nature publishing group

A Population-Based Twin Study on Sleep Duration and Body Composition

Rong Liu[1], Xin Liu[1], Lester M. Arguelles[1], Pallavi P. Patwari[2], Phyllis C. Zee[3], Ronald D. Chervin[4], Fengxiu Ouyang[1], Katherine K. Christoffel[1], Shanchun Zhang[5], Xiumei Hong[1], Guoying Wang[1], Xiping Xu[6] and Xiaobin Wang[1]

The aim of this study is to investigate the relationship between sleep duration and body composition and to estimate the genetic contribution of sleep duration and body composition in a Chinese twin population. This cross-sectional analysis included 738 men and 511 women aged 21–72 year. Anthropometric and dual-energy X-ray absorptiometry (DXA) measures of body composition were used. Sleep duration was obtained from a standard sleep questionnaire. Multiple regression models were used to examine the association between sleep duration and body composition measures. Structural equation modeling was used to assess the heritability of sleep duration and body composition. Compared with individuals in the 2nd and 3rd age-specific quartiles of sleep duration (reference group), shorter (1st quartile) sleep duration among women but not men was associated with higher *z*-scores (0.248–0.317) for all adiposity measures—BMI, fat mass index (FMI), percent body fat mass (%BF), and percent trunk fat mass (%TF), $P < 0.05$ for each—and with 0.306 lower *z*-scores for percent body lean mass (%LM) and 0.353 lower lean/fat mass ratio (LFR), $P < 0.01$ for each. The heritability of sleep duration was 0.27 in men and 0.29 in women, while the heritability of body composition was as high as 0.56–0.73 after adjustment for age in both genders. Short sleep duration was associated with increased body fat and decreased lean body mass in women but not in men. Sleep duration was largely influenced by environmental factors while adiposity measures were mainly influenced by genetic factors.

Obesity (2012) **20,** 192–199. doi:10.1038/oby.2011.274

INTRODUCTION

The prevalence of obesity has been increasing during recent decades in developed countries and is now dramatically on the rise in countries with middle- and low-income economies1. The rapid increase in the prevalence of obesity over recent decades is thought to be caused by changes in lifestyle rather than by changes in genetic endowment. The most common factors believed to contribute to obesity are increased food intake and physical inactivity. Accumulating evidence also suggests that short sleep duration deserves particular attention (2–5). In parallel with the obesity epidemic, there has been a progressive decrease in self-reported sleep duration (6) worldwide. For example, over the past 8 years, the number of Americans who slept less than 6 h per night rose from 13 to 20% (7). In population studies, the relationship between short sleep duration and high BMI has been reported in different populations and different age groups (4,8–12). Short sleep duration also has been found to be associated with weight gain in longitudinal studies (13–15). Mechanistically, sleep duration may affect the regulation of metabolism and body composition, including fat mass and lean mass, which involve complex physiological systems (16,17).

In our previous study, we showed that short-sleep duration was associated with higher adiposity measures and lower lean body mass in Chinese adolescent girls but not in boys (4). In this current study, we sought to determine whether chronic short-sleep duration is associated with adiposity measures as assessed by BMI and direct measures of adiposity and body composition measured by dual-energy X-ray absorptiometry (DXA) and whether there is a gender difference, in a large sample of rural Chinese adult twins. Furthermore, the cotwin design offered us the opportunity to estimate heritability of sleep duration and body composition, which had not previously been examined in a Chinese population. Such information is particularly helpful in light of developing clinical and public health information. If sleep duration is primarily

[1]Mary Ann and J. Milburn Smith Child Health Research Program, Department of Pediatrics, Northwestern University Feinberg School of Medicine and Children's Memorial Hospital and Children's Memorial Research Center, Chicago, Illinois, USA; [2]Center for Autonomic Medicine in Pediatrics (CAMP), Department of Pediatrics, Northwestern University Feinberg School of Medicine and Children's Memorial Hospital, Chicago, Illinois, USA; [3]Department of Neurology and Sleep Medicine Center, Northwestern University, Chicago, Illinois, USA; [4]Sleep Disorders Center and Department of Neurology, University of Michigan, Ann Arbor, Michigan, USA; [5]Department of Epidemiology and Health Statistics, School of Medicine, Zhejiang University, Hangzhou, China; [6]Center for Population Genetics, University of Illinois at Chicago School of Public Health, Chicago, Illinois, USA. Correspondence: Xiaobin Wang (xbwang@childrensmemorial.org)

Received 23 March 2011; accepted 27 July 2011; published online 25 August 2011. doi:10.1038/oby.2011.274

environmentally determined, it would constitute a simple and effective intervention to prevent or reduce the risk of obesity in the population.

METHODS AND PROCEDURES

Study population

Subjects were men and women aged 21–72 years who participated in an ongoing National Institutes of Health–funded study of metabolic syndrome in a large population-based twin cohort in Anqing, Anhui province, China. This study has been described in detail elsewhere (4). Briefly, a baseline study was carried out in eight rural counties of Anqing from 1998 to 2000, with follow-up evaluation 6 years later. Our current study used data collected from the follow-up evaluation. Participants were invited to a central office and stayed overnight to complete an interview-based questionnaire including the Pittsburgh Sleep Quality Index (PSQI, Chinese version), a fasting blood chemistry test, physical examination, and DXA scans. Physical examinations were conducted by physicians trained specifically for the study to promote consistency of methods for obtaining body composition measures. Longitudinal analyses were not possible with baseline data because the sleep questionnaire was used only during the follow-up evaluation. The study protocol was approved by the institutional review board of Children's Memorial Hospital and the Institute of Biomedicine, Anhui Medical University in Hefei, China. All participants gave written consent prior to their participation.

Individuals ($n = 281$) who had missing data for any of the key variables, such as sleep duration or adiposity, were excluded from the final analysis. Also, an additional 68 individuals reported having symptom(s) of depression and were excluded from the final analysis because of the known association of both sleep and appetite change with depression (18); appetite change may confound the association of sleep and adiposity. Thus, the present study included a total sample of 1,249 participants.

Sleep parameters

Data on sleep duration, snoring, and sleep disturbance were derived from the PSQI. The validity and reliability of the PSQI have been previously demonstrated (19,20). Sleep duration was calculated based on bedtime, wake-up time, and sleep latency (21). Subjects who reported loud and disturbing snoring more than three times/week were considered "habitual snorers." Sleep disturbance was defined according to the score for the component on sleep disturbance on the PSQI, ranging from 0 to 3. Subjects with scores ≥2 were considered to have a sleep disturbance (19).

Considering the wide age range and variable sleep duration by age in our study, we grouped individuals based on age-specific sleep duration quartiles. Each age subgroup spanned 10 years (21–29, 30–39, 40–49, and ≥50). We combined all individuals 50 years or older into one age group because of its small sample size. Age-specific sleep duration quartiles were used to define three sleep groups: short, moderate, and long. The first quartile of sleep duration was defined as short sleeper, the fourth quartile was defined as long sleeper, and the remaining (second and third quartiles) was defined as moderate sleep duration and served as a reference group in linear regression models.

Body composition measures

As previously reported (4), total body fat mass, lean body mass, and trunk fat mass were measured by DXA scan. Body composition indexes were calculated as follows:

$$\mathrm{BMI} = \frac{\text{Weight}\,(\text{kg})}{\text{Height squared}\,(\text{m}^2)};$$

$$\mathrm{FMI} = \frac{\text{Body fat mass}\,(\text{kg})}{\text{Height squared}\,(\text{m}^2)};$$

$$\%\mathrm{BF} = \left(\frac{\text{Body fat mass}}{\text{Body weight}}\right) \times 100;$$

$$\%\mathrm{LM} = \left(\frac{\text{Body lean mass}}{\text{Body weight}}\right) \times 100;$$

$$\%\mathrm{TF} = \left(\frac{\text{Trunk fat mass}}{\text{Body weight}}\right) \times 100;$$

$$\mathrm{LFR} = \frac{\text{Lean mass}}{\text{Fat mass}}.$$

Covariates

This was a relatively homogeneous population in that all of the participating individuals were from the same geographic region, ethnically Han Chinese, mostly farmers, and spoke the same local dialect. Our food frequency questionnaire showed that locally produced rice and vegetables were part of their regular diet. Therefore, covariates considered in the data analyses included age, gender, physical activity level, smoking, alcohol consumption, and menopause status. Physical activity was assessed using a short version of the International Physical Activity Questionnaire (IPAQ-Short, http://www.ipaq.ki.se), as previously described (4).

Statistical analysis

The Student's t-test was used to compare mean differences for continuous variables, and the χ^2-test was used to test differences in proportions between genders. The crude patterns of sleep duration and body composition measures across age were first explored by graphic plots. To compare the magnitude of association of sleep duration with each body composition measure, we converted each body composition measure into standardized z-scores, calculated as (observed value − gender-specific mean)/gender-specific s.d. Least square means were used to compare the difference between either the short-sleep group or the long-sleep group and the reference group. Gender-specific relationships between sleep duration and body composition z-score were further assessed by multiple linear regression models with adjustment for age (5-year range), education and physical activity, smoking, alcohol consumption (for men only), and menopause (for women only). To test the sensitivity of all regression models to snoring or sleep disturbance, we repeated each analysis after exclusion of subjects who reported either problem. Furthermore, the generalized estimating equation was applied to all regression models to account for intratwin pair correlation. Statistical significance was set at a two-sided $P < 0.05$. All analyses described above were performed with SAS 9.2 software (SAS Institute, Cary, NC).

Cotwin design, in which monozygotic (MZ) twins are genetically identical and dizygotic (DZ) twins are genetically related as siblings, enables us to estimate relative contributions of genetic and environmental factors to a specific phenotype. We used structural equation modeling to estimate heritability of sleep duration and body composition indexes. Age and gender were adjusted in the structural equation model. First we tested for each variable to see whether the variances could be set equal over zygosity groups and genders without a significant loss of statistical fit. If no significant difference is observed, the univariate Cholesky decomposition model is collapsed over gender; otherwise the gender-specific model is applied. Saturated models were fitted for additive genetic (A), common/familial environmental (C), and unique environmental (E) components (ACE models), for body compositional indexes and sleep duration. Alternative models for which A, C, or E was equated to zero, i.e., CE, AE, and AC models, also were fitted. Chi-square and Akaike information criterion were used as goodness-of-fit criterion to compare the fit of each model. Estimates from the best-fitted models were presented—the lowest Akaike information criterion that did not have a statistically significant (P value <0.05) worse fit compared with

Table 1 Characteristics of the study subjects

	Male (*n* = 738)	Female (*n* = 511)	
Variables	Mean (s.d.)		*P* value
Age (years)	40.7 (12.7)	38.7 (10.1)	0.002
BMI (kg/m^2)	21.8 (2.7)	22.1 (2.8)	0.048
FMI (kg/m^2)	3.2 (2.1)	6.4 (2.1)	<0.001
%TF	7.7 (4.7)	14.6 (4.1)	<0.001
%BF	13.8 (7.4)	28.3 (6.3)	<0.001
%LM	83.2 (7.5)	68.4 (6.2)	<0.001
LFR	8.3 (4.8)	2.6 (0.9)	<0.001
Sleep duration (hours)	8.3 (1.4)	8.4 (1.4)	0.424
Sleep duration (range, hours)			
Short (4.0–7.5)	6.5 (0.9)	6.6 (0.8)	0.089
Moderate (7.2–9.5)	8.4 (0.5)	8.4 (0.5)	0.976
Long (9.0–11.0)	10.0 (0.6)	10.0 (0.5)	0.380
	N (%)		
Age (years)			
21–	169 (22.9)	86 (16.8)	<0.0001
30–	187 (25.3)	191 (37.4)	
40–	198 (26.8)	166 (32.5)	
50–72	184 (25.0)	68 (13.3)	
Sleep categories			
Short	181 (24.5)	132 (25.8)	0.428
Moderate	379 (50.1)	241 (47.2)	
Long	180 (24.4)	138 (27.0)	
Physical activity level			0.545
Low	117 (15.9)	92 (18.0)	
Moderate	152 (20.6)	94 (18.4)	
High	364 (49.3)	260 (50.9)	
Unknown	105 (14.2)	65 (12.7)	
Literate	674 (91.3)	332 (65.0)	<0.001
Alcohol consumption	302 (40.9)	15 (2.9)	<0.001
Smoking	473 (64.1)	22 (4.3)	<0.001
Menopause	—	83 (16.2)	
Habitual snorer (*n* = 1247)	154 (20.9)	37 (7.2)	<0.001
Sleep disturbance (*n* = 1240)	63 (8.6)	75 (14.8)	<0.001

Student's *t*-tests were used to compare continuous variables, and χ^2-tests were used to compare categorical variables between men and women.
FMI, fat mass index; LFR, lean/fat mass ratio; %BF, percent body fat mass; %LM, percent body lean mass; %TF, percent trunk fat mass; %BL, percent body lean mass.

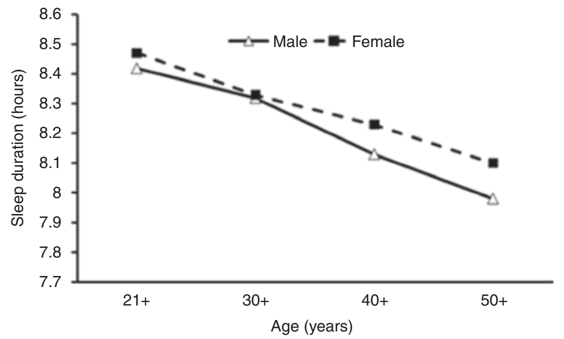

Figure 1 Sleep duration across age in men and women. There was no significant difference in mean sleep duration for each age group between genders.

the saturated model. Mx software (http:/www.psy.vu.nl/ mxbib/) was used for these analyses.

RESULTS

The general characteristics of the 1,249 subjects by gender (738 men and 511 women) are summarized in **Table 1**. Men had a slightly higher mean age and substantially higher literacy level than women. Smoking and alcohol consumption were common in men (64.1 and 40.9%, respectively) but rare in women (4.3 and 2.9%, respectively). As for measures of body composition, women had higher mean BMI, fat mass index (FMI), percent trunk fat mass (%TF), and percent body fat mass (%BF), while men had higher percent body lean mass (%LM) and lean/fat mass ratio (LFR). Physical activity levels were comparable between men and women ($P = 0.545$). The mean sleep duration was 8.3 h for men (s.d. = 1.4) and 8.4 h for women (s.d. = 1.4). No gender difference was found for sleep duration ($P = 0.424$). Men were more likely to report snoring, while women more frequently complained of sleep disturbance.

Sleep duration by age stratified by gender is shown in **Figure 1**. Sleep duration decreased on average by 9.7 min per decade ($P < 0.001$) for men and 8.8 min per decade ($P = 0.025$) for women. The average sleep duration in subjects aged 50 years and older was about half an hour less than that for subjects 20–29 years old in both genders.

Figure 2 depicts the relationships of body composition measures by age and gender. A roughly bell-shaped relationship for BMI by age was seen in both genders. In men, BMI was lower at a younger age, reached an acrophase at around age 35–40 years and then was lower with advanced age. In women, the acrophase of BMI appeared later on, around age 40–44 years. As for DXA measures of adiposity (%BF, %TF, and FMI), these were slightly lower across age groups in men. But in women, only FMI was higher across age while %TF and %BF remained almost unchanged across age groups. Women had consistently higher %BF, %TF, and FMI than men across all age groups, while men had higher %LM and LFR than women across all age groups.

Figure 3 and **Table 2** show the relationship between sleep duration and body composition. We observed significant associations of short-sleep duration with higher adiposity measures and lower %LM and LFR in women but not in men. Women with short sleep had the highest mean adiposity measures, and higher than average levels (all *z*-score >0), while the moderate sleep group and the long-sleep group had relatively low *z*-scores for each adiposity measure (**Figure 3**). Compared to subjects with moderate sleep durations, women with short-sleep durations had higher *z*-scores (0.248–0.317) for adiposity measures

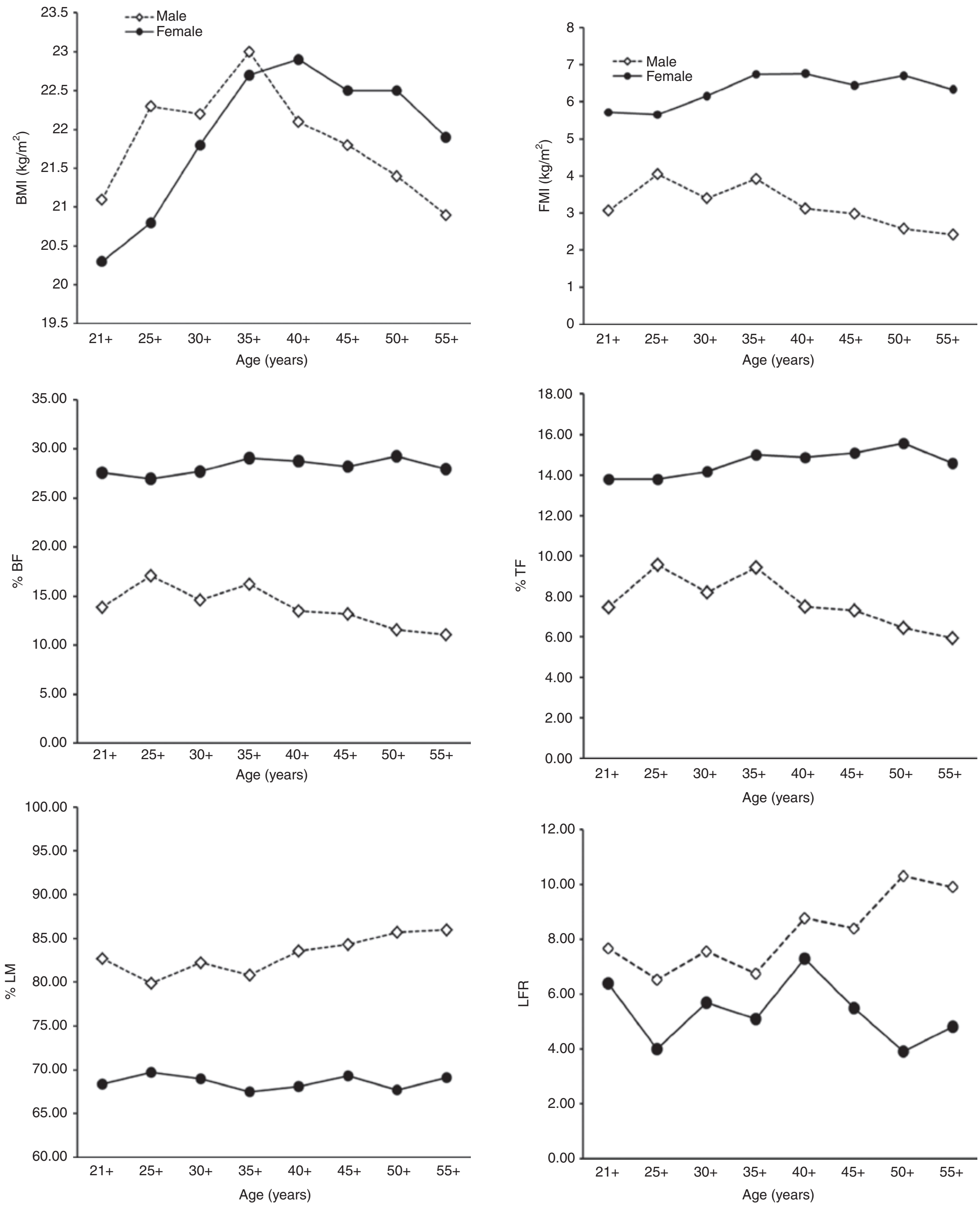

Figure 2 Mean body composition measures by age in males and females. FMI, fat mass index; LFR, lean/fat mass ratio; %BF, percent body fat mass; %LM, percent body lean mass; %TF, percent trunk fat mass; WC, waist circumference.

(BMI, FMI, %BF, and %TF, $P < 0.05$ for each) and lower %LM z-scores and LFR ($\beta = -0.306$, SE = 0.105, $P = 0.0035$, and $\beta = -0.353$, SE = 0.104, $P = 0.0007$) after adjustment for age, physical activity, education level, and menopause (**Table 2**). Using the same reference group (moderate sleep duration), no associations were found between long-sleep duration and any body composition measures in either gender. Similar relationships between sleep duration and each adiposity measure were observed when we excluded subsamples of participants who were determined to have habitual snoring or sleep disturbance (data not shown).

We examined the relative contribution of genetic and environmental influences on the body composition measures (BMI, FMI, %BF, %TF, %LM, and LFR) and sleep duration. We present here the gender-specific modeling result in **Table 3**, which includes 475 same-sex pairs only (male MZ = 110, DZ = 76; female MZ = 222, DZ = 66). BMI, FMI, %BF, %TF, %LM, and LFR were the traits that showed a strong genetic

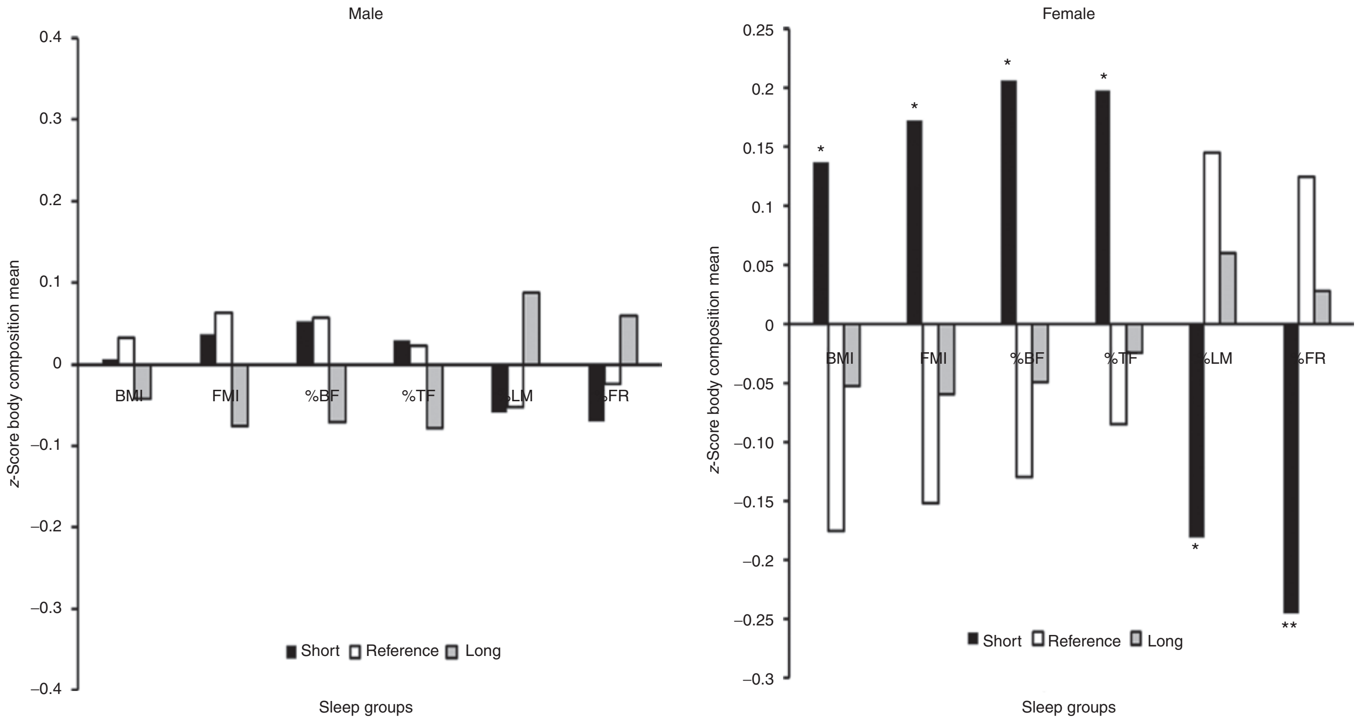

Figure 3 Least square means of gender-specific *z*-score of body composition measures by sleep duration groups. **P* < 0.01; ***P* < 0.001, compared with reference group. Adjusted for age and intratwin pair corelation. Sleep duration in the first and fourth age-specific quartile of sleep duration was classified as short and long, respectively, and the remaining (second and third quartiles) was classified as a reference group. FMI, fat mass index; LFR, lean/fat mass ratio; %BF, percent body fat mass; %LM, percent body lean mass; %TF, percent trunk fat mass.

component. This was reflected by a higher intrapair correlation (which measures the within-pair similarity of the traits) in MZ twins than in DZ twins (Table 3). After adjustment for age, the estimates of heritability (the ratio of genetic variance to the total phenotypic variance) for body composition measures (BMI, FMI, %BF, %TF, %LM, and LFR) ranged from 0.61 to 0.72 in women and 0.56 to 0.73 in men. In contrast, sleep duration appears to be primarily environmentally determined, with an estimated heritability of 0.27 (95% CI: 0.15–0.39) in men and 0.29 (95% CI: 0.13–0.44) in women. The statistical tests of gender difference for each measure are presented as **Supplementary Table S1** online, and the overall composite model, with men and women combined, **Supplementary Tables S2** and **S3** online.

DISCUSSION

To our knowledge, this is the first study to examine the gender-specific association of sleep duration with body composition as assessed by DXA and to estimate heritability of sleep duration using a cotwin design in a rural Chinese population. This study is further strengthened in that components of body composition (%BF, FMI, %LM, and LFR) were measured by DXA, a technique that can directly assess total and regional body fat mass. Besides sleep duration, we also considered other sleep quality factors such as sleep disturbance and habitual snoring. We performed additional sensitivity analyses to ensure that these potentially important factors did not confound the sleep duration–body composition relationships. In addition, we used age-specific quartiles of sleep duration to define short-sleep duration in this study because, even in healthy adults, sleep duration decreases with age (22).

This study has generated several new and interesting results. First, we found that women with short-sleep duration (the lowest quartile) had higher overall and central adiposity and lower lean body mass when compared to those with moderate sleep duration (2nd and 3rd quartiles). The association persisted even after exclusion of subjects who reported either habitual snoring or sleep disturbance, suggesting that the sleep duration itself is a potential determinant of increased adiposity. As we will further discuss below, both our previous study and the current study showed a clear gender difference in that men did not show the same effect that we saw in women, as reported above. Additionally, we found that sleep duration is primarily determined by environmental factors (as evident in low heritability). This is in contrast to body composition indexes, which have a strong genetic component (as evident in high heritability). Our findings are consistent with previous sleep studies (23) and body composition studies (24,25).

Our findings appear to be biologically plausible. The mechanisms underlying the association of sleep and weight have been demonstrated by a series of laboratory studies. Spiegel *et al.* found that sleep restriction in healthy young men resulted in reduced leptin and increased ghrelin, which was followed by an increase in appetite for nutrients with

Table 2 Association between sleep duration and sex-specific *z*-score of body composition measures

Sleep group	*z*-Score of adiposity measures			
	Male (n = 738)		Female (n = 511)	
	β (SE)	*P* value	β (SE)	*P* value
BMI				
Short	−0.041 (0.075)	0.581	0.302 (0.097)	0.0019
Reference		—		—
Long	−0.074 (0.075)	0.323	0.121 (0.093)	0.1931
FMI				
Short	−0.042 (0.077)	0.585	0.309 (0.106)	0.0035
Reference		—		—
Long	−0.139 (0.074)	0.059	0.084 (0.097)	0.3903
%BF				
Short	−0.020 (0.079)	0.805	0.317 (0.105)	0.0026
Reference		—		—
Long	−0.128 (0.075)	0.088	0.067 (0.101)	0.5113
%TF				
Short	−0.027 (0.079)	0.729	0.264 (0.103)	0.0102
Reference		—		—
Long	−0.109 (0.076)	0.150	0.047 (0.102)	0.6441
%LM				
Short	0.010 (0.078)	0.901	−0.306 (0.105)	0.0035
Reference		—		—
Long	0.141 (0.076)	0.062	−0.069 (0.101)	0.4941
LFR				
Short	−0.031 (0.082)	0.704	−0.353 (0.104)	0.0007
Reference		—		—
Long	0.087 (0.080)	0.275	−0.083 (0.109)	0.4460

All regression models were adjusted for age, education level, and physical activity level (for all subjects), smoking and alcohol consumption (for men only), and menopause (for women only). GEE models were applied to account for intratwin pair correlation.
FMI, fat mass index; GEE, generalized estimating equation; LFR, lean/fat mass ratio; %BF, percent body fat mass; %LM, percent body lean mass; %TF, percent trunk fat mass.

high carbohydrate content (26–28). Two population-based studies in adults, the Wisconsin Sleep Cohort Study (3) and the Quebec Family Study (2), also observed an association between short-sleep duration and changes in leptin and/or ghrelin consistent with an up-regulation of appetite. It also has been reported that growth hormone, secreted mainly during slow wave sleep, is suppressed by reduced sleep duration (29). Furthermore, reduced rapid eye movement sleep may increase cortisol levels (30). The metabolic effects (31,32) of decreased growth hormone and increased cortisol, both as a result of short sleep, also could contribute to obesity.

In our study, the association of short-sleep duration with body composition was just observed in women. This gender difference was consistent to what have been reported in our earlier adolescent study (4) and VISAT study (33), CARDIA study (34), and the Hong Kong Chinese study (5), but discrepant from what has been observed by Heslop *et al.* (35). We speculate that a growing gender difference in body composition at puberty and beyond might account for the gender-specific association we observed in adolescents in our earlier publication (4), and again in adults in the current study. There are some noticeable gender differences in the covariates. In our population, because few women smoked or consumed alcohol, we did not include smoking and alcohol consumption as covariates in the regression models. Yet, since the impact of menopause on weight and sleep is well known, we included menopause in the female regression models. The most remarkable gender difference was in the body composition. In contrast to a Western population, this study population was much leaner, especially the elderly male adults. Despite the fact that a number of studies (36–39) have reported age-related increases in body weight and fat mass, our data showed a noticeable reduction in direct adiposity measurements with increasing age in men. Such a preponderance of lean men in our study may have limited our ability to detect the association seen in populations with higher BMI. Our findings underscore the importance of performing gender-specific analyses in assessing sleep and adiposity relationships in both adolescents and adults.

Several limitations of this study should be noted. Using a cross-sectional design, it is impossible to determine the temporal relation between short-sleep duration and increased adiposity measures. In addition, we did not collect information on parity, which may affect a woman's sleep duration and fat mass. Also, because the sleep data were based on sleep questionnaires, reporting bias is a possibility. If it was nondifferential, it would bias the association toward null. If MZ pairs erroneously reported sleep more similarly than DZ pairs, this would have led to inflated heritability estimation; if it was the other way around, it would have led to attenuated heritability estimation. However, the questionnaires have been validated against quantitative sleep assessment (40). Moreover, the association between sleep and body composition may be because of yet unmeasured or unknown factors that may differ from population to population. The question also remains of whether or not Chinese twins generalize to other populations in terms of sleep quality and its relationship to body measures. Nevertheless, our findings are consistent with previous studies (8,9,12–13) in other populations on short-sleep duration and BMI.

In summary, in this relatively lean rural Chinese twin population, short-sleep duration was associated with increased body fat and decreased lean body mass in women but not in men. Sleep duration is largely influenced by environmental factors while body composition indexes are mainly influenced by genetic factors. While these findings remain to be confirmed in other populations, our study raises the possibility that the promotion of healthy sleep habits may be a simple and effective way to prevent or reduce the risk of obesity and its consequences in this population.

Table 3 Estimated genetic (A), common environmental (C), and unique environmental (E) effect on body composition indexes and sleep duration (s.d.) in Anqing Chinese twin study

	MZ[b]	DZ[b]	A (95% CI)	E (95% CI)	χ^2	*P* value	AIC
Male							
BMI	0.73	0.42	0.73 (0.66–0.78)	0.27 (0.22–0.34)	0.70	0.40	−1.303
FMI	0.65	0.46	0.66 (0.58–0.72)	0.34 (0.27–0.42)	2.70	0.10	0.695[c]
%BF	0.64	0.43	0.64 (0.56–0.71)	0.36 (0.29–0.44)	1.95	0.16	−0.054
%TF	0.63	0.45	0.65 (0.56–0.71)	0.35 (0.29–0.44)	2.75	0.10	0.753[c]
%LM	0.62	0.47	0.64 (0.55–0.71)	0.36 (0.29–0.44)	3.32	0.07	1.318[c]
LFR	0.54	0.37	0.56 (0.46–0.65)	0.44 (0.35–0.54)	0.96	0.33	−1.04
Sleep duration	0.28	0.14[a]	0.27 (0.15–0.39)	0.73 (0.61–0.85)	0.035	0.85	−1.97
Female							
BMI	0.71	0.31	0.72 (0.63–0.79)	0.28 (0.21–0.37)	0.24	0.62	−1.76
FMI	0.63	0.27	0.65 (0.55–0.72)	0.35 (0.28–0.45)	0.17	0.68	−1.83
%BF	0.56	0.29	0.61 (0.51–0.70)	0.39 (0.30–0.49)	0.10	0.75	−1.90
%TF	0.58	0.32	0.62 (0.52–0.70)	0.38 (0.30–0.48)	0.59	0.44	−1.41
%LM	0.59	0.29	0.63 (0.53–0.72)	0.37 (0.28–0.47)	0.00	—	−2.00
LFR	0.53	0.20[a]	0.61 (0.50–0.70)	0.38 (0.30–0.50)	0.00	—	−2.00
Sleep duration	0.29	0.17[a]	0.29 (0.13–0.44)	0.71 (0.56–0.87)	0.12	0.091	−1.99

Chi-square and AIC were used to compare the goodness of fit of the models (AE and CE models) with the saturated model (ACE model). Only the estimates from the best-fit models are presented. Male and female estimates were not statistically significant in variance of BMI, FMI, and sleep duration.
AIC, Akaike's Information Criterion; CI, confidence interval; DZ, dizygotic; FMI, fat mass index; MZ, monozygotic; %BF, percent body fat mass; %LM, percent body lean mass; %TF, percent trunk fat mass; LFR, lean/fat mass ratio.
[a]Not significant at $P < 0.05$. [b]Adjusted for age. [c]Although ACE is the best-fit according to AIC, the 95% CI included zero. So the AE models presented as χ^2-tests were not significant.

SUPPLEMENTARY MATERIAL

Supplementary material is linked to the online version of the paper at http://www.nature.com/oby

ACKNOWLEDGMENTS

This study is supported in part by grants R01 HD049059 from the National Institute of Child Health and Human Development; R01 HL0864619 from the National Heart, Lung, and Blood Institute; and R01 AG032227 from the National Institute of Aging. We thank Tami Bartell for intensive English editing.

DISCLOSURE

The authors declared no conflict of interest.

REFERENCES

1. Low S, Chin MC, Deurenberg-Yap M. Review on epidemic of obesity. *Ann Acad Med Singap* 2009;38:57–59.
2. Chaput JP, Després JP, Bouchard C, Tremblay A. Short sleep duration is associated with reduced leptin levels and increased adiposity: Results from the Quebec family study. *Obesity (Silver Spring)* 2007;15:253–261.
3. Taheri S, Lin L, Austin D, Young T, Mignot E. Short sleep duration is associated with reduced leptin, elevated ghrelin, and increased body mass index. *PLoS Med* 2004;1:e62.
4. Yu Y, Lu BS, Wang B *et al*. Short sleep duration and adiposity in Chinese adolescents. *Sleep* 2007;30:1688–1697.
5. Ko GT, Chan JC, Chan AW *et al*. Association between sleeping hours, working hours and obesity in Hong Kong Chinese: the 'better health for better Hong Kong' health promotion campaign. *Int J Obes (Lond)* 2007;31:254–260.
6. Van Cauter E, Knutson KL. Sleep and the epidemic of obesity in children and adults. *Eur J Endocrinol* 2008;159 Suppl 1:S59–S66.
7. 2009 Sleep in Americatm Poll Summary of Findings <http://www.sleepfoundation.org/article/sleep-america-polls/2009-health-and-safety> (2010).
8. Kohatsu ND, Tsai R, Young T *et al*. Sleep duration and body mass index in a rural population. *Arch Intern Med* 2006;166:1701–1705.
9. Chaput JP, Brunet M, Tremblay A. Relationship between short sleeping hours and childhood overweight/obesity: results from the 'Québec en Forme' Project. *Int J Obes (Lond)* 2006;30:1080–1085.
10. Chaput JP, Després JP, Bouchard C, Tremblay A. Association of sleep duration with type 2 diabetes and impaired glucose tolerance. *Diabetologia* 2007;50:2298–2304.
11. Ohayon MM. Interactions between sleep normative data and sociocultural characteristics in the elderly. *J Psychosom Res* 2004;56:479–486.
12. 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;24:1683–1688.
13. Chaput JP, Després JP, Bouchard C, Tremblay A. The association between sleep duration and weight gain in adults: a 6-year prospective study from the Quebec Family Study. *Sleep* 2008;31:517–523.
14. 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;27:661–666.
15. Gangwisch JE, Malaspina D, Boden-Albala B, Heymsfield SB. Inadequate sleep as a risk factor for obesity: analyses of the NHANES I. *Sleep* 2005;28:1289–1296.
16. Flier JS. Obesity wars: molecular progress confronts an expanding epidemic. *Cell* 2004;116:337–350.
17. Obici S. Minireview: Molecular targets for obesity therapy in the brain. *Endocrinology* 2009;150:2512–2517.
18. NIMH. Symptoms of Depression and Mania. <http://www.nimh.nih.gov/health/publications/men-and-depression/symptoms-of-depression-and-mania.shtml> (2011).
19. Buysse DJ, Reynolds CF 3rd, Monk TH, Berman SR, Kupfer DJ. The Pittsburgh Sleep Quality Index: a new instrument for psychiatric practice and research. *Psychiatry Res* 1989;28:193–213.
20. Liu X, Tang M. Reliability and Validity of Pittsburgh Sleep Quality Index. *Chinese Journal of Psychiatry* 1996;29:103–107.

21. Ouyang F, Lu BS, Wang B *et al*. Sleep patterns among rural Chinese twin adolescents. *Sleep Med* 2009;10:479–489.
22. Dijk DJ, Groeger JA, Stanley N, Deacon S. Age-related reduction in daytime sleep propensity and nocturnal slow wave sleep. *Sleep* 2010;33:211–223.
23. de Castro JM. The influence of heredity on self-reported sleep patterns in free-living humans. *Physiol Behav* 2002;76:479–486.
24. Zhang S, Liu X, Yu Y *et al*. Genetic and environmental contributions to phenotypic components of metabolic syndrome: a population-based twin study. *Obesity (Silver Spring)* 2009;17:1581–1587.
25. Ouyang F, Christoffel KK, Brickman WJ *et al*. Adiposity is inversely related to insulin sensitivity in relatively lean Chinese adolescents: a population-based twin study. *Am J Clin Nutr* 2010;91:662–671.
26. Spiegel K, Tasali E, Penev P, Van Cauter E. Brief communication: Sleep curtailment in healthy young men is associated with decreased leptin levels, elevated ghrelin levels, and increased hunger and appetite. *Ann Intern Med* 2004;141:846–850.
27. Spiegel K, Leproult R, Van Cauter E. Impact of sleep debt on metabolic and endocrine function. *Lancet* 1999;354:1435–1439.
28. Spiegel K, Leproult R, L'hermite-Balériaux M *et al*. Leptin levels are dependent on sleep duration: relationships with sympathovagal balance, carbohydrate regulation, cortisol, and thyrotropin. *J Clin Endocrinol Metab* 2004;89:5762–5771.
29. Spiegel K, Leproult R, Colecchia EF *et al*. Adaptation of the 24-h growth hormone profile to a state of sleep debt. *Am J Physiol Regul Integr Comp Physiol* 2000;279:R874–R883.
30. Van Cauter E, Leproult R, Plat L. Age-related changes in slow wave sleep and REM sleep and relationship with growth hormone and cortisol levels in healthy men. *JAMA* 2000;284:861–868.
31. Aron DC, Findling JW, Tyrrell JB. Hypothalamus & pituitary gland. In: Gardner DG, Shoback D (eds). *Greenspan's Basic & Clinical Endorinology*. The McGraw-Hill Companies Inc.: New York City, NY, 2007, pp 114–116.
32. Aron DC, Findling JW, Tyrrel JB. Glucocorticoids & adrenal androgens. In: Gardner DG, Shoback D (eds). *Greenspan's Basic & Clinical Endocrinology*. The McGraw-Hill Companies Inc.: New York City, NY, 2007, pp 356–363.
33. Cournot M, Ruidavets JB, Marquié JC *et al*. Environmental factors associated with body mass index in a population of Southern France. *Eur J Cardiovasc Prev Rehabil* 2004;11:291–297.
34. St-Onge MP, Perumean-Chaney S, Desmond R *et al*. Gender Differences in the Association between Sleep Duration and Body Composition: The Cardia Study. *Int J Endocrinol* 2010;2010:726071.
35. Heslop P, Smith GD, Metcalfe C, Macleod J, Hart C. Sleep duration and mortality: The effect of short or long sleep duration on cardiovascular and all-cause mortality in working men and women. *Sleep Med* 2002;3:305–314.
36. Roberts SB, Dallal GE. Effects of age on energy balance. *Am J Clin Nutr* 1998;68:975S–979S.
37. Guo SS, Zeller C, Chumlea WC, Siervogel RM. Aging, body composition, and lifestyle: the Fels Longitudinal Study. *Am J Clin Nutr* 1999;70:405–411.
38. Perissinotto E, Pisent C, Sergi G, Grigoletto F; ILSA Working Group (Italian Longitudinal Study on Ageing). Anthropometric measurements in the elderly: age and gender differences. *Br J Nutr* 2002;87:177–186.
39. Hughes VA, Frontera WR, Roubenoff R, Evans WJ, Singh MA. Longitudinal changes in body composition in older men and women: role of body weight change and physical activity. *Am J Clin Nutr* 2002;76:473–481.
40. Lockley SW, Skene DJ, Arendt J. Comparison between subjective and actigraphic measurement of sleep and sleep rhythms. *J Sleep Res* 1999;8:175–183.