



DOI: 10.4274/jcrpe.galenos.2026.2025-12-23

J Clin Res Pediatr Endocrinol 2026;18(3):488-497

Early Childhood Obesity: Multifactorial Influences with a Prominent Familial Contribution

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Cite this article as: Akbeyaz İH, Akın Y, Akbeyaz BB, Abalı S. Early childhood obesity: multifactorial influences with a prominent familial contribution. J Clin Res Pediatr Endocrinol. [Epub Ahead of Print] 2026;18(3):488-497

ABSTRACT

Objective: Early childhood obesity is an increasing public health problem with long-term consequences. Identifying risk factors is essential for effective preventative strategies. To evaluate prenatal, perinatal, and postnatal influences including: breastfeeding and nutritional practices; lifestyle and daily habits; household, sociocultural, and economic conditions; and parental anthropometric characteristics to better elucidate the determinants of obesity in early childhood.

Methods: This cross-sectional study included children aged 12-60 months, divided into children with obesity and non-obese controls. Standardized anthropometric assessments were performed in all participants. Parents completed a structured questionnaire capturing perinatal characteristics, infant feeding practices, dietary intake, medical history, lifestyle behaviors, and household-level factors.

Results: A total of 210 children (113 girls; 53.8%) were included, comprising 106 obese and 104 non-obese individuals, with comparable age between groups (3.4 ± 1.3 vs. 3.3 ± 1.3 years). Both maternal and paternal body mass index (BMI) values were significantly higher in the obese group, and maternal employment (27.4% vs. 5.8%) and maternal obesity (42.5% vs. 21.2%) were more prevalent. Maternal smoking during pregnancy (23.6% vs. 7.7%) and gestational diabetes (22.6% vs. 9.6%) were also more frequent. Feeding practices and current dietary patterns did not differ significantly between groups. In multivariable analysis, maternal employment (aOR 3.74), sibling obesity (aOR 2.21), and extended family obesity burden (aOR 1.19) independently predicted childhood obesity.

Conclusion: Familial obesity burden, including parents, siblings and extended family members, showed the strongest associations with obesity, highlighting the need for early identification of high-risk children. The implementation of family-centered and family-encompassing preventive strategies targeting physical activity, diet, and daily routines should be considered in these high-risk children.

Keywords: Obesity, early childhood, familial

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Conflict of interest: None declared

Received: 25.12.2025 **Accepted:** 14.03.2026 **Epub:** 16.03.2026 **Publication Date:** 08.09.2026



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What is already known on this topic?

Globally, the prevalence of childhood overweight has risen steadily over the past three decades, with approximately 35 million children under five years of age classified as overweight by 2024.

Childhood obesity has a multifactorial etiology, involving the interplay of genetic susceptibility with perinatal, nutritional, socioeconomic, environmental, familial, and lifestyle factors.

What this study adds?

Familial obesity burden, that is obesity in parents, siblings and other family members, emerged as a prominent etiological factor, highlighting the importance of early recognition of genetically and environmentally high-risk children. The implementation of family-centered and family-encompassing preventive strategies targeting physical activity, diet, and daily routines should be considered in these high-risk children. Sibling obesity and a greater burden of obesity among extended family members were independently associated with early childhood obesity. These findings highlight the importance of early identification of children at increased familial risk and family-centered preventive strategies targeting physical activity, diet, and daily routines.

Introduction

Childhood obesity in the preschool period has emerged as a major public health concern due to its strong tendency to persist into later childhood and adulthood, leading to long-term cardiometabolic and psychosocial consequences (1,2,3). Data from Türkiye reports an obesity prevalence of about 5% among children aged 0-84 months (4). Studies show that approximately one-third of obese preschoolers and nearly half of overweight school-aged children remain obese in adulthood (5).

The etiology of childhood obesity is multifactorial, encompassing genetic, perinatal, nutritional, socioeconomic, environmental, familial, and lifestyle determinants (1,2,6,7). Shifts in dietary habits, reduced physical activity, increased screen time, and broader lifestyle changes increasingly contribute to excess weight gain in young children (1,2,5). In early childhood, family structure, caregiving patterns, and shared household behaviors play a particularly influential role, as children are largely dependent on their immediate environment for nutrition, physical activity, and daily routines (2,3,5). Genetic susceptibility also plays a significant role, with studies indicating that more than half of the variance in obesity risk is attributable to heritable factors (6,7). Monogenic forms typically present with rapid, early weight gain and hyperphagia, and should be particularly considered in children younger than five years or when syndromic features are present (6,7).

Beyond genetic predisposition, the “first 1000 days” constitute a critical window in which early-life exposures, including maternal pre-pregnancy body mass index (BMI), gestational weight gain, and feeding practices, shape later obesity risk (8). Given this broad etiological spectrum, a comprehensive evaluation of modifiable early childhood risk factors will be essential for effective prevention efforts.

The aim of the present study was to evaluate prenatal, perinatal, and postnatal influences; breastfeeding and nutritional practices, lifestyle and daily habits; household, sociocultural, and economic conditions; and parental anthropometric characteristics to better elucidate the determinants of obesity in early childhood.

Methods

Study Design and Participants

This cross-sectional, case-control study included children aged 12-60 months who presented to the outpatient clinic over a six-month period. Children were classified as obese or non-obese controls according to World Health Organization BMI and weight-for-height standard deviation scores (SDS) criteria (BMI SDS $>+2.0$ and/or weight-for-height SDS $>+2.0$) (1,9). Children with chronic illness or on weight-affecting medications were excluded.

Written informed consent was obtained from parents or legal guardians. Ethical approval was obtained from the University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Scientific Research Evaluation and Support Board on 29 January 2016, under application number 89513.307/1009/531 and decision number 112.

Data Collection

Standardized anthropometric measurements of all patients were performed by the principal investigator with an infantometer and a stadiometer, both sensitive to 0.1 cm. Weight was measured using an electronic scale (Seca GmbH, Hamburg, Germany) (sensitive to 5 g). The BMI of each patient was calculated as weight (kg) divided by height (m) squared (kg/m^2). SDS for all anthropometric measurements were calculated according to Turkish reference standards (9,10,11). Children with a BMI $\geq+2$

SDS were classified as obese. Weight-for-height was calculated as $100 \times [\text{patient's weight (kg)} / \text{ideal weight at the 50}^{\text{th}} \text{ percentile for the child's length-age (kg)}]$, and a weight-for-height value $>120\%$ was defined as obesity (12). Waist circumference SDS was derived from reference data for Turkish children (13). Maternal and paternal weights and heights were measured and BMI values were recorded during outpatient visits. Data were collected using a structured questionnaire administered by the investigator to parents. The survey captured information across several domains, including demographic characteristics, perinatal history, feeding practices, medical history, lifestyle behaviors, and socioeconomic and household factors.

Perinatal information included maternal weight gain, smoking during pregnancy, gestational diabetes mellitus (GDM), delivery mode, gestational age, birth weight SDS, Neonatal Intensive Care Unit (NICU) admission, phototherapy, and neonatal hypoglycemia. Feeding and nutritional data covered exclusive and total breastfeeding duration, infant formula use, vitamin D supplementation, and the child's intake of sugary beverages, fast food, ultra-processed foods, packaged snacks, and high-carbohydrate foods. Medical history included infection-related hospitalizations (>24 hours), antibiotic use, nebulizer therapy usage, and vaccination status. Socioeconomic and household characteristics assessed household income, parental education, maternal employment, primary caregiver, passive smoking exposure, family history of obesity, and household size. Lifestyle factors included daycare attendance, outdoor play or physical activity, child temperament and habits and daily screen time.

Statistical Analysis

Statistical analyses were performed using NCSS 2007 and SPSS, version 25.0 (IBM Inc., Armonk, NY, USA). Group comparisons used independent t-tests, Mann-Whitney U tests, and chi-square tests as appropriate. The dependent variable was obesity status (obese vs. control). Variables associated with obesity at $p < 0.2$ in univariate analyses were entered into a multivariable logistic regression model using a backward stepwise method. Results

are expressed as adjusted odds ratios (aOR) with 95% confidence intervals (CI), and $p < 0.05$ was considered statistically significant.

Results

Of the study included 210 children, comprising 106 (50.5%) obese and 104 non-obese participants. The two groups were comparable with respect to age (3.4 ± 1.3 vs. 3.3 ± 1.3 years, $p = 0.398$) and gender (girls comprised 53.8% in both groups, $p = 0.992$). All anthropometric indices, including height SDS, weight SDS, BMI SDS, weight-for-height (%), weight-for-height SDS, and waist circumference SDS, are presented in Table 1.

Familial Characteristics

Familial characteristics are summarized in Table 2. The annual per-capita household income was significantly higher in the obese group (2502.5 ± 1533.4 vs 1943.7 ± 1317.5 USD, $p = 0.005$), whereas household size was significantly smaller (4.1 ± 1.1 vs 4.9 ± 1.6 persons, $p < 0.001$). Passive smoking exposure did not differ between groups ($p = 0.231$). Parents of obese children had higher educational attainment. Fathers had higher education levels ($p = 0.028$), mothers also had higher education levels ($p = 0.022$) and maternal employment was substantially more frequent in the obese group (27.4% vs. 5.8%, $p < 0.001$).

A family history of obesity emerged as a prominent determinant of childhood obesity. Parents of obese children exhibited significantly higher BMI values, with both paternal BMI ($p = 0.023$) and maternal BMI ($p = 0.009$) exceeding those observed in the control group. Consistently, maternal obesity was markedly more prevalent among obese children compared with controls (42.5% vs. 21.2%, $p = 0.002$) although paternal obesity rates did not differ between groups ($p = 0.13$). Furthermore, sibling obesity was significantly more frequent in the obese group (22.6% vs. 7.7%, $p = 0.005$), and the number of obese individuals in the extended family (excluding parents and siblings) was also significantly higher ($p < 0.001$), indicating a strong familial clustering of obesity (Table 2).

Table 1. Demographic and anthropometric characteristics

	Obese group	Control group	p value
Age, years	3.4 ± 1.3	3.3 ± 1.3	0.398*
Female n (%)	57 (53.8%)	56 (53.8%)	0.992 [#]
Height SDS	1.2 ± 1.1	0.2 ± 0.8	$<0.001^+$
Weight SDS	3.3 ± 1.1	0.1 ± 0.8	$<0.001^+$
Body mass index SDS	3.6 ± 1.0	0.0 ± 1.0	$<0.001^+$
Weight-for-Height (%)	141.9 ± 17.0	100.0 ± 8.1	$<0.001^+$
Weight-for-Height SDS	3.6 ± 1.0	-0.1 ± 0.9	$<0.001^+$
Waist circumference SDS	4.6 ± 2.6	0.3 ± 1.5	$<0.001^+$

*Independent t-test, [#]Chi-square test, ⁺Mann-Whitney U test
SDS: standard deviation score

Table 2. Familial characteristics

	Obese Group	Control Group	p value
Annual per capita household income, USD	2502.5±1533.4	1943.7±1317.5	0.005 ⁺
Household size, persons	4.1±1.1	4.9±1.6	<0.001 ⁺
Passive smoking exposure n (%)			
None	24 (22.6)	32 (30.8)	0.231 [#]
Indoor smoking	27 (25.5)	18 (17.3)	
Outdoor smoking	55 (51.9)	55 (51.9)	
Father's education, years	9.8±2.9	8.9±3.0	0.028 ⁺
Father's BMI	28.0±4.5	26.7±3.5	0.023 ⁺
Father with obesity n (%)	31 (29.2)	20 (19.2)	0.126 [#]
Father with diabetes n (%)	4 (3.8)	0 (0.0)	0.135 [#]
Mother's education years	9.5±3.3	8.4±3.2	0.022 ⁺
Maternal employment n (%)	29 (27.4)	6 (5.8)	<0.001 [#]
Mother's BMI	28.6±6.1	26.8±3.6	0.009 ⁺
Mother with obesity n (%)	45 (42.5)	22 (21.2)	0.002 [#]
Mother with diabetes n (%)	9 (8.5)	2 (1.9)	0.068 [#]
Sibling with obesity n (%)	24 (22.6)	8 (7.7)	0.005 [#]
Number of obese individuals in extended family (except parents and sibling) Median (IQR)	2.7±2.0 2 (1-5)	1.6±1.9 1 (0-3)	<0.001 [*]

⁺Independent t-test, ^{*}Mann-Whitney U test, [#]Chi-square test
USD: United States Dollars; BMI: body mass index, IQR: Interquartile range

Perinatal and Birth History

Perinatal and birth characteristics are presented in Table 3. Maternal gestational weight gain did not differ between groups ($p=0.254$). In contrast, maternal smoking during pregnancy was significantly more frequent in the obese group (23.6% vs. 7.7%, $p=0.003$), and the prevalence of GDM was also higher among mothers of obese children (22.6% vs. 9.6%, $p=0.018$).

Delivery mode, prematurity rates, and gestational age were comparable between groups. Although birth weight SDS was significantly higher in the obese group ($p=0.0037$), weight-for-gestational-age categories (small for gestational age, appropriate for gestational age, large for gestational age) did not differ significantly. Rates of NICU admission and phototherapy were similar but neonatal hypoglycemia was more frequently observed in obese children (11.3% vs. 1.9%, $p=0.014$).

Nutritional History and Lifestyle Factors

The proportion of children who had ever been exclusively breastfed did not differ significantly between groups ($p=0.090$). The duration of exclusive breastfeeding and total breastfeeding duration were slightly shorter in the obese group, although these differences were not statistically significant ($p=0.074$ and $p=0.080$, respectively). Infant formula use was significantly more commonly among obese children (72.6% vs. 57.7%, $p=0.033$), while the age at first formula introduction was similar between groups.

Current dietary habits, including ultra-processed food intake, fast-food consumption frequency, number of weekly fast-food meals, and high-carbohydrate food intake, did not differ significantly between groups.

Regarding medical history, hospitalization due to infection tended to be more frequent among obese children, although was not significant ($p=0.058$). No significant differences were observed in doctor visits due to infection, nebulizer therapy, vaccination status, or infantile colic severity.

Lifestyle factors showed notable differences. The distribution of primary caregivers differed significantly between groups ($p<0.001$), with mothers serving less frequently as primary caregivers in the obese group. Daycare attendance was significantly higher among obese children (27.4% vs. 11.5%, $p=0.004$), and physical activity was reported less frequently in this group (86.8% vs. 96.2%, $p=0.031$). Daily screen time did not differ between groups ($p=0.871$) (Table 4).

Multivariable Logistic Regression Analysis

To identify independent predictors of childhood obesity, a multivariable logistic regression model was constructed (Table 5). In the adjusted analysis, maternal employment (aOR 3.74, 95% CI 1.29-10.84, $p=0.015$), sibling obesity (aOR 2.21, 95% CI 1.08-4.51, $p=0.030$), and history of extended family members with obesity (aOR 1.19, 95% CI 1.01-1.41, $p=0.038$) emerged

as independent predictors of childhood obesity, indicating both intergenerational and household-level influences. Other familial, perinatal, and lifestyle variables were not independently associated with obesity in the multivariable model (Figure 1).

Discussion

This study provides insight into the determinants of obesity specifically during early childhood, a developmental period in which family environment and caregiving patterns may exert a stronger influence than individual lifestyle choices. Children aged 12-60 months were evaluated to examine the associations of infant feeding and nutritional practices, socioeconomic and household characteristics; lifestyle behaviors and daily habits; and familial obesity burden with early childhood obesity.

Several studies conducted in Türkiye have examined these risk factors. In a study by Kondolot et al. (14) investigating obesity risk among school-aged children, higher socioeconomic status, irregular eating patterns, insufficient physical activity, and a family history of obesity were identified as key contributors. These results highlight the pivotal role of familial obesity burden, socioeconomic context, and modifiable lifestyle-related factors in the development of obesity, beginning as early as childhood, and our results are consistent with this (14).

Familial and Environmental Characteristics

The association between socioeconomic status and childhood obesity is complex and may differ according to a country's level of economic development (15,16,17). In Türkiye, higher socioeconomic status has been associated with an increased

risk of childhood obesity in several studies (14,15,16,17,18,19), whereas an inverse socioeconomic gradient, with a greater burden of obesity among socioeconomically disadvantaged groups, is more commonly observed in high-income countries.

Parental educational attainment and employment status may also influence childhood obesity risk. In the present study, both maternal and paternal education levels were significantly higher in the obese group. In addition, maternal employment was more frequent among families of obese children. This observation is consistent with previous reports indicating a higher prevalence of childhood obesity in households where primary caregivers are engaged in full-time employment. Importantly, maternal employment should not be interpreted as a causal or maternal-specific risk factor. Rather, it likely reflects broader family dynamics, including contributing to higher family socioeconomic status, caregiving arrangements, time constraints, and reliance on convenience foods, all of which may influence early childhood eating behaviors and activity patterns (18,20,21,22).

Perinatal and Birth History

Maternal factors before and during pregnancy are consistently highlighted as important determinants of childhood obesity risk (23). GDM is a significant risk factor for childhood obesity. A study found that GDM doubles the probability of childhood overweight and obesity at ages 2-5 years (24). In our cohort, GDM was more frequent in the obese group in univariate analyses; however, GDM did not remain an independent predictor in multivariate models.

Table 3. Perinatal and birth history

	Obese group	Control group	p value
Maternal weight gain kg	14.96±7.50	13.88±6.10	0.254*
Smoking in pregnancy n (%)	25 (23.6)	8 (7.7)	0.003 [#]
Gestational diabetes n (%)	24 (22.6)	10 (9.6)	0.018 ⁺
Delivery mode n (%) Cesarean section Spontaneous vaginal	72 (67.9) 34 (32.1)	62 (59.6) 42 (40.4)	0.267 [#]
Prematurity n (%)	11 (10.4)	8 (7.7)	0.662 [#]
Gestational age, weeks	38.7±2.2	38.9±1.3	0.059*
Weight for gestational age n (%) SGA AGA LGA	6 (5.7) 75 (70.8) 25 (23.6)	8 (7.7) 80 (76.9) 16 (15.4)	0.301 [#]
Birth weight SDS	0.50±1.45	-0.05±1.26	0.004*
NICU admission n (%)	29 (27.4)	18 (17.3)	0.114 [#]
Phototherapy n (%)	24 (22.6)	20 (19.2)	0.662 [#]
Neonatal hypoglycemia n (%)	12 (11.3)	2 (1.9)	0.014 [#]

*Independent t-test, ⁺Mann-Whitney U test, [#]Chi-square test

SGA: small for gestational age; AGA: appropriate for gestational age; LGA: large for gestational age

Maternal smoking during pregnancy was significantly found to be more common in the obese group in univariate analyses. This is consistent with evidence showing that maternal smoking during pregnancy increases the risk of childhood overweight, independent of maternal pre-pregnancy BMI and genetic predisposition (25).

Children born large or small for gestational age, as well as those born preterm, have an increased risk of later obesity, particularly in the presence of maternal factors or rapid postnatal catch-up growth. Consistent with this, higher birth weight SDS was

associated with obesity in univariate analyses in our study, although, once again, it did not remain an independent predictor in multivariable analysis (26,27,28,29,30).

Nutritional History and Dietary Intake

Breastfeeding is widely recognized as a potent protective mechanism against early-onset obesity (31,32). Increased duration of breastfeeding is often linked to a decreased risk of overweight later in life (33,34). Total breastfeeding duration tended to be lower in the obese group, although the difference

Table 4. Nutrition and lifestyle characteristics

	Obese group	Control group	p value
Infants exclusively breastfed at any time, n (%)	54 (50.9)	66 (63.5)	0.090 [#]
Duration of exclusive breastfeeding, months	3.74±2.72	4.38±2.45	0.074 [*]
Total duration of breastfeeding, months	13.65±8.89	15.78±8.66	0.080 [*]
Use of infant formula, n (%)	77 (72.6)	60 (57.7)	0.033[#]
Age at first infant formula introduction, months	3.52±3.84	4.05±4.50	0.468 [*]
Current nutrition and dietary practices			
Ultra processed food intake (portions per day)			0.739 [#]
None	9 (8.5)	6 (5.8)	
Rare-1/day	25 (23.6)	26 (25.0)	
≥2/day	72 (67.9)	72 (69.2)	
Fast food consumption n (%)			0.493 [#]
None	43 (40.6)	16 (32.7)	
Occasional	21 (19.8)	66 (23.1)	
Frequent	42 (39.6)	22 (44.2)	
Fast-food meals per week	0.89±1.23	0.92±1.26	0.874 [*]
High-carbohydrate foods (portions per day)	1.94±0.94	1.90±0.91	0.758 [*]
Medical history			
History of hospitalization due to infection n (%)	25 (23.6)	38 (36.5)	0.058 [#]
Doctor visits due to infection n/year	5.47±4.07	5.79±4.12	0.576 [*]
Any nebulizer therapy n (%)			0.658 [#]
None	42 (39.6)	42 (40.4)	
At hospital	32 (30.2)	36 (34.6)	
At home	32 (30.2)	26 (25.0)	
Vaccination status (schedule-compatible) n (%)	103 (97.2)	98 (94.2)	0.477 [#]
Infantile colic history n (%)			0.392 [#]
None	38 (35.8)	46 (44.2)	
Mild	41 (38.7)	32 (30.8)	
Severe	27 (25.5)	26 (25.0)	
Lifestyle factors			
Primary caregiver n (%)			<0.001 [#]
Mother	65 (61.3)	88 (84.6)	
Nanny	10 (9.4)	2 (1.9)	
Grandmother	29 (27.4)	12 (11.5)	
Single parent	2 (1.9)	0 (0.0)	
Daycare attendance n (%)	29 (27.4)	12 (11.5)	0.004[#]
Physically active n (%)	92 (86.8)	100 (96.2)	0.031[#]
Screen time, hours per day	2.94±2.15	2.99±2.03	0.871 [*]

^{*}Independent t-test, [#]Mann-Whitney U test, [#]Chi-square test

^{*}Independent t-test, [#]Mann-Whitney U test, [#]Chi-square test

Table 5. Multivariable logistic regression analysis of factors associated with childhood obesity

Variable	Adjusted OR	95% CI	p value
Maternal employment	3.74	1.29-10.84	0.015
Neonatal hypoglycemia	2.91	0.54-15.6	0.212
Sibling obesity	2.21	1.08-4.51	0.030
Gestational diabetes	2.11	0.83-5.36	0.115
Mother with diabetes	2.01	0.31-13.1	0.460
Daycare attendance	1.72	0.69-4.30	0.243
Maternal obesity	1.62	0.77-3.43	0.208
NICU admission	1.29	0.55-3.05	0.560
Extended family history of obesity	1.19	1.01-1.41	0.038
Mother's BMI, kg/m ²	1.04	0.97-1.12	0.287
Paternal obesity	1.09	0.64-1.87	0.756
Father's education, years	1.04	0.91-1.18	0.561
Mother's education, years	1.03	0.92-1.16	0.621
Father's BMI, kg/m ²	1.02	0.94-1.11	0.611
Household size, persons	0.80	0.60-1.06	0.116

BMI: body mass index, OR: odds ratio, CI: confidence interval, NICU: neonatal intensive care unit

was not significant, a trend consistent with evidence supporting a protective role of breastfeeding against childhood obesity. However, no significant associations were observed with exclusive breastfeeding or its duration (35,36).

The association between infant formula feeding and childhood obesity remains controversial. Early formula use has been linked to accelerated infant weight gain (37,38). Consistent with this, formula use was significantly more frequent (73% vs 58%) among obese children in our cohort.

In our cohort, fast-food consumption, weekly fast-food meal frequency, and ultra-processed food intake did not differ significantly between groups. Nevertheless, previous studies have consistently reported that higher consumption of ultra-processed products, such as packaged snacks, confectionery, and sugar-sweetened beverages, has been associated with increased obesity risk and unfavorable trajectories in BMI, fat mass index, and waist circumference throughout childhood (39,40). These foods are typically energy-dense and nutrient-poor, often displacing healthier dietary options and thereby promoting excess weight gain (41).

Medical History and Lifestyle Factors

A higher proportion of children in the obese group had a history of hospitalization due to infection compared with controls, although this difference was not statistically significant. Similarly, no significant between-group differences were observed in

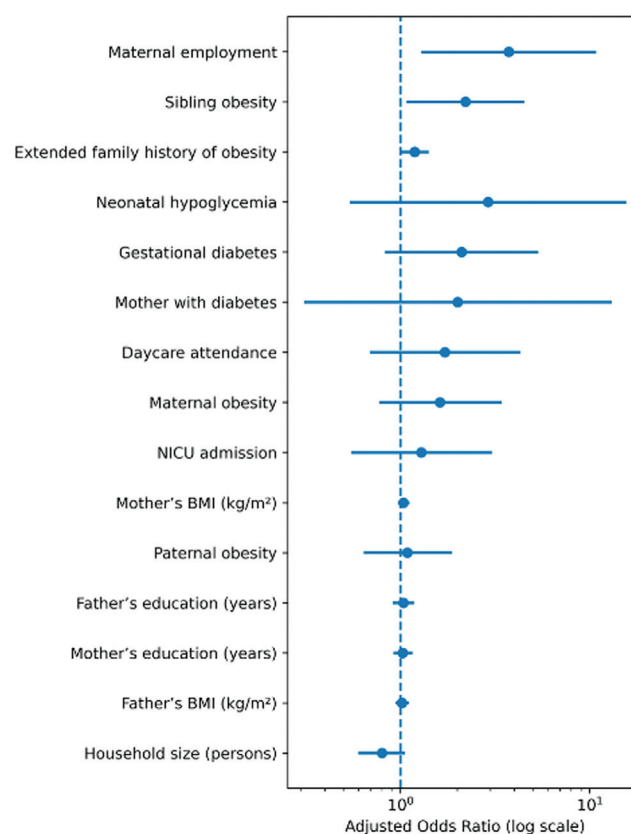


Figure 1. Multivariable logistic regression analysis of factors associated with childhood obesity. Forest plot showing adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for factors associated with childhood obesity. Maternal employment, sibling obesity, and extended family history of obesity were independently associated with an increased risk of childhood obesity. The vertical reference line indicates an OR of 1.0. Variables with confidence intervals not crossing 1.0 are considered statistically significant. NICU: neonatal intensive care unit, BMI: body mass index

nebulizer therapy, vaccination status, or infantile colic severity. These findings suggest that early-life clinical exposures play a limited role in obesity development, consistent with previous studies reporting no clear association with later adiposity (42,43,44).

Children in the obese group were more often cared for by non-parental caregivers and attended daycare more frequently, consistent with evidence linking extra-familial childcare arrangements to childhood obesity. This association likely reflects broader contextual factors rather than caregiver-specific effects, and its impact appears heterogeneous across caregiving settings (45,46).

Physical activity is a well-established protective factor against childhood obesity (14). In our study, parent-reported physical activity was significantly lower among obese children compared with controls. Consistent with this finding, objective assessments

in the literature, including accelerometry-based studies, have demonstrated strong associations between lower activity levels and increased adiposity in preschool-aged children (47).

Screen time exposure, including use of mobile phones, tablets, and television, did not differ significantly between groups in our study. Although no association with screen time was observed in this study, previous literature suggests that excessive sedentary media exposure may contribute to obesity risk as children grow older (48,49).

Eating behaviors established in early childhood, beginning around three years of age and consolidated by school entry, are thought to confer a cumulative risk for later obesity, highlighting the importance of early-life interventions (50). In contrast, our findings indicated that early childhood obesity was more strongly associated with familial obesity burden than with lifestyle-related factors. In this age group, the home food environment likely represents the predominant dietary exposure shared across family members. Alternatively, these observations may suggest that genetic determinants exert a relatively greater influence on obesity development before five years of age, or a complex combination of genetic and social factors may be at play. Overall, our findings suggest that in early childhood, familial obesity burden may outweigh individual lifestyle behaviors as a determinant of obesity, underscoring the importance of family-centered preventive strategies initiated early in life.

Study Limitations

This study has several limitations that should be considered when interpreting the findings. First, physical activity and screen time were assessed using parent-reported measures rather than objective tools such as accelerometers or digital tracking devices, which may have attenuated true associations. Second, several potentially relevant factors, including parental dietary patterns, detailed household food environment, sleep duration and quality, and parental mental health, were not assessed and may have influenced obesity risk.

Although familial clustering of obesity was a prominent finding, the absence of genetic or polygenic risk analyses limits our ability to disentangle shared genetic susceptibility from shared environmental influences. Therefore, familial aggregation observed in this study likely reflects a combination of genetic predisposition and common household behaviors rather than a predominantly genetic effect.

Finally, the single-center, metropolitan setting and sociocultural characteristics of the study population may limit the generalizability of our findings to rural areas or populations with different socioeconomic or cultural backgrounds. These limitations highlight the need for future multicenter studies

incorporating objective behavioral measures and genetic data.

Conclusion

Our findings were consistent with the hypothesis that early childhood obesity arises from the interaction between familial predisposition and modifiable environmental factors. Although multiple variables were associated with obesity in univariate analyses, maternal employment and a stronger familial burden of obesity were associated with substantially increased odds of obesity in this metropolitan Turkish cohort. Thus, preventive strategies should include early identification of high-risk children/families while targeting modifiable behaviors, including physical activity, dietary patterns, and household routines.

Ethics

Ethics Committee Approval: Ethical approval was obtained from the University of Health Sciences Türkiye, Kartal Dr. Lütfi Kırdar City Hospital, Scientific Research Evaluation and Support Board (approval no.: 112, date: 29.01.2016).

Informed Consent: Written informed consent was obtained from parents or legal guardians.

Acknowledgements

We would like to thank the children and their parents for their participation.

Authorship Contributions

Surgical and Medical Practices: İsmail Hakkı Akbeyaz, Saygın Abalı, Concept: İsmail Hakkı Akbeyaz, Yasemin Akın, Design: İsmail Hakkı Akbeyaz, Saygın Abalı, Data Collection or Processing: İsmail Hakkı Akbeyaz, Berkin Berk Akbeyaz, Saygın Abalı, Analysis or Interpretation: İsmail Hakkı Akbeyaz, Berkin Berk Akbeyaz, Saygın Abalı, Literature Search: İsmail Hakkı Akbeyaz, Yasemin Akın, Saygın Abalı, Writing: İsmail Hakkı Akbeyaz, Saygın Abalı.

Financial Disclosure: No funding was received in the publication of this article.

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