Acute and Chronic Effects of Sleep Duration on Blood Pressure

WHAT’S KNOWN ON THIS SUBJECT: Inconsistent results have been reported on the association between sleep duration and blood pressure (BP) in children, likely as a result of inadequate adjustment for confounders and the use of different time frames in assessing sleep duration.

WHAT THIS STUDY ADDS: Short sleep duration and poor sleep quality are associated with higher BP in normal-weight adolescents. One night of adequate sleep may partially ameliorate the risk of high BP but cannot completely reverse the effect of chronic sleep insufficiency.

abstract

OBJECTIVE: To evaluate the association between ambulatory blood pressure (ABP) and sleep duration as measured by 7-day sleep diary and nocturnal polysomnography in normal-weight adolescents without significant obstructive sleep apnea.

METHODS: Subjects aged 10 to 17.9 years with an obstructive apnea hypopnea index <5 underwent polysomnography for 9.5 hours and 24-hour ABP monitoring commencing at noon on the same day. ABP was divided into prepolysonmography, in bed during polysomnography, and postpolysomnography periods for separate analyses. Sleep duration (SpD7) was obtained from a 7-day sleep diary, reflecting the sleep pattern in the week before admission. Total sleep time (TST) and sleep efficiency (SpE) were obtained from polysomnography.

RESULTS: A total of 143 adolescents participated. SpD7 was inversely associated with systolic blood pressure (SBP) in prepolysonmography, in-bed, and postpolysomnography periods (all \(\beta = -2 \text{ mm Hg} \)) and with diastolic blood pressure (DBP) in prepolysonmography and in-bed periods (all \(\beta = -1 \text{ mm Hg} \)). TST was inversely associated with SBP in the postpolysonmography period (\(\beta = -1.5 \text{ mm Hg} \)). SpE was inversely associated with SBP in in-bed period (\(\beta = -0.1 \text{ mm Hg} \)) and with DBP in in-bed (\(\beta = -0.1 \text{ mm Hg} \)) and postpolysonmography (\(\beta = -0.2 \text{ mm Hg} \)) periods. Neither TST nor SpE was associated with SBP and DBP in prepolysonmography period.

CONCLUSIONS: Short sleep duration as reflected by 7-day sleep diary was associated with higher blood pressure in normal-weight adolescents. Occasional adequate sleep may partially ameliorate the risk of high blood pressure but may not completely reverse the effect of long-term sleep insufficiency. Pediatrics 2014;133:e64–e72

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KEY WORDS adolescents, ambulatory blood pressure monitoring, obstructive sleep apnea, obesity, short sleep

ABBREVIATIONS ABP—ambulatory blood pressure
ABPM—ambulatory blood pressure monitoring
BP—blood pressure
DBP—diastolic blood pressure
OAHI—obstructive apnea hypopnea index
OSA—obstructive sleep apnea
SBP—systolic blood pressure
SD—average daily sleep duration obtained from a 7-day sleep diary
SE—sleep efficiency
TST—total sleep time

Mr Au conceptualized and designed the study, coordinated and supervised data collection, carried out data analyses, and drafted the initial manuscript; Dr Wing conceptualized and designed the study and critically reviewed the manuscript; Mr Ho performed data collection, carried out initial analyses, and reviewed and revised the manuscript; Dr Lam reviewed and revised the manuscript; Dr Li conceptualized and designed the study, supervised data collection, and critically reviewed the manuscript; and all authors approved the final manuscript as submitted.

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Inadequate sleep is a common and global phenomenon in modern society. The reasons for short sleep duration in children and adolescents are likely multifactorial and include family, school, media use, and individual factors. In this regard, inadequate sleep duration has been linked with obesity and ath- erogenic dyslipidemia in children and adolescents.

Blood pressure (BP) is an important marker of future health because elevated BP in childhood is associated with higher cardiovascular risk in later life. Therefore, understanding the risk factors associated with elevated BP may help to avoid cardiovascular adverse events in the future. Compared with spot BP measurement, ambulatory BP monitoring (ABPM) is a more reliable and comprehensive tool in assessing the circadian variation in BP levels, which is especially important in sleep-related research where accurate documentation of nocturnal events is essential.

Several studies investigating the association between sleep duration and BP in children or adolescents have been published. The reported results, however, are conflicting and inconsistent (Supplemental Table 5). Most studies did not control for the confounding effects of obesity and obstructive sleep apnea (OSA). The interrelationship between sleep duration, obesity, OSA, and BP is complicated. Obesity is a well-established risk factor for OSA, which has been demonstrated to lead to elevated BP.

Anthropometric variables including weight, height, waist, and hip circumferences were measured. Waist circumference and BMI were converted to z scores according to normal reference. Sexual maturity rating was evaluated by a validated gender-specific self-assessment questionnaire. Information on parental history of hypertension and use of antihypertensive medications was obtained directly from parents.

Sleep Diary
All subjects had to fill in a sleep diary that asked about bedtime, get-up time,
sleep latency, nap duration, and caffeine use prospectively for the week immediately before hospital admission for assessment (Fig 1). The average sleep duration over the week (SD7) was calculated as the average of nocturnal sleep duration (ie, the amount of time between bedtime and get-up time minus the length of sleep latency) plus daytime nap duration over the 7-day recording period.

Polysomnography
An overnight polysomnography was performed in a dedicated sleep laboratory with the CNS 1000P polygraph (Chanhassen, MN) as described in our previous publication. All computerized sleep data were further manually edited by experienced polysomnography technologists and clinicians according to standard criteria. All studies were set to record the time in bed as 9.5 hours ± 5 minutes, starting at 21:30 ± 30 minutes and ending at 07:00 ± 30 minutes the next morning (Fig 1). TST was the amount of actual sleep time during the recording period. SE was calculated as the TST divided by the amount of time in bed between lights out and final awakening.

ABPM
Twenty-four-hour ABP was measured by using an oscillometric monitor (SpaceLabs 90217; SpaceLabs Medical, Redmond, WA), which has been validated for use in children. Systolic BP (SBP) and diastolic BP (DBP) were measured every 30 minutes during nighttime starting from 21:30 to 07:00 and every 15 minutes from 07:00 to 21:30 (daytime period). The proper cuff chosen according to the length of the subject’s arm was placed in the non-dominant arm. A recording was considered adequate and valid when it contained at least 40 readings for the 24-hour period, with a minimum of 1 successful reading per hour. Mean SBP and DBP over 24 hours were converted into z scores by using the LMS reference values (relative to gender and height) published by Wühl et al. To examine the effects of SD7 and the acute change in sleep pattern induced by the standardized bedtime and get-up time at polysomnography, the recorded ABP readings were divided into prepolysomnography, in-bed, and postpolysomnography periods for separate analyses. The prepolysomnography period was defined as the time between the start of ABPM and bedtime. The in-bed period referred to the time between bedtime and get-up time. The postpolysomnography period referred to the time between get-up time and the end of ABPM (Fig 1). Average SBP and DBP were calculated for each of these periods. Nocturnal dipping was calculated as the difference between daytime BP and nighttime BP and expressed as a percentage of mean daytime BP. Morning surge was defined as the morning BP (the 2-hour average of BP readings just after get-up) minus the lowest nighttime BP (the average of the lowest pressure and the 2 readings immediately preceding and after the lowest value).

Statistical Analysis
Means ± SDs and numbers (percentages) were calculated for continuous and categorical data, respectively. The subjects were divided into 5 groups according to their SD7: ≤7 hours, 7.01 to 8 hours, 8.01 to 9 hours, 9.01 to 10 hours, and >10 hours. Linear contrast test and linear-by-linear association tests were used to examine the linear trends across groups for continuous and dichotomous data, respectively. Log-transformation was used to convert data with nonnormal distribution to satisfy the assumption of normality. Pearson correlation analyses were used to assess the association between variables. Multiple linear regression analyses were used to examine the association between ABP measures and sleep quantity and quality while adjusting for age, gender, BMI z score, OAHI, and parental history of hypertension. All the analyses were performed by using the SPSS statistical software package, version 13.0 for Windows (SPSS for Windows, Chicago, SPSS Inc).

RESULTS
Subject Characteristics
A total of 162 normal-weight adolescents were recruited, of whom 19 with an OAHI ≥ 5 were excluded. All subjects had adequate and valid ABP readings...
for analysis. Among the remaining 143 subjects, 83 (58%) were boys and their mean ± SD age was 14.3 ± 1.8 years (range: 10.8–17.8 years). Demographic and anthropometric data for subjects with different SD7 are presented in Table 1. Groups with shorter SD7 consisted of a greater proportion of boys (P for linear trend = .012). No significant linear trends could be found in body size, sexual maturity rating, and parental history of hypertension.

**Sleep Pattern**

Groups of shorter SD7 exhibited later bedtime (P = .004) and earlier get-up time (P < .001) during the week before hospital admission. No significant difference in the proportion of subjects with caffeine use was found between groups. According to the polysomnography results, groups of shorter SD7 had shorter sleep latency (P = .001) and longer TST (P = .038). No significant between-group differences in sleep architecture, respiratory indexes, and arousal index were found (Table 2).

**ABP Measures**

Groups of shorter SD7 had higher 24-hour SBP and DBP and their z scores (all P for linear trend < .05) (Table 3). Pearson correlation analysis revealed that SD7 was inversely associated with both 24-hour SBP and DBP z scores (P < .001 and P = .008, respectively; Fig 2). No significant linear trends were found in nocturnal dipping and morning surge (Table 3). Groups of shorter SD7 had significantly higher SBP in prepolysomnography, in-bed, and postpolysomnography periods (all P for linear trend < .001). For DBP, readings for groups of shorter SD7 also tended to be higher in the prepolysomnography and in-bed periods, but the linear trend did not reach statistical significance (P = .066 and .079, respectively; Fig 3).

To examine the independent effects of TST, SE, and SD7 on ABP, multiple linear regression analyses were used to adjust for confounders including age, gender, BMI z score, OAHI, and presence of parental history of hypertension. Because TST was highly correlated with SE (r = 0.83, P < .001), they had to be assessed in 2 separate regression models. Model 1 examined TST and SD7 together, whereas model 2 tested for SE and SD7 simultaneously (Table 4). The results revealed that SD7 was inversely associated with SBP in prepolysomnography, in-bed, and postpolysomnography periods (all P < .01) as well as with DBP during the in-bed period in both model 1 (P = .017) and 2 (P = .007). It was also inversely associated with prepolysomnography DBP and positively associated with nocturnal dipping of SBP in model 2 (P = .041 and .044, respectively) but not in model 1 (P = .063 and .072, respectively). However, SD7 was not associated with DBP in the postpolysomnography period in either of the models (both P > .4). On the other hand, TST was inversely associated with SBP in the postpolysomnography period (P = .047). In addition, SE was significantly associated with SBP in the in-bed period (P = .038) and with DBP in the in-bed (P = .023) and postpolysomnography (P = .006) periods, as well as with morning DBP surge (P = .042). The association between SE and SBP in the postpolysomnography period was also close to significance (P = .058). Neither TST nor SE was associated with SBP and DBP in the prepolysomnography period (all P > .1) (Table 4).

**DISCUSSION**

This study investigated the independent relationship between sleep duration and BP by minimizing the confounding effects of obesity and OSA. The principal finding was that habitual short sleep duration recorded in the previous 7 days was independently associated

### TABLE 1 Anthropometric Data for Different Sleep Duration Groups

<table>
<thead>
<tr>
<th>Mean Sleep Duration Over the Past 7 Days</th>
<th>P (for linear trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤7 Hours (n = 18)</td>
<td>7.01–8 Hours (n = 37)</td>
</tr>
<tr>
<td>Age, y</td>
<td>15.0 ± 1.7</td>
</tr>
<tr>
<td>Male gender, n (%)</td>
<td>16 (89%)</td>
</tr>
<tr>
<td>Weight, kg</td>
<td>52.5 ± 8.3</td>
</tr>
<tr>
<td>Height, cm</td>
<td>163 ± 8</td>
</tr>
<tr>
<td>BMI, kg/m²</td>
<td>18.8 ± 2.1</td>
</tr>
<tr>
<td>BMI z score</td>
<td>0.26 ± 0.71</td>
</tr>
<tr>
<td>Waist circumference, cm</td>
<td>64.0 ± 6.9</td>
</tr>
<tr>
<td>Waist circumference</td>
<td>−0.33 ± 1.05</td>
</tr>
<tr>
<td>z score</td>
<td>4 ± 1</td>
</tr>
<tr>
<td>Sexual maturity rating</td>
<td>3 (17)</td>
</tr>
</tbody>
</table>

Continuous and dichotomous data are presented as means ± SDs and n (%), respectively. P values were obtained from linear contrast tests and linear-by-linear association tests for the comparisons of continuous and dichotomous data, respectively. HTN, hypertension.

* Parental HTN refers to either the father or mother or both were hypertensive or antihypertensive drug users.

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**FIGURE 1**

Example figure showing a bar chart with data points and error bars. The chart should be described in the text for accessibility. Below is a legend for the figure if needed: **Legend:** Bar1, Bar2, Bar3, Y-axis, X-axis, Title, Description.
with elevated ABP in normal-weight adolescents, especially in the pre-polysomnography and in-bed periods. An average of 1 hour of reduced sleep duration was associated with an increase of 2 mm Hg in SBP and 1 mm Hg in DBP. In addition, sleep duration and SE as recorded on polysomnography were independently associated with BP in the postpolysomnography period. Although the effect size was modest, a previous study revealed that a small difference in BP was associated with significant left ventricular abnormalities,21 which are important markers of future cardiovascular adverse events.32 Thus, our findings could have important long-term health implications.

### TABLE 3 ABP of Different Sleep Duration Groups

<table>
<thead>
<tr>
<th>Mean Sleep Duration Over the Past 7 Days</th>
<th>P (for linear trend)</th>
</tr>
</thead>
<tbody>
<tr>
<td>≤7 Hours (n = 18)</td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>116 ± 8</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>70 ± 5</td>
</tr>
<tr>
<td>SBP z score</td>
<td>0.09 ± 0.94</td>
</tr>
<tr>
<td>DBP z score</td>
<td>0.43 ± 0.83</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>9.7 ± 4.7</td>
</tr>
<tr>
<td>Morning surge, mm Hg</td>
<td>19.9 ± 8.1</td>
</tr>
<tr>
<td>≥7 Hours (n = 49)</td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>111 ± 8</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>68 ± 5</td>
</tr>
<tr>
<td>SBP z score</td>
<td>-0.38 ± 0.92</td>
</tr>
<tr>
<td>DBP z score</td>
<td>-0.25 ± 0.84</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>8.8 ± 6.1</td>
</tr>
<tr>
<td>Morning surge, mm Hg</td>
<td>16.8 ± 7.5</td>
</tr>
<tr>
<td>&gt;10 Hours (n = 26)</td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>109 ± 6</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>68 ± 4</td>
</tr>
<tr>
<td>SBP z score</td>
<td>-0.37 ± 0.85</td>
</tr>
<tr>
<td>DBP z score</td>
<td>-0.16 ± 0.73</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>10.2 ± 3.8</td>
</tr>
<tr>
<td>Morning surge, mm Hg</td>
<td>19.3 ± 6.4</td>
</tr>
<tr>
<td>≥10 Hours (n = 13)</td>
<td></td>
</tr>
<tr>
<td>SBP, mm Hg</td>
<td>106 ± 7</td>
</tr>
<tr>
<td>DBP, mm Hg</td>
<td>68 ± 4</td>
</tr>
<tr>
<td>SBP z score</td>
<td>-0.47 ± 0.81</td>
</tr>
<tr>
<td>DBP z score</td>
<td>-0.31 ± 0.70</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>8.0 ± 5.1</td>
</tr>
<tr>
<td>Morning surge, mm Hg</td>
<td>15.4 ± 6.4</td>
</tr>
</tbody>
</table>

Data are presented as means ± SDs. P values were obtained from linear contrast tests.
The link between short sleep duration and high BP may be explained by several mechanisms. Previous studies found that short sleepers had higher salivary cortisol response to awakening and higher salivary cortisol nadir when compared with average or long sleepers. In addition, another study showed that acute total sleep deprivation in healthy young adults blunted both the endothelium-dependent and -independent microvascular reactivities, as well as increased vascular endothelial adhesion markers. Furthermore, decreased endothelium-dependent venodilation was also observed in the partially sleep-deprived group, indicating impaired endothelial function. A more recent study found that 5 nights of partial sleep deprivation (≤5 hours) induced changes in heart rate and BP variability, and a prospective community study reported higher 24-hour urinary catecholamines in short sleepers, suggesting increased sympathetic activity as an important link.

From the literature, several studies investigating sleep duration and BP have reported conflicting results (Supplemental Table 5). Most published studies were limited by the use of a single question to assess sleep duration, use of spot BP measurements, and failure to control for OSA. OSA has been documented to lead to elevated BP in children and adolescents. Therefore, in assessing the relationship between sleep duration and BP, the OSA status has to be ascertained and controlled for. Most of the previous studies included both obese and nonobese subjects and analyzed the 2 groups together. Obesity is associated not only with BP levels but also with sleep duration and severity of sleep-disordered breathing. It could be difficult to eliminate its confounding effect simply by statistical means. The current study excluded overweight and obese adolescents to minimize the confounding effect of obesity.
In this study, subjects with an OAHI > 5 were excluded because there is evidence to support that children with more severe OSA have significantly higher cardiovascular risk than healthy controls and children with milder disease.20,37 Another merit of this study was that overnight polysomnography was performed on the same day as 24-hour ABPM, both immediately after completion of the 7-day sleep diary. This study design allowed us to separately delineate the relatively long-term (7 days) and acute effects of sleep duration on BP. By standardizing the start time (21:30 ± 30 minutes) and end time (07:00 ± 30 minutes) of polysomnography recording to provide a fixed time in bed of 9.5 hours ± 5 minutes for all subjects, acute changes in their sleep pattern could be demonstrated. Our results revealed that subjects with shorter SD7 had shorter sleep latency and longer TST as reflected in their polysomnography results. This could be considered as a sign of sleep rebound from sleep deprivation in the preceding week. By analyzing the ABP data in prepolysomnography, in-bed, and postpolysomnography periods separately, it was found that, in general, SD7 was more closely related to ABP measured before and during polysomnography, whereas TST and SE as obtained from polysomnography were more closely associated with ABP during and after polysomnography. The latter was especially true for DBP. These findings suggested that BP was sensitive to acute changes in sleep pattern, meaning that BP level was influenced by the sleep quantity and quality in the previous night. However, it did not imply that 1 night of adequate sleep could compensate for the adverse effect of accumulated sleep debt. Our results revealed that SD7 was not only associated with SBP in prepolysomnography and in-bed periods but also in the postpolysomnography period. This finding suggested that there was a carryover effect of short sleep duration accumulated in the previous days on SBP, and the effect was not totally cancelled out by 1 night of “compensated” sleep. This finding has implications for weekend sleep compensation, which is a phenomenon commonly adopted by adolescents. Similar to our previous finding that weekend compensation may partially ameliorate the effect of short sleep duration on obesity,2 we demonstrated that similar sleep compensation may modulate BP changes. Thus, our current study supported that weekend compensation may have some beneficial effect on BP, but the effect of long-term short sleep duration on BP could not be completely eliminated.

The major limitation of this study was that prepolysomnography sleep was only recorded subjectively by sleep diaries but not objectively by actigraphy. Sleep diaries may be subject to reporting error that may over- or underestimate sleep duration. However, several studies revealed that actigraphy may also lead to errors by overestimating the duration of wake after sleep onset and underestimate TST, when compared with polysomnography and sleep diary.58-60 There is evidence that subjective and objective sleep variables in adolescents, including sleep start time, sleep end time, and sleep duration, were highly correlated.41 Our previous study also reported good agreement between actigraphy-measured and subjectively reported sleep duration (intraclass correlation coefficient = 0.72).3 Polysomnography and ABPM were performed only at a single time point, which hindered a better understanding of the association between changes in sleep duration, sleep architecture, and BP. Moreover, performing polysomnography and ABPM on the same day may affect the results obtained from either assessment. Nevertheless, having the 2 assessments on the same day provided us an opportunity to delineate the

### Table 4: Association Between ABP and Sleep Quantity and Quality

<table>
<thead>
<tr>
<th>Dependent Variable</th>
<th>Model 1</th>
<th>Model 2</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>SD7 (hours)</td>
<td>P</td>
</tr>
<tr>
<td>SBP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prepolysomnography period</td>
<td>-1.6 (-2.7 to -0.5)</td>
<td>0.005</td>
</tr>
<tr>
<td>In-bed period</td>
<td>-2.0 (-3.1 to -1.0)</td>
<td>&lt;0.01</td>
</tr>
<tr>
<td>Postpolysomnography period</td>
<td>-1.6 (-2.7 to -0.4)</td>
<td>0.007</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>0.7 (-0.1 to 1.4)</td>
<td>0.07</td>
</tr>
<tr>
<td>Morning surge</td>
<td>0.5 (-0.7 to 1.8)</td>
<td>0.39</td>
</tr>
<tr>
<td>DBP (mm Hg)</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Prepolysomnography period</td>
<td>-0.8 (-1.6 to 0.04)</td>
<td>0.06</td>
</tr>
<tr>
<td>In-bed period</td>
<td>-1.1 (-2.0 to -0.2)</td>
<td>0.02</td>
</tr>
<tr>
<td>Postpolysomnography period</td>
<td>-0.2 (-1.1 to 0.8)</td>
<td>0.76</td>
</tr>
<tr>
<td>Nocturnal dipping, %</td>
<td>0.8 (-0.3 to 1.9)</td>
<td>0.14</td>
</tr>
<tr>
<td>Morning surge</td>
<td>1.0 (-0.2 to 2.2)</td>
<td>0.10</td>
</tr>
</tbody>
</table>

Data are presented as β (95% confidence interval).

* Independent variables included age, gender, BMI z score, OAHI, parental history of hypertension, TST measured on polysomnography, and sleep duration recorded by sleep diary.

† Independent variables included age, gender, BMI z score, OAHI, parental history of hypertension, SE measured on polysomnography, and sleep duration recorded by sleep diary.
chronic and acute effects of sleep quantity and quality on BP separately.

**CONCLUSIONS**

On the basis of a sample of normal-weight adolescents without any significant sleep-disordered breathing, this study revealed that short sleep duration and poor sleep quality adversely affected ABP. Despite a small effect size, our findings have important long-term cardiovascular health implications. Intermitting adequate sleep on weekends may partially ameliorate the risk of high BP but could not completely reverse the effect of chronic sleep deprivation. Longitudinal and interventional studies are warranted to provide further evidence for the causal effect of sleep quantity and quality on BP.

**REFERENCES**


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