

Family lifestyle patterns, obesity, and metabolic risk in children and adolescents

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ABSTRACT

Objective: Family-related lifestyle behaviors are associated with childhood obesity and obesity-related metabolic dysfunction. The Family Nutrition and Physical Activity (FNPA) instrument provides a multidimensional assessment of lifestyle behaviors related to diet, physical activity, screen exposure, and sleep routines. This study aimed to compare FNPA scores between children and adolescents with and without obesity and to examine the associations between FNPA scores and obesity-related metabolic risk indicators.

Materials and Methods: In this single-center, cross-sectional study, 194 children and adolescents aged 6–18 years (97 with obesity and 97 normal-weight controls) were included. Anthropometric measurements were obtained using standardized protocols, and parental height and weight were measured directly to calculate body mass index (BMI). Parents completed the FNPA questionnaire and structured sociodemographic surveys. Screen time and sleep duration were categorized according to international guidelines. In the obesity group, fasting glucose, insulin, HbA1c, lipid profile, and Homeostasis Model Assessment of Insulin Resistance (HOMA-IR) were assessed. Multivariable linear and logistic regression analyses were performed to identify independent determinants of FNPA scores and factors associated with obesity.

Results: Total FNPA scores were significantly lower in participants with obesity than in controls ($p=0.013$). Longer daily screen exposure was independently associated with lower FNPA scores, whereas adherence to age-specific sleep duration recommendations was positively associated with higher FNPA scores. In multivariable analyses, higher FNPA scores were independently associated with lower odds of obesity. Within the obesity group, FNPA scores were weakly but significantly inversely correlated with BMI Standard Deviation Score (SDS) ($r_s=-0.235$, $p=0.020$) and remained inversely associated with fasting insulin ($r_p=-0.243$, $p=0.017$) and HOMA-IR ($r_p=-0.223$, $p=0.029$) after adjustment for pubertal status.

Conclusion: Lower FNPA scores were associated not only with obesity but also with markers of insulin resistance within the obesity group. These findings suggest that the associations between lower FNPA scores and markers of insulin resistance are independent of pubertal status and that multidimensional assessment tools such as the FNPA may provide clinically useful information in the evaluation of pediatric obesity.

Keywords: Childhood obesity, insulin resistance, life style patterns, screen time, sleep duration

Introduction

Childhood and adolescent obesity has become a major global public health concern, with a substantial increase in prevalence over recent decades and a growing population at risk for early cardiometabolic dysfunction (1). Childhood obesity is associated with metabolic and endocrine complications, including insulin resistance, dyslipidemia, nonalcoholic fatty liver disease, and impaired glucose metabolism (2–4). Early identification of modifiable factors contributing to obesity and obesity-related metabolic abnormalities has therefore become increasingly important in pediatric clinical practice.

Although genetic susceptibility contributes to obesity development, family-level environmental and behavioral factors

play a major role in shaping metabolic risk during childhood and adolescence. Household dietary patterns, physical activity opportunities, sleep routines, and screen-related sedentary behaviors are closely associated with energy balance, weight trajectories, and metabolic health (5,6). Family-based models further demonstrate that parental monitoring and role modelling strongly affect children's eating and physical activity behaviors, whereas sedentary lifestyles and short sleep duration contribute to obesogenic and metabolically unfavorable environments (7,8).

Recent studies suggest that family lifestyle patterns may influence not only obesity risk but also obesity-related metabolic disturbances. In particular, prolonged screen

exposure, irregular meal patterns, and inadequate sleep duration have been associated with insulin resistance and impaired glucose metabolism in pediatric populations (2,5,9–11). In addition, parental BMI and household behavioral characteristics may contribute to clustering of metabolic risk factors within families (5,12,13).

Biochemical markers such as fasting insulin, HOMA-IR, glucose metabolism parameters, and lipid profiles are widely used in the evaluation of children with obesity to identify early cardiometabolic risk (2,3). However, although the Family Nutrition and Physical Activity (FNPA) instrument has been widely used to evaluate family lifestyle environments and obesity risk, its relationship with metabolic abnormalities beyond adiposity has been investigated less extensively. Furthermore, children with similar degrees of obesity may exhibit markedly different metabolic profiles, suggesting that family lifestyle factors may be associated with metabolic risk independently of body weight alone. Therefore, evaluating FNPA scores in relation to metabolic parameters may provide clinically relevant information for the early identification of children at increased cardiometabolic risk.

The aim of this study was to compare FNPA scores between children and adolescents with obesity and normal weight and to investigate whether FNPA scores are associated with obesity-related metabolic risk indicators, particularly markers of insulin resistance, including analyses adjusted for pubertal status.

Material and Methods

Study design and participants

This single-center, cross-sectional analytical study was conducted prospectively at the Pediatric Endocrinology Outpatient Clinic of Ankara Bilkent City Hospital between October 2025 and January 2026. Children and adolescents aged 6–18 years who were diagnosed with obesity and evaluated in the outpatient clinic were enrolled, along with a control group of children and adolescents with normal weight (without overweight or obesity) and no chronic diseases who presented for routine follow-up visits.

The participants in the obesity and control groups were selected for comparison in terms of age and sex. The sample size calculation was based on the total FNPA score, which was defined as the primary outcome of the study. Using the mean difference in the total FNPA score reported in a previously published study ($\Delta=3.5$) and a pooled standard deviation of approximately 7.12, the calculation was performed with G*Power version 3.1 (14). The effect size was estimated as Cohen's $d=0.49$. Assuming a two-sided significance level of $\alpha=0.05$ and a statistical power of 90%, a minimum of 87 participants per group was needed. To account for potential data loss, the sample size was increased by approximately 10%, and at least 95 participants were planned for inclusion in each group, yielding a total sample of 190 participants.

Inclusion and exclusion criteria

The inclusion criteria were as follows:

- Age between 6 and 18 years;
- Written informed consent was obtained from a parent or legal guardian, and assent was provided by the child;

- For the obesity group, BMI at or above the 95th percentile according to the Turkish reference standards developed by Neyzi et al. (15);
- For the control group, the BMI was between the 5th and 85th percentiles.

The exclusion criteria were as follows:

- In both groups, the presence of chronic diseases or the use of medications that could directly affect body weight or metabolism, including long-term systemic corticosteroids or chemotherapy;
- Inability of parents to reliably complete the questionnaires because of severe cognitive or neurologic disorders.

Anthropometric measurements and definition of obesity

Height was measured via a fixed stadiometer with shoes removed and the head positioned in the Frankfurt plane. Body weight was measured to the nearest 0.1 kg via a calibrated digital scale. Parental height and weight were also measured by the same health care personnel using the same equipment, and parental body mass index (BMI; kg/m²) was calculated.

BMI percentiles and standard deviation scores (BMI SDS) were determined according to the Turkish reference curves developed by Neyzi et al. (15). Individuals with a BMI $\geq 95^{\text{th}}$ percentile were classified as obese, whereas those between the 5th and 85th percentiles were classified as normal weight. Pubertal staging was performed according to the Tanner criteria.

Data collection and assessment parameters

Parents completed structured questionnaires on sociodemographic characteristics and lifestyle behaviors together with the FNPA instrument. The FNPA was originally developed by Ihmels et al. (16), and its Turkish validity and reliability were established by Ozdemir et al. (17) (Cronbach's alpha: 0.76).

The FNPA includes 20 items across 10 domains evaluating family eating patterns, screen-time monitoring, beverage choices, family activities, reward practices, food preferences, meal structure, family routines, participation in activities, and home environment. Items are rated on a four-point Likert scale, yielding a total score of 20–80. Since no cut-off value exists, higher scores indicate lower obesity risk, whereas lower scores reflect a more obesogenic family environment.

Assessment of sleep duration and screen time

Sleep duration was assessed on the basis of parental reports and categorized according to age-specific recommendations. In accordance with guidelines from the American Academy of Sleep Medicine and the American Academy of Pediatrics, 9–12 hours for children aged 6–12 years and 8–10 hours for adolescents aged 13–18 years were considered within guideline ranges, whereas shorter durations were classified as below guidelines and longer durations as above guidelines (18).

The daily screen time was recorded via parental reports in three categories: ≤ 2 h/day, 2–4 h/day, and ≥ 4 h/day. This classification was based on recommendations from the American Academy of Pediatrics regarding limits on recreational screen use among school-aged children and adolescents (19).

Parental educational level and household income were recorded in addition to the anthropometric measurements for all the participants. In the obesity group, fasting glucose, insulin, HbA1c, lipid profile, and biochemical data required for calculation of HOMA-IR were obtained as part of routine clinical evaluation. Because metabolic screening tests such as HbA1c, insulin, and lipid profiles are not routinely performed in children without obesity unless clinically indicated, these parameters were evaluated only in the obesity group in the present study.

Written informed consent was obtained from the parents or legal guardians of all participants prior to data collection, and assent was obtained from the children when appropriate.

Statistical analysis

Statistical analyses were performed via IBM SPSS Statistics for Windows, version 25.0 (IBM Corp., Armonk, NY, USA). The distribution of continuous variables was assessed via the Kolmogorov–Smirnov test and visual inspection of histograms and Q–Q plots. Normally distributed data are presented as the means and standard deviations, whereas nonnormally distributed data are reported as medians (interquartile ranges, IQRs). Categorical variables are expressed as numbers (n) and percentages (%).

Between-group comparisons were conducted via Student's *t* test or one-way analysis of variance (ANOVA) for normally distributed continuous variables and the Mann–Whitney *U* test for nonnormally distributed variables. Categorical variables were compared via Pearson's chi-square test. Bonferroni correction was applied for multiple comparisons.

To identify factors associated with the total FNPA score, multivariable linear regression analysis (enter method) was performed. Factors associated with obesity were evaluated via binary logistic regression analysis, and the results are reported as odds ratios (ORs; Exp(B)), 95% confidence intervals, and *p* values. Covariates included in the logistic regression model were selected on the basis of their clinical relevance, previously reported associations with childhood obesity, and potential confounding effects, and were entered simultaneously into the model. Model fit was assessed via the Hosmer–Lemeshow test. Regression model assumptions and diagnostics were assessed prior to the final analyses. Multicollinearity was assessed using variance inflation factors (all VIFs < 5). For the linear regression model, residual normality and influential observations were evaluated using the Shapiro–Wilk test and Cook's distance, respectively. No substantial violations were identified. In the obesity group, associations between total FNPA score and anthropometric and biochemical parameters were examined via Spearman rank correlation analysis. To account for the potential confounding effect of pubertal maturation on insulin resistance, partial correlation analyses controlling for pubertal status were additionally performed for variables demonstrating statistically significant zero-order associations.

A *p* value < 0.050 was considered to indicate statistical significance for all analyses. For post-hoc pairwise comparisons involving three-group analyses, Bonferroni correction was applied and statistical significance was set at *p* < 0.017.

Table I: Sociodemographic and clinical characteristics of the study groups

Variable	Control Group	Obese Group	<i>p</i>
Number of patients	97	97	–
Age*	13.16±2.83 (6.80–17.70)	13.55±3.07 (7.50–17.80)	0.370
Sex†			0.447
Male	35 (36.1)	30 (30.9)	
Female	62 (63.9)	67 (69.1)	
BMI ‡	20.40 (18.30–22.00)	30.60 (27.10–35.30)	<0.001
BMI SDS†	0.20 (–0.40–1.00)	2.50 (2.10–3.00)	<0.001
BMI percentile†	57.00 (31.00–82.00)	99.40 (98.00–99.80)	<0.001
Tanner stage†			0.298
Stage 1	17 (17.5)	14 (14.4)	
Stage 2	9 (9.3)	14 (14.4)	
Stage 3	13 (13.4)	7 (7.2)	
Stage 4	5 (5.2)	2 (2.1)	
Stage 5	53 (54.6)	60 (61.9)	
Maternal BMI‡	25.59 (23.03–30.10)	28.23 (24.98–32.05)	0.001
Paternal BMI‡	26.83 (25.06–29.04)	28.58 (26.57–31.56)	<0.001
Maternal education level†			0.240
Primary school or less	19 (19.6)	22 (22.7)	
Middle school	11 (11.3)	18 (18.6)	
High school	35 (36.1)	36 (37.1)	
University	32 (33.0)	21 (21.6)	
Paternal education level†			0.092
Primary school or less	11 (11.3)	16 (16.5)	
Middle school	8 (8.2)	17 (17.5)	
High school	34 (35.1)	33 (34.0)	
University	44 (45.4)	31 (32.0)	
Household income†			0.405
Low	37 (38.1)	33 (34.0)	
Middle	36 (37.1)	45 (46.4)	
High	24 (24.7)	19 (19.6)	
Screen time†			0.892
< 2 h/day	26 (26.8)	25 (25.8)	
2–4 h/day	45 (46.4)	43 (44.3)	
≥ 4 h/day	26 (26.8)	29 (29.9)	
Sleep duration†			0.388
Below guideline	35 (36.1)	44 (45.4)	
Within guideline	56 (57.7)	49 (50.5)	
Above guideline	6 (6.2)	4 (4.1)	

*, mean±SD (min–max) (Student's *t* test), †: n(%) (Pearson's chi-square test), ‡: median (IQR) (Mann–Whitney *U* test)

Results

Participant characteristics and sociodemographic findings

A total of 194 children and adolescents were included in the analysis, with 97 participants in the obesity group and 97 in the control group. The mean age of the control group was 13.16±2.83 years (range, 6.80–17.70), whereas the mean age of the obesity group was 13.55±3.07 years (range, 7.50–17.80). There was no statistically significant difference in age between the groups (*p*=0.370) (Table I).

With respect to sex distribution, the control group included 35 boys (36.1%) and 62 girls (63.9%), whereas the obesity

Table II: Comparison of total FNPA scores and subscale scores between the obese and control groups

Variable	Control Group	Obese Group	p
Total number of patients	97	97	-
FNPA total score*	53.32±7.09; 53.0 (48.0–58.0)	50.65±7.69; 50.0 (46.0–55.0)	0.013*
Family meals†	6.19±1.63; 7.0 (5.0–8.0)	5.67±1.82; 6.0 (5.0–7.0)	0.045†
Family eating habits†	5.89±1.06; 6.0 (5.0–7.0)	5.37±1.34; 6.0 (4.0–6.0)	0.007†
Food choices†	6.05±1.13; 6.0 (5.0–7.0)	5.75±1.35; 6.0 (5.0–7.0)	0.136†
Beverage choices†	4.73±1.17; 5.0 (4.0–5.0)	4.69±1.32; 5.0 (4.0–6.0)	0.783†
Restriction/reward†	6.00±1.03; 6.0 (5.0–7.0)	6.08±0.95; 6.0 (5.0–7.0)	0.745†
Screen time†	4.96±1.49; 5.0 (4.0–6.0)	4.77±1.56; 5.0 (4.0–6.0)	0.304†
Healthy environment†	4.65±1.51; 5.0 (3.0–6.0)	4.63±1.32; 4.0 (4.0–6.0)	0.925†
Family activity†	4.91±1.30; 5.0 (4.0–6.0)	4.68±1.35; 5.0 (4.0–6.0)	0.238†
Child activity†	4.30±1.63; 4.0 (3.0–5.0)	3.67±1.68; 3.0 (2.0–4.0)	0.003†
Family schedule/sleep†	5.65±1.79; 6.0 (4.0–7.0)	5.33±1.75; 5.0 (4.0–6.0)	0.213†

*: Student's *t* test, †: Mann–Whitney *U* test

Table III: Univariable and multivariable linear regression analysis for total FNPA score

Variable	Univariable				Multivariable				
	B	SE	95% CI	p	B	SE	β	95% CI	p
Age	-0.95	0.170	-1.29 to -0.62	<0.001	-0.82	0.193	-0.323	-1.20 to -0.44	<0.001
Sex (ref: male)	-2.27	1.131	-4.50 to -0.04	0.046	-2.37	0.933	-0.150	-4.21 to -0.53	0.012
BMI SDS	-1.16	0.362	-1.88 to -0.45	0.002	-1.42	0.590	-0.275	-2.58 to -0.25	0.018
Maternal BMI	-0.28	0.102	-0.49 to -0.08	0.006	-0.05	0.095	-0.035	-0.24 to 0.14	0.592
Paternal BMI	0.00	0.139	-0.27 to 0.28	0.992	0.14	0.122	0.070	-0.10 to 0.38	0.264
Group (obese/control)	-2.67	1.062	-4.76 to -0.58	0.013	1.85	1.690	0.124	-1.48 to 5.18	0.275
Household income	2.41	0.697	1.04 to 3.79	0.001	0.89	0.668	0.089	-0.43 to 2.21	0.185
Screen time	-3.81	0.676	-5.15 to -2.48	<0.001	-3.25	0.590	-0.322	-4.42 to -2.09	<0.001
Sleep duration	3.65	0.898	1.87 to 5.42	<0.001	2.10	0.756	0.162	0.60 to 3.59	0.006
Maternal education	2.28	0.469	1.36 to 3.21	<0.001	0.77	0.543	0.112	-0.30 to 1.84	0.158
Paternal education	2.30	0.494	1.33 to 3.28	<0.001	0.76	0.581	0.106	-0.39 to 1.91	0.191
Pubertal status	-2.98	1.456	-5.85 to -0.10	0.042	2.92	1.517	0.143	-0.08 to 5.91	0.056

B: unstandardized regression coefficient, **SE:** standard error, **β:** standardized regression coefficient, **CI:** confidence interval. Multivariable model: $R = 0.428$, adjusted $R^2 = 0.390$, $F(12,181) = 11.276$, $p < 0.001$

group included 30 boys (30.9%) and 67 girls (69.1%). The difference in sex distribution between the groups was not statistically significant ($p = 0.447$) (Table I).

Anthropometric assessments revealed that the BMI, BMI SDS, and BMI percentile values were significantly greater in the obesity group than in the control group (all $p < 0.001$). Similarly, maternal and paternal BMI values were significantly greater in the obesity group than in the control group ($p = 0.001$ and $p < 0.001$, respectively). No significant differences were observed between the groups with respect to Tanner stage ($p = 0.298$).

Comparisons of maternal and paternal educational levels and household income distributions revealed no statistically significant differences between the obesity and control groups. In addition, no significant between-group differences were found for screen time or sleep duration categories ($p = 0.892$ and $p = 0.388$, respectively) (Table I).

Comparison of FNPA scores between groups

The total FNPA scores and domain-specific subscale scores for the obesity and control groups are presented in Table II. The total FNPA score was significantly greater in the control group than in the obesity group (53.32±7.09 vs 50.65±7.69; $p = 0.013$).

Among the subdomains, scores for family meals, family eating habits, and child activity were significantly higher in the control

group than in the obesity group ($p = 0.045$, $p = 0.007$, and $p = 0.003$, respectively). In contrast, no statistically significant differences were detected between the groups with respect to food choices ($p = 0.136$), beverage choices ($p = 0.783$), restriction/reward ($p = 0.745$), screen time ($p = 0.304$), healthy environment ($p = 0.925$), family activity ($p = 0.238$), or family schedule/sleep ($p = 0.213$) subdomains.

Factors associated with total FNPA scores

Multiple linear regression analysis (enter method) was performed to identify factors associated with the total FNPA score. Age, sex, BMI SDS, maternal and paternal BMI, group (obesity/control), household income, screen time, sleep duration, maternal and paternal educational levels, and pubertal status were included in the model. The results are presented in Table III.

The analysis demonstrated that age ($B = -0.82$; 95% CI, -1.20 to -0.44; $p < 0.001$), sex ($B = -2.37$; 95% CI, -4.21 to -0.53; $p = 0.012$), BMI SDS ($B = -1.42$; 95% CI, -2.58 to -0.25; $p = 0.018$), and screen time ($B = -3.25$; 95% CI, -4.42 to -2.09; $p < 0.001$) were independently and negatively associated with the total FNPA score. In contrast, sleep duration was positively associated with the total FNPA score ($B = 2.10$; 95% CI, 0.60 to 3.59; $p = 0.006$). Pubertal status showed a borderline association that did not reach statistical significance ($B = 2.92$; 95% CI, -0.08 to 5.91; $p = 0.056$). Among the determinants of total FNPA

Table IV: Univariable and multivariable logistic regression analyses of factors associated with childhood obesity

Variable	Univariable		Multivariable		
	OR (95% CI)	p	B	Adjusted OR (95% CI)	p
FNPA total score	0.952 (0.915-0.990)	0.014	-0.061	0.941 (0.893-0.991)	0.022
Maternal BMI	1.105 (1.041-1.174)	0.001	0.076	1.079 (1.006-1.157)	0.033
Paternal BMI	1.196 (1.094-1.307)	<0.001	0.156	1.169 (1.064-1.285)	0.001
Sleep duration (ref: below guideline)					
Within guideline	0.696 (0.387-1.251)	0.226	-0.057	0.944 (0.460-1.939)	0.876
Above guideline	0.530 (0.139-2.027)	0.354	-0.430	0.651 (0.154-2.748)	0.559
Screen time (ref: <2 h/day)					
2-4 h/day	0.994 (0.498-1.981)	0.986	-0.468	0.626 (0.280-1.400)	0.254
≥4 h/day	1.160 (0.541-2.487)	0.703	-0.568	0.567 (0.226-1.423)	0.227
Household income (ref: low)					
Middle	1.402 (0.737-2.664)	0.303	0.511	1.667 (0.786-3.536)	0.183
High	0.888 (0.414-1.904)	0.760	0.373	1.452 (0.589-3.583)	0.418
Pubertal status (ref: prepuberte)	1.260 (0.583-2.724)	0.557	0.229	1.257 (0.523-3.019)	0.609

OR: odds ratio, **CI:** confidence interval, **B:** unstandardized logistic regression coefficient (log-odds). Sleep duration, screen time, and household income were entered as categorical (indicator/dummy-coded) covariates, each contributing 2 degrees of freedom relative to the stated reference category; FNPA total score, maternal BMI, and paternal BMI were entered as continuous covariates, and pubertal status as binary (1 df). Age, sex, and BMI SDS were not entered into this model (see Methods for rationale). The multivariable model was statistically significant, $\chi^2(10)=33.458$, $p<0.001$; Nagelkerke $R^2=0.211$; Hosmer–Lemeshow $\chi^2(8)=12.074$, $p=0.148$; overall correct classification=63.4%.

Table V: Correlations between total FNPA score and metabolic parameters in the obesity group

Variable	Spearman r_s	p	Partial r_p^*	p*
BMI SDS	-0.235	0.020	—	—
HOMA-IR	-0.234	0.021	-0.223	0.029
Fasting insulin	-0.255	0.012	-0.243	0.017
HbA1c	0.113	0.271	—	—
Fasting glucose	0.026	0.801	—	—
Triglycerides	-0.140	0.170	—	—
HDL cholesterol	0.014	0.894	—	—
LDL cholesterol	-0.019	0.852	—	—
ALT	-0.135	0.188	—	—

FNPA: Family Nutrition and Physical Activity, **BMI:** body mass index, **SDS:** standard deviation score; r_s : Spearman's rank correlation coefficient, r_p : partial correlation coefficient. *: Partial correlation analyses were performed after adjustment for pubertal status and only for variables that showed statistically significant zero-order correlations. Bold values indicate statistical significance ($p<0.05$).

scores, screen time and sleep duration showed the strongest associations the distribution of FNPA scores across screen time and sleep duration categories was consistent with these findings (Table III).

Factors associated with obesity

The results of the multivariable logistic regression analysis are presented in Table IV. Higher total FNPA scores were independently associated with lower odds of obesity (OR=0.941; 95% CI, 0.890–0.995; $p=0.022$). In addition, higher maternal BMI (OR=1.079; 95% CI, 1.006–1.158; $p=0.033$) and paternal BMI (OR=1.169; 95% CI, 1.065–1.284; $p=0.001$) were independently associated with increased odds of childhood obesity, whereas pubertal status was not significantly associated with obesity ($p=0.609$).

Associations between FNPA scores and metabolic parameters in the obesity group

Correlations between total FNPA scores and obesity-related metabolic parameters are presented in Table V. Total FNPA

scores were weakly but significantly inversely correlated with BMI SDS ($r_s=-0.235$; $p=0.020$), HOMA-IR ($r_s=-0.234$; $p=0.021$), and fasting insulin levels ($r_s=-0.255$; $p=0.012$). After adjustment for pubertal status, the inverse correlations with HOMA-IR ($r_p=-0.223$; $p=0.029$) and fasting insulin ($r_p=-0.243$; $p=0.017$) remained statistically significant. No significant correlations were observed between total FNPA scores and HbA1c ($p=0.271$), fasting glucose ($p=0.801$), triglycerides ($p=0.170$), HDL cholesterol ($p=0.894$), LDL cholesterol ($p=0.852$), or ALT levels ($p=0.188$).

Discussion

In this study, we aimed to evaluate whether the FNPA environment, assessed using the FNPA instrument, was associated not only with obesity status but also with metabolic risk indicators in children and adolescents. The principal finding was that total FNPA scores were significantly lower in children and adolescents with obesity than in their normal-weight peers, and higher FNPA scores were independently associated with lower odds of obesity. More importantly, within the obesity group, lower FNPA scores were associated with higher fasting insulin levels and HOMA-IR values even after adjustment for pubertal status, indicating that these associations were not solely explained by pubertal maturation. Lower FNPA scores were also weakly associated with higher BMI SDS. Together, these findings suggest that family lifestyle patterns may be linked to metabolic heterogeneity beyond obesity status alone and extend previous FNPA research, which has primarily focused on obesity risk and weight status, by demonstrating associations with early markers of insulin resistance in children and adolescents with obesity. Among the FNPA subdomains, family meal patterns, family eating habits, and child physical activity scores were significantly less favorable in the obesity group, consistent with the relationship between obesogenic family environments and higher BMI reported in foundational FNPA studies (16,8). A Turkish validation study of the FNPA instrument likewise demonstrated that the scale captures family behavioral patterns associated with children's weight status (17).

In another study conducted in a Turkish population, higher parental monitoring behaviors were associated with higher FNPA scores, whereas restrictive feeding practices were linked to lower scores, findings that are consistent with our observations regarding family meal structure and eating habits (20). Moreover, the independent inverse association between the total FNPA score and the odds of obesity in our multivariable analyses suggests that the FNPA may reflect clinically relevant lifestyle patterns associated with pediatric obesity rather than merely describing household behavioral characteristics.

Another important finding of our study was that screen time and sleep duration emerged as strong independent determinants of the total FNPA score. Greater screen exposure was associated with significantly lower FNPA scores, whereas adherence to guideline-recommended sleep duration was associated with higher FNPA scores. These findings suggest that excessive screen exposure and inadequate sleep may coexist with less favorable family meal patterns, lower physical activity, and lifestyle behaviors associated with adverse metabolic profiles in children with obesity.

Large cohort studies have demonstrated that short sleep duration and prolonged sedentary screen exposure are associated not only with obesity risk but also with insulin resistance and metabolic dysfunction in pediatric populations (5,9,21,22). In this context, the use of sleep duration categories aligned with recommendations from the American Academy of Sleep Medicine and the American Academy of Pediatrics strengthens the clinical relevance of our findings and supports the interpretation that family lifestyle patterns may be associated with early metabolic abnormalities beyond excess adiposity alone.

These findings further support the value of the FNPA instrument as a multidimensional assessment tool capable of capturing clinically relevant lifestyle patterns rather than isolated behaviors alone. This may help explain why significant differences in FNPA-related behavioral profiles and metabolic associations were observed despite the lack of between-group differences in individual lifestyle variables. The apparent discrepancy between the regression models may reflect the multidimensional structure of the FNPA instrument. Although screen exposure and sleep duration were strong determinants of FNPA scores, they were not independently associated with obesity status after adjustment. This finding may suggest that the FNPA captures broader family behavioral organization and lifestyle quality rather than isolated obesity-related behaviors alone.

The lack of an independent association between obesity status and FNPA scores in multivariable models suggests that the FNPA may reflect lifestyle behaviors associated with metabolic risk rather than obesity status alone. In particular, the strong associations observed with screen exposure and sleep duration indicate that the FNPA captures behavioral patterns linked to sedentary lifestyle and metabolic dysregulation. Consistent with this interpretation, the original FNPA development studies emphasized that the instrument was designed to evaluate household routines, parental monitoring, and lifestyle practices rather than body weight itself (16).

Previous studies have similarly demonstrated that prolonged screen exposure and inadequate sleep duration are associated with insulin resistance, adverse metabolic profiles, and obesity-related cardiometabolic risk in children and adolescents (5,9). Therefore, the relationship between FNPA scores and these behavioral factors may partly explain the associations observed between lower FNPA scores and markers of insulin resistance in our cohort.

Large-scale studies and systematic reviews have consistently identified parental adiposity as one of the strongest determinants of childhood obesity (23). Increased parental BMI is thought to contribute not only to obesity susceptibility but also to familial clustering of adverse metabolic characteristics through shared genetic predisposition, intrauterine influences, and family lifestyle behaviors (5,6).

Consistent with previous studies, both maternal and paternal BMI values were significantly greater in the obesity group and remained independently associated with the odds of childhood obesity in multivariable analyses. These findings support the concept that familial predisposition is strongly associated with pediatric obesity through shared genetic susceptibility, intrauterine influences, and common lifestyle patterns. In contrast, the absence of independent associations between parental BMI or socioeconomic indicators and FNPA scores suggests that the FNPA may primarily reflect modifiable lifestyle behaviors rather than fixed structural or anthropometric characteristics.

Previous studies have demonstrated that family-related behavioral factors, including meal structure, screen exposure, physical activity support, and sleep routines, may independently influence obesity risk and metabolic health in children (5,9). The ability of the FNPA instrument to discriminate lifestyle patterns across different socioeconomic strata further supports its utility as a multidimensional assessment tool for identifying clinically relevant behavioral profiles associated with pediatric obesity (16,17,24).

Taken together, our findings suggest that family-based lifestyle routines may be linked to metabolic risk independently of socioeconomic conditions and that comprehensive instruments such as the FNPA may provide clinically useful information regarding behavioral patterns associated with insulin resistance and obesity-related metabolic dysfunction (7,8).

The weak but statistically significant inverse correlations observed between total FNPA scores and BMI SDS, HOMA-IR, and fasting insulin levels within the obesity group suggest that an unfavorable family lifestyle environment may be associated with early insulin resistance and metabolic dysfunction in pediatric obesity. These findings are clinically relevant because HOMA-IR and fasting insulin are recognized as early markers of cardiometabolic risk that may become abnormal before overt disturbances in glucose metabolism develop (2,3).

Recent pediatric studies have demonstrated that lifestyle factors shaped within the family environment—including sedentary behaviors, irregular meal patterns, prolonged screen exposure, and short sleep duration—are associated with insulin resistance and impaired glucose metabolism (3,5,12,25). In this context,

the observed associations between lower FNPA scores and markers of insulin resistance support the concept that family-based lifestyle patterns may be associated not only with obesity but also with heterogeneity in metabolic risk among children with obesity. These findings suggest that, within children and adolescents with obesity, multidimensional lifestyle assessment tools such as the FNPA may provide clinically useful information regarding behavioral patterns associated with insulin resistance markers. Importantly, these associations remained statistically significant after adjustment for pubertal status, indicating that the observed relationships were not solely explained by pubertal maturation.

In contrast, the absence of significant associations between FNPA scores and lipid parameters or liver enzyme levels may indicate that the FNPA is more closely related to early metabolic derangements, particularly insulin resistance, rather than to later obesity-related complications such as dyslipidemia or hepatic steatosis. These complications are known to be influenced by multiple interacting factors, including duration of adiposity exposure, pubertal maturation, and genetic susceptibility (2,26–29). Contemporary pediatric obesity literature likewise emphasizes the heterogeneous metabolic course of obesity and the variable relationship between lifestyle factors and advanced metabolic complications.

Limitations

This study has several limitations. First, the cross-sectional design precludes causal inference and does not clarify the temporal relationship between family lifestyle patterns and obesity-related metabolic risk. Although the FNPA instrument assesses habitual household behaviors, it remains unclear whether unfavorable lifestyle patterns preceded or resulted from obesity and metabolic abnormalities.

Second, participants were recruited from a tertiary pediatric endocrinology outpatient clinic, which may limit generalizability. Families seeking specialized care may differ from the general pediatric population in health awareness, obesity severity, or lifestyle behaviors, potentially introducing selection bias.

Third, certain lifestyle variables, including screen time and sleep duration, were based on parental self-reports and may therefore be subject to recall or social desirability bias. In addition, metabolic parameters were evaluated only in the obesity group, preventing direct biochemical comparisons with normal-weight participants.

Finally, although statistically significant associations were observed, several correlations were modest. Nevertheless, the relationships between FNPA scores and insulin resistance markers suggest that family-based lifestyle patterns may be associated with clinically relevant variability in metabolic risk among children with obesity.

Despite these limitations, the study has several strengths. The obesity and control groups were recruited from the same center during the same period and were balanced for age and sex. Anthropometric measurements of both children and parents were obtained using standardized protocols and calibrated equipment rather than self-reported values. The multidimensional

FNPA instrument enabled comprehensive assessment of family-related lifestyle patterns associated with obesity and metabolic risk. Furthermore, multivariable regression modelling allowed adjustment for potential confounders and strengthened the internal validity of the findings.

Conclusion

This study demonstrated that FNPA patterns are associated not only with obesity status but also with metabolic risk indicators in children and adolescents. Lower FNPA scores in children with obesity and the inverse association between FNPA scores and obesity risk suggest that unfavorable family lifestyle patterns are closely linked to pediatric obesity.

More importantly, the observed associations between lower FNPA scores and higher BMI SDS, fasting insulin, and HOMA-IR values indicate that the family lifestyle environment may be associated with early insulin resistance and metabolic dysfunction beyond excess adiposity alone. The strong relationships between FNPA scores, screen exposure, and sleep duration further support an association between family-based behavioral patterns and metabolic risk profiles in pediatric obesity.

Taken together, these findings suggest that multidimensional assessment tools such as the FNPA may provide clinically useful information regarding lifestyle behaviors associated with adverse metabolic outcomes and may help support family-based approaches in the clinical management of pediatric obesity.

Ethics committee approval

This study was conducted in accordance with the Helsinki Declaration Principles. The study was approved by Ankara Bilkent City Hospital (19.11.2025, reference number: 1/1868/2025).

Contribution of the authors

Study conception and design: DD, SO, MB, FG, data collection: IG, CAG, EG, analysis and interpretation of results: DD, SO, PK, DT, statistical analysis: DD, draft manuscript preparation: DD, critical revision of the manuscript: PK, DT, MB, FG. All authors reviewed the results, approved the final version of the manuscript, and agreed to be accountable for all aspects of the work.

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Conflict of interest

The authors declare that there is no conflict of interest.

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