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Sociodemographic and Cultural Determinants of Sleep Deficiency: Implications for Cardiometabolic Disease Risk

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Abstract

Sleep is a biological imperative associated with cardiometabolic disease risk. As such, a thorough discussion of the sociocultural and demographic determinants of sleep is warranted, if not overdue. This paper begins with a brief review of the laboratory and epidemiologic evidence linking sleep deficiency, which includes insufficient sleep and poor sleep quality, with increased risk of chronic cardiometabolic diseases such as obesity, diabetes and hypertension. Identification of the determinants of sleep deficiency is the critical next step to understanding the role sleep plays in human variation in health and disease. Therefore, the majority of this paper describes the different biopsychosocial determinants of sleep, including age, gender, psychosocial factors (depression, stress and loneliness), socioeconomic position and race/ethnicity. In addition, because sleep duration is partly determined by behavior, it will be shaped by cultural values, beliefs and practices. Therefore, possible cultural differences that may impact sleep are discussed. If certain cultural, ethnic or social groups are more likely to experience sleep deficiency, then these differences in sleep could increase their risk of cardiometabolic diseases. Furthermore, if the mechanisms underlying the increased risk of sleep deficiency in certain populations can be identified, interventions could be developed to target these mechanisms, reduce sleep differences and potentially reduce cardiometabolic disease risk.

Introduction

The impact of cardiometabolic diseases such as cardiovascular disease (CVD), diabetes and obesity worldwide is enormous. In 2005, approximately 1.5 billion adults globally were overweight and of these, more than 200 million men and nearly 300 million women were obese (Kelly et al., 2008). In addition, more than 300 million people worldwide have diabetes (Danaei et al., 2011), and over 80 million Americans have CVD (Lloyd-Jones et al., 2009). Cardiovascular disease, diabetes and overweight/obesity are closely linked conditions and are therefore considered together as “cardiometabolic diseases”. Risk factors for cardiometabolic diseases are not distributed equally across the globe or even within populations. Many sociocultural and behavioral factors are associated with increased cardiometabolic disease risk, including lower socioeconomic position, deficient diet and sedentary lifestyles. An additional factor that may increase cardiometabolic disease risk is sleep deficiency.

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This paper will begin by briefly reviewing evidence for a link between sleep deficiency, which is defined as too little sleep, poor sleep quality or sleep problems (National Center on Sleep Disorders Research, 2011), and increased risk of cardiometabolic disease. Given this potential link, we must now consider the biopsychosocial determinants of sleep deficiency. Thus, the majority of this paper will review these determinants, including age, gender, psychosocial factors, socioeconomic position, race/ethnicity and culture. One goal of this paper is to stimulate additional future research into human variation in sleep characteristics and potentially increase our understanding of differences in sleep and cardiometabolic disease. Understanding these determinants may provide novel insight into cardiometabolic disease prevention.

Characteristics of sleep

There are several different methods of assessing sleep that provide different dimensions of sleep. The gold standard is called polysomnography (PSG) and involves applying electrodes to the head, face and chest. Sleep architecture can only be assessed with PSG and is comprised of two major sleep stages, rapid-eye-movement (REM) sleep and non-rapid-eye-movement (NREM) sleep. NREM is further divided into stages 1, 2 and 3 (stage 3 is also called slow-wave sleep; SWS) and each successive stage represents deeper sleep. Sleep can also be estimated using wrist actigraphy, which involves wearing a wristwatch-like device that counts wrist movements (less movement indicates sleep) and simply identifies sleep versus wake (no stages). The advantage of actigraphy is that can be easily used for multiple days, which provides a better measure of habitual behavior. Finally, subjective estimates of sleep duration, quality and sleepiness can be collected.

Sleep Deficiency and Cardiometabolic Disease

Evidence from both experimental and epidemiologic studies indicates that decreased sleep duration and/or quality may be risk factors for cardiometabolic diseases. Specific sleep disorders, particularly obstructive sleep apnea, are also associated with cardiometabolic diseases, but are beyond the scope of this article.

Experimental Studies

Experimental studies have demonstrated that both sleep restriction and reduced sleep quality lead to adverse changes in glucose metabolism and appetite regulation (see Table 1). For example, restricted bedtimes have been associated with reduced glucose tolerance and reduced insulin sensitivity (Buxton et al., 2010; Spiegel et al., 1999), which are associated with diabetes risk. Laboratory studies also found changes in appetite regulating hormones, including lower levels of leptin, a hormone that signals satiety, and increased levels of ghrelin, an appetite stimulant after sleep restriction, despite controlled food intake and physical activity (Spiegel et al., 2004a; Spiegel et al., 2004b). Indeed some studies observed increased subjective appetite (Spiegel et al., 2004b), increased food intake (St-Onge et al., 2011) and increased snacking (Nedeltcheva et al., 2009) after sleep restriction. Finally, studies that impaired sleep quality (either by reducing SWS or fragmenting sleep) were associated with reduced glucose tolerance and reduced insulin sensitivity (Stamatakis & Punjabi, 2010; Tasali et al., 2008). Finally, studies have also observed increased blood pressure after a night of sleep restriction (Lusardi et al., 1996; Tochikubo et al., 1996). Taken together, these experimental studies suggest that both reduced sleep duration and reduced sleep quality are associated with adverse cardiometabolic effects.

Epidemiologic Studies

Observational studies from around the world have reported significant cross-sectional associations between short sleep duration and increased prevalence of obesity or higher BMI

(see Marshall et al., 2008 for a review). A recent meta-analysis found that for adults, short sleepers (≤5 hours/night) were 55% more likely to be obese and for children, short sleepers (≤10 hours/night) were 89% more likely to be obese (Cappuccio et al., 2008). Other studies reported that worse subjective sleep quality (Asplund & Aberg, 2001; Jennings et al., 2007) and less SWS (Rao et al., 2009) were associated with higher BMI and/or obesity. Two large epidemiologic studies found a positive association between sleep duration and leptin levels and one also found an inverse association between sleep duration and ghrelin levels (Chaput et al., 2007; Taheri et al., 2004), which is consistent with the experimental studies. Observational studies also found that shorter sleep durations were associated with increased caloric intake, particularly from fat (Grandner et al., 2010a; Weiss et al., 2010) and unhealthy eating habits such as skipping breakfast (Nakade et al., 2009; Nishiura et al., 2010) and snacking (Imaki et al., 2002). Finally, some prospective studies have observed greater weight gain associated with shorter sleep durations (Chaput et al., 2008; Hairston et al., 2010; Mozaffarian et al., 2011; Nishiura & Hashimoto, 2010; Patel et al., 2006; Watanabe et al., 2010), but not all (Lauderdale et al., 2009; Stranges et al., 2008a). Sleep loss therefore may disturb the neuroendocrine regulation of appetite.

Many studies reported that those who reported short sleep or sleep problems had an increased risk of incident diabetes, hypertension and CVD (Knutson, 2010). A meta-analysis found that those reporting sleeping ≤5–6 hours/night had a 28% higher risk of developing diabetes, and that those reporting difficulty initiating sleep or difficulty maintaining sleep had a 57% and 84% increased risk, respectively (Cappuccio et al., 2009). Longitudinal analysis of the first National Health and Nutrition Examination Survey indicated that sleeping ≤5 hours/night was associated with over two times the risk of incident hypertension over 8–10 years in adults aged 32 to 59 years (Gangwisch et al., 2006). The CARDIA Sleep Study observed a significant association between shorter sleep duration based on wrist actigraphy and increased risk of incident hypertension and increased risk of incident coronary artery calcification over 5 years in 35–50 year olds (King et al., 2008; Knutson et al., 2009). Finally, another meta-analysis found that short sleep was associated with a greater risk of developing or dying of coronary heart disease and stroke (Cappuccio et al., 2011). Thus, observational studies indicate that sleep deficiency is associated with increased risk of cardiometabolic diseases.

Determinants of sleep deficiency

Given the link between sleep deficiency and cardiometabolic disease, it is critical that we examine determinants of sleep deficiency. Table 2 presents a summary of the most consistent associations between these factors and sleep. The potential determinants discussed below were selected based on published associations with sleep, however these factors are not mutually independent nor is this list exhaustive. Indeed, additional research is required to understand better all determinants of sleep deficiency, and more importantly, the mechanisms underlying the associations. Social factors, cultural practices and complex behavior play a critical role in human health and we cannot underestimate their importance in the relationship between sleep and cardiometabolic disease risk.

Age and Gender

Two demographic factors whose associations with sleep are well-documented are age and gender. Sleep characteristics change across the lifecycle. Total sleep duration declines from birth to adulthood ranging from an average of 14 hours at 6 months to 8 hours at 16 years of age (Iglowstein et al., 2003). Many studies in the United States have reported a decline in sleep duration and quality with advancing age. A meta-analysis with over 3,500 people aged 5–102 years found that total sleep time, sleep efficiency (percentage of time in bed spent sleeping), percentage of SWS, and percentage of REM sleep significantly decreased linearly

with age (Ohayon et al., 2004). Thus, as we age we are at an increased risk of insufficient or poor quality sleep. It is important to note, however, that some have argued that the decline in sleep with aging may simply be due to co-morbidities (Vitiello, 2009). In addition, sleep deficiency in older adults may be due to factors other than illness, including social isolation and social support. Elderly adults with strong social support systems may have superior sleep quality than those lacking social connections, and this could be explained by psychosocial factors (described below). Moreover, the social positions and roles of elderly adults can vary greatly among different societies and could explain some variation in the association between sleep and aging. Whether sleep quality declines with age in regions other than the U.S. and Western Europe needs to be examined, particularly since identification of the reasons for sleep deficiency at older ages could have important implications for geriatric well-being.

Differences in sleep between men and women have also been observed. One striking difference pertains to SWS. Specifically, during middle age up through menopause, women exhibit greater amounts of SWS than men (Dijk et al., 1989; Redline et al., 2004), which may be due to reproductive hormones. Thus, if SWS is associated cardiometabolic disease risk, this suggests that men may be more vulnerable, but whether SWS mediates gender differences in disease risk has not been examined. Studies using wrist actigraphy have found that habitual sleep duration is longer and sleep quality is higher on average in women than men (Jean-Louis et al., 2000; Lauderdale et al., 2006). Subjective reports of sleep quality, however, tend to be lower and insomnia complaints higher in women than men (Ohayon, 2002; van den Berg et al., 2009). This contradiction has not been fully explained but may be related to gender roles and expectations. For example, women may be more willing to admit to sleep difficulties than men. Alternatively, increased anxiety and depression among women may account for increased sleep complaints (Voderholzer et al., 2003). Considering that substantial variation in gender roles exist among different societies, the degree to which the sleep of men and women differ throughout the world remains an interesting and unanswered question.

Although differences according to gender and age are probably conditioned by cultural and behavioral factors, gender and age are not modifiable. At the other end of the spectrum, however, are the aspects of our identities that are predominantly influenced or determined by sociocultural forces, as discussed in the following sections.

Psychosocial Factors

Several psychosocial factors are associated with sleep, including depression, stress and loneliness. The prevalence or incidence of depression has been associated with increased sleep disturbances (Riemann et al., 2001). For example, patients with depression exhibit alterations in sleep architecture, including reduced sleep efficiency and reduced SWS, and commonly complaint of insomnia. Conversely, individuals with insomnia experience elevated symptoms of depression and anxiety compared to those who sleep well.

Two other psychosocial factors, stress and loneliness, may also impair sleep. Animal studies have observed impairments in sleep quality after a stressor (Cui et al., 2008), but fewer studies of humans exist. Among caregivers, subjective sleep quality was significantly worse and perceived stress levels were higher compared to non-caregivers (Brummett et al., 2006). In a study that used PSG, sleep efficiency was lower and amount of wake was greater after a day with high subjective stress levels (Akerstedt et al., 2007). Increased subjective loneliness has also been associated with reduced sleep efficiency in undergraduate college students (Cacioppo et al., 2002) and with increased sleep fragmentation in a communal, religious society in South Dakota (Kurina et al., 2011).

These psychosocial factors may constitute an important link between the social environment and sleep. To the extent that people in different social positions are more or less vulnerable to stress, loneliness or depression, these psychosocial factors will play an important role in the associations between sleep deficiency and both socioeconomic position and race/ethnicity, as discussed below.

Socioeconomic Position (SEP)

Most studies of sleep and SEP among adults examined subjective sleep quality but a few included sleep duration and/or objective sleep measures. Studies in Taiwan, South Korea, and Italy found a higher prevalence of self-reported sleep problems (including difficulty falling asleep, staying asleep or waking too early) associated with lower SEP (Geroldi et al., 1996; Nomura et al., 2010). Several U.S. studies reported that lower SEP was associated with poorer subjective sleep quality, increased sleepiness and/or increased sleep complaints (Baker et al., 2009; Friedman et al., 2005; Gellis et al., 2005; Grandner et al., 2010b; Hall et al., 2009; Patel et al., 2010). Shorter average sleep duration may also be more common among lower SEP. A large U.S. study found increased odds of short sleep associated with lower SEP, which was partially due to differences in chronic health conditions and depressive symptoms (Stamatakis et al., 2007). A study that examined data from the U.S. and the U.K. reported that after adjustment for covariates such as age, sex, BMI, smoking, alcohol use, physical activity and health measures, lower SEP was significantly associated with short sleep only in the U.K. (Stranges et al., 2008b), suggesting that health behaviors or health status may confound the association between SEP and sleep in the U.S.

Other sociodemographic factors may modify the association between SEP and subjective sleep quality. For example, a study in the U.K. reported that higher social class was associated with fewer sleep problems only at younger ages (20–44 years) (Hunt et al., 1985), indicating age may modify the association. A Japanese study found a significant inverse association between SEP and subjective sleep quality in men but not in women (Sekine et al., 2006), suggesting gender differences. Another study examined perceived social status and found that it was negatively associated with subjective sleep quality for Asian Americans and African Americans but not for whites (Goodin et al., 2010), which suggests potential sociocultural differences in the association between sleep and SEP.

A few studies examined associations between SEP and objective measures of sleep. Among older women (61–90 years) lower SEP was associated with lower sleep efficiency (Friedman et al., 2005). Another study of women in midlife found that financial strain was associated with more wake and lower sleep efficiency from PSG (Hall et al., 2009). A study of U.S. adults aged 46–78 years found that lower SEP was associated with more wake based on PSG, but observed no association between SEP and sleep stages (Mezick et al., 2008). Among 128 American adults aged 18–52 years, childhood SEP was associated with more stage 2 and less SWS, even after adjusting for current SEP (Tomfohr et al., 2010). The few studies available indicate positive associations between objectively-measured sleep quality and SEP.

Some studies among children have observed an association between higher SEP and increased sleep duration and quality, but not all. In American children aged 12–60 months, those in families with lower SEP had later rise times, more nocturnal wake minutes, lower sleep efficiency and more night-to-night variability in bedtime based on wrist actigraphy (Acebo et al., 2005). A U.S. study of children aged 5–17 years reported that children from families whose highest wage earner had <12 years of education and children in poverty were less likely to sleep ≥9 hours/night (Cornelius, 1991). In Kentucky, children aged 2–7 years living in zip codes with low median income were more likely to exhibit excessive daytime sleepiness (McLaughlin Crabtree et al., 2005). A study of adolescents found lower SEP was

associated with shorter sleep durations, later bedtimes and greater irregularity in bedtimes (Marco et al., 2011). A study of Parisian children aged 8.5–11.5 years, however, observed no association between parental occupational status and self-reported sleep (Guerin et al., 2001). Although there are only a few studies in children, most suggest a positive association between sleep and SEP.

We need to elucidate the mechanisms linking low SEP to sleep deficiency particularly if sleep deficiency increases risk of cardiometabolic diseases. Social pressures and poor environmental conditions may cause decrements of sleep quality and quantity in lower SEP groups, and sleep may therefore mediate the relationship between SEP and health (Van Cauter & Spiegel, 1999). In addition to absolute differences in material standards, including environmental effects of noise, light and air pollution, differences in relative wealth, which can increase psychosocial stress, may modify associations between SEP and sleep deficiency in different societies. Indeed, Wilkinson (1997) argued that greater income equality within a society is associated with better health because of greater social cohesion, and this same phenomenon may operate in determining sleep differences by SEP. The possibility that sleep may mediate socioeconomic disparities in cardiometabolic diseases requires further investigation, as inexpensive, non-pharmacologic, behavioral interventions may be designed to improve health in those at risk. Indeed, Moore and others (Moore et al., 2002) found that self-reported sleep quality mediated the relationship between SEP and both physical health and psychological distress.

Race/ethnicity

As described in the previous section, lower SEP is associated with reduced sleep duration and quality. Health disparities among different racial and ethnic minorities are confounded by differences in SEP and differences in sleep likely are too. Indeed, racial/ethnic differences in sleep are probably due to social factors that get under the skin and disrupt normal physiological function. It is also important to note that “race” and “ethnicity” are socially constructed categories and not biologically or genetically determined groups. As the American Anthropological Association stated, “present-day inequalities between so-called racial groups are not consequences of their biological inheritance but products of historical and contemporary social, economic, educational and political circumstances” (“AAA statement on race,” 1998). Nonetheless, “race” and “ethnicity” can be meaningful constructs in societies that are “race conscious” such as the U.S. (Jones, 2000). Therefore, this section will review evidence for sleep differences between racial and ethnic groups, but these differences are most likely driven by sociocultural factors.

Most studies that examined racial or ethnic differences in sleep have compared African Americans to whites. Among studies that used subjective reports of sleep duration, most found that African Americans report short or long sleep durations. For example, according to the National Health Interview Survey, African Americans were more likely to report being short sleepers (≤6h) and long sleepers (≥9h) compared to whites, while Mexican Americans did not differ from whites (Hale & Do, 2007). The Alameda County Study in 1965–1983 also found that African Americans were more likely report short sleep (≤6h) than whites (Stamatakis et al., 2007). The association with African American race, however, was reduced by 32% when adjusting for living conditions (home ownership, household size), income and education but was reduced by only 13% when adjusting for chronic health conditions, health behaviors and depression, suggesting that socioeconomic factors may play a bigger role in these racial differences than health characteristics.

Studies of subjective sleep quality have had inconsistent results. Two large multi-ethnic studies reported that African Americans had higher odds of excessive daytime sleepiness (Baker et al., 2009; Baron et al., 2010). Physical health variables attenuated the racial

differences, suggesting that health and health behavior play an important role in this difference. Chinese and Hispanics were not significantly more likely than whites to have excessive daytime sleepiness. In Pennsylvania, African Americans regardless of income, Latinos who were poor and whites who were poor were significantly more likely to report poor sleep quality compared to whites who were not poor (Patel et al., 2010). After adjusting for sociodemographic and health characteristics, only the white poor and the African Americans who were not poor remained at an increased risk of reporting poor sleep quality, indicating that the increased risk of poor sleep quality among poor minorities may be due to health problems. Several studies have not observed worse subjective sleep quality in African Americans. For example, in a sample of 575 adults, self-reported symptoms of insomnia did not differ between African Americans and whites (Gellis et al., 2005). In the Behavioral Risk Factor Surveillance System, a national U.S. telephone study of over 150,000 adults, African American, Latina and Asian women were actually less likely to report frequent sleep complaints than white women, while in men only Asians were less likely to report frequent sleep complaints compared to whites (Grandner et al., 2010b). These results are in contrast to objective measures of sleep (discussed below), and this discrepancy underscores the need to understand better both the determinants and the consequences of subjective complaints of sleep quality.

Many studies included objective measures of sleep, such as actigraphy and PSG. A study in San Diego used wrist actigraphy and reported significantly shorter mean sleep duration (5.9 vs. 6.3 hours) and lower sleep efficiency (79% vs. 83%) for minorities compared to whites (Jean-Louis et al., 2000). A second study that used wrist actigraphy in adults aged 35–50 years also found that sleep duration and quality was significantly lower in African Americans, particularly men, compared to white women, even after adjustment for numerous sociodemographic and behavioral factors (Lauderdale et al., 2006). A study in Massachusetts compared the sleep of non-Hispanic African/Caribbean immigrant employees in long-term health care to white employees using actigraphy and found that the African/Caribbean immigrants slept an hour less on average (Ertel et al., 2011). Once socioeconomic and occupational characteristics were adjusted for, the difference was reduced to 38 minutes, suggesting that SEP and work factors partially account for the difference. Several studies that used PSG observed less SWS in African Americans than in whites (Hong et al., 2005; Mezick et al., 2008; Mokhlesi et al., 2011; Profant et al., 2002; Rao et al., 1999; Redline et al., 2004; Stepnowsky et al., 2003; Tomfohr et al., 2010). A meta-analysis found that African Americans had poorer sleep efficiency, shorter duration, less SWS, and a greater proportion of REM (Ruiter et al., 2011).

A few studies reported sleep differences in racial/ethnic groups in addition to African Americans. The Study of Women across the Nation enrolled white, African American and Chinese women in midlife (mean age 50 years). Similar to other studies, African Americans had more subjective sleep quality complaints and less sleep time, lower sleep efficiency, and less SWS based on PSG, even after adjustment for covariates including SEP (Hall et al., 2009). Chinese women also had less SWS than whites but did not differ on any other sleep characteristic. A large study in the U.S. found that in adjusted analyses, American Indians had less SWS than all other ethnic groups and that Hispanics did not differ from whites (Redline et al., 2004). It is important to recognize that the broad “racial” or “ethnic” categories typically used in research, including African Americans, Hispanics/Latinos, and Asian Americans, are actually extremely heterogeneous and any meaningful examination of sleep within racial and ethnic groups needs to focus on more specific communities.

A few studies have examined the association between race/ethnicity and sleep in children. For example, among 2–8 year olds, the age-related decline in napping frequency was more gradual for African American children than whites and by age 8, 39% of African American

children took naps compared to only 5% of white children (Crosby et al., 2005). In this same study, parentally reported nocturnal sleep duration on weekdays was shorter for African American children from ages 3 to 8 years, but because of napping, total sleep duration was the same. Thus, there may be important sociocultural differences in the distribution of sleep across the day, and examining only nocturnal sleep duration in children may underestimate total sleep. Nonetheless, in the Kentucky study of 2–7 year olds, African American children had greater daytime sleepiness and shorter sleep durations based on parental reports than white children, even after adjusting for SEP (McLaughlin Crabtree et al., 2005). A national survey in the U.S. found that African American teenagers were more likely to report sleeping 6 hours or less than all other ethnicities (Cornelius, 1991), however this association was not adjusted for any potential confounders. In Texas, a study of over 5,000 adolescents found that Chinese adolescents were at approximately half the risk of reporting insomnia symptoms while Mexican adolescents had about 25% greater risk compared to whites (Roberts et al., 2000). African American, Mexican and Central American adolescents were more likely to report problems with excessive sleep compared to whites (Roberts et al., 2000). These results conflict with the national survey, suggesting regional differences in the race-sleep association. One study conducted PSG in African American and white children aged 5–7 years but observed no significant racial differences in sleep architecture (O'Brien et al., 2007), which suggests the reduced SWS observed among African American adults may develop later.

Some attempts to identify mediators of the association between race/ethnicity and sleep have been made. A study in Pittsburgh of adults aged 46–78 years that found reduced sleep quality in African Americans explored several potential mediators of the race-sleep association, including negative affect, stressful life events, health behaviors, physical activity, and sleep environment, but none were significant mediators of the racial differences (Mezick et al., 2008). One study examined whether racial differences in sleep were in fact due to SEP in a sample of full-time college students (Goodin et al., 2010). They argued that minority ethnic groups may be more susceptible to negative social encounters (e.g., discrimination) and possess limited access to resources (e.g., well-paying jobs), which could affect their self-perceived social status and subsequent sleep quality. In this study, however, the racial/ethnic groups did not differ in terms of perceived social status, which may be due to the fact that they were all full-time college students. There is substantial evidence that African Americans get less sleep and have less SWS than whites, even after adjustment for some potential confounders. Thus, the degree to which SEP accounts for racial differences in sleep, and specifically which components of SEP are involved, remains to be determined.

We need to identify the underlying explanations for sleep deficiency among some ethnic minorities. Skin pigmentation or language use *per se* certainly does not impair sleep, therefore other factors common among these groups must. Racism and attendant discrimination presumably play an important role and can have a variety of effects. Some are psychological, such as lowered self esteem, greater stress, higher rates of mental illness, while some of the effects are more structural, such as the unequal distribution of socioeconomic positions in society by race, as well as access to health care. Experiences of marginalization, disrespect and devaluation can increase psychosocial factors, such as stress and loneliness, which impair sleep. We must expand our view to incorporate the social environment and not just individual-level factors. Aspects of the social environment, including neighborhood characteristics such as crime, safety, air pollution and noise, could be salient for understanding differences in sleep. Moreover, cultural beliefs and practices pertaining to sleep in particular communities need to be explored further, as these may provide explanations for associations that are group specific but not necessarily the result of discrimination. A better understanding of differences in sleep by racial and ethnic identity is necessary, particularly if these differences in sleep mediate disparities in cardiometabolic

disease. Indeed, these sleep differences parallel the disparities in obesity and diabetes the U.S., the rates of which are substantially higher among African Americans.

Culture

Culture is an often ill-defined concept, particularly in medical research. Anthropologists consider culture to be a complex set of behaviors, beliefs, attitudes and practices, all of which can have a substantial impact on health and health behaviors. Since sleep is in some respects a behavior, its pattern will be shaped by culture. Cultural practices pertaining to sleep, including when, where and with whom one sleeps, can all impact sleep duration and quality. Unfortunately, there is currently a dearth of research looking into cultural determinants of sleep. Undoubtedly, human variation in sleep practices and patterns exist, but little documentation of cross-cultural variation in sleep is available.

Some studies have compared sleep patterns in different countries, and although there is cultural heterogeneity within countries, differences between countries may reflect some shared cultural values or practices within each country as a whole. One study collected self-reported sleep habits among over 40,000 children aged 11 to 16 years from eleven mostly European countries, including Austria, Belgium, Finland, Hungary, Israel, Norway, Scotland, Spain, Sweden, Switzerland and Wales (Tynjälä et al., 1993). Bedtimes varied by only 1 hour between the eleven countries, the Hungarian and Swiss children went to bed the earliest and the Spanish children went to bed the latest (9:30 PM vs. 10:30 PM). Furthermore, Finnish children experienced difficulties falling asleep the most and the Spanish children the least while the Swiss slept the longest across all ages (9–10 hours/night) and Israeli children the least (8.3–9 hours/night). Some potential reasons for these differences include that these data was collected during the winter, which could have a greater impact on sleep disturbances in the Northern countries where winters are colder and light levels lower. Also, because Spanish children go to bed later, it may be easier for them to fall asleep and thus have fewer difficulties. Still, variation in cultural beliefs concerning appropriate bedtimes for children could account for some differences.

A second study collected sleep questionnaires in adults from ten countries around the world, including China, Japan, Slovakia, South Africa, Austria, Brazil, German, Portugal, Spain, and Belgium (Soldatos et al., 2005). Bedtimes were earliest in Brazil, Germany, Slovakia and South Africa (10:00 PM) and latest in Portugal and Spain (12:00 AM), while the shortest sleep duration was observed for Japan (6.9 hours) and the longest for Portugal (8.4 hours). Finally, the percentage of respondents reporting poor sleep quality varied from 10% in Austria to 32% in Belgium. It appears that substantial variation in subjective sleep characteristics exists worldwide, but why they exist is unknown.

One study compared 776 Italian adolescents in Rome to 572 American adolescents in Mississippi, 78% of whom were African American (LeBourgeois et al., 2005). Italian adolescents reported substantially better sleep quality and better sleep hygiene (factors that promote better sleep) than did American adolescents. Finally, data from France and the U.S. indicate a higher proportion of short sleepers among U.S. adults (44% vs. 33%) and adolescents (87% vs. 78%) compared to French adults and adolescents (Morselli et al., 2010). Variation in sleep between countries seems to be present for children, teenagers and adults.

Another sleep-related behavior that is likely to vary between cultures is napping. Among an agricultural population in Brazil, 72.3% of adults napped during the day at least once per week and 35.9% of males and 20.5% of females napped every day (Reimao et al., 2000). By comparison, in a national telephone poll in the U.S. 55% of adults said they take at least one nap during the week (National Sleep Foundation, 2005). The practice of napping in the

afternoon, the “siesta”, is a supposed part of many Mediterranean and Latin American countries. A study conducted among full-time college students in Mexico City reported that the siesta culture was absent (Valencia-Flores et al., 1998), but college students in a large, urban city may not maintain the traditional siesta practice. In contrast, among 471 adolescents in rural and semi-urban areas in Greece, almost 90% reported taking a siesta at least occasionally and 42% took a siesta regularly (Paraskakis et al., 2008). In the study of 10 countries around the world, the proportion of adults reporting regular napping varied greatly from 12% in Japan to 42% in Brazil (Soldatos et al., 2005). Whether or not taking a nap during the day is beneficial for cardiometabolic health is currently unknown.

Conclusion

Several critical yet understudied issues have been raised by this paper. First, the mechanisms by which aspects of the social environment condition health outcomes, including sleep, need to be identified. We can theorize that greater stress leads to worse sleep, but stress is a vague term and response to stressors can vary between individuals. Thus we must ask which particular stressors impair sleep and in whom.

Another issue is the age-old problem of disentangling SEP from race/ethnicity. How much of the association between race and sleep deficiency is due to poverty and the social environment? The data presented here show mixed results, but many observed worse sleep among African Americans even after adjusting for SEP indicators. Can membership in certain communities produce habits that are beneficial or detrimental to sleep? Many communities undoubtedly include both beneficial and detrimental habits and we must identify both. For example, beneficial factors could include membership in social organizations, such as churches, which may have positive psychosocial effects on sleep. Psychosocial predictors of sleep deficiency, such as depression, stress and loneliness, are correlated with social position and focusing on these factors may hold the key for understanding psychological links between class, race and sleep. For example, low self-esteem (related to depression) and loneliness (related to social integration) are clearly both related to social factors and sleep problems. Future research needs to operationalize pertinent components of SEP and the social environment and explicitly test whether they explain vulnerability to sleep deficiency.

One of the most understudied aspects of sleep is the cross-cultural perspective. This lack of attention is at least partly a reflection of the long-held assumption that sleep is entirely biological. Progress is being made in this regard as some anthropologists have identified sleep behaviors strongly conditioned by cultural beliefs and practices (Worthman & Melby, 2002). Furthermore, cross-cultural studies of sleep have the potential to show that the emphasis on consolidated nighttime sleep – among other commonly held assumptions about what constitutes a good night’s sleep – may not be a universal human standard.

Evidence from both experimental and observational studies indicates sleep deficiency may be a risk factor for cardiometabolic diseases and it is therefore imperative to identify and understand the determinants of sleep deficiency. Identification of salient determinants of sleep deficiency could be novel targets for interventions to prevent or improve cardiometabolic disease. This paper has reviewed demographic factors, such as age and gender, psychosocial factors, as well as socioeconomic position and race and ethnicity as possible determinants. The possibility of cultural influences on sleep patterns was discussed, but insufficient attention has thus far been paid to human variation in sleep around the globe. Moreover, whether this variation has any health implications needs to be verified, particularly if sleep differences partially account for cardiometabolic health disparities.

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Research highlights

- Sleep deficiency is associated with increased risk of obesity, diabetes and cardiovascular disease
- Possible determinants of sleep deficiency include older age, depression, stress and loneliness
- Lower socioeconomic position and African American race are also associated with sleep deficiency
- Cultural practices may be beneficial or detrimental to sleep but cultural variation in sleep behavior remains understudied
- Additional research on biopsychosocial determinants of sleep and underlying mechanisms is needed

Table 1

Effects of experimental studies of sleep manipulation on glucose tolerance and appetite regulation.

Sleep Manipulation	n; age; BMI	Outcome Effect	Reference
6d 4h TIB v 6d 12 h TIB	n=11 men Age: 18–27 y BMI <27kg/m^2	↓ Glucose Tolerance (−40%) ↓ Acute Insulin Response to Glucose (−30%)	(Spiegel et al., 1999)
6d 4h TIB v 6d 12h TIB	n=11 men Age: 18–26 y BMI <27kg/m^2	↓ Leptin (−19%)	(Spiegel et al., 2004a)
2d 4h TIB v 2d 10h TIB	n=12 men Mean ± SD age: 22 ± 2 y Mean ± SD BMI: 23.6 ± 2.0 kg/m2	↓ Leptin (−18%) ↑ Ghrelin (+28%)	(Spiegel et al., 2004b)
7d 5h TIB v 8d 10h TIB	n=20 men Age: 20–35 y BMI 20–30 kg/m^2	↓ Glucose Tolerance (−14%) ↓ Insulin Sensitivity (−20%)	(Buxton et al., 2010)
3d SWS suppression v 1 baseline night	n=5 men; 4 women Age: 20–31 y BMI<25 kg/m^2	↓ Glucose Tolerance (−23%) ↓ Insulin Sensitivity (−25%)	(Tasali et al., 2008)
2d sleep fragmentation v 1 baseline night	n=9 men, 2 women Age: 18–29 y Mean ± SD BMI: 24.3±3.0 kg/m^2	↓ Insulin Sensitivity (−25%)	(Stamatakis & Punjabi, 2010)
5d 4h TIB v 5d 9h TIB	n=15 men, 15 women Age: 30–49 y BMI 22–26 kg/m^2	↑ Caloric Intake (+12%) ↑ Fat Intake (+25%)	(St-Onge et al., 2011)
14d 5.5h TIB v 14d 8.5h TIB	n=6 men, 5 women Age: 34–49 y BMI: 24–29 kg/m^2	↑ caloric intake from snacks (+26%) No difference in total caloric intake	(Nedeltcheva et al., 2009).
2d 4.25h TIB v 2d 7.25h TIB	n=15 men Age: 20–40 y BMI<25 kg/m^2	No effect on leptin No effect on ghrelin No effect on food intake	(Schmid et al., 2009)

Abbreviations: y: years; d: days; h: hours; TIB: time in bed; SWS: slow wave sleep;

Symbols: ↑: increased; ↓: decreased

Table 2

A summary of the most consistent associations between specific determinants and sleep characteristics (see text for more detail)

Determinant	Association with sleep	Sleep Measure Used
Older Age	↓ TST	PSG
	↓ Sleep Quality	PSG
	↓ SWS	PSG
Female Sex	↑ SWS	PSG
	↑ Sleep Duration & Quality	Actigraphy
	↓ Sleep Quality	Self-report
Depression	↓ Sleep Quality	PSG
	↓ SWS	PSG
	↑ Insomnia	Self-report
Stress	↓ Sleep Quality	PSG & self-report
Loneliness	↓ Sleep Quality	Actigraphy & "Nightcap"[a]
Low Socioeconomic Position		
Adults	↓ Sleep Quality	PSG & Self-report
	↓ Sleep Duration	Self-report
Children	↓ Sleep Quality	Actigraphy
	↓ Sleep Duration	Self-report
African American Race (vs. White)		
Adults	↑ Short Sleep Duration (≤6h)	Self-report
	↑ Long Sleep Duration (≥9h)	Self-report
	↓ or = Sleep Quality	Self-report
	↓ Sleep Duration & Quality	Actigraphy
	↓ SWS	PSG
Children	↑ Napping	Parental report
	↓ or = Sleep Duration	Parental report
	= SWS	PSG

[a]"Nightcap" is an objective measure of sleep that uses a few electrodes and can distinguish NREM from REM sleep. It is not considered PSG, however.

Abbreviations: TST: total sleep time; PSG: polysomnography; SWS: slow-wave sleep.

Symbols: ↑: increased; ↓: decreased; =: equivalent/no difference