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 2015;166:632-9)*.

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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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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,

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