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Sleep problems in adolescence and overweight/obesity in young adults: is there a causal link?

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ABSTRACT

Objective: This study aims to explore if there is a causal association between adolescence sleep problems and overweight/obesity (Ow/Ob) in young adults.
Methods: Youth self-reports were used to explore if adolescence (mean age 13.90 years, SD ±0.32) sleep problems lead to general Ow/Ob, computed from body mass index (n = 1075), or abdominal Ow/Ob, computed from waist circumference and waist to height ratio (n = 1179), in young adults (mean age 20.65 years, SD±0.82). Directed acyclic graphs were used to identify potential confounders, modified Poisson regression with a robust error variance was used to model the associations, and inverse probability weights were used to account for loss to follow-up.
Results: At adolescence, 27.37% of the subjects reported having frequent sleep problems, and about a fifth of these subjects (22.65%) developed general Ow/Ob at young adulthood.
Unadjusted regression analysis indicates a link between adolescent sleep problems and general Ow/Ob in young adults (incidence rate ratio [IRR]: 1.34, 95% confidence interval [CI]: 1.08-2.03), and this link was robust to adjustment for potential confounders (IRR: 1.47, 95% CI: 1.07-2.02). However, no such association was seen for adolescence sleep problems, and abdominal Ow/Ob computed from waist circumference (IRR: 1.30, 95% CI: 0.91-1.87) and waist to height ratio (IRR: 1.27, 95% CI: 0.86-1.88).
Conclusion: Although there is evidence for a causal association between sleep problems and general Ow/Ob, the link between sleep problems and abdominal Ow/Ob needs more research to produce conclusive results. Nonetheless, behavioral interventions encouraging healthy sleep practice in young subjects are likely to influence future Ow/Ob outcome.

Introduction

The transition from adolescence to young adulthood is considered to be one of the fastest weight gain phases of life, as concurrent changes in growth, development, behaviors, and lifestyle often create conditions favorable to weight gain.[1] Weight gained during adolescence also tends to persist later into adulthood, with only a small percentage of obese adolescents growing out of obesity in young adulthood.[2] The high rates of obesity incidence and persistence in young subjects highlight the need for preventive measures for adolescent obesity and thus reducing its persistence into adulthood.

Many studies report that behavioral interventions aiming at traditional modifiable risk factors, for example, physical activity and diet, can help prevent weight gain. Among nontraditional risk factors, inadequate sleep is emerging to be a major risk factor for overweight and obesity (Ow/Ob). Although there is sufficient epidemiological evidence to indicate a role for sleep duration in Ow/Ob,[3] the impact of sleep problems is still relatively unexplored, especially in young subjects.

Considering that some recent studies have provided substantial evidence for growing rates of sleep problems in young subjects,[4] we decided to explore the sleep and obesity association in the context of sleep problems. Although preliminary evidence from some studies indicates a positive association between sleep problems and Ow/Ob,[5,6] there are critical gaps in the existing epidemiologic evidence that limit drawing definitive conclusions. Firstly, due to cross-sectional nature of studies, it is not possible

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to ascertain the directionality of association between sleep problems and Ow/Ob.[7,8] Many studies failed to account for the role of critical confounders and, therefore, there are conflicting results on the role of sleep problems and Ow/Ob outcome.[9–11]

Another important limitation of the existing literature on the sleep-obesity link is preferentially relying on only body mass index (BMI) for obesity assessment. BMI, although simple to calculate and interpret, has limitations in distinguishing between fat and lean mass, and it is perhaps less strongly related to visceral adipose tissue than measures of abdominal obesity.[12] Some studies suggest that BMI may be inadequate and may misclassify adiposity in some individuals.[13] Abdominal obesity, assessed through waist circumference (WC) and waist to height ratio (WHtR), is reported to be a better indicator of Ow/Ob for metabolic syndrome, diabetes, and cardiovascular outcomes than BMI.[14,15] Additionally, the WHtR is found to be a better surrogate than WC and BMI for percent body fat assessment.[16] Therefore, findings based on alternative adiposity measures that reflect the degree of central fat distribution would perhaps be more reliable for exploring the role of sleep problems in cardiovascular outcomes.

Considering the gaps in the literature, we explored evidence from a prospective cohort to see if there is a causal association between adolescent sleep problems and Ow/Ob in young adults independent of other known risk factors. The main aim of this study is to assess the causal link between adolescence sleep problems and general Ow/Ob computed from BMI in young adults. We will also evaluate whether sleep problems affect abdominal obesity computed from WC and WHtR.

Participants and methods

This study is based on the sleep problems data from the Mater-University of Queensland Study of Pregnancy (MUSP) cohort obtained from 14 and 21 years of follow-up. Data were obtained from the 7223 mothers and their offspring who participated between 1981 and 1984.[17] These mothers and their offspring have been followed up prospectively, with assessments done when the offspring were 6 months and 5, 14, and 21 years of age. Written informed consent from the mothers was obtained at all data collection phases and from the young adults at the 21-year follow-up of the study. Ethics committees at the Mater Hospital and the University of Queensland approved each phase of the study. Full details of the study participants and measurements have been previously reported.[17]

Sleep problems assessment

Sleep problems can be defined as problems with sleep initiation, maintenance, and patterns that interfere with the refreshing nature of sleep.[6] At 14 years, the offspring provided information for the prevalence of 5 types of sleep issues in the past 6 months, that is, nightmares (prevalence rate = 27.88%), sleep more than others during the day and/or night (prevalence rate = 37.39%), sleep less than others (prevalence rate = 38.71%), trouble sleeping (prevalence rate = 40.6%), and feeling overtired (prevalence rate = 60.33%). Each question was rated as often (score = 2), sometimes (score = 1), or rarely/never (score = 0). We developed a composite sleep scale using sleep items in the Child Behavior Checklist (CBCL) and Youth Self-Report (YSR). It is reported that using a composite score is reliable when the aim is to examine overall sleep functioning and external correlates of sleep.[18,19]

The criteria to categorize sleep problems from the composite sleep problem score were done using an approach used in a previous study based on the Diagnostic Interview for Children and Adolescents criteria for sleep problem assessment.[20] If the study subject reported "often" on any of the sleep problems items or "sometimes" on all items, it was considered as "often" occurring sleep problem. A report of "sometimes" on 2 to 4 items was considered as "sometimes" occurring sleep problems. In all other cases, sleep problems were considered as "absent."

Assessment of anthropometric indices

Participant's height was measured without shoes using a portable stadiometer to the nearest centimeter, and weight was measured in light clothing with a scale accurate to 0.2 kg. Two measures of weight were taken with a 5-minute interval, and the mean of these 2 measures was used in all analyses. Adolescents' BMI (weight in kilograms divided by the square of height in meters) was calculated based on measured height and weight and was categorized as normal, overweight, or obese according to standard definitions derived from international surveys by Cole et al.[21] For young adults, BMI was categorized into normal (<25 kg/m^2), overweight (25-29 kg/m^2), and obese (≥30 kg/m^2) using the World Health Organization classification of BMI cut-offs.[22]

WC was used as one of the measures to assess assessing abdominal Ow/Ob. WC was measured horizontally using a tape (provided by Sullivans Haberdashery & Craft Wholesalers, Australia) roughly in line with the participant's navel or belly button, directly against the skin without compressing the skin. The average of the 2 measures was taken. WC was categorized as follows: for males: <94 cm as normal, 94-<102 cm as overweight, and ≥102 cm as obese; and for females: <80 cm as normal, 80-<88 cm as overweight, and ≥88 cm as obese.[23] However, the main limitation of WC is the inability to differentiate the cardiovascular risk level of people with different heights.[24] It was also seen that the predictive power of WC for incident hypertension improved when WC was corrected with height or hip circumference.[25]

A more recently proposed measure, WHtR, provides a more accurate reflection of body fat distribution.[26] Some studies indicate WHtR to be superior to BMI, WC, and waist to hip ratio because WHtR will change only when there is a change in the waist because height remains constant in adults.[27] We used WHtR as another measure of abdominal obesity. WHtR was calculated as the ratio of waist circumference to height. WHtR was categorized as follows: for males: <0.52 as normal, 0.53-<0.62 as overweight, and ≥0.63 as obese; and for females: <0.48 as normal, 0.49-<0.57 as overweight, and ≥0.58 as obese.[28]

Confounding factors

Covariates were selected for inclusion in the models using a directed acyclic graph (DAG), which is a method for selecting variables on which it is necessary to condition to control for confounding in the estimation of causal effects.[29] DAG models were created, and the minimum confounder set was selected using the DAGitty platform.[30]

In the creation of the DAG, relationships between each of the variables were assigned based on a priori knowledge considering that all confounding variable must affect the exposure and outcome variables, and should not be on the causal pathway. All potential confounders that have been identified, examined, and discussed in the literature were included in the DAG.[31,32] These variables are detailed in Table S1.

Statistical and epidemiological analysis

Descriptive statistics were used to present young adults' BMI, WC, and WHtR by adolescence sleep problems and lifestyle variables. Categorical variables were examined using the χ^2 test. An inverse probability weighted modified Poisson regression with a robust error variance was fitted to the data[33] using a log-link function and a binary response outcome variable to model the association between adolescence sleep problems and Ow/Ob in young adults. To increase the

statistical precision, we combined overweight and obese (Ow/Ob) categories.

Because we were interested in a causal association, the DAG approach was used to define a minimum set of potential confounders out of those available that would minimize bias in the estimation of the total effect of adolescent sleep problems on Ow/Ob in young adults. Figure 1 illustrates the schematic view of adjustment and output for the "minimally sufficient" confounding variables using a DAG. Based on the latter, 2 variables were dropped (maternal anxiety/depression in pregnancy and parental BMI), whereas adjustments were made for race, birth weight, sex, family income, health, food choices, pubertal stage, TV hours, and exercise within the model.

Exclusion criteria

There were some lifestyle variables, for example, smoking and drinking, that could be both the precursor of sleep problems as well as the outcome of sleep deprivation. Consequently, their inclusion in the DAG could have violated the conditions required for creating the DAG. According to Australian Institute of Health and Welfare, the average age when 14- to 24-year-old Australians first tried alcohol was 15.7 years.[34] Therefore, considering the baseline age of study subjects (13.9 years, SD ±0.34), it is less likely that lifestyle risk factors (eg, drinking, smoking) preceded sleep problems. Hence, exclusion of these variables seems justifiable.

For establishing causality, the essential requirement is that the cause (sleep problems) must precede the effect (Ow/Ob). Therefore, we considered only new cases of Ow/Ob and had to exclude the preexisting cases, that is, subjects who were overweight or obese during childhood or adolescence, from the analysis.

Missing data

As observed in other cohort studies, MUSP cohort also witnessed some loss to follow-up. Therefore, standard analytic methods were applied to correct for potential selection bias induced by attrition. The main study had revealed that participants who were lost to follow-up in MUSP study were more likely to have been teenagers when they delivered, of lower educational status, single or cohabitating, had 3 or more children, used tobacco and alcohol during their pregnancy, and had been anxious and depressed at their first prenatal visit.[17] For this study, the losses to follow-up at 21 years were accounted for by running an inverse probability weighted regression.[35] Inverse probability weighting was based on allocating a weight to each selected subject so that the subject accounts in the analysis not only for self but also for those with similar characteristics who were not selected. The weight is the inverse of the probability of selection. We did this by first computing the propensity (probability) to remain in the study by regressing (using logistic regression) an indicator variable for nonmissing on the baseline explanatory variables. The fitted model gave a predicted probability of being present on follow-up based on participant characteristics. The final modified Poisson regression analysis was then weighted by $1/P$, where P was each individual's fitted probability of participation from the logistic regression. Measures that predicted presence at the 21-year follow-up included race, sex, maternal age at childbirth, maternal education, maternal anxiety during last trimester of pregnancy, maternal smoking and drinking habits, adolescent depression/anxiety, and adolescent smoking and drinking. Significance levels were set at <.05 unless otherwise stated. All statistical analyses were done using Stata version 13, College Station, TX. The DAG analysis was created and evaluated using the DAGitty v2.3 platform (www.dagitty.net).

Results

In this study, a considerable percentage of children (16.52%) and adolescents (22.62%) were Ow/Ob at their respective follow-ups. After excluding these subjects, sleep problems information was available for a total of 1966 non-obese and non-overweight adolescents, with 27.37% reporting frequent sleep problems, (mean age 13.90 years, SD ±0.32) (53.60% male), mostly belonging to the white race (91.78%).

Sleep and Ow/Ob link was explored using adolescent sleep problems and general Ow/Ob, computed from BMI (n = 1075), as well as abdominal Ow/Ob, computed from WC and WHtR (n = 1179), in young adults (mean age 20.65 years, SD±0.82), with mean BMI,

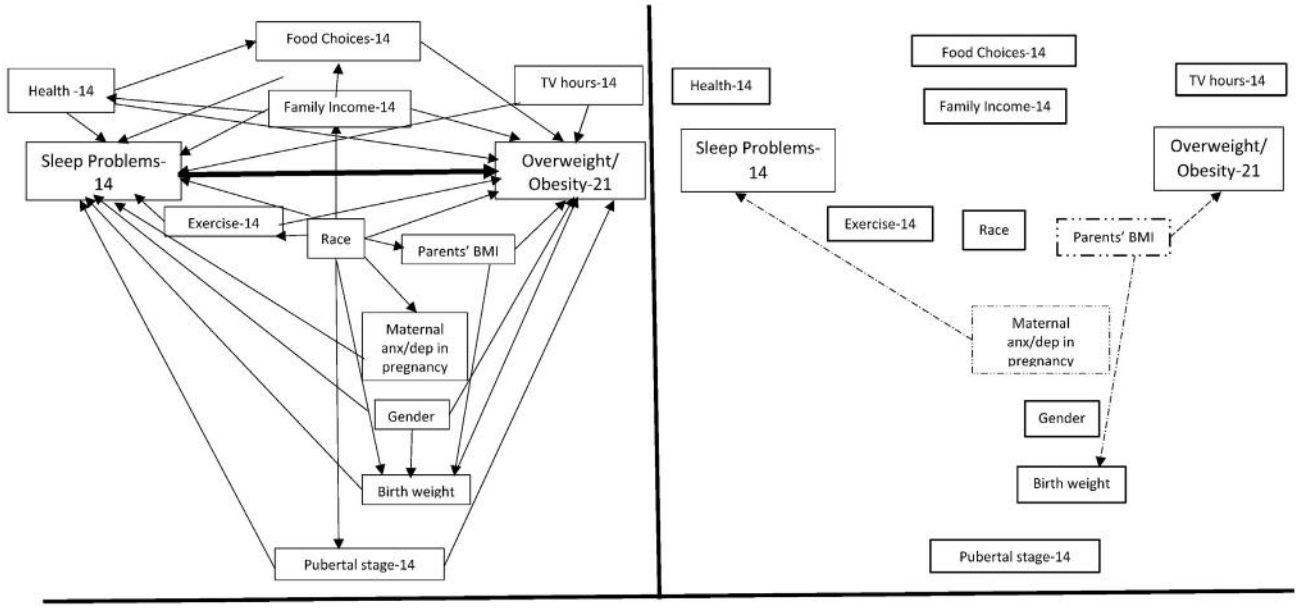

Fig. 1. Application of a DAG to explore the causal impact of adolescent sleep problems on Ow/Ob in young adults. Figure on the left-hand side indicates all variables considered for drawing DAG; figure on the right-hand side indicates minimally sufficient variables (variables inside the boxes with solid lines), identified by DAG, to control for confounding in the association between sleep problems and Ow/Ob.

Table 1
Distribution of sleep problems, lifestyle variables at adolescence, and anthropometric categories at young adulthood (after excluding Ow/Ob subjects at childhood and adolescence)

Variable	n (%)
Age (mean ± SD)	
Age at adolescence	13.90 (0.32)
Age at young adulthood	20.65 (0.82)
BMI at 21 y (kg/m^2) (mean ± SD)	22.59 (3.37)

	Ow/Ob at age 21 y								
	BMI n (%)			WC n (%)			WHtR n (%)		
	Normal	Ow/Ob	*P* value	Normal	Ow/Ob	*P* value	Normal	Ow/Ob	*P* value*
Sleep problems (14 y)									
Absent	284 (83.0)	58 (17.0)	.07	321 (86.52)	50 (13.48)	.207	329 (88.68)	42 (11.32)	.08
Sometimes	329 (76.3)	102 (28.7)		403 (83.61)	79 (16.39)		414 (86.07)	67 (13.93)	
Often	238 (78.8)	64 (21.2)		267 (81.65)	60 (18.35)		271 (82.87)	56 (17.13)	
Race									
White	802 (78.47)	220 (21.53)	.85	941 (83.87)	181 (16.13)	.99	965 (86.08)	156 (13.92)	.76
Asian	39 (79.59)	10 (20.41)		45 (83.33)	9 (16.67)		45 (83.33)	9 (16.67)	
Aboriginal-Islander	30 (75)	10 (25)		35 (83.33)	7 (16.67)		35 (83.33)	7 (16.67)	
Exercise (14 y)									
No-<1 d/wk	129 (81.13)	30 (18.87)	.66	143 (80.34)	35 (19.66)	.33	150 (84.27)	28 (15.73)	.152
2-3 d/wk	287 (78.85)	77 (21.15)		336 (81.36)	77 (18.64)		345 (83.74)	67 (16.26)	
>3 d/wk	470 (77.81)	134 (22.19)		557 (86.49)	87 (13.51)		565 (87.73)	79 (12.27)	
Weekday TV h (14 y)									
<1-3 h/d	572 (78.90)	153 (21.10)	.68	668 (84.03)	127 (15.97)	.87	684 (86.04)	111 (13.96)	.88
>3 h/d	309 (77.83)	88 (22.17)		364 (83.68)	71 (16.32)		372 (85.71)	62 (14.29)	
Soft drinks (14 y)									
Rarely/never	316 (80.82)	75 (19.18)	.24	362 (84.19)	68 (15.81)	.54	372 (86.71)	57 (13.29)	.78
1-3 times/wk	371 (76.18)	116 (23.82)		446 (82.59)	94 (17.41)		460 (85.19)	80 (14.81)	
Most days	192 (79.34)	50 (22.66)		220 (85.60)	37 (14.40)		220 (85.60)	37 (14.40)	
Sweets (14 y)									
Rarely/never	385 (80.38)	94 (19.62)	.09	458 (85.77)	76 (14.23)	.23	471 (88.37)	62 (11.63)	.08
1-3 times/wk	415 (76.01)	131 (23.99)		487 (81.99)	107 (18.01)		497 (83.67)	97 (16.33)	
Most days	75 (84.27)	14 (15.73)		77 (83.70)	15 (16.30)		79 (85.87)	13 (14.13)	

* *P* values are based on χ^2 association.

WC, and WHtR being 22.53 (SD 3.37) kg/m^2, 78.34 (SD 9.49) cm, and 0.82 (SD 0.08), respectively (Table 1).

Results from descriptive analysis indicate that about a fifth of the subjects (22.65%) with adolescence sleep problems were found to develop general Ow/Ob at young adulthood. While the incidence rate of abdominal Ow/Ob was slightly lower with only 17.18% and 15.22% of young adults found to develop Ow/Ob, computed from WC and WHtR, respectively. The sex difference was seen for WC and WHtR but not for BMI.

For Ow/Ob categories computed using BMI, unadjusted regression analysis indicates higher rates of Ow/Ob in subjects with sleep problems (incidence rate ratio [IRR]: 1.34, 95% confidence interval [CI]: 1.02-1.75). The causal link between sleep problems and general Ow/Ob was still evident even when adjusted for important sociodemographic and lifestyle variables (IRR: 1.47, 95% CI: 1.07-2.02) (Table 2).

For abdominal Ow/Ob categories computed from WC, both unadjusted (IRR: 1.27, 95% CI: 0.95-1.72) and adjusted regression analyses (IRR: 1.30, 95% CI: 0.91-1.87) indicate uncertainty around the role of adolescence sleep problems in abdominal Ow/Ob in young adults. Similarly, for Ow/Ob computed from WHtR, both unadjusted regression analysis (IRR: 1.34, 95% CI: 0.97-1.27) as well as adjusted regression analysis (IRR: 1.27, 95% CI: 0.86-1.88) indicated even more uncertainty. However, the potential role of sleep problems on abdominal Ow/Ob cannot be ruled out.

Discussion

In this investigation of the impact of sleep problems on future Ow/Ob, we found evidence suggesting a causal association involving adolescence sleep problems and general Ow/Ob in young adults. Although the relationship with abdominal obesity was statistically nonsignificant, nevertheless, the effect size warrants further exploration of the role of sleep problems in abdominal obesity. Our results suggest that sleep problems play a more prominent role in general obesity when compared with abdominal obesity, and the discrepancy may be attributed to the different pathogenic mechanisms for abdominal vs general obesity.[36]

The findings of this study demonstrate a significant role of sleep problems in future Ow/Ob. Our results are in line with a recent case-control study that used objective methodology (polysomnography)

Table 2
Regression analysis results exploring causal link between adolescence sleep problems and Ow/Ob in young adults

Anthropometric index	Unadjusted rate ratio		Adjusted rate ratio[a]	
	IRR	95% CI	IRR	95% CI
Ow/Ob (BMI)	1.34	1.02-1.75	1.47	1.07-2.02
Ow/Ob (WC)	1.27	0.95-1.72	1.30	0.90-1.87
Ow/Ob (WHtR)	1.34	0.97-1.27	1.27	0.86-1.88

Sleep problem categories: often/sometimes, absent.
BMI categories: normal (<25 kg/m^2), overweight (25-29 kg/m^2), and obese (≥30 kg/m^2).
WC categories (for males): normal <94 cm, overweight 94-<102 cm, and obese ≥102 cm.
WC categories (for females): normal <80 cm, overweight 80-<88 cm, and obese ≥88 cm.
WHtR categories (for males): normal <0.52, overweight 0.53-<0.62, and obese ≥0.63.
WHtR categories (for females): normal: <0.48, overweight 0.49-<0.57, and obese ≥0.58.
[a] Adjusted for race, birth weight, sex, family income, health, food choices, pubertal stage, TV hours, and exercise.

to assess inadequate sleep and established a link between sleep problems and obesity.[11] Although the overall evidence for the role of sleep problems in Ow/Ob is weaker when compared to the evidence for the role of short sleep duration and Ow/Ob, from a public health perspective, interventions encouraging healthy sleep practice in young subjects are likely to influence future Ow/Ob prevalence.

There is plenty of evidence on puberty-associated changes in circadian rhythms, that is, "sleep phase delay" that is linked with a tendency for later bedtimes and rise times, irregular sleep patterns and daytime drowsiness.[37,38] Studies that have explored sleep-obesity association in adolescence without adjusting for the role of pubertal stages might have missed the contribution of an important confounder in the sleep-obesity link. The findings of this study suggest that the link between sleep problems and Ow/Ob is robust to adjustment for Tanner stages. It is also important to consider that the subjects who have short sleep duration are also seen to complain about sleep problems and vice versa,[39] We suggest that future epidemiological studies should explore a more appropriate indicator of inadequate sleep, that is, poor sleep quality that comprises both sleep duration and sleep problems, to conclusively establish a link, if any, between inadequate sleep and central obesity.

Several studies have attempted to assess possible mechanisms that link sleep problems and obesity. It is seen that during the deeper stage of slow wave sleep (SWS) which is associated with robust elevations of growth hormone levels, brain glucose utilization and sympathetic nervous activity decrease, and parasympathetic nervous activity increases.[40] Hence, SWS is likely to play a significant role in total body glucose regulation. Subjects with sleep issues or poor quality sleep are seen to spend less time in SWS and have alterations in REM sleep, inducing hormonal changes.[41] Inadequate sleep is also linked to alterations in the regulation of thyroid-stimulating hormones which, in turn, may affect the thyroid hormones—an important regulator of carbohydrate metabolism—resulting in the development of glucose dysregulation and insulin resistance and predisposing to diabetes in the longer term.[42] The evidence from experimental studies suggests that sleep loss appears to influence hormones associated with appetite and satiety (ie, leptin and ghrelin) that could result in higher energy intake and lower energy expenditure.[43] Thus, hormonal changes associated with sleep loss are likely to be crucial to metabolic outcomes which lead to Ow/Ob. More experimental studies are therefore needed for better understanding of this relationship.

Although the longitudinal nature of our study helped in assessing the temporality of sleep problems and Ow/Ob association, overcoming the drawbacks of many cross-sectional studies, there are some limitations as well. First, we relied on sleep items in CBCL/YSR for sleep problems assessment which is not the best choice for assessing sleep issues and disorders. The prevalence rates for sleep problems found in this study need to be replicated with a stronger measurement approach based on a multimethod assessment and objective measures of sleep problems. Nonetheless, many of CBLC/YSR sleep items have good agreement with the objective evaluation of sleep problems,[44] and the composite score from CBCL sleep items is seen to be more strongly correlated with validated measure for sleep assessment, for example, the Children's Sleep Habits Questionnaire total score and Sleep Disorders Inventory for Students sleep disturbance indexes, than any of the individual CBCL sleep items.[19] We believe that a total score will help in tapping multiple and at times unrelated domains of sleep functioning. Therefore, our composite scale was an appropriate measure to assess sleep problems.

Another limitation of our study is using parent or self-reports to assess physical activity and diet, which often introduce recall and social desirability bias and are not as reliable as objective measures of physical activity (eg, accelerometer). Lastly, we did not have any information on the role of possible gene-level influence on the causal association between sleep and obesity. Our study has some noteworthy strengths as well, which include a large sample size, a long follow-up period, using a DAG to more accurately adjust for confounding, and relying on multiple anthropometric measures to assess the role of sleep problems in general as well as abdominal Ow/Ob.

Conclusion

In summary, although our results provide evidence for a causal association between sleep problems and general Ow/Ob, there was uncertainty associated with the evidence for abdominal Ow/Ob. Considering that abdominal obesity is a stronger risk factor than BMI for cardiovascular diseases, it is important to highlight the uncertainty in the association between sleep problems and abdominal Ow/Ob. Therefore, further evidence from longitudinal studies is needed to explore the role of inadequate sleep in abdominal Ow/Ob. Also, there is a need for evidence from twin-pairs studies for unbiased evaluation of the roles of genetical and environmental factors in shaping sleep-obesity link. Finally, we recommend that lifestyle interventions targeting young subjects should include a component emphasizing the benefits of sleep health and the impact of disturbed sleep on future weight gain.

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Conflict of interest statement

The authors declare no conflict of interest.

Authors' contributions

YF was responsible for literature review, data analysis, and writing the manuscript. AAM and SD helped in data analysis and critically reviewed the draft of the manuscript.

Disclosure

The authors declare no conflict of interest.

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