A Review of Evidence for the Link Between Sleep Duration and Hypertension

James E. Gangwisch

There are lines of evidence from experimental sleep deprivation studies, population-based epidemiological studies, and an interventional study that point to the potential efficacy of adequate quality sleep to prevent and treat hypertension. Experimental sleep restriction has been shown to raise blood pressure and heart rate. Insufficient sleep on a chronic basis can raise average 24-hour blood pressure and lead to structural adaptions that entrain the cardiovascular system to operate at an elevated blood pressure equilibrium and increase the risk for hypertension. Disruptions in the timing and duration of sleep could also disrupt circadian rhythmicity and autonomic balance, which can increase the prevalence of the nondipping pattern, disturb diurnal rhythm of cardiac output, and increase blood pressure variability. Short sleep duration has been found to be associated with higher blood pressure and hypertension in both cross-sectional and longitudinal epidemiological studies. The association appears stronger in middle-aged adults and in women. Experimental sleep extension has been shown to significantly reduce blood pressure in individuals with prehypertension or stage 1 hypertension. The observed association between sleep duration and hypertension raises the hypothesis that interventions to extend sleep and improve sleep quality could serve as effective primary, secondary, and tertiary preventive measures for hypertension.

Keywords: blood pressure; circadian rhythm; epidemiology; hypertension; metabolic syndrome; sleep.

doi:10.1093/ajh/hpu071

The primary behavioral interventions for the prevention and treatment of hypertension have been changes in diet and physical activity, but there is growing evidence that improvements in sleep could also represent a behavioral target to combat high blood pressure. There are lines of evidence from experimental sleep deprivation studies, population-based epidemiological studies, and an interventional study that point to the potential efficacy of adequate quality sleep to prevent and treat hypertension. An evolutionary perspective provides a useful conceptual framework to organize and present these lines of evidence.

MECHANISMS LINKING SLEEP AND BLOOD PRESSURE

The central biological clock, or suprachiasmatic nucleus, evolved to synchronize activity, rest, and consumption to the circadian and circannual cycles using the autonomic nervous system. To entrain autonomic rhythms to the external environment, the suprachiasmatic nucleus requires repeated cues from light exposure, sleep, activity, and nutrient intake. These cues would have varied seasonally for our hunter-gatherer ancestors who lived away from the equator. The length of the daily photoperiod at the equator is 12 hours, and as the distance from the equator increases, the seasonal variation in daylight increases to the extremes at the poles of continuous daylight in summer and continuous darkness in winter. It is likely that the sleep durations of early humans were longer than 8 hours during most of the year, with those who lived away from the equator having been exposed seasonally to both shortened and lengthened photoperiods, inducing shorter sleep durations in summer and longer sleep durations in winter. Because blood pressure dips by an average of 10%–20% during sleep,1 less sleep in summer months resulted in higher average 24-hour blood pressures. Less sleep in summer also extended exposure to elevated sympathetic nervous system activity and to waking physical and psychosocial stressors. For our ancestors, the effects of seasonally shortened sleep on blood pressure were brief, but today short sleep durations and sleep disruptions are common throughout the year. More than 57% of participants in the National Sleep Foundation’s 2013 International Bedroom Poll reported getting less sleep than needed on workdays.2 We have many potential modern distractions, including shift work, 24-hour shopping, the Internet, cable television, dual careers, smart phones, and travel across time zones that can compete with sleep time and throw off sleep schedules. Irregular sleep schedules can contribute toward sleep maintenance insomnia characterized by nocturnal awakenings. Even if those with insomnia get adequate sleep by compensating for nocturnal awakenings by sleeping more during the day, chronic insomnia has been found to be associated with higher nighttime systolic blood pressure and blunted day-to-night systolic blood pressure dipping.3

Correspondence: James E. Gangwisch (jeg64@columbia.edu).

Initially submitted January 24, 2014; date of first revision February 26, 2014; accepted for publication March 12, 2014; online publication April 28, 2014.

1Division of Experimental Therapeutics, Department of Psychiatry, College of Physicians and Surgeons, Columbia University, New York, New York.

© American Journal of Hypertension, Ltd 2014. All rights reserved.

For Permissions, please email: journals.permissions@oup.com
Chronically insufficient sleep can function to develop and maintain hypertension. Experimental studies with both normotensive and hypertensive subjects have shown significant increases in blood pressure and sympathetic nervous system activity after nights where sleep was restricted. Sleep deprivation is a stressful condition, and stress has been shown to promote salt appetite and suppress renal salt-fluid excretion. Short-term sleep curtailment has been found to increase appetite, with particular cravings for salty snacks. Prolonged exposure to the increased total 24-hour hemodynamic load from inadequate sleep could lead to structural adaptations, such as arterial and left ventricular hypertrophic remodeling, that gradually entrain the entire cardiovascular system to operate at an elevated pressure equilibrium. There is also evidence that the pathophysiology of hypertension could contribute toward shortening sleep duration, which, in turn, could further raise blood pressure.

Habitually short sleep, typically defined as less than 5 or 6 hours per night in adults, can also increase the risk for hypertension by disrupting circadian rhythmicity and autonomic balance. Dramatic alterations in the timing and duration of external and internal zeitgebers in modern industrialized society are theorized to cause the environment sensed by the suprachiasmatic nucleus to become metabolically flattened and arrhythmic, disrupting the circadian rhythmicity of blood pressure in susceptible individuals. Hypertension is characterized by disturbances in the circadian rhythmicity of many physiological variables, such as an increased prevalence of the nondipping pattern, a shifting of the daily blood pressure profile to higher values, disturbances in the diurnal rhythm of cardiac output, and increased blood pressure variability. Hypertensive subjects who died of myocardial infarction or brain hemorrhage have been shown to have reductions of >50% in the 3 main neuronal populations of the suprachiasmatic nucleus compared with normal subjects.

### SLEEP AND METABOLISM

Short sleep duration has also been shown to affect metabolic parameters that are risk factors for hypertension. These metabolic alterations could lie along the causal pathway between short sleep duration and hypertension and therefore act as partial mediators of the relationship. The theory of the seasonal expression of the thrifty genotype provides a useful way to conceptualize the links between metabolism, circannual rhythms, and sleep. The thrifty genotype is theorized to have evolved to be expressed during seasons of high food availability to facilitate the deposition of fat reserves through insulin resistance and other metabolic changes to prepare for later seasons of relative food scarcity. In the extreme case of hibernators, the fat-storing phase is characterized by hyperinsulinemia and insulin insensitivity to promote large gains in weight, mainly through the accumulation of stores of triglycerides in adipose tissue. These changes are transitory, however, and reverse themselves once hibernation begins. The stored fat, predominantly from large abdominal depots, provides the primary metabolic fuel for the entire winter hibernation season. Although humans do not hibernate, it is likely that we share at least some of the genes and pathways involved in orchestrating seasonal weight fluctuations. The cues to signal summer, and therefore to trigger the seasonal expression of the thrifty genotype, include extended photoperiods, shorter sleep durations, and consumption of seasonally available fruits and carbohydrates. Only in very recent history has industrialization dramatically altered our environments, allowing the choice to bask in artificial light, get very little sleep, and gorge on processed sweets throughout the year. These modern changes could persistently activate a thrifty phenotype that had previously evolved to be expressed only on a seasonal basis. Metabolic changes that evolved to be adaptive on a seasonal basis for our hunter-gatherer ancestors could now, because of our modern lifestyles, increase our risks for diabetes, hypercholesterolemia, obesity, and the metabolic syndrome. Experimental sleep restriction has been shown to decrease glucose tolerance, compromise insulin sensitivity, increase total and low-density lipoprotein cholesterol levels, decrease leptin levels, increase ghrelin levels, and elevate hunger and appetite ratings with particular cravings for sweets, starch, and salty snacks. Short sleep duration has been shown to be associated with the incidences of diabetes, hypercholesterolemia, obesity, and the metabolic syndrome. Diabetes and obesity have been shown to act as partial mediators of the association between short sleep duration and the incidence of hypertension, providing evidence that metabolic alterations lie along the causal pathway between short sleep duration and hypertension.

### EPIDEMIOLOGICAL STUDIES

There have been many cross-sectional and longitudinal epidemiological studies on the relationship between sleep duration and hypertension (Table 1). The studies have included diverse populations from around the world and have been comprised of age groups ranging from children to the elderly. Results have varied by age group, sex, and whether the studies were cross-sectional or longitudinal. One meta-analysis of longitudinal studies found short sleep duration to be associated with increased risk for hypertension incidence.

### Children and Adolescents

Results from epidemiological studies on the relationship between sleep duration and blood pressure in children and adolescents have been mixed. Two longitudinal studies with children found decreases in total sleep time to be associated with increases in blood pressure at follow-up. Results from cross-sectional studies with children have been inconsistent. One cross-sectional study with children found only a marginally lower mean arterial pressure in children with the longest sleep vs. those with the shortest sleep, whereas another study found no association between sleep quantity and 24-hour ambulatory blood pressure. A cross-sectional study that included female and male children and adolescents found short sleep duration to be associated with hypertension prevalence only in male children between the ages of 11 and 14 years. Three cross-sectional studies that included only adolescents showed negative relationships between...
Table 1. Cross-sectional and longitudinal studies of the relationship between sleep duration and hypertension

<table>
<thead>
<tr>
<th>Source</th>
<th>Sample</th>
<th>Follow-up period</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Cross-sectional studies</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Bansil et al.22</td>
<td>10,308 US women and men aged ≥18 years</td>
<td>N/A</td>
<td>Combination of short sleep duration with sleep disorders associated with hypertension prevalence. Short sleep alone not associated with hypertension prevalence</td>
</tr>
<tr>
<td>Bayer et al.23</td>
<td>7,701 German female and male children aged 3–10 years</td>
<td>N/A</td>
<td>Sleep duration had no or only marginal association with blood pressure</td>
</tr>
<tr>
<td>Bjorvatn et al.24</td>
<td>8,860 Norwegian women and men, aged 40–45 years</td>
<td>N/A</td>
<td>Short sleep duration associated with significantly higher systolic blood pressure</td>
</tr>
<tr>
<td>Cappuccio et al.25</td>
<td>5,766 English women and men, aged 35–55 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence only in women</td>
</tr>
<tr>
<td>Choi et al.26</td>
<td>4,222 Korean women and men, aged ≥ 20 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence only in those aged &lt;60 years</td>
</tr>
<tr>
<td>Fang et al.27</td>
<td>71,455 US women and men, aged ≥ 18 years</td>
<td>N/A</td>
<td>Short and long sleep duration in hypertension prevalence in those aged &lt;45 years, in middle-aged men, and in older women</td>
</tr>
<tr>
<td>Faraut et al.28</td>
<td>1,046 French women and men, aged ≥ 40 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence.</td>
</tr>
<tr>
<td>Gangwisch et al.21</td>
<td>82,130 US women aged 40–65 years</td>
<td>N/A</td>
<td>Short and long sleep duration associated with hypertension prevalence in all age groups</td>
</tr>
<tr>
<td>Hall et al.31</td>
<td>8,674 US women aged 37–64 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Gottlieb et al.29</td>
<td>5,910 US women and men aged 40–100 years</td>
<td>N/A</td>
<td>Short and long sleep duration associated with hypertension prevalence</td>
</tr>
<tr>
<td>Guo et al.30</td>
<td>4,902 Chinese female and male children and adolescents aged 5–18 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence in male subjects aged 11–14 years only</td>
</tr>
<tr>
<td>Hall et al.31</td>
<td>1,214 US women and men aged 30–54 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Javaheri et al.32</td>
<td>238 US female and male adolescents aged 13–16 years</td>
<td>N/A</td>
<td>Short sleep duration associated with prehypertension</td>
</tr>
<tr>
<td>Kawada et al.33</td>
<td>4,941 Japanese men aged 36–60 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Kim et al.34</td>
<td>5,393 Korean women and men aged 19–99 years</td>
<td>N/A</td>
<td>Short sleep associated with hypertension in adults aged &lt;65 years but not in adults aged ≥65 years</td>
</tr>
<tr>
<td>Knutson et al.35</td>
<td>578 US women and men aged 33–45 years</td>
<td>N/A</td>
<td>Short sleep associated with higher systolic and diastolic blood pressure levels</td>
</tr>
<tr>
<td>Lima-Costa et al.36</td>
<td>1,423 Brazilian women and men with mean age of 69 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Lopez-Garcia et al.37</td>
<td>3,686 Spanish women and men aged ≥60 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Magee et al.38</td>
<td>218,155 Australian women and men aged ≥45 years</td>
<td>N/A</td>
<td>Short and long sleep duration associated with hypertension prevalence in those aged &lt;70 years but not in those aged ≥70 years</td>
</tr>
<tr>
<td>Martikainen et al.39</td>
<td>231 Finnish female and male children aged 8 years</td>
<td>N/A</td>
<td>Sleep quantity not associated with 24-hour ambulatory blood pressure</td>
</tr>
<tr>
<td>Mezick et al.40</td>
<td>246 US female and male adolescents aged 14–19 years</td>
<td>N/A</td>
<td>Shorter sleep associated with higher 48-hour blood pressure, higher nighttime blood pressure, and a higher systolic blood pressure sleep/wake ratio</td>
</tr>
<tr>
<td>Stang et al.41</td>
<td>4,766 German women and men aged 45–74 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence only in women</td>
</tr>
<tr>
<td>Stranges et al.42</td>
<td>6,472 British women and men aged 45–69 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence only in women</td>
</tr>
</tbody>
</table>

(Continued)
sleep duration and blood pressure. Short sleep duration in adolescents has been found to be associated with prehypertension, increased systolic blood pressure, higher 48-hour blood pressure, higher nighttime blood pressure, and a higher systolic blood pressure sleep/wake ratio.

Young and middle-aged adults

The strongest evidence for an association between sleep duration and hypertension has come from studies with young and middle-aged adults. Five longitudinal studies showed significant associations between short sleep duration and hypertension incidence in young and middle-aged adults, and eleven cross-sectional studies showed relationships with hypertension prevalence. Three cross-sectional studies found individuals with short sleep to have significantly higher blood pressures. Bansil et al. found short sleep duration in combination with sleep disorders, but not short sleep duration alone, to be associated with hypertension prevalence. Another study found short sleep duration in combination with chronic insomnia, but not short sleep duration alone, to be associated with hypertension incidence. One longitudinal study and two cross-sectional studies that included middle-aged adults found no association between sleep duration and hypertension.

Elderly adults

Differences between middle-aged and older adults have been found in the relationship between sleep length and hypertension. No longitudinal studies have shown a

---

**Table 1. Continued**

<table>
<thead>
<tr>
<th>Source</th>
<th>Sample</th>
<th>Follow-up period</th>
<th>Results</th>
</tr>
</thead>
<tbody>
<tr>
<td>Stranges et al.</td>
<td>3,027 US women and men, aged 35–79 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence only in women, with a stronger association in premenopausal women</td>
</tr>
<tr>
<td>van den Berg et al.</td>
<td>5,058 Dutch women and men, aged 58–98 years</td>
<td>N/A</td>
<td>No association between sleep duration and hypertension prevalence</td>
</tr>
<tr>
<td>Vgontzas et al.</td>
<td>1,741 US women and men, aged ≥20 years</td>
<td>N/A</td>
<td>Short sleep duration associated with hypertension prevalence</td>
</tr>
<tr>
<td>Wells et al.</td>
<td>4,452 Brazilian male and female adolescents aged 10–12 years</td>
<td>N/A</td>
<td>Short sleep duration associated with increased systolic blood pressure</td>
</tr>
</tbody>
</table>

**Longitudinal studies**

| Archbold et al. | 334 US female and male children aged 6–11 years | 5 years | Decrease in total sleep time associated with increases in systolic blood pressure |
| Beunza et al. | 11,837 Spanish university graduates, women and men, with an average age of 36 years | 3 years | No significant difference found in the relationship between sleep quartiles and hypertension incidence |
| Capuccio et al. | 3,691 English women and men aged 35–55 years | 7 years | Short sleep associated with hypertension incidence only in women |
| Fernandez-Mendoza et al. | 1,395 US adult women and men | 7.5 years | Short sleep duration in combination with chronic insomnia associated with hypertension incidence but not short sleep duration alone |
| Gangwisch et al. | 8,992 US women and men aged 32–86 | 8 to 10 years | Short sleep associated with hypertension incidence only in those aged 32–59 years |
| Gangwisch et al. | 82,130 US women aged 40–65 years | 6 years | Short sleep associated with hypertension incidence only in younger women aged >60 years |
| Kim et al. | 4,965 Korean women and men aged 40–69 years | 6 years | Short sleep associated with hypertension incidence only in premenopausal women |
| Knutson et al. | 578 US women and men aged 33–45 years | 5 years | Short sleep associated with hypertension incidence and adverse changes in systolic and diastolic blood pressure levels |
| Lopez-Garcia | 3,686 Spanish women and men aged ≥60 years | 2 years | No association between sleep duration and hypertension incidence |
| Wells et al. | 4,452 Brazilian female and male adolescents aged 10–12 years | 11–12 years | Short sleep duration associated with significantly higher systolic and diastolic blood pressure at follow-up |
significant relationship between sleep duration and hypertension incidence in elderly participants. One likely explanation for the lack of an association in longitudinal studies relates to the fact that hypertension is a common disease that becomes more prevalent as age increases. Because longitudinal studies exclude prevalent cases of hypertension at baseline, prospective analyses with elderly cohorts are confined to those individuals at an age when sleep difficulties are common. The sleep of elderly subjects can be affected by age-related changes in sleep architecture, compensatory daytime sleep, and medication side effects. Survival bias due to increased mortality associated with hypertension and other components of the metabolic syndrome may also help explain the different relationships found between sleep duration and hypertension in the younger and older age groups. Many cross-sectional analyses that were confined to older adults found no association between sleep duration and hypertension prevalence in elderly participants. Some studies that pooled results from young, middle-aged, and elderly adults showed positive associations between sleep duration and hypertension prevalence, likely due predominantly to the influence of the younger participants. Only one study found a significant association between short sleep duration and hypertension in an analysis that included a sample of strictly elderly participants. The relationship between sleep duration and hypertension could differ between younger and older adults because of distinct influences of increased sympathetic activation from short sleep duration on the types of hypertension that predominate in middle-aged and elderly adults. Increased sympathetic activation could influence systolic and diastolic hypertension, which is common in middle-aged populations, but not isolated systolic hypertension resulting from age-related loss of arterial compliance, which accounts for nearly 60% of hypertension in elderly populations.

Females

Many epidemiological studies that conducted analyses stratified by sex indicate that the relationship between short sleep duration and hypertension is stronger in female subjects. Two longitudinal studies found short sleep duration to be associated with the incidence of hypertension only in women, with 1 of the studies finding the association only in premenopausal women. Four cross-sectional studies also found short sleep duration to be associated with hypertension prevalence only in female subjects. Cappuccio et al. pointed out a number of potential mechanisms that could help explain sex differences in the association. First, the average age of their sample was approximately 55 years, corresponding with the menopausal period that is associated with major hormonal changes and psychosocial stressors that could increase the risk for adverse health outcomes such as hypertension. Second, Cappuccio et al. found the distribution of correlates of short sleep duration, such as depression, to differ between men and women, therefore potentially contributing toward the observed associations. Third, the authors theorized that differential self-reporting of sleep habits could have affected the results.

Long sleep duration

Long sleep duration has been found to be associated with hypertension prevalence in some cross-sectional studies, but no published longitudinal studies have shown an association between long sleep duration and hypertension incidence. Longitudinal studies have the benefit of a temporal order between variables to strengthen the counterfactual argument that if the independent variable had not occurred then the dependent variable would not have occurred. Cross-sectional studies, on the other hand, are more susceptible to the potential bidirectional relationship between the theorized independent and dependent variables. Reverse causation could be a contributing factor in the cross-sectional relationship between long sleep duration and hypertension. The inflammatory process has been shown to play key roles in the pathogenesis and pathophysiology of metabolic disorders such as cardiovascular disease. Proinflammatory cytokines contribute toward sleepiness and fatigue, presumably as an evolutionary adaptation to promote rest and recovery from illness. Proinflammatory cytokines have been shown to be elevated in those suffering from hypertension. There is also little evidence that long sleep duration has adverse health effects, making it likely that long sleep duration is an epiphenomenon of hypertension as opposed to a cause.

Other sleep characteristics contributing toward hypertension

Duration is not the only characteristic of sleep that has been shown to increase the risk for hypertension. Insomnia coupled with short sleep duration has been shown to synergistically and significantly increase the risk for hypertension incidence. Shift work, which is associated with changes in the timing of sleep and circadian reversal, has been shown to be a risk factor for hypertension. Variations in sleep architecture, such as decreased percentage of time in slow-wave sleep in elderly men and low non-rapid eye movement sleep delta power in midlife women, have been shown to increase the risk for hypertension incidence.

Intervention study

Investigators from Harvard Medical School conducted a 6-week sleep extension interventional study with 22 participants with prehypertension or stage 1 hypertension. Participants were randomized to either a sleep maintenance group or to a sleep extension group with the aim to increase the amount of time in bed by 1 hour each day. The sleep extension intervention involved setting habitual bed times 30 minutes earlier and wake times 30 minutes later. The sleep durations of subjects in the sleep extension group increased by an average of 35 minutes, and their average 24-hour systolic and diastolic beat-to-beat blood pressures decreased significantly (P < 0.05) by an average of 14 and 8 mm Hg, respectively, from before to after intervention. The blood pressure reduction in the intervention group did not differ significantly from the sleep maintenance group (P = 0.15), but both groups were provided with and asked to follow a
set of sleep hygiene recommendations designed to improve overall sleep habits. The sleep hygiene instructions included emphasizing the importance of maintaining a regular sleep/wake schedule, establishing an undisturbed and comfortable sleeping environment, timing exercise at optimal periods, and controlling alcohol consumption. The results from this study point out the potential for sleep interventions to decrease blood pressure in individuals with prehypertension or stage 1 hypertension. Improvements in sleep could augment the other common behavioral interventions to prevent and treat hypertension. Because sleep deprivation is associated with increased hunger, sufficient sleep could help people stick to dietary interventions and lose weight. Adequate sleep could also allow for the necessary energy to engage in regular physical activity.

Common limitations

Many of the studies on the relationship between sleep duration and hypertension have similar limitations. Sleep duration measures are often based upon self-report in large epidemiological studies because more objective measures such as actigraphy and polysomnography can be cost-prohibitive on a larger scale. Conversely, studies that do include objective sleep measures often have smaller sample sizes with less statistical power to test hypotheses. One study with a relatively large sample size (n = 1,395) found a nominally higher risk of hypertension when sleep was measured objectively with polysomnography. Many longitudinal studies also lack repeated measures of sleep duration, making it difficult to determine how representative the baseline sleep measure is of the sleep durations over the follow-up period. The hypertension outcome in many studies has been based upon self-report of physician diagnosis, which could result in bias. Hypertension also frequently goes undiagnosed and we have no way of knowing whether sleep duration affects the likelihood of seeking or receiving treatment for hypertension.

Comparing results from studies on sleep duration and blood pressure can be problematic. Sleep duration can be categorized or retained as a continuous variable in various ways, making it difficult to compare results between studies. Different studies also include various covariables and can conceptualize them as either potential confounders or mediators of the relationship.

Implications

There is strong evidence that sleep duration impacts blood pressure and acts as a risk factor for hypertension. Future epidemiological studies would ideally include repeated objective sleep and blood pressure measures and include all relevant potentially confounding and mediating variables. Enough evidence exists to encourage the funding of larger interventional studies to explore the efficacy of sleep extension and sleep improvement to lower blood pressure. Getting enough sleep has many known benefits and few drawbacks other than the need to prioritize adequate time to rest. Whether teaching proper sleep hygiene techniques and providing encouragement to devote the necessary time to sleep could serve as effective primary, secondary, and tertiary preventive measures for hypertension needs further study.

ACKNOWLEDGMENT

This work was supported by National Institutes of Health grant HL091443 from the National Heart, Lung, and Blood Institute.

DISCLOSURE

The author declared no conflict of interest.

REFERENCES

39. Sleep duration are associated with prevalent cardiovascular disease in Magee CA, Kritharides L, Attia J, McElduff P, Banks E. Short and long
JR, Rodriguez-Artalejo F. Self-reported sleep duration and hyperten
Lopez-Garcia E, Faubel R, Guallar-Castillon P, Leon-Muñoz L, Banegas
Study (BHAS).
32. Usual sleep duration is not asso
10. Lopez-García E, Faubel R, Guallar-Castillon P, Leon-Muñoz L, Banegas JR, Rodríguez-Artalejo F. Self-reported sleep duration and hyperten
Hypertension 2009; 57:663–668.
11. Vgontzas AN, Liao D, Bixler EO, Chrousos GP, Vela-Bueno A. Insomnia with objective short sleep duration is associated with a high risk for hyperten
Hypertension 2009; 32:491–497.
Hypertension 2012; 60:929–935.
