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Assessment of neighborhood-level disadvantage and pediatric obstructive sleep apnea severity

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Abstract

Objectives: To examine the relationship between neighborhood-level advantage and severe obstructive sleep apnea (OSA) in children.

Methods: A retrospective case-control study was conducted on 249 children who underwent adenotonsillectomy and had full-night polysomnography conducted within 6 months prior. Patients were divided into more or less socioeconomically disadvantaged groups using a validated measure, the area deprivation index (ADI). The primary outcomes were the relationship between the apnea-hypopnea index (AHI) and the presence of severe OSA, and the secondary outcome was residual moderate or greater OSA after tonsillectomy.

Results: Of the 249 children included in the study, 175 (70.3%) were socially disadvantaged (ADI > 50). The median (interquartile range [IQR]) age was 9.4 (7.3–12.3) years, 129 (51.8%) were male, and the majority were White (151, 60.9%), Black (51, 20.6%), and/or of Hispanic (155, 62.5%) ethnicity. A total of 140 (56.2%) children were obese. The median (IQR) AHI was 8.9 (3.9–20.2). There was no significant difference in the median AHI or the presence of severe OSA between the more and less disadvantaged groups. Severe OSA was found to be associated with obesity (odds ratio [OR] = 3.13, 95% confidence interval [CI] = 1.83–5.34), and residual moderate or greater OSA was associated with older age (OR = 1.20, 95% CI = 1.05–1.38).

Conclusions: The ADI was not significantly associated with severe OSA or residual OSA in this cohort of children. Although more neighborhood-level disadvantage may increase the risk of comorbidities associated with OSA, it was not an independent risk factor in this study.

Level of Evidence: Level 4.

KEYWORDS

area deprivation index, neighborhood-level disadvantage, pediatric obstructive sleep apnea, socioeconomic status

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1 | INTRODUCTION

Obstructive sleep apnea (OSA) is a sleep disorder characterized by recurrent upper airway obstruction that disrupts ventilation and sleep.¹ It is diagnosed using level 1 or 2 polysomnography (PSG). In children, untreated OSA can have various negative consequences, including behavioral problems, cognitive deficits, excessive daytime sleepiness, impaired academic performance, and long-term effects on cardiovascular and endocrine systems.^{2,3} Despite several studies examining the relationship between socioeconomic status (SES) and OSA in children, the evidence is inconclusive. Some studies have identified certain socioeconomic factors associated with increased OSA severity, such as race, maternal education, and family income.^{4–7} In contrast, others have found that these associations are not statistically significant when controlling for confounders or using other measures of SES.^{5,8}

The area deprivation index (ADI) is a metric of neighborhood-level disadvantage that combines 17 social determinants of health, including education, employment, housing, and poverty measures, as reported in the American Community Survey.^{9,10} It offers a comprehensive index that incorporates multiple facets of SES. The ADI has been used in other studies of pediatric health disparities.¹¹⁻¹⁴ However, to our knowledge, it has yet to be used to investigate the relationship between neighborhood-level deprivation and OSA severity or outcomes following adenotonsillectomy (T&A) as a treatment for OSA in children.

The primary objective of this study is to use the ADI to examine the relationship between neighborhood-level deprivation and OSA severity as measured by PSG. The secondary objective is to examine the level of residual moderate or greater OSA (defined as an apneahypopnea index [AHI] > 5) present following T&A. We hypothesize that a significant relationship exists between socioeconomic disadvantage, as measured by the ADI, and the severity of OSA in children.

2 | METHODS

The study was a retrospective case-control study conducted at the Children's Medical Center of Dallas/University of Texas Southwestern Medical Center between December 2018 and December 2019. The study included patients 18 years or younger who underwent T&A and excluded those who did not complete PSG within 6 months before surgery, previously had a tonsillectomy or had a tracheostomy. The study was approved by the UT Southwestern Institutional Review Board and was exempt from consent due to its retrospective design.

Demographic data collected included age, weight, height, BMI, sex, race/ethnicity, language, insurance type, and ADI. ADI was divided into more (ADI ≤ 50) or less disadvantaged (ADI > 50) categories in accordance with previous validation studies. Comorbidities collected included birth status, allergic rhinitis, gastroesophageal reflux disease (GERD), asthma, Down syndrome, airway support, and developmental delay/impaired cognitive status. Tonsillar hypertrophy was also recorded, with grades 3 and 4 on the Brodsky grading scale defined as the presence of tonsillar hypertrophy and grades 1 and 2 as an absence.

Variables collected from PSG included AHI, obstructive AHI, central apnea index, percentage of time in rapid eye movement (REM) and non-rapid eye movement (NREM) sleep, sleep efficiency, arousal index, oxygen saturation nadir, peak end-tidal carbon dioxide (CO₂), time spent with end-tidal CO₂ level >50 mmHg, and time spent with oxygen saturation under 90%. OSA severity was classified based on AHI: mild (1.0–4.0), moderate (5.0–9.9), and severe (>10). Severe OSA was further classified as very severe if AHI was >30. Residual moderate or greater OSA was defined as AHI >5, as patients with this level of residual OSA following T&A are referred for positive airway pressure (PAP) therapy in the home institution.

Bivariate analysis of more or less disadvantaged groups was compared using the Kruskal–Wallis rank test for continuous and Pearson's chi-squared test for categorical variables. We performed a simple logistic regression model of severe and residual OSA risk and neighborhood disadvantage as a binary variable. Furthermore, we performed a multiple logistic regression model to assess predictors of severe and residual OSA. We used a purposeful selection of variables to include in the model. Variables with a $p \le .25$ in the bivariate models of demographics and OSA risk (severe and residual) were included. Variables with a p > .05 were dropped until the final model was formed. Final models were validated using jackknife regression, and their goodness of fit was assessed using the Hosmer–Lemeshow goodness-of-fit test.

All statistical analyses were performed using Stata Statistical Software, Version 17, and statistical significance was set at p < .05. This study adhered to the STROBE reporting guidelines.

3 | RESULTS

The present study included 249 children aged 18 years or younger who underwent PSG before T&A at the Children's Medical Center of Dallas/University of Texas Southwestern Medical Center between December 2018 and December 2019. The demographic and clinical characteristics of the study population are summarized in Table 1.

The median (interquartile range [IQR]) age at sleep study was 9.4 years (7.3–12.3), and the study population consisted of 129 males (51.8%) and was predominantly White (151, 60.9%), Black (51, 20.6%), and/or of Hispanic ethnicity (155, 62.5%). The most common insurance type was public (83.5%), followed by private (14.9%) and other (1.6%). Of the study population, 175 (70.3%) were classified as having neighborhood-level disadvantages (ADI national rank >50). The median (IQR) AHI from PSG was 8.9 (3.9–20.2). The median (IQR) body mass index (BMI) *z*-score was 24.0 (18.0–29.5), and 140 (56.2%) patients were obese (i.e., BMI ≥95th percentile). Most had tonsillar hypertrophy of grade 3 or 4 (200, 80.3%). Comorbidities included a history of preterm birth (11.4%), allergic rhinitis (47.4%), GERD (7.2%), asthma (30.1%), Down syndrome (6.0%), developmental delay or impaired cognitive status (13.3%), and history of PAP therapy (1.6%).

 TABLE 1
 Demographic and clinical characteristics of children with obstructive sleep apnea (OSA) based on social disadvantage.

	Disadvantaged			
	No 74 (29.7%)	Yes 175 (70.3%)	Total 249 (100.0%)	p Value
Age	9.4 (7.0-12.3)	9.3 (7.4-12.3)	9.4 (7.3-12.3)	.738
Weight	43.3 (30.0-55.9)	44.4 (29.7-68.6)	44.0 (29.9-65.0)	.413
Height	134 (121-149)	139 (124–152)	139 (124-151)	.560
BMI	22.1 (18.1-27.7)	24.2 (17.9-30.4)	24.0 (18.0-29.5)	.470
BMI percentile	95.8 (76.5-99.1)	97.5 (78.4-99.1)	97.0 (77.4-99.1)	.728
BMI z-score	1.7 (0.7–2.4)	2.0 (0.8-2.4)	1.9 (0.8–2.4)	.696
Obese				
No	36 (48.6%)	73 (41.7%)	109 (43.8%)	.313
Yes	38 (51.4%)	102 (58.3%)	140 (56.2%)	
Gender				
Male	35 (47.3%)	94 (53.7%)	129 (51.8%)	.354
Female	39 (52.7%)	81 (46.3%)	120 (48.2%)	
Ethnicity				
Non-Hispanic	36 (49.3%)	57 (32.6%)	93 (37.5%)	.013
Hispanic	37 (50.7%)	118 (67.4%)	155 (62.5%)	
Race				
Asian	1 (1.4%)	1 (0.6%)	2 (0.8%)	.443
Black	11 (15.1%)	40 (22.9%)	51 (20.6%)	
White	49 (67.1%)	102 (58.3%)	151 (60.9%)	
Other	12 (16.4%)	32 (18.3%)	44 (17.7%)	
Language				
English	61 (82.4%)	117 (66.9%)	178 (71.5%)	.042
Spanish	13 (17.6%)	57 (32.6%)	70 (28.1%)	
Other	0 (0.0%)	1 (0.6%)	1 (0.4%)	
ADI national rank	34.5 (25.0-44.0)	76.0 (64.0-87.0)	66.0 (47.0-82.0)	<.001
Payer				
Private	19 (25.7%)	18 (10.3%)	37 (14.9%)	.004
Public	53 (71.6%)	155 (88.6%)	208 (83.5%)	
Other	2 (2.7%)	2 (1.1%)	4 (1.6%)	
Allergic rhinitis				
No	40 (54.1%)	91 (52.0%)	131 (52.6%)	.767
Yes	34 (45.9%)	84 (48.0%)	118 (47.4%)	
GERD				
No	66 (89.2%)	165 (94.3%)	231 (92.8%)	.156
Yes	8 (10.8%)	10 (5.7%)	18 (7.2%)	
Asthma				
No	59 (79.7%)	115 (65.7%)	174 (69.9%)	.028
Yes	15 (20.3%)	60 (34.3%)	75 (30.1%)	
Down syndrome				
No	70 (94.6%)	164 (93.7%)	234 (94.0%)	.790
Yes	4 (5.4%)	11 (6.3%)	15 (6.0%)	
Developmental delay/impaired cognitive status				
No	63 (85.1%)	153 (87.4%)	216 (86.7%)	.626
Yes	11 (14.9%)	22 (12.6%)	33 (13.3%)	

TABLE 1 (Continued)

	Disadvantaged			
	No 74 (29.7%)	Yes 175 (70.3%)	Total 249 (100.0%)	p Value
Tonsillar hypertrophy				
No	12 (16.2%)	37 (21.1%)	49 (19.7%)	.372
Yes	62 (83.8%)	138 (78.9%)	200 (80.3%)	
Preterm				
No	62 (87.3%)	148 (89.2%)	210 (88.6%)	.684
Yes	9 (12.7%)	18 (10.8%)	27 (11.4%)	
PAP therapy				
No	73 (98.6%)	172 (98.3%)	245 (98.4%)	.835
Yes	1 (1.4%)	3 (1.7%)	4 (1.6%)	

Note: p Value based on Kruskal–Wallis rank test for continuous variables, and Pearson χ^2 test for categorical variables.

Abbreviations: ADI, area deprivation index; BMI, body mass index adjusted for age and sex; GERD, gastroesophageal reflux disease; PAP, positive airway pressure; Tonsillar hypertrophy, Brodsky 3+ and 4+.

TABLE 2	Polysomnographic (PSG	parameters in children with	obstructive sleep apr	nea (OSA) based o	n social disadvantage.

	Disadvantaged			
	No 74 (29.7%)	Yes 175 (70.3%)	Total 249 (100.0%)	p Value
REM	72.5 (53.0-91.0)	76.0 (53.0-97.0)	75.0 (53.0-94.0)	.370
Sleep efficiency	82.6 (74.7-89.5)	81.4 (73.6-88.4)	81.4 (73.8-88.4)	.472
Arousal index	13.9 (8.9–18.3)	13.5 (9.5–19.3)	13.5 (9.4–19.1)	.765
Respiratory distress index	8.2 (3.9-24.4)	9.1 (4.0–19.8)	8.9 (4.0-20.3)	.788
Obstructive apnea-hypopnea index	7.7 (3.4-22.9)	8.7 (3.8-19.6)	8.6 (3.8-20.2)	.681
Central apnea index	0.0 (0.0–0.5)	0.1 (0.0-0.4)	0.1 (0.0-0.4)	.516
Apnea-hypopnea index	7.2 (3.6-22.9)	9.1 (4.1–19.8)	8.9 (3.9-20.2)	.461
Low SaO ₂ nadir	88.5 (83.0-92.0)	89.0 (84.0-92.0)	89.0 (84.0-92.0)	.389
Peak CO ₂	52.0 (48.0-54.0)	51.0 (48.0-54.0)	51.0 (48.0-54.0)	.876
TST 50 CO ₂	0.9 (0.0-8.9)	0.5 (0.0-6.0)	0.5 (0.0-7.4)	.627
TST <90%	0.1 (0.0-0.9)	0.1 (0.0-0.6)	0.1 (0.0-0.6)	.959
Severe OSA				.579
No	43 (58.1%)	95 (54.3%)	138 (55.4%)	
Yes	31 (41.9%)	80 (45.7%)	111 (44.6%)	

Note: Continuous variable are median with interquartile ranges. p Value based on Kruskal-Wallis.

Abbreviations: AHI, apnea-hypopnea index; CAI, central apnea index; low SaO_2 nadir, lowest pulse oximetry measured hemoglobin saturation; No social disadvantage, ADI national rank <50; NREM, non-rapid eye movement sleep time (min); OAHI, obstructive apnea-hypopnea index; REM, rapid eye movement sleep time (min); Sleep efficiency, percentage of time the patient was asleep; Social disadvantage, ADI national rank >50; TST <90% O₂, percentage of total sleep time spent with oxygen saturation <90%; TST 50 >CO₂, total sleep time spent at greater than 50 mmHg blood CO₂ saturation.

Table 1 also compares the demographic and clinical characteristics of the study population between those who had more disadvantages (ADI national rank > 50) and less disadvantages (ADI \leq 50). The neighborhood-level disadvantage was significantly associated with Hispanic ethnicity (p = .013), public insurance (p = .004), and asthma (p = .028). Comparisons of polysomnographic parameters in children with more and less neighborhood-level disadvantages are shown in Table 2. The median (IQR) AHI was 8.9 (3.9–20.2). There was no significant association between neighborhood-level disadvantage and BMI *z*-score (p = .69), as well as any PSG parameter, including AHI, NREM, and REM sleep, sleep efficiency, oxygen saturation nadir, and peak end-tidal carbon dioxide (CO₂) level.

Table 3 compares the characteristics of patients with mild to moderate OSA and severe OSA. Tables 4 and 5 present the characteristics and PSG results of the subset of patients who underwent repeat PSG, divided into those with residual moderate or greater OSA (defined as AHI \geq 5) and those without. Severe OSA was significantly associated with obesity (p < .001). Severe, very severe, and residual

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	Severe OSA			
	No 138 (55.4%)	Yes 111 (44.6%)	Total 249 (100.0%)	p Value
Age	9.4 (7.4–11.6)	9.2 (7.3–13.1)	9.4 (7.3-12.3)	.526
Weight	40.9 (27.7–57.7)	50.5 (33.0–78.3)	44.0 (29.9–65.0)	.007
BMI	20.8 (17.4-27.1)	25.9 (19.5–31.5)	24.0 (18.0-29.5)	<.001
BMI percentile	93.1 (67.1-98.9)	98.3 (85.1-99.3)	97.0 (77.4-99.1)	<.001
Gender				
Male	69 (50.0%)	60 (54.1%)	129 (51.8%)	.525
Female	69 (50.0%)	51 (45.9%)	120 (48.2%)	
Ethnicity				
Non-Hispanic	54 (39.4%)	39 (35.1%)	93 (37.5%)	.489
Hispanic	83 (60.6%)	72 (64.9%)	155 (62.5%)	
Race				
Asian	1 (0.7%)	1 (0.9%)	2 (0.8%)	.212
Black	30 (21.9%)	21 (18.9%)	51 (20.6%)	
White	88 (64.2%)	63 (56.8%)	151 (60.9%)	
Other	18 (13.1%)	26 (23.4%)	44 (17.7%)	
Language				
English	107 (77.5%)	71 (64.0%)	178 (71.5%)	.042
Spanish	31 (22.5%)	39 (35.1%)	70 (28.1%)	
Other	0 (0.0%)	1 (0.9%)	1 (0.4%)	
ADI national rank	64.0 (44.0-82.0)	68.0 (48.0-82.0)	66.0 (47.0-82.0)	.505
Allergic rhinitis				
No	68 (49.3%)	63 (56.8%)	131 (52.6%)	.240
Yes	70 (50.7%)	48 (43.2%)	118 (47.4%)	
GERD				
No	127 (92.0%)	104 (93.7%)	231 (92.8%)	.614
Yes	11 (8.0%)	7 (6.3%)	18 (7.2%)	
Asthma				
No	99 (71.7%)	75 (67.6%)	174 (69.9%)	.476
Yes	39 (28.3%)	36 (32.4%)	75 (30.1%)	
Down syndrome			(,	
No	129 (93.5%)	105 (94.6%)	234 (94.0%)	.713
Yes	9 (6.5%)	6 (5.4%)	15 (6.0%)	
Developmental delay	. (,	- (,	(,	
No	119 (86,2%)	97 (87,4%)	216 (86.7%)	789
Yes	19 (13.8%)	14 (12.6%)	33 (13.3%)	
Tonsillar hypertrophy		_ (,	()	
No	24 (17,4%)	25 (22,5%)	49 (19.7%)	.311
Yes	114 (82.6%)	86 (77 5%)	200 (80.3%)	.011
PAP therapy	111(02.070)	00 (, , .0,0)	_00 (00.070)	
No	137 (99 3%)	108 (97 3%)	245 (98.4%)	217
Ves	1 (0 7%)	3 (2 7%)	A (1 6%)	.21/
105	1 (0.7%)	3 (2.770)	4 (1.0%)	

TABLE 3 Demographic and clinical characteristics of children with obstructive sleep apnea (OSA) divided into those with mild to moderate OSA and severe OSA.

Note: Continuous variable are median with interquartile ranges. *p* Value based on Kruskal–Wallis rank test for continuous variables, and Pearson χ^2 test for categorical variables.

Abbreviations: ADI, area deprivation index; BMI, body mass index adjusted for age and sex; GERD, gastroesophageal reflux disease; PAP, positive airway pressure; Tonsillar hypertrophy, Brodsky 3+ and 4+.

TABLE 4	Demographic and clinical characteristics of children who had residual obstructive sleep apnea (OSA) following
adenotonsille	ectomy (T&A).

	Residual OSA			
	No 61 (70.9%)	Yes 25 (29.1%)	Total 86 (100.0%)	Test
Age	9.0 (7.3-12.3)	11.9 (8.9–15.2)	9.3 (7.4–13.0)	0.009
Weight	44.0 (30.0-68.6)	64.1 (36.4-88.6)	46.1 (30.0-78.3)	0.064
BMI	24.4 (18.1-30.7)	27.2 (21.4-34.6)	25.9 (18.3-31.4)	0.146
BMI percentile	97.7 (82.6–99.2)	98.5 (93.3-99.5)	98.0 (86.0-99.3)	0.446
Gender				0.982
Male	34 (55.7%)	14 (56.0%)	48 (55.8%)	
Female	27 (44.3%)	11 (44.0%)	38 (44.2%)	
Ethnicity				0.732
Non-Hispanic	22 (36.1%)	10 (40.0%)	32 (37.2%)	
Hispanic	39 (63.9%)	15 (60.0%)	54 (62.8%)	
Race				0.455
Asian	0 (0.0%)	1 (4.0%)	1 (1.2%)	
Black	14 (23.0%)	5 (20.0%)	19 (22.1%)	
White	34 (55.7%)	13 (52.0%)	47 (54.7%)	
Other	13 (21.3%)	6 (24.0%)	19 (22.1%)	
Language				0.522
English	37 (60.7%)	17 (68.0%)	54 (62.8%)	
Spanish	24 (39.3%)	8 (32.0%)	32 (37.2%)	
ADI national rank	72.0 (54.0-85.0)	66.0 (48.0-85.0)	70.0 (51.0-85.0)	0.765
Allergic rhinitis				0.362
No	30 (49.2%)	15 (60.0%)	45 (52.3%)	
Yes	31 (50.8%)	10 (40.0%)	41 (47.7%)	
GERD				0.278
No	54 (88.5%)	24 (96.0%)	78 (90.7%)	
Yes	7 (11.5%)	1 (4.0%)	8 (9.3%)	
Asthma				0.185
No	41 (67.2%)	13 (52.0%)	54 (62.8%)	
Yes	20 (32.8%)	12 (48.0%)	32 (37.2%)	
Down syndrome				0.402
No	57 (93.4%)	22 (88.0%)	79 (91.9%)	
Yes	4 (6.6%)	3 (12.0%)	7 (8.1%)	
Developmental delay/impaired cognitive status				0.016
No	53 (86.9%)	16 (64.0%)	69 (80.2%)	
Yes	8 (13.1%)	9 (36.0%)	17 (19.8%)	
Tonsillar hypertrophy				0.410
No	10 (16.4%)	6 (24.0%)	16 (18.6%)	
Yes	51 (83.6%)	19 (76.0%)	70 (81.4%)	
Disadvantaged				0.295
No	13 (21.3%)	8 (32.0%)	21 (24.4%)	
Yes	48 (78.7%)	17 (68.0%)	65 (75.6%)	

Note: Continuous variable are median with interquartile ranges. p Value based on Kruskal–Wallis rank test for continuous variables, and Pearson χ^2 test for categorical variables.

Abbreviations: ADI, area deprivation index; BMI, body mass index adjusted for age and sex; GERD, gastroesophageal reflux disease; PAP, positive airway pressure; Tonsillar hypertrophy, Brodsky 3+ and 4+.

TABLE 5 Polysomnographic (PSG) parameters at the initial sleep study in children with obstructive sleep apnea (OSA) divided into those with and without residual OSA.

	Residual OSA			
	No 61 (70.9%)	Yes 25 (29.1%)	Total 86 (100.0%)	p Value
REM	73.0 (55.0-94.0)	79.0 (29.0-91.0)	75.5 (50.0-94.0)	.648
Sleep efficiency	81.2 (75.7-87.6)	83.4 (71.2-88.6)	81.4 (75.4-87.6)	.909
Arousal index	15.4 (12.2-21.7)	18.3 (12.7-32.4)	16.1 (12.5–23.2)	.164
Respiratory distress index	15.9 (10.1–27.9)	29.6 (19.9-56.3)	19.8 (10.4–33.6)	.006
Obstructive apnea-hypopnea index	15.9 (9.8–25.6)	29.6 (12.8-55.4)	19.1 (9.8-33.0)	.020
Central apnea index	0.1 (0.0-0.4)	0.3 (0.0-1.2)	0.1 (0.0-0.6)	.155
Apnea-hypopnea index	15.9 (10.1-27.9)	28.1 (12.9-56.3)	19.6 (10.1-32.8)	.025
Low SaO ₂ nadir	88.0 (81.0-90.0)	79.0 (74.0-87.0)	86.0 (78.0-90.0)	.028
Peak CO ₂	52.0 (48.0-55.0)	51.0 (50.0-55.0)	52.0 (49.0-55.0)	.361
TST 50 CO ₂	1.1 (0.0-8.3)	2.7 (0.1-16.2)	1.3 (0.0–10.2)	.172
TST <90%	0.2 (0.0-1.4)	1.5 (0.4–3.6)	0.4 (0.0–2.0)	.009

Note: Continuous variable are median with interquartile ranges. p Value based on Kruskal-Wallis.

Abbreviations: AHI, apnea-hypopnea index; CAI, central apnea index; low SaO2 nadir, lowest pulse oximetry measured hemoglobin saturation; NREM, non-rapid eye movement sleep time (min); OAHI, obstructive apnea-hypopnea index; REM, rapid eye movement sleep time (min); sleep efficiency, percentage of time the patient was asleep; TST <90% O_2 , percentage of total sleep time spent with oxygen saturation <90%; TST 50 >CO₂, total sleep time spent at greater than 50 mmHg blood CO₂ saturation.

Variable	Category	Odds ratio	p Value	95% Confidence interval
Obese				
No	(base)	1		
Yes		3.13	<.001	1.83-5.34
Language				
English	(base)	1		
Spanish		1.96	.028	1.07-3.57
_cons		0.34	<.001	0.22-0.53

TABLE 6 Multiple logistic regression of odds of severe OSA among pediatric patients with sleep disordered breathing referred for polysomnography (PSG).

Note: This logistic regression model predicts severe OSA among a group of 249 children. _cons represents the baseline odds when all predictor variables in the model are set to their reference categories. In this case, the reference categories are children who are not obese and whose language is English. Pearson χ^2 goodness of fit = 3.28; p = .07. Predictors evaluated in the model—race, ethnicity, ADI, payer, GERD, asthma, weight, BMI, developmental delay, allergic rhinitis, and preoperative positive airway pressure therapy.

OSA were not significantly associated with race, ethnicity, or insurance type. Residual moderate or greater OSA was found to be associated with older age (p = .009) and AHI at the initial sleep study (p = .02), but not with neighborhood-level disadvantage, that is, national ADI rank (p = .29).

Our assessment of a patient's level of disadvantage, as determined by their ADI national ranking, reveals that children who are classified as disadvantaged are 1.17 times more likely to have severe OSA than those who are classified as less deprived. However, it is important to note that this relationship is not considered precise due to the wide confidence interval (95% CI = 0.99-1.01). In addition, our model's pseudo- R^2 value and likelihood χ^2 test indicate that the relationship is not significant ($R^2 = 0.0013$; χ^2 test = 0.44). These findings suggest that while there may be a slight association between disadvantage status and OSA severity, further research is needed to determine a more definitive relationship.

Multiple logistic regression was used to predict demographic and clinical variables associated with severe OSA and is displayed in Table 6. The predictors evaluated in the model included variables where the *p* value in Tables 1 and 3 were ≤ 0.25 . These variables included the following: race, ethnicity, preferred language, ADI national ranking, primary payer, GERD, asthma, weight, BMI, allergic rhinitis, developmental delay, and preoperative PAP therapy. The results show that children with obesity have 3.13 higher odds of severe OSA than those who are not obese, and patients whose families preferred language is Spanish have 1.96 times higher odds of severe OSA. According to the χ^2 test (*p* < .001), the model was statistically significant and accounted for 6.83% of the variation in severe

OSA (pseudo- $R^2 = 0.068$). The model's intercept estimates the baseline odds of the event occurring to be 25%.

An additional logistic regression model was used to predict variables associated with residual OSA. The simple regression model of residual OSA and more disadvantages was not significant (OR = 0.57, 95% CI = 0.20–1.63). The predictors evaluated in the multiple logistic models included variables for which the p values in Tables 1 and 4 were ≤ 0.25 . These variables included: ethnicity, preferred language, ADI national rank, payer, GERD, asthma, age, weight, BMI, and development delay. This model showed that for a 1-year increase in age at the time of the sleep study, residual OSA risk increased by 1.20 (95% CI = 1.05 - 1.38). The model was statistically significant (p < .001) according to the likelihood ratio χ^2 test, and the pseudo-R² value suggests that the model explains about 7.1% of the variation in residual OSA. The model estimated the baseline odds of the residual OSA was 5.3%. The predicted probability of residual OSA after tonsillectomy and the AHI >5 occurring is 12.3% for a 5-year-old child, while the chance increases to 19.6% for a 17-year-old. No other effect modifiers or statistical interactions were found.

4 | DISCUSSION

In this study, we evaluated the demographic and clinical characteristics of 249 children who underwent PSG and subsequent T&A at a single medical center. The median (IQR) age of the children was 9.4 years (7.3–12.3). We found that neighborhood-level deprivation, as measured by the ADI ranking, was not a predictor of OSA severity or residual moderate or greater OSA after surgery. Furthermore, we did not observe any association between OSA severity or residual OSA and race or ethnicity. However, we did find that severe OSA was associated with obesity and that residual OSA was associated with older age at the initial sleep study but not with obesity or sex.

The relationship between SES and pediatric OSA has been investigated in previous research with mixed results. For example, a study by Wang et al. found that higher poverty rates and a higher percentage of single-female-headed households were associated with a higher AHI in a sample of 774 children from six American cities.⁸ In contrast, a study by Park et al. found that certain markers of SES, such as maternal education and urban location, were associated with a higher AHI in a Canadian sample,⁵ but did not find a significant association with perceived SES based on the MacArthur Scale of Subjective Social Status. In addition, Xie et al. found no statistically significant differences in markers of SES, including median household income, single-parent households, public insurance coverage, race, or age, between children with refractory and non-refractory OSA following T&A.¹⁵ These findings suggest that the relationship between SES and pediatric OSA may be complex and may vary depending on the specific measures of SES used and the characteristics of the study population.

The majority of these previous studies used individual measures to serve as proxies for SES. Our study is the first to use ADI, a comprehensive index of neighborhood-level deprivation, to examine pediatric OSA severity and outcomes following T&A treatment. Although more social disadvantages may increase the risk of comorbidities associated with OSA, it was not an independent risk factor when using ADI in our study.

Studies on the relationship between race and pediatric OSA have been similarly conflicting. In a Canadian study, Brouillette et al. found that Black children were 3.35 times more likely to have OSA than non-Black children.⁶ Weinstock et al. also found that, after controlling for confounders-including maternal education, family income, age, sex, obesity, and medical comorbidities-African American race was associated with an approximately 20% increase in AHI.⁴ However, Wang et al. found in their study, mentioned above, that after controlling for poverty rate and single-female-headed households, the association between African American race and AHI was no longer significant.⁸ Similarly, Baker et al. found that race was not a predictor for increased AHI in adolescents.¹⁶ In our study, we found that African American race was not associated with AHI in the pediatric population. However, it is worth noting that our study population had a high number of Hispanic patients, which could potentially be a confounding variable as Hispanic children may have different genetic, environmental, or behavioral exposures that may influence the development or manifestation of OSA thereby limiting the impact of race on OSA in this cohort. It is interesting to note our regression model found that severe OSA was associated with the Spanish language, but not Hispanic ethnicity. It is possible that there are unique social factors-for example, language barriers and/or lack of access to carethat may delay the diagnosis and treatment of OSA, leading to an increased likelihood of severe OSA in this population. However, this observation is a secondary finding that should be interpreted with caution and warrants further study.

Our study shows an association between obesity and OSA severity, a relationship that has been well-documented in the literature.^{16–20} While tonsillar hypertrophy has been shown to be associated with OSA,^{16,19,20} this was not the case for our population. Dayyat et al. reported that in obese children, tonsillar hypertrophy has a smaller contribution to OSA, as measured by changes in OAHI, than in nonobese children.²¹ Thus, the effects of tonsillar hypertrophy on OSA severity may not have been significant in our population as obesity was prevalent. This finding emphasizes the importance of using PSG to evaluate patients with small tonsil size, especially in the setting of obesity. However, we also recognize that, given the majority of our patients had tonsillar hypertrophy (80.1%), there may not have been enough patients without tonsillar hypertrophy to show a significant difference.

In our study, we found that older age and higher AHI at the initial sleep study was associated with residual moderate or greater OSA, but there was no significant association with BMI *z*-score or sex. This finding contrasts with previous research that has found a relationship between these factors and residual OSA.^{15,22} However, our study used a different definition of residual OSA (AHI \ge 5) compared to some previous studies, which may account for the discrepancy. In addition, comorbidities like obesity, Down syndrome, and hypotonia may be more relevant predictors of residual OSA than demographic

factors.^{15,23} It is also important to note that this was another secondary finding which merits cautious interpretation. Continued research is needed to understand better the factors contributing to residual OSA in children following tonsillectomy.

The limitations of this study should be noted. While our sample size was relatively large, the majority (70.3%) of our population was disadvantaged, which may have limited the power of the study to detect differences in this group. In addition, our population was predominantly Hispanic (62.5%), which may not be representative of the overall pediatric OSA population. While ADI provides a comprehensive measure of neighborhood-level disadvantage, it does not capture all aspects of disadvantage, such as racism and discrimination, which can have negative impacts on health.^{23,24} Individual levels of SES may better reflect some families' social standing (e.g., family income, insurance status, or employment). Our study was also limited by its retrospective design, which may have introduced selection bias. It is worth noting that while there is a wellestablished relationship between obesity and socioeconomic disadvantage, this relationship is not seen in our study between ADI and BMI. However, it is important to note this study was primarily designed to examine the relationship between ADI and OSA. It is possible that the effect of BMI and ADI may not be identifiable in our study due to children with obesity having a larger representation (56.2% vs. 43.8%), as children with obesity are more likely to be referred for PSG in clinical practice. In addition, individual-level SES and lifestyle behaviors may have a stronger influence on obesity in this sample. Strengths of this study include the use of a validated measure of neighborhood deprivation and the inclusion of a large and ethnically diverse population. Further research is needed to fully understand the relationship between socioeconomic measures and OSA severity and treatment outcomes and identify risk factors for residual OSA in older children.

5 | CONCLUSION

In conclusion, this study of 249 children aged 20 months to 18 years found that neighborhood-level disadvantage and race were not associated with OSA severity or residual moderate or greater OSA following T&A. However, obesity was identified as a risk factor for OSA severity, while older age was a risk factor for residual OSA. Further prospective studies examining the influence of these socioeconomic, demographic, and clinical factors on pediatric OSA are needed to better understand and address the prevalence of OSA in children.

CONFLICT OF INTEREST STATEMENT

The authors declare no conflict of interest.

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