MANAGEMENT OF INSOMNIA IN THE OLDER ADULT

by

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Abstract

Insomnia is the most common sleep disorder in the population and is one of the most common sleep complaints among primary care patients. Multiple factors increase the risk of developing insomnia disorder in older age. This condition generally does not resolve on its own and in older adults is associated with cognitive decline, increased risk of dementia, increased risk of falls, and higher rates of mortality. Pharmacotherapy is most frequently used to manage insomnia despite recommendations to try cognitive and behavioural strategies first. Given the prevalence of insomnia in older adults and its potential adverse consequences, primary care providers, including family nurse practitioners, need to be aware of non-pharmacological and pharmacological methods of insomnia management in this age group. For this paper a literature review was done to explore pharmacological and non-pharmacological management strategies for insomnia disorder in adults over the age of 65 in the primary care setting. Insomnia prevalence, considerations for diagnosis, and changes in sleep with ageing are also discussed. The literature review informed the creation of a poster to be presented at the UBC Nursing Graduate Student Research Symposium in order to disseminate the findings on recommendations of insomnia disorder management in older adults. With increased knowledge, family nurse practitioners should be better equipped to identify, diagnose, and manage this prevalent disorder in primary care.

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Management of Insomnia in the Older Adult

Introduction

It is estimated that 52% to 64% of patients in primary care have sleep complaints (Davidson, 2012). Of these complaints, insomnia is among the most commonly encountered (Morin, 2012). Among sleep-wake disorders, insomnia disorder is also one of the most common (Chun & Lee, 2016). The reported prevalence of insomnia in studies is highly variable, likely due to varying definitions of insomnia as well as differing assessment methods (Morin, LeBlanc, Belanger, Ivers, Merette, & Savard, 2011). Factors associated with insomnia include older age, being female, and poor self rated physical and mental health (Morin et al., 2011). While sleep complaints are extremely common in primary care, most clinicians are not well versed in sleep medicine, and particularly not in insomnia and its treatment (Rybarczyk, Lund, Garroway, & Mack, 2013).

In various studies about a third of the population reports insomnia symptoms like difficulty initiating or maintaining sleep, or early awakening (De Crescenzo et al., 2016). If daytime impairment, which is necessary for an insomnia diagnosis, is considered this falls to about 10% to 15%. If main insomnia diagnostic criteria are used, the prevalence of insomnia disorder is about 6% to 10%. In people over 75 prevalence of insomnia has been reported to be 20% (Rojas-Fernandez & Chen, 2014). One study of 5 886 community dwelling adults over 65 found over 70% prevalence of at least one insomnia symptom (Jaussent et al., 2011). Changes in sleep architecture with normal ageing, polypharmacy, and comorbidities increase the risk of developing sleep difficulties and insomnia disorder as people age (Gleason & McCall, 2015).

While family physicians and family nurse practitioners (NPs) are primary care providers, for the purposes of this paper NPs will be the target group. The aim of this paper is to provide family NPs with background information on the assessment and diagnosis of insomnia disorder in community dwelling adults over 65, and to focus on evidence for non-pharmacological and pharmacological management of insomnia in this population.

Insomnia generally does not resolve on its own and is associated with a number of adverse outcomes for the individual and for society (Morin, 2012). Those with insomnia have been shown to have impaired working memory and episodic memory, as well as impaired problem solving (Fortier-Brochu, Beaulieu-Bonneau, Ivers, & Morin, 2012). Insomnia can also lead to mood disturbances, with an increased risk of developing psychiatric disorders (Morin, 2012). In addition, insomnia leads to increased disability and higher healthcare utilization costs (Morin, 2012). In older individuals, insomnia is even more detrimental than in younger groups (Carrier, Lafortune, & Drapeau, 2012). In this age group, sleep disturbances and insomnia are associated with cognitive decline, increased risk of dementia, increased risk of falls, and higher rates of mortality (Almondes, Costa, Malloy-Diniz, & Diniz, 2016; Carrier et al., 2012).

Despite the high prevalence of insomnia and its known potential adverse consequences, insomnia remains under diagnosed and undertreated (Belanger, LeBlanc, & Morin, 2012; Morin, 2012). Moreover, when people do seek treatment this most often results in pharmacotherapy, despite an urge in the literature to first try non-pharmacological approaches (Morin et al., 2011). This is especially true in older adults, who are at increased risk of drug-drug interactions and adverse events from medications,

particularly sedatives and hypnotics (Carrier et al., 2012). Hypnotic use in those over the age of 65 is estimated to be between 3% and 21% for men, and between 7% and 29% for women, while in younger individuals rates range between 2% to 4% (Carrier at al., 2012). Canada's population aged 65 and over is 16.9%, now outnumbering children aged zero to 14 years, and is set to continue growing (Statistics Canada, 2016). In fact, it is estimated that by 2036 25% of Canadians will be 65 and older and by 2056 10% will be 80 and older (Sheets & Gallagher, 2012). Given this, insomnia will continue to become a more prevalent concern.

Primary care is the best place to manage insomnia (Davidson, 2012). Given the growing elderly population, the high prevalence of insomnia disorder, and the increased risks associated with pharmacotherapy in the older adult, it is important for the family nurse practitioner (NP) to have a good understanding of the assessment and management of insomnia disorder in adults over 65. Furthermore, there are higher rates of sleep complaints in primary care than there are actual rates of insomnia disorder, making it important for NPs to know diagnostic criteria for insomnia disorder to differentiate insomnia as a symptom of another sleep disorder like sleep apnea from insomnia as a disorder. In evaluating and educating older patients on insomnia, NPs also need to have an understanding of normal sleep cycle changes that occur with ageing to avoid evaluating and managing an older adult's sleep habits in the same way as those of a younger adult. Finally, NPs need to know about options for non-pharmacological as well as pharmacological therapies in older adults, and evidence of benefits and risks associated with each.

Project Goals and Objectives

The aim of this project is to provide the entry-level primary care NP with the current evidence based information regarding the non-pharmacological and pharmacological management options for insomnia disorder in adults over 65 based on a review of relevant literature. The non-pharmacological options will include cognitive behavioural therapy for insomnia (CBT-I), as well as its various components such as sleep hygiene, stimulus control therapy, sleep restriction therapy, relaxation therapy, and cognitive therapy. Other non-pharmacological options such as light therapy are less well studied and not recommended as a first choice in treatment, and will therefore not be included here. An understanding of the effectiveness of CBT-I is important for the entry-level primary care NP. However, the knowledge of how to engage in full CBT-I requires special training and is beyond the scope of the novice NP without such training. It will therefore not be included in this paper. Pharmacological therapy will include discussion of drugs available and commonly used for insomnia disorder in Canada. These are benzodiazepines, benzodiazepine receptor agonists, sedating antidepressants, antipsychotics, and over the counter preparations. Secondary goals of the project are to provide background knowledge on factors that contribute to insomnia development in older adults, the diagnostic criteria, and evaluation.

A poster presentation will be created for the UBC Nursing Graduate Student Research Symposium 2018. The aim is the creation of an educational poster to help the entry-level family NP to become more knowledgeable about insomnia diagnosis and the non-pharmacological and pharmacological treatments for insomnia in older adults in primary care.

It will not be possible to assess the impact of the Culminating Project as it will be presented in the form of a poster presentation. However, this project can potentially impact NP practice by increasing awareness of geriatric insomnia as a prevalent disorder, educating NPs that geriatric insomnia should not be considered a normal part of ageing, and by informing NPs about available non-pharmacological and pharmacological treatments.

Search Strategy

To inform this project literature was obtained from a search of PubMed, CINAHL, and MEDLINE. Search terms included older adult(s), senior(s), geriatric(s), elderly, insomnia, treatment, therapy, and management in various combinations. Only articles in English and with available online full text were retrieved. The search was limited to articles published within the past 10 years, unless earlier articles were deemed relevant and important to include. Abstracts were scanned to determine appropriateness. Reference lists of relevant papers were also reviewed, and articles from these were included if deemed relevant. RxTx was accessed through the University of British Columbia library and used to obtain relevant drug information. Choosing Wisely Canada was also reviewed for their recommendations.

Description of the Problem

Background

Diagnosis. The Diagnostic and Statistical Manual of Mental Disorders (DSM), currently in its fifth edition, and the International Classification of Sleep Disorders (ICSD), currently in its third edition, are the main classification systems used for sleep disorders (Ong, Arnedt, & Gehrman, 2017). Commonalities between these two systems

in criteria needed to diagnose insomnia disorder include difficulty initiating sleep, maintaining sleep, or early morning awakenings with resulting distress or impairment in functioning despite an adequate opportunity for sleep (Ong et al., 2017). This impairment needs to occur at least three times a week for at least three months, and the insomnia should not be better explained by another comorbid sleep disorder, mental disorder, or medical condition or the effects of a substance. The ICSD-3 refers to this diagnosis as chronic insomnia disorder, while the DSM-5 lists this as insomnia disorder. The DSM-5 also has various specifiers to indicate when comorbidities are present, or specifiers to indicate episodic (symptoms lasting one to three months), persistent (symptoms lasting at least three months), or recurrent (two or more episodes within a year) insomnia.

Insomnia has long been thought of as arising due to a comorbid disorder, but there is also evidence that insomnia itself is a precursor and risk factor for new psychiatric disorders (Morin & Jarrin, 2013). Previous classification systems differentiated between primary and secondary insomnia, but these systems have not persisted due to lack of empirical support and with evidence that often insomnia persists and needs treatment despite remission of the primary comorbid disorder (Ong et al., 2017). Regardless of the coexisting appropriately managed comorbid condition that a patient presents with, treatment for chronic insomnia itself is essentially the same (Sateia, 2014). The lack of differentiation in current diagnosis of insomnia between primary and secondary suggests that treatment of insomnia should be initiated along with treatment of the comorbid disease rather than waiting to see what effect treating the primary disease will have on the insomnia (Suzuki, Miyamoto, & Koichi, 2017).

Sleep cycle and sleep architecture. Insomnia is often under diagnosed in primary care. One contributing factor in older adults is that it is assumed that poor sleep is an inevitable consequence of ageing (Belanger et al., 2012). Epidemiological studies suggest that ageing itself is not responsible for higher prevalence of insomnia in older individuals (Jaussent et al., 2011). It is important to understand that while sleep architecture does change with age, these normal changes are not associated with the distress and daytime impairment that characterize insomnia disorder (Belanger et al., 2012). Insomnia should be appropriately addressed in older adults to decrease its associated negative consequences such as daytime fatigue and impairment, mood issues, memory and cognitive problems, increased risk of falls, and increased risk of depression and its negative impact on prognosis of medical conditions (Belanger et al., 2012).

The normal sleep cycle includes non rapid eye movement (NREM) sleep and rapid eye movement (REM) sleep (Gooneratne, & Vitiello, 2014). NREM sleep includes stage N1, stage N2, and stage N3. Stage N1 is the transition from wakefulness to sleep and is the lightest sleep stage. Stage N3 is particularly important because it is the period of deep sleep with slow wave electroencephalographic activity that correlates with growth hormone release. Other hormones relating to energy balance and metabolism also vary with the sleep-wake cycle (Gooneratne, & Vitiello, 2014). It is thought that NREM sleep improves physical functioning while REM sleep re-energizes the brain and emotional functioning, although the function of REM sleep is still being studied (Rybarczyk et al., 2013). Most people will cycle through these stages four or five times nightly.

As people age, total sleep time decreases from six and a half to eight and a half hours nightly in young adults to about five to seven hours nightly (Gooneratne, & Vitiello, 2014). The trend of decreased total sleep time does not continue past age 60. The time it takes to fall asleep generally does not change with healthy ageing. However, there are more night time awakenings, especially in the second half of sleep, and lighter sleep with less time spent in stage N3 slow wave sleep and more time spent in superficial stages N1 and N2 sleep (Crowley, 2011; Gooneratne & Vitiello, 2014). Circadian rhythm control changes occur and sleep onset occurs earlier, with subsequent earlier morning awakening (Carrier et al., 2012). There is more daytime sleep, as well as changes in specific waves during NREM sleep. Older adults also have less resilience to sleep schedule changes and have more challenges adjusting to issues impacting their biological clock, such as shift work or jet lag (Carrier et al., 2012). Sleep is lighter overall and auditory stimuli during sleep are more arousing (Rybarczyk et al., 2013). Retired individuals also have less strict schedules and so are more likely to deviate from regular sleep habits. Some literature has shown that older individuals are less likely to complain of sleep problems compared to younger adults possibly owing to older adults being more tolerant of sleep deprivation or because of adjustments to sleep status (Gooneratne & Vitiello, 2014). Understanding physiologic changes in sleep architecture and helping to educate older patients on these may help to adjust their expectations related to decreased need for sleep (Schroeck, Ford, Conway, Kurtzhalts, Gee, Vollmer, & Mergenhagen, 2016).

3P model of insomnia. Factors involved in chronic insomnia development include predisposing, precipitating, and perpetuating factors (Ebben & Narizhnaya,

2012). This is known as the 3P model of insomnia, proposed by Spielman, Caruso, and Glovinskin in 1987 (as cited in Ebben & Narizhnaya, 2012). It is a common model used to explain insomnia development and maintenance. Predisposing factors are things like genetic predisposition or tendencies to worry or ruminate. Hyperarousal, or a state of elevated emotional and physiological tension, is thought to be a major predisposing factor and a driver of insomnia (Ebben & Narizhnaya, 2012). Precipitating factors are acute events like an illness or a stressor such as a death of a loved one. How much control the individual perceives to have over the stressor and the perception of it contribute to the stressor's impact. Perpetuating factors cause ongoing sleep issues. These are usually behavioural things like napping throughout the day or staying in bed despite being awake. While the person may think that staying in bed longer will lead to the chance for more sleep, this actually leads to associating the bed with wakefulness, resulting in conditioned arousal (Ebben & Narizhnaya, 2012). Anxiety trying to fall asleep can result, leading to associating the bedroom with anxiety.

Insomnia evaluation. A single tool for evaluating insomnia does not exist (Chun & Lee, 2016). In evaluating for possible insomnia, practitioners should take a thorough history including a sleep history, medical history, psychiatric history, substance use history, medication list, any relevant physical exam, and investigate potential contributing factors. The bed partner should be questioned to determine issues the patient may be unaware of such as sleep apnea (Carrier et al., 2012). A sleep diary of one to two weeks can help evaluate sleep habits like sleep/wake schedule, total sleep time, and naps (Suzuki et al., 2017). While there is a lack of standardized sleep diaries in the literature, a consensus sleep diary has been created and three versions of this diary are provided in

Appendix A (Carney, Buysse, Ancoli-Israel, Edinger, Krystal, Lichstein, & Morin, 2012). The core sleep diary represents essential items, while the other two versions have optional items. It is important to be aware that a sleep diary can not include everything needed for assessment and should be augmented with other questions to make sure patients can adequately relay their sleep experience (Carney et al., 2012).

Insomnia often co-exists with other conditions. One study of 1506 community dwelling individuals aged 55 to 84 found that 83% had at least one of the 11 medical conditions the study assessed, and a quarter had four or more; these conditions were often comorbid with sleep complaints (Foley, Ancoli-Israel, Britz, & Walsh, 2004). For example, depression, heart disease, pain, and memory problems were associated with more prevalent insomnia symptoms. Another study found that 93% of elderly patients with insomnia had associated comorbid conditions (Foley, Monjan, Brown, Simonsick, Wallace, & Blazer, 1995 as cited in Chun & Lee, 2016). While in the past insomnia was frequently thought to be a symptom of other conditions like depression, it has been shown that it often persists after comorbid conditions are treated, and therefore requires its own evaluation and treatment (Mitchell, Gehrman, Perlis, & Umscheid, 2012).

Numerous factors should be explored in the interview (Ong et al., 2017). Discussion should take place around whether there is difficulty initiating sleep, maintaining sleep, or early morning awakening, as well as the frequency and duration of the issue, whether there were previous similar episodes, and precipitating factors. Daytime consequences like fatigue, sleepiness, mood changes, and cognition or performance issues should be sought. The Insomnia Severity Index is one possible tool to assess distress and insomnia severity (Medalie & Cifu, 2017). This tool is valid, reliable,

and sensitive to changes in insomnia treatment (Ritterband et al., 2017). Any treatments used now or in the past should be discussed. Behavioural practices like watching television before bed or physical activity are important. Cognitive beliefs should be explored, such as belief that everyone should sleep eight hours. Factors interfering with sleep, levels of stress, medications, and medical or psychiatric conditions are important considerations as the cause of insomnia is often multifactorial (Chun & Lee, 2016).

Contributing factors that can adversely impact sleep quality in older adults include behavioural and environmental things like noise, light, poor sleep hygiene, or physical inactivity, psychosocial factors like stress, bereavement, or isolation, psychiatric disorders like depression, anxiety, or schizophrenia, medical issues like cardiovascular disease, menopause, or chronic pain, nocturia, and drugs and medications (Carrier et al., 2012). For example, caffeine has effects on sleep that are similar to ageing in that it decreases slow wave sleep and increases wakefulness (Carrier et al., 2012). Stress has a greater negative impact on the sleep of older compared to younger people whereby stress hormones have been shown to decrease slow wave sleep and increase waking in middle aged men while having no effect in young men (Carrier et al., 2012). NPs should consider a referral for further evaluation if sleep disorders other than insomnia, like sleep apnea, seem likely.

Insomnia management and associated issues. Particularly in older adults insomnia disorder does not generally resolve on its own, making treatment important (Morin et al., 2009). Currently pharmacotherapy is the most common treatment method for insomnia despite recommendations that initially non-pharmacological approaches are preferred (Goodie & Hunter, 2014; Morin et al., 2011). Non-pharmacological treatments

include stimulus control therapy (SCT), sleep hygiene, relaxation therapy, sleep restriction therapy (SRT), and cognitive behavioural therapy for insomnia (CBT-I), all of which are appropriate for older adults (Chun & Lee, 2016). Difficulty in implementing non-pharmacological interventions and time constraints in primary care can limit their use (Taylor & Weiss, 2009). Furthermore, patients often expect to get a prescription. Despite time constraints, many sleep hygiene measures fall under SCT and so can be effectively performed in primary care. SRT aims to reduce awake time in bed and can be accomplished in primary care over multiple visits. CBT-I incorporates cognitive therapy, sleep hygiene, relaxation, SCT, and SRT. CBT-I is just as effective as pharmacotherapy in the short term and more effective in the long term, but few clinicians are informed about it (Rybarczyk, et al., 2013). For these reasons, a goal of this paper is to discuss non-pharmacological interventions for insomnia in older adults in addition to pharmacological ones.

In addition to lack of or difficulty in implementation of non-pharmacological methods, another problem is the potentially inappropriate use of pharmacotherapy. In Canada, variables associated with regular use of prescribed medications for insomnia include female sex, lower education, poor physical health, and older age (Morin et al., 2011). Older adults are more vulnerable to the effects of medications and have a higher incidence of adverse events (Schroeck et al., 2016). An increase in body fat, decrease in total body water, and fewer plasma proteins result in increased elimination half lives (Schroeck et al., 2016). Changes in phase I metabolism (oxidation, reduction, and hydrolysis) include a possible reduction in the cytochrome P450 system, which can impact clearance of drugs such as diphenhydramine or zolpidem. Drugs cleared through

phase II metabolism (conjugation reactions, for example glucuronidation) are not affected by older age. Thus, drugs that may be used in younger people can be inappropriate or have a greater risk of harm in older ones.

An issue contributing to potentially inappropriate medication use is the lack of studies evaluating safety and efficacy of drug therapy in older adults. Commonly used drugs for insomnia include antihistamines like diphenhydramine, antidepressants like trazodone or doxepin, melatonin, atypical antipsychotics like quetiapine, and sedative-hypnotics like benzodiazepines (Taylor & Weiss, 2009). Many drugs that are used for insomnia appear on the American Geriatrics Society Beers criteria as potentially inappropriate medications in older adults (Fick et al., 2015). Examples include benzodiazepines and non-benzodiazepine drugs like zopiclone or zolpidem. Choosing Wisely Canada also recommends against using benzodiazepines and non-benzodiazepine hypnotics as first line for older patients with insomnia (Canadian Geriatrics Society, 2017). Such issues pose challenges to practitioners in deciding whether and what to prescribe to their patients. Given these challenges, another goal of this paper is to address pharmacological agents used and considerations for their use in managing insomnia in older patients.

Literature Review

Non-pharmacological Management

Current literature advocates for psychological treatments to be used first line, as they are effective and safe interventions for insomnia disorder at any age (Chun & Lee, 2016). The clinical guidelines committee of the American College of Physicians (ACP) published a guideline for management of chronic insomnia disorder in adults (Medalie &

Cifu, 2017; Qaseem et al., 2016). While this guideline does not focus specifically on older adults, it makes a strong recommendation based on moderate quality evidence that all adults should receive CBT-I as the initial treatment for chronic insomnia. It goes on to make a weak recommendation using low quality evidence that for those for whom CBT-I is unsuccessful, the clinician should discuss risks, benefits, and costs of short term medication use. CBT-I combines behavioural interventions like SCT, SRT, sleep hygiene, and relaxation training with cognitive therapy (Medalie & Cifu, 2017). While the exact efficacy of each of the behavioural or cognitive components of CBT-I is not known, all of the components delivered together in the form of CBT-I are more effective than just trying them individually (Trauer, Quian, Dooyle, Rajaratnam, & Cunnington, 2015).

Sleep restriction therapy. Traditionally SRT has been the starting point of CBT-I, aiming to overpower developed conditioned arousal through temporary sleep deprivation (Rybarczyk et al., 2013). It aims to restore the homeostatic regulation of sleep. For SRT the patient should maintain a sleep log for two to three weeks prior to starting therapy to calculate their average total sleep time (Chun & Lee, 2016). The practitioner then prescribes nightly time in bed, which is calculated as the average of the total sleep time from the sleep log. Another approach is to prescribe total time in bed as the average of the total sleep time plus a normal amount of nocturnal wakefulness time, which is typically about 30 minutes. Time in bed should not exceed seven and a half hours in older adults (Chun & Lee, 2016). It should also be noted that wake up time is fixed, bedtime should be no later than two in the morning, and minimum time in bed is five hours. At weekly follow up sessions the total time in bed should be increased by

about 15 to 20 minutes as long as sleep efficiency is over 85%, based on ongoing sleep logs. In older patients if sleep efficiency is under 80%, 15 to 20 minutes should be subtracted (Ebben & Narizhnaya, 2012). Sleep efficiency is defined as time asleep divided by time in bed. Because wake up time is fixed, bed time should be advanced by 15-20 minutes. Modifications may need to be made for those over age 85 which may include not being as restrictive with time in bed (Rybarczyk et al., 2013). SRT can also be used during tapering of hypnotic medications to counteract rebound insomnia.

Stimulus control therapy. SCT emphasizes the need to associate being in bed with sleep (Chun & Lee, 2016). Patients should only go to bed when tired and should be instructed to leave the bed if they can not fall asleep within 15 minutes, returning only when sleepy (Rybarczyk et al., 2013). They should not watch television in bed. The bed should not be associated with anything other than sleep and intimate activity.

Sleep hygiene. Discussion of sleep hygiene includes the importance of maintaining a regular sleep pattern on weekdays and weekends, avoiding substances that can interfere with sleep like alcohol, caffeine, and nicotine, having a regular relaxing bedtime routine, importance of physical activity, and appropriate room temperature (Rybarczyk et al., 2013). Excessive use of electronics like computers before bed can suppress nocturnal melatonin production and have an adverse effect on the circadian rhythm (Chun & Lee, 2016). Aerobic exercise can promote deeper and more restful sleep, as well as have benefits for mood and anxiety reduction. Exercise should occur earlier in the day, rather than before bed to promote deeper sleep (Suzuki et al., 2017). Napping is also not recommended for those with insomnia as it can decrease the natural tendency for sleep at night (Carrier et al., 2012; Chun & Lee, 2016). In older people without insomnia

however, napping is not associated with negative outcomes and can even increase total sleep time as well as alertness in the evenings (Carrier et al., 2012). It is important to know that when sleep hygiene is the only intervention, it is not particularly effective (Schroeck et al., 2016). This is likely because poor sleep hygiene can contribute to insomnia but not cause it, so targeting sleep hygiene alone is not a sufficient treatment (De Crescenzo et al., 2016).

Relaxation therapy. Relaxation therapy includes any relaxation techniques that the person finds reduce cognitive arousal and decrease muscle tension (Trauer et al., 2015). Examples include meditation, mindfulness, guided imagery, or breathing techniques. Some authors write that relaxation therapy is not an essential component of CBT-I because it may not actually improve outcomes (Rybarczyk et al., 2013).

Cognitive behavioural therapy for insomnia. CBT-I involves the behavioural interventions discussed above plus cognitive therapy (Medalie & Cifu, 2017). Cognitive therapy tries to identify, challenge, and replace dysfunctional beliefs about sleep and insomnia (Trauer et al., 2015). Examples of such beliefs in older adults include thinking sickness will result or medical conditions will worsen without eight hours of sleep, that they will not be able to function the next day, that they should be able to sleep the same as when they were younger, or that they can not fix their sleep due to age (Rybarczyk et al., 2013). Research shows that CBT-I is just as effective as pharmacotherapy in the short term and is more effective in the long term (Rybarczyk et al., 2013). Moreover, it does not have the risks or side effects associated with medication use. The ACP Clinical Guidelines Committee report low to moderate quality evidence that in older adults CBT-I improves Insomnia Severity Index scores by 3.6 points, sleep onset latency by 8.2

minutes, wake after sleep onset by 37.6 minutes, and sleep efficiency (Qaseem et al., 2016). Limitations of CBT-I are that improvements are not immediate and may be seen only after two to four weeks, and that when first starting SRT patients will be more tired due to sleep deprivation. This could cause some patients to stop treatment. Implementation and utilization of CBT-I by the primary care NP requires the NP to have additional training and experience and is therefore beyond the scope of this paper. However, knowledge of CBT-I efficacy in the treatment of chronic insomnia is essential for the novice NP.

A systematic review and meta-analysis of CBT-I in adults with mean age of 55 found various improvements in sleep time variables that were associated with significant improvement of subjective symptoms (Trauer et al., 2015). Analysis included studies incorporating at least four of the five components of CBT-I, with SRT always included as this is deemed to potentially be one of the most effective strategies. Further, only studies including at least four one on one in person sessions were included. Among the included studies, patients with insomnia had sleep onset latency (average time to fall asleep) of 57.6 minutes, wakefulness after sleep onset (average awake time during the night after initial sleep onset) of 76 minutes, total sleep time of 344.1 minutes, and sleep efficiency (total sleep time divided by average time in bed, multiplied by 100) of 71.8%. Studies suggest that people without insomnia have sleep efficiency of 80% to 90% (Trauer et al., 2015). Compared to insomnia subjects, control participants without insomnia have been shown to vary in sleep onset latency by 23 minutes, wakefulness after sleep onset by 36 minutes, total sleep time by 95 minutes, and sleep efficiency by 16% (Trauer et al., 2015). With CBT-I, sleep onset latency was improved by 19 minutes, wakefulness after

sleep onset by 26 minutes, total sleep time by 7.6 minutes, and sleep efficiency by 9.9%. The authors report that these effect sizes are similar to ones of benzodiazepines and Z-drugs from meta-analyses of these hypnotics, with the difference that effects of CBT-I persist after stopping treatment.

It may pose challenging to practitioners to recommend CBT-I if patients have not heard of it and if they are expecting to receive a medication for their insomnia. Being aware of evidence comparing CBT-I with medications can help NPs in this conversation. A six week randomized controlled trial of adults aged 55 and over (mean age of 60.8) with insomnia evaluated CBT-I versus 7.5mg zopiclone nightly versus placebo with the objective of comparing short and long term efficacy of these interventions for insomnia (Sivertsen et al., 2006). The CBT-I involved six weekly 50 minute sessions on sleep hygiene, SRT, SCT, relaxation, and cognitive therapy. Polysomnography and sleep diaries were used to determine total wake time, total sleep time, sleep efficiency, and slow wave sleep. For the most part zopiclone did not differ from placebo at six weeks. At six weeks total wake time decreased by 52% in the CBT group, while in the zopiclone group it decreased by 4%, and in the placebo group by 16%. There was no significant difference in total sleep time between groups. Sleep efficiency was improved from 81.4% to 90.1% at six months in the CBT group, while in the zopiclone group sleep efficiency dropped from 82.3% to 81.9%. The CBT group also spent significantly more time in slow wave sleep, while the zopiclone group spent significantly less time in slow wave sleep after treatment. Adverse effects were reported in the zopiclone group but none were reported in the CBT group. Treatment effects were stronger in the CBT group at six months than at six weeks.

Another randomized controlled trial looked at CBT alone versus CBT plus 10mg of zolpidem nightly in 160 adults with a mean age of 50.2 (range 30 to 72 years) (Morin et al., 2009). Both groups included weekly sessions of CBT for six weeks, followed by six months of extended therapy. After six weeks there were significant decreases in insomnia in both groups, with 60%-61% of people responding to treatment. Both groups had significant improvements in sleep latency, time awake after sleep onset, and sleep efficiency. At the end of the six weeks people treated with combined CBT plus zolpidem had higher insomnia remission rates compared to just CBT. During the following six months of extended therapy, patients treated with CBT alone were split into two groups which had either monthly CBT sessions or no additional treatment, while the CBT plus zolpidem group was split to receive either a monthly CBT session or CBT plus as needed zolpidem. At the six month follow up people in the CBT alone group had higher remission rates (68%) versus the CBT plus as needed zolpidem group (42%). In this study zolpidem was useful during acute insomnia treatment, but better remission was obtained when medication was stopped after this and treatment continued with CBT only. This study did not look exclusively at older adults, which is a limitation for generalizability to this population.

A systematic review looking at CBT-I versus drug therapy found moderate quality evidence suggesting that CBT-I is superior to benzodiazepines and low quality evidence that it is superior to benzodiazepine receptor agonists for sleep in the long term (6 to 24 months after treatment completion) (Mitchell et al., 2012). The medications in the reviewed trials were zopiclone, zolpidem, temazepam, and triazolam. The authors concluded that there was moderate grade evidence suggesting CBT-I is superior to

zolpidem and zopiclone in the short term and very low grade evidence that temazepam may be more effective in the short term for improving sleep. CBT-I improved sleep latency by about 30-45 minutes, improved total sleep time by 30-60 minutes, and sleep efficiency by 8% to 16%. In the long term medications do not resolve the condition and are associated with adverse effects, dependence, and tolerance. Patients should also be aware that effects of drug therapy decline with time (Mitchell et al., 2012). Moreover, there is a lack of data on safety and efficacy of medications for long term use.

The efficacy of CBT-I is comparable between younger and older adults with studies including older adults showing moderate to strong effect sizes and sustained results in follow up (Rybarczyk et al., 2013). Karlin, Trockel, Spira, Taylor, and Manber (2015) conducted a study evaluating CBT-I for younger versus older veterans. The veterans in the older group were all over 65, with mean age of 69.2 years. The treatment consisted of six CBT-I sessions. 64% of the younger patients and 77% of the older patients finished either all six sessions or finished early due to symptom relief. Of those who finished early, most had symptom relief after five sessions, some after three to four sessions, and one after two sessions. The Insomnia Severity Index was used as the measure of insomnia severity. The younger group had 47% improvement of symptoms on the Insomnia Severity Index, while the older group was comparable at 48% improvement on average. The study also found significant improvements in physical, psychological, and social indices of quality of life in both groups.

Unfortunately there is a lack of clinicians trained in CBT-I and therefore a lack of referral options for patients with insomnia disorder (Rybarczyk et al., 2013). To circumvent these drawbacks and appointment time constraints in primary care, NPs can

refer patients to online resources. Most of these come at a cost. However, this cost is likely less than what a patient would pay a private therapist, if they could find one practicing CBT-I. Examples include www.cbtforinsomnia.com and www.myshuti.com (Fleming, 2017).

A recent randomized controlled trial evaluating adults with chronic insomnia found similar results in sleep outcomes in individuals using the Sleep Healthy Using the Internet program (SHUTi) at www.myshuti.com as compared to rates reported in other trials of face to face CBT-I, with sustained results (Ritterband et al., 2017). Participants either did the six week SHUTi automated program or received some online information about insomnia. Treatment effects in the SHUTi group were maintained at one year, with 56.6% achieving insomnia remission, and 69.7% achieving insomnia improvement. As the study did not include participants over age 65, more research needs to be done to determine whether such online programs would benefit older individuals. Particularly in older adults, individualized considerations such as computer literacy should be taken into account when referring to an online program.

Pharmacological Management

Despite recommendations for CBT-I to be used first for insomnia disorder, medications are most frequently used for management. It is important for clinicians and patients to understand that medications do not address the underlying mechanism perpetuating insomnia, and that patients will usually re-experience their insomnia symptoms once medications are withdrawn (Ebben & Narizhnaya, 2012). However, when medications are used as an adjunct to cognitive and behavioural treatments or when CBT-I is not successful, NPs need to know the associated benefits and risks. Goals of

drug therapy include to improve sleep quality and quantity and to improve daytime functioning (Schroeck et al., 2016). Principles to keep in mind when prescribing include using the lowest effective dose, using intermittent dosing such as two to four times a week, not using drugs for more than three to four weeks, choosing drugs with shorter half lives and less potential for daytime sedation, and using drugs that are less likely to cause rebound insomnia on discontinuation (Krishnan & Hawranik, 2008). This section will provide a general overview of drugs used for insomnia.

Sedative-hypnotics. Sedative-hypnotics like benzodiazepines and benzodiazepine receptor agonists, also called Z-drugs (zopiclone and zolpidem), are the most common treatment for insomnia (Trauer et al., 2015). In older adults, these drugs have an almost five times increased risk of adverse cognitive events, a 2.6 times increased risk of adverse psychomotor events, a five times increased risk of hospital admission after a motor vehicle accident, and increased risk of falls and hip fractures (McMillan, Aitken, & Holroyd-Leduc, 2013). The number needed to treat to improve sleep quality is 13, while the number needed to harm is six. Chronic use of hypnotics has also been associated with increased risk of depression and suicide, as well as mortality (Rybarczyk et al., 2013).

Choosing Wisely Canada recommends against using these drugs as first choice therapy (Canadian Geriatrics Society, 2017). Based on high quality evidence, the American Geriatrics Society also strongly recommends against their use as part of their Beers criteria (Fick et al., 2015). Despite recommendations against the use of these agents, 16% to 33% of community dwelling adults use benzodiazepines, with 54% of them using these drugs daily (McMillan et al., 2013). 64% of them report using these drugs for insomnia. Studies have generally not evaluated long term use of

benzodiazepines for insomnia (Schroeck et al., 2016). Benzodiazepine use of just several days, depending on the drug, can result in physical dependence, and longer term use may require discontinuation tapering to avoid withdrawal (Schroeck et al., 2016). Benzodiazepines have also been reported to reduce stage N3 sleep, or deep sleep (Shrivastava, Jung, Saadat, Sirohi, & Creswon, 2014).

Benzodiazepines work for insomnia by shortening sleep onset, reducing time to REM sleep, and reducing nocturnal awakening (Schroeck et al., 2016). They work by binding to the gamma aminobutyric acid (GABA) receptors in the central nervous system and decreasing neuronal excitation (Schroeck et al., 2016). Older individuals are more sensitive to benzodiazepine effects and have altered pharmacokinetics and pharmacodynamics, which increases the chances of sedation, cognitive impairment, ataxia, and other adverse effects.

Flurazepam, nitrazepam, temazepam, and triazolam are the four benzodiazepines in Canada that are officially indicated for insomnia (Fleming, 2017). However, Flurazepam and nitrazepam have long half lives, have more hangover effects, and cause higher cortical impairment leading to confusion and falls in the elderly, and are therefore not recommended in anyone (Fleming, 2017).

Temazepam is immediate acting, with onset of action of about an hour. Its half life is 10 to 20 hours (CPhA, 2015). It will generally not cause hangover effects, although it can cause a dose dependent next day impairment of activities despite the patient feeling alert (Fleming, 2017). It may also be associated with falls in the elderly (Fleming, 2017; Schroeck et al., 2016). It is metabolized exclusively through glucuronidation, which is not affected by ageing, and so its half life is not prolonged

(Schroeck et al., 2016). It has no clinically significant metabolites. Many other benzodiazepine hypnotics are metabolized via additional pathways, leading to prolonged half lives in the elderly. Temazepam can be used for sleep maintenance at a dose of 7.5mg in older individuals (Schroeck et al., 2016).

Clonazepam has been used for insomnia as well. In the elderly it poses risks and daytime impairment due to its long half life of 20 to 60 hours, which is prolonged in older people (CPhA, 2015). Clonazepam, is metabolized through oxidation and reduction and has no active metabolites (CPhA, 2015). Due to its half life, it works better for sleep maintenance rather than onset.

Triazolam should be avoided in the elderly due to its dose related side effect profile (Fleming, 2017). In some studies, however, it has been used at a dose of 0.125mg to improve sleep onset latency (Schroeck et al., 2016). Its side effects include rebound insomnia, anterograde amnesia, dependence, and anxiety, among others.

While not officially approved by Health Canada as a hypnotic, oxazepam is used for sleep onset insomnia given 60 to 90 minutes prior to sleep, and for sleep maintenance given at bedtime (Fleming, 2017). For healthy young adults the dose is 15mg for insomnia, and for older adults the general recommendation is one half to one third of the younger adult dose (CPhA, 2015). It is intermediate acting with a half life of five to 25 hours, no active metabolites, and without half life prolongation in the elderly.

Compared with benzodiazepines, benzodiazepine receptor agonists have better safety profiles, and less dependence and withdrawal risk (Schroeck et al., 2016). These drugs bind with the GABA receptor complex but have more selective binding to a specific subunit than benzodiazepines, leading to sedation with reduced risk of some

other side effects. For example, fracture risk and risk of falls is lower, although still present (Chun & Lee, 2016). Zopiclone can have a residual daytime hangover due to its longer half life (5-6 hours) compared to zolpidem (2.5-3 hours). Half lives of both drugs are increased in those over age 65 (Schroeck et al., 2016). Side effects of zolpidem also include memory disturbances and complex sleep behaviours like somnambulism or sleep driving (Fleming, 2017). Dosage recommendations for older individuals are not the same as in younger adults. Recommended maximum dose for both zolpidem and zopiclone for those 65 and older is 5mg, with a recommended starting dose of zopiclone is 3.75mg (Chun & Lee, 2016; Fleming, 2017). Some authors advocate for no more than 2.5mg of zolpidem to be used (Schroeck et al., 2016). Both these medications can help to improve sleep onset latency, while zopiclone can also be used for sleep maintenance. Zolpidem and zopiclone have minimal rebound insomnia. As with other drugs, research in the elderly is limited, and they are recommended to be used for no longer than four weeks as their long term use has not been studied (Chun & Lee, 2016; McMillan et al., 2013).

Antipsychotics. There is a growing use of second generation antipsychotics such as off label use of quetiapine for insomnia despite a lack of evidence and safety data for this and recommendations against such use (Trauer et al., 2015). Quetiapine use should be limited to situations where it is indicated, such as for psychotic disorders or severe disturbances in behaviour (Fleming, 2017). Despite this recommendation, low dose quetiapine is widely used off label for insomnia treatment (Anderson & Vande Griend, 2014).

Quetiapine is thought to induce its sedating effects through antagonism at the histamine H1 receptors and at 5-HT2A receptors (Anderson & Vande Griend, 2014).

Some of its side effects include orthostatic hypotension, weight gain, hyperglycemia, and hyperlipidemia. Studies evaluating quetiapine for insomnia have been limited in number and duration of treatment, and have included variable populations with small sample sizes using mostly subjective evaluations like questionnaires (Anderson & Vande Griend, 2014). In the elderly quetiapine is associated with increased risk of death (Anderson & Vande Griend, 2014). Given the limited data, the benefit of quetiapine for sleep has not been proven to outweigh its potential risks.

Antidepressants. Low doses of sedating antidepressants are not recommended to be used for insomnia unless comorbid depression is present (Fleming, 2017; Schroeck et al., 2016). Examples of such drugs include doxepin, trazodone, mirtazapine, and amitriptyline. However, there is some evidence of using very low dose doxepin for insomnia, and it is approved by Health Canada for sleep maintenance insomnia (Fleming, 2017; Schroeck et al., 2016). Given this, only doxepin will be discussed in this paper.

Doxepin is a tricyclic antidepressant with strong antihistamine effects at low doses with selectivity for the H1 receptors (Fleming, 2017). There have been three trials evaluating its use in older adults (Rojas-Fernandez & Chen, 2014; Schroeck et al., 2016). The usual dose used for depression is 25mg to 300mg, while the evaluated dosage for insomnia is 1mg, 3mg, and 6mg. At this low dose, doxepin mostly acts as an antihistamine to produce sedation and improve sleep maintenance, rather than having anti muscarinic or noradrenergic effects as seen in doses over 25mg (Rojas-Fernandez & Chen, 2014). The Beers criteria list doxepin as appropriate for use in doses not exceeding 6mg daily, and write that its safety profile in these low doses is comparable to placebo (Fick at al., 2015).

Doxepin should be taken within 30 minutes of bedtime and not within three hours of food as high fat meals can delay time to peak concentration by three hours (Schroeck et al., 2016). The half life is about 15 hours, and as doxepin undergoes oxidative metabolism via CYP2D6 and CYP2C19, its half life is prolonged in the elderly. NPs should be cognizant of potential drug interactions prior to prescribing doxepin, as some inhibitors of either CYP enzyme pathway include drugs like proton pump inhibitors or bupropion (Schroeck et al., 2016). A published review of the three trials of low dose doxepin in older individuals found the trials showed no difference between the 3mg and 6mg dose (Rojas-Fernandez & Chen, 2014). In this review the authors concluded that evidence shows doxepin improves and sustains sleep maintenance and duration without affecting sleep onset. There are no residual next day effects or discontinuation effects. The drug is well tolerated by people over 65 with the incidence of side effects like headache and sedation being comparable to placebo. A limitation of the current research is that all the studies compared doxepin to placebo only and did not evaluate its use long term; more studies need to be done to compare its effects against commonly used sleep aids. In these low doses doxepin is only available under the brand name Silenor, which can be a limitation if older individuals have financial difficulties buying a brand name drug.

Alcohol. A 2011 study of insomnia in Canada found that 4.6% of people with insomnia self treat with alcohol (Morin et al., 2011). Alcohol use can contribute to insomnia. Patients may see alcohol as helping them to fall asleep as it decreases sleep onset latency by increasing initial somnolence but it also increases sleep fragmentation, increasing nightly awakenings as blood alcohol levels decrease (Gooneratne, & Vitiello,

2014). Use of alcohol for sleep should be assessed as part of the history taking and is discouraged as part of sleep hygiene interventions for insomnia (Gooneratne, & Vitiello, 2014).

Over the counter preparations. Patients with insomnia often self medicate with over the counter products (Krishnan & Hawranik, 2008). This practice needs to be discussed and patients should be informed of potential adverse effects. For example, diphenhydramine should not be used as antihistamine use is associated with sedation, confusion, urinary retention, and dry mouth especially in the elderly. (Krishnan & Hawranik, 2008; Schroeck et al., 2016). After a few days of use antihistamines can also result in residual sedation, impaired cognitive and psychomotor functioning, orthostatic hypotension, blurred vision and tolerance (Krishnan & Hawranik, 2008). The Beers criteria advise against use of diphenhydramine due to its anticholinergic effects (Fick et al., 2015).

Melatonin plays an important role in the sleep wake cycle and is secreted by the pineal gland mostly at night due to its production being suppressed by light (Krishnan & Hawranik, 2008). The suprachiasmic nucleus of the hypothalamus regulates secretion of melatonin by the pineal gland, which helps to regulate circadian rhythm (Schroeck et al., 2016). The secretion of this hormone decreases with age. A meta-analysis showed that melatonin decreases sleep onset latency, increases total sleep time, and improves sleep quality (Ferracioli-Oda, Oawasmi, & Bloch, 2013). However, its effect is smaller than that of other pharmacological options. The general dosage range is usually 1-5mg (Fleming, 2017). However, the optimal dosage in older adults is not known (Vural, van Munster, & de Rooij, 2014). While melatonin is thought to have minimal side effects,

some research suggests that higher doses can more easily lead to supra-physiologic blood levels in older people compared to younger ones, which could increase the risk of drowsiness or unsteadiness the following day. Doses as low as 0.3mg have resulted in physiologic levels of melatonin, with doses above 3mg resulting in supra-physiologic levels (Chun & Lee, 2016). Currently its use and benefits for insomnia are controversial, and data are mixed regarding its effectiveness (Boivin & Boudreau, 2013; McMillan et al., 2013). The ACP reports insufficient evidence for the effectiveness of melatonin in older adults (Qaseem et al., 2016).

Description of the Project

A poster was created to be presented at the UBC Nursing Graduate Student Research Symposium in 2018 (Appendix B). The poster includes background information, diagnostic criteria of insomnia disorder, sleep changes that occur with ageing, elements of history taking and insomnia evaluation, information about CBT-I and its components, and pharmacological options with their respective risks and benefits.

A limitation of the poster is that it will only reach the people who attend the symposium, thus a lot of people will not have access to the information. Another limitation is that it is not possible to assess impact and there is no follow up evaluation of what individuals exposed to the poster learned, apart from asking them about this at the symposium. A benefit of doing a poster presentation is that I will be present and able to answer questions, as well as to evaluate what information attendees may have wanted to see on the poster to help determine whether it is able to adequately address the aims of my project.

The primary target audience of this paper is NPs. The symposium will have many non-NP attendees in addition to NPs and NP students. Disseminating insomnia information to registered nurses as well as NPs can be a benefit of presenting at the symposium. While prescribing is only within the scope of NP attendees, it is my opinion that all attendees will benefit from the poster as nurses have a lot of patient care contact, and can therefore serve to educate patients about insomnia and can potentially have an influence on medications patients use in hospital or community for insomnia. Furthermore, increasing awareness that non pharmacological treatments like CBT-I are first choice for insomnia in any population, and particularly in older adults, may help to address the societal trend of expecting medications to treat insomnia.

This paper addressed the primary aim of the project by discussing the literature on CBT-I as well as currently used pharmacotherapies and potential risks and benefits associated with these to give NPs knowledge of recommended approaches to management in primary care. It also addressed the secondary aims of presenting information on factors contributing to insomnia development in older adults, the diagnostic criteria of insomnia disorder, and approaches to evaluation.

Conclusion

Older individuals are at increased risk of developing sleep difficulties. Contributory factors have been discussed and include things like changes in the sleep cycle and sleep architecture, as well as a higher number of comorbid conditions. NPs need to have a good understanding of these factors in order to be able to assess and discuss these changes with their patients. Having the knowledge that insomnia is not part of healthy ageing should help NPs to recognize and treat this important disorder in their

patients. Patients themselves may not realize their sleep will be different from their younger years, such as the sleep phase shift and increasing fragmentation of their sleep. Even though NPs in primary care may not have the time or additional training to implement CBT-I with patients, they could start addressing potential erroneous beliefs at the start of treatment, which could address patient expectations of their sleep and perhaps alleviate some anxiety.

Given the evidence on insomnia disorder treatment for older adults from articles reviewed for this paper, CBT-I should be the initial approach in all patients. Sleep hygiene recommendations alone should not be used as this is not likely to be effective. A limitation of CBT-I is that it requires special training and time, which may not be available in the primary care setting. It also does not take effect immediately, and patients may be more fatigued within the first week of treatment due to sleep restriction therapy; this could potentially cause some patients to stop treatment. Due to the increased risks of adverse effects associated with medication use in older adults, it is difficult to determine whether initially starting treatment for chronic insomnia with CBT-I and medications during the first three to four weeks could be recommended in older adults. To take a more conservative approach given the risks of medication use in older adults, it is my opinion that the safer management would include only CBT-I, with later thought being given to adding medications if this is not successful.

There are an increasing number of online resources becoming available for CBT-I given the lack of providers trained in this treatment. NPs should be knowledgeable about some of these to be able to refer their patients. Prior to referring to a particular website, NPs should also be aware that there is a possibility that different websites can have

different results. The Sleep Healthy Using the Internet web based CBT-I program has had research showing results equivalent to in person CBT-I. However, the population included only people aged up to 65 (Ritterband et al., 2017). Future research should focus on addressing whether these online CBT-I programs are effective in older individuals. Older adults can have different cognitive beliefs about their insomnia and also have alterations in sleep architecture, which a program directed at all ages may or may not address.

If cognitive and behavioural approaches do not work, a conservative trial of pharmacotherapy can be used. However, most of the drugs which are currently used for insomnia in the elderly do not have evidence supporting their use, or are recommended against due to increased risk of adverse effects. Moreover, there is a lack of long term safety and efficacy studies for various drugs like benzodiazepines, benzodiazepine receptor agonists, and low dose doxepin. Melatonin shows mixed results with some evidence of potential for supra-physiologic levels in older populations, leading potentially to risk of daytime impairment. More research needs to take place with adults over 65 to evaluate drug use in this population given their altered pharmacokinetics and pharmacodynamics. Drug therapy should be approached cautiously and recommendations are that it be for periodic and short term use, usually not more than four weeks.

Benzodiazepines are discouraged in older adults, especially for long term use as in this population the benefits may not justify the potential adverse effects (Schroeck et al., 2016). An interesting issue to consider is that the family clinician may not be the initial prescriber of benzodiazepines for insomnia. These drugs may in fact have been started during a recent hospital admission (McMillan et al., 2013). This means that it is

important for family NPs to periodically re-evaluate medications their patients are on, and ask what is being used for sleep. NPs should also be knowledgeable about benzodiazepine tapering protocols, and be aware of the increased risk of these drugs in older individuals.

Current evidence suggests that the benzodiazepine receptor agonists have a better safety profile compared to benzodiazepines, although their use is also recommended against. An issue contributing to understanding drug therapy and safety in those over 65 is the lack of studies in this population. For future, more research needs to look at long term considerations of use of such drugs in the elderly.

Out of the current medications used for insomnia in Canada, doxepin and melatonin appear to have the lowest associated risks. Low dose doxepin, under the brand name Silenor, is not listed as an unsafe drug for the elderly in the Beers criteria (Fick et al., 2015). Given that physiological changes with ageing lead to early sleep times, more night awakenings, and early wake up times, doxepin may be a good choice for addressing insomnia in this population as it is indicated for sleep maintenance, rather than sleep onset. A downside is the cost of the drug because there is no low dose generic equivalent, which can be a barrier for some older adults on a fixed income. More research needs to be done to evaluate its long term use and effectiveness as compared to other sleep aids in older adults, as thus far the research has compared it against placebo and studies have been short term. If CBT-I and a conservative trial of pharmacotherapy fail, the NP should consider a referral to a sleep specialist for further evaluation and management.

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Appendix A

Sleep diaries taken from: Carney, C. E., Buysse, D. J., Ancoli-Israel, S., Edinger, J. D., Krystal, A. D., Lichstein, K. L., & Morin, C. M. (2012). The consensus sleep diary: Standardizing prospective sleep self-monitoring. *Sleep, 35*(2), 287. doi:10.5665/sleep.1642

Consensus Sleep Diary-Core ID/Name:______________________

	Sample							
Today's date	4/5/11							
1. What time did you get into bed?	10:15 p.m							
2. What time did you try to go to sleep?	11:30 p.m							
3. How long did it take you to fall asleep?	55 min.							
4. How many times did you wake up, not counting your final awakening?	3 times							
5. In total, how long did these awakenings last?	1 hour 10 min.							
6. What time was your final awakening?	6:35 a.m.							
7. What time did you get out of bed for the day?	7:20 a.m							
8. How would you rate the quality of your sleep?	☐ Very poor ☑ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good
9. Comments (if applicable)	I have a cold							

Appendix A

Consensus Sleep Diary-M (Please Complete Upon Awakening) **ID/NAME: ____________________**

	Sample							
Today's Date	4/5/11							
1. What time did you get into bed?	10:15 p.m.							
2. What time did you try to go to sleep?	11:30 p.m.							
3. How long did it take you to fall asleep?	55 min.							
4. How many times did you wake up, not counting your final awakening?	6 times							
5. In total, how long did these awakenings last?	2 hours 5 min.							
6a. What time was your final awakening?	6:35 a.m.							
6b. After your final awakening, how long did you spend in bed trying to sleep?	45 min.							
6c. Did you wake up earlier than you planned?	☑ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No	□ Yes □ No
6d. If yes, how much earlier?	1 hour							
7. What time did you get out of bed for the day?	7:20 a.m.							
8. In total, how long did you sleep?	4 hours 10 min.							
9. How would you rate the quality of your sleep?	□ Very poor ☑ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good	□ Very poor □ Poor □ Fair □ Good □ Very good
10. How rested or refreshed did you feel when you woke-up for the day?	□ Not at all rested ☑ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested	□ Not at all rested □ Slightly rested □ Somewhat rested □ Well-rested □ Very well-rested

Consensus Sleep Diary-M Continued **ID/NAME: ____________________**

	Sample							
Today's Date	4/5/11							
11a. How many times did you nap or doze? 11b. In total, how long did you nap or doze?	2 times 1 hour 10 min.							
12a. How many drinks containing alcohol did you have? 12b. What time was your last drink?	3 drinks 9 :20 p.m.							
13a. How many caffeinated drinks (coffee, tea, soda, energy drinks) did you have? 13b. What time was your last drink?	2 drinks 3 :00 p.m.							
14. Did you take any over-the-counter or prescription medication(s) to help you sleep? If so, list medication(s), dose, and time taken	☑ Yes □ No Medication(s): Relaxo-Herb Dose: 50 mg Time(s) taken: 11 pm	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:	□Yes □No Medication(s): Dose: Time(s) taken:
15. Comments (if applicable)	I have a cold							

Appendix A

Consensus Sleep Diary - E (Please Complete Upon Awakening) **ID/NAME: ____________________**

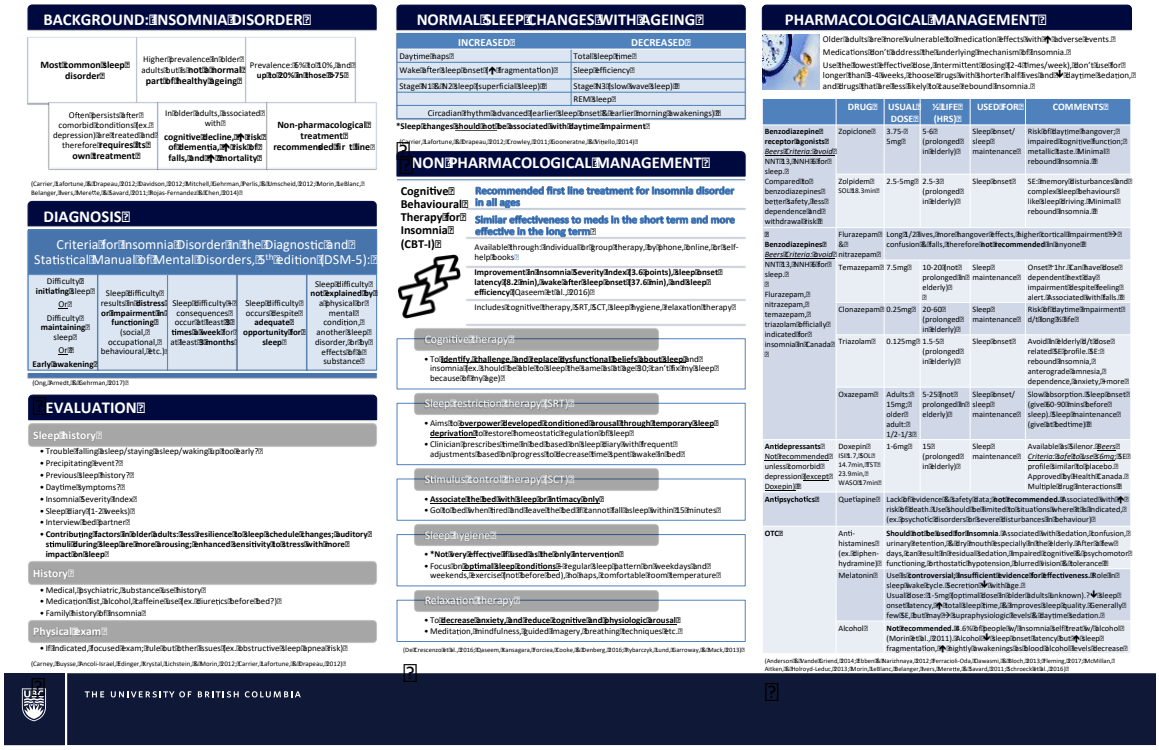
	Sample							
Today's Date	4/5/11							
1. What time did you get into bed?	10:15 p.m.							
2. What time did you try to go to sleep?	11:30 p.m.							
3. How long did it take you to fall asleep?	55 min.							
4. How many times did you wake up, not counting your final awakening?	6 times							
5. In total, how long did these awakenings last?	2 hours 5 min.							
6a. What time was your final awakening?	6:35 a.m.							
6b. After your final awakening, how long did you spend in bed trying to sleep?	45 min.							
6c. Did you wake up earlier than you planned?	☑ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No	☐ Yes ☐ No
6d. If yes, how much earlier?	1 hour							
7. What time did you get out of bed for the day?	7:20 a.m.							
8. In total, how long did you sleep?	4 hours 10 min.							
9. How would you rate the quality of your sleep?	☐ Very poor ☑ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good	☐ Very poor ☐ Poor ☐ Fair ☐ Good ☐ Very good
10. How rested or refreshed did you feel when you woke-up for the day?	☐ Not at all rested ☑ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested	☐ Not at all rested ☐ Slightly rested ☐ Somewhat rested ☐ Well-rested ☐ Very well-rested

Consensus Sleep Diary - E (Please Complete Before Bed) **ID/NAME: ____________________**

	Sample							
Today's Date	4/4/11							
11a. How many times did you nap or doze? 11b. In total, how long did you nap or doze?	2 times 1 hour 10 min.							
12a. How many drinks containing alcohol did you have? 12b. What time was your last drink?	3 drinks 9 :20 p.m.							
13a. How many caffeinated drinks (coffee, tea, soda, energy drinks) did you have? 13b. What time was your last drink?	2 drinks 3 :00 p.m.							
14. Did you take any over-the-counter or prescription medication(s) to help you sleep? If so, list medication(s), dose, and time taken	☑ Yes ☐ No Medication(s): Relaxo-Herb Dose: 50 mg Time(s) taken: 11 pm	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:	☐Yes ☐No Medication(s): Dose: Time(s) taken:
15. Comments (if applicable)	I have a cold							

Appendix B

Culminating Project Poster for the Graduate Student Research Symposium



Insomnia Management In Older Adults

Valerie Moshchenko, BSc, BSN. Supervisors: Fairleth McCuaig, MSN, NP(F); Jillian Harding, MN-NP, NP(F)
University of British Columbia, School of Nursing

BACKGROUND: INSOMNIA DISORDER

Most common sleep disorder

Higher prevalence in older adults but is not a normal part of healthy ageing

Prevalence: 5% to 10%, and up to 20% in those 65-75

Often persists after comorbid conditions (ex. depression) are treated and therefore requires its own treatment

In older adults, associated with cognitive decline, ↑ risk of dementia, ↑ risk of falls, and ↑ mortality

Non-pharmacological treatment recommended for 1st line

(Carrier, Lafortune, Drapeau, 2012; Davidson, 2012; Mitchell, Gehrman, Perlis, Kim scheid, 2012; Morin, Bellanc, Belanger, Ivers, Merette, Savard, 2011; Rojas-Fernandez, Chen, 2014)

DIAGNOSIS

Criteria for Insomnia Disorder in the Diagnostic and Statistical Manual of Mental Disorders, 5th edition (DSM-5):

Difficulty initiating sleep Or Difficulty maintaining sleep Or Early awakening	Sleep difficulty results in distress or impairment in functioning (social, occupational, behavioural, etc.)	Sleep difficulty occurs at least 3 times a week for at least 3 months	Sleep difficulty occurs despite adequate opportunity for sleep	Sleep difficulty not explained by a physical or mental condition, another sleep disorder, or by effects of a substance

(Ong, Arnedt, Gehrman, 2017)

EVALUATION

Sleep history
- Trouble falling asleep/staying asleep/waking up/too early?
- Precipitating event?
- Previous sleep history?
- Daytime symptoms?
- Insomnia Severity Index
- Sleep diary (1-2 weeks)
- Interview bed partner
- **Contributing factors in older adults: less resilience to sleep schedule changes; auditory stimuli during sleep are more arousing; enhanced sensitivity to stress with more impact on sleep**

History
- Medical, psychiatric, substance use history
- Medication list, alcohol, caffeine use (ex. diuretics before bed?)
- Family history of insomnia

Physical exam
- If indicated, focused exam; rule out other issues (ex. obstructive sleep apnea risk)

(Carney, Buysse, Ancoli-Israel, Edinger, Krystal, Lichstein, Morin, 2012; Carrier, Lafortune, Drapeau, 2012)

NORMAL SLEEP CHANGES WITH AGEING

INCREASED	DECREASED
Daytime naps	Total sleep time
Wake after sleep onset (fragmentation)	Sleep efficiency
Stage N1 & N2 sleep (superficial sleep)	Stage N3 (slow wave sleep)
	REM sleep
Circadian rhythm advanced (earlier sleep onset & earlier morning awakenings)	

Sleep changes should not be associated with daytime impairment

(Carrier, Lafortune, Drapeau, 2012; Crowley, 2011; Eisenreatne, & Vitiello, 2014)

NON-PHARMACOLOGICAL MANAGEMENT

Cognitive Behavioural Therapy for Insomnia (CBT-I)

Recommended first line treatment for insomnia disorder in all ages

Similar effectiveness to meds in the short term and more effective in the long term

Available through: individual, group, therapy, by phone, online, brief self-help books

Improvement in Insomnia Severity Index (3.6 points), sleep onset latency (8.2 min), wake after sleep onset (37.6 min), and sleep efficiency (Qaseem et al., 2016)

Includes: cognitive therapy, SRT, SCT, sleep hygiene, relaxation therapy

Cognitive therapy
- To **identify, challenge, and replace dysfunctional beliefs about sleep** and insomnia (ex. 'should be able to sleep the same as I did age 30; I can't fix my sleep' because of my age)

Sleep restriction therapy (SRT)
- Aims to **overpower developed conditioned arousal through temporary sleep deprivation** to restore homeostatic regulation of sleep
- Clinician prescribes time in bed based on sleep diary with frequent adjustments based on progress to decrease time spent awake in bed

Stimulus control therapy (SCT)
- **Associate the bed with sleep or intimacy only**
- Go to bed when tired and leave the bed if cannot fall asleep within 15 minutes

Sleep hygiene
- ***Not very effective if used as the only intervention**
- Focus on **optimal sleep conditions**: regular sleep pattern on weekdays and weekends, exercise (not before bed), no naps, comfortable room temperature

Relaxation therapy
- To **decrease anxiety, and reduce cognitive and physiologic arousal**
- Meditation, mindfulness, guided imagery, breathing techniques, etc.

(DelCresceno et al., 2016; Qaseem, Kansagara, Forciea, Cooke, & Denberg, 2016; Rybarczyk, Lund, Garroway, & Mack, 2013)

PHARMACOLOGICAL MANAGEMENT

Older adults are more vulnerable to medication effects with ↑ adverse events

Medications don't address the underlying mechanism of insomnia

Use the lowest effective dose, intermittent dosing (2-4 times/week), don't use for longer than 3-4 weeks, choose drugs with shorter half lives and ↓ daytime sedation, and drugs that are less likely to cause rebound insomnia

DRUG	USUAL DOSE	½ LIFE (HRS)	USED FOR	COMMENTS
Benzodiazepine receptor agonists *Beers Criteria: Avoid!* NNT 13, NNH 6 for sleep	Zopiclone	3.75-5 5mg	Sleep onset/ sleep maintenance	Risk for daytime hangover, impaired cognitive function, metallic taste. Minimal rebound insomnia.
Compared to benzodiazepines better safety, less dependence and withdrawal risk	Zolpidem SOL 8.3 min	2.5-5mg	2.5-3 (prolonged in elderly)	Sleep onset	SE: memory disturbances and complex sleep behaviours like sleep driving. Minimal rebound insomnia.
Benzodiazepines *Beers Criteria: Avoid!*	Flurazepam & nitrazepam	Long 1, 2 lives, more hangover effects, higher cortical impairment & confusion & falls, therefore **not recommended** in anyone		
NNT 13, NNH 6 for sleep	Temazepam	7.5mg	10-20 (not prolonged in elderly)	Sleep maintenance	Onset 1hr. Can have dose dependent next day impairment despite feeling alert. Associated with falls.
Flurazepam, nitrazepam, temazepam, triazolam diffically indicated for insomnia in Canada	Clonazepam	0.25mg	20-60 (prolonged in elderly)	Sleep maintenance	Risk for daytime impairment and long life
	Triazolam	0.125mg	1.5-5 (prolonged in elderly)	Sleep onset	Avoid in elderly & dose related. Short profile. Some rebound insomnia, anterograde amnesia, dependence, anxiety, more
	Oxazepam	Adults 15mg, older adult 1/2-1/3	5-25 (not prolonged in elderly)	Sleep onset/ sleep maintenance	Slow absorption. Sleep onset (give 60-90 mins before sleep). Sleep maintenance (give at bedtime)
Antidepressants Not recommended unless comorbid depression Except Doxepin	Doxepin Is 7, SOL 14.7 min, TST 23.9 min, WASO 17 min	1-6mg	15 (prolonged in elderly)	Sleep maintenance	Available as Silenor. *Beers Criteria: Safer dose 3mg.* SE profile similar to placebo. Approved by Health Canada. Multiple drug interactions
Antipsychotics	Quetiapine	Lack of evidence & safety data; **not recommended.** Associated with ↑ risk of death. Use should be limited to situations where it is indicated, (ex. psychotic disorders or severe disturbances in behaviour)		
OTC	Anti-histamines (ex. diphenhydramine)	**Should not be used for insomnia.** Associated with sedation, confusion, urinary retention, & dry mouth especially in the elderly. After a few days, can result in residual sedation, impaired cognitive & psychomotor functioning, orthostatic hypotension, blurred vision & tolerance		
	Melatonin	Used **controversially. Insufficient evidence for effectiveness.** Role in sleep wake cycle. Secretion ↓ with age. Usual dose: 1-5mg (optimal dose in older adults unknown). ? ↓ sleep onset latency, ? ↑ total sleep time, & improves sleep quality. Generally well tolerated, but may ? ↑ supraphysiologic levels & daytime sedation.		
	Alcohol	**Not recommended.** 33.6% of people with insomnia self treat with alcohol (Morin et al., 2011). Alcohol ↓ sleep onset latency, but ↑ sleep fragmentation, ↑ nightly awakenings as blood alcohol levels decrease		

(Anderson & Vander Eriend, 2014; Ebben & Narinhaya, 2012; Ferracioli-Oda, Etsewier, & Bloch, 2013; Fleming, 2017; McMillan, Aitken, & Honroyd-Leduc, 2013; Morin, Bellanc, Belanger, Ivers, Merette, & Savard, 2011; Schroeck et al., 2016)

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