The Association Between Short Sleep Duration and Obesity in Young Adults: a 13-Year Prospective Study

Gregor Hasler, MD[1,2]; Daniel J. Buysse, MD[3]; Richard Klaghofer, PhD[4]; Alex Gamma, PhD[2]; Vladeta Ajdacic, PhD[2]; Dominique Eich, MD[2]; Wulf Rössler, MA, MD[2]; Jules Angst, MD[2]

[1]Mood and Anxiety Disorders Program, Intramural Research Program, National Institute of Mental Health, National Institutes of Health, Bethesda, MD, USA; [2]Psychiatric University Hospital, Zurich, Switzerland; [3]University of Pittsburgh School of Medicine, Department of Psychiatry, Pittsburgh, PA, USA; [4]Department of Psychosocial Medicine, Zürich University Hospital, Switzerland

Study Objectives: Obesity has become a major health problem with increasing prevalence. Given the limited availability of effective treatment of weight problems, the identification of potentially modifiable risk factors may lead to preventive approaches to obesity. The objective of this study was to test the hypothesis that short sleep duration is associated with obesity and weight gain during young adulthood.

Design: Prospective single-age cohort study of young adults. Information was derived from 4 interviews when participants were ages 27, 29, 34, and 40 years.

Setting: Community setting.

Participants: 496 young adults.

Measurements and Results: Trained health professionals administered a semistructured interview for psychiatric and medical conditions and health habits. This study showed an association between short sleep duration and obesity (at age 27 years, odds ratio: 7.4, 95% confidence interval: 1.3-43.1) and a negative association between sleep duration and body mass index in young adults. These associations persisted after controlling for a variety of potentially confounding variables, including family history of weight problems, levels of physical activity, and demographic variables. Associations between sleep duration and obesity diminished after age 34 years. There was a trend ($P = .08$) for average change rate of weight gain to be negatively associated with average change rate of sleep duration.

Conclusions: Because sleep duration is a potentially modifiable risk factor, these findings might have important clinical implications for the prevention and treatment of obesity.

Key Words: sleep duration, obesity, cohort study

Citation: Hasler G; Buysse DJ; Klaghofer R et al. The association between short sleep duration and obesity in young adults: a 13-year prospective study. *SLEEP* 2004;27(4):661-6.

INTRODUCTION

OBESITY HAS BECOME A MAJOR HEALTH PROBLEM[1] BECAUSE IT SHOWS INCREASING PREVALENCE AND IS RELATED TO MULTIPLE MEDICAL CONSEQUENCES SUCH AS INCREASED RISK FOR DIABETES, HEART DISEASE, ARTHRITIS, AND CANCER.[2] In addition, obesity is associated with important social and economic consequences,[3] and weight-related morbidity is estimated to account for 6.8% of healthcare costs in the United States.[4] Given the limited effectiveness of treatment for weight problems, the identification of potentially modifiable risk factors for increased body weight could lead to preventive strategies. Prior research indicates a range of such risk factors, including low levels of physical activity and easy access to high-fat foods. However, the identification of other potentially novel risk factors might provide insights into other possible preventative approaches to obesity.

There is accumulating evidence that shorter sleep duration is becoming another pervasive problem in today's society.[5] In humans, experimental reduction of sleep duration results in metabolic alterations, including glucose intolerance.[6] In rats, prolonged sleep deprivation can lead to increased food intake, changes in appearance of the animals, decrease in body temperature, decrease in plasma thyroxine, and increased mortality.[7]

Short sleep duration has been associated with health outcomes, including death,[8] diabetes,[9] and heart disease.[10] A longitudinal study in women has provided evidence for an association between short sleep duration and later diabetes.[9] Increased body mass index (BMI) is one of the most consistently reported correlates of short sleep duration. Based on a prospective study in women, Ayas and coworkers[9] concluded that the causal relationship between short sleep duration and diabetes could be in either direction and that sleep restriction might mediate its effect on diabetes through weight gain. Based on their large epidemiologic study,[8] Kripke et al reported a negative association between sleep duration and BMI in men and a U-shaped relation between sleep duration and BMI in women. Vioque and coworkers[11] showed an inverse association between obesity and sleep duration, with a prevalence odds ratio for obesity being 24% lower for each additional hour of sleeping time in a cross-sectional community study. Even among 5- and 6-year-old children, von Kries[12] and coworkers showed that the prevalence of obesity decreases by duration of sleep, independent of other risk factors for childhood obesity.

In contrast to previous cross-sectional studies, this study examines longitudinal associations between sleep duration and obesity and BMI (level, change per year) in young adults. Based on previous studies,[9,8,11,12] we hypothesized that sleep duration of less than 6 hours would be associated with increased BMI and obesity. In addition, we hypothesized that short sleep duration would be associated with increased weight gain in young adults.

METHODS

Sample

The Zurich Cohort Study comprises a cohort of 4,547 subjects (2201 men, 2346 women) representative of the canton of Zurich in Switzerland, who were assessed in 1978 with a psychological symptom questionnaire, the Symptom Checklist 90-R (SCL-90-R)[13] and a questionnaire for sociodemographic data. The study is based on a stratified sample with an overrepresentation of risk cases for psychiatric disorders. In order to increase the probability of somatic and psychological syndromes, a subsample of 591 subjects (292 men, 299 women) was selected for interview, with two thirds consisting of high scorers (defined by the 85th percentile or more of the SCL-90-R) and a random sample of those with scores below the 85th percentile. After a complete description of the study to the subjects, informed consent was obtained from subjects according to the requirements of the Swiss National Science Foundation. The screening took place in 1978 when the participants were aged 19 years, the first and second interviews in 1979 and 1981, the third and fourth interviews in 1986 and 1988, the fifth interview in 1993, and the sixth in 1999. The number of study participants and type of assessment at each interview are shown in Table 1.

For the current study, data from interviews in 1986, 1988, 1993, and 1999 were used and included 496 subjects. These assessments were selected because they included interview data on sleep duration. The following proportions participated in specific numbers of interviews: 62.5% in all 4 interviews, 77.4% in 3 interviews, and 90.9% in at least 2 interviews. Those who had dropped out did not differ significantly from participants who participated in all interviews regarding the major predictor and outcome variables of this study, including baseline sleep duration, baseline BMI, level of physical activity, family history of weight problems, and childhood depression (analyzed by χ^2 and t tests).

Diagnostic Interview

The diagnostic instrument used in the Zurich study was the Structured Psychopathological Interview and Rating of the Social Consequences for Epidemiology (SPIKE), a semistructured interview for psychiatric and medical conditions and health habits that was developed for epidemiologic studies.[14] Health professionals with extensive clinical training administered the SPIKE in the participants' homes.[15] This interview schedule assesses a number of somatic syndromes, including sleep behaviors and sleep disorder symptoms; weight problems; headache; and gastrointestinal, cardiovascular, respiratory, perimenstrual, and sexual syndromes. In addition, it assesses psychological syndromes, including depression, hypomania, anxiety, phobia, obsessive-compulsive disorder, posttraumatic stress disorder, substance abuse, and suicidality. Personal and family history of the syndromes was assessed for all subjects, irrespective of endorsement of the diagnostic screening question for each section. Diagnoses of psychiatric disorders and insomnia were made by algorithms based on the criteria of the *Diagnostic and Statistical Manual of Mental Disorders* (DSM-III-R and DSM-IV).

Assessment of BMI

BMI is frequently used to estimate body fat in clinical practice and epidemiologic research, partially because of the ease with which it is measured.[16] Among middle-aged adults, BMI is strongly correlated with fat mass measured densitometrically and adjusted for height (r is approximately 0.9 for both men and women).[17] Although self-reported BMI is measured with some bias, validation studies suggest that this bias is unlikely to affect conclusions about associations between BMI and other health variables, particularly in longitudinal studies.[18-20] Specifically, a Swiss national survey showed that BMI underreporting depends on age, being minimal in young adults between ages 20 and 40 years[21] (ie, the underestimation of self-reported BMI was around 1 kg/m^2). For the Zurich cohort study, height was determined by self-report in 1979, and weight was determined by self-report at each interview. Following the United States guidelines for healthy weight,[16] we defined obesity as having a BMI $\geq$ 30.

Assessment of Sleep Duration

Previous studies comparing self-reported sleep duration with quantitative rest-activity measurements with actigraphy found good agreement between these methods[22,23]; particularly good correlations were found when comparing the measurement of sleep onset ($r = 0.77$) and sleep offset ($r = 0.88$), while the measurement of sleep latency was poorly correlated ($r = 0.12$). For the Zurich study, 3 questions concerning sleep duration were assessed by interview: (a) time (hours and minutes) subjects went to bed, (b) time (hours and minutes) subjects arose, and (c) time (minutes) subjects needed to fall asleep. Sleep duration was calculated as duration spent in bed (a, b) minus time needed to fall asleep (c). Based on previous studies,[9,11] we defined short sleep duration as less than 6 hours. In addition to the questions related to sleep duration, there were questions concerning daytime sleepiness (yes or no), awakenings during the sleep episode (yes or no), and (subjective) quality of sleep (not impaired, moderately impaired, severely impaired).

Statistical Analyses

We conducted all analyses for the whole sample as well as separately for men and women and for the SCL-90-R high-scorer and low-scorer samples. All analyses were tested for sex-by-predictor and stratified sampling (SCL-90-R high vs low scorer)-by-predictor variable interactions as predictors of obesity. Sex and stratified sampling were included in the analyses. SAS for Windows release 8.02 (SAS, Inc., Cary, NC) was used for all statistical analyses. We used the macro COLLIN from SAS-L to calculate collinearity diagnostics from variance-covariance matrix in nonlinear regression models.[24]

We examined associations between sleep duration and obesity defined both by a cut-off on the BMI distribution and BMI as a continuous variable, since these 2 analytic approaches answer conceptually different questions. The categorical approach considers the association with an abnormal category associated with severe medical consequences; the continuous approach, in contrast, examines the associations between sleep duration and normal variations in relative weight, as distributed throughout the population. An initial data exploration revealed a nonlinear relationship between sleep duration and BMI with a virtually mono-

tonic trend toward lower BMI among those with longer sleep duration. Because BMI in subjects with intermediate sleep duration did not statistically differ form BMI in subjects with long sleep duration, we decided to dichotomize sleep duration (short vs intermediate/long sleep duration) as well as to use sleep duration as continuous/multilevel variable.

To test for associations between sex, stratified sampling, and socioeconomic status and sleep duration, we used repeated measures analysis of variance and included sex, stratified sampling (0 = SCL-90-R low scorer, 1 = SCL-90-R high scorer), and socioeconomic status as main effects and sleep duration as response variable. To express cross-sectional associations between short sleep duration (< 6 hours) and obesity (BMI > 30), we calculated odds ratios adjusted for sex and stratified sampling. For a multivariable longitudinal model for obesity, we calculated robust estimates for several sleep characteristics, such as sleep duration, sleep quality, insomnia, awakenings during the sleep episode, and daytime sleepiness, by generalized estimating equations (SAS PROC GENMOD) using measures from interviews in 1986, 1988, 1993, and 1999 with subject as cluster and a first-degree autoregressive within-cluster correlation structure. In this model, we included 21 covariates (age, sex, stratified sampling, levels of physical activity, education level, socioeconomic status, household income, family history of weight problems, major depression, childhood depression, level of self-esteem, use of antidepressants, subjective oversleeping, use of hypnotics, generalized anxiety disorder, panic disorder, phobic disorders, binge eating, conduct disorder symptoms, tobacco dependence, alcohol abuse/dependence, drug abuse/dependence) because these variables were considered in previous studies as potential confounders.[3,8,9,25-28] We reduced the number of covariates by backward elimination to avoid multicollinearity (probability level for removal: 10%).

To graphically depict the associations between sleep duration and BMI, we used the average sleep duration from interviews in 1986, 1988, 1993, and 1999 and BMI data from the last interview approximating the cumulative effect of sleep duration on BMI as outcome. In addition, we applied a simple linear regression model of each subject in which his or her assessed BMI values was the dependent variable and the age at the time of the assessment, the independent variable; we used the slope coefficient as the individual's BMI change trend of direction and magnitude (BMI slope). Using the same method, we also calculated slope coefficients for the individual sleep duration change trend with age. We used simple linear regression to test an association between average change rate of BMI and average change rate of sleep duration, including sex and stratified sampling as covariates.

To test associations between sleep-duration categories and BMI at each age, we applied analyses of variance using data from each interview separately. To statistically test associations between sleep duration and BMI level and BMI change rate across interviews, we used random regression (SAS PROC MIXED). The form of the random effects model is identical to that used in ordinary multiple regression, but the methods used to estimate the regression coefficients are modified to account for the correlation between repeated measures on the same subject. We modeled the repeated measures of BMI as response variable with subject as random effect and subject as cluster using a first-order autoregressive within-cluster correlation structure. Assuming a nonlinear relationship between BMI and sleep duration, we included sleep duration as indicator variables coding for 6 sleep-duration categories (< 5, 6, 7, 8, > 9 hours). These categories, other sleep characteristics, and 21 covariates (see above) were included as fixed effects. We reduced the number of covariates by backward elimination to avoid multicollinearity (probability level for removal: 10%).

RESULTS

Table 1 shows mean sleep duration in men and women at ages 27, 29, 34, and 40 years. On average, women slept more than men, and sleep duration decreased with age. Subjects with low socioeconomic status slept less than those with higher socioeconomic status (data not shown). A repeated measures analysis of variance with the repeated measures of sleep duration as response variable yielded significant main effects for sex ($F_{1,290}$ = 54.43, $P < .0001$), socioeconomic status ($F_{3,290}$ =3.53, $P < .05$), and age ($F_{3,870}$ 13.55, $P < .0001$); stratified sampling (ie, level of SCL-90-R scores in 1979) did not have a significant influence on sleep duration.

As shown in Table 2, there were significant cross-sectional associations between short sleep duration and obesity at ages 27, 29, and 34 years; however, there was no association between short sleep and obesity at age 40 years. In addition, all associations between short sleep and obesity at previous and later adjacent interviews were significant. In the subsample of subjects having low SCL-90-R scores at screening (n = 158), the magni-

Table 1—Sample and design of the Zurich Cohort Study, self-reported BMI, and self-reported sleep duration

Year	No.	Age, y	Mean BMI, kg/m² (SD)	Assessment	Mean sleep duration, h (SD)	
					Women	Men
1978	4547	19	—	Screen	—	—
1979	591	20	21.2 (2.5)	Interview	—	—
1980	501	21	—	Questionnaire	—	—
1981	456	22	21.6 (2.7)	Interview	—	—
1986	457	27	21.8 (2.9)	Interview	7.70 (0.94)	7.13 (0.97)
1988	424	29	22.0 (3.0)	Interview	7.76 (0.93)	7.14 (0.94)
1993	407	34	22.5 (3.4)	Interview	7.63 (0.94)	7.02 (0.92)
1999	367	40	23.3 (3.8)	Interview	7.31 (0.98)	6.90 (1.00)

BMI refers to body mass index.

tude of the cross-sectional associations between short sleep duration and obesity were slightly higher in 1986 (odds ratio = 12.0 [1.7-85.8]) and in 1988 (odds ratio = 9.0 [1.3-64.2]) and did not differ in 1993 and 1999 as compared to the total sample. There were no significant sex-by-predictor and sampling-by-predictor variable interactions as predictors of obesity.

Figure 1 shows least-squared means and 95% confidence intervals of BMI at age 40 years by categories of average sleep duration. There was a virtually monotonic trend toward lower BMI among those with longer sleep duration. Figure 2 shows least-squared means and 95% confidence intervals of BMI slope (change per year) by categories of average sleep duration. There was also a virtually monotonic trend toward lower weight gain among those with longer sleep duration.

Although these figures graphically depict relationships between BMI and sleep duration, these relationships were statistically assessed using multivariable methods. A multivariable longitudinal model for obesity using the general estimating equations approach (ie, a regression method for correlated data and binary response variable) is shown in Table 3. This model included repeated measures of obesity (BMI ≥ 30) as a response variable, sleep duration in hours and sleep disorder symptoms as a predictor variable, and a variety of potentially confounding variables as covariates. Sleep duration was a strong and significant

predictor of obesity. Impaired sleep quality, insomnia, awakenings during the sleep episode, and daytime sleepiness were not associated with obesity. None of the covariates had a considerable influence on the association between sleep duration and obesity.

There were cross-sectional associations between sleep duration categories and BMI in 1986, 1988, and 1993; however, there was no significant association between sleep-duration categories and BMI in 1999. The random effects model for BMI (ie, a regression method for correlated data and continuous response variable) including a variety of covariates yielded a significant association between sleep-duration categories and BMI and a significant interaction between sleep-duration categories and age related to BMI. Other sleep characteristics, including sleep quality, insomnia, awakenings during the sleep episode, and daytime sleepiness, were not associated with BMI in this model.

Using simple linear regression, change rate in sleep duration in minutes per year tended to be negatively associated with change rate in BMI per year after adjusting for stratified sampling and sex (β = -0.082, P = .08).

Table 2—Associations between short sleep duration (< 6 hours) and concurrent, past, and previous obesity*

Age, y	Cross-sectional associations OR (95% CI)	Association between short sleep duration and later obesity OR (95% CI)	Association between short sleep duration and previous obesity OR (95% CI)
27	7.4 (1.3-43.1)†	8.2 (1.9-36.3)‡	—
29	8.1 (1.8-37.4)‡	4.6 (1.3-16.5)†	11.8 (1.6-86.5)†
34	4.7 (1.5, 14.8)‡	3.5 (1.0-12.2)†	5.1 (1.2-22.7)†
40	1.1 (0.3, 4.0)	—	3.5 (1.1-11.5)†

*All odd ratios (OR) were adjusted for sex and stratified sampling (high scorer vs low scorer on the Symptom Checklist 90-R). CI refers to confidence interval.
†P < .05
‡P < .01

Table 3—Multivariable associations between sleep characteristics and obesity in subjects between ages 27 and 40 years

| Variable	OR*	95% CI
Age, in 10-y increases	2.9‡	1.7-4.7
Sleep duration, h	0.5†	0.3-0.8
Impaired sleep quality	0.6	0.3-1.0
Insomnia	0.5	0.2-1.1
Awakenings during the sleep episode	1.2	0.6-2.4
Daytime sleepiness	0.9	0.4-2.3

*Odds ratios (OR) estimated by generalized estimating equations; the OR are adjusted for sex, stratified sampling, education level, level of physical activity, smoking, binge eating, childhood depression, and a family history of weight problems; the variables are dichotomous when no (unit) is indicated. CI refers to confidence interval.
†P < .01
‡P < .001
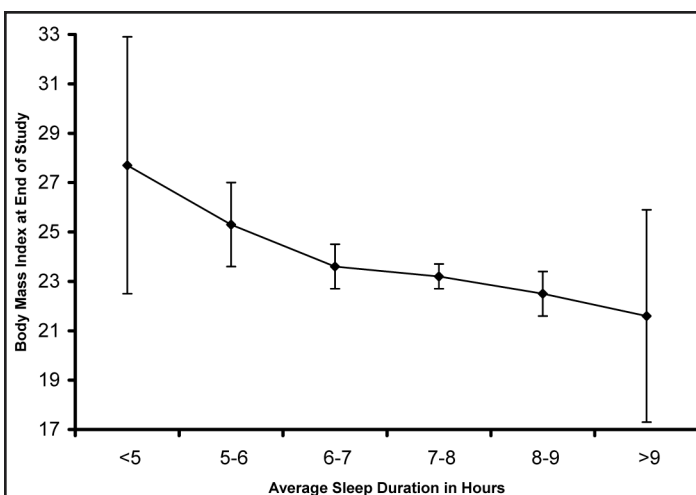
Figure 1—Least-squared means and 95% confidence intervals of body mass index at age 40 years adjusted for sex and stratified sampling by categories of average sleep duration in young adults between ages 27 and 40 years.
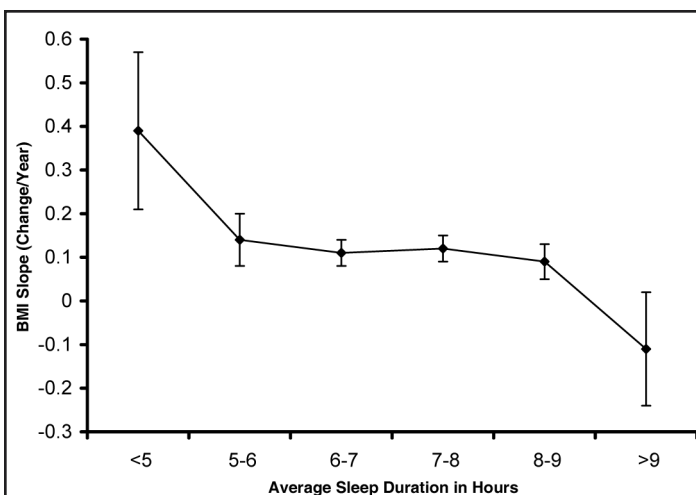
Figure 2—Least-squared means and 95% confidence intervals of body mass index (BMI) slope adjusted for sex and stratified sampling by categories of average sleep duration in young adults between ages 27 and 40 years.

DISCUSSION

Consistent with our first hypothesis, we observed strong associations between short sleep duration (< 6 hours) and obesity. Cross-sectionally, we found this association at ages 27, 29, and 34 but not at age 40 years. This is in line with previous studies that showed stronger associations between sleep duration and BMI in samples with predominantly children and younger adults than in samples with predominantly older adults[8,9,11,12] and is consistent with reports that point to age-specific pathophysiologic mechanisms of obesity.[29,30] In addition, potential differences in the assessment methods may have contributed to age-specific variations, although almost identical interviews were applied between 1986 and 1999.

The multivariable longitudinal model for obesity showed that sleep duration as a continuous variable persisted as a strong and statistically significant correlate of obesity after controlling for a variety of potentially confounding variables. Further confirming these observations, we found a significant association between sleep-duration categories and BMI. The associations between sleep duration and obesity and BMI suggest a dose-response relationship. However, results of previous studies concerning BMI and other outcomes associated with long sleep duration are inconsistent.[8,9] In the current study, the specific association between long sleep duration and BMI was inconsistent over time: at age 29, subjects who slept more than 9 hours had a numerically higher BMI than did subjects sleeping 7 or 8 hours. Therefore, we do not assume a dose-response relationship and do not draw any conclusion about the association between long sleep duration related to BMI.

The odds ratios estimating the obesity-sleep duration associations showed considerable imprecision, and the confidence intervals of mean BMI of extreme sleep duration categories were wide due to the small number of subjects in these categories. However, standard deviations of BMI and BMI slope were similar across sleep duration categories.

Associations between sleep duration and previous and later obesity did not reveal causality. However, the random-effects model for BMI revealed an interaction between sleep-duration categories and age, and there was a trend for the rate of change in sleep duration being negatively associated with the rate of change in BMI. Because this association did not reach statistical significance, it has to be interpreted with caution and needs confirmation by future studies.

The lack of association between sleep quality and insomnia and obesity is in line with previous studies that showed that insomnia was not associated with, or was even negatively associated with, increased mortality.[8,31] This apparent paradox may result from the fact that insomnia complaints are not strongly associated with sleep duration[8] or that insomnia is pathophysiologically distinct from short sleep.

Sleep-disordered breathing is associated with awakenings during the sleep episode, daytime sleepiness, and impaired sleep quality,[32] as well as with obesity, glucose intolerance, and insulin resistance.[33] The finding, however, that awakenings during the sleep episode and especially daytime sleepiness were not associated with obesity tempers the plausible suggestion that obstructive sleep apnea may mediate the obesity–sleep-duration relationship.

This study has several methodological limitations. The main limitation is the recruitment of the sample with two thirds of SCL-90-R high scorers, which reduces the generalizability of the results. However, we tested all associations in the SCL-90-R low-scorer sample separately and found somewhat stronger associations between short sleep and obesity relative to the SCL-90-R high-scorer sample. For instance, subjects with major depression did not show any association between sleep duration and BMI. These findings might suggest an even stronger association between short sleep duration and weight problems in a more representative sample. Moreover, the assessments of sleep duration and of body weight were based on self-report, and no objective sleep measures were available to validate the self-reported data. However, the associations of low socioeconomic status and male sex with short sleep duration, and the decrease in sleep duration with age, are in line with results of previous studies that measured sleep by objective measures.[31,34,35] Finally, we did not have any objective outcome measure such as blood pressure or blood glucose.

Nevertheless, the strengths of this study suggest the findings are worthy of further consideration. Specifically, the sample was community-based; the study design was longitudinal over 20 years; experienced well-trained clinicians administered standardized interviews; and the main hypothesis (association between short sleep duration and obesity) was based on results found in larger community samples, suggesting a high a priori probability of confirmation.

In conclusion, this study shows an association between short sleep duration and obesity that diminished above age 34 years and an association between sleep duration and BMI in young adults. Because sleep duration is a potentially modifiable risk factor, these findings might have important clinical implications for the prevention and treatment of obesity.

REFERENCES

1. Mokdad AH, Serdula MK, Dietz WH, Bowman BA, Marks JS, Koplan JP. The spread of the obesity epidemic in the United States, 1991-1998. JAMA 1999;82:1519-22.
2. Must A, Jacques PF, Dallal GE, Bajema CJ, Dietz WH. Long-term morbidity and mortality of overweight adolescents. A follow- up of the Harvard Growth Study of 1922 to 1935. N Engl J Med 1992;327:1350-5.
3. Gortmaker SL, Must A, Perrin JM, Sobol AM, Dietz WH. Social and economic consequences of overweight in adolescence and young adulthood. N Engl J Med 1993;329:1008-12.
4. Wolf AM, Colditz GA. Social and economic effects of body weight in the United States. Am J Clin Nutr 1996;63:466S-9.
5. Bonnet MH, Arand DL. We are chronically sleep deprived. Sleep 1995;18:908-11.
6. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. Lancet 1999;354:1435-9.
7. Rechtschaffen A, Bergmann BM, Everson CA, Kushida CA, Gilliland MA. Sleep deprivation in the rat: X. Integration and discussion of the findings. Sleep 1989;12:68-87.
8. Kripke DF, Garfinkel L, Wingard DL, Klauber MR, Marler MR. Mortality associated with sleep duration and insomnia. Arch Gen Psychiatry 2002;59:131-6.
9. Ayas NT, White DP, Al-Delaimy WK, et al. A prospective study of self-reported sleep duration and incident diabetes in women. Diabetes Care 2003;26:380-4.
10. Ayas NT, White DP, Manson JE, et al. A prospective study of sleep duration and coronary heart disease in women. Arch Intern Med 2003;163:205-9.

11. Vioque J, Torres A, Quiles J. Time spent watching television, sleep duration and obesity in adults living in Valencia, Spain. Int J Obes Relat Metab Disord 2000;24:1683-8.

12. von Kries R, Toschke AM, Wurmser H, Sauerwald T, Koletzko B. Reduced risk for overweight and obesity in 5- and 6-y-old children by duration of sleep—a cross-sectional study. Int J Obes Relat Metab Disord 2002;26:710-6.

13. Derogatis LR. Administration, Scoring and Procedures Manual-I for the R (revised) Version and Other Instruments of the Psychopathology Rating Scale Series. Baltimore: Johns Hopkins School of Medicine; 1977.

14. Angst J, Dobler-Mikola A, Binder J. The Zurich Study—a prospective epidemiological study of depressive, neurotic and psychosomatic syndromes. Eur Arch Psychiatry Clin Neurosci 1984;234:13-20.

15. Angst J, Dobler-Mikola A. The Zurich study. VI. A continuum from depression to anxiety disorders? Eur Arch Psychiatry Clin Neurosci 1985;235:179-186.

16. Willett WC, Dietz WH, Colditz GA. Guidelines for healthy weight. N Engl J Med 1999;341:427-34.

17. Willett WC. Nutritional Epidemiology. New York: Oxford University Press; 1998.

18. Stevens J, Keil JE, Waid LR, Gazes PC. Accuracy of current, 4-year, and 28-year self-reported body weight in an elderly population. Am J Epidemiol 1990;132:1156-63.

19. Stewart AL. The reliability and validity of self-reported weight and height. J Chronic Dis 1982;35:295-309.

20. Stunkard AJ, Albaum JM. The accuracy of self-reported weights. Am J Clin Nutr 1981;34:1593-9.

21. Schutz Y, Woringer V. Obesity in Switzerland: a critical assessment of prevalence in children and adults. Int J Obes Relat Metab Disord 2002;26 Suppl 2:S3-11.

22. Hauri PJ, Wisbey J. Wrist actigraphy in insomnia. Sleep 1992;15:293-301.

23. Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. J Sleep Res 1999;8:175-83.

24. Davis CE, Hyde JE, Bangdiwala SI, Nelson JJ. An example of dependencies among variables in a conditional logistic regression. In: Moolgavkar SH, Prentice RL, eds. Modern Statistical Methods in Chronic Disease Epidemiology. New York: John Wiley & Sons; 1986:140-7.

25. Buysse DJ, Ganguli M. Can sleep be bad for you? Can insomnia be good? Arch Gen Psychiatry 2002;59:137-8.

26. Friedman MA, Brownell KD. Psychological correlates of obesity: moving to the next research generation. Psychol Bull 1995;117:3-20.

27. Kripke DF, Klauber MR, Wingard DL, Fell RL, Assmus JD, Garfinkel L. Mortality hazard associated with prescription hypnotics. Biol Psychiatry 1998;43:687-93.

28. Pine DS, Cohen P, Brook J, Coplan JD. Psychiatric symptoms in adolescence as predictors of obesity in early adulthood: a longitudinal study. Am J Public Health 1997;87:1303-10.

29. Comings DE, Gade R, MacMurray JP, Muhleman D, Peters WR. Genetic variants of the human obesity (OB) gene: association with body mass index in young women, psychiatric symptoms, and interaction with the dopamine D_2 receptor (DRD2) gene. Mol Psychiatry 1996;1:325-35.

30. Stunkard AJ, Sorensen TI, Hanis C, et al. An adoption study of human obesity. N Engl J Med 1986;314:193-8.

31. Wingard DL, Berkman LF. Mortality risk associated with sleeping patterns among adults. Sleep 1983;6:102-7.

32. Malhotra A, White DP. Obstructive sleep apnoea. Lancet 2002;360:237-45.

33. Punjabi NM, Ahmed MM, Polotsky VY, Beamer BA, O'Donnell CP. Sleep-disordered breathing, glucose intolerance, and insulin resistance. Respir Physiol Neurobiol 2003;136:167-78.

34. Jean-Louis G, Kripke DF, Ancoli-Israel S, Klauber MR, Sepulveda RS. Sleep duration, illumination, and activity patterns in a population sample: effects of gender and ethnicity. Biol Psychiatry 2000;47:921-7.

35. Van Cauter E, Spiegel K. Sleep as a mediator of the relationship between socioeconomic status and health: a hypothesis. Ann N Y Acad Sci 1999;896:254-61.