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ORIGINAL ARTICLE

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Association of body anthropometry and obstructive sleep apnea in children: Variations observed in Hispanic children

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Abstract

Objectives: Obesity is a risk factor for obstructive sleep apnea (OSA) in children. Childhood obesity rates vary amongst different ethnic groups. Here the interaction of Hispanic ethnicity and obesity on OSA risk was evaluated.

Methods: Retrospective cross-sectional analysis of consecutive children undergoing polysomnography and anthropometry using bioelectrical impedance from 2017 to 2020. Demographics obtained from the medical chart. Children who had also undergone cardiometabolic testing were identified and the relationship of cardiometabolic markers with OSA and anthropometry was assessed.

Results: Data from 1217 children revealed Hispanic children were more likely to have moderate-severe OSA (36.0%) compared to Non-Hispanic children (26.5%), $p < 0.001$. Hispanic children had greater Body mass index (BMI), BMI percentile and percent body fat, $p < 0.0001$. In children that underwent cardiometabolic testing, Hispanic children had significantly greater serum alanine aminotransferase levels (ALT) levels. Following adjustment of age and sex, Hispanic ethnicity was not found to moderate the association of anthropometry with OSA, anthropometry with cardiometabolic markers, and OSA with cardiometabolic markers.

Conclusions: OSA was more likely in Hispanic children; this relationship was likely driven by obesity status rather than ethnicity. Among children undergoing cardiometabolic testing, Hispanic children were observed to have greater ALT concentrations however ethnicity did not impact the association of anthropometry and ALT or other cardiometabolic markers.

KEYWORDS
anthropometry, childhood obesity, ethnicity, obstructive sleep apnea

Sonia Jain, Rakesh Bhattacharjee denotes both authors served equally as senior authors.

1 | INTRODUCTION

Obstructive sleep apnea (OSA) is a common condition in which normal sleep is disrupted by periodic collapse of the upper airway causing sleep fragmentation and abnormal gas exchange, including intermittent oxygen desaturation. In children, the reported estimated prevalence of OSA is about 2%–3%.[1–4] While hypertrophy of adenotonsillar tissue comprises as the major risk factor for OSA in children,[4,5] particularly young children, the surge in childhood obesity[6] has resulted in an increase in prevalence with up to 6% of all children being affected by OSA.[7–11] Furthermore, the degree of adenotonsillar hypertrophy necessary for airway obstruction during sleep is significantly less in children with obesity.[12] In context of the high prevalence of childhood obesity, OSA should now be considered a common disease of childhood.

Obesity impacts sleep related breathing via independent mechanisms. Fatty deposits in the anterior neck encroaches the upper airway, narrowing the diameter of the upper airway leading to pharyngeal collapsibility during sleep.[13,14] In addition, abdominal obesity restricts normal diaphragmatic expansion, particularly during supine sleep, thereby reducing lung volumes and predisposing children to gas exchange abnormalities typical of OSA.[7,15]

In children, studies have implicated OSA in neurocognitive and behavioral consequences[16–22] and recently cardiovascular morbidity.[23] Of note, like childhood obesity, OSA has also been associated with several metabolic derangements. Lipid profiles obtained from pre-pubertal children with OSA or adenotonsillar hypertrophy correlate with elevations in low-density lipoprotein cholesterol levels, and reductions in high-density lipoprotein cholesterol levels which reversed following treatment.[24] Similar findings have been corroborated in post-pubertal children.[25] While it is noteworthy that obesity is a significant risk factor for childhood metabolic disease, the coexistence of OSA in many children with obesity may augment metabolic disease even further. Adding to the complexity of this relationship, studies have suggested that sleep restriction or lack of sleep[26,27] and sleep fragmentation induced by OSA[28] may increase obesity risk in children.

Ethnicity is a well ascribed major risk factor for childhood obesity,[29] and impacts pediatric OSA prevalence.[4,30–33] Notwithstanding few studies have addressed the influence of Hispanic ethnicity on OSA.[34] Here a large population of children undergoing both sleep study testing and body anthropometric measurements was formally assessed. The goal of the study was to assess whether Hispanic ethnicity influences the prevalence of OSA as well as body anthropometry in children and evaluate the interaction of Hispanic ethnicity with anthropometric measures on OSA. In addition, those patients that had undergone cardiometabolic testing within 1 year of their sleep study were evaluated to assess the relationship of OSA and anthropometry on cardiometabolic markers and assess the influence of Hispanic ethnicity status. The hypothesis of the study was that Hispanic ethnicity interacts with the relationship of obesity and OSA.

2 | MATERIALS AND METHODS

Criteria for study inclusion consisted of all children (age<18 years) that had a diagnostic polysomnogram (PSG) as well as anthropometry measured using the InBody 570® Body composition Analyzer (Queensland, Australia). All testing was conducted at the Rady Children's Hospital Center for Healthy Sleep, San Diego, CA from January 2017 to March 2020. Demographic data including age, sex, ethnicity, medical history was extracted from the electronic medical record (EMR). Children undergoing polysomnography for the purpose of positive airway pressure titration were excluded. During the study period, if children had undergone repeat polysomnography to assess sleep following airway surgery, only presurgical polysomnograms were chosen.

Of all patients included in the study, a subset of patients was also identified using the EMR if they had undergone cardiometabolic blood testing within 1 year of the date of the time of their sleep lab visit. These patients were separately analyzed to evaluate the relationship of sleep apnea or anthropometry on cardiometabolic profiles.

Overnight laboratory diagnostic PSGs were conducted and scored by sleep technologists and interpreted by a pediatric sleep medicine physician according to American Academy of Sleep Medicine criteria.[35] Polysomnogram parameters examined included the total sleep apnea hypopnea index (AHI), obstructive apnea-hypopnea index (OAHI), oxygen desaturation index (ODI), and oxygen saturation nadir during sleep. Normal, mild, and moderate/severe OSA was defined as having an OAHI less than 1.5, 1.5 to ≤5, and >5 events/hour respectively. Where an OAHI of 5 to 10 events/hr is consistent with moderate OSA and an OAHI≥10 events/hr is consistent with severe OSA; both groups were merged together, as typically these groups are most likely to be treated, which can include initiating positive airway pressure therapy, whereas observation or watchful waiting may be indicated in mild OSA.[36]

Body mass index (BMI) was calculated using weight and height as measured by a digital stadiometer. Body mass index percentiles were calculated using Centers for Disease and Control (CDC) normative data in children. Body composition was measured using a bioelectrical impedance analyzer (InBody 570®). The analyzer measures body water through bioelectrical impedance using electrodes that were situated beneath the subject's feet on the platform and on the palms and thumbs attached to handles on the device. As a result, the device was limited to children who can stand independently. Bioelectrical impedance was used to derive dry lean mass and calculate body fat mass and percentage of body fat. This technique has been shown to strongly correlate with dual-energy X-ray absorptiometry scanning.[37]

2.1 | Statistical analysis

Descriptive statistics were used to summarize demographic characteristics of children. All normally distributed data were presented with sample mean ± standard deviation, data that were not normally

distributed were presented with median and interquartile range. Group comparisons between Hispanic and Non-Hispanic children used Wilcoxon rank sum test for continuous variables and Fisher's exact test for categorical variables. Logistic regression model was used to assess whether the association of each anthropometric variable or cardiometabolic measure with the presence of OSA differed between Hispanic versus Non-Hispanic children. Linear regression model was used to study whether Hispanic ethnicity moderated the association between each anthropometric variable and cardiometabolic outcome. To minimize data skew, cardiometabolic outcomes underwent log transformation in these analyses. All models were adjusted for age and sex. Analyses were conducted in R (version 3.6.1).

All study data were collected retrospectively through approval by the Institutional Review Board at the University of California, San Diego (#180458) (consistent with the World Medical Association Declaration of Helsinki regarding ethical conduct of research involving human subjects).

3 | RESULTS

Anthropometric and sleep study data were available from 1333 children. Patients who had sleep studies less than 240 min were deemed as unsuccessful sleep studies and were thereby excluded from the analysis (n = 99). Thirteen subjects were excluded due to a current age of greater than 18 years. Four subjects were excluded due to absence of ethnicity data. A total of 1217 children (509 females or 41.8%) during the study period were included. Demographic sleep study characteristics are outlined in Table 1. Of the study population, 659 (54.1%) children were of Hispanic ethnicity. There were no significant differences in sex distribution between Hispanic and Non-Hispanic children. There was however a statistically significant difference in age with the mean ± sd age of Hispanic children found to be 10.9 ± 3.5 years compared to 10.4 ± 3.9 years in Non-Hispanic children ($p = 0.0043$), although this difference was unlikely to be clinically significant.

The median (IQR) AHI and OAHI were 3.2 (1.5–8.3) events/hr and 2.3 (0.9–7.1) events/hr of the entire population. Hispanic children were observed to have significantly larger median AHI and OAHI values at 3.8 events/hr and 3.0 events/hr compared to Non-Hispanic children 2.6 events/hr and 1.9 events/hr (Table 1) ($p < 0.0001$ for both measures. In addition, Hispanic children were observed to have significantly larger median ODI levels and significantly lower median oxygen saturation nadirs. Overall, 472 (38.8%) of the 1217 children were observed to have normal sleep studies (OAHI≤1.5 events/hr), 360 children (29.6%) were found to have mild OSA (1.5<OAHI≤5 events/hr) and 385 children (31.6%) were found to have moderate-severe OSA (OAHI>5 events/hr). Hispanic children were more likely to have moderate-severe OSA (237 of 659 children or 36.0%) compared to Non-Hispanic children (148 of 558 children or 26.5%), and less likely to have had normal sleep studies (227 of 659 children or 34.5%) compared to Non-Hispanic children (245 of 558 children or 43.9%), $p < 0.001$.

Anthropometric measures revealed that Hispanic children were found to have greater BMI, BMI percentiles, body fat mass, and percent body fat (Table 2). However, despite the observed larger polysomnographic indices suggestive of OSA and anthropometric indices in Hispanic children, following adjustment for age and sex, there was no significant interaction of Hispanic ethnicity on the relationship of body anthropometry and the presence of moderate-severe OSA (OAHI>5 events/hr) (Table 3).

In the subset of patients who undergone cardiometabolic evaluation within a year of their sleep study, there were no significant

TABLE 1 Demographic characteristics and sleep apnea outcomes of study population

	Total ($n = 1217$)	Hispanic ($n = 659$)	Non-Hispanic ($n = 558$)	p Value
Sex				>0.999
Female	509 (41.8%)	276 (41.9%)	233 (41.8%)	
Male	708 (58.2%)	383 (58.1%)	325 (58.2%)	
Age (years) [Mean ± SD, range]	10.6 ± 3.7 [1–18]	10.9 ± 3.5 [1–18]	10.4 ± 3.9 [3–18]	**0.0043**
AHI (events/hr) [Median, IQR]	3.2 (1.5–8.3)	3.8 (1.6–10.3)	2.6 (1.3–6.6)	**<0.0001**
OAHI (events/hr) [Median, IQR]	2.3 (0.9–7.1)	3.0 (1.1–8.7)	1.9 (0.7–5.4)	**<0.0001**
ODI (events/hr) [Median, IQR]	0.7 (0.2–2.4)	0.8 (0.2–3.0)	0.6 (0.2–1.8)	**<0.0001**
SaO2 Nadir (%) [Median, IQR]	93.0 (90.0–94.0)	92.0 (89.0–94.0)	93.0 (90.0–95.0)	**<0.0001**
OSA category [Number, %]				**<0.001**
Normal (OAHI < 1.5)	472 (38.8%)	227 (34.5%)	245 (43.9%)	
Mild (1.5 ≤ OAHI <5)	360 (29.6%)	195 (29.6%)	165 (29.6%)	
Moderate - severe (OAHI ≥ 5)	385 (31.6%)	237 (36.0%)	148 (26.5%)	

Note: The Bold Values represented significant of p value (i.e. <0.05)

Abbreviations: AHI, apnea hypopnea index; OAHI, obstructive apnea hypopnea Index; ODI, oxygen desaturation Index; OSA, obstructive sleep apnea; SaO2, oxygen saturation.

differences observed in triglyceride and total cholesterol levels (Table 4) when comparing Hispanic to Non-Hispanic children. There were no differences in inflammation detected as determined by C-reactive protein or the percentage of Hemoglobin A1c. There was however a significant elevation in alanine aminotransferase levels (ALT) in Hispanic children 41.4 ± 37.5 U/L compared to 31.4 ± 21.5 U/L in Non-Hispanic children ($p < 0.0001$).

Following adjustment for age and sex, there were no statistically significant differences observed in any of the cardiometabolic markers of focus in those children with normal sleep studies compared to those children with moderate-severe OSA, regardless of Hispanic ethnicity (Table 5). Hispanic ethnicity was also not found to significantly moderate the association between anthropometric measures and any of the cardiometabolic markers (Table 6).

TABLE 2 Anthropometric measures of study population

Anthropometric measure	Total ($n = 1217$)	Hispanic ($n = 659$)	Non-Hispanic ($n = 558$)	p Value
BMI	23.7 ± 7.9	26.0 ± 8.0	21.0 ± 6.7	**<0.0001**
BMI percentile	66.0 ± 34.7	75.9 ± 31.2	54.4 ± 35.0	**<0.0001**
Dry lean Mass	21.4 ± 8.90	22.2 ± 8.8	20.6 ± 9.1	**<0.0001**
Body fat Mass	44.5 ± 36.8	55.6 ± 38.0	31.4 ± 30.5	**<0.0001**
Percent body fat	30.9 ± 14.1	36.2 ± 12.3	24.6 ± 13.3	**<0.0001**

Note: Mean ± SD was reported.

Abbreviation: BMI, body mass index.

TABLE 3 Associations between anthropometry and obstructive sleep apnea (OSA) (moderate-severe (OAHI > 5) versus normal (OAHI < 1.5)) and interaction of Hispanic ethnicity

Anthropometric measure	Ethnicity	Odds Ratio (OR)	OR (lower limit)	OR (upper limit)	p Value	Interaction p Value
BMI	Non-Hispanic	2.055	1.559	2.708	**<0.001**	0.709
	Hispanic	2.191	1.746	2.749	**<0.001**	
BMI percentile	Non-Hispanic	1.478	1.195	1.827	**<0.001**	0.876
	Hispanic	1.513	1.223	1.873	**<0.001**	
Dry lean Mass	Non-Hispanic	1.721	1.249	2.371	**<0.001**	0.591
	Hispanic	1.868	1.392	2.506	**<0.001**	
Body fat Mass	Non-Hispanic	2.019	1.529	2.666	**<0.001**	0.811
	Hispanic	2.104	1.664	2.659	**<0.001**	
Percent body fat	Non-Hispanic	1.860	1.477	2.342	**<0.001**	0.474
	Hispanic	2.093	1.653	2.650	**<0.001**	

Note: Separate logistic regression models were performed to assess whether the association of each anthropometric measure (in standardized scale) with OSA (defined as moderate-severe if OAHI>5/hr vs. normal if OAHI<=1.5/hr) was moderated by ethnicity status (Hispanic vs. non-Hispanic). All models adjusted for age and sex. OR represents the odds of having moderate-severe OSA per standardized unit increase in each anthropometric measure. Interaction p Value indicates whether the association differed between Hispanic and Non-Hispanic children. The Bold Values represented significant of p value (i.e. <0.05)

Abbreviation: BMI, body mass index.

TABLE 4 Cardiometabolic measures of study patients having undergone blood testing

	Total	n	Hispanic	n	Non-Hispanic	n	p Value
Triglycerides (mg/dl)	110 (85–156)	205	112 (88–157)	136	103 (78–150)	69	0.153
Total cholesterol (mg/dl)	155 (131–177)	204	151 (129–177)	136	158 (136–176)	68	0.297
C-reactive protein (mg/L)	1 (0.5–3.9)	89	1.2 (0.5–3.9)	53	0.6 (0.5–3.2)	36	0.263
ALT (U/L)	29 (21–42)	365	32 (23–45)	220	26 (19–34)	145	**<0.0001**
HbA1c (%)	5.4 (5.1–5.5)	106	5.4 (5.2–5.5)	74	5.4 (5.1–5.5)	32	0.531

Note: Median (IQR) was reported.

Abbreviations: ALT, alanine aminotransferase; HbA1c, hemoglobin A1c.

TABLE 5 Associations between cardiometabolic measures and obstructive sleep apnea (OSA) (moderate-severe (OAHI>5) versus normal (OAHI<1.5)) and interaction of Hispanic ethnicity

Cardiometabolic marker	Ethnicity	Odds Ratio (OR)	OR (lower limit)	OR (upper limit)	*p* Value	Interaction *p* Value
Triglycerides (n = 150)	Non-Hispanic	1.009	0.998	1.019	0.117	0.294
	Hispanic	1.002	0.994	1.009	0.670	
Total cholesterol (n = 150)	Non-Hispanic	1.017	0.994	1.041	0.137	0.075
	Hispanic	0.994	0.981	1.007	0.332	
C-reactive protein (n = 68)	Non-Hispanic	1.038	0.947	1.138	0.415	0.590
	Hispanic	1.080	0.966	1.207	0.173	
ALT (n = 259)	Non-Hispanic	1.005	0.987	1.023	0.605	0.883
	Hispanic	1.006	0.995	1.017	0.270	
HbA1c (n = 69)	Non-Hispanic	43.827	0.736	2608.760	0.069	0.314
	Hispanic	3.755	0.271	51.954	0.318	

Note: Separate logistic regression models were performed to assess whether the association of each cardiometabolic markers with OSA (defined as moderate-severe if OAHI>5/hr vs. normal if OAHI<=1.5/hr) was moderated by ethnicity status (Hispanic vs. Non-Hispanic). All models adjusted for age and gender. Odds Ratio (OR) represents the odds of having OSA per unit increase in each cardiometabolic marker. Interaction *p* Value indicates whether the association differed between Hispanic and Non-Hispanic children.

Abbreviations: ALT, alanine aminotransferase; HbA1c, hemoglobin A1c.

4 | DISCUSSION

The findings reveal that in a very large clinical population of children undergoing polysomnography, Hispanic children were more likely to have moderate-severe OSA, more likely to have anthropometric measures that suggest obesity, and finally a greater likelihood of having elevated serum ALT levels. However, following adjustment for age and sex, Hispanic ethnicity did not moderate the relationship of OSA and body anthropometry. Notwithstanding, these findings revealed that obesity was more common in Hispanic children and given the propensity toward obesity in Hispanic children, they were at a greater risk for OSA. The study findings may be important motivation for preventative strategies including diet and exercise in all children particularly in those of Hispanic ethnicity. Moreover, the findings should improve awareness and recognition of OSA and OSA related complications in Hispanic children.

In addition to the findings on OSA, the study did reveal differential susceptibilities of cardiometabolic risk namely ALT, a surrogate for hepatic steatosis, in Hispanic children. However, following adjustment for age and sex, Hispanic ethnicity did not significantly moderate the association of anthropometry and ALT or other cardiometabolic markers of interest. A recent study of Korean children,[38] has also reported elevation of ALT in children with OSA and obesity implying the link of obesity and OSA with cardiometabolic disease, however this study did not assess the impact of different ethnic groups.

Despite the study drawing conclusions from a very large dataset of children (n = 1217), there were several limitations with the study. First, the relationship of cardiometabolic risk with OSA status and body anthropometry was limited by a smaller sample size, as not all children underwent metabolic blood testing. Further, those children that underwent metabolic blood testing were more likely to have testing done for a specific reason, for example, family history of cardiovascular disease or diabetes risk. Further work will be required to look at the generalizability of the findings. Second, the study did not evaluate hard cardiometabolic outcomes, such as liver biopsy, or cardiac MRI. Thus, the study relied on surrogate measures which are typically used clinically. However, the findings are supportive of further efforts to perform more definitive outcome measures. Third, based on the nature of the study design, a clinical sample was evaluated with an inherent referral bias. For example, it is plausible that the Hispanic children with obesity were more likely to be referred to the sleep clinic for purposes of screening related to obesity complications. Because the goal was to perform a real-world study in a clinical sample, the findings of this study are important. Nonetheless, efforts to evaluate community cohorts may provide additional insights including identifying children at greater risk for OSA and thus considered for earlier screening via diagnostic PSG. Fourth, the study did not evaluate specific phenotypical traits associated with OSA that may be more prevalent in certain ethnic groups such as mandibular cortical width[39] or neck circumference.[40] Fourth, the findings were correlative, but provide supportive data to inform the proper design of rigorous randomized clinical trials to evaluate further the impact of treatment such as positive airway pressure on ethnically diverse children with OSA.

5 | CONCLUSIONS

Data from a large population of children showed that prevalence rates of OSA and anthropometric measures of obesity were significantly higher in Hispanic children compared to Non-Hispanic children. Following adjustment, Hispanic ethnicity however did not moderate the association of anthropometry and OSA, OSA and cardiometabolic risk and finally anthropometry and cardiometabolic risk. These findings imply that while the clinical presentation and potential

TABLE 6 Associations between anthropometric measures and cardiometabolic markers interaction of Hispanic ethnicity

Cardiometabolic measure	Ethnicity	Beta	Beta (lower limit)	Beta (upper limit)	*p* Value	Interaction *p* Value
Triglycerides (n = 205)						
BMI	Non-Hispanic	0.217	0.110	0.324	**<0.001**	0.081
	Hispanic	0.107	0.034	0.179	**0.004**	
BMI percentile	Non-Hispanic	0.184	0.069	0.299	**0.002**	0.480
	Hispanic	0.129	0.027	0.231	**0.014**	
Dry lean Mass	Non-Hispanic	0.197	0.058	0.337	**0.006**	0.385
	Hispanic	0.142	0.025	0.258	**0.018**	
Body fat Mass	Non-Hispanic	0.192	0.085	0.299	**0.001**	0.072
	Hispanic	0.080	0.005	0.154	**0.036**	
Percent body fat	Non-Hispanic	0.185	0.043	0.326	**0.011**	0.452
	Hispanic	0.118	0.017	0.219	**0.022**	
Total cholesterol (n = 204)						
BMI	Non-Hispanic	0.022	−0.031	0.074	0.416	0.231
	Hispanic	−0.015	−0.051	0.020	0.393	
BMI percentile	Non-Hispanic	0.015	−0.041	0.071	0.603	0.476
	Hispanic	−0.012	−0.061	0.037	0.623	
Dry lean Mass	Non-Hispanic	−0.008	−0.074	0.058	0.814	0.434
	Hispanic	−0.032	−0.087	0.024	0.262	
Body fat Mass	Non-Hispanic	0.019	−0.032	0.071	0.460	0.150
	Hispanic	−0.024	−0.059	0.012	0.190	
Percent body fat	Non-Hispanic	0.017	−0.051	0.085	0.616	0.353
	Hispanic	−0.022	−0.070	0.026	0.370	
C-reactive protein (n = 89)						
BMI	Non-Hispanic	0.491	−0.001	0.983	0.050	0.774
	Hispanic	0.573	0.242	0.904	**0.001**	
BMI percentile	Non-Hispanic	0.483	0.064	0.901	**0.024**	0.981
	Hispanic	0.490	0.089	0.890	**0.017**	
Dry lean Mass	Non-Hispanic	0.014	−0.549	0.576	0.961	0.246
	Hispanic	0.332	−0.119	0.783	0.147	
Body fat Mass	Non-Hispanic	0.448	−0.028	0.924	0.064	0.962
	Hispanic	0.435	0.092	0.778	**0.014**	
Percent body fat	Non-Hispanic	0.638	0.222	1.054	**0.003**	0.692
	Hispanic	0.527	0.154	0.900	**0.006**	
ALT (n = 365)						
BMI	Non-Hispanic	0.153	0.055	0.252	**0.002**	0.899
	Hispanic	0.161	0.090	0.232	**<0.001**	
BMI percentile	Non-Hispanic	0.151	0.063	0.239	**0.001**	0.784
	Hispanic	0.168	0.083	0.253	**<0.001**	
Dry lean Mass	Non-Hispanic	0.171	0.053	0.289	**0.005**	0.583
	Hispanic	0.204	0.108	0.299	**<0.001**	
Body fat Mass	Non-Hispanic	0.140	0.040	0.240	**0.006**	0.723

(Continues)

TABLE 6 (Continued)

Cardiometabolic measure	Ethnicity	Beta	Beta (lower limit)	Beta (upper limit)	*p* Value	Interaction *p* Value
	Hispanic	0.161	0.088	0.235	**<0.001**	
Percent body fat	Non-Hispanic	0.145	0.042	0.247	**0.006**	0.615
	Hispanic	0.180	0.087	0.272	**<0.001**	
HbA1c (n = 106)						
BMI	Non-Hispanic	0.022	−0.011	0.054	0.186	0.979
	Hispanic	0.022	0.003	0.041	**0.021**	
BMI percentile	Non-Hispanic	0.010	−0.019	0.038	0.503	0.831
	Hispanic	0.014	−0.018	0.046	0.383	
Dry lean Mass	Non-Hispanic	−0.011	−0.051	0.029	0.581	0.815
	Hispanic	−0.007	−0.039	0.025	0.665	
Body fat Mass	Non-Hispanic	0.022	−0.012	0.055	0.198	0.814
	Hispanic	0.018	−0.001	0.047	0.068	
Percent body fat	Non-Hispanic	0.043	0.001	0.085	**0.046**	0.479
	Hispanic	0.025	−0.002	0.052	0.065	

Note: Separate linear regression models were performed to assess whether the association of each anthropometric measures (in standardized scale) with each cardiometabolic outcomes (in log scale) was moderated by ethnicity status (Hispanic vs. non-Hispanic). All models adjusted for age and gender. Beta represents the mean difference in each outcome per standardized unit increase in each anthropometric measure. Interaction *p* Value indicates whether the association differed between Hispanic and non-Hispanic children. The Bold Values represented significant of *p* value (i.e. <0.05)
Abbreviations: ALT, alanine aminotransferase; BMI, body mass index; HbA1c, hemoglobin A1c.

cardiometabolic consequences may vary when comparing Hispanic to Non-Hispanic children, the effect was largely driven by the presence of obesity rather than ethnicity specifically.

AUTHOR CONTRIBUTIONS

Emily B. Bhattacharjee participated in study concept, data analysis, data interpretation, and manuscript preparation; Xiaoying Sun participated in data analysis; Atul Malhotra participated in data analysis, data interpretation and manuscript preparation, Kelan G. Tantisira participated in manuscript preparation, Sonia Jain participated in data analysis and data interpretation; Rakesh Bhattacharjee participated in study concept, data collection, data analysis, data interpretation and manuscript preparation.

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CONFLICT OF INTEREST

Emily B. Bhattacharjee does not report any conflict of interests. Xiaoying Sun does not report any conflict of interests. Atul Malhotra reports funding by the National Institutes of Health. He reports income from Corvus, Equillium and Livanova related to medical education. Resmed provided a philanthropic donation to UCSD. Kelan G. Tantisira reports funding by the National Institutes of Health. JSLG does not report any conflict of interests. Sonia Jainreports funding by the National Institutes of Health. Rakesh Bhattacharjee has received consulting and speaker's fees from Jazz Pharmaceuticals.

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