Sleep Duration, Quality, or Stability and Obesity in an Urban Family Medicine Center

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Purpose: Inadequate sleep has negative metabolic consequences that may contribute to obesity. A priori hypotheses posit relationships between sleep characteristics, carbohydrate and lipid metabolism, appetite, fatigue, and obesity in laboratory, clinical, and population settings. There are few reports from primary care; and none that address sleep duration, quality, and stability. This study examines the relationship between three sleep characteristics—duration, quality, or stability—and obesity in our urban hospital affiliated family medicine center in Akron, Ohio.

Methods: A systematic sampling process yielded 225 representative patients who completed the Pittsburgh Sleep Quality Index, the Berlin Apnea Questionnaire, and the Sleep Timing Questionnaire. Demographic, body mass, hypertension, and insurance data were obtained from the electronic medical record. Associations between self-reported sleep characteristics and obesity were examined via contingency tables and regression models.

Results: Seventy-eight percent (78%) reported poor quality sleep, 59% had elevated Berlin apnea-risk scores, 12% reported restless legs symptoms, and 9% reported a prior

diagnosis of sleep apnea; 62% were obese. We found significant ($p < 0.05$) associations between sleep quality, duration or bedtime stability, and obesity. The association between sleep quality and obesity was negative and linear (69%, 72%, 56%, 43%), while the association between sleep duration and obesity was U-shaped (74%, 53%, 53%, 62%; linear term $p = 0.02$ and quadratic term $p = 0.03$). Less stable bedtimes during the week ($OR = 2.3$, $p = 0.008$) or on the weekend ($OR = 1.8$, $p = 0.04$) were also associated with obesity. The association between sleep quality and obesity was not explained by patient demographics or snoring ($OR_{adj} = 2.2$; $p = 0.008$).

Conclusion: This study adds to the sparse literature on the relationship between three self-reported sleep characteristics and obesity in urban primary care settings which typically differ from both general population and specialty outpatient settings.

Keywords: Sleep quality, sleep duration, sleep timing, obesity, primary care

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Over the past 40 years, there have been dramatic increases in the prevalence of obesity throughout the United States.¹ During the same period, there have been increases in the prevalence of voluntary sleep restriction, sleeplessness, and poor quality sleep.²⁻⁴ Since there are physiologic linkages between the “sleep system” and the “metabolic system,” and a variety of supporting circumstantial data, investigators have hypothesized that inadequate sleep contributes to the obesity epidemic by increasing stress, appetite, and fatigue.^{5,6} The hypothesized inadequate sleep-to-obesity pathway is distinct from the more familiar obesity-to-sleep apnea pathway.^{7,8} Circumstantial data supporting the inadequate sleep-to-obesity hypothesis include information from laboratory, clinical, and population settings.⁸⁻¹⁰ However, there are only a few relevant reports from primary care.¹¹⁻¹³ This cross-sectional study examines the relationship between sleep duration, quality, or stability and obesity in our urban hospital affiliated family medicine center.

METHODS

The target population and sampling frame for the study was all adult outpatients seen at our family medicine center during the 10 weeks that a student assistant was available. Patients

BRIEF SUMMARY

Current Knowledge/Study Rationale: A variety of observational and experimental data suggest that there are etiologic linkages between inadequate sleep and consequent obesity in the general population. However, there are few reports describing these linked phenomena in primary care, where both inadequate sleep and obesity are likely to be prevalent and overlooked.

Study Impact: Data from prior cross-sectional studies in primary care may be limited by selection and measurement issues. Stronger methods and relevant results in this study argue for more attention to the sleep-to-obesity hypothesis in primary care and elsewhere.

were selected for inclusion using a pseudo-random sampling method.¹⁴ The research assistant selected a patient according to time of the scheduled appointment (15 min or 45 min past the hour on Tuesdays and Thursdays, and on the hour or half hour on Mondays, Wednesdays, and Fridays). If more than one patient shared an appointment time, the research assistant chose the person to be interviewed based on the position of their last name in the alphabet. Two hundred twenty-five patients completed the IRB-approved consent process and a sleep habits interview using validated questionnaires. The characteristics of the patients in the study sample were compared with the

Table 1—Population and sample characteristics

Characteristic	Population Parameter (N = 1,964) Percentage	Sample Estimate* (n = 225) Percentage	95% Confidence Interval for the Sample Estimate* (95% CI)
Age group			
18-39	42.4	48.0	(41.6, 54.5)
40-64	44.2	41.9	(35.5, 48.3)
65-84	12.0	10.5	(6.5, 14.4)
85+	1.4	1.3	(-0.2, 2.8)
Gender			
Females	71.2	73.8	(68.1, 79.5)
Males	28.8	26.2	(20.5, 31.9)
Race/ethnicity			
European-American	53.3	55.9	(49.5, 62.3)
African-American	41.3	40.2	(33.8, 46.5)
Other	5.4	4.8	(2.0, 7.6)
Marital Status			
Married	24.5	25.3	(19.7, 31.0)
Divorced/legally separated	10.7	8.3	(4.7, 11.9)
Widowed	2.6	3.5	(1.1, 5.9)
Single & unknown	62.3	64.6	(58.4, 70.8)
BMI group (kg/m ² interval)			
Underweight (< 18)	1.0	1.4	(-0.2, 3.0)
Normal (18-24)	20.1	14.6	(9.8, 19.3) [†]
Overweight (25-29)	26.2	26.3	(20.4, 32.2)
1° obesity (30-34)	21.0	20.2	(14.8, 25.6)
2° obesity (35-39)	14.7	19.7	(14.4, 25.1)
3° obesity (40+)	16.9	21.6	(16.1, 27.1)
Hypertension			
Yes	40.3	38.9	(32.6, 45.2)
No	59.7	61.1	(54.8, 67.4)
Insurance type			
Medicaid	43.2	40.6	(34.3, 47.0)
Commercial	18.2	18.3	(13.3, 23.4)
Medicare	20.1	17.9	(12.9, 22.9)
Organized charity	12.5	18.8	(13.7, 23.8) [‡]
No insurance	6.1	7.0	(3.7, 10.3)

*Adjusted Wald estimates. [†]Normal approximation to binomial $p = 0.02$; exact binomial $p = 0.49$. [‡]Normal approximation to binomial $p = 0.01$; exact binomial $p = 0.33$.

characteristics of the population of all adult patients (N = 1964) who had an office visit during the recruiting period. Ninety-five percent confidence intervals and goodness of fit tests were used to quantify the likely impact of random error on the sample characteristics.¹⁴

Sleep habits were assessed by the student assistant using the Sleep Timing Questionnaire (STQ), the Pittsburgh Sleep Quality Index (PSQI), the Berlin Apnea Questionnaire (BAQ), and a standard restless legs syndrome (RLS) screening item.¹⁵⁻¹⁸ The STQ reliably simulates a 2-week sleep diary.¹⁵ Bedtimes were “stable” if they occurred within 31 min of one another according to STQ response categories (0-15 or 16-30 min). The PSQI has acceptable internal homogeneity, test-retest reliability, and validity for clinical practice and research.¹⁶ The BAQ has sensitivity of 0.86, a specificity of 0.77, and a positive predictive value of 0.89 for an elevated respiratory distress index (RDI > 5) within primary care settings.¹⁷ The RLS screening item has a sensitivity of 100% and specificity of 97% for clinical RLS according to the 4 International Restless Legs

Syndrome Study Group criteria.^{18,19} Demographic data, recent heights and weights, or body mass indices (BMI), International Classification of Diseases, 9th Revision codes for hypertension (for the BAQ), and insurance type were downloaded from the electronic medical record system (eCW) via a MYSQL to SAS interface and linked with the interview data.

Associations between sleep quality or duration quartile (1, 2, 3, 4) and obesity (BMI ≥ 30 kg/m²) were assessed by 2×4 contingency tables. The association between bedtime stability (a dichotomous variable) and obesity was assessed by a 2×2 contingency table. Logistic regression models were used to adjust for potential confounders. SAS and Excel software were used for statistical analysis.

RESULTS

Table 1 displays the characteristics of the primary care population (N = 1,964) and the study sample (n = 225). The 95% confidence intervals around the sample estimates generally

Table 2—Prevalence of sleep issues among study patients (N = 225)

Sleep Problem	Number	Prevalence (per 100)	(95% CI) for Prevalence
Overall poor quality sleep (PSQI)	176	78.2	(73, 84)
Poor subjective sleep quality	101	45.5	(39, 52)
Long sleep latency	129	57.3	(51, 64)
Short sleep duration	93	41.3	(35, 48)
Low sleep efficiency	100	45.0	(39, 52)
Frequent sleep disturbances	120	53.6	(47, 60)
Use medicines to induce sleep	64	29.1	(23, 35)
Frequent daytime dysfunction	60	27.4	(22, 33)
High (Berlin) apnea risk	133	59.1	(53, 66)
Daily restless legs symptoms	26	11.6	(7, 16)
Patient reports apnea diagnosis	21	9.3	(6, 13)

Figure 1—Sleep quality quartile and obesity

χ^2 (df = 3) = 10.1; p = 0.01; 16 values missing

Figure 2—Sleep duration quartile and obesity

χ^2 (df = 3) = 6.2; p = 0.10; 19 values missing

include the corresponding population parameters (24 of 26 intervals included the parameter, and the exceptions were close to the parameter). This suggests that selection bias was small. The goodness of fit tests supported this interpretation. In other words, these data suggest that the study sample was statistically similar to the target population of interest, at least in terms of the displayed characteristics.

Table 2 displays the prevalence of sleep issues among patients that were interviewed. We note the high prevalence of poor quality sleep (78%) and elevated Berlin apnea scores (59%). Five of the 7 individual PSQI components have prevalence estimates near 50%. The exceptions were use of medicines to induce sleep (29%) and frequent daytime dysfunction (27%). Positive RLS screens were 12%, slightly higher than patient reports of sleep apnea (9%).

Figure 1 documents a significant near step-wise relationship between sleep quality (p = 0.003 from a Wald test) and obesity. That is, participants with the best sleep quality had the lowest prevalence of obesity, and participants with the worst sleep quality had the highest prevalence of obesity. A logistic model indicated that the relationship between poor sleep quality

and obesity was independent (p = 0.005) of age, gender, and ethnicity. The estimates for these covariates were not significant. Other models considered the possible impact of hypertension and snoring (Berlin category 1 symptoms) and daytime dysfunction (Berlin category 2 symptoms) on obesity. The estimates for hypertension or Berlin category 2 symptoms were not significant. However, the presence of Berlin category 1 symptoms (p < 0.0001) and high PSQI scores (p = 0.007) were both significant and independently related to obesity.

The data summarized in **Figure 2** are consistent with a nonlinear (quadratic) relationship between sleep duration and obesity. That is, the prevalence of obesity is lowest among participants who reported 5 to 7 h of sleep per night and higher among participants who sleep less or more. This interpretation is supported by a logistic model which had significant linear and quadratic terms (**Table 3**).

Table 4 documents a likely effect of bedtime stability on the prevalence of obesity. That is, patients with less stable bedtimes were more likely to be obese. The bedtime stability finding is important because causality is less likely to be reversed, that is, obesity is probably not a cause of bedtime instability on both

Figure 3—Conceptual model **Table 3**—Logistic model testing for non-linear relationship between duration and obesity

Parameter	DF	Estimate	STD Error	χ^2	p-value
Intercept	1	2.50	0.88	8.00	0.005
Duration	1	-1.80	0.77	5.52	0.019
Duration ² (duration squared)	1	0.33	0.15	4.90	0.027

Table 4—Bedtime stability and obesity

Bedtime Stability	Obese	Not Obese
Week day*		
Stable	70 (53.0)	62 (47.0)
Not stable	50 (72.5) [†]	19 (27.5)
Week end [‡]		
Stable	63 (54.3)	53 (45.7)
Not stable	57 (68.7) [§]	26 (31.3)

Values reported are number (percent). *24 values missing. [†] χ^2 (df = 1) = 7.11; p = 0.008. [‡]26 values missing. [§] χ^2 (df = 1) = 4.17; p = 0.04.

weekdays and weekends, while obesity may be a cause of poor quality sleep or shorter sleep.

Figure 3 shows the conceptual model that drives our research. We do not have adequate data on the relationships in the model, but the model suggests that the general hypothesis of inadequate sleep-to-obesity and the observations from this study are plausible from a biopsychosocial perspective. In addition, a pilot randomized trial conducted by our group suggests that exposure to sleep hygiene education and sleep focused

cognitive behavioral therapy accelerates weight loss in the short term.¹³

DISCUSSION

This cross-sectional investigation appears to be the first study with an unbiased probability sample of primary care patients to show that sleep duration, poor sleep quality, and unstable bedtimes are associated with obesity. According to our model (**Figure 3**) short-sleep, poor quality sleep, or unstable bedtimes can initiate a metabolic response with wide ranging consequences that can affect appetite, fatigue, behavior, and fat mass. The U-shaped sleep duration-obesity relationship observed in the current study has been previously reported and remains unexplained.^{11,12}

Five recent reviews summarize the sleep duration-obesity literature.²⁰⁻²⁴ Magee and Hale reviewed 20 longitudinal studies published between 2004 and 2010. Results for adults were inconsistent, while studies of children consistently showed a positive relationship between short sleep and obesity.²⁰ Nielson et al. looked at 23 original studies that were not in recent reviews and report similar conclusions.²¹ Cappuccio et

al. conducted a rigorous meta-analysis of 45 cross-sectional or prospective studies of adults or children and found a pooled short sleep-obesity odds ratio of 1.6 (1.4-1.7) for adults and 1.9 for children (1.5-2.4).²² Marshal et al. described the nature of the association (none, negative linear, or U-shaped) between sleep duration and obesity in cross-sectional and prospective studies of children and adults.²³ The conflicting data drove the authors to conclude that the evidence does not warrant “public health advice about sleep duration as risk factor for obesity” (p. 289).²³ Patel and Hu also considered cross-sectional and prospective studies for children and adults separately.²⁴ The results from both study designs were consistent in children, while studies of adults were less so.²⁴ Patients younger than 18 years of age were excluded from the current study (see **Table 1**). Explanations of the nonlinear association include age-related changes in the relevant biology or mismatches between sleep transitions and follow-up periods;²⁰ bi-directional causality, measurement bias, confounding by depression or stress, or measuring the wrong sleep parameter.²¹ A single adult primary care sample was listed in the five reviews; neither sleep quality nor sleep timing was addressed.^{11,24} The competing explanations for the nonlinear findings are bias or a complex etiology. However, the negative linear relationship between *sleep quality and obesity* and the relationship between *sleep timing and obesity* are easier to understand from the perspective of the causal model. Sleep timing is also amenable to intervention.

Vorona et al. found an association between reduced sleep and obesity after collecting data from 924 “consecutive” patients in four primary care practices.¹¹ They state that 95% of the approached patients consented to the interview, but there is no information regarding the patients who were not approached or why they were omitted. In addition, they treated sleep duration as a dependent variable rather than as an independent variable, thus making direct comparisons with our work and that of Buscemi et al. difficult.¹² For example, the authors report the results from an analysis of variance that compared the mean total sleep time among normal weight patients, overweight patients, obesity class 1 and 2 patients *combined*, and class 3 obesity patients. No justification is given for combining obesity class 1 and 2 patients, despite the fact that the relative large sample size should have supported a finer-grained analysis. This is an important point because the combined class 1-class 2 obesity group appears to have had the lowest mean total sleep time. A more standard BMI grouping strategy could shift the low (inflection) point of the mean total sleep time curve, change standard deviations, and affect p-values.

Buscemi et al. hypothesized a general association between shorter sleep durations and obesity in an ethnically diverse convenience sample of 200 internal medicine patients with obesity-related chronic conditions; but the central feature of their data was a U-shaped relationship between sleep duration and obesity in 125 women, but not in 75 men.¹² However, the gender-sleep duration interaction was not hypothesized a priori, their ability to explore the interaction statistically was limited by the small sample size, and the authors did not present an adequate biopsychosocial explanation of the interaction. Buscemi et al. could have hypothesized that the U is explained by age-related changes in metabolism or competing

pathways such as female short-sleepers experiencing high levels of stress, while long-sleepers may be depressed.²¹ Statistical interactions are more interesting when there is a testable biopsychosocial hypothesis that could account for the result and stimulate further research. Sleep quality was not assessed. Bedtime stability was assessed with unvalidated items, and no results were reported.

Despite methodological differences, our results are consistent with those of Vorona et al. and Buscemi et al., large population surveys, and with studies of highly selected volunteers.^{25,6} For example, Wheaton et al. model “the number of days in the past 30, when the respondent did not get enough rest or sleep.”²⁵ This construct appears to be an amalgam of sleep quantity and quality and may account for the absence of a U-shaped relationship in their data.²⁵ St-Onge et al. compared volunteers with 6 days of restricted sleep (4 h of sleep per night) or 6 days of habitual sleep (9 h of sleep per night) in a crossover design and report that functional MRI data indicated greater interest in food stimuli after sleep restriction.⁶

Our study has several strengths. Based on comparisons with the target population, our sample is representative with respect to multiple characteristics. Thus selection bias should be small. We assessed sleep duration, sleep quality, and bedtime stability—three aspects of sleep adequacy that are associated with obesity—with valid self-report measures instead of ad hoc items. The associations between sleep quality or bedtime stability and obesity in a primary care sample are new findings consistent with the underlying biopsychosocial model (**Figure 1**). High sleep quality and stable bedtimes may reduce stress, normalize metabolism, and encourage appetite control and increased exercise. Understanding the U-shaped relationship between sleep duration and obesity is more challenging. The possibility of directionality and confounding bias in a cross-sectional study cannot be eliminated. Short sleep may activate a stress pathway and unhealthy behaviors, or short sleep may be the result of anxiety or stress, while long sleep may be caused by depression, which also encourages unhealthy behaviors.²⁶ Further experimental work may settle the issue.^{6,27,28}

Limitations of our study include the cross-sectional design and the fact that we did not confirm patient reports of sleep problems with polysomnography or actigraphy. Our relatively small sample size and the fact that data were collected from one center also limit results. However, there are data suggesting that our urban center is similar to other primary care sites locally and across the country.²⁹

Randomized trials with high quality interventions and measures are needed to further examine the propositions depicted in the conceptual model and advance the field. Thus far, we have conducted a small pilot randomized trial that compared a standard behavioral intervention for obesity with a combination of behavioral interventions for obesity and inadequate sleep.¹³ The combined intervention group experienced accelerated weight loss over a 12-week period ($p < 0.05$). However, sleep adequacy measures did not change in the expected manner. This work needs to be replicated with a larger sample and a longer treatment and follow-up period. The likely public health impact of addressing sleep and obesity in primary care justifies additional clinical and research resources.

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