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Pediatric Sleep Disorders and Special Educational Need at 8 Years: A Population-Based Cohort Study

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KEY WORDS

sleep-disordered breathing, behavior sleep problem, longitudinal, special education

ABBREVIATIONS

ALSPAC—Avon Longitudinal Study of Parents and Children
BSP—behavioral sleep problem
CI—confidence interval
OR—odds ratio
SDB—sleep disordered breathing
SEN—special educational need
SES—socioeconomic status

All authors meet the criteria for authorship. Dr Bonuck conceptualized and designed the study, drafted the initial manuscript, reviewed and modified the analyses in collaboration with Dr Xu, and incorporated co-author feedback into the final manuscript. Dr Xu developed the methods, carried out all statistical analyses, and reviewed and revised the final manuscript. Dr Rao drafted sections of the manuscript and reviewed the final version.

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WHAT'S KNOWN ON THIS SUBJECT: Sleep disordered breathing (SDB) and behavioral sleep problems (BSPs) affect cognitive, behavioral, and language development. No studies have examined associations between SDB and BSPs across early childhood, and later special education need (SEN), on a population basis.

WHAT THIS STUDY ADDS: A history of SDB through 5 years of age was associated with ∼40% increased odds of SEN at 8 years, among >11 000 children. BSPs were associated with 7% increased odds of SEN, for each additional ∼12 months of reported BSPs.

abstract

NIH

OBJECTIVES: To examine associations between sleep-disordered breathing (SDB) and behavioral sleep problems (BSPs) through 5 years of age and special educational need (SEN) at 8 years.

METHODS: Parents in the Avon Longitudinal Study of Parents and Children reported on children's snoring, witnessed apnea, and mouth-breathing at 6, 18, 30, 42, and 57 months, from which SDB symptom trajectories, or clusters, were derived. BSPs were based on report of ≥5 of 7 sleep behaviors at each of the 18-, 30-, 42-, and 57-month questionnaires. Parent report of SEN (yes/no) at 8 years was available for 11 049 children with SDB data and 11 467 children with BSP data. Multivariable logistic regression models were used to predict SEN outcome by SDB cluster and by cumulative report of SEN.

RESULTS: Controlling for 16 putative confounders, previous history of SDB and BSPs was significantly associated with an SEN. BSPs were associated with a 7% increased odds of SEN (95% confidence interval [CI] 1.01–1.15), for each ∼1-year interval at which a BSP was reported. SDB, overall, was associated with a near 40% increased odds of SEN (95% CI 1.18–1.62). Children in the worst symptom cluster were 60% more likely to have an SEN (95% CI 1.23–2.08).

CONCLUSIONS: In this population-based longitudinal study, history of either SDB or BSPs in the first 5 years of life was associated with increased likelihood of SEN at 8 years of age. Findings highlight the need for pediatric sleep disorder screening by early interventionists, early childhood educators, and health professionals. *Pediatrics* 2012;130:1–9

Pediatric sleep disorders result in disrupted, inefficient, and inadequate sleep.[1,2] The most prevalent and pernicious are behavioral sleep problems (BSPs) and sleep disordered breathing (SDB). Both may affect brain development and cause neuronal damage, particularly during critical early development periods.[1–3] Slow wave sleep, the most restorative form of sleep, is largely governed by the frontal cortex, which mediates higher functions, such as decision-making, ambition, and emotional regulation. Disrupting this restorative process via either sleep fragmentation or hypoxemia may affect frontal cortex functioning and lead to aspects of the behavioral phenotype seen with childhood obstructive sleep apnea.[1,2,4]

BSPs, characterized by inadequate and fragmented sleep, affects behavior[5,6] and cognition[5–7] and language development.[8] Similarly, SDB is linked to delayed development,[9] speech-language impairments,[10,11] and adverse behavioral[2,12] and cognitive[2,13,14] effects. Thus, both disorders can affect school functioning and educational need, in addition to being 2 to 3 times as prevalent among children with developmental delay or disability versus the typically developing child.[15] In the United States, 3 million 6- to 21-year-olds receive special education for conditions associated with sleep disorders (ie, developmental delay, learning disability, or autism); 40% to 80% also have attention-deficit disorder/attention-deficit hyperactivity disorder.[16]

While sleep disorders in early childhood may affect special educational needs, just a few studies have analyzed this association. Parents of children in Spain (mean age 11–12 years) in special versus mainstream schools reported significantly higher rates of both BSPs (32.3% vs 10.5%) and SDB (26.8% vs 5.7%),[17] affirming results of an earlier UK study of 4- to 12-year-olds from special versus mainstream educational venues for both BSP (23.8% vs 11.6%) and SDB (19.8% vs 9.0%).[18] An Australian study of 6- to 15-year-olds found similar results, although a low response rate, small sample size, and poor matching[19] limit generalizability.

This is the first prospective, population-based study of the associations between SDB and BSPs throughout early childhood and effect on later special education need (SEN). Given the dynamic, multisymptom expression of SDB's hallmark symptoms (snoring, apnea, and mouth-breathing) SDB was examined as a combined trajectory of these symptoms. This is a secondary analysis of observational data collected during the peak period in the development of SDB and BSPs, in a cohort of >11 000 children. The study had 2 specific research questions. Is cumulative report of BSP across 4 intervals of ~12 to 15 months from 18 to 57 months of age associated with an increased likelihood of an SEN determination at 8 years of age? Similarly, are SDB symptom trajectories, or clusters, from 6 to 57 months of age associated with a greater likelihood of SEN at 8 years of age?

METHODS

Population

The Avon Longitudinal Study of Parents and Children (ALSPAC) is a geographically based cohort study of children. ALSPAC enrolled ~85% of pregnant women (N = 14 541) residing in a defined section of southwest England with an expected date of delivery between April 1991 and December 1992. This study uses data from ALSPAC because it is the only known longitudinal, population-based cohort with measures of SDB, non–respiratory-related sleep problems, and school outcomes in early childhood.

The cohort, described in detail elsewhere,[20] was generally representative of the UK population. Our analyses, which excluded twins and triplet and quadruplet births, children who did not survive to 1 year, and children with conditions such as major congenital disorders that are likely to affect SDB or SEN yielded an initial base sample of 13 467 infants.

Ethical approval for the ALSPAC study was obtained from the ALSPAC Law and Ethics Committee and the local research ethics committees. All participants provided informed consent. This secondary data analysis was considered exempt from the lead investigator's committee on human subjects.

Assessment of SEN

"SEN identified" is the primary outcome variable. Comparable to the US categories for special education, SEN categories include speech, language, and communication needs; specific learning difficulty; and behavioral, emotional, and social difficulties. In 1999–2000, when this study's SEN data were obtained, ~17% of children in England had an SEN identified.[21] Most such children have their needs met in mainstream schools with an individualized education plan. We did not use the legal "statement of SEN," a much higher level of need, obtained for just ~3% of children in England who usually attend specialized schools, as this would exclude the ~14% of children generally served in mainstream schools.[22] For comparison, during this same period, ~13% of children were classified under the US Individuals with Disabilities Education Act as having a disability entitling them to special education.[23]

Assessment of SDB and Sleep Problems

SDB Symptoms

Parents reported on their child's snoring, mouth-breathing, and apnea at 6, 18, 30, 42, and 57 months of age in response to ALSPAC's mail questionnaires. ALSPAC's Likert scaled items are similar to items validated against polysomnography data; objective sleep evaluation measures were unavailable. Snoring was assessed with the question: "Does she snore for more than a few minutes at a time?" Mouth-breathing was assessed with the item: "Does she breathe through her mouth rather than her nose?" Witnessed apnea was assessed with the question: "When asleep, does she seem to stop breathing or hold breath for several seconds at a time?"

We derived a series of unique (ie, statistically distinct) patterns of these SDB symptoms across the 5 time points via a methodology reported elsewhere.[24] Clusters were derived for children with SDB measures at ≥2 of these 5 time points. Briefly, this process yielded 5 unique symptom patterns, or clusters (see Fig 1 A–E), depicting the prevalence of snoring, mouth-breathing, and apnea in SD or *z* scores, at 6, 18, 30, 42, and 57 months of age.

These 5 clusters classified children as (1) normals, asymptomatic throughout (37% of sample); (2) peak at 6 months, all 3 symptoms peak at 6 months but abate thereafter (19% of sample); (3) peak at 18 months, all 3 symptoms peak at 18 months but lessen thereafter (17% of sample); (4) worst, elevated symptom levels beginning at 18 months that remain high, with a 30-month peak (9% of sample); and (5) late symptom, modestly elevated symptoms first appear at 42 months (18% of sample).

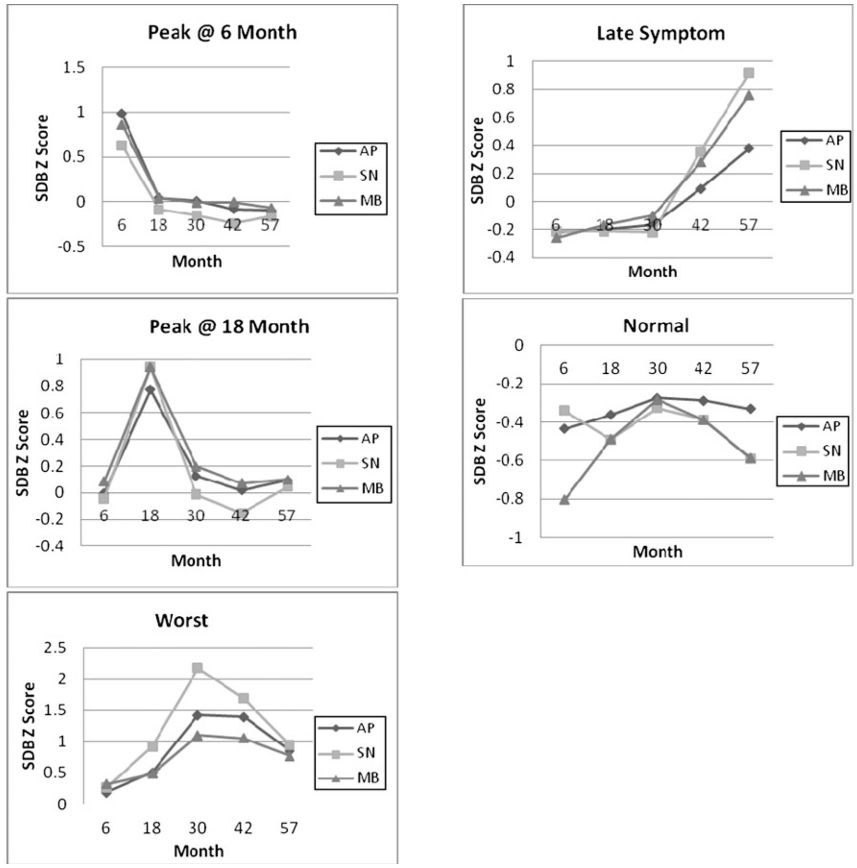

FIGURE 1
SDB clusters of combined apnea (AP), snoring (SN), and mouth-breathing (MB) in sample from 6 to 57 months of age.

BSPs

At the 18-, 30-, 42-, and 57-month questionnaires, parents were asked 7 items about their child's sleep. Most pertained to BSPs in the past year (except at 30 months when no recall period was given). Items included whether (yes/no) the child refused to go to bed, regularly woke early, regularly had difficulty sleeping, regularly had nightmares, regularly got up after being put to bed, regularly woke in the night, and regularly got up after a few hours. As in previous ALSPAC analyses,[25,26] we combined these into an index. We applied a cut-off score of ≥5 of these 7 items as a BSP, given that 15%, 27%, 24%, and 21% of the sample responded affirmatively to ≥5 of the 7 items at 18, 30, 42, and 57 months, respectively, consistent with previously published prevalence rates.[27,28] These items were, appropriately, not assessed at 6 months.

Descriptive Characteristics

The literature guided the selection of potential covariates and mediating variables. SDB is associated with multiple socioeconomic status (SES) variables, such as parental education and employment,[9,29,30] as well as maternal risk factors, such as maternal smoking,[9,30] race,[9,31] birth weight and gestational age,[32] and breastfeeding.[33,34] Optimal sleep hygiene reduces BSP risk[35] and is significantly related to race, SES, family structure, and household characteristics.[36] In several studies, child gender and race,[37,38] SES,[38,39] and parental education[40] moderated the effects of poor sleep on cognitive functioning and academic achievement. SDB is associated with reduced IQ, which may not resolve postadenotonsillectomy.[41]

Based on these associations, putative covariates included (1) maternal cigarette smoking: "ever" versus "never" before pregnancy; (2) ethnicity of child: white or nonwhite; (3) housing inadequacy: a composite variable for crowdedness (<1 room per person) and/or homelessness from birth to 4 years of age; (4) paternal social class: manual versus professional; (5) maternal education: low versus high, with "low" denoting the end of compulsory education, resulting in a school leaving certificate at 16 in the United Kingdom; (6) family adversity index: 18 stressor items (eg, maternal psychopathology, crime, financial insecurity) used in other ALSPAC analyses[42]; (7) Home Observation for the Measurement of the Environment (HOME): an inventory[43] of the quality of parenting and home environment; (8) birth weight and gestational age: low birth weight was defined as <2500 g and premature as <37 weeks' gestation; (9) breastfeeding: whether the child was ever breastfed; (10) adenoidectomy/tonsillectomy: questionnaire at 57 months asked if child ever had tonsils or adenoids removed (exact age at surgery was not assessed); and (11) IQ: per the Wechsler Intelligence Scale for Children, Third Edition (WISC-III), administered at ~8 years of age. Consistent with other ALSPAC work, an IQ <80 was denoted as low.[44]

Statistical Analyses

We used χ^2 and analysis of variance tests for categorical covariates and *t* tests for continuous covariates to describe differences between or among: children with missing versus nonmissing data for the sleep or SEN variables, children with versus without an SEN, the SDB clusters, and children with BSPs at 0, 1, 2, 3, or 4 time points. Logistic regression was used to examine unadjusted relationships between BSP and SEN and between SDB and SEN. For BSP, the odds ratios (OR) and 95% confidence intervals (CIs) represent the odds of SEN associated with each additional time period of having a BSP (range 0–4). For SDB, ORs (95% CIs) were derived both for each of the 4 symptomatic clusters versus normals, as well as for all 4 symptomatic clusters combined versus normals.

Initial multivariate logistic regression models included all putative covariates, but only significant ($P < .05$) covariates were retained in final models. Models were run including and excluding IQ, as well as with IQ as an interaction term with SDB in the SDB model, and with race, gender, maternal education, and paternal employment with BSP in the BSP model. In addition, BSP models analyzed race, gender, maternal education, and paternal employment as interaction terms, based on earlier work. To address multicollinearity, variance inflation factors were derived to assess the effects of individual independent variables on variance. A conservative variance inflation factor threshold of 10 was used in model testing.[45] Analyses were conducted by using SAS version 9.1 (SAS Institute Inc, Cary, NC).

RESULTS

Sample Size

Excluding children of multiple births, children who did not survive to 1 year, and children with conditions related to sleep disorders or SEN, there were 13 024 children with either BSP or SDB (exposure variables) or with the SEN outcome measure. Of these, 11 026 children had SEN outcomes data, 11 049 children were reflected in the SDB clusters (ie, SDB measures for ≥2 of 5 time points), and 11 467 had BSP data (ie, ≥1 BSP measure at 18, 30, 42, or 57 months of age).

Sample Characteristics and Association With SEN

Table 1 presents the characteristics of the analytic sample of 13 024 children. Compared with the initial base sample of 13 467, the 443 children missing either sleep exposure variable and/or the SEN outcome variable had more adverse SES and family risk characteristics but did not differ by gestational age, birth weight, gender, or IQ (not shown). Among the 11 026 children with SEN outcomes, 16.6% (1825)

TABLE 1 Sample Demographics and Special Education Need

	Base Samplea	SEN Among Total Sample	
		Yes	No
N	13 024	1825	9201
Maternal characteristics			
Smoked during pregnancy, any, %***	24.8	33.7	24.3
Alcohol during pregnancy, any, %	54.8	53.6	54.3
Age at delivery, mean y (SD)***	28 (4.94)	27.91 (4.84)	26.78 (5.02)
Breastfed this child, ever, %***	75.6	66.0	74.6
Child characteristics			
Gender, male, %***	51.5	68.7	47.5
Race, white, %	97.6	97.3	98.0
Premature, <37 wk, %***	4.9	6.6	4.7
Low birth weight, <2500 g, %***	4.2	5.9	4.0
Adenoids removed, ever, %***	7.4	11.6	6.7
Tonsils removed, ever, %*	4.5	6.0	4.2
IQ, <80, %***	12.5	41.5	10.1
IQ, mean (SD)***	104.33 (16.42)	89.97 (16.68)	104.83 (15.32)
Socioeconomic/family characteristics			
Maternal education, lower, %***	64.7	78.4	67.3
Paternal employment, manual, %***	44.1	59.8	46.2
Housing, inadequate, %***	12.4	18.1	11.7
Family adversity index, range 0–18, mean (SD)***	1.80 (1.98)	2.17 (2.22)	1.72 (1.93)
HOME score, range 0–8, mean (SD)***	5.74 (1.66)	5.58 (1.75)	5.75 (1.64)
Parity, ≥1, %***	53.7	62.7	54.9
Sleep disorders			
SDB, symptomatic cluster, %[b]**	63.1	71.5	61.6
BSP, reported at >1 time point, %**	44.3	48.4	43.5

HOME, Home Observation for the Measurement of the Environment.

[a] These 13 024 constitute the base sample used to derive the SDB clusters, sleep problems score, and SEN outcomes presented.

[b] In any of the 4 symptomatic clusters versus the 1 asymptomatic cluster.

* $P < .05$; ** $P < .01$; *** $P < .001$.

had an SEN. Children who did not have an SEN (n = 9201) differed from those with an SEN on nearly every characteristic (Table 1).

SDB Cluster Association With Sample Characteristics

There were significant differences among the clusters for 16 of the 17 putative covariates (Table 2). Children in the symptomatic clusters had the most adverse risk profile, led by those in the "Worst" cluster, and followed by those in the "Late Symptoms" cluster. In contrast to SEN associations with sample characteristics, the normals were significantly less likely to be premature or low birth weight or to have had mothers who reported smoking or drinking alcohol in pregnancy. There was a 4-point IQ difference between the "Worst" (mean 102.4, SD 16.3) and normal (mean 106.4, SD 16.1) clusters.

Cumulative Sleep Problem Association With Sample Characteristics

Cumulative report of BSPs differed by maternal risk factors as well as SES and family characteristics (Table 3). A higher proportion of children with BSPs had disadvantaged profiles; for most significant variables, this association appeared to be linear. There was nearly a 4-point IQ difference between children with BSPs at all 4 time points (mean 101.80, SD 15.61) versus children with no reported BSPs (mean 105.76, SD 16.28) In contrast, to SDB, neither gender, race, prematurity, nor low birth weight was associated with duration of BSP.

BSP Associations With SEN

Table 4 presents crude and adjusted effects of each additional time point report of a BSP. In crude analyses, each additional time point with a BSP was associated with a 12% increased odds of SEN (95% CI 1.06–1.18). As neither the IQ × BSP nor the child race × BSP, gender × BSP, maternal education × BSP, or paternal employment × BSP interaction terms were significant, these variables were entered as covariates into logistic regression models. In adjusted analyses without IQ, BSP remained significant (OR 1.07, 95% CI 1.01–1.15). In analyses adjusted for IQ, BSP nearly attained significance (OR 1.08, 95% CI 1.00–1.17), even when controlling for the strong, significant effect of IQ (OR 6.17, 95% CI 5.10–7.48).

SDB Associations With SEN

The combined symptomatic clusters (Table 5) were associated with a 56% increased odds of SEN in unadjusted analyses (95% CI 1.37–1.77); children in the "Worst" cluster had the highest increased odds: 83% (OR 1.83, 95% CI 1.48–2.25). In adjusted analyses without IQ, the combined symptomatic cluster effect attenuated to 38% (95% CI 1.18–1.62). The "Worst" cluster continued to have the strongest effect: 60% (95% CI 1.23–2.08), while other cluster effects ranged from 30% to 40%. Adjusting for IQ only slightly attenuated the combined symptomatic cluster effect to 30% (95% CI 1.05–1.61), but the "Peak at 18" and "Late" cluster effects no longer reached significance. For streamlining purposes, significant covariates are not shown (table legend identifies which were significant).

TABLE 2 SDB Cluster Associations With Sample Characteristics

	Peak at 6 mo	Peak at 18 mo	Worst	Late	Normal
N	2142	1830	934	2005	4138
Maternal					
Smoked during pregnancy, any, %***	24.6	26.6	30.0	23.8	17.5
Alcohol during pregnancy, any, %*	53.3	54.3	57.4	54.0	56.8
Age at delivery, mean y (SD)***	28.28 (4.73)	27.98 (4.90)	27.73 (5.03)	28.11 (4.79)	29.04 (4.64)
Breastfed this child, ever, %***	75.6	74.8	70.4	75.1	79.3
Child					
Gender, male, %***	54.0	53.5	55.9	49.6	49.2
Race, white, %**	98.2	96.7	97.7	98.1	98.2
Premature, <37 wk, %*	4.5	4.5	7.0	4.8	4.3
Low birth weight, <2500 g, %**	3.3	4.9	5.2	4.1	3.3
Adenoids removed, ever, %***	4.6	6.3	31.7	9.8	3.1
Tonsils removed, ever, %***	2.7	3.3	20.7	6.0	2.0
IQ, <80, %***	11.4	14.0	15.6	14.8	9.6
IQ, mean (SD)***	104.32 (16.14)	103.3 (17.05)	102.4 (16.25)	102.7 (16.28)	106.41 (16.07)
Socioeconomic and family					
Maternal education, lower, %***	65.3	66.0	68.3	66.4	56.9
Paternal employment, manual, %***	43.7	46.4	47.2	44.4	37.7
Housing, inadequate, %***	15.3	14.8	16.2	13.3	9.12
Family adversity index, range of 0–18, mean (SD)***	2.15 (2.11)	2.20 (2.09)	2.37 (2.19)	1.93 (2.00)	1.57 (1.81)
HOME score, range of 0–8, mean (SD)	5.7 (1.67)	5.73 (1.64)	5.70 (1.67)	5.78 (1.64)	5.79 (1.65)
Parity, ≥1, %**	54.7	54.1	54.1	51.6	56.6

HOME, Home Observation for the Measurement of the Environment.
* $P < .05$; ** $P < .01$; *** $P < .001$.

DISCUSSION

This is the first population-based study of the association between respiratory-related (SDB) and behavioral (BSP) sleep

TABLE 3 BSP Association With Sample Characteristics, by Number of Time Points With Reported BSPs

	No. of Time Points With a BSP				
	0	1	2	3	4
N	6522	2527	1366	780	272
Maternal characteristics					
Smoked during pregnancy, any, %***	19.9	25.7	27.8	31.0	30.7
Alcohol during pregnancy, any, %*	54.3	54.6	58.5	58.4	53.2
Age at delivery, mean y (SD)***	28.61 (4.78)	28.09 (4.84)	27.94 (5.05)	27.6 (4.89)	27.96 (4.72)
Breastfed this child, ever, %	77.2	76.6	74.7	73.4	73.8
Child characteristics					
Gender, male, %	50.8	53.5	51.0	52.4	50
Race, white, %	98.0	97.7	97.5	97.1	98.5
Premature, <37 wk, %	4.6	4.3	6.2	5	3.3
Low birth weight, <2500 g, %*	3.6	3.9	4.7	5.7	3.7
Adenoids removed, ever, %**	6.7	7.2	9.6	9.9	7.0
Tonsils removed, ever, %	4.0	4.7	5.3	6.3	4.3
IQ, <80, %***	10.6	13.2	14.6	18.1	15.1
IQ (mean, SD)***	105.76 (16.28)	103.33 (16.54)	102.67 (16.27)	100.99 (16.35)	101.80 (15.61)
Socioeconomic/family characteristics					
Maternal education, lower, %***	60.1	63.8	65.1	71.9	75
Paternal employment, manual, %***	39.9	45.0	45.5	52.0	48.0
Housing, inadequate, %***	10.5	14.3	15.3	18.2	20.6
Family adversity index, range 0–18, mean (SD)***	1.64 (1.81)	2.03 (2.08)	2.36 (2.15)	2.64 (2.39)	2.87 (2.27)
HOME score, mean 0–8, mean (SD)	5.76 (1.64)	5.77 (1.66)	5.75 (1.65)	5.69 (1.68)	5.71 (1.79)
Parity, ≥1, %***	57.5	53.5	49.9	46.8	48.9

HOME, Home Observation for the Measurement of the Environment.
* $P < .05$; ** $P < .01$; *** $P < .001$.

TABLE 4 BSP Odds of SEN Associated With Each Time Point of Reported BSP

Crude	OR (95%CI)
Crude	
Cumulative sleep problem score	1.12 (1.06–1.18)
Adjusted, without IQ[a]	
Cumulative sleep problem score*	1.07 (1.01–1.15)
Maternal age at delivery, higher vs lower	0.97 (0.95–0.98)
Child gender, male vs female	2.63 (2.26–3.06)
Child birth weight, low vs high	1.65 (1.16–2.34)
Paternal employment, manual vs professional	1.42 (1.22–1.65)
Family adversity index	1.10 (1.07–1.14)
Parity	1.29 (1.09–1.53)
1 vs 0	
≥2 vs 0	1.74 (1.42–2.14)
Adjusted, with IQ[a]	
Cumulative sleep problem score	1.08 (1.00–1.17)*
Child gender, male vs female	2.41 (2.00–2.90)
Family adversity index, increased	1.10 (1.06–1.15)
IQ <80	6.17 (5.10–7.48)

[a] Only significant covariate effects shown.
* $P < .062$.

problems, throughout early childhood, and SEN. Children with a history of BSPs and of SDB in the first 5 years of life were more likely to have an SEN at 8 years of age; even controlling for 16 putative confounders, BSPs were associated with a 7% increased odds of SEN, for each ~1-year interval. Thus, for example, children with a BSP in at least 2 of the 4 intervals (~1 of 5 children) had a 15% increased likelihood of SEN. SDB, overall, was associated with a near 40% increased odds of SEN. Children with the worst SDB symptoms were 60% more likely to have an SEN. Sleep problem effects remained significant, even after controlling for IQ, which itself was associated with five- to sixfold increased odds of SEN for both BSPs and SDB. Residual confounding is possible. Specifically, children with underlying neurodevelopmental issues, who are more likely to have SENs, may indeed have more sleep problems. However, given the persistence of sleep problem effects in analyses controlling for multiple putative confounders including IQ, such confounding is unlikely to negate our findings.

Our study differs from the few publications on sleep problems and SEN, in several ways. First, earlier work compared children from specialized versus mainstream schools.[18,19,46] In contrast, we use a population-based approach. Correspondingly, we applied the inclusive SEN designation, rather than the narrower "statement of SEN," most applicable to children in specialized schools. Second, in contrast to earlier work, our longitudinal cohort study's strengths include large sample size, control for multiple potential confounders, and ability to examine temporal relationships and estimate effects. Previous cross-sectional work analyzed data from ~100 children or fewer from specialized schools, with limited control for confounders. Third, our work assesses SDB and BSP from infancy through 5 years of age, the peak period

TABLE 5 SDB Clusters Effects on SEN

	Crude OR (95% CI)	Adjusted,[a] Without IQ, OR (95% CI)	Adjusted,[b] With IQ, OR (95% CI)
Combined symptomatic vs normal	1.56 (1.37–1.77)***	1.38 (1.18–1.62)***	1.30 (1.05–1.61)*
Peak at 6 mo	1.52 (1.28–1.79)***	1.32 (1.08–1.62)**	1.40 (1.09–1.80)**
Peak at 18 mo	1.47 (1.24–1.75)***	1.31 (1.06–1.63)**	1.14 (0.87–1.50)
Worst	1.83 (1.48–2.25)***	1.60 (1.23–2.08)***	1.45 (1.05–2.00)*
Late	1.56 (1.32–1.85)***	1.43 (1.16–1.75)***	1.26 (0.98–1.63)

* $P < .05$; ** $P < .01$; *** $P < .001$.

[a] In adjusted models without IQ (based on symptomatic versus normal SDB clusters), the following variables were significant: maternal age at delivery, child gender (male), low birth weight, paternal employment (manual), family adversity (increased), and parity (≥2).

[b] In adjusted models with IQ (based on symptomatic versus normal SDB clusters), the following variables were significant: child gender (male), family adversity (increased), and IQ (<80).

for both disorders,[27,47] enabling us to predict cumulative effects during this vulnerable period on later SEN.

Several population-based studies have examined cognitive and academic outcomes (ie, not special education) with mixed results. In 1 study, school-aged children with objectively measured mild SDB did no poorer on most intelligence measures assessed,[48] in contrast to most research linking SDB to poorer academic performance,[12,14] while in another, objectively measured SDB was significantly associated with cognitive outcomes.[49] Regarding BSPs, our findings differ from an Australian study finding no concurrent or longitudinal association between "sleep problems" and cognitive outcomes measured at 4 to 5 and 6 to 7 years of age.[50] In that study, "sleep problems" was defined by just 1 item (ie, whether the parent considered the child to have a sleep problem [none, mild, moderate, severe]), in contrast to our scale-based measure of 7 specific sleep behaviors.

Regarding school outcomes of interventions, adenotonsillectomy to treat SDB is associated with improved neurocognition among 4-year-olds[51,52] and school performance among the lowest performing first graders.[53] Limited data are available on school-related outcomes of BSP interventions, despite their known efficacy among young children.[54–57] The 1 published randomized controlled trial did not find improved learning outcomes among 5- to 6-year-olds identified via school screening. Authors posit that the brief instrument that was used may have been insufficiently sensitive to detect changes in skills (eg, working memory) that affect school outcomes or that learning effects might lag beyond the study's 6-month follow-up.[58]

This study has several limitations. First, neither sleep problem was assessed with validated pediatric sleep questionnaires,[18,19,46] in part, because none were available at the time.[59] Still, the SDB items are similar to those validated against objective measures,[60–64] while the clusters themselves may better capture the dynamic, multisymptom expression of SDB.[24] Our BSP measure corresponds to one used in earlier ALSPAC analyses.[25,26] Second, ALSPAC data did not specify the disabilities that qualified a child for SENs. At the time, the bulk of classifications were for learning versus socioemotional or physical disorders.[21] In contrast, "behavioral, emotional, and social difficulty," "speech, language, and communication difficulty," and "autism spectrum disorder" are now more prevalent among those with SENs, compared with when our study's SEN outcomes data were collected.[65] Given overlap between the functional effects of these disabilities and sleep disorders,[66] our effect sizes may actually be underestimates based on present SEN classifications.

Findings presented here strongly support an association between early childhood sleep problems and later SEN, on a population basis. This highlights the need for early screening, because early treatment is often effective for SDB[67] and BSPs.[56] The magnitude of potential benefit from early screening and treatment is greatest for young children with behavioral, cognitive, and language delays/disabilities, because sleep disorders affect functioning in these areas. Currently, US-based early intervention programs do not systematically screen for sleep disorders,[68] which are underdiagnosed in routine pediatric care.[69,70] Future research should focus on timely and systematic screening and on testing potential interventions, particularly for BSPs, among young children at risk for developmental delay/disability.

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