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Research Article

Associations Between Dietary Diversity Score and Adiposity Indexes in Obese Adolescents

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What is already known on this topic?
Visceral adipose tissue is considered an independent risk factor for cardiometabolic risk. Diet and lifestyle changes can affect visceral adipose tissue. However, the relationship between dietary diversity and adiposity-related biomarkers used to determine visceral adiposity and predict cardiometabolic risk is unknown.

What does this study adds?
High dietary diversity is associated with low insulin resistance and low visceral adiposity, triglyceride/glucose, and lipid accumulation product indices, which predict cardiometabolic risk.

ABSTRACT
Objective: Nutrition can affect visceral adipose tissue, but the effect of dietary diversity on visceral adiposity is unknown. This study aimed to determine the relationship between dietary diversity and visceral adiposity, triglyceride/glucose, lipid accumulation product, and body shape indices in obese adolescents.
Methods: The study included 141 obese adolescents (70 males, 71 females) aged between 12 and 18. Participants' biochemical parameters, anthropometric measurements, and blood pressures were measured. Two days of retrospective food intake records were collected from the adolescents, and Dietary Diversity Scores (DDS) were calculated and divided intotertiles.. A DDS score of <4.09 was classified as tertile 1; 4.09-4.96 as tertile 2; and >4.96 as tertile 3. Visceral adiposity, triglyceride/glucose, lipid accumulation product, and body shape indexes were calculated according to the formulas specified in the literature.
Results: Insulin and Homeostasis Model assessment for Insulin Resistance (HOMA-IR) values were found to be higher in individuals in Tertile 1 compared to those in other tertiles ($p<0.001$). The triglyceride/glucose index value was found to be lower in individuals in Tertile 3 compared to those in Tertile 1 ($p=0.028$). In individuals in Tertile 3, fibre ($p=0.002$), vegetable ($p<0.001$), and whole grain ($p<0.001$) intake were higher than in other tertiles, while refined grain ($p<0.001$) and meat consumption ($p=0.013$) were lower than in other tertiles. A negative correlation was found between the DDS and fasting blood glucose (rho = -0.177; $p = 0.036$), insulin (rho = -0.633; $p < 0.001$), triglycerides (rho = -0.223; $p = 0.008$), HOMA-IR (rho = -0.656; $p < 0.001$), visceral adiposity index (rho = -0.228; $p = 0.007$), triglyceride/glucose index (rho = -0.251; $p = 0.003$), and lipid accumulation product index (rho = -0.200; $p = 0.018$). When confounding factors were controlled for, fasting blood glucose emerged as the significant factor affecting DDS
Conclusion: High dietary diversity scores in obese adolescents are associated with low visceral adiposity, triglyceride/glucose, and lipid accumulation product index, indices associated with visceral obesity. As dietary diversity scores increase, fasting blood sugar, insulin, triglyceride, and HOMA-IR levels decrease.
Keywords: Dietary diversity, visceral adiposity, triglyceride/glucose index, lipid accumulation product, body shape indices

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INTRODUCTION

Obesity is one of the most critical chronic medical conditions that affects cell metabolism and causes an increase in adipose tissue, thereby increasing the risk of metabolic syndrome, such as cardiovascular disease, hypertension, and type 2 diabetes (1). The increase in the prevalence of obesity among children and adolescents is a risk factor for chronic diseases in adulthood and affects future morbidity/mortality. Identifying risk factors for obesity in adolescents is the most appropriate way to intervene in obesity and reduce cardiovascular risk (2).
In recent years, researchers have focused more on the location and distribution of fat in the body than on the total amount of fat. The location and distribution of fat in the body are associated with morbidity and mortality from disease. In general, visceral obesity is reported to play a central role in the development of chronic disease compared to regional or general obesity (3). Visceral adipose tissue is a hormonally active component of the body's fat mass stored in the abdominal cavity, near the digestive organs (4). It is considered an independent risk factor for metabolic syndrome due to its role in regulating glucose, lipid metabolism, and blood pressure (5). It has been found to be associated with cardio-metabolic pathologies, and its level is valuable in determining risk for these diseases (6). To date, body composition variables used as predictors of metabolic syndrome include Body Mass Index (BMI), Waist Circumference (WC), or Waist-to-Hip Ratio (WHR). At the same time, visceral fat tissue can be measured using costly and impractical methods such as bioelectrical impedance analysis, dual-energy X-ray absorptiometry, Computed Tomography, and Magnetic Resonance Imaging (7,8). For example, since BMI cannot distinguish between muscle mass and body fat mass, an increase in muscle mass may be diagnosed as excess weight or obesity. Therefore, additional anthropometric indicators are needed to assess abdominal visceral obesity (9). In the past few years, some lipid and visceral obesity-related indices, such as visceral adiposity index, triglyceride/glucose index, lipid accumulation product index, atherogenic index of plasma, cardiometabolic index, and body roundness index, have also been proposed as supplementary indices to to estimate the existence of obesity and the distribution pattern of adipose tissue, especially visceral ones (10). Visceral adiposity index, triglyceride/glucose index, lipid accumulation product index, and body shape index are better predictors of insulin resistance and metabolic syndrome risk than traditional indices in pediatric population. These indices are mathematical models calculated using anthropometric data, lipid, and glucose profiles. They are used to indicate visceral adiposity, adiposity dysfunction, Homeostasis Model Assessment for Insulin Resistance (HOMA-IR),

metabolic dysfunction, and cardiometabolic risk. Thus, they can help predict significant health risks with a simple formulation and facilitate early intervention (11).

Current evidence shows that lifestyle changes and diet affect visceral adipose tissue (4). When examining the effects of diet on health, the importance of dietary diversity is emphasised. Eating the same types of food can lead to an insufficient intake of certain nutrients. Dietary diversity ensures a more balanced intake of nutrients and other non-nutrient components into the body (12). Studies have found a negative correlation between dietary diversity scores and the incidence of cardiovascular disease, cancer, metabolic syndrome, and osteoporosis (13-15). A study found that increased consumption of plant-based diets was associated with better anthropometric measurements, increased HDL cholesterol levels, and reduced lipid accumulation products (3). Another study suggests that increased dietary protein intake and animal-derived monounsaturated fatty acids may be positively associated with changes in visceral fat dysfunction and visceral adiposity index (16). In contrast to these studies, another study found no effect of Western-style, healthy, and combined diets on triglyceride/glucose index and visceral fat levels (4). However, the relationship between dietary diversity and adiposity-related biomarkers is unknown. Therefore, this study aims to investigate the effects of dietary diversity on visceral adiposity, triglyceride/glucose, lipid accumulation products, and body shape indices, which are used to determine visceral adiposity and predict cardiometabolic risk in obese adolescents.

Materials and methods

Study design, setting, and participants

This study was carried out in 141 obese adolescents (70 male, 71 female) aged 12-18 years who applied to the Pediatric Endocrinology Outpatient Clinic at Gazi University Faculty of Medicine Hospital between February 2025 and May2025. The inclusion criteria were obese adolescents (Body Mass Index (BMI) ≥95th percentile) who did not have any chronic disease diagnosed by a doctor, did not take hormone therapy, and did not use medication. Exclusion criteria included having any concomitant chronic medical disease (syndromic, metabolic, or neurological) except for metabolic syndrome secondary to obesity or not having clinically normal mental development.

Clear explanations were provided with regard to the purpose of the study, after which written informed consent was obtained from the adolescents in accordance with the Declaration of Helsinki. Approval was obtained from the Gazi University Ethics Committee (with approval number:2025-164).

Data Collection and Evaluation

Data was collected in face-to-face interviews through a questionnaire that included adolescent socio-demographics, dietary habits, anthropometric measurements, body composition analyses, biochemical findings, dietary diversity score table, and two -day food consumption records.

Anthropometric Measurements and Body Composition Analysis

Body weight measurement and body composition analysis (fat mass, percentage of fat, Fat-Free Mass [FFM]) were conducted by using the InBody 720 (1–1000 kHz; InBody Co., Ltd., Korea). Height was measured (cm) with feet close together and the head in Frankfort plane with a portable stadiometer with a 0.1-cm accuracy. Body Mass Index was calculated as weight (kg)/height (m^2). BMI-SDS and Body weight-SDS were calculated according to the standards established for Turkish children (17). Waist Circumference (cm) (WC) was measured from the midpoint between the lowest rib and the iliac crest. Hip Circumference (cm) (HC) was measured horizontally at the largest circumference of the hip.

Biochemical parameters and blood pressure

The fasting blood glucose, fasting insulin, total cholesterol, LDL-cholesterol (LDL-C), HDL-cholesterol (HDL-C), triglyceride, ALT, and AST levels of the children, which are routinely analyzed at Gazi University, Faculty of Medicine, Department of Pediatric Endocrinology, were recorded. Venous blood samples were obtained from all patients from the antecubital region between 8.00 and 8.30 am after an 8-12 hour overnight fast. Fasting glucose was measured with the enzymatic UV (hexokinase method) test method using the autoanalyzer AU5800 (Beckman Coulter Inc., Brea, CA,USA). HDL-C, LDL-C, total cholesterol, and triglyceride levels were measured with the enzymatic colorimetric method using the autoanalyzer AU5800 (Beckman Coulter Inc., Brea, CA,USA). Insulin levels were measured with a one-step principle enzymatic immunoassay method using the autoanalyzer UniCel DxI 800 (Beckman Coulter Inc., Brea, CA,USA). Serum AST and ALT levels were measured with the kinetic UV method using the autoanalyzer AU5800 (Beckman Coulter Inc., Brea, CA,USA). Blood pressure measurements of the adolescent were taken by the researchers in accordance with the standard measurement protocol (18). The "Homeostasis Model Assessment for Insulin Resistance" (HOMA-IR) value was calculated using the "fasting blood glucose (mg/dl) x fasting insulin (μU/mL)/405" formula (19). Unit changes have been made in the parameters in accordance with the formulas.

Cardio-metabolic risk markers

Cardio-metabolic risk markers are calculated as;

Visceral adiposity index = in girls [WC/((36.58) + (1.89 x BMI))] x (TG/0.81) x (1.52/HDL-C),
in boys = [WC/((39.68 + (1.88 x BMI))] x (TG/1.03) x (1.31/HDL-C) (20)

Triglyceride/glucose index = ln [fasting triglyceride (mg/dl) x fasting blood glucose (mg/dl)/2] (21)

Lipid accumulation product index = (WC- 58) x TG in girls, (WC- 65) x TG in boys (22)

Body shape index = $WC/(BMI^{⅔} \times height^{½})$ (23).

Dietary intake and calculation of Dietary Diversity Score (DDS)

DDS is most often determined by counting the number of selected food groups consumed by individuals over a reference period, which usually ranges between 1 and 3 days. Two-day food consumption records were obtained from the participants. Adolescents were trained by the dietitian on how to keep food consumption records. The Food and Nutrient Photo Catalogue was used to ensure that patients correctly specified the amount of food they consumed. The food diversity score table was filled in by the researcher according to retrospective food consumption records, 1 day on weekdays and 1 day on weekends. The DDS score was calculated according to the completed food diversity table.

The food diversity score table consists of five main food groups:grains,, vegetables, fruits, meat, and dairy products. Under these five groups, 23 sub-food groups were evaluated in terms of score. These subgroups were:

1. Grains group with seven subgroups: white bread, biscuits, pasta, whole grains, cereals, rice, refined grains,
2. Fruits were divided into 2 subgroups: berries and citrus, other fruits, and juices.
3. Vegetables group with seven subgroups: vegetables, potatoes, tomatoes, other starchy vegetables (corn, pea, eggplant, squash), legumes (pease, beans, corn, lentils), yellow vegetables (carrots and pumpkin), and other green vegetables (bell peppers, all kinds of cabbage, broccoli, celery, cucumbers, garlic, onion, green beans, zucchini, leeks, parsley, lettuce, radish, spinach, turnips).
4. The meat group was divided into four subgroups: red meat, poultry, fish, and eggs.
5. The dairy group was divided into three subgroups: milk (low-fat and full-fat), yogurt (low-fat and full-fat), cheese

Fats and sugars were excluded from the dietary diversity score calculation.

To be considered as a consumer of a food group, it is necessary that at least a half-serving of that food group should be consumed per day by individuals. Each of the five main groups is evaluated at two scores.-each. This two scores was divided among the subgroups. If all food groups - are consumed, the DDS is 10 points. The points obtained from five groups are summed, and the total DDS score is obtained (24).

Statistical Analysis

Data were analyzed using ~~IBS~~ IBM SPSS 29.0 package program (IBM Corp, Armonk, NY, USA). Compliance with normal distribution was examined using Shapiro-Wilk and Kolmogorov-Smirnov tests. Mean ± standard deviation and median (Q1 - Q3: IQR-quartile range) values

were used in descriptive statistics for continuous variables. Independent two-sample t-test was used to compare normally distributed data according to paired groups, and Mann-Whitney U test was used to compare non-normally distributed data. To create balanced groups according to DDSdietary diversity status was divided into tertiles based on 33.3% and 66.6% percentiles of the series. Individuals in the top 33.3 percent were classified as having low dietary diversity, while those in the 33.3 percent to 66.6 percent decile were considered to have moderate dietary diversity. Those with values above 66.6 percent were classified as having high dietary diversity.A DDS<4.09 was classified as tertile 1 (46 individuals), 4.09-4.96 as tertile 2 (48 individuals), and >4.96 as tertile 3 (47 individuals). One-way analysis of variance was used to compare normally distributed data for groups of three and more than three, and multiple comparisons were analyzed with the Tukey HSD test. Kruskal-Wallis test was used to evaluate non-normally distributed data for groups of three and more than three, and multiple comparisons were analyzed with Dunn's test. Pearson correlation analysis was used for normally distributed data, and Spearman correlation analysis was used for non-normally distributed data. Multiple Linear Regression Analysis was performed to determine the effect of independent variables on the dependent variable (DDS). Variables that showed a significant correlation with DDS were included in the multiple regression model, taking into account confounding factors (age, gender, BMI-SDS, total energy intake, etc.).Statistical significance value was accepted as $p<0.05$.

Results

The mean age of the adolescents was 14.81±1.94 years. Of the individuals, 49.6% were boys, and 50.4% were girls. Table 1 shows the distribution of general characteristics of the individuals according to gender. A statistically significant difference was found between genders in BMI-SDS, VA-SDS, waist circumference, fat percentage, systolic and diastolic blood pressure, visceral adiposity index, and body shape index ($p<0.05$). It was found that the BMI-SDS and Weight-SDS scores of boys were lower than those of girls ($p<0.05$). While waist circumference was higher in boys ($p=0.004$), girls had higher fat percentage ($p=0.012$) and lower systolic and diastolic blood pressure than boys ($p<0.05$). The visceral adiposityindex was higher in girls ($p=0.048$), while the body shape index was higher in boys ($p=0.020$). Other demographic data, anthropometric measurements, biochemical findings, and index scores were similar between genders ($p>0.05$).

Table 1 here

When looking at DDS by gender in Table 2, it is found that boys have higher fruit group DDS ($p=0.005$) and milk group DDS ($p=0.018$) than girls. No statistically significant difference was found between the meat group DDS, the vegetable group DDS, the grain group DDS, and total DDS according to gender ($p>0.05$).

Table 2 here

Anthropometric measurements, biochemical findings, and index scores according to DDS are compared in Table 3. Statistically significant differences were observed between tertiles in terms of insulin ($p<0.001$), HOMA-IR ($p<0.001$), and Triglyceride/Glucose Index ($p = 0.034$) values. Insulin and HOMA-IR values differed among the three tertiles, with individuals in Tertile 1 having higher insulin and HOMA-IR values than those in the other tertiles ($p<0.001$). The triglyceride/glucose index value was found to be lower in individuals in Tertile 3 compared to those in Tertile 1 ($p=0.028$). In individuals in Tertile 3, fibre ($p=0.002$), vegetable ($p<0.001$), and whole grain ($p<0.001$) intake was higher than in other tertiles, while refined grain ($p<0.001$) and meat consumption ($p=0.013$) were lower than in other tertiles. Our study found no statistically significant differences between individuals in other parameters according to tertiles ($p>0.05$).

Table 3 here

Table 4 shows the relationship between DDS and anthropometric measurements, biochemical findings, and index scores. DDS was negatively correlated with fasting blood sugar (rho=-0.177; $p=0.036$), insulin (rho=-0.633; $p<0.001$), triglycerides (rho=-0.223; $p=0.008$), HOMA-IR (rho=-0.656; $p<0.001$), visceral adiposityindex (rho=-0.228; $p=0.007$), triglyceride/glucose index (rho=-0.251; $p=0.003$), and lipid accumulation product index (rho=-0.200; $p=0.018$).

Table 4 here

The results of the multiple regression analysis are shown in Table 5. In the regression analysis, at least one of the independent variables was found to be a significant factor (DDS: $F=6.917$ and $p<0.001$). Fasting blood glucose ($B=-0.017$; $p=0.039$) was found to have significant effects on DDS and explained approximately 35% of the variance ($R^2adj=0.355$).

Table 5 here

Discussion

Identifying risk factors for obesity in adolescents is the most appropriate way to intervene in obesity and reduce cardiovascular risk (24). Diet, insulin resistance, and adiposity, particularly visceral fat, contribute significantly to the development of obesity (4). Previous studies have examined the potential contribution of diet to serum insulin levels and body composition (4,25,26). However, there are insufficient studies on its effects on visceral adiposity, triglyceride/glucose, lipid accumulation product, and body shape indices, which are strong predictors of cardiometabolic risk. This study observed that as the dietary diversity score increased, the scores for the visceral adiposity index, triglyceride/obesity index, and lipid accumulation product index used to predict metabolic obesity decreased. Dietary diversity score is an important parameter used to assess nutrient adequacy, overall diet quality, and the diet-disease relationship. It has been reported that a higher dietary diversity score is closely associated with a healthy diet with better nutrient adequacy and diet quality (24). A study has shown that DDS has an inverse relationship with metabolic syndrome and also with high blood pressure, high triglyceride levels, and abnormal glucose homeostasis (27). In our study, a negative relationship was observed between DDS and fasting blood sugar, insulin, triglycerides, and HOMA-IR. When we excluded confounding factors, it was seen that the factor affecting DDS was fasting blood glucose. Visceral adipose tissue is considered an independent risk factor for cardiovascular diseases due to its role in regulating glucose, lipid metabolism, and blood pressure (9). The visceral adiposity index has been identified as a new cardiometabolic risk marker in recent decades because it reflects abdominal fat distribution and dyslipidaemia. The triglyceride/glucose index and lipid accumulation product index are good markers of insulin sensitivity and are associated with insulin resistance (4). It has been reported that visceral adipose tissue is affected by changes in diet and lifestyle (4,9). In our study, as the DDS scores of obese adolescents increased, the visceral adiposity index, triglyceride/glucose index, and lipid accumulation product index used to predict metabolic obesity decreased. In a study examining the effects of different dietary patterns on body composition, it was found that the Western-style dietary pattern positively affected the fat mass index/fat-free mass index ratio, while the 'vegetable and fruit'-based dietary pattern negatively affected the fat mass index/fat-free mass index ratio (28). A study on children suggested that high consumption of fruits, vegetables, grains, and vegetable oils may increase lean body mass index (29). It has been reported that adolescents with low DDS have a higher body fat percentage than adolescents with high DDS (30). However, in our study, no relationship was found between DDS and anthropometric measurements such as BMI, fat percentage, mass, waist, and hip circumference. The fact that the study sample consisted of obese individuals and that energy, carbohydrate, fat, and protein intake were similar across DDS groups may have contributed to this finding.

The inverse relationship between DDS and metabolic risks may be attributed to the increased consumption of healthier food groups associated with high DDS (24). This study revealed that although adolescents' energy intakes were similar according to their DDS scores, adolescents with high DDS consumed more fibre, vegetables, whole grains, legumes, and less refined grains and meat. Vizzuso et al (11) found that energy intake was positively associated with BMI z-score, but no association was found with visceral adiposity index. In this study, while total energy intake was not associated with cardiometabolic risk markers, it was observed that meal pattern affected DDS and DDS was associated with cardiometabolic risk markers. In a study, healthy plant-based diet index scores were found to be associated with better anthropometric measurements and HDL levels compared to unhealthy plant-based diet index scores, and were also found to reduce LAP levels (3). Another study reported that healthy diet models had no effect on triglyceride/glucose indices compared to a Western-style

diet or a mix of healthy and Western-style diets, but they did cause a decrease in LAP levels. Individuals with the highest healthy diet model scores were 71% less likely to have high LAP levels compared to those in the lowest category (4). Mazidi and colleagues also reported positive correlations between the visceral adiposity index and glucose/insulin homeostasis markers and the consumption of carbohydrates and sugar, total fat and saturated fatty acids, as well as negative correlations between fibre, vitamin, and mineral intake and the visceral adiposity index and lipid accumulation product indices, similar to our study. Additionally, it has been noted that there is an inverse relationship between a diet rich in monounsaturated and polyunsaturated fatty acids and fasting blood glucose and lipid accumulation product indices (26). Studies have reported a negative relationship between the DASH diet index, which is based on increasing the consumption of vegetables, fruits, whole grains, legumes, and white meat, while reducing the consumption of red meat, refined carbohydrates, and sugary beverages, and the visceral adiposity index (31), and a negative relationship between the anti-inflammatory diet and triglyceride/glucose indices (4). A study examining the relationship between whole grain consumption and insulin resistance, glucose homeostasis, and inflammation found that the group with high whole grain consumption had lower C-reactive protein, apolipoprotein B, FBG, insulin, homeostatic model assessment of insulin resistance (HOMA-β, haemoglobin A1c, and glucose levels were lower in the group with high whole grain product consumption compared to the group with low whole grain product consumption (26).

Adolescents are generally a group that is influenced by their peers, has freedom of choice in food, and tends to choose unhealthy foods. Good growth and development require a variety of foods from various food groups (vegetables, fruits, whole grains, and animal-based foods) and a balanced intake of vitamins. Dietary diversity consists of all food groups (grains, vegetables, fruits, meat, and dairy products) necessary for growth and development, and high dietary diversity is associated with healthy food groups such as vegetables, fruits, and fibre (24). In our study, individuals with high dietary diversity were found to have a reduced risk of metabolic syndrome. The proposed mechanism explaining the results obtained from implementing a dietary model with high dietary diversity is that the higher fibre content of vegetables, whole grain products, and legumes may lead to lower nutrient absorption or energy intake, affecting total fat mass and visceral fat accumulation (1,26). Higher fibre intake has been shown to improve insulin resistance and reduce visceral adiposity (4,32). High fibre intake, which is broken down into short-chain fatty acids by the gut microbiota, is known to improve insulin sensitivity or insulin resistance (3). Low-glycaemic index carbohydrates found in vegetables, whole grains, and legumes may also reduce insulin resistance (33). Additionally, the anti-inflammatory and antioxidant properties of vegetables, fibre, and legumes may be associated with lower systemic inflammation. Chronic, low-grade inflammation has been shown to contribute to obesity and be associated with multiple metabolic complications (1,33). Soluble fibre, in particular, binds to bile acids in the small intestine, increasing the excretion of bile salts in the faeces, lowering cholesterol, and regulating postprandial insulinemic and glycaemic responses (34. High intake of antioxidants and micronutrients from plant foods also represents another potential cardioprotective mechanism. This antioxidant capacity, combined with the potential to modulate nitric oxide production, enhances the ability of polyphenolic compounds to maintain vascular homeostasis (35). In our study, refined grain and meat consumption were significantly reduced in the tertile with the highest dietary diversity. Low intakes of animal protein and saturated fatty acids have been reported to prevent obesity effectively. Additionally, animal proteins are rich in other nutrients such as iron, sodium, and nitrites obtained from processed meats, increasing the risk of cardiometabolic diseases (36). Refined grains have high carbohydrate content, which leads to a high dietary glycaemic load. Compared to whole grain products, refined grains are rapidly absorbed due to their high glycaemic load, leading to increased fasting blood sugar and insulin resistance. Unlike refined grains, whole grain products are high in dietary fibre, trace elements, and phytochemicals, and their nutrients and nutritional components have beneficial effects on metabolic syndrome (37).

Study Limitations

This study has some limitations. The study included only obese adolescents from a single tertiary center. Without a healthy control group, the ability to generalize the findings or assess relative risk is limited. Diet history method has some limits in accuracy. All self-reported dietary assessment methods are subject to both random and systematic measurement errors. However, retrospective food consumption records do not have the potential recall bias caused by food consumption frequency questionnaires. The cross-sectional nature of the study, preventing any causal inferences, the division of dietary diversity scores into tertiles, potentially leading to misclassification in the data, and the small sample size are the other limitations of the study. This study has many strengths. It is the first study to examine the effect of dietary diversity on cardiometabolic risk markers such as visceral adiposity, triglyceride/glucose ratio, lipid accumulation product, and body shape indices. Other strengths include the control of a wide range of potential confounding factors to obtain an independent relationship, the calculation of dietary diversity scores from food consumption records by a trained dietitian, and the homogeneity of the sample due to the adolescents participating in the study being from the same geographical region and sharing similar cultures, lifestyles, and eating habits. Additionally, using food combinations rather than single foods to examine the specified relationships provides accurate information and should be considered an additional strength.

Conclusion

In conclusion, dietary diversity scores were inversely associated with visceral adiposity, triglyceride/glucose, and lipid accumulation product indices in obese adolescents. Furthermore, as dietary diversity scores increased, fasting blood sugar, insulin, triglyceride, and HOMA-IR levels decreased. Dietary diversity was found to be associated with metabolic syndrome. Strategies aimed at increasing dietary diversity through nutritional interventions may have a positive effect, particularly on insulin resistance and cardio-metabolic risk. Extensive prospective studies focusing on different populations are needed to confirm these findings.

Ethics

Ethics Committee Approval: Approval was obtained from the Gazi University Ethics Committee (with approval number: 2025-164)
Informed Consent: Clear explanations were provided with regard to the purpose of the study, after which written informed consent was obtained from the adolescents in accordance with the Declaration of Helsinki
Authorship Contributions: Conceptualization and data collection (R.B, A.B, M.O.Ç, A.K.U, E.D, M.A.O, U.A); data cleaning and analysis (R.B, M.A.O, U.A, A.K.U, E.D); draft manuscript preparation, writing the manuscript (R.B.); critical review and editing of manuscript (R.B, M.O.Ç, A.B, A.K.U, E.D, M.A.O, U.A); final approval of manuscript (A.B.) All authors reviewed and approved the final version.
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Table 1. Distribution of general characteristics of individuals by gender				
	Boy (n=70)	**Girl (n=71)**	**Total (n=141)**	**p**
Age (years)	14.50±1.82	15.11±2.02	14.81±1.94	0.061*
Mother's age (years)	40.47±5.64	41.25±5.98	40.87±5.81	0.426*
Father's age (years)	43.96±5.32	44.90±5.64	44.43±5.48	0.308*
BMI-SDS	2.01 (1.61-2.55)	2.39 (1.76-2.99)	2.16 (1.72-2.75)	**0.012****
Body weight-SDS	1.73±0.91	2.35±1.42	2.04±1.23	**0.003***
Waist circumference (cm)	102.50 (98-108)	96 (89-105)	100 (91-108)	**0.004****
Hip circumference (cm)	110.50 (104-119)	108 (101-115)	109 (102-117)	0.145**
Fat mass (kg)	24.30 (19.90-34.80)	26.20 (21.20-32.70)	25.50 (20.70-33.10)	0.561**
Fat percentage (%)	33.65±8.28	36.65±5.44	35.16±7.13	**0.012***
Fasting blood sugar (mg/dL)	90 (84-96)	88 (82-93)	89 (84-94.60)	0.178**
Insulin (µU/L -)	20.61 (13.04-31.11)	23.15 (13.70-29.38)	21.70 (13.70-30.75)	0.928**
Total cholesterol (mg/dL)	161.81±33.68	164.01±32.06	162.92±32.78	0.692*
LDL (mg/dL)	94 (78-108.92)	93 (77-102)	93 (78-105)	0.585**
HDL (mg/dL)	43 (38.30-49.70)	46.50 (40.21-51.50)	44 (39.50-50.70)	0.136**
Triglycerides (mg/dL)	110.30 (84.90-159.90)	101 (76-131.60)	102.60 (80.80-150.80)	0.158**
Systolic blood pressure (mmHg)	130 (120-140)	130 (120-130)	130 (120-140)	**0.022****
Diastolic blood pressure (mmHg)	75 (70-80)	70 (70-75)	70 (70-80)	**0.006****
HOMA-IR	4.48 (2.93-6.96)	5.03 (3.15-6.92)	4.87 (3.13-6.93)	0.916**
Visceral Adiposity Index	1.55 (1.01-2.38)	1.82 (1.37-2.68)	1.68 (1.13-2.50)	**0.048****
Triglyceride/Glucose Index	8.58 (8.26-8.84)	8.35 (8.13-8.68)	8.43 (8.17-8.82)	0.104**
Lipid Accumulation Product Index	44.60 (33.91-70.23)	38.43 (28.39-64.67)	41.72 (30.17-65.33)	0.362**
Body Shape Index	0.0834±0.0045	0.0814±0.0054	0.0824±0.0051	**0.020***

BMI-SDS: Body mass index standard deviation score, VA-SDS: Body weight standard deviation score, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, *Independent two-sample t-test value is given as mean ± standard deviation, ** Mann-Whitney U test value is given as median (Q1 - Q3: IQR-interquartile range). p<0.05.

Table 2. Distribution of dietary diversity scores by gender				
	Boy (n=70)	**Girl (n=71)**	**Total (n=141)**	**p**
Meat group DDS	1.13 (0.72-1.43)	1.23 (0.87-1.51)	1.19 (0.81-1.46)	0.126**
Fruit group DDS	1.00 (0.66-1.49)	0.76 (0.39-1.12)	0.88 (0.51-1.23)	**0.005****
Vegetable group DDS	0.92 (0.51-1.15)	0.90 (0.55-1.29)	0.90 (0.51-1.18)	0.479**
Dairy group DDS	0.71 (0.41-1.00)	0.54 (0.37-0.77)	0.65 (0.39-0.90)	**0.018****
Cereal group DDS	0.90 (0.29-1.33)	0.91 (0.48-1.46)	0.90 (0.43-1.37)	0.349**
Total DDS	4.56±1.12	4.43±0.79	4.50±0.97	0.425*

DDS Dietary diversity score, *Independent two-sample t-test value given as mean ± standard deviation, ** Mann-Whitney U test value given as median (Q1 - Q3: IQR-interquartile range). p<0.05.

Table 3. Comparison of anthropometric measurements, biochemical findings and index scores according to tertiles of Dietary Diversity Score				
	1st Tertile [<4.09] (n=46)	**2nd Tertile [4.09-4.96] (n=48)**	**3rd Tertile [>4.96] (n=47)**	**p**
BMI-SDS	2.17 (1.76-2.75)	2.11 (1.72-2.77)	2.20 (1.71-2.79)	0.946**
Body weightSDS	2.12±1.24	2.17±1.08	1.84±1.36	0.371*
Waist circumference (cm)	100.50 (92-107)	99.50 (92-108)	101 (89-108)	0.865**
Hip circumference (cm)	108 (103-118)	109 (102-116.50)	110 (101-118)	0.981**
Fat mass (kg)	25.75 (21.20-34.00)	25.75 (21.40-32.85)	24.40 (19.30-33.90)	0.716**
Fat percentage (%)	35.03±7.45	35.04±7.15	35.40±6.93	0.961*
Fasting blood sugar (mg/dL)	91 (87-96)	86 (80.15-94.30)	88 (83-93)	0.072**
Insulin µU/L (-)	31.72 (23.17-42.83)[a]	21.70 (14.84-27.37)[b]	13.04 (10.38-20.68)[c]	**<0.001****
Total cholesterol (mg/dL)	161.92±34.53	167.31±26.82	159.41±36.54	0.489*
LDL (mg/dL)	93.50 (79-108.28)	90 (77.50-103)	94 (77.80-107.20)	0.782**
HDL (mg/dL)	42.65 (39.90-48)	46.15 (40-53)	43.70 (37.20-50.70)	0.266**
Triglycerides (mg/dL)	111.75 (89.40-166.50)	102.80 (81.85-155.90)	98.30 (66.10-131.60)	0.074**
Systolic blood pressure (mmHg)	130 (120-140)	130 (120-140)	125 (120-140)	0.369**

Diastolic blood pressure (mmHg)	70 (70-80)	70 (70-80)	70 (70-80)	0.910**
HOMA-IR	7.07 (5.03-10.05)[a]	4.79 (3.46-6.49)[b]	2.80 (2.28-4.40)[c]	**<0.001****
Visceral Adiposity Index	2.03 (1.26-2.77)	1.60 (1.39-2.53)	1.52 (0.95-2.30)	0.081**
Triglyceride/Glucose Index	8.57 (8.27-8.90)[a]	8.46 (8.17-8.87)[a,b]	8.31 (8.05-8.63)[b]	**0.034****
Lipid Accumulation Product Index	48.31 (34.44-71.50)	45.04 (32.32-72.03)	37.93 (25.70-61.16)	0.106**
Body Shape Index	0.0816±0.004	0.0829±0.004	0.0828±0.005	0.435*
Energy (kcal)	2118.26±103.44	2133.61±188.13	2097.81±216.22	0.319*
Carbohydrates (%)	49.08±3.5	48.62±2.9	48.79±3.5	0.459*
Proteins (%)	14.04±2.66	13.87±3.04	14.21±2.15	0.501*
Fats (%)	35.92±2.74	36.23±3.12	35.61±2.18	0.662*
Fiber (g)	20.61.±2.19[a]	19.74±3.15[a]	24.69±2.57[b]	**0.002***
Whole Grains (g)	103.17.±25.23[a]	147.47±15.19[a]	239.52±13.08[b]	**<0.001***
Refined grains (g)	352.14±18.35[a]	271.22±20.15[a]	205.35±31.28[b]	**<0.001***
Fruits (g)	296.55±126.17	319.63±149.37	336.13±105.40	0.103*
Vegetable (g)	288.24±100.15[a]	269.38±93.63[a]	361.79±65.48[b]	<0.001*
Dairies (g)	455.86±76.84	478.5±101.62	446.32±92.17	0.516*
Meat (g)	171.77±62.11[a]	163.80±54.22[a]	127.52±55.13[b]	**0.013***
Legumes (g)	19.88±10.14[a]	17.65±11.29[a]	23.93±14.86[b]	**0.004***

BMI-SDS: Body mass index standard deviation score, BW-SDS: Body weight standard deviation score, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, *One-way analysis of variance value is given as mean ± standard deviation, **Kruskal-Wallis test value is given as median (Q1-Q3: IQR-interquartile range). a-c: There is no difference between groups with the same letter.

Table 4. Relationship between Dietary Diversity Score and anthropometric measurements, biochemical findings, and index scores

	Dietary Diversity Score	
	r	**p**
Body weight-SDS	-0.080	0.347*
Fat percentage (%)	-0.024	0.780*
Total cholesterol (mg/dL)	-0.062	0.468*
Body Shape Index	0.074	0.384*
Energy (kcal)(day)	-0.077	0.369*
	rho	**p**
BMI-SDS	-0.009	0.920**
Waist circumference (cm)	-0.039	0.649**
Hip circumference (cm)	0.000	0.997**
Fat mass (kg)	-0.075	0.379**
Fasting blood sugar (mg/dL)	-0.177	**0.036****
Insulin (µU/L -)	-0.633	**<0.001****
LDL (mg/dL)	-0.018	0.834**
HDL (mg/dL)	0.047	0.580**
Triglycerides (mg/dL)	-0.223	**0.008****
Systolic blood pressure (mmHg)	-0.144	0.088**
Diastolic blood pressure (mmHg)	-0.038	0.657**
HOMA-IR	-0.656	**<0.001****
Visceral Adiposity Index	-0.228	**0.007****
Triglyceride/Glucose Index	-0.251	**0.003****
Lipid Accumulation Product Index	-0.200	**0.018****

BMI-SDS: Body mass index standard deviation score, BW-SDS: Body weight standard deviation score, HDL: High-density lipoprotein, LDL: Low-density lipoprotein, HOMA-IR: Homeostatic Model Assessment for Insulin Resistance, * Pearson correlation analysis, ** Spearman correlation analysis. p<0.05.

Table 5. Multiple regression results for different factors affecting the Dietary Diversity Score
Result of multiple regression analysis

		Unstandardized Coefficients		**Standardized Coefficients**		
Dependent	**Independent**	**B± SE**	**95% CI**	**Beta**	**p**	**Significance of the model**
Dietary Diversity Score	**Constant**	9.505±2.862	3.841/15.169		**0.001**	F=6.917 p<0.001 R^2=0.355
	Fasting blood sugar (mg/dL)	-0.017±0.008	-0.032/-0.001	-0.204	**0.039**	
	Insulin (µU/L)	-0.035±0.018	-0.071/0.001	-0.592	0.057	
	Triglycerides (mg/dL)	-0.008±0.005	-0.018/0.003	-0.594	0.155	

	HOMA-IR	0.022±0.078	-0.133/0.177	0.087	0.782	
	Visceral Adiposity Index	0.094±0.106	-0.116/0.305	0.212	0.378	
	Triglyceride/Glucose Index	-0.017±0.394	-0.796/0.762	-0.009	0.966	
	Lipid Accumulation Product Index	0.006±0.006	-0.005/0.018	0.299	0.279	
B: Unstandardized coefficient, SE: Standard Error, CI: Confidence Interval						