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Autism Spectrum Disorder Diagnosis and Other Child, Family, and Community Risk Factors for Obesity among Children and Adolescents Aged 10–17 Years in the United States: A Mediation Analysis

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Abstract

Background: This study explored whether there are mediated effects of child and family risk in the association between community and organizational risk and obesity among children and adolescents aged 10–17 years using 2017–2018 National Survey of Children's Health (NSCH) data, addressing autism spectrum disorder (ASD) and co-occurring conditions.

Methods: This cross-sectional study ($N = 27,157$) used 2017–2018 NSCH data. Frequency distributions and chi-square tests were used to describe participants with and without ASD. Cumulative risk indices were created for child, family, community, and organizational level risk, and mediation analyses were conducted in a two-mediator model (X_1: community risk, X_2: organizational risk, M_1: child risk, M_2: family risk) for the dichotomous outcome (Y: obesity). Path analyses were performed using generalized structural equation modeling in Stata 16.0.

Results: Direct effects for all four risk indices were associated with obesity in single index models (all $p < 0.001$); only child and family risk indices were associated with obesity in a full model with all four risk indices (both $p < 0.001$). When child and family risk indices were assessed as mediators, the indirect effects of community and organizational risk were significant (all $p < 0.0001$). The total effect of community risk on obesity was significant with family risk as a mediator ($p = 0.002$). The total effect of organizational risk was not significant with either mediator.

Conclusion: Findings suggest that child and family factors play a strong role in obesity risk and that ASD contributes to this risk. Community risk may be another strong predictor of obesity, mediated by family risk. Additional research on social-ecological risk factors for obesity is needed to identify leverage points to improve obesity risk in children and adolescents with and without ASD.

Keywords: adolescence; ASD; childhood; cross-sectional; structural equation modeling

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Introduction

Childhood obesity is a pressing public health concern, with a current estimated prevalence of 18.5% in the United States.[1] Children with obesity are more likely to have obesity and chronic diseases such as type 2 diabetes, cardiovascular disease, and certain types of cancer as adults.[2,3] Children with autism spectrum disorder (ASD) have an increased risk of having overweight or developing obesity compared with typically developing children, with an estimated obesity prevalence of 22.2% and a 41.1% greater risk of developing obesity compared with typically developing children.[4] ASD, characterized by social communication and behavioral challenges, is one of the fasting growing developmental disabilities; the estimated prevalence of ASD in the United States is 23 per 1000 (1 in 44).[5] Known risk factors for obesity in children with ASD include unhealthy eating behaviors, increased sedentary behavior, genetic vulnerabilities, and medication use to manage symptoms of ASD and co-occurring conditions.[6]

As obesity has been associated with various social-ecological determinants,[7–12] a useful framework to develop a comprehensive understanding of obesity determinants is the Social Ecological Model (SEM).[11,13] Variations of the SEM may include five levels of prevention that interact with each other: individual, such as age, education, and income; interpersonal, including family influences; institutional/organizational, including health care factors; community, such as school, workplace, and neighborhood factors; and policy, including laws and policies.[14,15] The Institute of Medicine recommends the SEM to examine determinants of childhood obesity and provide a foundation for intervention research.[16] However, there is a lack of integration of studies examining child characteristics with contextual factors of the family, community, and health care-related environment.[17,18] Research on obesity in children and adolescents with and without ASD exploring synergistic effects among multiple social-ecological risk factors is limited. Moreover, research on obesity risk factors including ASD diagnosis have focused on child-level factors, rather than family, community, and health care-related factors.[6]

Research on child, family, community, and organizational factors associated with obesity that includes ASD and co-occurring conditions is warranted. Prior research using 2007–2008 National Survey of Children's Health (NSCH) data has suggested that children with ASD have increased levels of poor neighborhood social capital, greater parental aggravation and difficulty coping, and lower levels of relationship satisfaction and mental health.[19] As children with ASD are less likely than other children with special health care needs to receive supportive services, such as occupational therapy and speech therapy,[20] health care-related factors, including forgone medical care, should also be examined. Using 2016–2017 NSCH data, previous studies have indicated health insurance may be a protective factor against overweight and obesity in a general population sample of children and adolescents (*i.e.*, most do not have ASD).[21] Understanding the social-ecological context for obesity will aid in addressing disparities in life course outcomes present in children and adolescents, and particularly for children and adolescents with ASD and co-occurring conditions as they transition to adult health care.[22]

There is a need to identify significant independent associations with obesity that could represent potential mediating pathways in the relationship between child and family level risk factors and obesity. The purpose of this study was to examine associations between child, family, community, and organizational risk factors and obesity and to explore whether there are any mediated effects of child and family risk indices in the relationship between community and organizational risk indices and obesity among children and adolescents aged 10–17 years, addressing ASD and co-occurring conditions, using 2017–2018 NSCH data.

Methods

This cross-sectional study used 2017–2018 data from the nationally representative NSCH with a reference population of noninstitutionalized children aged 0–17 years living in the United States.[23] Online surveys were completed by a parent or other adult caregiver with knowledge of the child's health and health care. The weighted response rates, which can approximate the proportion of the population for which data are available, were 37.4% and 43.1% for the 2017 and 2018 datasets, respectively.[24] For this study, observations for participants aged 10–17 years with and without ASD who had data available for BMI and ASD diagnosis were included, resulting in an unweighted sample size of 27,157 children and adolescents aged 10–17 years (911 children with current ASD diagnosis, weighted $n = 1,094,145$; 26,246 children without ASD; weighted $n = 32,082,679$). Missing data were treated with simultaneous imputation methods by the NSCH; race, sex, and ethnicity were imputed using hot-deck imputation, and household income was imputed using regression imputation methods.[25] Variables that were missing in error, that is, owing to respondent or system errors, were excluded from analyses. This study was reviewed by the University of South Florida Institutional Review Board and determined exempt.

Measures

Obesity. Using the BMI categories included in the NSCH dataset, obesity was coded as obesity vs. nonobesity (overweight, healthy weight, or underweight). The NSCH calculates BMI based on parent-reported height and weight ("What is this child's current height?" and "How much does this child currently weigh?") for the selected child and determines obesity status based on the CDC growth chart, as it defines BMI-for-age at 95th percentile or greater as obesity.[26]

Autism spectrum disorder. Children were classified as having ASD based on "yes" responses to the questions: (1) "Has a doctor or other health care provider ever told you that this child has Autism or Autism Spectrum Disorder (ASD)? (Including diagnoses of Asperger's Disorder or Pervasive Developmental Disorder (PDD)" and (2) "If yes, does (child) currently have the condition?" Participants were excluded from the analysis to minimize misclassification if the response to the first question was "yes" but the response to the second question was "no." Because question (2) is not based on the clinical evaluation, but rather based on the parents' self-report, we could not clearly verify whether the participants had a child with ASD or without ASD at the time of NSCH data collection.

Social-ecological risk indices. Four separate risk indices (child, family, community, and organizational) were developed based on chi-square tests for dichotomous variables and *t*-tests for continuous variables that indicated significant associations with obesity. Table 1 provides the characteristics used for each index. To allow equal weights for all variables in the indices, continuous variables were recoded on a 0–1 scale. The child risk index (0–6 points) included ASD diagnosis (current diagnosis vs. never diagnosed), co-occurring conditions [current diagnosis of one or more co-occurring conditions that were significantly associated with obesity (intellectual disability, learning disability, speech problems, developmental delay, depression, or anxiety) vs. never diagnosed with any co-occurring conditions], male sex (vs. female), Black race (vs. non-Black race), Hispanic ethnicity (vs. non-Hispanic ethnicity), and low household income (coded continuously from 0 to 1). Age, low birth weight, premature birth, and urbanicity were also examined and found not to be significantly associated with obesity.

The family risk index (0–16 points) included poor/average overall health of mother (excellent physical and mental health vs. one or both not excellent), poor/average overall health of father (excellent physical and mental health vs. one or both not excellent), household smoking (vs. no household smoking), working poor (vs. not working poor), food insufficiency ("We could always afford to eat good nutritious meals" vs. "We could always afford enough to eat but not always the kinds of food we should eat," "Sometime we could afford enough to eat," or "Often we could not afford enough to eat"),[27] family adversity (coded continuously from 0 to 1), parental aggravation (coded continuously from 0–1), lack of parent education (coded continuously from 0 to 1), and adverse childhood experiences (ACEs) count (coded continuously from 0 to 9). Based on the NSCH, "working poor" households have someone employed for 50 weeks in the past year and a total household income <100% federal poverty level (FPL). Family adversity was derived from four reverse-coded questions about family members talking about what to do when faced with problems, working together to solve problems, knowing they have strength to

draw on when faced with problems, and staying hopeful in difficult times when faced with problems. Parental aggravation was derived from three questions about parents feeling, during the past month: their child was harder than most to care for, their child did things that bothered them, and angry with their child. Lack of parent education was based on the "Highest education of adult in household" variable ("Less than high school," "High school degree or GED," "Some college or technical school," "College degree or higher"). ACEs were derived from a count of nine ACEs. Emotional support and lack of parent coping were also examined and found not to be significantly associated with obesity.

The community risk index (0–4 points) included four variables that were coded continuously from 0 to 1: lack of neighborhood support, lack of school safety, lack of neighborhood amenities, and neighborhood detracting elements. Lack of neighborhood support was derived from three reverse-coded questions about people in the neighborhood: helping each other out, watching out for each other's children, and knowing where to go for help. Lack of school safety was derived from reverse-coded Likert scale responses regarding parents perceiving that their children are safe at school. Lack of neighborhood amenities was derived from four questions about the neighborhood: not containing sidewalks or walking paths; not containing a park or playground area; not containing a recreation center, community center or Boys'/Girls' Club; not containing a library or bookmobile. Neighborhood detracting elements was derived from three questions about the neighborhood: having litter or garbage on sidewalks and streets, having poorly kept or dilapidated housing, and having broken windows or graffiti. Lack of neighborhood safety was also examined and found not to be significantly associated with obesity.

The organizational risk index (0–4 points) included: forgone care ("Yes" vs. "No" response to "During the past 12 months, was there any time when this child needed health care but it was not received?"), special education (currently receives special education or early intervention plan vs. does not currently receive special education or early intervention plan), special services (currently receives special services to meet child's developmental needs vs. does not currently receive special services to meet child's developmental needs), and lack of medical home (health care does not meet medical home criteria vs. health care meets medical home criteria), were all coded as dichotomous variables. The NSCH measures the presence of a medical home[28] based on 16 items from 5 domains: personal doctor or nurse, usual source for sick case, family-centered care, problems getting needed referrals, and effective care coordination when needed. Insurance was also examined and found not to be significantly associated with obesity.

Demographic characteristics. Age was measured as a numerical value from 10 to 17. Sex was a dichotomous variable including male and female. Race/ethnicity was a

Table 1. Prevalence of Child, Family, Community, and Organizational Characteristics in Adolescents 10–17 Years with and without Obesity

	Obesity				Without obesity				
	n	**Weighted n**	**%**	**n**	**Weighted n**	**%**	**Test statistic[a]**	**Degrees of freedom**	**p**
Child risk									
ASD diagnosis							12.68	26,001	0.0004**
Currently diagnosed with ASD	194	255,069	5.3	674	761,662	2.9			
Not diagnosed with ASD	3242	4,528,289	94.7	21,993	25,781,719	97.1			
Co-occurring conditions[b]							17.77	26,164	<0.0001***
Has one or more co-occurring conditions	977	1,118,209	23.3	4549	4,612,311	17.3			
Does not have any co-occurring conditions	2487	3,689,625	76.7	18,253	22,094,662	82.7			
Age, years							1.81	26,164	0.1789
10–13	1592	2,496,840	51.9	9802	13,125,508	50.9			
14–17	1872	2,310,994	48.1	13,000	13,581,465	49.1			
Gender							4.12	26,164	0.0424*
Male	2088	2,632,165	54.7	11,563	13,493,693	50.5			
Female	1376	2,175,668	45.3	11,239	13,213,280	49.5			
Race							24.03	26,164	<0.0001***
Black	378	981,974	20.4	1344	3,450,424	12.9			
Non-Black	3086	3,825,858	79.6	21,458	23,256,548	87.1			
Ethnicity							11.89	26,164	0.0006**
Hispanic	525	1,531,092	31.8	2418	6,525,655	24.4			
Non-Hispanic	2939	3,276,741	68.2	20,384	20,181,318	75.6			
Low birth weight							0.23	25,166	0.6303
Born with a low birth weight	265	443,917	9.7	1842	2,333,044	9.2			
Did not have a low birth weight	3043	4,123,045	90.3	20,118	23,144,385	90.8			
Premature birth							1.86	25,778	0.1725
Born premature	439	631,068	13.4	2525	3,075,886	11.7			
Not born premature	2973	4,092,318	86.6	19,943	23,266,240	88.3			
Urbanicity							0.90	26,164	0.3418
Lives in an urban area[c]	2352	4,012,560	83.5	15,366	22,556,139	84.5			
Does not live in an urban area	1112	795,273	16.5	7436	4,150,834	15.5			
Low household income[c]		0.54			0.41		−9.46	26,164	<0.0001***
Family risk									
Mother's health							47.27	23,140	<0.0001***
Mother has excellent physical and mental health	1503	1,961,968	49.5	13,519	14,864,679	64.0			
One or both of mother's physical and mental health is not excellent	1476	1,999,265	50.5	6744	8,363,643	36.0			
Father's health							24.88	20,025	<0.0001***

Table 1. Prevalence of Child, Family, Community, and Organizational Characteristics in Adolescents 10–17 Years with and without Obesity *continued*

	Obesity			Without obesity			Test statistic[a]	Degrees of freedom	p
	n	Weighted n	%	n	Weighted n	%			
Father has excellent physical and mental health	1304	1,618,920	55.8	12,140	13,218,412	67.3			
One or both of father's physical and mental health is not excellent	1067	1,282,682	44.2	5616	6,415,330	32.7			
Household smoking							33.82	25,795	<0.0001***
Someone in the household smokes	790	1,025,316	21.8	3165	3,742,693	14.3			
No one in the household smokes	2616	3,673,987	78.2	19,326	22,461,392	85.7			
Working poor							13.38	25,692	0.0003**
Working poor household	377	845,110	18.3	1658	3,275,018	12.6			
Not a working poor household	3012	3,785,084	81.7	20,747	22,740,356	87.4			
Food insufficiency							62.32	25,729	<0.0001***
Could always afford nutritious meals	1972	2,527,108	54.3	17,003	18,275,911	69.9			
Could not always afford nutritious meals	1425	2,130,303	45.7	5431	7,866,552	30.1			
Emotional support							−0.26	20,333	0.2851
Parents have emotional support	2616	3,357,648	71.0	18,073	19,251,184	73.2			
Parents do not have emotional support	800	1,373,477	29.0	4481	7,061,095	26.8			
Family adversity[c]		0.23			0.20		−2.16	25,512	0.031*
Lack of parent coping[c]		0.12			0.13		0.85	26,050	0.396
Parental aggravation[c]		0.19			0.18		2.48	25,785	0.013*
Lack of parent education[c]		0.43			0.30		−8.35	26,266	<0.0001***
ACE count[d]		1.35			0.97		−6.71	25,936	<0.0001***
Community risk									
Lack of neighborhood support[c]		0.28			0.25		−3.16	25,490	0.002**
Lack of school safety[c]		0.13			0.11		−2.75	25,534	0.006**
Lack of neighborhood amenities[c]		0.37			0.33		−2.98	25,432	0.003**
Lack of neighborhood safety[c]		0.86			0.87		1.18	25,776	0.0237*
Neighborhood detracting elements[c]		0.12			0.086		3.91	25,558	<0.0001***
Organizational risk									
Forgone care							8.17	26,089	0.0043**
Forgone health care	167	321,385	6.7	618	961,668	3.6			
No forgone health care	3287	4,442,913	93.3	22,119	25,623,161	96.4			
Special education							8.94	26,056	0.0028**
Receives special education or early intervention plan	512	609,216	12.7	2247	2,483,144	9.4			
Does not receive special education or early intervention plan	2935	4,169,375	87.3	20,464	24,073,543	90.6			
Special services							11.52	25,886	0.0007**
Receives special services	319	426,805	9.0	1285	1,520,893	5.8			
Does not receive special services	3114	4,317,723	91.0	21,270	24,860,583	94.2			

Table 1. Prevalence of Child, Family, Community, and Organizational Characteristics in Adolescents 10–17 Years with and without Obesity *continued*

	Obesity				Without obesity				
	n	Weighted *n*	%	*n*	Weighted *n*	%	Test statistic[a]	Degrees of freedom	p
Medical home							21.78	26,161	<0.0001***
Health care meets medical home criteria	1644	1,899,156	39.6	12,045	12,987,582	48.6			
Health care does not meet medical home criteria	1819	2,901,085	60.4	10,395	13,713,911	51.4			
Insurance							0.076	26,092	0.7829
Adequate health insurance	2206	3,046,823	63.8	14,764	17,084,812	64.3			
Not adequate health insurance	1245	1,729,705	36.2	7979	9,468,212	35.7			

Chi-square tests and *t*-tests were conducted to identify associations with obesity (vs. nonobesity).

[a]Design-based *F* statistic for chi-square tests and *t* statistic for *t*-tests.

[b]Intellectual disability, learning disability, speech problems, developmental delay, depression, or anxiety.

[c]Comparison of means for variables weighted on a 0–1 scale.

[d]Count of ACEs (0–9).

*$p < 0.05$; **$p < 0.01$; ***$p < 0.001$.

ACE, adverse childhood experience; ASD, autism spectrum disorder.

categorical variable including Hispanic; White, non-Hispanic; Black, non-Hispanic; and Multiracial/Other. Household income was a categorical variable including: 0%–99% FPL, 100%–199% FPL, 200%–399% FPL, and 400% FPL or greater. Household income refers to the combined annual income of all members in the household. FPL is the poverty threshold set forth by the US Census Bureau to estimate the number of Americans in poverty.

Statistical Analysis

Analyses were performed in STATA/SE 16.[29] Analyses were weighted to account for selection and nonresponse bias. An *a priori* conceptual model was designed based on the social-ecological framework (*i.e.*, having individual, interpersonal, community, and organizational levels) using risk indices and their corresponding indicators. The study sample was described for participants with and without ASD using frequency distributions and chi-square tests to identify bivariate associations for the development of risk indices and to describe demographic characteristics of children and adolescents with and without ASD. Variables that were not associated with the outcome of obesity were excluded from the risk indices and subsequent analyses.

Direct, indirect, and total effects were assessed using the mediation analysis framework proposed by Baron and Kenny.[30] A mediator is a variable that is part of the causal pathway between an exposure and outcome. Mediation quantifies the causal relationship between an exposure,

mediator, and outcome. The total effect of an exposure on the outcome can be broken down into the exposure's direct effect and indirect effect. The direct effect is the remaining effect from the exposure on the outcome after considering the mediator in the model. The indirect effect is the product of the effect of exposure on the mediator and the effect of the mediator on the outcome.

Based on analysis of direct effects of child, family, community, and organizational risk on obesity using separate models for each risk index, child and family level factors were identified as possible mediators, and community and organizational factors were identified as distal factors. Mediation analyses were conducted in a two-mediator model (X_1: community risk, X_2: organizational risk, M_1: child risk, M_2: family risk) for the dichotomous outcome (Y: obesity). Variables included in the risk indices are given in Table 1. As it was hypothesized that there would be a relationship between child and family risk indices, two plausible models were tested: one with and one without covariance between error variables for child and family risk indices. The model with covariance between the error variables was selected owing to a smaller Bayesian information criterion value. Path analyses were performed to examine indirect and total effects of community and organizational risk with child and family risk as mediators using generalized structural equation modeling (GSEM). Parameter estimation was performed with the maximum likelihood estimation method. The Bernoulli likelihood and logit link function were applied to the

Table 2. Demographic Characteristics in Adolescents 10–17 Years with and without Autism Spectrum Disorder

	ASD			Without ASD			F statistic	Degrees of freedom	p
	N	**Weighted *n***	%	*n*	**Weighted *n***	%	*F* statistic		*p*
Obesity							12.68	26,001	0.0004***
Obesity	194	255,069	25.1	3242	4,528,289	14.9			
Nonobesity	674	761,662	74.9	21,993	25,781,719	85.1			
Age, years							0.034	27,055	0.8548
10–13	397	557,998	51.0	11,500	16,105,515	50.2			
14–17	514	536,147	49.0	14,746	15,977,165	49.8			
Sex							26.62	27,055	<0.0001***
Male	710	823,791	75.3	13,388	16,130,743	50.3			
Female	201	270,354	24.7	12,858	15,951,936	49.7			
Race/ethnicity							2.052	27,055	0.1288
Hispanic	109	368,362	33.7	2997	8,220,393	25.6			
White, non-Hispanic	639	513,805	47.0	18,290	15,938,460	49.7			
Black, non-Hispanic	58	137,597	12.6	1785	4,736,533	14.8			
Multiracial/Other, non-Hispanic	105	74,382	6.8	3174	3,187,294	9.9			
Household income							3.52	27,055	0.0231*
0%–99% FPL	137	260,151	23.8	3067	6,403,188	20.0			
100%–199% FPL	194	335,184	30.6	4061	6,886,056	21.5			
200%–399% FPL	268	245,549	22.4	7794	8,676,192	27.0			
400% FPL or greater	312	253,260	23.2	11,324	10,117,243	31.5			

Chi-square tests were conducted to examine differences between adolescents with and without ASD.

$*p < 0.05$; $***p < 0.001$.

FPL, federal poverty level.

outcome variable of obesity. Unstandardized beta coefficients are reported owing to constraints of GSEM procedures in STATA.

Results

Obesity prevalence was 25.1% in children and adolescents with ASD and 14.9% in those without ASD ($p = 0.0004$). Children and adolescents with ASD were more likely to be male (75.3% vs. 50.3%, $p < 0.0001$), have lower household income (45.6% at 200% of the FPL or greater vs. 58.5%, $p = 0.0231$). Children and adolescents with ASD were 75.3% male, 47% non-Hispanic White, 33.7% Hispanic, 12.6% non-Hispanic Black, and 6.8% non-Hispanic Multiracial/Other. Children and adolescents without ASD were 50.3% male, 49.7% non-Hispanic White, 25.6% Hispanic, 14.8% non-Hispanic Black, and 9.9% non-Hispanic Multiracial/Other. Full demographic characteristics for children and adolescents with and without ASD are given in Table 2.

Direct Effects

The direct effects of child, family, community, and organizational risk indices on obesity were examined through separate models by social-ecological level and a full model with all four levels. In separate single index models, child, family, community, and organizational risk indices (all $p < 0.0001$) were associated with obesity. The direct effects of community and organizational risk were no longer significant, indicating child and family risk mediate most of the effects of community and organization risks on obesity. The results for direct effects are given in Table 3.

Indirect and Total Effects

When child risk and family risk were separately analyzed as mediators, the indirect effects of community risk ($\beta = 0.046$ and $\beta = 0.18$, respectively) and organizational risk ($\beta = 0.12$ and $\beta = 0.081$, respectively) were significant (all $p < 0.0001$). In the full model, the total effect of

Table 3. Direct Effects of Child, Family, Community, and Organizational Risk Indices on Obesity in Adolescents Aged 10–17 Years

	Separate models (path coefficient, p-value)	Full model (path coefficient, p-value)
Child risk	0.40, $p < 0.0001$***	0.26, $p < 0.0001$***
Family risk	0.18, $p < 0.0001$***	0.14, $p < 0.0001$***
Community risk	0.42, $p < 0.0001$***	0.082, $p = 0.343$
Organizational risk	0.29, $p < 0.0001$***	−0.016, $p = 0.827$

Path analyses were performed separately for each risk index and for the full model using generalized structural equation modeling.
***$p < 0.001$.

community risk on obesity was significant when family risk was analyzed as the mediator ($\beta = 0.26$, $p = 0.002$). The other three total effects examined were not significant. Indirect and total effects of community and organizational risk indices on obesity are given in Table 4, and the full model summarizing indirect and direct effects of community and organizational risk indices on obesity is given in Figure 1.

Discussion

This study investigated associations between ASD and other child, family, community/neighborhood, and orga-

Table 4. Indirect and Total Effects of Community and Organizational Risk Indices on Obesity in Adolescents Aged 10–17 Years in the Full Model with Child, Family, Community, and Organizational Risk Indices

	Indirect effect (coefficient, p-value)	Total effect (coefficient, p-value)
Child risk		
Community risk	0.046, $p < 0.0001$***	0.13, $p = 0.138$
Organizational risk	0.12, $p < 0.0001$***	0.11, $p = 0.113$
Family risk		
Community risk	0.18, $p < 0.0001$***	0.26, $p = 0.002$**
Organizational risk	0.081, $p < 0.0001$***	0.065, $p = 0.357$

Path analyses were performed to examine indirect and total effects of community and organizational risk with child and family risk as mediators using generalized structural equation modeling.
**$p < 0.01$; ***$p < 0.001$.

nizational risk factors and obesity and examined whether there were mediated effects of child and family risk indices in the associations between community and organizational risk indices and obesity among children and adolescents aged 10–17 years using 2017–2018 NSCH data. Several factors from child, family, community, and organizational levels were associated with obesity, and each of the risk indices was independently associated with obesity, indicating multilevel obesity risk factors in addition to ASD and co-occurring conditions. Although the indirect effects of community and organizational risk were significant with child and family risk as mediators, total effect was only significant for the relationship between community risk and obesity, mediated by family risk.

The results suggested that community risk may be a strong predictor of obesity ($\beta = 0.26$), mediated by family risk. These factors include important indicators such as neighborhood support, safety, amenities, and distracting elements, as well as school safety. As ASD was included in the child risk index along with co-occurring conditions, gender, race, ethnicity, and income, the finding that the relationship between community risk and obesity may be mediated by family risk is applicable to all children and adolescents. The community risk index may be a strong predictor for obesity owing to the inclusion of both school and neighborhood components.[31,32] Previous research has also suggested that community factors, including neighborhood characteristics, parent perceptions of neighborhood characteristics,[11] and the school environment[33] were associated with children's overweight/obesity status.

Although the organizational risk index was associated with obesity in a single index model ($p < 0.0001$), and the indirect effects were significant when both child and family risk indices were assessed as mediators, the total effects of organizational risk were not significant with either mediator. As only four variables (forgone care, special education, special services, and lack of medical home) were included in the organizational risk index, there may be a need for additional variables related to health care and service utilization. Among children and adolescents with ASD, unmet care needs may include information on services, family support, and respite care.[34] In the general population, prior research has found associations between health care access variables, such as having a usual source of care provider, well-child visits, and preventative dental use, and obesity among children,[35] but there is a lack of research using multivariable risk indices or latent variables to investigate health care or other organizational level risk factors or obesity among children and adolescents with or without ASD.

This study was limited by the cross-sectional methodology of the NSCH. There were no data on age of onset for the obesity outcome, so directionality of associations cannot be determined. Moreover, national-level data for children and adolescents with ASD or nationally representative data with ASD-specific weights are needed to conduct mediation analyses in samples of children and
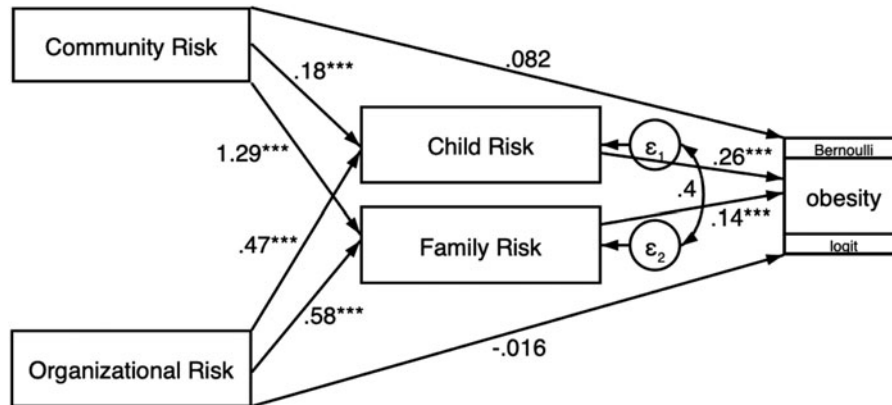
Figure 1. Full mediation model including child, family, community, and organizational risk indices. ^aValues represent unstandardized beta coefficients for indirect and direct effects. ***$p < 0.001$.

adolescents with ASD. In addition, risk indices for this study were created by applying weights to differences in scales such that continuous variables were coded 0–1.

Future research may improve upon this methodology by taking into account regression coefficients or otherwise standardizing values. This study was further limited by the fact that our risk indices included general variables for each level of the SEM model. Future analyses may define more specific family-level risk indices including variables such as family screen time and family eating meals together. Furthermore, as the study relies on self-report for all measures, there may be information bias, particularly for key variables including ASD diagnosis and BMI.

Nevertheless, this research provides insight into pathways for obesity risk in children and adolescents aged 10–17 years by applying the SEM to understand ASD and other risk factors using a large, nationally representative dataset. Twenty-five risk factors, including ASD, were included in four separate risk indices. Our findings buttress past research by elucidating pathways between these risk indices and including ASD and co-occurring conditions. A conceptual model will aid in directing coordinated care among health care providers by delineating evidence-based interaction among multiple levels of the SEM and potential pathways for intervention.

The pathways involving the child risk index highlight the significance of ASD in obesity risk, whereas the pathways involving the family risk index point to the importance of family, community, and organizational risk factors among all children and adolescents. The community risk index in particular emerged as a strong predictor of obesity. As the family risk index included nine variables (mother's health, father's health, household smoking, working poor, food insufficiency, family adversity, parental aggravation, lack of parent education, and count of ACEs), future research should investigate how these factors work together to influence obesity risk among children and adolescents.

The findings of this study may help to identify leverage points for advancing best practices to improve the physical and behavioral health of children and adolescents with and without ASD. For example, parents and families may support their children's healthy lifestyle behaviors through positive role modeling, and providers may encourage family involvement in promoting these behaviors for children and adolescents. Moreover, community stakeholders may be identified to help create healthy school and neighborhood environments. Investing in and increasing the availability of inclusive sports and recreation programs for children and adolescents may also be leveraged. Further research on social-ecological risk factors for obesity is needed to inform children and adolescents, parents, care providers, and policy makers alike[36] on key pathways for action. The risk indices used in this study can be used as a guiding framework for research and practice. Future research should investigate and define community and organizational level risk factors among children and adolescents, and obesity prevention interventions should target the community environment, including school and neighborhood safety and access to nutrient-dense food and physical activity opportunities in children and adolescents with and without ASD. Future studies may also include behavioral variables that have been established in the literature as obesity risk factors, such as physical activity, for a more comprehensive social-ecological analysis.

Disclaimer

This information or content and conclusions are those of the author and should not be construed as the official position or policy of, nor should any endorsements be inferred by Health Resources and Services Administration (HRSA), Health and Human Services (HHS), or the U.S. Government.

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