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Childhood obesity patterns and relation to middle-age sleep apnoea risk: the Bogalusa Heart Study

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Summary

Background—Obese adults have a higher risk of obstructive sleep apnoea (OSA); however, the relationship between childhood obesity and adult OSA risk is unclear.

Objectives—This study aimed to examine overweight/obesity (OW) in childhood and risk of OSA in middle age.

Methods—Childhood OW status was classified as never OW, weight cycling, persistent OW and incident OW. After 35 years of follow-up, high risk for OSA was determined by a positive score in 2 domains on the Berlin Questionnaire with obesity removed from scoring.

Results—At initial assessment, mean (SD) age was 9.9 (2.9) years, and 23.9% were OW. Overall, 25.7% had scores indicating a high risk for OSA. Compared with participants who were never OW, those with persistent OW and incident OW were 1.36 (95% CI: 1.04–1.77) and 1.47 (1.11–1.96) times more likely to be high risk for OSA, after adjustment for multiple risk factors

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Conflicts of Interest Statement

The authors have no conflicts of interest relevant to this article to disclose. The authors have no financial relationships relevant to this article to disclose.

Author contributions

L. A. B. conceptualized and designed the study, drafted the initial manuscript and approved the final manuscript as submitted. T. H. designed the study, carried out data analyses, drafted the initial manuscript and approved the final manuscript as submitted. S. M. B., E. W. H., W. C., J. G., L. Y., L. S. W. and S. R. reviewed and revised the manuscript, provided critical insights into the analysis and interpretation of data and approved the final manuscript as submitted. T. S. carried out the initial analyses, reviewed and approved the final manuscript as submitted. All authors were involved in writing the paper and had final approval of the submitted and published versions.

and adult OW status. Participants with an OW duration of 1–4 years, 5–8 years, and 8+ years were 0.96 (0.44–2.09), 1.20 (0.70–2.04) and 1.52 (1.22–1.90) times more likely to be high risk for OSA compared with those who were never OW (*P* for trend: 0.0002).

Conclusions—These results suggest that childhood OW is associated with a high risk of OSA in middle age.

Keywords

childhood obesity; cohort study; obesity duration; obstructive sleep apnoea

Introduction

Obstructive sleep apnoea (OSA) affects approximately 5–15% of middle-aged men and women, and its prevalence has increased substantially over the past several decades in tandem with increases in obesity (1). Intermittent hypoxaemia and sleep disruption are the hallmarks of OSA, which in turn are associated with a higher risk for cardiovascular and neurocognitive morbidity and mortality (2). Untreated OSA is also a significant cause of injuries, such as motor vehicle crashes (3).

Compelling evidence has suggested that obesity in adulthood is an important risk factor for OSA (4). Obesity is also a risk factor for OSA in adolescence and is increasingly recognized as a risk factor for OSA in childhood (5). Currently, approximately 17% of children aged 2 to 19 years are obese in the USA (6), more than double the prevalence in the 1980s (7). The increased prevalence of childhood obesity indicates a greater cumulative exposure to excess adiposity over the life course, which may have profound implications for future risk of obesity-related chronic diseases, such as OSA (8). In addition, this increase in childhood obesity has been associated with the earlier development of insulin resistance, diabetes, hypertension and dyslipidaemia, which are established risk factors for OSA (8,9). Childhood obesity is associated with a higher prevalence of cardiovascular disease, asthma and premature mortality in adulthood (10). Although childhood may be a particularly important period when the effects of changes in weight and metabolic factors may have long-term health consequences, little is known regarding the relationship between childhood obesity and the development of OSA in mid-life or later.

Most previous studies examining the relationship between childhood obesity and OSA risk were based on cross-sectional comparisons in paediatric populations (11,12), or used only a single measure of adiposity, and thus may have missed important associations related to the pattern of obesity in childhood (13). Given the high prevalence of childhood obesity, understanding the relationship between obesity in childhood and OSA in adulthood may provide critical insights for intervention. Therefore, we prospectively examined the association of overweight and obesity (OW) patterns, including OW status and duration in childhood, with subsequent risk of OSA in middle age using data from the Bogalusa Heart Study.

Methods

Study population

The Bogalusa Heart Study is a series of long-term prospective studies of a semi-rural biracial (65% white, 35% black) community in Bogalusa, Louisiana begun in 1973 by Dr Gerald Berenson. The population and study design of the Bogalusa Heart Study have been previously described (14). The present study initially included 914 participants who were followed from childhood through middle age. All participants responded to the Berlin Sleep Questionnaire (15). We excluded 66 participants who had <2 measures of adiposity in childhood (4–18 years) and four participants who had cardiovascular disease. The final sample for analysis included 844 individuals. All protocols were approved by the Institutional Review Board of Tulane University, and written informed consent was obtained from participants or from a parent or guardian if <18 years of age.

Data collection

Exposure measures—Trained examiners collected all data using standardized protocols, which have been described previously (14). At each examination, anthropometric indices were measured in duplicate or triplicate and then averaged. Height and weight were measured twice to the nearest 0.1 cm and nearest 0.1 kg, respectively. Averaged values were used to calculate body mass index (BMI) as weight in kilograms divided by height in meters squared. Childhood OW was defined as age-specific and sex-specific BMI ≥ 2 standard deviations from the population mean based on the World Health Organization recommendation (16,17).

The assessment of OW patterns in childhood was based on OW status over time and OW duration, from multiple measures of BMI in childhood. A total of ten childhood measurements were possible between 1973 and 1992. The OW status was categorized into four groups: never OW, weight cycling (OW then ever became normal weight), persistent OW (OW throughout) and incident OW (normal weight at first assessment then became and remained OW through last examination). Duration of OW was calculated using the presence or absence of OW at each follow-up examination. For participants who were normal weight at baseline then became and remained OW through the last examination (incident OW), or participants who were OW throughout (persistent OW), duration was calculated as the cumulative number of consecutive OW years. If participants were OW then ever became normal weight during childhood (weight cycling), duration was not calculated.

Outcome measures—Sleep-related outcomes in adulthood were collected in 2010 using the Berlin Questionnaire (15). High risk for OSA, the primary outcome, was determined using a modified Berlin Questionnaire score, which excluded obesity from risk assessment to minimize bias in the association between OW and OSA (18). A participant was categorized as high risk for OSA if he/she scored positive for at least two of the three domains: loud or frequent snoring or frequent breathing pauses, frequently tired after sleeping or during the daytime or having fallen asleep while driving and self-reported hypertension. Secondary outcomes included habitual snoring and excessive sleepiness. Habitual snoring was defined as two of the following: snoring ≥ 3 nights/week, snoring

louder than talking or very loud, or breathing pauses ≥ 3 nights/week. Excessive sleepiness was defined as two of the following: feeling tired ≥ 3 days/week after sleeping, feeling tired during time awake for ≥ 3 days/week or having fallen asleep while driving. The Berlin Questionnaire has been shown to have a high sensitivity (70–87%) but lower specificity (22.2%) for identifying an apnoea–hypopnoea index of >5 (15).

Covariates—Demographic characteristics and lifestyle risk factors were collected in surveys from childhood to adulthood. Questions on tobacco use were asked for 3rd graders and older from 1974 onward. Self-reported current smoking status (yes/no) was defined as the use of any cigarettes within the past year. Questions on alcohol consumption were asked for 7th graders or older from 1984 onward. Self-reported regular alcohol drinking (yes/no) was defined as drinking alcohol beverages >2 times a week. Self-reported leisure-time physical activity was assessed on a scale of 1 (very inactive) to 5 (very active) based on a validated questionnaire (19). Educational levels were categorized into $<$ high school and high school. Both leisure-time physical activity and educational levels were measured in adulthood. C-reactive protein, an inflammatory biomarker, was measured by latex particle-enhanced immunoturbidimetric assay on Hitachi 902 Automatic Analyzer in 2010. Dietary data were measured in adulthood using a validated 152-item food frequency questionnaire (20,21).

Statistical analysis—Descriptive statistics were employed to define population characteristics in childhood (baseline) and adulthood and adult sleep-related outcomes by the four childhood OW patterns. Given that the length of follow-up years varied little across study participants [Median (interquartile range, IQR): 35 (1) years], time effects were minimal. Therefore, the associations of OW patterns and duration in childhood with study outcomes were examined using polytomous logistic regression techniques. Models were adjusted for baseline age, race, sex, educational levels, current adult OW status, leisure-time physical activity, follow-up time and time-dependent covariates including regular alcohol drinking and current smoking status using a propensity score method with inverse probability weighting (IPW) (22). In this technique, a propensity score, which reflects participants' likelihood of being located in levels of independent variables, was computed first using a logistic regression model based on the values of covariates for each participant. Data were then weighted by the inverse probabilities of being in the observed group to account for covariates. Compared with standard statistical methods adjusting for individual covariates, IPW increases statistical efficiency of adjusting for confounding due to measured time-varying covariates affected by prior exposure (22). To assess the effectiveness of the IPW in reducing confounding effect by covariates, we compared the characteristics of the groups before and after applying the combined weights. In addition, age, sex and race differences were tested for interaction. All P values were two-sided, and statistical significance was defined as $P < 0.05$. Analyses were performed using SAS 9.3 (SAS Institute, Cary, NC, USA) and STATA 15.0 (StataCorp LP, College Station, TX, USA) for Windows.

Results

Childhood characteristics

Of the 844 participants included in the analysis, 42.3% were male and 33.6% were Black. At baseline, mean (SD) age was 9.9 (2.9) years, and the proportion of OW individuals was 23.9%. Of a possible 10 measurements, BMI was measured between two and six times in the analytical sample (median: three measures between the ages of 4 and 18 years). Numbers of exams did not differ significantly across categories of OW pattern.

There were 536, 104, 140 and 64 participants in the never OW, weight cycling, persistent OW and incident OW groups, respectively. Baseline characteristics are presented by patterns of childhood OW status (Table 1). The mean levels of BMI were greater for participants in the persistent OW group than those in the never OW group. Participants in the incident OW group were more likely to drink alcohol regularly as adults than those in the never OW group. Median time between the first and last measures of BMI in childhood was 5 years (IQR: 5 years) across the four groups. The means (SDs) of OW duration were 5 (2.5) years and 5 (1.8) years for the persistent OW and incident OW groups, respectively.

Follow-up

Participants were followed for an average of 35 years (IQR: 1 year). In 2010, when risk for OSA was measured, the mean age was 42.8 (4.5) years, 78.2% were OW, and the mean BMI was 31 (7.8) kg/m². Overall, 25.7% participants had elevated Berlin scores indicating high risk for OSA. Participants in the persistent OW group had significantly higher risk of OSA than those in the never OW group. The crude risk of habitual snoring in adulthood was higher in the persistent OW and the incident OW group than the never OW group (Table 1). Participants in the incident OW and persistent OW groups tended to be slightly younger and more often obese in adulthood than those in the never OW group (Table 1).

Association with sleep-related outcomes

In multivariate analyses, participants in the persistent OW and incident OW groups were 1.36 (95% CI: 1.04–1.77) times and 1.47 (1.11–1.96) times more likely to be high risk for OSA than those in the never OW group, after adjustment for baseline age, sex, race, educational levels, current adult OW status and leisure-time physical activity, follow-up time and time-dependent covariates including regular alcohol drinking and current smoking status (Table 2). In contrast, participants in the weight cycling group were not more likely to be at high risk of OSA or habitual snoring as compared with those in the never OW group.

Participants with an OW duration of 1–4 years, 5–8 years and 8+ years were 0.96 (95% CI: 0.44–2.09), 1.20 (0.70–2.04) and 1.52 (1.22–1.90) times more likely to be high risk for OSA as compared with those who were never OW. Significant linear trends were present across categories of OW duration (*P* for trend: 0.0002) (Fig. 1). A similar trend was observed for habitual snoring but not for excessive sleepiness (Table 3).

We performed a series of sensitivity analyses to test the robustness of study results. After replacing current adult OW status with adult BMI in the models, we found similar

significant results for OW duration and risk of sleep apnoea, but the association between persistent OW and risk of sleep apnoea was attenuated slightly. Results remained consistent when the patterns of childhood OW were defined based on the two most disparate exams in childhood. Among those with 3 visits, somewhat stronger associations were seen as compared with primary analyses, for instance, participants with an OW duration of 1–4 years, 5–8 years and 8+ years were 1.18 (0.47–2.92), 1.21 (0.54–2.71) and 1.68 (1.29–2.18) times more likely to be high risk for OSA as compared with those who were never OW (P for trend: 0.0001). Participants in the persistent OW group and the incident OW group were 1.69 (95% CI: 1.20–2.40) and 1.68 (1.18–2.39) times more likely to be high risk for OSA than those in the never OW group, after adjustment for the aforementioned covariates including current adult OW status. Further adjusting for C-reactive protein or consumption of sugar and caffeine measured in adulthood did not change the results. There was no significant effect modification by age, sex, or race in the association between OW pattern or duration and high risk for OSA.

Discussion

In this community-based prospective cohort study with approximately 35 years of follow-up, individuals who were persistently OW or who were normal weight and then became OW during childhood, were more likely to be at high risk for OSA and habitual snoring in middle age, compared with those who were never OW in childhood, independent of adult obesity. Dose–response analysis shows that the length or duration of OW in childhood was associated with progressively higher risk of sleep apnoea and habitual snoring in middle age. To our knowledge, this is the first study to examine the impact of childhood obesity pattern and duration on adult OSA risk, independent of the current adult BMI. Notably, the association between OW duration during childhood and sleep apnoea in adulthood was independent of obesity in adulthood. These novel findings highlight the importance of childhood OW as an independent predictor of sleep apnoea in middle age.

Few previous studies have examined the impact of childhood OW patterns on risk of OSA, and those that have are primarily cross-sectional or short-term follow-up studies that report conflicting results. Many of those studies were based on a single measure of BMI, and other than the present study, little or no information exists assessing the impact of childhood OW patterns over time (11–13). The relationship between OW and OSA varies with age across childhood, underscoring the importance in investigating the pattern of childhood OW over time in relation to OSA (11). Our study is unique in assessing two different aspects of the long-term patterns of weight changes in childhood, OW status and duration of OW, across an average of 35 years of follow-up.

Several factors may contribute to the association between prolonged OW and OSA. Prolonged OW may result in additional metabolic changes, which lead to increased risk of OSA and habitual snoring in adulthood. For example, in a nationally representative sample of US adults, persistent obesity from 25 years old to at least 35 years old was associated with higher odds of elevated blood pressure and C-reactive protein, compared with the current obesity, which reflected a shorter duration in men (23). Obese children are more likely to stay obese into adulthood and to develop associated chronic diseases (24).

Moreover, it is possible that persistent or longer duration of obesity in childhood results in systemic inflammation which has been linked to the pathogenesis of OSA (25). Longer exposure to obesity and exposure during critical periods of development may therefore contribute to the development of obesity-related comorbidities earlier and more frequently (24). Reciprocal relationships may also contribute in that sleep disturbance may promote obesity in children (26–28).

This study has important strengths. First, standardized protocols and quality control procedures for data collection were used in each examination. Second, our study includes a number of repeated examinations for each participant, which enables us to longitudinally assess the obesity patterns in childhood. Childhood may be a particularly sensitive period where changes in weight or metabolic parameters could have significance for long-term health outcomes, and thus, it may be important to capture OW pattern in childhood and describe it comprehensively. Finally, this study was conducted in a biracial community-dwelling population, enhancing the generalizability of the results.

Our results are subject to some limitations. Our estimation of the pattern and duration of childhood OW during follow-up was based on measurement of BMI at prospective cohort examinations. A more frequent number of BMI assessments would have resulted in a more accurate estimation of OW pattern and duration during childhood. Misclassification due to assessment schedule should be random and so may have resulted in an underestimation of the true association. In our sensitivity analysis among those with 3 BMI measures, we found consistent results. As with other observational studies, residual confounding due to unknown and unmeasured factors cannot be eliminated. Data on sleep fragmentation or physiological/psychological markers for stressors were not available. Our sensitivity analyses showed that the results did not change after adjustment for stimuli such as dietary caffeine and sugar and inflammatory biomarkers such as C-reactive protein. Questionnaires rather than diagnostic measurements were used to identify likelihood of OSA. The Berlin Questionnaire used in this study has been widely applied in population settings to indicate the risk of OSA with reasonable validity and reliability (18). There was no measurement of OSA in childhood. However, reverse causality is unlikely to be responsible for the results we observed, because the prevalence of OSA is relatively low (0.8% to 3%) in children and teenagers (5,29), as compared with middle-aged adults, and a recent study demonstrated that OSA in childhood usually remits in adolescence (30). Finally, C-reactive protein was not measured at baseline but only in 2010.

This study suggests that persons who became overweight in childhood or experienced prolonged periods of OW in childhood were more likely to be at high risk of OSA and habitual snoring in middle age, independent of adult OW. These findings underscore the importance of primordial prevention of obesity and the need for interventions targeted to childhood. In the context of the current epidemic of childhood obesity, the clinical implications of our findings include the need for increased awareness and testing of individuals at risk for OSA.

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References

1. Peppard PE, Young T, Barnet JH, Palta M, Hagen EW, Hla KM. Increased prevalence of sleep-disordered breathing in adults. *Am J Epidemiol*. 2013; 177:1006–1014. [PubMed: 23589584]
2. Bradley TD, Floras JS. Obstructive sleep apnoea and its cardiovascular consequences. *Lancet*. 2009; 373:82–93. [PubMed: 19101028]
3. Tregear S, Reston J, Schoelles K, Phillips B. Obstructive sleep apnea and risk of motor vehicle crash: systematic review and meta-analysis. *J Clin Sleep Med*. 2009; 5:573–581. [PubMed: 20465027]
4. Somers VK, White DP, Amin R, et al. Sleep apnea and cardiovascular disease: an American Heart Association/American College Of Cardiology Foundation Scientific Statement from the American Heart Association Council for High Blood Pressure Research Professional Education Committee, Council on Clinical Cardiology, Stroke Council, and Council On Cardiovascular Nursing. In collaboration with the National Heart, Lung, and Blood Institute National Center on Sleep Disorders Research (National Institutes of Health). *Circulation*. 2008; 118:1080–1111. [PubMed: 18725495]
5. Redline S, Tishler PV, Schluchter M, Aylor J, Clark K, Graham G. Risk factors for sleep-disordered breathing in children. Associations with obesity, race, and respiratory problems. *Am J Respir Crit Care Med*. 1999; 159:1527–1532. [PubMed: 10228121]
6. Ogden CL, Carroll MD, Kit BK, Flegal KM. Prevalence of childhood and adult obesity in the United States, 2011–2012. *JAMA*. 2014; 311:806–814. [PubMed: 24570244]
7. National Center for Health Statistics (US). Health, United States, 2011: With Special Features on Socioeconomic Status and Health. Department of Health and Human Services; Hyattsville, MD; U.S.A: 2012.
8. Dietz WH. Health consequences of obesity in youth: childhood predictors of adult disease. *Pediatrics*. 1998; 101:518–525. [PubMed: 12224658]
9. Alberga AS, Sigal RJ, Goldfield G, Prud'homme D, Kenny GP. Overweight and obese teenagers: why is adolescence a critical period? *Pediatrics Obesity*. 2012; 7:261–273.
10. Reilly JJ, Kelly J. Long-term impact of overweight and obesity in childhood and adolescence on morbidity and premature mortality in adulthood: systematic review. *Int J Obes (Lond)*. 2011; 35:891–898. [PubMed: 20975725]
11. Kohler MJ, Thormaehlen S, Kennedy JD, et al. Differences in the association between obesity and obstructive sleep apnea among children and adolescents. *J Clin Sleep Med*. 2009; 5:506–511. [PubMed: 20465015]
12. Kadiotis AG, Alexopoulos EI, Hatzi F, et al. Adiposity in relation to age as predictor of severity of sleep apnea in children with snoring. *Sleep Breath*. 2008; 12:25–31. [PubMed: 17684780]
13. Inge TH, King WC, Jenkins TM, et al. The effect of obesity in adolescence on adult health status. *Pediatrics*. 2013; 132:1098–1104. [PubMed: 24249816]
14. Berenson, GS.; McMahan, CA.; Voors, AW., et al. Cardiovascular Risk Factors in Children – The Early Natural History of Atherosclerosis and Essential Hypertension. Oxford University Press; New York: 1980.
15. Netzer NC, Stoohs RA, Netzer CM, Clark K, Strohl KP. Using the Berlin Questionnaire to identify patients at risk for the sleep apnea syndrome. *Ann Intern Med*. 1999; 131:485–491. [PubMed: 10507956]

16. de Onis M, Onyango AW, Borghi E, Siyam A, Nishida C, Siekmann J. Development of a WHO growth reference for school-aged children and adolescents. *Bull World Health Organ.* 2007; 85:660–667. [PubMed: 18026621]
17. World Health Organization (WHO). Multicentre Growth Reference Study Group. WHO child growth standards: length/height-for-age, weight-for-age, weight-for-length, weight-for-height and body mass index-for-age: Methods and development. Geneva: WHO; 2006.
18. Lauderdale DS, Knutson KL, Yan LL, et al. Objectively measured sleep characteristics among early-middle-aged adults: the CARDIA study. *Am J Epidemiol.* 2006; 164:5–16. [PubMed: 16740591]
19. Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. *Am J Clin Nutr.* 1982; 36:936–942. [PubMed: 7137077]
20. Rockett HR, Breitenbach M, Frazier AL, et al. Validation of a youth/adolescent food frequency questionnaire. *Prev Med.* 1997; 26:808–816. [PubMed: 9388792]
21. Rockett HR, Wolf AM, Colditz GA. Development and reproducibility of a food frequency questionnaire to assess diets of older children and adolescents. *J Am Diet Assoc.* 1995; 95:336–340. [PubMed: 7860946]
22. Cole SR, Hernan MA. Constructing inverse probability weights for marginal structural models. *Am J Epidemiol.* 2008; 168:656–664. [PubMed: 18682488]
23. Dowd JB, Zajacova A. Long-term obesity and cardiovascular, inflammatory, and metabolic risk in U.S. adults. *Am J Prev Med.* 2014; 46:578–584. [PubMed: 24842734]
24. Kelsey MM, Zaepfel A, Bjornstad P, Nadeau KJ. Age-related consequences of childhood obesity. *Gerontology.* 2014; 60:222–228. [PubMed: 24434909]
25. Waters KA, Mast BT, Vella S, et al. Structural equation modeling of sleep apnea, inflammation, and metabolic dysfunction in children. *J Sleep Res.* 2007; 16:388–395. [PubMed: 18036084]
26. Mendelson M, Borowik A, Michallet AS, et al. Sleep quality, sleep duration and physical activity in obese adolescents: effects of exercise training. *Pediatrics Obesity.* 2015 Mar 2. Epub ahead of print. doi: 10.1111/ijpo.12015
27. McDonald L, Wardle J, Llewellyn CH, et al. Sleep and nighttime energy consumption in early childhood: a population-based cohort study. *Pediatrics Obesity.* 2015
28. Scharf RJ, DeBoer MD. Sleep timing and longitudinal weight gain in 4- and 5-year-old children. *Pediatrics Obesity.* 2015; 10:141–148.
29. Bixler EO, Vgontzas AN, Lin HM, et al. Sleep disordered breathing in children in a general population sample: prevalence and risk factors. *Sleep.* 2009; 32:731–736. [PubMed: 19544748]
30. Spilsbury JC, Storfer-Isser A, Rosen CL, Redline S. Remission and incidence of obstructive sleep apnea from middle childhood to late adolescence. *Sleep.* 2015; 38:23–29. [PubMed: 25325456]

Figure 1.

Odds ratio (95% confidence interval) for the association between overweight/obesity (OW) duration in childhood and high risk of obstructive sleep apnoea in adulthood. The model was adjusted for baseline age, sex, race, education, adult OW status, leisure-time physical activity, follow-up years and time-dependent regular alcohol consumption and smoking status. * <0.05 compared with the never OW group.

Table 1

Characteristics at baseline and in adulthood (in 2010) by patterns of OW status in childhood

	Body mass index classification in childhood			
	Never OW	Weight cycling	Persistent OW	Incident OW
Number of participants	536	104	140	64
Male	45.9%	32.2%	38.6%	34.8%
African-American	32.5%	31.1%	33.6%	46.9%
Characteristics in childhood				
Number of exams ^a	3 (2–4)	3 (2–5)	3 (2–4)	3 (2.5–4)
Age at first measurement (year)	10.1 (2.9)	9.6 (2.8)	9.9 (2.5)	8.8 (2.6)
Body mass index (kg/m ²)	16.2 (1.8)	19.4 (3.3)	22.7 (4.2) ^d	16.6 (1.8)
Current smoking ^b	2.1%	3.3%	0%	0%
Regular alcohol drinking ^c	6.3%	11.1%	5.0%	17.2% ^d
Characteristics in adulthood				
Age (year)	43.3 (4.3)	42.9 (3.9)	42.4 (4.9) ^d	39.8 (4.7) ^d
Body mass index (kg/m ²)	28.2 (5.8)	32.4 (6.2) ^d	39.1 (8.6) ^d	34.7 (7.9) ^d
OW (%)	69%	92% ^d	98% ^d	92% ^d
Less than high school	59%	53%	53%	71%
Current smoking	26%	33%	32%	30%
Regular alcohol drinking ^c	50%	51%	47%	55%
Leisure-time physically active ^e	29%	24%	21%	24%
Sleep-related outcomes				
High-risk for OSA	117 (22%)	28 (27%)	52 (37%) ^d	17 (27%)
Habitual snoring	183 (34%)	31 (30%)	74 (52%) ^d	26 (41%) ^d
Excessive daytime sleepiness	125 (23%)	29 (28%)	48 (34%)	14 (22%)

OSA, obstructive sleep apnoea; OW, obesity or overweight.

Data were expressed as *N*(%) and mean (SD) when appropriate; otherwise noted.^aNumber of exams were expressed as median (interquartile range).^bSmoking was first included in 1974–1975 and was asked for 3rd graders and older.^cAlcohol consumption was not measured at baseline but first included in 1984–1985 and was asked for 7th graders or older. Regular alcohol drinking was defined as at drinking least three times a week.^d $P < 0.05$ compared with never OW.^eLeisure-time physical activity was defined as self-reported physically ‘active’ and ‘very active’.

Table 2

Adjusted odds ratio (95% confidence interval) for the associations of childhood OW patterns with sleep-related outcomes

	High risk for obstructive sleep apnoea^a	Habitual snoring^b	Excessive daytime sleepiness^c
Never OW	Ref	Ref	Ref
Weight cycling	1.20 (0.91, 1.59)	0.82 (0.64, 1.06)	1.26 (0.94, 1.66)
Persistent OW	1.36 (1.04, 1.77)	1.60 (1.26, 2.04)	1.17 (0.89, 1.54)
Incident OW	1.47 (1.11, 1.96)	1.45 (1.12, 1.90)	0.80 (0.59, 1.09)

OW, overweight or obesity

^aModels were adjusted for baseline age, sex, race, education, adulthood overweight status, leisure-time physical activity levels, follow-up year, and time-dependent regular alcohol drinking and current smoking status.

^bModels were additionally adjusted for excessive daytime sleepiness, compared with footnote a.

^cModels were additionally adjusted for habitual snoring compared with footnote a.

Table 3

Adjusted odds ratio (95% confidence interval) for the associations of OW duration with habitual snoring and excessive daytime sleepiness

	Habitual snoring ^a	Excessive daytime sleepiness ^b
OW duration		
Never	Ref	Ref
1–4 years	1.51 (1.17, 1.95)	0.86 (0.62, 1.19)
5–8 years	1.34 (1.04, 1.73)	0.97 (0.72, 1.30)
8+ years	2.06 (1.50, 2.83)	1.37 (0.95, 1.98)
<i>P</i> for linear trend	<0.0001	0.22

OW, overweight or obesity

^a Models were adjusted for baseline age, sex, race, education, adulthood overweight status, leisure-time physical activity levels, follow-up year, time-dependent regular alcohol drinking, current smoking status and excessive daytime sleepiness.

^b Models were adjusted for baseline age, sex, race, education, adulthood overweight status, leisure-time physical activity levels, follow-up year, time-dependent regular alcohol drinking, current smoking status and habitual snoring.