Published in final edited form as:
J Pediatr. 2012 July ; 161(1): 26–30. doi:10.1016/j.jpeds.2011.12.034.

Effects of sleep patterns and obesity on increases in blood pressure over a 5-year period: Report from the Tucson Childrens Assessment of Sleep Apnea Study

Kristen Hedger Archbold, RN, PhD[1], **Monica M. Vasquez, MPH**[2], **James L. Goodwin, PhD**[2], and **Stuart F. Quan, MD**[2,3]

[1]College of Nursing, Department of Biobehavioral Health Sciences, University of Arizona, Tucson, AZ

[2]Arizona Respiratory Center, College of Medicine, University of Arizona, Tucson, AZ

[3]Division of Sleep Medicine, Brigham and Women's Hospital and Harvard Medical School, Boston, MA

Abstract

Objectives—To determine associations between body mass index (BMI) and sleep on blood pressure over a 5-year period from childhood to adolescence.

Study design—A longitudinal, community-based sample of 334 children recruited at ages 6 through 11 years. Each participant underwent in-home polysomnography initially and then 5 years later. Individual systolic (SBP) and diastolic (DBP) blood pressures were calculated at both time points during wake periods and classified as hypertensive if SBP or DBP was ≥ 95th standardized percentiles for height and weight.

Results—Hypertension was present in 3.6% of the sample at time one and increased to 4.2% 5-years later. Obesity prevalence increased from 15.0% to 19.5%. Normal changes in sleep architecture were observed in the sample. Random effects modeling which controlled for age, sex and ethnicity indicated that change in obesity status and decrease in total sleep time were associated with increases in SBP. Change in obesity status was also associated with increases in DBP over the 5-year period. A trend for sleep-disordered breathing to increase SBP was noted.

Conclusions—Increases in SBP and DBP were associated with increasing BMI and decreased total sleep time over a 5-year period from childhood to adolescence.

Elevated blood pressure in childhood is known to be a risk factor for hypertension and cardiac disease in adulthood, but few longitudinal data exist to explicate the causal onset of elevated blood pressure in children(1). Modifiable factors known to contribute to the development of elevated blood pressures in childhood include dietary habits, obesity and sedentary lifestyle, but little is known about the contribution of sleep patterns to the development of blood pressure elevation in adolescent children.

The authors declare no conflicts of interest.

In adults, several cross sectional and prospective population-based cohort studies have linked sleep disordered breathing (SDB) with elevated blood pressure and cardiovascular disease (2, 3). Although obesity is a known risk factor for hypertension in adults, most, but not all of these studies indicate that SDB is an independent risk factor for the development of hypertension as well (2). Whether SDB is a risk factor for the development of elevated blood pressure in children is less clear. Several studies have demonstrated a linkage between the two conditions, however, to date, most of these studies have been performed in clinical populations and all have been cross-sectional. Furthermore, although reduced sleep duration has been linked to development of cardiovascular disease in adults(4), the effects of a reduction in sleep duration on heart health in children are not known.

The Tucson Childrens Assessment of Sleep Apnea Study (TuCASA) is a longitudinal cohort study whereby preadolescent children ages 6-11 years initially had in-home polysomnography, anthropometric and blood pressure assessments with follow up measurements repeated approximately 5 years later. Analyses of these data provide the opportunity to understand the relationships between sleep patterns, SDB, blood pressure and obesity in a general population cohort of children. We hypothesize that SDB, reduced sleep duration and obesity are independent risk factors for elevations in blood pressure in children.

METHODS

Details of the TuCASA study design have been published previously(5, 6). Briefly Hispanic and Caucasian children 6 to 11 years were recruited to undergo unattended home polysomnography and to have a neurocognitive assessment performed. Subjects were recruited through the Tucson Unified School District (TUSD) which has a large elementary school population. Parents were asked to complete a short screening questionnaire and to provide their contact information if they were willing to allow study personnel to contact them to determine if their child was eligible for the study. A total of 7,055 screening questionnaires were sent home with children in a “notes home” folder. Of these, 2,327 (33%) were returned. Recruitment information was supplied on 52% of the returned questionnaires from which we selected children, based on pre-established inclusion and exclusion criteria, to undergo polysomnography. An unattended home polysomnogram was scheduled as soon as possible after recruitment. From 1999-2004, 503 children aged 6-11 years completed home polysomnograms (Baseline). Approximately five years later (follow up, mean 4.7 years), 348 children participated in the second phase of the study; 319 children had home visits where acceptable in-home polysomnography was completed a second time. For blood pressure readings, there were 334 children who had BP recorded at both time 1 and time 2. On both occasions, all of the families completed sleep screening, sleep habits, and morning questionnaires. The TuCASA study was approved by the University of Arizona Institutional Review Board as well as the TUSD Research Committee.

The methods for obtaining data have been previously described(6). In brief, for both the initial and follow up assessments, a two person team arrived at the home approximately one hour prior to the child’s normal bedtime. Prior to performing any study procedures, parents gave informed consent and the child gave assent to the study using language appropriate IRB forms. Each child’s height, weight, neck circumference, and blood pressure were measured. A parent was asked to complete a comprehensive Sleep Habits Questionnaire (SHQ) that inquired about their child’s sleep history and sleep characteristics.

After a few minutes of rest while seated, the child’s BP was measured in triplicate from the right arm using a portable mercury sphygmomanometer and standardized techniques. The appropriate BP cuff was selected according to the measured arm size (upper arm circumferences of 16-22 cm for children, and 23-30 cm for regular-sized adults). The initial

cuff inflation pressure was determined by adding 30 mm Hg to the palpated systolic BP. Cuff deflation was at 2 mm/second. At least 30 seconds elapsed between each of the 3 successive measurements. The mean of the final 2 of 3 BP measurements was used for the analyses for this report. We defined hypertension as blood pressure in the 95th percentile or greater for age, height and sex, or a systolic level of > 120 mm/Hg or a diastolic value of > 80 mm/Hg.

Height and weight were collected on a platform scale. BMI was calculated according to a standardized equation from the Centers for Disease Control, and percentile of BMI adjusted for age, sex and ethnicity was calculated with a standardized data-analysis program (http://www.cdc.gov/nccdphp/dnpao/growthcharts/resources/sas.htm). Obesity was considered to be A single, unattended overnight polysomnogram was obtained using the Compumedics PS-2 system (Abbotsford, Victoria, Austrailia). The following signals were acquired as part of the TuCASA montage: C3/A2, C4/A1 electroencephalogram, right and left electrooculogram, a bipolar submental electromyogram, thoracic and abdominal displacement (inductive plethysmography), airflow (nasal/oral thermistor), nasal pressure cannula, finger pulse oximetry, ECG (single bipolar lead), snoring microphone, body position (Hg gauge sensor), and ambient light (sensor attached to the vest to record on/off). The sleep architecture variables considered in this study include respiratory disturbance index (RDI), Stage N1, Stage N2, Stage N3 (combined Rechtschaffen and Kales Stages 3 and 4), Stage REM, sleep efficiency, and total sleep time.

Scoring of sleep was performed by a single registered polysomnographic technologist using Rechtschaffen and Kales criteria. Arousals were identified using criteria published by the American Academy of Sleep Medicine. Apneas were scored if the amplitude (peak to trough) of the airflow signal using the thermistor decreased below at least 25% of the amplitude of baseline breathing (identified during a period of regular breathing with stable oxygen levels), if this change lasted for more than 6 seconds or 2 breathing cycles. Hypopneas were designated if the amplitude of any respiratory signal decreased below (approximately) 70% of the amplitude of baseline and if the thermistor signal did not meet the criterion for apnea. Central events were marked if no displacement was noted on both the chest and abdominal inductance channels. However, central events that occurred after movement were not included. Otherwise, events were scored as obstructive. After full scoring, analysis software was used to link each event to data from the oxygen saturation and EEG channels. The Respiratory Disturbance Index (RDI) was defined as the number of respiratory events (apneas and hypopneas) per hour of the total sleep time. For this analysis, a 3% oxygen desaturation was required for an event to be counted in the total RDI. We considered a child to have SDB if their RDI was greater than or equal to 1 event per hour of total sleep time. Use of this definition is supported by previous evidence that a RDI of one, based on events with a 3% oxygen desaturation, is clinically significant(7).

Data Analysis

Student paired t-test and the 2-sample test of proportion were conducted to compare differences in characteristics from study baseline to study follow-up. Characteristics of interest include ethnicity, sex, age, body mass index (BMI) percentile, blood pressure, and sleep architecture. Blood pressure was classified as systolic blood pressure (SBP), diastolic blood pressure (DBP), and was also used as a dichotomous variable to indicate the hypertensive state of the study subject. A random effects model was then used to investigate which characteristics were associated with a steeper increase or decrease of blood pressure over time. The random effects model accounts for the correlation of repeated measures taken on the same subject over time. It also allows for subject-specific intercepts and slopes measured for each individual subject. Models for SBP and DBP were calculated separately. Independent variables included in the models were SDB, obesity, and total sleep time. The

variables sex and ethnicity were included in the fully-adjusted model, and both models were adjusted for age.

RESULTS

The sample consisted of 334 children, 163 (48.8%) of whom were female, and 35.6% of whom were Hispanic. The average age was 9.0 ± 1.6 years at baseline and 13.7 ± 1.8 years at follow-up. Average systolic and diastolic blood pressures increased significantly from baseline to follow-up. At baseline, 15.0% of the sample was considered obese which increased to 19.5% at follow-up. In comparison with Caucasians, the prevalence of obesity was higher in Hispanics at both baseline and follow up: 11.2% vs. 21.9% ($p= 0.009$) and 13.6% vs. 29.4% ($p < 0.001$), respectively. Hypertension was found in 12 of 334 children (3.6%) at baseline and increased to 14 children (4.2%) 5-years later (p=NS) (Table I).

From year 1 to year 5, % time spent in stages N3 and sleep efficiency declined and % time spent in rapid eye movement (REM) sleep increased (all $p<0.01$). The average RDI at baseline for the sample was 1.05 and decreased significantly to 0.59 by year 5 (Table II).

Incident and prevalent hypertension at 5-year follow-up were not related to changes in sleep architecture, changes in SDB or obesity (data not shown). However, when adjusted for age, random effects modeling of the independent effects of SDB, obesity, total sleep time on systolic and diastolic blood pressure levels showed that systolic blood pressure levels at 5-year follow-up were positively associated with an increase in SDB and obesity and a decrease of total sleep time. Furthermore, there was a significant association between the presence of SDB and obesity on diastolic blood pressure levels (Model 1, Table III). When adjusted for age, ethnicity and sex, obesity was positively associated with an increase in systolic blood pressures, as was decreased total sleep time. Within the fully adjusted model, ethnicity and sex had no significant effect on either systolic or diastolic blood pressure levels and the presence of SDB became non-significant in its effect on systolic and diastolic blood pressures although a trend remained for an effect of SDB on SBP (Model 2, Table III).

DISCUSSION

In our cohort of 334 children followed over a period of 5 years from childhood to adolescence, we found a small, but non-significant increase in the prevalence of hypertension from 3.6% to 4.2%. However, the prevalence of obesity increased from 15% of the sample in childhood to 19% of the sample at adolescence. Most importantly, over the 5 year follow-up, increases in BMI and reductions in total sleep time were associated with higher levels of blood pressure in adolescents with a trend for an effect from SDB. These are important findings because not all obese children have SDB, but obese children who decrease their total sleep time as they age may be more likely to experience increases in systolic and diastolic blood pressures which could potentially lead to the development of frank hypertension in adulthood.

Our study found the prevalence of obesity increased from childhood to adolescence, and that increases in BMI were associated with higher levels of both systolic and diastolic blood pressure. Obesity is well-known to be associated with hypertension in adults and in children, and the findings of this study are not surprising from that perspective (8-12). However, there are few previous longitudinal studies that have demonstrated this relationship. In that general endothelial dysfunction begins early in life in obese children(13), our data add to the accumulating evidence that obese children should undergo routine blood pressure screenings throughout childhood development and also be made the focus of intensive lifestyle

modification programs in order to reduce susceptibility to hypertension and other morbidity in adulthood.

Although the prevalence of obesity was greater in Hispanics in our study, Hispanic ethnicity did not appear to be a significant factor in the elevation of systolic or diastolic blood pressure levels from childhood to adolescence. However, previous cross-sectional studies do illustrate clear ethnic differences with Hispanic and African American youth at greater risk for the development of obesity and hypertension(14). McCarthy et al(14) reported that in a sample of 199 inner-city Hispanic and African American children, higher BMI was associated with increased systolic and diastolic blood pressure. In addition, Sanchez-Zamorano(15) found that increased BMI was associated with increases in systolic and diastolic blood pressure in a sample of Mexican adolescents(15).

Although cross-sectional data exist to suggest a link between poor sleep quality and prehypertension in childhood and adolescence(16, 17), Bayer et al reported that blood pressure increases were not affected by total sleep time in adolescents(18). However, results from the current longitudinal study suggest that the relationship between total sleep time and elevations in blood pressure may indeed be present but at a more insidious level whereby decreased sleep time over a 5-year period elevates systolic but not diastolic blood pressure in the presence of an increasing BMI. This is consistent with observations indicating that decreased total sleep time is associated with increase in blood pressure and the presence of obesity in adults aged 40-55(19-22).

As a society, total sleep time is less than optimal for all age groups, but particularly so for children and adolescents. The recommended period of time for sleep in childhood is approximately 9.5 to 10 hours(5, 23), and children in this sample experienced approximately 8 hours decreasing to 7 hours of total sleep over a 5-year period. This shorter period of total sleep time was a significant contribution to elevated systolic blood pressure at 5-year follow-up in our study. Thus, our data reinforce the concept that more attention must be paid to the negative physiological effects of less than optimal sleep periods during childhood. Specifically, more information is needed concerning the physiologic impact of early school start times and late night use of electronic media, both of which negatively affect sleep duration.

In our current analyses, there was no significant relationship between the development of hypertension and either prevalent or incident SDB. Previous studies including a cross-sectional analysis of the TuCASA cohort during childhood have observed an association between SDB and hypertension. However, in our cohort, the overall prevalence of SDB was low, and mean RDI in the cohort decreased over the 5 year follow-up period. It is likely that a cohort with greater numbers of children with SDB and hypertension would be needed to better define this relationship. Nevertheless, our observation of a trend for increasing RDI to be associated with higher systolic blood pressures in adolescents suggests that SDB may yet prove to be an independent risk factor for incident hypertension in adolescents.

Limitations of the current study include the lack of racial and ethnic groups other than Hispanics and Caucasians and the loss of cohort members over the 5 year follow-up period, thus reducing the power to potentially detect the impact of BMI, sleep patterns and SDB on blood pressure levels, and frank hypertension. In addition, we measured BP at only 2 time points. It is possible that BP was artifactually elevated in some children due to the novelty and/or apprehension of having the procedure performed. However, such effects would be random over the entire cohort and unlikely to produce a systematic error. Another potential limitation is the use of PSG measured total sleep time with the attendant potential for “first night effect” and underestimation of sleep. However, most children slept quite well during

their PSGs, and any “first night effect” would be random across the cohort. Nevertheless, these limitations are counterbalanced by the ability to perform longitudinal analyses, and objective measures of sleep architecture and sleep duration.

In conclusion, this study found that increases in BP from childhood to adolescence were related to increasing BMI and reduction in sleep. A trend for an effect of SDB on increased BP was noted as well. These findings suggest mechanistic links explaining associations between childhood obesity and higher adult cardiovascular mortality and premature death(24).

Reference List

1. Falkner B, Gidding SS, Portman R, Rosner B. Blood pressure variability and classification of prehypertension and hypertension in adolescence. Pediatrics. 2008; 122:238–42. [PubMed: 18676538]
2. Punjabi NM, Caffo BS, Goodwin JL, Gottlieb DJ, Newman AB, O’Connor GT, et al. Sleep-Disordered Breathing and Mortality: A Prospective Cohort Study. PloS Med. 2009; 6:e1000132. [PubMed: 19688045]
3. Korcarz CE, Gepner AD, Peppard PE, Young TB, Stein JH. The Effects of Sleep-Disordered Breathing on Arterial Stiffness are Modulated by Age. Sleep. 2010; 33:1081–5. [PubMed: 20815190]
4. Sabanayagam C, Shankar A. Sleep Duration and Cardiovascular Disease: Results from the National Health Interview Survey. Sleep. 2010; 33:1037–42. [PubMed: 20815184]
5. Quan SF, Goodwin JL, Babar SI, Kaemingk KL, Enright PL, Rosen GM, et al. Sleep architecture in normal Caucasian and Hispanic children aged 6-11 years recorded during unattended home polysomnography: experience from the Tucson Children’s Assessment of Sleep Apnea Study (TuCASA). Sleep Med. 2003; 4:13–9. [PubMed: 14592355]
6. Goodwin JL, Morgan WJ, Kaemingk KL, Quan SF. Clinical presentation of sleep disordered breathing in 6-11 year old children: The Tucson Children’s Assessment of Sleep Apnea Study (TuCASA). Sleep. 2004; 27:102–3.
7. Zhao Q, Sherrill DL, Goodwin JL, Quan SF. Association Between Sleep Disordered Breathing and Behavior in School-Aged Children: The Tucson Children’s Assessment of Sleep Apnea Study. Open Epidemiol J. 2008; 1:1–9. [PubMed: 18568089]
8. Paradis G, Lambert M, O’Loughlin J, Lavallee C, Aubin J, Delvin E, et al. Blood pressure and adiposity in children and adolescents. Circulation. 2004; 110:1832–8. [PubMed: 15381642]
9. Adams MH, Carter TM, Lammon CA, Judd AH, Leeper J, Wheat JR. Obesity and blood pressure trends in rural adolescents over a decade. Pediatr Nurs. 2008; 34:381–6. 94. [PubMed: 19051841]
10. Kawabe H, Shibata H, Hirose H, Tsujioka M, Saito I, Saruta T. Determinants for the development of hypertension in adolescents. A 6-year follow-up. J Hypertens. 2000; 18:1557–61. [PubMed: 11081767]
11. Puri M, Flynn JT, Garcia M, Nussbaum H, Freeman K, DiMartino-Nardi JR. The frequency of elevated blood pressure in obese minority youth. J Clin Hypertens (Greenwich, Conn.). 2008; 10:119–24.
12. Rosa ML, Mesquita ET, da Rocha ER, Vde M Fonseca. Body mass index and waist circumference as markers of arterial hypertension in adolescents. Arq Bras Cardiol. 2007; 88:573–8. [PubMed: 17589633]
13. Bhattacharjee R, Alotaibi WH, Kheirandish-Gozal L, Capdevila OS, Gozal D. Endothelial dysfunction in obese non-hypertensive children without evidence of sleep disordered breathing. BMC Pediatr. 2010; 10:8. [PubMed: 20156343]
14. McCarthy WJ, Yancey AK, Siegel JM, Wong WK, Ward A, Leslie J, et al. Correlation of obesity with elevated blood pressure among racial/ethnic minority children in two Los Angeles middle schools. Prev Chronic Dis. 2008; 5:A46. [PubMed: 18341781]

15. Sanchez-Zamorano LM, Salazar-Martinez E, Anaya-Ocampo R, Lazcano-Ponce E. Body mass index associated with elevated blood pressure in Mexican school-aged adolescents. Prev Med. 2009; 48:543–8. [PubMed: 19286003]
16. Javaheri S, Storfer-Isser A, Rosen CL, Redline S. Sleep quality and elevated blood pressure in adolescents. Circulation. 2008; 118:1034–40. [PubMed: 18711015]
17. Sampei M, Dakeishi M, Wood DC, Murata K. Impact of total sleep duration on blood pressure in preschool children. Biomed Res (Tokyo, Japan). 2006; 27:111–5.
18. Bayer O, Neuhauser H, von Kries R. Sleep duration and blood pressure in children: a cross-sectional study. J Hypertens. 2009; 27:1789–93. [PubMed: 19633568]
19. Bjorvatn B, Sagen IM, Oyane N, Waage S, Fetveit A, Pallesen S, et al. The association between sleep duration, body mass index and metabolic measures in the Hordaland Health Study. J Sleep Res. 2007; 16:66–76. [PubMed: 17309765]
20. Friedman O, Shukla Y, Logan AG. Relationship between self-reported sleep duration and changes in circadian blood pressure. Am J Hypertens. 2009; 22:1205–11. [PubMed: 19745817]
21. Kim J, Jo I. Age-Dependent Association Between Sleep Duration and Hypertension in the Adult Korean Population. Am J Hypertens. 2010; 23:1286–91. [PubMed: 20706198]
22. Kotani K, Saiga K, Sakane N, Mu H, Kurozawa Y. Sleep status and blood pressure in a healthy normotensive female population. Int J Cardiol. 2008; 125:425–7. [PubMed: 17391783]
23. Ohayon M, Carskadon M, Guilleminault C, Vitiello WV. Evolution of PSG sleep parameters with age. Sleep. 2004; 27:134–5. [PubMed: 14998250]
24. Franks PW, Hanson RL, Knowler WC, Sievers ML, Bennett PH, Looker HC. Childhood obesity, other cardiovascular risk factors, and premature death. N Engl J Med. 2010; 362:485–93. [PubMed: 20147714]

J Pediatr. Author manuscript; available in PMC 2013 July 01.

Table 1

Average Blood Pressures and Demographic Data for Sample

	Baseline			Follow-Up			
	N	**Mean ± SD**	**Range**	**N**	**Mean ± SD**	**Range**	**p-value**
Systolic Blood Pressure	334	98.94 ± 9.37	60-142	334	105.57 ± 8.81	89-137	<0.01
Diastolic Blood Pressure	334	60.63 ± 9.24	30-90	334	63.45 ± 7.73	48-88	<0.01
BMI Percentile	334	59.98 ± 31.44	0-99.96	333	63.32 ± 30.75	0.01-99.72	0.14
Age	334	9.03 ± 1.63	6.2-12.6	334	13.72 ± 1.79	10.2-18.3	--
		No.	**%**		**No.**	**%**	
Ethnicity	334			334			--
Hispanic		119	35.63%		119	35.63%	
Caucasian		215	64.37%		215	64.37%	
Sex	334			334			--
Male		171	51.20%		171	51.20%	
Female		163	48.80%		163	48.80%	
Obesity	334			333			0.12
Yes		50	14.97%		65	19.52%	
No		284	85.03%		268	80.48%	
Hypertension	334			334			0.69
Yes		12	3.59%		14		4.19%
No		322	96.41%		320	95.81%	

Table 2

Sleep Architecture Characteristics from Baseline to Follow-Up (5-year period N=319)

	Baseline Mean (±SD)	**Follow-up Mean (±SD)**	**p-value**
Stage N1 (% of total sleep time TST)	4.30 (±3.3)	3.96 (±2.3)	0.09
Stage N2 (%TST)	54.3 (±11.1)	55.4 (±6.9)	0.12
Stage N3 (%TST)	21.3 (±8.1)	18.5 (±6.7)	< **0.01**
Stage REM (%TST)	20.1 (±6.4)	22.7 (±4.7)	< **0.01**
RDI	1.05 (±2.3)	0.59 (±1.9)	< **0.01**
Sleep Efficiency (%)	90.2 (±5.2)	87.14 (±7.3)	< **0.01**
Total Sleep Time (minutes)	481.46 (±91.8)	470.96 (±64.3)	0.07

Table 3

Random Effects Model for Blood Pressure and Longitudinal Covariates (N=334)

	SYSTOLIC BLOOD PRESSURE			DIASTOLIC BLOOD PRESSURE		
	Coefficients	**SE**	**p-value**	**Coefficients**	**SE**	**p-value**
Model 1[a]						
Sleep Disordered Breathing	1.967	0.788	**0.013**	1.693	0.802	**0.035**
Obesity	4.631	0.883	<**0.001**	4.475	0.850	<**0.001**
Total Sleep Time	−0.009	0.004	**0.027**	−0.007	0.004	0.105
Model 2[b]						
Sleep Disordered Breathing	1.387	0.786	0.078	0.971	0.804	0.227
Obesity	4.275	0.901	<**0.001**	4.341	0.881	<**0.001**
Total Sleep Time	−0.008	0.004	**0.042**	−0.006	0.004	0.144
Age at PSG	1.557	0.103	<**0.001**	0.768	0.113	<**0.001**
Ethnicity (Hispanic)	0.036	0.767	0.963	−0.311	0.680	0.647
Sex (Female)	−0.589	0.730	0.420	0.243	0.647	0.708

[a] Independent Effects, each adjusted for age at PSG

[b] Fully Adjusted Model

J Pediatr. Author manuscript; available in PMC 2013 July 01.