Sleep-Disordered Breathing, Sleep Duration, and Childhood Overweight: A Longitudinal Cohort Study

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Objectives To examine independent associations between sleep-disordered breathing (SDB), sleep duration from birth through 6.75 years, and body mass index (BMI) through 15 years of age in a population-based cohort.

Study design The Avon Longitudinal Study of Parents and Children collected parent questionnaire data on child sleep duration and SDB symptoms from birth through 6.75 years and child BMI from the Avon Longitudinal Study of Parents and Children research clinics (n = 1899). For SDB, logistic regression models—minimal, confounder, and confounder + sleep duration adjusted—examined associations with BMI at 7, 10, and 15 years of age. For short sleep duration (≤10th percentile), comparable SDB-adjusted models examined associations with BMI at 15 years of age.

Results Children with the worst SDB symptoms vs asymptomatic children, had increased odds of overweight at 7 (OR = 2.08, 95% CI = 1.04-4.17), 10 (OR = 1.79, 95% CI = 1.02-3.16), and 15 years of age (OR = 2.25, 95% CI = 1.27-3.97) in models adjusted for sleep duration. Similarly, short sleep duration at ≈5-6 years was associated with overweight at 15 years, independent of SDB. Children with short sleep duration at 4.75 years were more likely to be overweight at 15 years in minimally (OR = 2.21, 95% CI = 1.52-3.20), confounder (OR = 1.99, 95% CI = 1.34-2.96), and SDB-adjusted (OR = 2.04, 95% CI = 1.36-3.04) models.

Conclusions Both SDB and short sleep duration significantly and independently increase children's odds of becoming overweight. Findings underscore the potential importance of early identification and remediation of SDB, along with insufficient sleep, as strategies for reducing childhood obesity. *(J Pediatr 2014; ■: ■-■).*

See related article, p •••

Both sleep-disordered breathing (SDB) and short sleep duration are associated with childhood obesity.[1-4] SDB ranges from snoring to obstructive sleep apnea and peaks at 2-8 years of age.[5,6] Mechanisms linking SDB to obesity are multifactorial and complex. They are held to involve inflammation and insulin resistance,[1,4,7] appetite-regulating hormones,[8,9] and sleep disruption[1,3] often with reciprocal effects.[2,3,7] Adenotonsillar hypertrophy is the main remediable cause of SDB in young children.[10,11] In the context of a childhood obesity epidemic, a second "obesity phenotype" of SDB, more similar to that seen in adults, has been proposed.[12] Short sleep duration also increases obesity risk[13,14] in longitudinal data from early[15-17] and middle childhood,[18] through adolescence.[19] In fact, increasing young children's sleep is considered among the most promising strategies for reducing childhood obesity.[20-22] Mechanisms are similar to those for SDB,[1-4] but also include effects upon biological (circadian) and social (household) rhythms.[2,3] In recent years, short sleep duration has eclipsed SDB as a putative risk factor in the literature on childhood obesity.

Though SDB and short sleep duration are increasingly recognized as sharing potential pathways to obesity, their independent associations with obesity throughout childhood remain unexplored. Of the near dozen longitudinal studies of SDB in children[5,23-30] just 2 assessed body mass index (BMI) outcomes.[27,28] Both showed an association with higher BMI. However, neither tracked SDB from early childhood, assessed BMI beyond a single follow-up, or adjusted for multiple confounding factors, particularly sleep duration. This latter gap is significant because persistent short sleep from 2-6 years of age can elevate obesity risk by 4-fold[15] and because a sizable component of childhood obesity is set by 5-7 years of age.[31-33] Thus, failing to account for sleep duration in early childhood may lead to confounding of the association between SDB and subsequent obesity. Similarly, failure to account for SDB may lead to confounding of the association between sleep duration and obesity. Although 2 recent studies report that sleep timing[34] and duration[34,35] elevate obesity risk in children, independent of SDB, neither was longitudinal.

ALSPAC	Avon Longitudinal Study of Parents and Children
BMI	Body mass index
SDB	Sleep-disordered breathing
T&A	Tonsillectomy and/or adenoidectomy

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Supported by the National Heart, Lung, and Blood Institute (R21HL091241). L.H. is funded by a UK Medical Research Council fellowship (G1002375) and works in a unit that receives core funding from the UK Medical Research Council and the University of Bristol (MC_UU_12013/9). The authors declare no conflicts of interest.

http://dx.doi.org/10.1016/j.jpeds.2014.11.001

This study addresses the above gaps in knowledge, using data from a longitudinal cohort study. We examined the independent association between both SDB and sleep duration in early childhood on BMI later in childhood and adolescence (7, 10, and 15 years of age). We focused upon early childhood as the exposure period because sleep patterns in those years, compared with subsequent years, are more predictive of overweight in late childhood and adolescence[36] and because early childhood is a key period for excess weight gain.[37] SDB risk, in the absence of objective obstructive sleep apnea measures within this large cohort, was assessed as in our previous work through composite trajectories of its hallmark symptoms (ie, clusters) of snoring, mouth-breathing, and witnessed apnea, prior to 7 years of age.[5] Our primary research hypotheses were: (1) SDB symptom clusters are associated with obesity at 7, 10, and 15 years of age, independent of sleep duration; and (2) shorter sleep duration in early life is associated with obesity at 15 years of age, independent of SDB. Secondarily, we examined associations with underweight and short stature, which can occur with severe, untreated SDB in early life.[38] This study builds upon prior analyses from the Avon Longitudinal Study of Parents and Children (ALSPAC) that describe the natural history of SDB,[5] the SDB symptom clusters,[39] sleep duration,[40] and growth.[16,41,42] We undertook this analysis in ALSPAC because its longitudinal data offer a unique opportunity for exploring the above timely hypotheses.

Methods

The ALSPAC cohort study of child health and development enrolled pregnant women from southwest England with expected delivery dates between April 1991 and December 1992. A total of 14 541 pregnant women were enrolled. Described in detail elsewhere (http://www.bristol.ac.uk/alspac/), the cohort is broadly representative of the United Kingdom population in terms of socioeconomic status, with slight under-representation of ethnic minorities, and over-representation of wealthier families.[43]

We incorporated potential confounders based upon prior work. Maternal demographic variables included education (4 levels, "degree" = highest), age at delivery, prepregnancy BMI, and parity; all were reported by the mother in questionnaires during pregnancy. Child demographics included sex, birth weight (extracted from medical records), and weight and height at 6 months (as described in a previous publication).[44,45] Sleep duration was calculated from maternal report of typical weekday bed- and wake-times at ages 18 months, 2.5 years, 4.75 years, 5.75 years, and 6.75 years.[40] These timepoints were chosen to represent different stages of childhood during the period in which our exposure (SDB) is assessed. At each age, we divided sleep duration into 3 categories: ≤10th percentile (≤10, ≤10, ≤10.5, ≤10.5, and ≤9.5 hours, respectively), >10th and <90th percentile, and ≥90th percentile (≥12.5, ≥12.5, ≥12.1, ≥12, and ≥11.75 hours, respectively) and treated the measures as categorical variables because of possible nonlinear associations with other variables. Tonsillectomy and/or adenoidectomy (T&A) is the first line treatment for SDB.[46,47] Responses were grouped to indicate any or no T&A.

Height and weight at ages 7, 10, and 15 years were measured at ALSPAC research clinics, with the child in light clothing and no shoes. We calculated BMI as weight/height^2 (kg/m^2). We used International Obesity Task Force definitions of obesity as a BMI >95th percentile for age and sex, and underweight as a BMI <5th percentile for age and sex.[48] Short stature was defined as <5th percentile for age and sex using internally derived percentiles. We selected measures of BMI and height at ages 7, 10, and 15 years because they occur after our assessments of SDB, and, for most children, these ages represent the period immediately following adiposity rebound (7 years), just prior to puberty (10 years), and during puberty (15 years). ALSPAC questionnaires, mailed when children were 6, 18, 30, 42, 57, 69, and 81 months old, asked parents about their child's snoring, observed apnea, and mouth-breathing. These measures, consistent with guidelines for clinical diagnosis of SDB,[49] were: (1) snoring: "Does she snore for more than a few minutes at a time?"; (2) apnea: "When asleep, does she seem to stop breathing or hold breath for several seconds at a time?"; and (3) mouth-breathing: "Does she breathe through her mouth rather than her nose?". Responses were categorized along ordinal 3, 4, or 5 level scales. Given this variation in response categories, we extrapolated values to a common scale (0-100), anchored by the extreme "always" and "never" or "rarely/never" categories, with proportionate spacing in-between (ie, a 4 category scale was recoded as 0, 33, 66, 100). Variables were transformed to z-scores. Higher scores indicate more symptoms.

To capture SDB's multisymptom, changing nature, we classified snoring, witnessed apnea, and mouth-breathing into trajectories or "clusters." SDB z-scores were partitioned into clusters using the k-means model procedures of SAS FASTCLUS v 9.2 (SAS Institute, Cary, North Carolina). We examined the uniqueness of clusters with ANOVA tests; linear discriminant functional analysis was used to test for the significance of differences between them. Independent clinicians examined cluster plots for clinical relevance. To assess clinical validity, plots were analyzed in relation to 2 'criterion' variables associated with SDB: wheezing and tonsil and/or adenoid removal. Through this cluster analysis, described in more detail elsewhere,[39] we produced 5 conceptually and statistically distinct clusters for children with SDB data for ≥3 of 7 possible timepoints. They included 1 asymptomatic ("normals," 45% of sample) and 4 symptomatic (55% of sample) trajectories with distinct temporal distributions of symptoms.

Statistical Analyses

Analyses are based upon children with complete data for exposure variables (SDB and sleep duration) as well as for BMI and all potential confounders, at ages 7, 10, and 15 years. Sample characteristics are presented by SDB cluster, as numbers (percentages) and means (SDs). Associations between SDB cluster and sleep duration (unadjusted) are shown as numbers (percentages). Our a priori intention was to consider underweight,

Table I. Characteristics of participants (N = 1899)

Characteristics† (N = 1899*)	SDB clusters: No symptoms (N = 858)	Peak at 6 mo (N = 353)	Peak at 18 mo (N = 200)	Worst case (N = 126)	Late symptoms (N = 362)	*P* value**
Female sex	430 (46.0%)	166 (47.0%)	101 (50.5%)	50 (39.7%)	187 (51.7%)	.16
Birth weight (kg)	3.4 (0.5)	3.5 (0.5)	3.5 (0.5)	3.5 (0.6)	3.4 (0.5)	.87
Maternal education						
Less than O-level	102 (11.9%)	57 (16.2%)	37 (18.5%)	24 (19.1%)	68 (18.8%)	
O-level	289 (33.7%)	119 (33.7%)	61 (30.5%)	43 (34.1%)	128 (35.4%)	
A-level	261 (30.4%)	101 (28.6%)	66 (33.0%)	36 (28.6%)	103 (28.5%)	
Degree or above	206 (24.0%)	76 (21.5%)	36 (18.0%)	23 (18.3%)	63 (17.4%)	.04
Maternal age (y)	30.1 (4.3)	29.7 (4.1)	29.9 (4.8)	29.0 (4.4)	29.2 (4.2)	.01
Maternal parity						
0	406 (47.3%)	155 (43.9%)	107 (53.5%)	62 (49.2%)	171 (47.2%)	
1	285 (33.2%)	136 (38.5%)	60 (30.0%)	38 (30.2%)	126 (34.8%)	
2	117 (13.6%)	37 (10.5%)	26 (13.0%)	18 (14.3%)	47 (13.0%)	
3 or more	50 (5.8%)	25 (7.1%)	7 (3.5%)	8 (6.4%)	18 (5.0%)	.45
Maternal prepregnancy BMI (kg/m^2)	22.7 (3.4)	22.5 (3.6)	22.9 (3.8)	23.4 (4.0)	23.2 (3.9)	.05
T&A	17 (2.0%)	18 (5.1%)	9 (4.5%)	30 (23.8%)	29 (8.0%)	<.001
Weight at 6 mo (kg)	4.5 (0.5)	4.6 (0.5)	4.5 (0.6)	4.5 (0.6)	4.5 (0.5)	.64
Height at 6 mo (cm)	55.2 (1.7)	55.1 (1.8)	55.2 (1.8)	55.0 (2.0)	55.0 (1.7)	.65
BMI at age 7						
Underweight‡	30 (3.5%)	11 (3.1%)	8 (4.0%)	8 (6.4%)	8 (2.2%)	
Normal BMI	776 (90.4%)	323 (91.5%)	181 (90.5%)	103 (81.8%)	317 (87.6%)	
Obese§	52 (6.1%)	19 (5.4%)	11 (5.5%)	15 (11.9%)	37 (10.2%)	.02
BMI at age 10						
Underweight	22 (2.6%)	9 (2.6%)	9 (4.5%)	5 (4.0%)	10 (2.8%)	
Normal BMI	748 (87.2%)	306 (86.7%)	170 (85.0%)	98 (77.8%)	300 (82.9%)	
Obese	88 (10.3%)	38 (10.8%)	21 (10.5%)	23 (18.3%)	52 (14.4%)	.11
BMI at age 15						
Underweight	22 (2.6%)	11 (3.1%)	7 (3.5%)	8 (6.4%)	7 (1.9%)	
Normal BMI	753 (87.8%)	312 (88.4%)	171 (85.5%)	95 (75.4%)	302 (83.4%)	
Obese	83 (9.7%)	30 (8.5%)	22 (11.0%)	23 (18.3%)	53 (14.6%)	.003
Short stature at 7¶	21 (2.5%)	8 (2.3%)	7 (3.5%)	5 (4.0%)	11 (3.0%)	.77
Short stature at 10	13 (1.5%)	5 (2.5%)	5 (2.5%)	4 (3.2%)	10 (2.8%)	.42
Short stature at 15	13 (1.5%)	2 (0.6%)	5 (2.5%)	3 (2.4%)	6 (1.7%)	.40

*Based upon the 1899 participants with complete data on all variables.
†Values are numbers (percentages) or means (SDs).
‡Underweight defined as BMI less than the fifth percentile for age and sex, according to the International Obesity Task Force.
§Obese defined as BMI greater than the 95th percentile for age and sex, according to the International Obesity Task Force.
¶Short stature is defined as height less than the fifth percentile for age and sex.
**From χ^2 tests or linear regressions.

overweight, and short stature as outcomes in our analyses. However, given the low prevalence of short stature and underweight in our population, we only present descriptive statistics for these outcomes and do not carry out regression analyses. We used logistic regression to estimate the associations of SDB cluster (with "no symptoms" as the reference category) with obesity at ages 7, 10, and 15 years. We excluded participants with underweight BMI because we hypothesized that SDB might also be associated with weight status, and we, therefore, did not want to bias our results by mixing normal weight and underweight people. Results are presented as ORs and 95% CI. Three models are presented: (1) minimally adjusted; (2) confounder adjusted; and (3) confounder adjusted with additional adjustment for sleep duration. Minimally adjusted models incorporate child sex and exact age at BMI assessment; this model was a priori determined given known differences in obesity prevalence between males and females and variation in exact age at the 7, 10, and 15 year follow-ups. Confounder adjusted models included these variables, as well as maternal and child confounders as detailed above. The included confounders were determined a priori using subject knowledge about determinants of both SDB and obesity. Tests for statistical interactions were conducted to determine whether T&A history or sleep duration modified the association between SDB cluster and obese BMI; we a priori determined that these variables were potential effect modifiers. Logistic regression was used to assess the associations of sleep duration with BMI status at 15 years with and without adjustment for confounders and SDB.

Results

The participants with complete data on all variables are described by SDB clusters (n = 1899) (Table I). The 5 clusters (Figure) are: (1) no symptoms—asymptomatic throughout (45%); (2) peak at 6 months—symptoms peak at 6 months, then abate (18.5%); (3) peak at 18 months—symptoms peak at 18 months, then abate (10.5%); (4) worst case—symptoms rise at 18 months, peak at 30-40 months, then remain high (7%); and (5) late symptom—modest symptoms appear at 42 months, and remain high (19%). Maternal education, age, and prepregnancy BMI differed by cluster, as did child history of T&A and BMI at 7 and 15 years. Neither underweight,

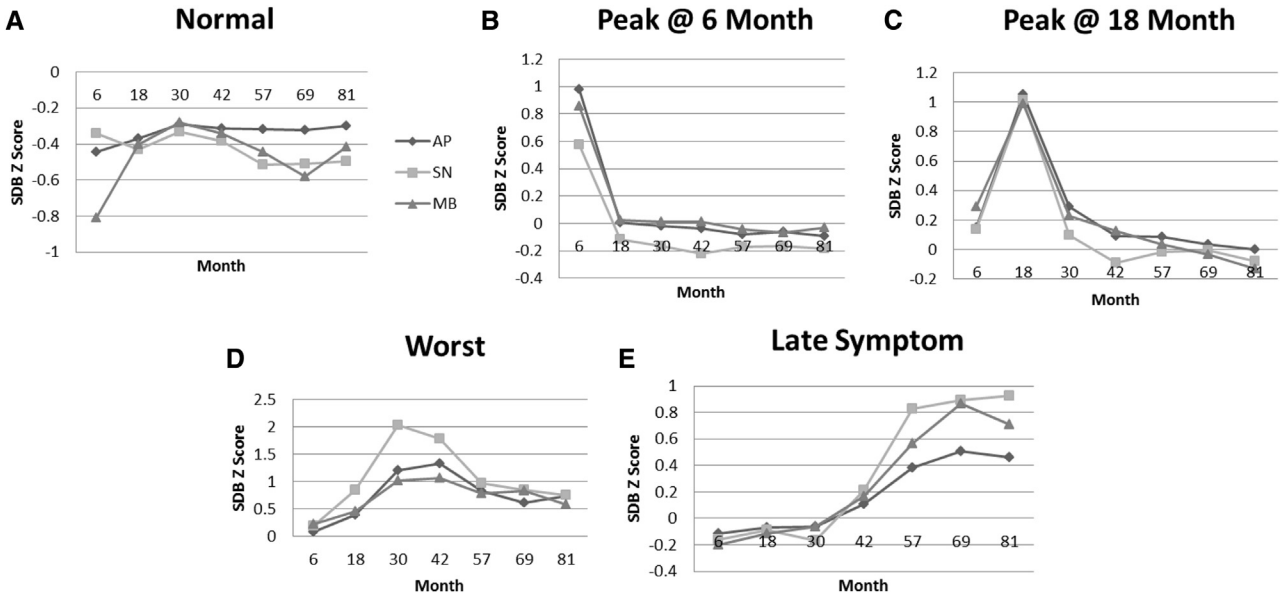

Figure. **A,** Normal cluster: apnea (AP), snoring (SN), and mouth-breathing (MB) symptom SD (Z) scores. **B,** Peak at 6-month cluster: AP, SN, and MB symptom SD (Z) scores. **C,** Peak at 18-Month cluster: AP, SN, and MB symptom SD (Z) scores. **D,** Worst symptom cluster: AP, SN, and MB symptom SD (Z) scores. **E,** Late symptom cluster: AP, SN, and MB symptom SD (Z) scores.

nor short stature appeared to differ by cluster. Given insufficient cell sizes for these outcomes, we did not pursue further analyses of them. Participants from the larger ALSPAC cohort excluded from our analysis because of incomplete data tended to be of lower maternal education, younger maternal age, and higher maternal BMI and were less likely to be in the "no symptoms" cluster.

Sleep Duration and SDB Cluster Associations across Childhood

We examined associations between each SDB cluster, and sleep duration at 18 months, 2.5 years, 5.75 years, and 6.75 years of age (Table II). None were significant; comparable proportions within each cluster were short, average, and long sleepers.

BMI Outcomes at ages 7, 10, and 15 Years (N = 1844)

Regardless of adjustment for any confounders or sleep duration, the "worst case" cluster had twice as high odds of becoming obese by 7, 10, and 15 years of age, compared with the asymptomatic group (Table III). Minimally adjusted results at 7 (OR = 2.15, 95% CI = 1.17-3.96), 10 (OR = 1.90, 95% CI = 1.14-3.16), and 15 years (OR = 2.18 95% CI = 1.31-3.64) mirrored confounder and sleep duration adjusted results at 7 (OR = 2.08, 95% CI = 1.04-4.17), 10 (OR = 1.79, 95% CI = 1.02-3.16), and 15 years (OR = 2.25, 95% CI = 1.27-3.97). Likewise, in the "late symptom" cluster, increased odds of overweight at 7 and 15 years (≈80% and ≈60%, respectively) did not vary across models. Children with "late symptoms" had a borderline significant increased odds of obesity at 10 years (OR = 1.45, 95% CI = 1.00-2.10) in minimally adjusted analyses, which became nonsignificant after adjustment. No other cluster-time associations were significant.

We sought to determine whether sleep duration modified the statistical effects of SDB upon BMI. Though we have insufficient power to examine this question definitively, the data do not suggest significant interactions for sleep duration at 18 months (P = .81), 2.5 years (P = .26), 4.75 years (P = .33), or at 5.75 years (P = .77). Interactions between T&A and clusters were not significant at 7 (P = .97), 10 (P = .32) or 15 years (P = .16); thus, having a T&A did not modify these outcomes (not shown).

Associations between Sleep Duration across Childhood and Obesity at Age 15 Years (N = 1844)

Regardless of adjustment for confounders or SDB clusters, short sleep duration at 4.75 and 5.75 years was significantly associated with increased odds of obesity at 15 years (Table IV). At 4.75 years, the minimally (OR = 2.21, 95% CI = 1.52-3.20), confounder (OR = 1.99, 95% CI = 1.34-2.96), and SDB cluster (OR = 2.04, 95% CI = 1.36-3.04) adjusted increased odds of subsequent obesity were nearly identical. Likewise, short sleep duration at 5.75 years was consistently associated with 55%-65% increased odds of obesity at 15 years. Short sleep duration at other time periods was borderline significant. Children with the longest sleep duration at 2.5 years were less likely to be obese at 15 years (OR = 0.50, 95% CI = 0.26-0.97) in minimally adjusted analyses only.

Discussion

Roots of obesity in late childhood, through to adulthood, most likely extend back to early childhood.[31,50-52] They

Table II. Associations between sleep duration across childhood and SDB clusters (N = 1899)

	SDB clusters					
	No symptoms, N (%)*	Peak at 6 mo, N (%)*	Peak at 18 mo, N (%)*	Worst case, N (%)*	Late symptoms, N (%)*	*P* value
Sleep duration at 18 mo†						
Short	118 (13.8%)	48 (13.6%)	32 (16.0%)	18 (14.3%)	46 (12.7%)	
Average	622 (72.5%)	266 (75.4%)	156 (78.0%)	94 (74.6%)	267 (73.8%)	
Long	118 (13.8%)	39 (11.1%)	12 (6.0%)	14 (11.1%)	49 (13.5%)	.21
Sleep duration at 2.5 y†						
Short	95 (11.1%)	48 (13.6%)	32 (16.0%)	18 (14.3%)	61 (16.9%)	
Average	679 (79.1%)	275 (77.9%)	153 (76.5%)	97 (77.0%)	271 (74.9%)	
Long	84 (9.8%)	30 (8.5%)	15 (7.5%)	11 (8.7%)	30 (8.3%)	.28
Sleep duration at 4.75 y†						
Short	100 (11.7%)	44 (12.5%)	27 (13.5%)	9 (7.1%)	46 (12.7%)	
Average	676 (78.8%)	283 (80.2%)	160 (80.0%)	106 (84.1%)	287 (79.3%)	
Long	82 (9.6%)	26 (7.4%)	13 (6.5%)	11 (8.7%)	29 (8.0%)	.61
Sleep duration at 5.75 y†						
Short	143 (16.7%)	57 (16.2%)	38 (19.0%)	15 (11.9%)	60 (16.6%)	
Average	548 (63.9%)	233 (66.0%)	134 (67.0%)	88 (69.8%)	226 (62.4%)	
Long	167 (19.5%)	63 (17.9%)	28 (14.0%)	23 (18.3%)	76 (21.0%)	.50
Sleep duration at 6.75 y†						
Short	95 (11.1%)	39 (11.1%)	31 (15.5%)	11 (8.7%)	43 (11.1%)	
Average	669 (78.0%)	282 (79.9%)	148 (74.0%)	104 (82.5%)	279 (77.1%)	
Long	94 (11.0%)	32 (9.1%)	21 (10.5%)	11 (8.7%)	43 (11.9%)	.63

*Numbers and % of people within each cluster who have short, average and long sleep duration; *P* values from χ^2 tests.
†At 18 months and 2.5 years, short sleep duration is ≤10 hours; average sleep duration is >10 and <12.5 hours, long sleep duration is ≥12.5 hours. At 4.75 years, short sleep duration is ≤10.5 hours; average sleep duration is >10.5 and <12.08 hours, long sleep duration is ≥12.08 hours. At 5.75 years, short sleep duration is ≤10.5 hours; average sleep duration is >10.5 and <12.0 hours, long sleep duration is ≥12.0 hours. At 6.75 years, short sleep duration is ≤9.5 hours; average sleep duration is >9.5 and <11.75 hours, long sleep duration is ≥11.75 hours.

include SDB and short sleep duration, which we assessed from birth to nearly 7 years of age, to determine associations with subsequent obesity. Compared with children without SDB symptoms, those with the worst symptoms (peak age ≈ 2.5 years) had double the odds of obesity at ages 7, 10, and 15 years, independent of sleep duration. Children whose SDB peaked later (≈5-6 years) had a 60%-80% increased odds, again, regardless of sleep duration. Overall,

Table III. The association of SDB clusters with overweight across childhood and adolescence (N = 1844)

	SDB clusters				
	No symptoms	Peak at 6 mo, OR (95% CI)†	Peak at 18 mo, OR (95% CI)	Worst case, OR (95% CI)	Late symptoms, OR (95% CI)
Minimally adjusted results*					
Obesity at age 7 years‡	1 (ref)	0.88 (0.51-1.51) *P* = .63	0.91 (0.47-1.78) *P* = .79	2.15 (1.17-3.96) *P* = .01	1.75 (1.12-2.72) *P* = .01
Obesity at age 10 years	1 (ref)	1.05 (0.70-1.57) *P* = .82	1.04 (0.63-1.72) *P* = .89	1.90 (1.14-3.16) *P* = .01	1.45 (1.00-2.10) *P* = .05
Obesity at age 15 years	1 (ref)	0.87 (0.56-1.35) *P* = .54	1.17 (0.71-1.92) *P* = .54	2.18 (1.31-3.64) *P* = .003	1.60 (1.10-2.31) *P* = .01
Confounder adjusted results§					
Obesity at age 7 years‡	1 (ref)	0.83 (0.47-1.47) *P* = .53	0.91 (0.46-1.83) *P* = .79	2.08 (1.05-4.11) *P* = .04	1.78 (1.11-2.85) *P* = .02
Obesity at age 10 years	1 (ref)	1.02 (0.67-1.56) *P* = .92	1.00 (0.59-1.69) *P* = 1.00	1.76 (1.01-3.08) *P* = .05	1.40 (0.95-2.07) *P* = .09
Obesity at age 15 years	1 (ref)	0.86 (0.54-1.36) *P* = .52	1.14 (0.68-1.92) *P* = .62	2.17 (1.24-3.81) *P* = .01	1.58 (1.07-2.35) *P* = .02
Confounder adjusted results with additional adjustment for sleep duration¶					
Obesity at age 7 years‡	1 (ref)	0.82 (0.46-1.46) *P* = .51	0.91 (0.45-1.84) *P* = .79	2.08 (1.04-4.17) *P* = .04	1.78 (1.11-2.87) *P* = .02
Obesity at age 10 years	1 (ref)	1.03 (0.67-1.57) *P* = .91	0.97 (0.57-1.66) *P* = .93	1.79 (1.02-3.16) *P* = .04	1.38 (0.93-2.05) *P* = .11
Obesity at age 15 years	1 (ref)	0.87 (0.55-1.38) *P* = .55	1.12 (0.66-1.90) *P* = .66	2.25 (1.27-3.97) *P* = .01	1.56 (1.05-2.33) *P* = .03

*Analyses are adjusted only for child's sex and age at BMI assessment.
†ORs (95% CIs) compare the odds of obesity vs normal BMI, with each SDB cluster compared with the 'no symptoms' cluster.
‡Obesity defined as BMI greater than the 95th percentile for age and sex, according to the International Obesity Task Force. Underweight children are excluded from these analyses.
§Analyses are adjusted for child's sex, age at BMI/height assessment and birth weight, child's estimated weight and height at 6 months, maternal education, age, parity and prepregnancy BMI, and T&A.
¶Analyses are adjusted for the same as above, with the addition of sleep duration (at 18 months, 2.5, 4.75, 5.75, and 6.75 years). Each measure of sleep duration is treated as a categorical variable; ≤10th percentile, >10th and <90th percentile, and ≥90th percentile.

Table IV. Associations between sleep duration across childhood and obesity at age 15 years (N = 1899)

	Minimally adjusted*, OR (95% CI)§	Confounder adjusted†, OR (95% CI)§	Confounder adjusted with additional adjustment for SDB cluster‡, OR (95% CI)§
Sleep duration at 18 mo§,¶			
Short	1.48 (1.00-2.17)	1.22 (0.80-1.84)	1.23 (0.81-1.86)
	$P = .05$	$P = .35$	$P = .34$
Average	1 (ref)	1 (ref)	1 (ref)
Long	1.16 (0.75-1.80)	1.05 (0.65-1.68)	1.05 (0.65-1.69)
	$P = .50$	$P = .85$	$P = .85$
Sleep duration at 2.5 y§,¶			
Short	1.44 (0.98-2.10)	1.38 (0.92-2.08)	1.33 (0.89-2.01)
	$P = .06$	$P = .12$	$P = .17$
Average	1 (ref)	1 (ref)	1 (ref)
Long	0.50 (0.26-0.97)	0.53 (0.26-1.05)	0.53 (0.27-1.06)
	$P = .04$	$P = .07$	$P = .07$
Sleep duration at 4.75 y§,¶			
Short	2.21 (1.52-3.20)	1.99 (1.34-2.96)	2.04 (1.36-3.04)
	$P < .001$	$P = .001$	$P = .001$
Average	1 (ref)	1 (ref)	1 (ref)
Long	1.08 (0.64-1.84)	0.82 (0.46-1.47)	0.86 (0.48-1.55)
	$P = .77$	$P = .51$	$P = .62$
Sleep duration at 5.75 y§,¶			
Short	1.54 (1.08-2.20)	1.62 (1.10-2.37)	1.64 (1.11-2.41)
	$P = .02$	$P = .01$	$P = .01$
Average	1 (ref)	1 (ref)	1 (ref)
Long	0.83 (0.55-1.25)	0.72 (0.46-1.12)	0.73 (0.47-1.13)
	$P = .38$	$P = .14$	$P = .16$
Sleep duration at 6.75 y§,¶			
Short	1.50 (1.00-2.25)	1.34 (0.87-2.06)	1.33 (0.86-2.06)
	$P = .05$	$P = .19$	$P = .20$
Average	1 (ref)	1 (ref)	1 (ref)
Long	0.94 (0.58-1.54)	0.81 (0.48-1.37)	0.82 (0.48-1.39)
	$P = .81$	$P = .43$	$P = .46$

*Analyses are adjusted only for child's sex and age at BMI assessment.
†Analyses are adjusted for child's sex, age at BMI/height assessment and birth weight, child's estimated weight and height at 6 months, maternal education, age, parity and prepregnancy BMI, and T&A.
‡Analyses are adjusted for the same variables as above, with the addition of SDB clusters.
§ORs (95% CIs) for obesity compared with normal weight at age 15 years; compare short and long sleep duration with average sleep duration. Obesity is defined as BMI greater than the 95% percentile for age and sex, according to the International Obesity Task Force.
¶At 18 months and 2.5 years, short sleep duration is ≤10 hours; average sleep duration is >10 and <12.5 hours, long sleep duration is ≥12.5 hours. At 4.75 years, short sleep duration is ≤10.5 hours; average sleep duration is >10.5 and <12.08 hours, long sleep duration is ≥12.08 hours. At 5.75 years, short sleep duration is ≤10.5 hours; average sleep duration is >10.5 and <12.0 hours, long sleep duration is ≥12.0 hours. At 6.75 years, short sleep duration is ≤9.5 hours; average sleep duration is >9.5 and <11.75 hours, long sleep duration is ≥11.75 hours.

25% of children in this population-based cohort had increased odds of obesity in association with early SDB symptoms. Conversely, short sleep duration at ≈5-6 years was associated with almost identical increased odds of obesity at 15 years (60%-100%), independent of SDB. Thus, even though SDB and sleep duration share multiple common pathways to obesity in children, our findings suggest that their effects are of comparable magnitude and independent of one another.

This study's strengths include a large longitudinal cohort with sleep exposures and BMI assessed at multiple timepoints, control for multiple confounders, and a previously established[39] SDB assessment for which each of 3 symptom constructs has been validated against polysomnography.[53] The study has limitations. SDB trajectories extended just through 6.75 years. Other work finds a 10% incidence of SDB between 8 and 13 years of age,[27] stable snoring prevalence from 4-12 years of age,[17] and adenotonsillar enlargement beyond 7-8 years of age in children who snore.[54] Thus, SDB beyond 6.75 years might have affected BMI at 10 and 15 years. Likewise, sleep duration at later periods may affect subsequent BMI. We censored our duration measure at 6.75 years to permit analysis of its contemporaneous confounding effects with SDB. More broadly, our focus upon earlier childhood sleep exposures reflects evidence that sleep patterns early in childhood compared with late childhood are more strongly associated with subsequent obesity, and that excess weight gain in early childhood tracks to later years.[31-33,37,50] Reverse causality is possible (ie, overweight may cause SDB). To address this, we adjusted for maternal prepregnancy BMI, and child's weight and length at 6 months of age, both of which are strong determinants of a child's later BMI. We did not adjust for later BMI measures as this would impose over-adjustment and likely preclude valid assessment of our hypothesis. As in any longitudinal study, loss to follow-up has been socially patterned and, therefore, our participants tended to be of higher socioeconomic position compared with those excluded because of missing data and were less likely to have SDB symptoms. Although this means that our population is not representative of the entire cohort, this is unlikely to have biased our results; in order to cause bias, the association between SDB and overweight/obesity would need to differ between those included and excluded from our analysis; we do not think this is likely.

Another limitation was the use of 3 symptom items, rather than gold-standard polysomnography, in the nearly

2000 subjects. These symptoms no doubt misclassified some subjects as having SDB, or not, as would be defined by objective testing.[55] However, these symptoms are highly likely to serve the intended research purpose: to identify effectively in aggregate a group of children at increased risk for SDB. These 3 symptoms (snoring, observed apnea, and mouth-breathing) correspond to 3 key, simply worded items from the Pediatric Sleep Questionnaire–Sleep-Related Breathing Disorders Scale, each of which was individually validated as predictive of polysomnographic results.[53] The scale itself has been highlighted by several reviews as one of the most appropriate instruments developed for this purpose.[56,57]

Regarding SDB, our findings are consistent with the 2 prior longitudinal studies of BMI outcomes in children. One study showed that SDB at baseline (mean age = 8.5 years) was associated with a 3-fold increased odds of obesity at 5-year follow-up.[27] In the other study, only children who were both overweight at baseline (mean age = 10.2 years) and had severe SDB, remained overweight at 4-year follow-up.[28] Notably, they employed polysomnography (the gold standard for assessing SDB), though neither was population-based or incorporated multiple potential confounders (including sleep duration). Still, even though these reports and the present study provide longitudinal rather than randomized controlled data that could prove cause-and-effect, they combine to suggest that SDB in early life could promote overweight in later life particularly among those children who have an initial proclivity of predisposition to overweight.

Regarding sleep duration, our findings are consistent with effect sizes in prior longitudinal studies of children, which range from 40%-100% increased risk of subsequent obesity,[14,58] despite variable definitions of short sleep duration. Several studies point to sleep duration at 3-7 years of age, as a critical risk period for obesity in children. Likewise, we found that short sleep duration (≤10.5 hours) at 4.75 and 5.75 years of age was associated with the likelihood of being obese at age 15 years.

Our findings affirm the concept of healthy sleep, broadly conceived, as a foundation for healthy weight throughout childhood and into adulthood. Clinically, this broad approach should encompass, as suggested by our new data, attention to symptoms of SDB, in addition to insufficient sleep. Despite the bright spotlight in recent years on the potential influence of insufficient sleep on obesity risk, the potential contribution of untreated childhood SDB has received scant attention. Previous research also suggests that early childhood SDB may increase risks for adverse neurobehavioral outcomes that become apparent only years later.[59,60] If morbidity such as obesity and attention deficit/hyperactivity disorder is in fact promoted by exposures to inadequate sleep early in life, new more sensitive and effective approaches for identification of those consequential yet remediable exposures will be required. These considerations highlight the challenges inherent with young patients, on rapid developmental trajectories in which outcomes can remain occult until later ages, yet have potential for lifelong effects on human health. ■

Submitted for publication Jul 8, 2014; last revision received Oct 6, 2014; accepted Nov 3, 2014.

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