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Anxiety sensitivity and racial differences in sleep duration: Results from a national survey of adults with cardiovascular disease

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ABSTRACT

Although Blacks sleep between 37 and 75 min less per night than non-Hispanic Whites, research into what drives racial differences in sleep duration is limited. We examined the association of anxiety sensitivity, a cognitive vulnerability, and race (Blacks vs. White) with short sleep duration (<7 h of sleep/night), and whether anxiety sensitivity mediated race differences in sleep duration in a nationally representative sample of adults with cardiovascular disease. Overall, 1289 adults (115 Black, 1174 White) with a self-reported physician/health professional diagnosis of ≥1 myocardial infarction completed an online survey. Weighted multivariable logistic regressions and mediation analyses with bootstrapping and case resampling were conducted. Anxiety sensitivity and Black vs. White race were associated with 4%–84% increased odds, respectively, of short sleep duration. Anxiety sensitivity mediated Black–White differences in sleep duration. Each anxiety sensitivity subscale was also a significant mediator. Implications for future intervention science to address sleep disparities are discussed.

1. Introduction

Racial disparities in sleep duration are well documented (Chen et al., 2015; Lauderdale, Knutson, Yan, Liu, & Rathouz, 2008; Ruiter, Decoster, Jacobs, & Lichstein, 2011). For example, prior research using objective measures of sleep such as wrist actigraphy has found that Black men sleep on average up to 75 min less per night than White men, while Black women sleep up to 45 min less per night than White women (Chen et al., 2015). Short sleep duration, often classified as sleeping less than 7 h per night, is also more common among Blacks than Whites (Jackson, Redline, & Emmons, 2015; Krueger & Friedman, 2009; Stamatakis, Kaplan, & Roberts, 2007). Indeed, several studies find that Black Americans are more than twice as likely to report short sleep duration than White Americans, and this pattern has been observed in healthy adults as well as those with cardiovascular disease (CVD) (Alcantara, Peacock, Davidson, Hiti, & Edmondson, 2014; Hale & Do, 2007; Jackson, Redline, Kawachi, Williams, & Hu, 2013). While racial differences in sleep duration are well characterized, potential modifiable factors driving racial differences in sleep duration are understudied in the general adult population broadly, and in those with cardiovascular disease, specifically. Importantly, short sleep duration is becoming increasingly recognized as a risk factor for cardiovascular disease and related health risk behaviors, as well as a marker of excess prognostic risks in those with CVD (St-Onge et al., 2016). Thus, knowledge into factors that drive racial differences in sleep patterns, particularly in adults with CVD, may inform future intervention efforts to improve sleep, reduce CVD health disparities, and in turn potentially advance health equity for all.

Prior research with healthy adults and patients with CVD suggests that anxious pathology and poor sleep often co-occur (Alvaro, Roberts, & Harris, 2013; Babson, Trainor, Feldner, & Blumenthal, 2010; Jackson et al., 2015). For example, in an experimental study, adults who were exposed to either anxious cognitive arousal (i.e., speech threat task) or physiological arousal (i.e., caffeine capsule) immediately before bedtime exhibited longer sleep onset latency and shorter total sleep time (Tang & Harvey, 2004). Observational longitudinal research has also found evidence of an adverse effect of anxiety on sleep. In a large sample of Finnish adults, a general predisposition toward anxious arousal, or "anxious liability," was associated with poor sleep quality in both men and women (Vahtera et al., 2007). People who endorsed high symptoms of

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sympathetic nervous system hyperactivity exhibited a higher vulnerability to report poor sleep quality, and this relationship was stronger in men compared to women, particularly under increasing severity of stressful life events (Vahtera et al., 2007). This finding demonstrates that subthreshold anxiety levels also affect self-reported sleep quality. Further, in adults with CVD, anxiety and sleep deficits have been shown to co-occur. At least one study finds that sleep latency and short slow wave sleep are associated with cognitive anxiety (Edell-Gustaffson, 2002). Importantly, far less research has focused on identifying underlying and modifiable cognitive vulnerabilities, such as anxiety sensitivity, and their associations with poor sleep.

Anxiety sensitivity, often referred to as "fear of fear," is defined as the fear of the negative social, physical, or cognitive consequences of anxiety-related sensations (Olatunji & Wolitzky-Taylor, 2009; Reiss & McNally, 1985; Reiss, Peterson, Gursky, & McNally, 1986). Anxiety sensitivity is conceptualized as a multi-dimensional and trait-like cognitive vulnerability that intensifies pre-existing levels of anxiety (Olatunji & Wolitzky-Taylor, 2009; Reiss & McNally, 1985; Reiss et al., 1986). Three lower-order and interrelated factors that pertain to fear of anxiety have been identified, and include: social concerns (e.g., belief that observable anxiety symptoms will lead to social rejection), physical concerns (e.g., belief that palpitations result in cardiac arrest/heart attack), and cognitive concerns (e.g., belief that concentration difficulties result in mental incapacitation) (Taylor, Rabian, & Fedoroff, 1999; Taylor et al., 2007). Individuals with high anxiety sensitivity are more likely to exhibit increased attention to signals and cues of anxious arousal, and to make negative and catastrophic interpretations about anxiety-related sensations, and in turn experience increased anxiety, creating a positive and escalating feedback loop (Reiss & McNally, 1985).

A growing body of empirical evidence suggests that anxiety sensitivity is adversely related to optimal sleep health. High anxiety sensitivity has been linked with poor sleep quality, difficulty initiating sleep, and longer sleep onset latency in anxiety disorder clinical patient samples, even after accounting for important confounders such as depressive symptoms, worry, and traumatic life events (Babson, Boden, Woodward, Alvarez, & Bonn-Miller, 2013; Babson, Trainor, Bunaciu, & Feldner, 2008; Hoge et al., 2011; Raines et al., 2015). Related research with veteran populations also found that anxiety sensitivity was positively associated with worsening subjective sleep quality (Babson et al., 2013).

There is also recent preliminary evidence to suggest that specific subscales of anxiety sensitivity may be particularly related to sleep in the general adult population. For example, concerns about mental incapacitation or cognitive concerns related to anxious arousal were associated with sleep-related impairment (Vincent & Walker, 2001) and sleep dysfunction (Calkins, Hearon, Capozzoli, & Otto, 2013). Similarly, other research has found that cognitive concerns related to anxiety sensitivity (and not physical concerns or social concerns) partially mediated the effect of unacceptable thoughts on insomnia symptoms in adults with obsessive compulsive disorder (Raines et al., 2015). In contrast, other research has found that only the physical concerns subscale of anxiety sensitivity moderated the relationship between sleep anticipatory physical concerns and sleep onset latency (Babson et al., 2008). Specifically, sleep anticipatory physical concerns were associated with longer sleep onset latency only in those with high vs. low anxiety sensitivity (Babson et al., 2008).

Although there is mounting cross-sectional and longitudinal observational evidence of a relationship between anxiety sensitivity and poor sleep, most of this research was conducted with psychiatric samples and therefore not in cardiovascular disease samples, and without attention to racial/ethnic differences. Yet, the prevalence of anxious pathology, cardiovascular morbidity, and poor sleep differ across races/ethnicities (Chen et al., 2015; Evangelista, Ter-Galstanyan, Moughrabi, & Moser, 2009; Himle, Baser, Taylor, Campbell, & Jackson, 2009). Indeed, Blacks compared to Whites exhibit greater risk of adverse cardiovascular health behaviors, including poor sleep, and greater risk of CVD incidence and morbidity compared to their White counterparts (Jackson et al., 2015; Williams, Mohammed, Leavell, & Collins, 2010). Further, both poor sleep and anxiety sensitivity are established independent risk factors for CVD and cardiovascular health risk behaviors such as medication nonadherence, exercise, and smoking (Alcantara, Edmondson et al., 2014; Cappuccio, Cooper, D'Elia, Strazzullo, & Miller, 2011; Frasure-Smith et al., 2012; Smits, Tart, Presnell, Rosenfield, & Otto, 2010).

We used data from a nationally representative sample of adults with CVD in the United States (US) to: (1) examine the association of anxiety sensitivity and race (Black vs. White) with short sleep duration (<7 h of sleep/night), (2) test whether anxiety sensitivity mediates the relationship between race and sleep duration, and (3) explore whether specific subscales of anxiety sensitivity are consistent mediators of the effect of race on sleep duration. We hypothesized that anxiety sensitivity and Black race relative to White race would be independently associated with increased odds of short sleep duration (<7 h of sleep per night). We also hypothesized that anxiety sensitivity would mediate the relationship between race and sleep duration. Our working conceptual model was that increasing levels of anxiety sensitivity would correspond with increased attention to and vigilance for any cues or signals that indicate anxious arousal. This increased attention would promote perseveration about anxiety sensations and their consequences, which in turn would reduce total sleep time because of time spent in bed perseverating (worrying or ruminating) about anxious arousal, and time not spent sleeping. We hypothesized that this type of vigilance and attention to anxious arousal would partly explain Black–White differences in sleep duration.

2. Method

2.1. Participants

Participants included 1333 self-identified Black or non-Hispanic White US adults with a self-reported history of a physician or a health professional diagnosis of at least 1 myocardial infarction (MI). We restricted our sample to those who self-identified as Black or non-Hispanic White, reported 5 or less lifetime MIs, and average sleep duration of less than 15 h per night. Our final analytic sample included 1289 participants (787 men, 502 women) with complete data for the predictor, outcome, covariate, and mediator variables. Participants had a mean age of 64.9 years (SE = 0.36).

2.2. Procedures

We conducted a nationally representative online survey of US adults who reported that a doctor or health professional informed them that they had been diagnosed with a MI. YouGov, a nonpartisan research firm, administered the survey in English and Spanish from May 2013 to July 2013 to 1500 participants recruited from an opt-in survey panel. Participants received incentives through a YouGov loyalty program for their participation in the survey. YouGov panel members earn points for completing online surveys; the points can then be redeemed for rewards. Sampling targets and sampling weights were set based on gender, age, and race distribution of MI according to the 2010 National Health Interview Survey. Response rates were 45.8% for the English language survey and 16.2% for the Spanish survey. The Institutional Review Board of Columbia University approved this study.