Original Contribution

Do Childhood Sleeping Problems Predict Obesity in Young Adulthood? Evidence from a Prospective Birth Cohort Study

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It has been suggested that sleeping problems are causally associated with obesity in early life, but most studies examining this association have been cross-sectional. The authors used a population-based birth cohort of 2,494 children who were born between 1981 and 1983 in Brisbane, Australia, to examine the prospective association between early-life sleeping problems (at ages 6 months and 2–4 years) and obesity at age 21 years. The authors compared mean body mass indices (BMIs; weight (kg)/height (m)²) and persons in the categories of overweight (BMI 25.0–29.9) and obesity (BMI ≥30) among offspring at age 21 years according to maternally reported childhood sleeping problems. They found that young adult BMI and the prevalence of obesity were greater in offspring who had had sleeping problems at ages 2–4 years than in those who had not had sleeping problems. These associations were robust to adjustment for a variety of potential confounders, including offspring sex, maternal mental health, and BMI, and several mediators, including adolescent dietary patterns and television-watching. These findings provide some evidence for a long-term impact of childhood sleeping problems on the later development of obesity.

adult; body mass index; child; obesity; overweight; sleep

Abbreviations: BMI, body mass index; CI, confidence interval; SD, standard deviation.

There is increasing research interest in the role of sleep in obesity and its associated insulin resistance and cardiovascular health, with prospective studies in adults showing shorter sleep duration to be associated with an increased risk of obesity, all-cause mortality, and cardiovascular mortality (1, 2). In a recent review of studies carried out in children, Taheri concluded that there was now sufficient experimental and population-based evidence to support an effect of sleep on energy balance and hence obesity, stating “good sleep could be part of the obesity prevention approach” (3, p. 881). However, of the 13 population-based studies reviewed, only two were prospective, with the remaining 11 being cross-sectional (3). Since obesity appears to be causally associated with sleep apnea and other breathing disorders (4), which could compromise sleep quantity and quality, it is unclear from cross-sectional studies whether obesity results in reduced hours of sleep (reverse causality) or vice versa.

Of the two longitudinal studies published to date (5, 6), one, a prospective study of a pregnancy cohort from southwestern England, examined 25 possible predictors of obesity, all assessed at age 30 months, with obesity being based on body mass index (BMI) calculated from weight and height measured at age 7 years (5). Those investigators found that eight of the 25 predictors were independently associated with obesity, and they found a dose response

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The study

The Mater-University Study of Pregnancy and Its Outcome is a prospective study of 7,223 women, and their offspring, who received antenatal care at a major public hospital in Brisbane, Australia, between 1981 and 1983 and delivered a live singleton child who was not adopted before leaving the hospital (7, 8). These mothers and their offspring have been followed prospectively, with maternal questionnaires being administered when the offspring were aged 6 months, 5 years, 14 years, and 21 years. In addition, at ages 5, 14, and 21 years, detailed physical, cognitive, and developmental examinations of the offspring have been undertaken, and at ages 14 and 21 years, the offspring have completed health, welfare, and lifestyle questionnaires.

In the current study, the main analyses were restricted to 2,494 offspring for whom we had prospective information on sleeping problems at ages 6 months and 2–4 years and data on measured height and weight at age 21 years. Half of the 2,494 young adults were males. On average, they were 5.02 years old (standard deviation, SD, 0.50) at the 5-year follow-up, 13.50 years old (SD, 0.41) at the 14-year follow-up, and 20.41 years old (SD, 0.83) at the 21-year follow-up. Written informed consent was obtained from the mothers in all data collection phases and from the young adults at the 21-year follow-up. Ethics committees at the Mater Hospital and the University of Queensland approved each phase of the study. Full details on the study participants, sociodemographic data, and measurements have been previously reported (7, 8).

**Outcomes.** Young adult BMI (weight (kg)/height (m)²) and the BMI categories of normal, overweight, and obese were the main outcomes in this study. In all assessments of height and weight, the average of two measurements of the participant’s weight, taken with a scale accurate to 0.2 kg while the participant was lightly clothed, was used. A portable stadiometer was used to measure height. BMI categories (normal = BMI 18.5–24.9, overweight = BMI 25.0–29.9, and obese = BMI ≥30) were defined according to the criteria of the World Health Organization (9).

**Exposures: defining sleeping problems.** Sleeping problems in young children are often defined on the basis of nighttime awakenings and problems with sleep onset; however, there is no widely accepted definition (10). In this study, at 6 months and 5 years of follow-up, mothers were asked whether their children had irregular or troubled sleeping. At 6 months of follow-up, the mother was asked, “How often does your baby have the following problems?” “Sleeplessness” was one of the options, with response categories being “almost every day,” “a few times a month,” “a few times a week,” “rarely,” or “never.” At 5 years of follow-up, the mother was asked, “As you remember your child’s behavior between 2 and 4 years of age, did he/she have irregular sleeping habits?” Response options were “often,” “sometimes,” “rarely,” and “never.” For this study, maternal responses were collapsed into three categories for each follow-up time: “rarely/never,” “sometimes” (corresponding to “a few times a month” at the 6-month follow-up and “sometimes” at the 5-year follow-up), and “often” (corresponding to “almost every day” or “a few times a week” at the 6-month follow-up and “often” at the 5-year follow-up).

**Confounders and mediators**

The following maternal and family characteristics were considered to be potentially confounding factors on the basis of their possible association with BMI in early adulthood and sleeping problems during childhood and adolescence: maternal age at child’s birth, maternal prepregnancy BMI (based on prepregnancy weight and height, as reported by mothers at the first antenatal clinic visit), maternal education at child’s birth (did not complete secondary school, completed secondary school, completed further/higher education), maternal depression (assessed with the Delusions-Symptoms-States Inventory (11); mothers were classified as depressed if they reported three or more of seven symptoms related to depression), marital status (married, de facto married (cohabiting or in committed relationship), single, or other), and maternal smoking (none, 1–9 cigarettes/day, or 10 or more cigarettes/day) at 6 months of follow-up. To examine the possibility that any associations of sleeping problems in childhood with obesity at age 21 years were mediated by childhood-adolescent diet or physical activity, we examined the effect of adjustment for diet, physical activity, and sports participation, assessed by maternal report when the offspring were aged 14 years, on these associations. Diet and physical activity were not assessed in early childhood, but since these characteristics track from childhood to adolescence, our measurements at age 14 years will have captured earlier characteristics to some extent. At the 14-year follow-up, mothers were asked to report the frequency of their child’s consumption of fast food, salad, soft drinks, and red meat (all with response options of “rarely or never,” “at least two or three times a week,” and “most days”), the frequency of the family’s taking meals together (at least once a day vs. a few times/once/less than once a week), the amount of time the child spent watching television each day (<1, 1–<3, 3–<5, or ≥5 hours), and the...
amount of time the child spent on sports or exercise per week (4–7 days vs. 0–3 days).

Statistical analyses

We first tabulated the distribution of BMI and its categories according to sleeping problems at ages 6 months and 2–4 years (table 1). The differences in BMI distributions were compared by one-way analysis of variance and by F test. Statistical evidence for a difference in effect between males and females was assessed by means of a likelihood ratio test of the interaction with sex. Since we found no statistical evidence that effects differed between the sexes, results are presented for males and females combined.

Because of missing data, increasing the number of confounders or mediators in the multivariable models decreased the number of children retained in the analysis. Therefore, we included only those variables that were significantly (p < 0.05) associated with sleeping problems. From the above-mentioned confounders or mediators, we found child and maternal ages, maternal marital status, maternal mental health, family meals, adolescent television-watching, and maternal BMI to be associated with child sleeping problems.

A series of multiple linear regression models was used to determine the mean difference in BMI according to sleeping problems, taking into account potentially confounding factors and examining possible mediating effects. In separate models, we examined the effect of participant sex and age (model 1), maternal age, maternal BMI, and maternal mental health variables (model 2). We then examined potential mediators (models 3 and 4). The final model combined all variables (model 5). Similarly, a series of multinomial regression models was used to assess the association between sleeping problems and young adult overweight and obesity at age 21 years. Full details on each model are provided in the table footnotes.

In the sensitivity analysis, we excluded participants who were overweight or obese at age 5 years from the analysis (overweight or obesity status was defined using Cole et al.'s (12) standard definition) to see whether any association was influenced by reverse causality (i.e., persons who are overweight or obese by age 5 years having sleeping problems). All analyses were undertaken using Stata, version 9.2 (Stata Corporation, College Station, Texas).

RESULTS

Table 1 shows the numbers and percentages of participants with sleeping problems at ages 6 months and 2–4 years and the distribution of BMIs at age 21 years according to sleeping problems in early life. Mean BMI at age 21 years increased with increasing frequency of experiencing sleeping problems at ages 2–4 years, but there was no association between sleeping problems at age 6 months and BMI at age 21 years. Similarly, the prevalences of overweight and obesity at age 21 years increased with increasing frequency of having experienced sleeping problems at ages 2–4 years.

Since there was no association between sleeping problems at age 6 months and BMI or BMI categories at age 21 years (table 1), we present multivariable associations between sleeping problems at ages 2–4 years and BMI or obesity at age 21.

Table 2 shows the mean difference in BMI at age 21 years, comparing children who experienced sleeping problems at ages 2–4 years with those who did not, with adjustment for potentially confounding and mediating factors in a series of multiple regression models. The results are presented for the 2,001 adolescents with complete data on all
variables included in any of the multivariable models. In the age- and sex-adjusted model, we found that the young adults who often experienced sleeping problems averaged nearly a one-unit increase in BMI (odds ratio = 1.06, 95 percent CI: 0.32, 1.79) compared with those who did not have sleeping problems. These associations were independent of potentially confounding and mediating factors.

Young adults who experienced sleeping problems in early life had increased odds of being obese (odds ratio = 1.90, 95 percent CI: 1.24, 2.93) as likely to be obese by age 21 years as young adults who did not. These associations were independent of adjustment for a variety of potential confounders, including offspring sex, maternal age, maternal mental health, and maternal BMI, and several mediators, including adolescent dietary patterns, family meals, and adolescent television-watching. The findings of this study suggest that having a childhood sleep disorder is an independent predictor of young adult BMI and obesity. These findings also provide some evidence for a long-term impact of childhood sleeping problems on the later development of obesity.

There are various methods for measuring sleep duration. Wrist actigraphy is considered reasonably accurate, although it has been recommended that it be used for a minimum of 7 nights (13) or used only in conjunction with sleep logs (14). For a large epidemiologic study, wrist actigraphy is relatively impractical. Maternal reporting of amount of time spent in bed is also not considered an accurate measure of children’s sleep durations, as children are unlikely to be asleep the entire time they are in bed (15).

We did not have information on sleep duration in our study, and therefore the correct interpretation of our findings is that maternal reports of childhood sleeplessness and

### Table 2

<table>
<thead>
<tr>
<th>Model†</th>
<th>Sometimes (n = 395)</th>
<th>Often (n = 177)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>Mean difference</td>
<td>95% CI†</td>
</tr>
<tr>
<td>Model 1</td>
<td>0.34</td>
<td>−0.19, 0.86</td>
</tr>
<tr>
<td>Model 2</td>
<td>0.13</td>
<td>−0.37, 0.64</td>
</tr>
<tr>
<td>Model 3</td>
<td>0.30</td>
<td>−0.23, 0.83</td>
</tr>
<tr>
<td>Model 4</td>
<td>0.31</td>
<td>−0.21, 0.84</td>
</tr>
<tr>
<td>Model 5</td>
<td>0.10</td>
<td>−0.40, 0.61</td>
</tr>
</tbody>
</table>

* Weight (kg)/height (m)^2.
† Reported by the mothers when the offspring were aged 5 years.
‡ Model 1: results adjusted for child age and sex. Model 2: results adjusted for child age, sex, maternal age at child's birth, mental health, maternal marital status at 5 years, and maternal body mass index. Model 3: results adjusted for child age, sex, and diet at age 14 years. Model 4: results adjusted for child age, sex, and television-watching at age 14 years. Model 5: results adjusted for all of the above factors.
§ Reference category: rarely/never (n = 1,429). The mean difference for the reference category was 0.
¶ CI, confidence interval.

### Table 3

<table>
<thead>
<tr>
<th>Model†</th>
<th>Sometimes (n = 395)</th>
<th>Often (n = 177)</th>
</tr>
</thead>
<tbody>
<tr>
<td></td>
<td>OR§</td>
<td>95% CI§</td>
</tr>
<tr>
<td>Model 1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight#</td>
<td>0.99</td>
<td>0.75, 1.31</td>
</tr>
<tr>
<td>Obese#</td>
<td>1.45</td>
<td>1.04, 2.03</td>
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<tr>
<td>Model 2</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>1.00</td>
<td>0.69, 1.24</td>
</tr>
<tr>
<td>Obese</td>
<td>1.31</td>
<td>0.93, 1.86</td>
</tr>
<tr>
<td>Model 3</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>0.97</td>
<td>0.73, 1.29</td>
</tr>
<tr>
<td>Obese</td>
<td>1.43</td>
<td>1.02, 2.01</td>
</tr>
<tr>
<td>Model 4</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>0.99</td>
<td>0.75, 1.32</td>
</tr>
<tr>
<td>Obese</td>
<td>1.42</td>
<td>1.02, 1.99</td>
</tr>
<tr>
<td>Model 5</td>
<td></td>
<td></td>
</tr>
<tr>
<td>Overweight</td>
<td>0.92</td>
<td>0.69, 1.24</td>
</tr>
<tr>
<td>Obese</td>
<td>1.29</td>
<td>0.91, 1.83</td>
</tr>
</tbody>
</table>

* Reported by the mothers when the offspring were aged 5 years.
† Model 1: results adjusted for child age and sex. Model 2: results adjusted for child age, sex, maternal age at child's birth, mental health, maternal marital status at 5 years, and maternal body mass index (weight (kg)/height (m)^2). Model 3: results adjusted for child age, sex, and diet at age 14 years. Model 4: results adjusted for child age, sex, and television-watching at age 14 years. Model 5: results adjusted for all of the above factors.
‡ Reference category: rarely/never (n = 1,429). The odds ratio for the reference category was 1.
§ OR, odds ratio; CI, confidence interval.
¶ Overweight was defined as body mass index 25.0–29.9.
# Obesity was defined as body mass index ≥30.
irregular sleeping habits are associated with greater BMI many years later, in early adulthood. These associations might reflect an association between shorter sleep duration in childhood and increased risk of greater BMI in later life. This interpretation is supported by recent findings from a study in which the direction and magnitude of the association between sleep duration and glycemic control were similar to those of the association between quality of sleep and glycemic control in adults (16). However, while methods for determining “quality of sleep” and the understanding of what “irregular sleep” and “quality of sleep” mean are likely to vary from one study population to another, “duration of sleep” is less likely to be influenced by such differences in interpretation. If the key exposure of interest is duration of sleep, then future studies should directly assess this factor, and methods should be developed for determining the best methods of accurately determining sleep duration. Finally, it is unclear whether our broad definition of sleep (i.e., any sleep, not restricted to nighttime sleep) had an impact on our results. Studies suggest that parental concern focuses on nighttime waking, and parents may have been more likely to notice and remember these incidents (10). In addition, children in this age group commonly sleep for 1–2 hours during the day, as opposed to the recommended 10–12 hours each night. Nonetheless, it is possible that our nondefined use of the word “sleep” slightly inflated the prevalence of problem sleeping in comparison with other studies.

There are at least three plausible explanations for the observed association between sleep distortion and greater BMI or obesity. First, children who sleep less have more time in which to eat, and this excess consumption results in obesity (3, 5, 17–19). Second, lack of sleep results in tiredness and less physical activity during the day, causing less energy expenditure than energy intake (5, 17, 20). Alternatively, less physical activity may result in reduced sleep quality or duration, and therefore the association with sleep quality or duration might not be causal but might reflect the established association of physical inactivity with obesity. For instance, television-viewing (which uses less energy) has been associated with shorter sleep duration in children (21). We adjusted for television-viewing and sports involvement, but not activity levels. Third, biologic mechanisms such as metabolism disruptions (3, 22–24) or changes in hormone secretion—for example, decreased levels of leptin (an appetite suppressant) (25–27), decreased nocturnal secretion of growth hormone (which maintains lipolysis throughout the night) (28, 29), increased levels of ghrelin (an appetite stimulant) (30), compromised insulin sensitivity (31), and changes in the autonomic nervous system (32)—may play a role.

Our results are in broad agreement with those of most cross-sectional studies. They also affirm the results from previous longitudinal studies. The main strength of this study was its capacity to test the long-term independent prospective association between sleeping problems at 2–4 years of age and young adult overweight and obesity, while controlling for a range of potentially confounding factors and exploring the role of potential mediators. However, our results should be seen in the context of some limitations. The loss to follow-up in our cohort was considerable. Participants who were lost to follow-up (i.e., who did not attend the 14- and 21-year follow-ups but were included in the study at delivery) were more likely to be male and of Asian and Aboriginal/Torres Strait Islander background (all p’s < 0.001). Their mothers were more likely to have been teenagers at their birth, to be less educated, to be single or cohabitating, to have three or more children, to have used tobacco and alcohol during pregnancy, and to have been anxious and depressed at their first antenatal visit (all p’s < 0.001). Our results would be biased if the associations we assessed did not exist in nonparticipants or pointed in the opposite direction in nonparticipants. The broad similarity between our findings and those of two previous prospective studies in children (5, 6) in which loss to follow-up was lower than in our study provides some support that our findings were not biased by loss to follow-up. We have compared our estimates of overweight or obesity at age 21 years with those of the Australian National Nutritional Survey (1995) for a similar age category (ages 20–24 years), and the results are comparable. At age 21 years, the prevalence of overweight in the Mater-University Study of Pregnancy and Its Outcomes was 34 percent. At ages 20–24 years, the prevalence of overweight in the National Nutritional Survey was the same (34 percent) (33). Nevertheless, we cannot be certain that some bias has not occurred. Another limitation includes our reliance on maternal reporting of sleeping problems, which is likely to be affected by a number of factors that could confound the associations we found (including, for example, maternal-offspring relationships). However, maternal reports about offspring are widely used in epidemiologic studies, since direct measurements of sleep duration and quality such as those used in sleep clinic studies are not feasible in large epidemiologic studies. In general, maternal reports are likely to be more reliable than reports obtained from other caregivers.

This study adds some evidence for the suggestion that less sleep during childhood may result in greater obesity in young adulthood. It remains uncertain, however, whether the association in children is with duration of sleep or with sleep disturbance of another type. The current pediatric and adult literature suggests that it is the duration of sleep that is important. If this is confirmed, it may necessitate the development and provision of educational materials for mothers, child-care centers, and other caregivers of young children emphasizing recommended amounts of sleep and how to achieve this, especially in children who are having sleeping problems. Since this is a potential mechanism for disrupting the obesity epidemic, further research is needed to determine whether this relation is really causal and to determine the magnitude of any effect of increasing sleeping duration on population levels of obesity.

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REFERENCES

3. Taheri S. The link between short sleep duration and obesity: we should recommend more sleep to prevent obesity. Arch Dis Child 2006;91:881–4.
17. Astrup A. Have we been barking up the wrong tree: can a good night’s sleep make us slimmer? Int J Obes (Lond) 2006;30:1025–6.