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Addressing sleep disturbances: An opportunity to prevent cardiometabolic disease?

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Abstract

There is increasing awareness of the role of sleep disturbance as an important factor in health and disease. Although subclinical sleep disturbances (insufficient sleep duration or inadequate sleep quality) may be difficult to assess with conceptual and/or methodological clarity, this review attempts to summarize and synthesize these findings. First, the concept of sleep disturbance in a public health context is introduced, to provide context and rationale. Second, operational definitions of ‘cardiometabolic disease’ and ‘sleep disturbance’ are offered, to address many unclear operationalizations. Third, the extant literature is summarized regarding short or long sleep duration and/or insufficient sleep, insomnia and insomnia symptoms, general (non-specific sleep disturbances), circadian rhythm abnormalities that result in sleep disturbances, and, briefly, sleep-disordered breathing. Fourth, the review highlights the social/behavioural context of sleep, including discussions of sleep and race/ethnicity, socio-economic position, and other social/environmental factors, in order to place these findings in a social-environmental context relevant to public health. Fifth, the review highlights the issue of sleep as a domain of health behaviour and addresses issues regarding development of healthy sleep interventions. Finally, a research agenda of future directions is proposed.

Introduction

Sleep disorders, such as insomnia, sleep apnoea, and others, have been researched extensively regarding aetiology, pathophysiology, and effects on health and functioning. Still, much work needs to be done to achieve a comprehensive understanding of these conditions. Less attention has focused on unique problems associated with sleep disturbances that may or may not be indicative of a sleep disorder. Many epidemiological studies, for example, assess sleep disturbance broadly. These assessments can be problematic, in that it is often unclear whether these measurements capture disorders, symptoms of disorders, or transient/non-significant complaints. With the relatively recent recognition of the significant public health implications of insufficient sleep duration and/or inadequate sleep quality (sometimes referred to as ‘sleep deficiency’), increased attention

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has focused on sleep as a domain of health behaviour over and above specific disorders. This recognition has come in several forms; for example, healthy sleep has been the focus of two Institute of Medicine reports (Colten et al., 2006; Ulmer et al., 2009), adequate sleep has been included as a federal health and funding priority in Healthy People 2020 (Office of Disease Prevention and Health Promotion, 2011), and the public health impact of 'sleep deficiency' was a major focus of the recent research agenda published by the joint taskforce of the Sleep Research Society and the American Academy of Sleep Medicine (Zee et al., 2014).

Thus, healthy sleep, in a general sense, has been implicated as a major research and public health issue. This is largely because of the large number of studies that have implicated generally unhealthy sleep as a risk factor for many chronic diseases. Some of these relationships will be discussed in this review. The focus of this review is on 'sleep disturbance' which (as described below) can refer to several types of sleep complaints. The review also focuses on 'chronic disease' which has also been variously defined in the literature and is clarified below. The structure of this paper is intended to (1) introduce the concepts of chronic disease and sleep disturbance as they are used in this review, (2) summarize the extant literature linking general sleep disturbances to chronic cardiometabolic disease, (3) discuss the issue of sleep disturbance in a public health context, (4) address the possibility that intervening on sleep disturbance represents an opportunity to prevent chronic cardiometabolic disease, and (5) propose a research agenda for the future.

What is meant by 'cardiometabolic disease?'

Approximately 100 years ago, the leading causes of death in the USA were more likely to be infectious diseases (tuberculosis and pneumonia were both in the top three). Currently, the leading causes of death include heart disease, cancer, chronic lower respiratory diseases, and other diseases that are not infectious (Kung et al., 2008). These conditions typically arise insidiously over years and represent outcomes of complex pathways of illness. Many of these leading causes of death in the USA are likely caused by lifestyle behavioural risk factors (Mokdad et al., 2004). Understanding these risk factors is crucial to understanding how to ameliorate the effects of (and possibly prevent) these diseases. Many of the health conditions that account for the most deaths have related pathways that include obesity, diabetes, and cardiovascular disease. A global term for these related conditions is 'cardiometabolic disease'.

There are other important chronic diseases as well. For example, cancer is a chronic disease that is a major cause of death. It should be noted that there are many studies documenting sleep disturbances in cancer (Sharma et al., 2012), and several studies showing the benefits of treating sleep disorders in the context of cancer (Garland et al., 2011, 2014; Roscoe et al., 2007; Sharma et al., 2012). Also, sleep disturbances have been described in other chronic diseases as well, such as pain syndromes (Besteiro Gonzalez et al., 2011; Landis, 2011; Lindstrom et al., 2012; Perlis et al., 1997; Wagner et al., 2012), dementias (Jausent et al., 2012; Jirong et al., 2013; Zhou et al., 2012), human immunodeficiency virus (HIV) (Gamaldo et al., 2013b; Saberi et al., 2013; Taibi, 2013), and others (Cengic et al., 2012; Chakravorty et al., 2013; Jensen et al., 2013; Parhami et al., 2012; Wallhauser-Franke et al.,

2013). This review focuses primarily on cardiometabolic disease, since the goal is to understand the potential role of sleep disturbance in preventing chronic disease. Unfortunately, there is no available evidence that addressing sleep disturbances may aid in the prevention of pain, cancer or HIV infections, for example. There are studies showing that improving sleep can potentially improve outcomes in these disorders, though (Jungquist et al., 2010; Woodward, 2011). Since cardiometabolic disease can be postponed or prevented using lifestyle behavioural approaches (that would likely include healthy sleep), these are the focus of this review.

What do we mean by ‘sleep disturbances?’

The term ‘sleep disturbance’ is problematic in that it is non-specific. The term may refer to diagnosed or undiagnosed sleep disorders including insomnia and/or sleep apnoea, subclinical sleep continuity disturbances, non-restorative sleep, insufficient sleep, or even abnormally short or long sleep duration in the absence of overt symptoms. (A similar criticism applies to the recently adopted term ‘sleep deficiency’ (Zee et al., 2014).) The major limitation of this term is that because it is non-specific, it is difficult to gain conceptual or methodological clarity regarding its meaning and role in causal pathways. On the other hand, the use of general sleep disturbance can capture sleep problems in a global way. For epidemiological data collections, this has generally been the only way to assess sleep at a population level. Although there is a lack of precision in these assessments, this is balanced by an increase in generalizability and sample size. For example, typical polysomnographic studies have up to dozens of subjects, whereas population-level surveys can study thousands.

In some cases the ‘sleep disturbance’ was measured in terms of a specific symptom (e.g. difficulty initiating sleep at least 15 days/month (Grandner et al., 2013d)) and in some cases it was compounded (e.g. difficulty falling asleep, staying asleep, or sleeping too much, for at least 6 of 14 days (Grandner et al., 2012a)). In some cases, there was a chronicity component (as in the previous examples), and sometimes it only involved a subjective measure of severity. In some cases (particularly smaller studies), previously validated measures were used, whereas in most larger studies, these were simple survey questions. For the purposes of this review, the term ‘sleep disturbances’ is meant broadly, and throughout this review, the specific operationalization of the term will be described.

Cardiometabolic disease risk associated with sleep disturbance

Since sleep disturbances are variously defined, this section will deal separately with short and/or insufficient sleep duration, general (non-specific) sleep disturbance, and insomnia, with mentions of issues related to circadian rhythm disorders and sleep-disordered breathing. Many laboratory studies have demonstrated adverse effects of sleep deprivation that suggest mechanistic links between sleep loss and chronic disease (Grandner et al., 2010a; Knutson, 2010, 2012, 2013). However, these are beyond the scope of the present review. Rather, this review will focus on studies of individuals who experience sleep disturbances in a naturalistic setting, rather than studies of individuals for whom sleep disturbances are artificially and acutely induced.

Short or long sleep duration and insufficient sleep

For approximately 50 years and across approximately 50 studies, both short and long sleep duration have been associated with increased mortality risk (Cappuccio et al., 2010; Gallicchio & Kalesan, 2009; Grandner et al., 2010c). These studies used a variety of methods and designs, studied a variety of different populations, and had varying levels of statistical controls. Taken together, though, it seems that a robust finding linking both short and long sleep duration with mortality emerges. The mechanisms for this relationship are unclear, though several studies have shown that adverse outcomes are associated with both short and long sleep.

Among long sleepers, results have been mixed. This may be due to the fact that long sleepers tend to be individuals who have excessive sleep opportunity and frequently include individuals who are retired, unemployed, or disabled (Grandner & Drummond, 2007; Patel et al., 2012). That said, several studies have shown adverse outcomes associated with long sleep duration, including depression (Patel et al., 2006), obesity (Buxton & Marcelli, 2010), diabetes (Buxton & Marcelli, 2010; Kachi et al., 2012), carotid intima-media thickness (a measure of atherosclerosis risk) (Abe et al., 2011), and presence of cardiovascular disease in general (Buxton & Marcelli, 2010; Cappuccio et al., 2011) and heart attack (Altman et al., 2012; Magee et al., 2009) and stroke (Altman et al., 2012; Kakizaki et al., 2013) in specific.

Among short sleepers, dozens of studies have been conducted showing associations to chronic disease and/or associated risk factors. These have been summarized in several recent reviews (Faraut et al., 2012a; Grandner et al., 2010a, 2010c, 2013e; Knutson, 2010, 2012, 2013; Knutson et al., 2007; Knutson & Van Cauter, 2008; Liu et al., 2012a; Lucassen et al., 2012; Nielsen et al., 2011; Patel, 2009; Patel & Hu, 2008; Solarz et al., 2012; Wang et al., 2012; Zimberg et al., 2012). Briefly, short sleep duration has been associated with obesity-related outcomes, diabetes-related outcomes, and cardiovascular-related outcomes. Regarding obesity-related outcomes, short sleep duration has been associated with incident weight gain (Kobayashi et al., 2012; Nagai et al., 2013), incident obesity (Watanabe et al., 2010), current body mass index (Patel, 2009), and current adiposity (Chaput et al., 2007; Liu et al., 2012b; Moraes et al., 2013; Taveras et al., 2011; Yu et al., 2007). Regarding diabetes-related outcomes, short sleep duration has been associated with diabetes (Buxton & Marcelli, 2010; Grandner et al., 2014a; Hsieh et al., 2011; Kachi et al., 2012), insulin and/or glucose dysregulation (Beihl et al., 2009; Byberg et al., 2012; Chang et al., 2012b; Chaput et al., 2009; Javaheri et al., 2011; Liu et al., 2011; Lucassen et al., 2012; Zuo et al., 2012), and dysregulation of metabolic hormones (Chaput et al., 2007; Gangwisch, 2009; Hayes et al., 2011; Martinez-Gomez et al., 2011). Regarding cardiovascular-related outcomes, short sleep duration has been associated with incident hypertension (Cappuccio et al., 2011; Meng et al., 2013), current hypertension (Altman et al., 2012; Fang et al., 2012; Faraut et al., 2012b; Grandner et al., 2014a; Wang et al., 2012), dyslipidemia (Altman et al., 2012; Gangwisch et al., 2010a; Grandner et al., 2014a), atherosclerosis risk factors (King et al., 2008; Nakazaki et al., 2012; Sands et al., 2012), inflammation (Grandner et al., 2013e), and cardiovascular disease in general (Buxton & Marcelli, 2010), as well as history of heart attack (Altman et al., 2012) and stroke (Altman et al., 2012; Cappuccio et al., 2011; Eguchi et al., 2010; Hamazaki et al., 2011). More information about these and other associations can be found in

recent reviews (Adenekan et al., 2013; Cappuccio et al., 2008, 2010, 2011; Cappuccio & Miller, 2011; Faraut et al., 2012a; Gallicchio & Kalesan, 2009; Gangwisch, 2009; Grandner et al., 2010a, 2010c, 2013e; Knutson, 2010, 2012, 2013; Knutson et al., 2007; Knutson & Van Cauter, 2008; Patel, 2009; Patel & Hu, 2008; Salarz et al., 2012; Van Cauter & Knutson, 2008).

One of the reasons that short sleep duration may be considered a form of sleep disturbance despite the fact that many people engage in this behaviour volitionally is because short sleep duration may confer risk for, or serve as a proxy for, sleep disturbances. For example, short sleep duration overlaps significantly with daytime dysfunction due to sleepiness, tiredness, and/or fatigue (Grandner & Kripke, 2004). In addition, individuals who report short sleep duration are also more likely to report difficulty initiating sleep, difficulty maintaining sleep, early morning awakenings, and non-restorative sleep (Grandner & Kripke, 2004).

If this is the case, especially if to the degree to which short sleep duration confers risk for sleepiness and/or fatigue, short sleep duration may be a proxy for insufficient sleep. Insufficient sleep has been variously defined in the literature and has rarely been explored as a construct separate from short sleep duration (which may include individuals with naturally reduced sleep need)(Altman et al., 2012). However, several studies have looked at self-reported insufficient sleep as a health risk factor. For example, Shankar and colleagues found a negative association between unmet sleep need and cardiometabolic health (2010). Earlier studies also found a significant positive correlation between perceived sleep insufficiency and reported poor general health, functional deficits and increased cortisol secretion and abnormal growth hormone metabolism (Copinschi, 2005).

Although the concept of insufficient sleep may overlap with short sleep duration, few studies have attempted to clarify these constructs. In an attempt at this, Altman and colleagues (2012) used data from the 2009 Behavioral Risk Factor Surveillance System (BRFSS) to compare sleep duration and sleep insufficiency in the prediction of BMI, obesity, diabetes, hypertension, hypercholesterolaemia, diabetes, heart attack, and stroke. In this study, both short sleep and insufficient sleep were related to outcomes. After adjusting for 18 sociodemographic and health covariates, both short sleep duration and insufficient sleep were associated with elevated BMI, obesity, hypertension and hypercholesterolaemia; only short and long sleep duration were associated with heart attack and stroke. When sleep duration and insufficiency were combined in the same model, most of the effects of insufficiency were subsumed by sleep duration, except for hypertension (which demonstrated unique effects for both variables) and hypercholesterolaemia (which was no longer significantly explained by sleep duration). This demonstrates that for obesity, much of the relationship with insufficiency is due to sleep duration, but this is not the case for high blood pressure and cholesterol. Further research is needed to better clarify the independent and/or interdependent roles of insufficiency versus duration.

Mechanisms of this relationship are not fully known. Several reviews have attempted to schematically represent potential pathways linking short sleep and cardiometabolic disease (Knutson et al., 2007; Patel, 2009). In general, these schematics have several potential pathways in common. One of the most commonly discussed pathways includes the

metabolic hormones leptin and ghrelin. Leptin is a hormone secreted by fat cells that signals satiety via the hypothalamus. Laboratory sleep deprivation studies have shown that sleep deprivation is associated with decreased leptin levels, suggesting decreased satiety (Spiegel et al., 2004a, 2004b). Ghrelin is a hormone secreted by the stomach that stimulates hunger. Studies of sleep deprivation in the laboratory show increased ghrelin associated with sleep deprivation (Copinschi, 2005; Knutson & Van Cauter, 2008; Spiegel et al., 2004b; Van Cauter et al., 2008). This decrease in leptin and increase in ghrelin may lead to an orexigenic eating pattern that may predispose to weight gain and cardiometabolic disease, and is reflected in some of the changes in energy balance (Markwald et al., 2013; St-Onge et al., 2011) and hunger ratings (Spiegel et al., 2004b) observed in laboratory studies. Another potential pathway is via alterations in insulin and glucose functioning. Sleep deprivation studies have shown insulin and glucose dysregulation to be associated with sleep deprivation (Broussard et al., 2012; Buxton et al., 2010; Darukhanavala et al., 2011; Knutson & Van Cauter, 2008; Padilha et al., 2011; Reynolds et al., 2012). As insulin also plays an important role in leptin and other metabolic hormones, this pathway may intersect with the previous one as well. A third potential pathway involves the role of sleep deprivation as a proinflammatory state. Many studies have shown that sleep deprivation is associated with increased levels of inflammatory cytokines and other markers (for review see Grandner et al., 2013e). Inflammation is itself an important risk factor for cardiometabolic disease, as it is related to endothelial function, oxidative stress, and chronic stress. Interestingly, certain inflammatory pathways known to be associated with sleep deprivation also play a role in insulin regulation. A fourth potential pathway involves neurocognitive processing. Sleep deprivation has been frequently shown to impair cognitive function (Banks & Dinges, 2007; Durmer & Dinges, 2005; Lal et al., 2012). In particular, sleep deprivation impairs decision-making (Killgore, 2010; Killgore et al., 2007, 2008a, 2008b, 2009a, 2009b, 2012). In addition to the humoral pathways described above, sleep deprivation may lead to an impaired ability to make healthy food choices (St-Onge et al., 2012). This, coupled with the propensity of sleep-deprived individuals to indulge in late-night snacks (Markwald et al., 2013; Nedeltcheva et al., 2009; Weiss et al., 2010), may represent another pathway. Other mechanistic links have been suggested, such as the role of protein kinase B (Broussard et al., 2012), adiponectin (Simpson et al., 2010), and others.

Insomnia and insomnia symptoms

In the epidemiologic literature, insomnia is rarely assessed using gold-standard measures. Usually, insomnia symptoms are asked individually (e.g. Do you have difficulty falling asleep at the beginning of the night?), usually with Likert scales. Since these assessments are not standardized, they should be interpreted with some level of caution. Still, these studies allow for assessments of relationships with initial insomnia (difficulty initiating sleep), middle insomnia (difficulty maintaining sleep), and late insomnia (early morning awakenings), since these symptoms are often asked separately.

There is an emerging literature documenting adverse cardiometabolic risk factors associated with insomnia. Several studies have shown that insomnia symptoms are associated with hypertension in the population duration (Gangwisch et al., 2006; Gangwisch et al., 2010b; Knutson et al., 2009; Phillips & Mannino, 2007; Suka et al., 2003), especially when the

insomnia is accompanied by short sleep duration (Fernandez-Mendoza et al., 2012b; Vgontzas et al., 2009a). It is currently unclear whether insomnia itself poses a risk, or whether insomnia with short sleep duration is required.

Meng and colleagues (2013) reviewed studies of insomnia and incident hypertension in a meta-analysis, showing that overall insomnia is associated with a small but significant (5%) increased risk of incident hypertension. Separately, although initial insomnia was not significantly associated with incident hypertension, middle insomnia was associated with a 20% increased risk and late insomnia was associated with a 14% increased risk. In addition to hypertension, several studies have shown insomnia to be associated with inflammation (Jain et al., 2012; Laugsand et al., 2012; Motivala, 2011; Okun et al., 2011), impaired heart rate variability (De Zambotti et al., 2011; Israel et al., 2012; Spiegelhalder et al., 2011; Stein & Pu, 2012), and other risk factors (Balbo et al., 2010; Lattova et al., 2011; Laugsand et al., 2013; Strand et al., 2013).

In a recent study, Sands-Lincoln and colleagues evaluated the relationship of insomnia to coronary heart disease and cardiovascular events among > 85,000 women enrolled in the Women's Health Initiative (Sands-Lincoln et al., 2013b). They found that in fully adjusted models, insomnia symptoms were associated with a 19% increased risk of coronary heart disease and an 11% increased risk of cardiovascular events.

In addition to these studies, there is a literature that has suggested that the cardiometabolic risks associated with insomnia are particularly evident in the subset of sufferers that also experience short sleep duration (Vgontzas et al., 2013). In this way, there is an interaction between sleep duration and insomnia such that those with insomnia and short sleep duration demonstrate greater risks than those with either condition alone. Several studies have explored this phenomenon. For example, individuals with both short sleep and insomnia have been shown to be at increased risk for hypertension (Fernandez-Mendoza et al., 2012b; Vgontzas et al., 2009a), diabetes (Vgontzas et al., 2009b), depression (Fernandez-Mendoza et al., 2012a), and mortality (Vgontzas et al., 2010), relative to short sleepers without insomnia and individuals with insomnia without short sleep. These results may be moderated by unknown factors. For example, Sands-Lincoln and colleagues found that among postmenopausal women in a national sample, although there was a sleep duration by insomnia interaction in the prediction of coronary heart disease, they found that it was insomnia coupled with long sleep that was associated with the highest risk (Sands-Lincoln et al., 2013b).

General sleep disturbance (non-specific)

Although the use of individual insomnia symptoms presents methodological and interpretive challenges due to non-standardized measures, these challenges become even greater with assessments of general sleep quality. That said, there are several studies which only had access to general sleep quality data for analyses. Grandner and colleagues found that sleep disturbance, assessed as difficulty falling asleep, staying asleep, or sleeping too much in at least 7 of the past 14 days (assessed using the 2006 BRFSS), was associated with a 35% increased risk of obesity, a 54% increased risk of diabetes, an 80% increased risk of heart attack, a 102% increased risk of stroke, and a 98% increased risk of any coronary artery

disease (Grandner et al., 2012a). All of these relationships remained significant (though attenuated) after adjustment for demographics, socio-economics, mental health, healthcare access, smoking, alcohol use and BMI (for outcomes other than obesity). When overall physical health was entered into the model, the relationships with diabetes and stroke were no longer significant. Still, this study showed that poor sleep quality in general is an important health risk factor. A separate analysis of the 2006 BRFSS data asked a different question. Rather than using sleep disturbance to predict outcomes of interest, is it the case that those with obesity and diabetes sleep poorly overall, and is this mediated by access to exercise? In this separate study (Grandner et al., 2011), those with obesity and diabetes were more likely to report both night-time sleep disturbance and daytime tiredness. When exercise (assessed as any exercise in the past 30 days) was entered into the model, the relationships did not change (no mediation). Presumably, the amount of exercise measured by this variable includes small amounts of activity, which likely do not impact obesity or diabetes. However, simply reporting any activity was associated with a decreased likelihood of reporting sleep disturbance (around 30% decrease) and daytime tiredness (around 50% decrease). This suggests that general sleep disturbance is strongly associated with other general lifestyle health factors, such as physical activity.

Other studies have also assessed links between sleep disturbance and cardiometabolic disease risk. For example, Troxel and colleagues found that sleep complaints were associated with greater risk of metabolic syndrome (Troxel et al., 2010a). Also, Sands-Lincoln and colleagues found that self-reported non-restorative sleep was associated with hypertension in a nationally representative sample of US adults (Sands-Lincoln et al., 2013a).

Circadian rhythm disturbances

Circadian rhythm disturbances frequently interfere with sleep. Circadian rhythms refer to the ~ 24-h rhythms in the body responsible for regulation of a large host of physiological processes. When these rhythms are disturbed, sleep is also frequently disturbed as well. For example, advanced sleep phase refers to the condition where an individual's natural bed and rise times are much earlier than they prefer; similarly, delayed sleep phase refers to natural bed and rise times that are later than preferred. There is evidence that preferred sleep phase is, to some degree, driven by development (Sadeh et al., 2009; Tarokh et al., 2012) and genetic predisposition (Kerman et al., 2012; Lamont et al., 2007; Saini et al., 2011), but it can also be entrained by behaviours and exposure to light at inappropriate times (Chang et al., 2012a; Corbett et al., 2012; Kavcic et al., 2011; Sharkey et al., 2011). Another example of a circadian disturbance occurs with shift work. In this case, individuals are tasked with maintaining wakefulness and performance during hours for which this is not biologically optimal. This leads to several situations, including consequences of repeatedly maintaining wakefulness when biologically predisposed to sleep, consequences of the mismatch between internal and external rhythms, and consequences of the inability for the internal rhythm to adjust, especially in the case of rotating schedules or weekends off (Bannai & Tamakoshi, 2014; Cheng et al., 2014; Darwent et al., 2012; Guyette et al., 2013; Lin et al., 2012; Machi et al., 2012; Roenneberg et al., 2013; Ruggiero et al., 2012; Walia et al., 2012).

Most studies of circadian rhythm disturbances (including circadian rhythm sleep disorders) tend to focus on either basic physiological processes or, when they examine outcomes, these outcomes tend to be in the psychological domain. For example, several studies have shown that advanced and delayed sleep phase are both associated with insomnia (Kohyama, 2011; Martinez & Lenz Mdo, 2010; Reid & Zee, 2009), affective disorders (Kripke et al., 2008; Mendlewicz, 2009; Okawa, 2011; Robillard et al., 2013; Saxvig et al., 2012) and functional impairments (Bijlenga et al., 2013; Dowling & Mastick, 2010; Hida et al., 2012; Miller et al., 2012; Saxvig et al., 2012; Zhou et al., 2011).

Studies of shift work, on the other hand, have focused attention on health consequences of shift work. This is relevant to the current discussion since shift work is very frequently associated with abbreviated or otherwise disturbed sleep (Ferguson et al., 2012; Lin et al., 2012; Machi et al., 2012; Mahan et al., 1990; Roach et al., 2012). For example, Cheng and colleagues found that men working longer than 60 h/week were at approximately double the risk for coronary heart disease (Cheng et al., 2014). Notably, short sleep duration in this group was found to be a significant moderator of the effect, with those sleeping < 6 h on average being at the highest risk. This is consistent with a recent review by Bannai and Tamakoshi, which found that long working hours is detrimental to health and functioning (Bannai & Tamakoshi, 2014). In addition to the longer hours usually associated with shift work, a recent review by Szosland summarizes data that implicates shiftwork in diabetes, ischaemic heart disease, and metabolic syndrome (Szosland, 2010). Successful acclimation to the night work may alleviate many of these risks. For example, Boudreau and colleagues found that circadian adaptation to night work resulted in improved sleep, improved performance, and improved heart rate variability (Boudreau et al., 2013).

Sleep-disordered breathing

Many reviews have been written on the cardiometabolic risk factors associated with sleep-disordered breathing and sleep apnoea (Butt et al., 2010; Fava et al., 2011; Golbidi et al., 2012; Kent et al., 2011; Loke et al., 2012; Lurie, 2011; Monahan & Redline, 2011; Morselli et al., 2012; Pak et al., 2014; Phillips et al., 2013; Primhak & Kingshott, 2012). Thus, this issue is largely outside the scope of this paper. But it should be noted that sleep apnoea often presents as daytime sleepiness or non-restorative sleep (Alam et al., 2012; Cruz et al., 2012; Lal et al., 2012; Lee et al., 2012; Sunwoo et al., 2012) and sometimes as insomnia (Al-Jawder & Bahammam, 2012; Glidewell et al., 2012). Sleep apnoea is associated with significant sleep fragmentation (Bianchi et al., 2012) which may be one of the causal pathways linking this disorder to poor sleep, though the risks are probably largely due to the intermittent hypoxia and co-morbid obesity (Pak et al., 2014).

In summary, sleep disturbances, which could be defined as short or long sleep duration, perceived insufficient sleep, inadequate sleep quality, insomnia, circadian rhythm disturbance, and/or sleep-disordered breathing, are related to cardiometabolic disease risk factors. These relationships depend on the sleep variable studied. For example, short sleep duration has been associated with many adverse in mostly cross-sectional (but some longitudinal) designs. Long sleep duration and perceived insufficient sleep, however, have not been studied as thoroughly. Insomnia and its symptoms are strongly related to mental

health outcomes, but cardiometabolic relationships have just recently been explored. It is possible that much of the relationship between insomnia and cardiometabolic disease involves an interaction with short sleep duration. Circadian rhythm problems can also predispose individuals to adverse outcomes, especially when accompanied by long work hours. Sleep-disordered breathing, especially (untreated) obstructive sleep apnoea, is a well-known cardiometabolic risk factor, associated with cardiac and vascular outcomes, diabetes, and mortality.

Given that these sleep disturbances are prevalent and are associated with important clinical outcomes, it is important to consider the broader public health implications. If the above outcomes represent the downstream effects, attention must also be paid to the upstream determinants of sleep in the population. Understanding the contextual factors that give rise to sleep disturbances may better aid in the identification of high-risk groups, the understanding of mechanisms leading to adverse health outcomes, and the conceptualization of effective interventions. The next section addresses issues regarding the social and behavioural context.

The social and behavioural context of sleep disturbance

The issue of chronic disease management is a major public health concern. The burden of chronic health conditions such as obesity, diabetes, and hypertension are disproportionately borne by racial/ethnic minorities and those of lower socio-economic position (Centers for Disease Control and Prevention, 2011). This may be an important reason why life expectancy is lower among many racial/ethnic minority groups compared to white groups (Levine et al., 2001). Understanding the nature of these disparities is an important step in developing strategies for ameliorating them. In the context of sleep and health, it is important to understand the degree to which the burden of poor sleep is experienced among racial/ethnic minorities and those who are socio-economically disadvantaged. In addition, it is important to consider other behavioural and social factors that may explain some of this increased burden and point to potential intervention components.

Sleep and race/ethnicity

Several studies have shown that racial/ethnic minorities are more likely to be short sleepers. For example, Stamatakis and colleagues found that African-American and 'other' race/ethnicities (but not Hispanic) were consistently associated with short sleep duration (Stamatakis et al., 2007). Also, data from Hale and Do showed that black/African-Americans were also more likely to be long sleepers (Hale & Do, 2006).

A recent study by Whinnery and colleagues represents the most comprehensive analysis of sleep duration among racial/ethnic minorities in the US (Whinnery et al., 2014). This study used data from the 2007–2008 National Health and Nutrition Examination Survey (NHANES). In adjusted analyses, black/African-Americans, non-Mexican Hispanics/Latinos, and Asians/others were more likely to report very short sleep (< 5 h), black/African-Americans and Asians/others were more likely to report short sleep (5–6 h), Mexican-Americans were less likely to report long sleep (9 + h), those born in Mexico were less likely to report short sleep, and speaking Spanish only at home was associated with

lower likelihood of reporting very short sleep. Other studies have found other patterns. For example, Hale and Do found that in addition to short sleep, black/African-Americans were more likely to report longer sleep as well (Hale & Do, 2007). The effect of the environment may also be a major explanatory factor. For example, Gamaldo and colleagues studied middle-aged urban adults and found no significant differences in the likelihood of short or long sleep between non-Hispanic white adults and black/African-Americans (Gamaldo et al., 2013a). Interestingly, within groups, different factors predicted shorter sleep, with stress and education playing a larger role in the non-Hispanic white group, and inflammation playing a larger role in the black/African-American group.

To assess patterns in sleep disturbance, Grandner and colleagues used the BRFSS 2006 sleep disturbance item (described above)(Grandner et al., 2010b). Among men, no significant differences were seen between non-Hispanic white groups and black/ African-Americans, Hispanics/Latinos, or those reporting more than one race/ethnicity, and a significant difference was seen among Asians/others, who reported fewer sleep disturbances. Among women, non-Hispanic white women reported significantly more sleep disturbance than black/African-Americans, Hispanics/Latinos, and Asians/others, and less sleep disturbance than those reporting more than one race/ethnicity. This finding was surprising – despite increased likelihood of short sleep duration and associated outcomes, racial/ethnic minorities tended to report fewer sleep disturbances.

A subsequent study by Grandner and colleagues examined the 2007–2008 NHANES data (Grandner et al., 2013d). In this case, rather than sleep disturbance, specific variables were assessed: sleep latency > 30 min, difficulty initiating sleep, difficulty maintaining sleep, early morning awakenings, non-restorative sleep, daytime sleepiness, choking/gasping during sleep (suggesting sleep apnoea), and snoring. In models that included age, sex, race/ethnicity, marital status, immigrant status, income, education, access to health insurance, and household food security, the following results were found. Black/African-Americans were 58% more likely to report sleep latency > 30 min, compared to non-Hispanic white adults, and no other differences were seen for this variable. Contrastingly, self-reported difficulty falling asleep was reported less frequently among black/ African-Americans (odds ratio (OR) = 0.57), Mexican-Americans (OR = 0.59), and other Hispanics/ Latinos (OR = 0.73). Similarly, difficulty maintaining sleep, early morning awakenings, non-restorative sleep, and daytime sleepiness were all reported less frequently among black/African-Americans and Mexican-Americans, and both non-restorative and sleep daytime sleepiness were also reported less frequently among other Hispanics/Latinos. The non-Mexican Hispanics/ Latinos were more likely to report choking/gasping during sleep and snoring, however.

These findings mirror those of other studies, which inconsistently describe sleep disturbances among racial/ethnic minorities. For example, some studies have found overall sleep quality to be worse among minorities (Boss et al., 2011; Kripke et al., 2004; Moore et al., 2011; Patel et al., 2010b), whereas others have failed to find this association (Gellis et al., 2005; Grandner et al., 2010b; Hale & Rivero-Fuentes, 2011). Several reasons for this are possible. First, it is possible that differences in measurement introduce error across studies. Second, there may be fundamental differences among cohorts. Third, effects of geographical location and other environmental variables may moderate these relationships (Grandner et

al., 2012b). Fourth, there may be differences in levels of bias in reporting. For example, questions asked in a non-judgemental way (e.g. sleep duration, sleep latency) may lead to different reporting patterns than questions asked in a way that asks for an explicit complaint (e.g. difficulty initiating or maintaining sleep). Another important issue is a potential interaction between race/ethnicity and poverty. Patel et al. (2010) and colleagues found that, relative to the non-poor non-Hispanic white group, poor individuals of all races/ethnicities (including the non-Hispanic white group) had worse sleep overall. The only non-poor group to differ from the non-poor white group was non-poor black/African-Americans, who had worse sleep overall, but to a lesser degree than their poor counterparts.

Sleep and socio-economic position

Socio-economic position is frequently assessed using variables such as education level, household income, and occupational status. Previous studies that have examined associations between sleep quality and socio-economic factors have tended to report increased sleep disturbance (represented as general overall sleep disturbance or specific symptoms such as difficulty falling asleep, staying asleep or waking too early) among individuals of lower socio-economic position (Friedman et al., 2005; Gellis et al., 2005; Geroldi et al., 1996; Grandner et al., 2010b, 2013c; Hall et al., 2009; Nomura et al., 2010; Patel et al., 2010a). In addition, several studies have shown that those of lower socio-economic position are more likely to be short sleepers (Stamatakis et al., 2007; Whinnery et al., 2014). Although few studies have measured this relationship using sleep assessed with polysomnography, these have also generally found that lower socio-economic position is associated with poorer sleep quality (Friedman et al., 2005; Hall et al., 2009; Mezick et al., 2008; Tomfohr et al., 2010).

In the aforementioned study of general sleep disturbance measured using the BRFSS 2006 (Grandner et al., 2010b), increased sleep disturbance was more likely amongst those who did not graduate college, were of lower income (especially < US\$25,000 among men), and were unemployed. In the study by Whinnery and colleagues (Whinnery et al., 2014), in fully adjusted models, very short sleep was more likely to be reported among those earning < \$20,000 and \$65,000–75,000 (relative to > \$75,000), those who did not complete college, those with public insurance only (relative to no health insurance at all), and those with marginal or very low food security. Short sleep was more likely to be reported among those who attended high school but did not graduate from college, and those with very low food security. Long sleep was more frequently reported among those earning \$20,000–25,000 per year and those with public insurance only. In the complementary study by Grandner and colleagues (Grandner et al., 2013d), sleep latency > 30 min was associated with less than a college education, no access to private health insurance, and marginal, low, or very low food security. Insomnia symptoms (difficulty falling asleep, difficulty maintaining sleep and early morning awakenings) were only associated with food security, and not other factors. Non-restorative sleep, sleepiness, snorting/gasping and snoring were all associated with lower education levels and lower food security. Interestingly, relationships to income (seen in unadjusted analyses) were rendered non-significant after adjustment. For income, it seems that the once the benefits of increased income are realized (e.g. access to employment, education, food and healthcare, little unique variance is explained by income alone.

Sleep and other social/environmental factors

In addition to (and perhaps interacting with) race/ ethnicity and socio-economic position, a whole host of contextual factors play a role in sleep disturbance. For example, recent studies have shown that geography explains sleep disturbance (Grandner et al., 2012b) and insufficient sleep (Grandner et al., 2013f) in the US. Preliminary analyses suggest that a whole host of factors may be relevant to these geographical differences, including differences in racial make-up of the population, temperature and weather patterns, regional health issues, regional healthcare access, and others (Grandner et al., 2012b). On a more granular level, there is a growing body of literature showing that neighbourhood factors play a role in sleep quality. Not only have studies shown that neighbourhoods that are perceived to be dirty, dangerous, and disconnected are also home to individuals more likely to report poor sleep quality (Hale & Do, 2006; Hale et al., 2010, 2013; Hill et al., 2009), but this increased poor sleep quality may partially mediate the detrimental effect that these neighbourhoods have on health (Hale et al., 2010).

In addition to geographic factors, there may be cultural beliefs and attitudes about sleep that may play a role. For example, Sell and colleagues found that Mexican-Americans who were not acculturated to American norms were less likely to be able to demonstrate basic knowledge about sleep disorders and what the symptoms of sleep disorders look like (Sell et al., 2009). In addition, a study in the Philadelphia area compared beliefs and attitudes about sleep matched for income level among older women matched for income (Grandner et al., 2013b). This study found that black/African-American women were less likely to report beliefs and attitudes consistent with healthy sleep habits. These data are consistent with other studies that show that black individuals who are at higher risk of obstructive sleep apnoea demonstrate more dysfunctional beliefs and attitudes about sleep (Pandey et al., 2011). Some cultural differences in sleep-related beliefs and attitudes may be reflected early. For example, Sadeh and colleagues (Sadeh et al., 2011) found that the definition of a child's 'sleep problem' differed between countries relative to primary race/ethnicity. In primarily white countries, a 'sleep problem' was largely determined by length of sleep episode, nocturnal wakefulness, number of night wakings and total sleep time, whereas in primarily Asian countries, children's 'sleep problems' were more likely to be determined by socio-demographic and/or socio-economic factors, such as the child's age, parents' age, and parental education level.

In summary, poor sleep is a health concern that may be disproportionately felt by minorities and/or individuals of lower socio-economic position. As Simon Williams writes, 'When we sleep, where we sleep, and with whom we sleep are all important markers or indicators of social status, privilege, and prevailing power relations' (Williams, 2005). The next section considers the upstream determinants of sleep disturbance and the downstream cardio-metabolic (and other) adverse effects of sleep disturbance to consider a theoretical framework for considering the role of sleep in public health and the development and implementation of public health interventions.

Conceptualizing sleep disturbance in the context of public health

Although most, if not all, sleep disorders have targeted interventions (e.g. PAP therapies for sleep apnoea (Avlonitou et al., 2012; Bertisch & Patel, 2012; Campbell et al., 2012; Ciccone et al., 2012; Filtiness et al., 2012; Hegglin et al., 2012; Zhao et al., 2012), hypnotics or cognitive behavioural therapy for insomnia (CBTI) (Doghramji, 2010; Ebben & Narizhnaya, 2012; Gehrman & Gooneratne, 2010; Hall-Porter et al., 2010; Jarnefelt et al., 2012; Krystal, 2009; Morin & Willett, 2009; Pinto et al., 2010; Saddichha, 2010; Siebern & Manber, 2010; Siebern et al., 2012; Sullivan, 2010; Taylor et al., 2010), dopamine agonism for RLS and PLMD (Aurora et al., 2012; Benes et al., 2009; Earley et al., 2011; Earley & Silber, 2010; Garcia-Borreguero & Williams, 2010; Khalid et al., 2009; Natarajan, 2010; Quilici et al., 2008; Trenkwalder & Paulus, 2010; Wetter, 2010)), no intervention has been developed or evaluated for insufficient sleep or other aspects of general sleep disturbance. One potential reason is that the data needed to frame this form of sleep as important and relevant has only been recently aggregated in a way that makes the case compelling.

Sleep as a domain of health behaviour

One way to conceptualize insufficient sleep and other aspects of general sleep disturbance is as a disorder. This perspective is one that has been adopted by the sleep medicine community, given the inclusion of insufficient sleep syndrome in the International Classification of Sleep Disorders (American Academy of Sleep Medicine et al., 2006).

Another way to conceptualize insufficient sleep is as a domain of health behaviour.

Insufficient sleep shares many characteristics with other behaviours, such as smoking, poor diet, and lack of physical activity, in that the reasons for insufficient sleep involve beliefs and attitudes (Carney & Edinger, 2006; Grandner et al., 2013b, 2014b; Smith et al., 2004; Yang et al., 2011), home and work demands (Basner & Dinges, 2009; Basner et al., 2007; Grandner et al., 2014b), and environmental constraints (Barclay et al., 2012; Buxton et al., 2012; Grandner et al., 2014b; McCall et al., 2012; Roenneberg et al., 2013; Verhaert et al., 2011; Yoder et al., 2012).

With this in mind, a social-ecological model of sleep and health was recently proposed by our group (Grandner et al., 2010c), based on the social-ecological theory of Bronfenbrenner (1977). This model is summarized in Figure 1 and suggests that sleep disturbances (and resultant adverse outcomes) depend on individual-level factors (e.g. behaviour, physiology), which are embedded within social factors (e.g. neighbourhood, work), which are themselves embedded within societal factors (e.g. 24/7 society).

This model brings together the upstream social/ behavioural determinants of sleep and the downstream adverse consequences of sleep disturbance. According to the model, the individual-level factors (which would include that individuals' specific demographics, genetics, physiology, health, knowledge, beliefs, attitudes, and choices) directly impact on that individual's sleep. However, the model goes a step further and recognizes that all of these individual factors are embedded within a social framework that includes people that exist separate from the individual but directly or indirectly impact the individual-level factors. For example, a noisy or crime-ridden neighbourhood, a culture that engenders certain beliefs/attitudes, a racial or ethnic group that is subject to certain social stresses, a

work schedule that impinges on sleep time, family obligations that may begin early in the morning, or a social network that encourages unhealthy attitudes, all play a role in an individual's sleep. The model even goes further to recognize that even these social-level factors exist in the context of larger, societal-level factors such as globalization, technology, 24/7 society, and others, which play a role in shaping social and eventually individual attitudes, beliefs, and practices regarding sleep.

This model suggests that all of these levels are relevant to understanding sleep. For example, recognizing the social-level factors aids in the understanding of the effects of neighbourhoods on sleep and sleep disorders (Brouillette et al., 2011; Hale & Do, 2006; Hale et al., 2010, 2013; Hill et al., 2009; Omlin et al., 2011; Platt et al., 2009), geographic patterns of sleep (Grandner et al., 2012b; McKnight-Eily et al., 2009), sleep and the physical environment (e.g. green space, weather, and traffic and other noise) (Astell-Burt et al., 2013; Buxton et al., 2012; Elmenhorst et al., 2012; Pandey et al., 2005; Pirrera et al., 2010), family and relationship factors (Ailshire & Burgard, 2012; Lallukka et al., 2013; Rhoades et al., 2012; Rogojanski et al., 2013; Troxel, 2010; Troxel et al., 2010b, 2010c; von Kaanel et al., 2012), and even the effects of a changing technological landscape (Brunborg et al., 2011; Crowder et al., 2012; Garmy et al., 2012; Lowden et al., 2011; Munezawa et al., 2011; Thomee et al., 2011, 2012) suggest that individual-level factors need to be viewed in the context of these social-level and societal-level factors. For example, the study by Gamaldo and colleagues mentioned above found no differences in sleep duration by race/ethnicity when the individuals were otherwise relatively homogeneous in terms of age and living environment (Gamaldo et al., 2013a).

This model also has implications for the development of interventions to address sleep and health. To change sleep-related behaviours, individual-level factors that affect sleep need to be taken into account, including knowledge, beliefs, attitudes, and practices. But social-level factors also need to be considered, such as work/school obligations, home/family obligations, and the social and environmental context. For example, community-based approaches may work better for some versus other groups. Thus, a behavioural intervention for insufficient sleep needs to address sleep as an aspect of health behaviour, cast in a larger context.

Developing a sleep health intervention

To develop an intervention it would be important to consider models of health behaviour change. These models are necessary because unhealthy behaviours (such as poor diet, sedentary lifestyle, smoking) are complex and resistant to change. Examples include the health belief model, the transtheoretical model, and behavioural economics. Briefly, the health belief model (Champion & Skinner, 2008; Rosenstock, 1966) states that individuals will alter their behaviour based on (1) perceived risk of maintaining existing behaviour, (2) perceived benefits of the new behaviour, (3) perceived barriers in engaging in the new behaviour, (4) perceived ability to carry out the behaviour, and (5) cues to action (reminders). The transtheoretical model (Prochaska & DiClemente, 1982; Prochaska et al., 2008, 2009), briefly, posits that behaviour change occurs on a dimension of readiness to change, which includes precontemplation (not yet thought about change), contemplation

(thought but no decision to act), preparation (decision but no action), action, and maintenance. The model dictates that interventions based on action should only be implemented by those who are ready to engage in that action. Behavioural economics theory (Hursh, 1984), briefly, deals with the ways that complex health-promoting and health-compromising behaviours are/may be supported by complementary reinforcers (e.g. alcohol reinforces smoking behaviour) and weakened by alternative reinforcers (e.g. nicotine patch delivers nicotine without smoking). Since many health-compromising behaviours have benefits that may vary across individuals, financial incentives may act as a universal, simple, adaptable, alternative reinforcer (Halpern et al., 2011; John et al., 2011, 2012; Kim et al., 2012; Kimmel et al., 2012; Kullgren et al., 2013; Loewenstein et al., 2013; Shea et al., 2012; Volpp et al., 2008, 2011). As such, several studies have documented the benefits of financial incentives to change relatively intractable health-compromising behaviours (Cahill & Perera, 2011; Halpern et al., 2011; John et al., 2011; Kim et al., 2012; Kimmel et al., 2012; Kullgren et al., 2013; Volpp et al., 2008). It should also be noted that financial incentives may backfire in that they may serve to undermine an individual's intrinsic motivation to improve their health (Hagger et al., 2014; Ma et al., 2014; Moller et al., 2012, 2013; Promberger & Marteau, 2013).

Does addressing sleep disturbance impact chronic disease?

There are no studies of interventions for general sleep disturbance, so it is difficult to say whether such an intervention would have a long-term impact on chronic disease. Despite this, if sleep shares characteristics with other health behaviours, it is reasonable to suspect that a sleep intervention could have lasting impact. After all, reduced cardiometabolic risk has been produced with interventions that successfully improve diet and exercise (Stephens et al., 2014) and reduce smoking (Rigotti & Clair, 2013). Regarding insomnia, although there are several studies showing associations with cardiometabolic disease, there are still no studies investigating whether insomnia treatment improves cardiometabolic outcomes. Regarding sleep apnoea, there are several studies that have shown improvements in cardiovascular outcomes (Gottlieb et al., 2013). For example, in studies by Martinez-Garcia and colleagues, treatment of sleep apnoea was associated with significantly reduced cardiovascular mortality (Campos-Rodriguez et al., 2012; Martinez-Garcia et al., 2009).

Future directions

Eventually, the goal is that there will be a more complete understanding of the role of sleep in health, and successful interventions will be ready for implementation. This is an important goal and was expressed by reports from the Institute of Medicine (Colten et al., 2006), the US Department of Health and Human Services (Office of Disease Prevention and Health Promotion, 2011) and the Joint Task Force of the American Academy of Sleep Medicine and Sleep Research Society (Zee et al., 2014). To achieve this goal, much work is needed.

First, the concept of 'sleep disturbance' (and the similar concept of 'sleep deficiency') needs to be unpacked. What is it about poor sleep that confers risk? What are the additive or interactive contributions of difficulty initiating sleep, difficulty maintaining sleep, decreased sleep ability, sleep fragmentation, insufficient sleep, excessive sleep, and so on? For example, epidemiological studies should move away from non-standard survey item

measures of sleep and begin to adopt standardized, validated items or screening questionnaires. Also, retrospective, subjective measures of sleep need to be accompanied by prospective measures (e.g. sleep diary) and objective measures (e.g. actigraphy). This will allow for conceptual and methodological clarity regarding the role of sleep. Existing studies focused on specific sleep disorders help in this regard, but it should be noted that the domain of 'sleep disturbance' extends beyond the boundaries of traditional sleep disorders.

Second, additional research needs to clarify the causal relationships between sleep and cardiometabolic disease. Epidemiological studies should focus more on longitudinal designs. Since most cardiometabolic outcomes (e.g. obesity, metabolic syndrome, coronary heart disease) represent years of accumulated risk, and sleep assessments typically only retrospectively capture 1–4 weeks, it is difficult to argue in cross-sectional designs that the sleep problems preceded the cardiometabolic problems. For this reason, prospective, longitudinal designs are critical. Intervention studies may also help. If a sleep-based intervention can improve cardiometabolic health risk factors or even outcomes, this would lend further support to the notion that sleep changes are causally related to changes in cardiometabolic health.

Third, there needs to be an increased recognition of the role of sleep in health beyond the traditional construct of sleep disorders. Sleep likely represents a major domain of health behaviour. Studying sleep this way requires a different conceptual framework, likely involves different intervention strategies, and necessitates the examination of social and behavioural influences on health such as race/ethnicity and socio-economics. These perspectives will aid in gaining a better understanding of who is at increased risk of poor sleep (Grandner et al., 2013d; Whinnery et al., 2014), how this increased risk may be differentially associated with poor outcomes (Grandner et al., 2013a, 2014a; Sands-Lincoln et al., 2013a), and how to develop more effective interventions.

Summary and conclusions

'Sleep disturbance' is variously defined. It may represent short or long sleep duration (potentially reflecting insufficient or excessive sleep), or it may represent inadequate sleep quality, represented as a sleep disorder (or symptoms of a sleep disorder), such as insomnia, a circadian rhythm sleep disorder, or sleep-disordered breathing. Alternatively, it may be represented as a general perception of overall sleep quality. Taken together, the evidence suggests that sleep disturbances (especially short sleep duration, short sleep in the context of insomnia, and sleep disordered breathing) have been reliably associated with cardiometabolic disease. Less data is available for insomnia without short sleep, circadian-rhythm disorders, and general poor sleep quality, though there are a number of studies to suggest that these, too, may be associated with cardiometabolic disease. These relationships seem to generally remain consistent across domains, suggesting the importance of sleep in regulating cardiometabolic function. Several studies have evaluated potential mechanistic pathways, including metabolic, inflammatory, and neurocognitive possibilities. Overall, though, sleep represents an important issue for public health.

In examining the role of sleep in the context of public health, several upstream determinants of poor sleep have been explored. In particular, evidence that minority status and lower socio-economic position is associated with poor sleep represents the majority of this literature. Other studies, though, have addressed acculturation, geographic environment, beliefs and attitudes, and other factors. Putting all of this evidence together, a social-ecological model of sleep and health shows sleep at the intersection of these upstream determinants and downstream consequences. This conceptualization may be useful in framing the role of sleep in health and may also aid in the development of some of the first interventions for poor sleep at the population level.

Several important future directions are evident from the available data. First, the concept of ‘sleep disturbance’ needs to be more fully fleshed out. Put another way, more research is needed to understand the various types of situations in which someone does or does not report or experience ‘sleep disturbance’. This will help us better understand the phenotype of interest and better target mechanistic or interventional studies. Second, more research is needed to elucidate the causal pathways. More longitudinal research is needed especially in this regard. Third, the role of sleep alongside other more recognized components of lifestyle and cardiometabolic health (such as diet and exercise) needs to be better conceptualized. With this research agenda realized, we can begin to develop and implement successful interventions for healthy sleep at the population level and potentially help to ameliorate the cardiometabolic disease burden attributable to poor sleep.

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Figure 1.
Socio-ecological model of sleep and health. Adapted from Grandner et al. (2010c).