Title:
Examining Cardiovascular Symptoms in Adults with Chronic Insomnia

Authors: Suzanne Fredericks, Souraya Sidani, Joyal Miranda, Mary Fox

Correspondence:

Abstract

Keywords: insomnia, anxiety, depression, cardiovascular symptoms, physical activity, alcohol, caffeine, smoking

Introduction

Chronic insomnia is characterized by difficulty initiating sleep, difficulty maintaining sleep, waking up too early, non-restorative sleep, and daytime impairment, experienced for six or more weeks (American Academy of Sleep Medicine, 2005). Its prevalence is estimated at 50.0% of the general population worldwide (Irish et al., 2015; Kredlow et al., 2015). The experience of chronic insomnia is associated with an increased risk for chronic diseases, in particular cardiovascular diseases (King et al., 2008; Tsunoda et al., 2015). Evidence from cross-sectional and longitudinal studies demonstrates a significant relationship between insomnia and risk of cardiovascular diseases, after controlling for sleep disorders that are physiological in nature (e.g. sleep apnea) and for other risks of cardiovascular diseases such as smoking, low levels of physical activity, and alcohol and caffeine consumption (Foley et al., 2004; Hovenaar-Blom et al., 2011; Phillips et al., 2007). This evidence suggests that non-physiological factors contribute to the risk of cardiovascular diseases in persons with chronic insomnia. Stress, manifested in anxiety and depression, is often experienced by persons with chronic insomnia (Specchio et al., 2004; Taylor et al., 2007), and those with anxiety report cardiovascular symptoms such as chest pain and dyspnea (Ketterer et al., 2008). There is limited research that explored the contribution of psychological (i.e. insomnia, anxiety, and depression) or physical factors (smoking, physical activity, and alcohol and caffeine consumption) to the experience of cardiovascular symptoms (which are indicative of cardiovascular diseases) among adults with chronic insomnia.

Aims

The overall purpose of this study was to examine the relationships between physical and psychological factors, and the experience of cardiovascular symptoms among adults with chronic insomnia.

The specific objectives were to: 1) describe the experience of cardiovascular symptoms (i.e. dizziness, fainting, chest pain, heart racing, difficulty breathing) in adults with chronic insomnia; and 2) examine the relationships between physical (smoking, physical activity, and alcohol and caffeine consumption) and psychological (insomnia severity, state anxiety, depression) factors, and the experience of cardiovascular symptoms (i.e. pain). Understanding these relationships is useful in informing the development of educational and behavioral interventions for managing the factors and preventing cardiovascular diseases.

Related literature

Physical factors

Smoking, alcohol and caffeine consumption, and low levels of physical activity are well known physical factors that contribute to chronic insomnia and cardiovascular diseases. Studies have found that smoking leads to arousal and wakefulness, which disrupt sleep (Phillips et al., 2007). Smoking has also been found to be a major cause of coronary heart disease; it increases the risk of developing thrombosis and atherosclerosis of narrowed vessels (Burns, 2003). This risk increases with the amount of cigarettes smoked and the duration of smoking (Burns, 2003).

Similar to smoking, alcohol also affects sleep. Alcohol consumption can increase arousal during the second half of the night (Irish et al., 2015). As well, individuals who

consume significantly less alcohol had a lower risk for the development of cardiovascular factors (i.e. increased systolic blood pressure, interleukin-6 levels, waist circumference, and body mass index) associated with the onset of cardiovascular disease (Holmes et al., 2014; Rimm et al., 1991). These findings suggest that a reduction in alcohol consumption, even for light to moderate drinkers, may be beneficial for cardiovascular health. The findings challenge the concept of a cardio-protective effect associated with light to moderate alcohol consumption reported in observational studies and suggest that this effect may be due to selection bias (Holmes et al., 2014; Rimm et al., 1991). The findings support the hypothesis that an inverse relation between alcohol consumption and risk of coronary disease is causal (Holmes et al., 2014; Rimm et al., 1991).

Another physical factor that can affect sleep is caffeine consumption. Caffeine shifts sleep architecture toward lighter sleep (Drake et al., 2013). However, unlike alcohol and cigarette smoking, caffeine, in the form of cocoa, coffee, and tea can have optimal health effects on the prevention of cardiovascular diseases (Bøhn et al., 2012; Di Castelnuovo et al., 2012). The phytochemicals found within caffeine can protect against the onset of cardiovascular disease by regulating the vascular tone of vessels, improving glucose metabolism, increasing reverse cholesterol transport and enhancing platelet function. However, the positive effect of caffeine vanishes with heavy consumption (Bøhn et al., 2012; Di Castelnuovo et al., 2012).

Lastly, a positive association between levels of engagement in physical activity and sleep disturbances has been demonstrated. Results of systematic reviews and meta-analyses showed that engagement in physical activity, consisting of moderate

intensity exercises, performed for ≥ 30 minutes per day on 5 days of the week over 10-52 weeks, was beneficial in improving subjective sleep quality and reducing sleep onset latency (Kredlow et al., 2015; Passos et al., 2012) in persons with and without sleep problems (Hartescu et al., 2014). As well, low levels of physical activity have been shown to significantly affect the onset of cardiovascular disease (Bastien et al., 2014; Ford et al., 2012) and to promote increased body fat distribution, which can alter the interaction between vital organs (i.e. heart) and tissues resulting in narrowed arteries and/or formation of clots (Bastien et al., 2014).

Psychological factors

Similar to the physical factors, insomnia, anxiety and depression are interrelated and influence cardiovascular symptoms. Anxiety and depression are well recognized factors that precipitate and perpetuate insomnia (Griffith et al., 2005; Thase, 2005). Evidence indicates that anxiety and depression are inversely related to sleep (Specchio et al., 2004). Specifically, Taylor et al. (2007) found that persons experiencing insomnia were 10 times more likely to have depression and 17 times more likely to report anxiety than good sleepers. The relationship between insomnia and cardiovascular diseases has been reported in epidemiological studies (Foley et al., 2004; Suka et al., 2003; Sutton et al., 2001) and prospective cohort studies (Phillips et al., 2007). Insomnia, anxiety and depression are prevalent among patients with cardiovascular diseases. Carney et al. (1990) found that 39.0% of patients reported insomnia and 23.0% met the criteria for major depression prior to myocardial infarction. Stress and related psychological reactions (anxiety and depression) were found to correlate with myocardial infarction events (Gulletter et al., 1997). Similarly, Ketterer et al. (2008) explored the association

between psychological distress and cardiovascular symptoms (i.e. chest pain, dyspnea, presyncope, and palpitation); anxiety was positively related to chest pain and dyspnea, accounting for 10.0% of the variance in each symptom, after controlling for other physical factors (e.g. smoking) that increase the risk of cardiovascular diseases.

Methods

Design

Data were obtained from participants in a trial of cognitive behavioral therapies (CBT) for insomnia. Baseline data were analyzed to address the study objectives. Eligible participants attended the baseline data collection session at the study research office. At the beginning of the session, the researcher described the study in detail, clarified participants' rights and risks and benefits of their involvement, addressed any question participants may still have, and secured their written consent. Consenting participants completed the pretest questionnaire measuring socio-demographic characteristics and the study variables.

Sample

Persons with insomnia were eligible for the study if they 1) were community-dwelling, non-institutionalized middle or older-aged adults ($\geq$ 40 years of age), 2) had the ability to read and write English (required for providing written consent), and 3) complained of difficulty initiating and/or maintaining sleep, manifested in sleep onset latency and/or time awake after sleep onset of 30 minutes or more per night, for a minimum of 3 nights per week, for a duration of at least 6 weeks (National Institute of Health, 2005), ascertained with relevant questions of the Insomnia Interview Schedule (Morin, 1993). Exclusion criteria were: 1) cognitive impairment as ascertained by the

Mini-Mental State Exam (MMSE) score < 24 (Crum et al., 1993); and 2) confirmed medical diagnosis and current treatment for sleep apnea as reported by participants.

Participants with insomnia were recruited through community media advertisement made in local newspapers and community newsletters targeting middle and older-aged adults. Flyers advertising the study were distributed to community health centers, hospital ambulatory services or outpatient clinics and sleep clinics. Advertisements and flyers specified voluntary participation in a research study, general selection criteria (age and complaint of insomnia), and the phone number to contact if interested.

Sample size

A total of 204 participants provided baseline data. This sample was more than adequate to run the multiple regression analysis (addressing objective 2), based on the rule of having 10 cases per predictors x 7 predictors (smoking, physical activity, alcohol consumption, caffeine consumption. insomnia severity, anxiety and depression.

Variables and Measures

Screening variables

The Insomnia Interview Schedule (IIS) was used to screen for insomnia (difficulty initiating or maintaining sleep), duration of sleep onset latency and wake after sleep onset (≥ 30 minutes), duration of insomnia, and diagnosis of sleep apnea. The IIS was developed and tested for content validity by Barlow (1993).

Cognitive impairment was measured with the MMSE, which is an 11-item interview format questionnaire with established reliability and validity (Crum et al., 1993).

Demographic characteristics

Participants' demographic characteristics were measured with standard questions gathering information on age, sex, marital status, level of education, employment status, and ethnicity.

Insomnia severity

Insomnia severity was measured with the Insomnia Severity Index (ISI) developed by Morin (1993). The ISI is comprised of 7 items assessing severity of insomnia, satisfaction with sleep patterns and distress with the sleep problem. The ISI has demonstrated acceptable internal consistency reliability ($\alpha \geq .85$) and validity as evidenced by correlation with other subjective and objective measures of insomnia severity (Morin et al., 2011).

Anxiety

Anxiety was assessed with the 20-item State Trait Anxiety Inventory (STAI) – State scale developed by Spielberger et al. (1970). The STAI has been used with various patient populations; it has demonstrated internal consistency reliability ($\alpha > .85$), as well as construct validity (Spielberger et al., 1994).

Depression

Depression was measured with the Center for Epidemiology Studies – Depression (CESD) scale developed by Radloff (1977). The CESD assesses mood in general population surveys and has been used with different populations in experimental studies. It is internally consistent ($\alpha > .80$) and valid evidenced by its correlation with other measures of depression (Naughton et al., 1996).

Smoking

Smoking was assessed with two items, one related to whether or not participants smoked and the other related to the number of cigarettes smoked per day.

Physical activity

Physical activity was assessed with two items: whether or not participants engaged in physical activity and the number of physical activities they engaged in per day.

Caffeine consumption

Caffeine consumption was assessed with two items: whether or not participants drink caffeinated beverages and the number of cups per day.

Alcohol consumption

Alcohol consumption was also assessed with two items: whether or not participants drink alcohol and the number of drinks they have, on average, per day.

Cardiovascular symptoms

Cardiovascular symptoms of dizziness, fainting, chest pain, heart racing, difficulty breathing were measured with relevant items derived from the Symptom Checklist (SCL) – 90R. The items ask participants to indicate the frequency and level of distress experienced with each symptom. The items were used by Ketterer et al. (2008) to measure symptoms attributed to cardiovascular diseases, and were found to be related to psychological distress (i.e. anxiety and depression).

Data analysis

Descriptive statistics were used to describe the experience of cardiovascular symptoms by adults with chronic insomnia. Multiple regression analyses were used to examine the relationships between physical (smoking, physical activity, caffeine and

alcohol consumption) and psychological (insomnia severity, state anxiety, depression) factors, and cardiovascular symptom experience. Factors were entered (forced entry) into three blocks: 1) physical factors, 2) anxiety, and 3) insomnia severity. Depression was excluded due to its high correlation with anxiety and insomnia severity

Results

Description of sample

On average, participants were middle-aged adults (60.4 $\pm$ 10.4, range: 40-89 years). Most (70.6%) were women. The number of formal education years ranged from 4 to 34 years, with a mean of 16.5 $\pm$ 4.3 years. About one half were unemployed (37.4% were retired and 12.3% had no job), whereas 35.0% were employed on a full time basis and 14.8% on a part-time basis. Fifty one percent (51.0%) of the participants were married and 49.0% were not married (24.5% single, 16.4% divorced, 4.4% widowed, 3.9% separated). The majority were white (77.0%); 15.2% were Asian, and the remaining reported different racial backgrounds including Black, Hispanic, and First Nation.

Study variables

The overwhelming majority of participants (95.1%) did not smoke. The 10 smokers reported having 1 to 23 cigarettes per day. Similarly, the majority of participants (94.6%) reported engagement in physical activity 0 to 5 times per day, with each time lasting between 10 and 480 minutes. Slightly more than half (59.7%) of the participants reported having 0 to 4 alcoholic drinks per day. In total, 81 (40.3%) participants reporting no alcohol consumption. Most participants (79.3%) indicated that

they had caffeine beverages ranging from 0 to 6.5 cups per day. As well, 42 (20.7%) participants reporting no alcohol consumption.

Participants experienced difficulty initiating sleep (61.8%), difficulty maintaining sleep (97.8%) and early morning awakening (83.1%). On average, they experienced clinical insomnia of moderate severity, evidenced by a mean ISI score of 17.7 $\pm$ 4.6 (range: 5 – 28).

In general, participants reported low levels of anxiety (36.5 $\pm$ 10.7, range: 22 – 75) and depression (15.3 + 10.0, range: 0 – 48). Participants reported experiencing and being distress about cardiovascular symptoms to varying degrees (Table 1). On average, the total score indicated that participants rarely experienced these symptoms (0.3 $\pm$ 0.4, range: 0 – 2) and were distressed a little about them (0.9 $\pm$ 0.8, range: 0 – 3).

Relationship between physical and psychological factors and cardiovascular symptoms

The results of the regression analyses are presented in Table 2. For cardiovascular symptom frequency, the contribution of physical factors (smoking, and alcohol and caffeine consumption) was no longer statistically significant once the psychological factors were entered into the equation, suggesting a possible mediated relationship. High levels of anxiety and increased severity of insomnia were positively associated with the frequent experience of cardiovascular symptoms. In contrast, smoking was the only physical factors with a statistically significant influence on cardiovascular symptom distress; this influence was still significant when anxiety and insomnia were entered into the regression equation. Increased levels of smoking, anxiety and insomnia severity were associated with increased levels of distress regarding cardiovascular symptom experience.

Discussion

Psychological (insomnia severity and anxiety) and physical factors (smoking, physical activity, and alcohol and caffeine intake) explained approximately 5.0%, 13.0%, and 15.0 % of the variation in cardiovascular symptom frequency respectively, and about 7.0%, 18.0%, and 20.0% of the variation in cardiovascular symptom distress. The relatively small variation in both cardiovascular symptom frequency and distress accounted for by the physical factors may be due to the distribution of the responses on these variables. The dataset that was used in the regression analyses was obtained from a larger study that examined the effects of CBT for insomnia. The number of participants with insomnia who smoke and consume alcohol and caffeine was low; this may be explained by their adherence to sleep hygiene recommendations. Sleep hygiene is a widely disseminated intervention for insomnia that consists of recommendations to promote sleep, including avoiding nicotine, caffeine and alcohol before bedtime. (Stepanski & Wyatt, 2003).

Even though the majority of participants reported minimal to moderate levels of caffeine consumption, a relatively small variation in cardiovascular symptom frequency and distress was noted. This may be due to the overwhelmingly positive effects associated with moderate caffeine intake that include improvement in endothelial cell function, which can result in an increase in blood flow throughout the circulatory system (Papamichael et al., 2005; Ranheim & Halvorsen, 2005). Caffeine is also a rich source of heterocyclic, which is a compound that contains many antioxidant properties. This is significant, as antioxidants are used to prevent disease by fighting free radicals

substances. Without adequate amounts of antioxidants, free radicals can travel throughout the body, damaging cells (Fuster et al., 2000).

As well, approximately 20.0% of the variation noted in cardiovascular symptom distress was related to insomnia and anxiety. Increased levels of anxiety have been shown to increase an individual's vulnerability to arrhythmias, as a result of decreased secretion of adrenaline (Esler, 2006). Furthermore, research findings have noted heightened levels of anxiety can result in an increase in the level of platelet clotting (Aschbacher et al., 2008). Both arrhythmias and the formation of clots are indications of cardiovascular disease.

Finally, results from a systematic review of all available prospective studies that investigated the association between insomnia and cardiovascular diseases suggest that insomnia is strongly associated with an increased risk of developing and/or dying from cardiovascular diseases (Laugsand et al., 2011; Sofi, 2014). Sofi (2014) reported that participants who suffered from insomnia had a 45.0% increased risk of morbidity and/or mortality from cardiovascular disease in comparison to those who did not suffer from insomnia. In particular, Sofi's (2014) study results indicate poor quantity and/or quality of sleep, alterations in duration of sleep, and the presence of non-restorative sleep stimulates changes in the metabolic and endocrine systems that result in elevated levels of inflammatory cytokines, leading to the early onset of cardiovascular disease. From a metabolic perspective, reduced sleep has been shown to increase the risk of type 2 diabetes, through impaired glucose tolerance, which can result in the onset of cardiovascular disease (Laugsand et al., 2011; Sofi, 2014).

Similarly, obesity, which has been described as a significant indicator for cardiovascular disease (Mozaffarian, 2016), has been shown to develop in healthy young adults who experience sleep related issues (Spiegel et al., 2009). Spiegel (2009) reasoned that sleep impairment increases cortisol secretion, which then alters the circulating levels of growth hormone, leptin and ghrelin, resulting in an increased risk for obesity.

Implications

Based on the findings, relatively small variations in both cardiovascular symptom frequency and distress were related to insomnia severity and anxiety. Even though the percentage of variation is somewhat small, there is still a need to manage both anxiety and insomnia to prevent the onset of cardiovascular symptoms.

Nurses and health care providers should consider integrating CBT and relaxation therapy into their plan of care for patients who present with insomnia and who may be at high risk of developing of cardiovascular diseases. These types of therapy have been shown to be effective in producing significant improvements in total sleep time and sleep efficiency (Morin et al., 2006). CBT and relaxation therapy can be integrated to assist persons with insomnia successfully address insomnia and anxiety, and therefore reduce the risk of experiencing cardiovascular symptoms. CBT that nurses and health care providers may consider using for individuals with insomnia include: 1) a cognitive therapy module to correct misconceptions about sleep requirements; 2) stimulus control instructions (i.e. standard wake up time, need to get out of bed during prolonged awakening, and need to eliminate day time napping); and 3) sleep restriction therapy that focuses on developing a regular sleep-wake schedule (Edinger et al., 2001).

Music can also be used as a relaxation therapy to assist individuals in relaxing before going to sleep, as it affects brain waves (Johnson, 2003). In particular, the use of music at before bedtime has been shown to decrease anxiety, arousal, and time to sleep onset. In order to effectively implement this therapy, nurses and other healthcare providers will need to ensure that the music selected is based on the preferences identified by the individual.

Furthermore, relaxation therapies have demonstrated a positive effect in reducing anxiety and improving sleep quality in individuals with insomnia (Means, et al., 2000) and at increased risk of developing cardiovascular disease (Gagne & Toye, 1994). Relaxation therapies are any type of procedure that helps individuals achieve a state of calmness and serenity (Gagne & Toye, 1994). Therapeutic touch, muscle massage, meditation, visualization, and deep breathing exercises are examples of relaxation activities that nurses and health care providers can incorporate into their plan of care.

Finally, the empirical literature should be systematically reviewed to determine the link and/or nature of association between insomnia, anxiety, and cardiovascular disease. Using the findings generated from this review, a study with a rigorous design, containing a larger, heterogeneous sample should be conducted to examine the mediated relationship between insomnia, anxiety, and the onset of cardiovascular disease. The findings from these studies can be used to design single cost-effective therapies that can be implemented to manage a number of chronic conditions (insomnia, anxiety, and cardiovascular diseases). The long-term effects of insomnia may also link to cardiovascular issues.

Conclusion

This study examined the relationships between physical and psychological factors, and the experience of cardiovascular symptoms among adults with chronic insomnia. Results suggested minimal variations in both cardiovascular symptom frequency and distress which were deemed to be related to insomnia severity and anxiety more so than to physical factors. The use of CBT, music, and relaxation based therapies can be applied to manage both anxiety and insomnia to prevent the onset of cardiovascular symptoms.

References

American Academy of Sleep Medicine, diagnostic and coding manual (2005) International classification of sleep disorders. Westchester, IL: 148-52

Aschbacher K, Mills PJ, von Känel R et al (2008) Effects of depressive and anxious symptoms on norepinephrine and platelet P-selectin responses to acute psychological stress among elderly caregivers. *Brain Behav Immun* **22(4):** 493-502

Barlow DH (ed) (1993) Insomnia: Psychological assessment and management New York: Guilford Press.

Bastien M, Poirier P, Lemieux I, Després JP (2014) Overview of epidemiology and contribution of obesity to cardiovascular disease. *Prog Cardiovasc Dis* **56(4)**: 369-381

Bøhn SK, Ward NC, Hodgson JM, Croft KD (2012) Effects of tea and coffee on cardiovascular disease risk. *Food Funct* **3(6):** 575-591

Burns DM (2003) Epidemiology of smoking-induced cardiovascular disease. *Prog Cardiovasc Dis* **46(1):** 11-29

Carney RM, Freedland KE, Jaffe AS (1990) Insomnia and depression prior to myocardial infarction. *Psychosom Med* **52(6):** 603-609

Crum RM, Anthony JC, Bassett S, Folstein MF (1993) Population-based norms for the Mini-Mental State Examination by age and educational level *JAMA* **269 (18):** 2386-2391

Di Castelnuovo A, Di Giuseppe R, Iacoviello L, de Gaetano G (2012) Consumption of cocoa, tea and coffee and risk of cardiovascular disease. *Eur J Intern Med* **23(1):** 15-25

Drake C, Roehrs T, Shambroom J, Roth T (2013) Caffeine effects on sleep taken 0, 3, or 6 hours before going to bed. *J Clin Sleep Med* **9(11):** 1195-1200

Edinger JD, Wohlgemuth WK, Radtke RA, et al (2001) Cognitive behavioral therapy for treatment of chronic primary insomnia: a randomized controlled trial. *JAMA* **285(14):** 1856-1864

Esler M, Alvarenga M, Pier C et al (2006) The neuronal noradrenaline transporter, anxiety and cardiovascular disease. *J Psychopharmacol* **20(4 suppl):** 60-66

Foley D, Ancoli-Israel S, Britz P, Walsh J (2004) Sleep disturbances and chronic disease in older adults: results of the 2003 National Sleep Foundation Sleep in America Survey. *J Psychosom Res* **56(5):** 497-502

Ford ES, Caspersen, CJ (2012) Sedentary behaviour and cardiovascular disease: A review of prospective studies. *Int J Epidemiol* **41(5):** 1338-1353.doi: 10.1093/ije/dys078

Fuster MD, Mitchell AE, Ochi H, Shibamoto T (2000) Antioxidative activities of heterocyclic compounds formed in brewed coffee. *J Agric Food Chem* **48(11):** 5600-5603

Gagne D, Toye RC (1994) The effects of therapeutic touch and relaxation therapy in reducing anxiety. *Arch Psychiatr Nurs* **8(3):** 184-189

Griffiths MF, Peerson A (2005) Risk factors for chronic insomnia following hospitalization. *J Adv Nurs* **49(3):** 245-253

Guletter EC, Blumenthal JA, Babyak M et al (1997) Effects of mental stress on myocardial ischemia during daily life. *JAMA* **277(19)**, 1521-1526

Hartescu I, Morgan K, Stevinson CD (2015) Increased physical activity improves sleep and mood outcomes in inactive people with insomnia: a randomized controlled trial. *J Sleep Res* **24(5)** 526-534

Harvey Moldofsky MD (2001) Insomnia and health problems in Canadians. *Sleep*, **24(6):** 665

Hoevenaar-Blom MP, Spijkerman AM, Kromhout D, et al (2011) Sleep duration and sleep quality in relation to 12-year cardiovascular disease incidence. *Sleep* **34(11):** 1487-1492

Holmes MV, Dale CE, Zuccolo L et al (2014) Association between alcohol and cardiovascular disease: Mendelian randomisation analysis based on individual participant data. *BMJ* **349**: g4164

Irish LA, Kline CE, Gunn HE, et al (2015) The role of sleep hygiene in promoting public health: A review of empirical evidence. *Sleep Med Rev* **22(1):** 23-36

Ketterer MW, Knysz W, Keteyian SJ, et al (2008) Cardiovascular symptoms in coronary-artery disease patients are strongly correlated with emotional distress. *Psychosom* **49(3):** 230-234

Johnson JE (2003) The use of music to promote sleep in older women. *J Community Health Nurs* **20(1):** 27-35

King AC, Pruitt LA, Woo S et al (2008) Effects of moderate-intensity exercise on polysomnographic and subjective sleep quality in older adults with mild to moderate sleep complaints. *J Gerontology Series* **63(9)**: 997-1004

Kredlow MA, Capozzoli MC, Hearon BA, et al (2015) The effects of physical activity on sleep: a meta-analytic review. *J behav med* **38(3):** 427-449

Laugsand LE, Vatten LJ, Platou C, Janszky I (2011) Insomnia and the risk of acute myocardial infarction a population study. *Circ* **124(19):** 2073-2081

Means MK, Lichstein KL, Epperson MT, Johnson CT (2000) Relaxation therapy for insomnia: nighttime and day time effects. *Behav Res Ther* **38(7):** 665-678

Morin CM, Belleville G, Bélanger L, Ivers H (2011) The Insomnia Severity Index: psychometric indicators to detect insomnia cases and evaluate treatment response. *Sleep* **34(5):** 601-608

Morin CM, Bootzin RR, Buysse DJ, et al (2006) Psychological and behavioral treatment of insomnia: update of the recent evidence (1998-2004). *Sleep* **29(11):** 1398

Mozaffarian D (2016) Dietary and Policy Priorities for Cardiovascular Disease, Diabetes, and Obesity A Comprehensive Review. *Circ* **133(2):** 187-225

National Institutes of Health (2005) National Institutes of Health State of the Science Conference statement on Manifestations and Management of Chronic Insomnia in Adults. *Sleep* **28(9):** 1049

Naughton MJ, Shumaker SA, Anderson RT, Czajkowski SM (1996) Psychological aspects of health-related quality of life measurement: tests and scales. Quality of Life and Pharmaco economics in *Clinl Trials* **15(1):** 117-131

Papamichael CM, Aznaouridis KA, Karatzis EN, et al (2005) Effect of coffee on endothelial function in healthy subjects: the role of caffeine. Clinical Science 109(1): 55-

60.Passos, G.S., Poyares, D.L.R., Santana, M.G., et al (2012) Is exercise an alternative treatment for chronic insomnia? *Clinics* **67(6):** pp.653-660

Phillips B, Mannino DM (2007) Do insomnia complaints cause hypertension or cardiovascular disease. *J Clin Sleep Med* **3(5):** 489-494

Radloff LS (1977) The CES-D scale a self-report depression scale for research in the general population. *Appl Psychol Meas* **1(3):** 385-401

Ranheim T, Halvorsen B (2005) Coffee consumption and human health–beneficial or detrimental?–Mechanisms for effects of coffee consumption on different risk factors for cardiovascular disease and type 2 diabetes mellitus. *Mol Nutr Food Res* **49(3):** 274-284

Rimm EB, Giovannucci EL, Willett WC, et al (1991) Prospective study of alcohol consumption and risk of coronary disease in men. *Lancet* **338(8765):** 464-468

Specchio LM, Iudice A, Specchio N, et al (2004) Citalopram as treatment of depression in patients with epilepsy. *Clin Neuropharmacol* **27(3):** 133-136

Sofi F, Cesari F, Casini A, et al (2014) Insomnia and risk of cardiovascular disease: A meta-analysis. *Eur J Prev Cardiol* **21(1):** 57-64

Spiegel K, Tasali E, Leproult R, Van Cauter E (2009) Effects of poor and short sleep on glucose metabolism and obesity risk. *Nature Rev Endocrinol* **5(5):** 253-261

Spielberger CD, Gorsuch RL, Lushene RE (1970) State-trait anxiety inventory (self-evaluation questionnaire). Consulting Psycholol gists Press

Spielberger CD, Sydeman SJ (1994) State-Trait Anxiety Inventory and State-Trait Anger Expression Inventory

Stepanski EJ, Wyatt JK (2003) Use of sleep hygiene in the treatment of insomnia. *Sleep Med Rev* **7(3):** 215-225

Suka M, Yoshida, K, Sugimori H (2003) Persistent insomnia is a predictor of hypertension in Japanese male workers. *J Occup Health* **45(6):** 344-350

Taylor DJ, Lichstein KL, Durrence HH, et al (2005) Epidemiology of insomnia, depression, and anxiety. *Sleep* **28(11):**1457

Thase ME (2005) Correlates and consequences of chronic insomnia. *Gen Hosp Psychiatry* **27(2):** 100-112

Tsunoda K, Kitano N, Kai Y, et al (2015) Prospective study of physical activity and sleep in middle-aged and older adults. *Am J Prev Med* **48(6):** 662-673

Table 1
Cardiovascular symptom experience

Symptom	Percentage of participants not experiencing symptom	Percentage of participants experiencing symptom	Mean (SD) score
Dizziness	67.6	32.4	0.50 (0.85)
Distress	41.2	58.8	0.98 (1.01)
Fainting	98.5	1.5	0.01 (0.12)
Distress	92.3	7.7	0.15 (0.60)
Chest pain	75.5	24.5	0.36 (0.75)
Distress	42.0	58.0	1.11 (1.18)
Palpitation	66.0	34.0	0.51 (0.84)
Distress	43.0	57.0	1.01 (1.12)
Dyspnea	83.8	16.2	0.22 (0.58)
Distress	64.8	35.2	0.66 (1.01)

Table 2
Contributing factors

Dependent Variable = Cardiovascular symptom frequency	
Block 1: adj R2 = .057	
Smoking	β = .18 (p = .01)
Alcohol	β = - .49 (p = .04)
Caffeine	β = -.04 (p > .05)
Physical activity	β = - .11 (p > .05)
Block 2: adj R2 = .134	
Smoking	β = .10 (p > .05)
Alcohol	β = - .10 (p > .05)
Caffeine	β = -.09 (p > .05)
Physical activity	β = - .10 (p > .05)
Anxiety	β = .30 (p < .001)
Block 3: adj R2 = .152	
Smoking	β = .10 (p > .05)
Alcohol	β = - .09 (p > .05)
Caffeine	β = -.08 (p > .05)
Physical activity	β = - .09 (p > .05)
Anxiety	β = .25 (p = .001)
Insomnia severity	β = .16 (p = .024)

DV = Cardiovascular symptom distress	
Block 1: adj R2 = .077	
Smoking	β = .29 (p = .001)
Alcohol	β = .04 (p > .05)
Caffeine	β = -.08 (p > .05)
Physical activity	β = - .13 (p > .05)
Block 2: adj R2 = .178	
Smoking	β = .18 (p = .033)
Alcohol	β = .11 (p > .05)
Caffeine	β = -.16 (p > .05)
Physical activity	β = - .10 (p > .05)
Anxiety	β = .35 (p < .001)
Block 3: adj R2 = .197	
Smoking	β = .19 (p = .027)
Alcohol	β = .10 (p > .05)
Caffeine	β = -.14 (p > .05)
Physical activity	β = - .08 (p > .05)
Anxiety	β = .298 (p = .002)
Insomnia severity	β = .168 (p = .052)