World Health Organization. The International Classification of Diseases. 1992; ICD-10 10th rev.
2. American Association of Sleep Medicine. Diagnostic and coding manual. 2. 2005. International classification of sleep disorders.
3. McCrae CS, Wilson NM, Lichstein KL, et al. 'Young old' and 'old old' poor sleepers with and without insomnia complaints. *J Psychosom Res.* 2003; 54(1):11–19. [PubMed: 12505551]
4. Foley DJ, Monjan AA, Brown SL, Simonsick EM, Wallace RB, Blazer DG. Sleep complaints among elderly persons: an epidemiologic study of three communities. *Sleep.* 1995; 18(6):425–432. [PubMed: 7481413]
5. Mellinger GD, Balter MB, Uhlenhuth EH. Insomnia and its treatment. Prevalence and correlates. *Arch Gen Psychiatry.* 1985; 42(3):225–232. [PubMed: 2858188]
6. Bliwise DL, King AC, Harris RB, Haskell WL. Prevalence of self-reported poor sleep in a healthy population aged 50–65. *Soc Sci Med.* 1992; 34(1):49–55. [PubMed: 1738856]
7. American Psychiatric Association. Diagnostic and statistical manual of mental disorders. 1994
8. Morin CM, Mimeault V, Gagne A. Nonpharmacological treatment of late-life insomnia. *J Psychosom Res.* 1999; 46(2):103–116. [PubMed: 10098820]
9. Morgan K, Clarke D. Risk factors for late-life insomnia in a representative general practice sample. *Br J Gen Pract.* 1997; 47(416):166–169. [PubMed: 9167321]
11. Dodge R, Cline MG, Quan SF. The natural history of insomnia and its relationship to respiratory symptoms. *Arch Intern Med.* 1995; 155(16):1797–1800. [PubMed: 7654114]
12. Vitiello MV, Larsen LH, Moe KE. Age-related sleep change: Gender and estrogen effects on the subjective-objective sleep quality relationships of healthy, noncomplaining older men and women. *J Psychosom Res.* 2004; 56(5):503–510. [PubMed: 15172206]
13. Ohayon MM. Epidemiology of insomnia: what we know and what we still need to learn. *Sleep Med Rev.* 2002; 6(2):97–111. [PubMed: 12531146]
14. Rajput V, Bromley SM. Chronic insomnia: a practical review. *Am Fam Physician.* 1999; 60(5):1431–1438. discussion 1441–1432. [PubMed: 10524487]
15. Vitiello MV, Moe KE, Prinz PN. Sleep complaints cosegregate with illness in older adults: clinical research informed by and informing epidemiological studies of sleep. *J Psychosom Res.* 2002; 53(1):555–559. [PubMed: 12127171]
16. Foley D, Ancoli-Israel S, Britz P, Walsh J. Sleep disturbances and chronic disease in older adults: results of the 2003 National Sleep Foundation Sleep in America Survey. *J Psychosom Res.* 2004; 56(5):497–502. [PubMed: 15172205]

17. Brooks JO 3rd, Friedman L, Bliwise DL, Yesavage JA. Use of the wrist actigraph to study insomnia in older adults. *Sleep*. 1993; 16(2):151–155. [PubMed: 8446835]
18. Hohagen F, Kappler C, Schramm E, et al. Prevalence of insomnia in elderly general practice attenders and the current treatment modalities. *Acta Psychiatr Scand*. 1994; 90(2):102–108. [PubMed: 7976454]
19. Katz DA, McHorney CA. Clinical correlates of insomnia in patients with chronic illness. *Arch Intern Med*. 1998; 158(10):1099–1107. [PubMed: 9605781]
20. National Institutes of Health. National Institutes of Health State of the Science Conference Statement. *Sleep*. 2005; 28(9):1049–1057. [PubMed: 16268373]
21. Lichstein KL, Wilson NM, Johnson CT. Psychological treatment of secondary insomnia. *Psychol Aging*. 2000; 15(2):232–240. [PubMed: 10879578]
22. Ozminkowski RJ, Wang S, Walsh JK. The direct and indirect costs of untreated insomnia in adults in the United States. *Sleep*. 2007; 30(3):263–273. [PubMed: 17425222]
23. Roth T, Roehrs TA. Issues in the use of benzodiazepine therapy. *J Clin Psychiatry*. 1992; 53 (Suppl):14–18. [PubMed: 1613014]
24. Morgan K, Clarke D. Longitudinal trends in late-life insomnia: implications for prescribing. *Age Ageing*. 1997; 26(3):179–184. [PubMed: 9223712]
25. Roth T, Zorick F, Wittig R, Roehrs T. Pharmacological and medical considerations in hypnotic use. *Sleep*. 1982; 5 (Suppl 1):S46–52. [PubMed: 6125025]
26. Borbely AA. A two process model of sleep regulation. *Hum Neurobiol*. 1982; 1(3):195–204. [PubMed: 7185792]
27. Weitzman ED, Moline ML, Czeisler CA, Zimmerman JC. Chronobiology of aging: temperature, sleep-wake rhythms and entrainment. *Neurobiol Aging*. 1982; 3(4):299–309. [PubMed: 7170047]
28. Czeisler CA, Dumont M, Duffy JF, et al. Association of sleep-wake habits in older people with changes in output of circadian pacemaker. *Lancet*. 1992; 340(8825):933–936. [PubMed: 1357348]
29. Ohayon MM, Carskadon MA, Guilleminault C, Vitiello MV. Meta-analysis of quantitative sleep parameters from childhood to old age in healthy individuals: developing normative sleep values across the human lifespan. *Sleep*. 2004; 27(7):1255–1273. [PubMed: 15586779]
30. Van Cauter E, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in healthy men. *Jama*. 2000; 284(7):861–868. [PubMed: 10938176]
31. Bonnet MH, Arand DL. Hyperarousal and insomnia. *Sleep Med Rev*. 1997; 1(2):97–108. [PubMed: 15310517]
32. Fichten CS, Creti L, Amsel R, Brender W, Weinstein N, Libman E. Poor sleepers who do not complain of insomnia: myths and realities about psychological and lifestyle characteristics of older good and poor sleepers. *J Behav Med*. 1995; 18(2):189–223. [PubMed: 7563046]
33. Doghramji PP. Detection of insomnia in primary care. *J Clin Psychiatry*. 2001; 62 (10 Suppl):18–26. [PubMed: 11388586]
34. Doghramji K. Assessment of excessive sleepiness and insomnia as they relate to circadian rhythm sleep disorders. *J Clin Psychiatry*. 2004; 65 (16 Suppl):17–22. [PubMed: 15575800]
35. Doghramji PP. Recognizing sleep disorders in a primary care setting. *J Clin Psychiatry*. 2004; 65 (16 Suppl):23–26. [PubMed: 15575801]
36. Ancoli-Israel S, Cooke JR. Prevalence and comorbidity of insomnia and effect on functioning in elderly populations. *J Am Geriatr Soc*. 2005; 53(7 Suppl):S264–271. [PubMed: 15982375]
37. Bailes S, Baltzan M, Rizzo D, Fichten CS, Amsel R, Libman E. A diagnostic symptom profile for sleep disorder in primary care patients. *J Psychosom Res*. 2008; 64(4):427–433. [PubMed: 18374743]
38. Smyth CA. Evaluating sleep quality in older adults: the Pittsburgh Sleep Quality Index can be used to detect sleep disturbances or deficits. *Am J Nurs*. 2008; 108(5):42–50. quiz 50–41. [PubMed: 18434798]
39. Brown RT, Freeman WS, Brown RA, et al. The Role of Psychology in Health Care Delivery. *Professional Psychology: Research and Practice*. 2002; 33(6):536–545.

40. Morin CM, Gramling SE. Sleep patterns and aging: comparison of older adults with and without insomnia complaints. *Psychol Aging*. 1989; 4(3):290–294. [PubMed: 2803622]
41. Petit L, Azad N, Byszewski A, Sarazan FF, Power B. Non-pharmacological management of primary and secondary insomnia among older people: review of assessment tools and treatments. *Age Ageing*. 2003; 32(1):19–25. [PubMed: 12540343]
42. Sanford SD, Lichstein KL, Durrence HH, Riedel BW, Taylor DJ, Bush AJ. The influence of age, gender, ethnicity, and insomnia on Epworth sleepiness scores: a normative US population. *Sleep Med*. 2006; 7(4):319–326. [PubMed: 16713340]
43. Morin CM, Stone J, Trinkle D, Mercer J, Remsberg S. Dysfunctional beliefs and attitudes about sleep among older adults with and without insomnia complaints. *Psychol Aging*. 1993; 8(3):463–46. [PubMed: 8216967]
44. Wohlge-muth WK, Edinger JD, Fins AI, Sullivan RJ Jr. How many nights are enough? The short-term stability of sleep parameters in elderly insomniacs and normal sleepers. *Psychophysiology*. 1999; 36(2):233–244. [PubMed: 10194970]
45. Friedman L, Benson K, Noda A, et al. An actigraphic comparison of sleep restriction and sleep hygiene treatments for insomnia in older adults. *J Geriatr Psychiatry Neurol*. 2000; 13(1):17–27. [PubMed: 10753003]
46. McCrae CS, Rowe MA, Dautovich ND, et al. Sleep hygiene practices in two community dwelling samples of older adults. *Sleep*. 2006; 29(12):1551–1560. [PubMed: 17252886]
47. Sivertsen B, Omvik S, Pallesen S, et al. Cognitive behavioral therapy vs zopiclone for treatment of chronic primary insomnia in older adults: a randomized controlled trial. *JAMA*. 2006; 295(24):2851–2858. [PubMed: 16804151]
48. Morgenthaler T, Alessi C, Friedman L, et al. Practice parameters for the use of actigraphy in the assessment of sleep and sleep disorders: an update for 2007. *Sleep*. 2007; 30(4):519–529. [PubMed: 17520797]
49. American Sleep Disorders Association. Practice parameters for the treatment of snoring and obstructive sleep apnea with oral appliances. *Sleep*. 1995; 18(6):511–513. [PubMed: 7481422]
50. Lichstein KL, Stone KC, Nau SD, McCrae CS, Payne KL. Insomnia in the Elderly. *Sleep Med Clin*. 2006; 1(2):221–230.
51. Krakow B, Melendrez D, Ferreira E, et al. Prevalence of insomnia symptoms in patients with sleep-disordered breathing. *Chest*. 2001; 120(6):1923–1929. [PubMed: 11742923]
52. Antai-Otong D. Risks and Benefits of Non-Benzodiazepine Receptor Agonists in the Treatment of Acute Primary Insomnia in Older Adults. *Perspect Psychiatr Care*. 2006; 42(3):196–200. [PubMed: 16916422]
53. Bain KT. Management of Chronic Insomnia in Elderly Persons. *Am J Geriatr Pharmacother*. 2006; 4(2):168–192. [PubMed: 16860264]
54. Dolder C, Nelson M, McKinsey J. Use of non-benzodiazepine hypnotics in the elderly: Are all agents the same? *CNS Drugs*. 2007; 21(5):389–405. [PubMed: 17447827]
55. McCrae CS, Ross A, Stripling A, Dautovich ND. Eszopiclone for Late-Life Insomnia. *Clin Interv Aging*. 2007; 2(3):313–326. [PubMed: 18044182]
56. Reynolds CFI, Buysse DJ, Miller MD, Pollock BG, Hall M, Mazumdar S. Paroxetine Treatment of Primary Insomnia in Older Adults. *Am J Geriatr Psychiatry*. 2006; 14(9):803–807. [PubMed: 16943177]
57. Edinger JD, Sampson WS. A primary care ‘friendly’ cognitive behavioral insomnia therapy. *Sleep*. 2003; 26(2):177–182. [PubMed: 12683477]
58. McCrae CS, McGovern R, Lukefahr R, Stripling AM. Research evaluating brief behavioral sleep treatments for rural elderly (RESTORE): A preliminary examination of effectiveness. *Am J Geriatr Psychiatry*. 2007; 15(11):979–982. [PubMed: 17974868]
59. McCurry SM, Logsdon RG, Teri L, Vitiello MV. Evidence-Based Psychological Treatments for Insomnia in Older Adults. *Psychol Aging*. 2007; 22(1):18–27. [PubMed: 17385979]
60. Lichstein, KL. Relaxation. In: Lichstein, KL.; Morin, CM.; Lichstein, KL.; Morin, CM., editors. *Treatment of late-life insomnia*. Thousand Oaks, CA US: Sage Publications, Inc; 2000. p. 185–206.
61. Bootzin RR. A stimulus control treatment for insomnia. *Proc Am Psychol Assoc*. 1972:395–396.

62. Bootzin, RR.; Epstein, DR. Stimulus control. In: Lichstein, KL.; Morin, CM.; Lichstein, KL.; Morin, CM., editors. *Treatment of late-life insomnia*. Thousand Oaks, CA US: Sage Publications, Inc; 2000. p. 167-184.
63. Davies R, Lacks P, Storandt M, Bertelson AD. Countercontrol treatment of sleep-maintenance insomnia in relation to age. *Psychol Aging*. 1986; 1(3):233–238. [PubMed: 3267403]
64. Pallesen S, Nordhus IH, Kvale G, et al. Behavioral treatment of insomnia in older adults: An open clinical trial comparing two interventions. *Behav Res Ther*. 2003; 41(1):31–48. [PubMed: 12488118]
65. Puder R, Lacks P, Bertelson AD, Storandt M. Short-term stimulus control treatment of insomnia in older adults. *Behav Ther*. 1983; 14(3):424–429.
66. Friedman L, Benson K, Noda A, et al. An Actigraphic Comparison of Sleep Restriction and Sleep Hygiene Treatments for Insomnia in Older Adults. *J Geriatr Psychiatry Neurol*. 2000; 13(1):17–27. [PubMed: 10753003]
67. Friedman L, Bliwise DL, Yesavage JA, Salom SR. A preliminary study comparing sleep restriction and relaxation treatments for insomnia in older adults. *J Gerontol*. 1991; 46(1):1–8.
68. Lichstein KL, Riedel BW, Wilson NM, Lester KW, Aguillard RN. Relaxation and sleep compression for late-life insomnia: A placebo-controlled trial. *J Consult Clin Psychol*. 2001; 69(2):227–239. [PubMed: 11393600]
69. Riedel BW, Lichstein KL, Dwyer WO. Sleep compression and sleep education for older insomniacs: Self-help versus therapist guidance. *Psychol Aging*. 1995; 10(1):54–63. [PubMed: 7779317]
70. Germain A, Moul DE, Franzen PL, et al. Effects of a brief behavioral treatment for late-life insomnia: Preliminary findings. *J Clin Sleep Med*. 2006; 2(4):403–406. [PubMed: 17557467]
71. Sivertsen B, Omvik S, Pallesen SI, et al. Cognitive Behavioral Therapy vs Zopiclone for Treatment of Chronic Primary Insomnia in Older Adults: A Randomized Controlled Trial. *JAMA*. 2006; 295(24):2851–2858. [PubMed: 16804151]
72. Morin CM, Colecchi C, Stone J, Sood R, Brink D. Behavioral and pharmacological therapies for late-life insomnia: A randomized controlled trial. *JAMA*. 1999; 281(11):991–999. [PubMed: 10086433]
73. McCrae, CS.; Dautovich, N.; Dzierzewski, JM. *Short-term and Group Treatment Approaches*. New York: Informa Healthcare; In press
74. Morin CM, Kowatch RA, Barry T, Walton E. Cognitive-behavior therapy for late-life insomnia. *Journal of Consulting and Clinical Psychology*. 1993; 61(1):137–146. [PubMed: 8450099]
75. Arent SM, Landers DM, Etnier JL. The effects of exercise on mood in older adults: A meta-analytic review. *J Aging Phys Act*. 2000; 8(4):407–430.
76. Colcombe S, Kramer AF. Fitness effects on the cognitive function of older adults: A meta-analytic study. *Psychol Sci*. 2003; 14(2):125–130. [PubMed: 12661673]
77. Breen L, Stewart CE, Onambélé GL. Functional benefits of combined resistance training with nutritional interventions in older adults: A review. *Geriatr Gerontol Int*. 2007; 7(4):326–340.
78. King AC, Oman RF, Brassington GS, Bliwise DL, Haskell WL. Moderate-intensity exercise and self-rated quality of sleep in older adults. A randomized controlled trial. *JAMA*. 1997; 277(1):32–37. [PubMed: 8980207]
79. King AC, Pruitt LA, Woo S, et al. Effects of moderate-intensity exercise on polysomnographic and subjective sleep quality in older adults with mild to moderate sleep complaints. *J Gerontol A Biol Sci Med Sci*. 2008; 63(9):997–1004. [PubMed: 18840807]
80. McCurry SM, Reynolds CF, Ancoli-Israel S, Teri L, Vitiello MV. Treatment of sleep disturbance in Alzheimer's disease. *Sleep Med Rev*. 2000; 4(6):603–628. [PubMed: 12531038]
81. McCurry SM, Gibbons LE, Logsdon RG, Vitiello MV, Teri L. Nighttime insomnia treatment and education for Alzheimer's disease: a randomized, controlled trial. *J Am Geriatr Soc*. 2005; 53(5):793–802. [PubMed: 15877554]
82. McCurry SM, Logsdon RG, Teri L, Vitiello MV. Sleep disturbances in caregivers of persons with dementia: Contributing factors and treatment implications. *Sleep Med Rev*. 2007; 11(2):143–153. [PubMed: 17287134]

83. Rybarczyk B, Lopez M, Schelble K, Stepanski E. Home-based video CBT for comorbid geriatric insomnia: a pilot study using secondary data analyses. *Behav Sleep Med*. 2005; 3(3):158–175. [PubMed: 15984917]
84. Lichstein KL, Wilson NM, Johnson CT. Psychological treatment of secondary insomnia. *Psychol Aging*. 2000; 15(2):232–240. [PubMed: 10879578]
85. Soeffing JP, Lichstein KL, Nau SD, et al. Psychological treatment of insomnia in hypnotic-dependant older adults. *Sleep Med*. 2008; 9(2):165–171. [PubMed: 17644419]
86. Baillargeon L, Landreville P, Verreault R, Beauchemin J-P, Grégoire J-P, Morin CM. Discontinuation of benzodiazepines among older insomniac adults treated with cognitive-behavioural therapy combined with gradual tapering: A randomized trial. *Can Med Assoc J*. 2003; 169(10):1015–1020. [PubMed: 14609970]
87. Morin CM, Bastien CI, Guay B, Radouco-Thomas M, Leblanc J, Vallières A. Randomized Clinical Trial of Supervised Tapering and Cognitive Behavior Therapy to Facilitate Benzodiazepine Discontinuation in Older Adults With Chronic Insomnia. *Am J Psychiatry*. 2004; 161(2):332–342. [PubMed: 14754783]

Table 1

Common Sleep Education Components for Older Adults.

•	Increased Prevalence of Sleep Disturbance
•	Increase in SOL
•	Increase in WASO
•	Increase in NWAK
•	Increase in Hypnotic Use
•	Increase in Napping
•	Decreased TST
•	Good Sleep can be Re-learned

Note: The above describe commonly observed age-related changes in sleep. SOL = sleep onset latency; WASO = wake after sleep onset; NWAK = number of nocturnal awakenings; TST = total sleep time.

Table 2

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

Table 3

Common Relaxation Practices.

•	Progressive Muscle Relaxation
•	Passive Muscle Relaxation
•	Autogenic Phrases
•	Diaphragmatic/Deep Breathing
•	Mental Imagery
•	Meditation
•	Biofeedback

Note: All the above forms of relaxation aim to engender reductions in physiological and cognitive arousal.

Table 4

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 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

Note: Sleep may initially worsen. This should be expected, but may result in a sleep debt that may facilitate later positive changes.

Table 5

Sleep Restriction and Compression Instructions.

Sleep Restriction	Sleep Compression
<ul style="list-style-type: none"> • Calculate average TIB and TST for the previous 1–2 Weeks • If average 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 	<ul style="list-style-type: none"> • Calculate average TIB and TWT for the previous 1–2 Weeks • Divide TWT by Number of Proposed Treatment Sessions • Reduce TIB Slowly by above Calculated Increment • Wake at the Same Time Every Morning • Avoid Daytime Napping

Note: TIB = Time in Bed, TST = Total Sleep Time, SE = Sleep Efficiency, TWT = Total Wake Time.

* If SE is between 85% and 90% do not adjust TIB.