ABSTRACT

Objective: To investigate the relationship between short sleep and obesity among Thai adults. Design: Both 4-year longitudinal and cross-sectional analyses of a large national cohort. Setting: Thai adults residing nationwide from 2005 to 2009. Participants: Cohort members were enrolled as distance learners at Sukhothai Thammathirat Open University (N=87,134 in 2005 and 60,569 at 2009 follow-up). At 2005 baseline, 95% were between 20 and 49 years of age. Measures: Self-reported sleep duration was categorised as <6, 6-7, 8 and ≥9 h. For all analyses (2005 and 2009 cross-sectional and 2005-2009 longitudinal), we used multinomial logistic regression models to assess the effect of sleep duration on abnormal body size (underweight, overweight-at-risk, obese). Results were adjusted for an array of relevant covariates. Results: At the last cohort follow-up in 2009, cross-sectional associations linked short sleep (<6 h) and obesity: adjusted ORs (AOR) = 1.49, 95% CIs 1.32 to 1.68 for women and AOR = 1.36, 95% CI 1.21 to 1.52 for men. The earlier cross-sectional baseline results in 2005 were quite similar. Longitudinal analysis (2005-2009) of 4-year incremental weight gain (5 to <10%, 10 to <20% and 20%+) strongly supported the short sleep—obesity relationship (significant AORs of 1.10, 1.30 and 1.69, respectively). Conclusions: The results are internally consistent (2005 and 2009) and longitudinally confirmatory of a short sleep effect on obesity among Thai adults. Further research is needed to elucidate causal mechanisms underlying the sleep—obesity relationship.

INTRODUCTION

Short sleep has increased in prevalence over the last century and may now be contributing to the obesity epidemic. A causal association is most accepted for children and adolescents. Evidence remains inconclusive for adults. But recent research has shown that short sleeping young adults are at increased risk of obesity and of pre-diabetic levels of glycosylated haemoglobin. And many reports suggest potential biological mechanisms linking short sleep and obesity, including obstructive sleep apnoea, insulin resistance, stress, high levels of cortisol and ghrelin, low levels of leptin and adiponectin and disordered thermoregulation.

The evidence for a short sleep—obesity effect in adults is mostly based on cross-sectional data. However, two large prospective adult cohort studies in the USA report substantive longitudinal weight gain associated with short sleep. Other supportive longitudinal evidence of a short sleep—obesity effect in prospective cohort studies was found among adults in Canada, young adults in Switzerland and older adults in Spain. There have been very few longitudinal studies of adults in Asia but one retrospective cohort study...
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in Japan24 and another study of serial population panels in Korea25 both show consistent and significant associations between short sleep and obesity.

We examine short sleep and obesity in our large national cohort study of 87 134 Thai adults. Our report is based on a longitudinal analysis for the 4-year period from 2005 to 2009 and on cross-sectional analyses at baseline (2005) and follow-up (2009). We investigated the association between sleep duration and weight, adjusting for a wide array of potential confounders.

METHODS AND ANALYSIS

This study is part of an overarching research project entitled The Thai Health-Risk Transition: a National Cohort Study, which is examining the ongoing transition from traditional to modern patterns of risk and disease. In 2005, the study recruited 87 134 distance learning adult students enrolled at the Sukhothai Thammathirat Open University and residing nationwide.26 The 20-page baseline questionnaire covered socio-demographic characteristics, personal income categories (baht/month), rural–urban geographical residence, self-reported health risk behaviour including smoking (never, current and previous) or drinking (days/week), fruit and vegetable intakes (serves/day), vigorous or moderate physical activity (sessions/week), screen time (hours/day), doctor-diagnosed depression and doctor-diagnosed chronic disorders including type I and type II diabetes, high cholesterol, high blood pressure, heart disease, stroke, cancers (liver, lung, stomach, colon, breast and others), goitre, epilepsy, liver disease, lung disease, arthritis and asthma. These covariates were chosen based on our experience with risk factors of obesity in our cohort as well as international literature.35 36

We analysed men and women separately as our data show the occurrence of abnormal body size, and the socioeconomic associations vary by sex.32 33 For data scanning and editing, we used Thai Scandevet, SQL and SPSS software. For analysis, we used SPSS V.16 and Stata V.10. Individuals with missing data (<10%) were excluded from multivariable analyses.

RESULTS

We present the most recent cross-sectional results (2009) and the longitudinal results for 2005–2009 data. The 2005 cross-sectional data were analysed, but results are not shown because they were very similar to 2009. At the follow-up in 2009, cohort weight results were as follows: 9.5% underweight (BMI <18.5), 48.5% normal (18.5 to <23), 18.4% overweight-at-risk (23 to <25) and 21.7% obese (≥25). Underweight was most common among women aged between 20 and 29 years (23.5%), while overweight-at-risk and obesity were most common among men aged ≥45 years (28.2% and 40.3%).

Among the cohort members responding in 2009, there is a parallel trend between increasing BMI and older age, being married and higher income (table 1). Overweight and obesity increased in frequency with less than four daily serves of fruits and vegetables (especially among men), less than three physical activity sessions per week (especially among women) and >5 h of screen time per day (both for men and women). Increased BMI also was more prevalent among those with doctor-diagnosed chronic conditions.

In 2009, 15% of the cohort reported short sleep duration (<6 h/day) (table 2) and one-fourth and one-third of cohort members reported sleeping 7 or 8 h daily. Overweight-at-risk was associated with short sleep among women (adjusted ORs (AOR) = 1.33, 95% CI 1.18 to 1.51). Obesity had a stronger association with short sleep for women (AOR=1.49, 95% CI 1.32 to 1.68) but was also associated with short sleep for men (AOR=1.36, 95% CI 1.21 to 1.54). Also noted was an association between long sleep duration and obesity for both sexes.
For longitudinal analysis, we performed multinomial logistic regression of sleep duration on incident weight gain, adjusting for the same covariates as for the cross-sectional analyses. The dependent variable was substantive weight gain between 2005 and 2009, defined by three weight increase categories (5% to <10%, 10% to <20% and 20+%). Each category of weight gain was separately compared with no weight increase (ie, <5%) for calculation of ORs. Progressively larger increments in weight gain show progressively stronger significant associations with short sleep (<6 h), after adjusting for covariates (AOR=1.10, 95% CI 1.02 to 1.20; AOR=1.30, 95% CI 1.18 to 1.44 and AOR=1.69, 95% CI 1.39 to 2.05).

**DISCUSSION**

Our large cohort study revealed a consistent and substantial association between short sleep and obesity among Thai adults. Significant covariate AORs for the short sleep–obesity effect were 1.36 for men and 1.49 for women in 2009; corresponding ORs were similar in 2005. Longitudinal 2004–2009 cohort data confirmed a short sleep–weight gain relationship with a notable dose–response.

Cohort evidence from the USA is supportive. The Nurses Health Study analysed sleep and weight in a cohort of women for 16 years and found that those with ≤5 h sleep had more weight gain (additional...
and obesity.14 17

occurs, there is a strong relationship to both short sleep to obstructive sleep apnoea and when that condition


Ratios (HRs) were 1.28 and 1.10 for

extreme with less sleep: for a 15 kg weight gain, Hazard

respectively.19 Even more extreme sleep deprivation was

were underweight, and other reports suggest that this

may be related to comorbidity.41 42 However, our results

were adjusted for depression and 17 chronic health

conditions, so we do not have an explanation to link

long sleep and underweight. Nor do we understand the

U-shaped risk pattern whereby both short and long sleep

were associated with obesity in our Thai cohort. Such

a pattern has been observed in other reports.8 9 35

We investigated personal environments and found that

short sleep among our Thai cohort was more

common for those who lack a fan or an air conditioner

at home; at baseline in 2005, this affected 26% of short

sleepers and 13% of others. However, we found no link

between noisy home environments, hours of work, rural

or urban location and short sleep duration. But we did

note an occupational travel time effect: in 2005, short

sleep was reported by 17%, 14% and 12% of those

travelling

1 h, 30 min to 1 h and 30 min to work,

One limitation of this study was the subjective nature

of self-reporting sleep duration, a problem noted by

many others conducting sleep research.9 10 Weight and

height of cohort members were also self-reported, but

our previous validation study has found these numbers to

be reliable.27 We also noted the relatively higher level of

education among cohort members, but it is unclear

how this could affect the results.

The strength of this study was the large sample,

longitudinal data, national representation, involvement of

young adults and the opportunity to control for many
covariates in the analyses. We noted similar cross-

sectional results for both 2005 and 2009 although on

both occasions, cohort members were unaware of any

hypothesised relationship between sleep and weight.

Even more compelling was the evidence from the 4-year

longitudinal analyses yielding similar results.

<table>
<thead>
<tr>
<th>Sleep duration (men)</th>
<th>Overall, N=60569</th>
<th>Weight outcomes*</th>
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<tbody>
<tr>
<td>Sleep duration</td>
<td>n (%)</td>
<td>Underweight versus normal AOR† (95% CI)</td>
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<tr>
<td>&lt;6 h</td>
<td>4070 (14.9)</td>
<td>1.16 (0.90 to 1.50)</td>
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<tr>
<td>6 h</td>
<td>4815 (17.6)</td>
<td>0.90 (0.71 to 1.16)</td>
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<td>7 h</td>
<td>5719 (20.9)</td>
<td>Ref</td>
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<td>8 h</td>
<td>8664 (31.6)</td>
<td>1.15 (0.94 to 1.42)</td>
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<tr>
<td>≥9 h</td>
<td>4139 (15.1)</td>
<td>1.08 (0.84 to 1.39)</td>
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<tr>
<td>Sleep duration (women)</td>
<td>Overall, N=60569</td>
<td>Weight outcomes*</td>
</tr>
<tr>
<td>Sleep duration</td>
<td>n (%)</td>
<td>Underweight versus normal AOR† (95% CI)</td>
</tr>
<tr>
<td>&lt;6 h</td>
<td>4728 (14.3)</td>
<td>0.97 (0.85 to 1.11)</td>
</tr>
<tr>
<td>6 h</td>
<td>5120 (15.4)</td>
<td>0.99 (0.87 to 1.12)</td>
</tr>
<tr>
<td>7 h</td>
<td>6965 (21.0)</td>
<td>Ref</td>
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<tr>
<td>8 h</td>
<td>11007 (33.2)</td>
<td>1.05 (0.95 to 1.16)</td>
</tr>
<tr>
<td>≥9 h</td>
<td>5342 (16.1)</td>
<td>1.21† (1.07 to 1.37)</td>
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</table>

*Body mass index (BMI) categories: normal = 18.5 to <23; underweight = <18.5; overweight-at-risk = 23 to <25; obese = ≥25. For each ‘abnormal’ category, multinomial logistic regression compares the outcome odds to the outcome odds of a ‘normal’ BMI, with the results expressed as an Odd Ratio (OR).

†Adjusted ORs—covariates included age, marital status, personal income, geographical rural—urban residence, physical activity, fruit and vegetable intakes, screen time, smoking, alcohol drinking, doctor-diagnosed depression and chronic health conditions (see the Methods section).

†Bolded values indicate statistical significance at p<0.05.
CONCLUSIONS

Our large adult cohort in Thailand shows a consistent relationship between short sleep duration and obesity. This is one of the few sleep–obesity studies in a middle-income country, one of the first in Southeast Asia and one of only a few longitudinal studies investigating this topic worldwide. The epidemiological evidence available now points to a consistent, substantial and presumably causal association for adults but mechanisms should be further explored.

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Competing interests

None.

Patient consent

Obtained.

Ethics approval

Obtained from the Sukhothai Thammathirat Open University Research and Development Institute (protocol 0522/10) and the Australian National Health and Medical Research Council (2004344 and 2009570). Informed consent was obtained from all participants.

Contributors

EVT and CB conceptualised and designed the study. AS and SS devised and directed the cohort study. VY analysed and drafted the manuscript, with input by AS. All authors approved the final manuscript submission.

Provenance and peer review

Not commissioned; externally peer reviewed.

Data sharing statement

Additional unpublished data are available upon request, please contact Principal Investigators Professor Adrian C Sleigh (adrian.sleigh@anu.edu.au) or Associate Professor Sam-ang Seubsman (sam-ang.seu@stou.ac.th).

REFERENCES