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Nutritional status, dietary intake, and family predisposition to overweight and obesity in relation to the FTO rs9939609 polymorphism

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A – Research concept and design, B – Collection and/or assembly of data, C – Data analysis and interpretation, D – Writing the article, E – Critical revision of the article, F – Final approval of the article

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Abstract

Introduction and Objective. The FTO rs9939609 gene variant has been associated with obesity in humans; however, its role in the regulation of dietary intake remains unclear. The aim of the study is to examine the associations between dietary intake, nutritional status, and familial predisposition to overweight and obesity, as well as to evaluate the contribution of the FTO rs9939609 polymorphism in this context.

Materials and Method. The study included 112 adults (74% women; mean age: 40.1 ± 13.2 years). Body composition was assessed using bioelectrical impedance analysis; weight status was classified according to body mass index (BMI). Dietary intake was evaluated using a three-day food records and a food frequency questionnaire. Genotyping of the FTO rs9939609 polymorphism (AA, AT, and TT) was performed using fast real-time PCR on DNA isolated from buccal swabs. Statistical analyses included analysis of variance, non-parametric tests, and multivariate methods.

Results. The frequency of the A allele was 0.46. Genotype frequencies (TT = 0.26, AT = 0.56, AA = 0.18) were consistent with Hardy–Weinberg equilibrium. The FTO rs9939609 polymorphism was associated with a higher prevalence of excessive body weight. However, no significant differences were observed in anthropometric measurements or body composition parameters between genotype groups. The polymorphism explained 7.82% of the variability in indicators of nutritional status.

Conclusions. The study supports an association between the rs9939609 polymorphism of the FTO gene and increased susceptibility to excessive body mass, particularly among carriers of the A allele, and a significant influence of relatives along the maternal line. These observations highlight the potential contribution of shared genetic susceptibility and familial environment to obesity.

Key words

obesity, adults, nutrition, body composition, genetic obesity, rs9939609 polymorphism

INTRODUCTION

Polygenic obesity, the most common form of obesity, results from a complex interplay of behavioural, environmental, and genetic factors [1]. Although sustained positive energy balance directly leads to weight gain, the limited long-term efficacy of hypocaloric diets suggests the involvement of additional mechanisms. Environmental factors, including unhealthy dietary patterns and low physical activity, are important; however, family and twin studies indicate that 40–70% of interindividual variability in obesity risk is genetically determined [2]. Among genes regulating body weight, the FTO (fat mass and obesity-associated) gene is one of the most influential [3], with variants on chromosome 16 consistently linked to obesity and metabolic disturbances [4].

Experimental evidence shows that the FTO plays a key role in the development of energy homeostasis and adipose tissue. While FTO-deficient mice exhibit fat mass comparable to wild-type controls regardless of diet [5], FTO overexpression appears critical for adipogenesis [6]. Single-nucleotide polymorphisms (SNPs) in FTO have also been associated with eating behaviours and dietary intake, although results are inconsistent. Some studies report higher fat and lower carbohydrate intake among carriers of the rs9939609 A allele, without differences in total energy intake [7], whereas others found no association with food preferences [8] or observed higher added sugar intake among TA/AA carriers [9]. These inconsistencies highlight the need for further research on the relationships between FTO variants, dietary intake, and modifiable behaviours.

Despite extensive research on FTO, limited attention has been paid to its relationship with familial patterns of obesity. Familial clustering of excess body weight reflects shared genetic susceptibility and environmental exposures; therefore, examining overweight and obesity among relatives may provide additional insight into the phenotypic expression of FTO variants, particularly in relation to dietary behaviours and nutritional status.

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OBJECTIVE

The aims of the study are: (1) to determine the prevalence of FTO rs9939609 genotypes (TT, TA, and AA) in the study cohort; (2) to assess nutritional status across genotype groups; (3) to analyze dietary intake according to genotype; and (4) to evaluate the associations between the FTO rs9939609 polymorphism and dietary habits. An additional and novel component of this study was assessment of the relationship between excessive body weight in participants and the occurrence of overweight or obesity among their relatives in the context of FTO genotype.

MATERIALS AND METHOD

Study design and ethical approval. The study protocol was approved by the Ethics Committee of the Institute of Food and Nutrition in Warsaw, Poland. All procedures were conducted in accordance with the ethical principles outlined in the Declaration of Helsinki. The study was conducted in two stages: a pilot study (n=30) followed by a main study (n=82), yielding a total sample of 112 participants. Data collection included participant questionnaires, anthropometric measurements (height, weight, waist and hip circumference), BMI calculation, body composition analysis using bioelectrical impedance (BIA), dietary assessment (FFQ-6 and a three-day dietary record in the main study), and genotyping of FTO polymorphisms using cheek swabs, DNA isolation, and real-time PCR with probes. All data were subsequently subjected to statistical analysis to evaluate associations between FTO variants, dietary patterns, and nutritional status.

Selection of participants. Participants were recruited using purposive sampling based on predefined inclusion and exclusion criteria. The inclusion criteria were: body mass index (BMI) ≥ 18.5 kg/m^2; age between 18–65 years; stable, habitual dietary patterns; and willingness to participate in the study, as evidenced by written informed consent. Exclusion criteria: prior surgical or pharmacological treatment for obesity; adherence to restrictive or therapeutic diets that could alter habitual eating patterns; diagnosed eating disorders (including anorexia nervosa, bulimia nervosa, and binge-eating disorder); diagnosed epilepsy; presence of a cardiac pacemaker; diagnosed endocrine disorders (including diseases of the thyroid, adrenal glands, or gonads); pregnancy; age below 18 or above 65 years; and failure to provide informed consent.

Nutritional status. Anthropometric measurements, including body weight, height, waist circumference, and hip circumference, were obtained in accordance with standardized measurement procedures [10]. Based on these data, body mass index (BMI; weight (kg)/height [m^2]) [11] waist-to-hip ratio (WHR; waist circumference (cm)/hip circumference [cm]) [12], and waist-to-height ratio (WHtR; waist circumference (cm)/height [cm]) were calculated [13]. Overweight was defined as a BMI between 25.0 and 29.9 kg/m^2, whereas obesity was defined as a BMI ≥ 30.0 kg/m^2. Body composition was assessed using bioelectrical impedance analysis (BIA) with a body composition analyzer (BIA-101, AKERN-Srl) in accordance with the guidelines provided in the Body Composition Procedures Manual [10].

Dietary intake. Energy and nutrient intake was assessed using a standard three-day food record that included two non-consecutive weekdays and one weekend day. Mean daily energy and nutrient intakes were calculated with DIETA 6®, based on the Polish food composition database for foods and composite dishes [14]. Dietary variables were standardized (per 1,000 kcal or per kg body mass), and nutrient densities (fat, protein, carbohydrates, dietary fibre, alcohol) were expressed as proportions of energy. Macronutrient intakes were adjusted for total energy intake using the residual method [15], with linear regression models fitted for each macronutrient and energy-adjusted values calculated as residuals, reflecting dietary composition independent of total energy intake. Habitual dietary patterns and frequency of consumption of selected foods were assessed using the validated Polish Food Frequency Questionnaire (FFQ-6) [16], which includes 62 food items and six frequency categories ranging from 'never' or 'almost never' to 'several times a day' over the preceding 12 months. All dietary data were collected using paper questionnaires, thoroughly explained during the initial face-to-face meeting, completed independently by participants, and subsequently verified and clarified individually by the principal investigator during a second meeting, with ongoing access to researcher support and detailed, written instructions provided throughout the study.

Genotyping. Biological material was collected at the Nutrigenomics Laboratory at the University of Life Sciences in Warsaw, during the initial study visit. Buccal swabs were obtained under standardized conditions after at least 1 h of fasting (water permitted), avoidance of tooth brushing, and removal of foreign objects from the oral cavity; two swabs were collected from each participant. DNA was isolated from oral epithelial cells using the ReliaPrep gDNA Tissue Miniprep System (Promega), according to the manufacturer's protocol. DNA concentration was measured by UV-Vis spectrophotometry using a NanoDrop 2000 (Thermo Scientific) with 2 µL samples; DNA purity was assessed based on absorbance at 260 and 280 nm. In all samples, DNA concentration and purity were sufficient for further analyses. Genotyping of the FTO rs9939609 polymorphism was performed using the allelic discrimination method with fast real-time PCR. Reactions were carried out using TaqMan Genotyping Master Mix and TaqMan SNP Genotyping Assays (Applied Biosystems) on a LightCycler 96 real-time PCR system (Roche) with LightCycler® 96 SW 1.1 software. Analyses were conducted in 96-well plates with a total reaction volume of 25 µL containing 1.25 µL DNA template, 12.5 µL master mix, 1.25 µL SNP assay, and 10 µL DNase-free water. Thermal cycling included pre-denaturation at 95 °C for 10 min, followed by 45 cycles of denaturation at 92 °C for 15 s and annealing/extension at 60 °C for 60 s, with a final melting step. Negative controls without DNA were included on each plate, and selected samples were analyzed in duplicate. All reactions produced valid results.

Statistical analyses. Statistical analyses assessed associations between FTO rs9939609 genotypes and dietary intake, nutritional status, socio-economic and lifestyle factors, and the presence of overweight or obesity among family members. One-way ANOVA was used to examine relationships between genotype and mean values of bioimpedance and anthropometric traits, as no significant

gender effect was observed. Consumption frequency data were analyzed using the Kruskal–Wallis test after grouping questionnaire responses. Normality was evaluated with the Shapiro–Wilk test, followed by ANOVA or Kruskal–Wallis tests, as appropriate; nominal data (BMI and WHR categories) were analyzed using the chi-square test. Principal component analysis (PCA) assessed overall variability, while correspondence analysis identified features differentiating genetic groups. Statistical significance was set at $p \leq 0.05$, and analyses were conducted using Statistica version 13.

RESULTS

The study included 112 adults aged 18–65 years (mean age: 40.1 ± 13.24 years; mean BMI: 30.1 ± 7.36 kg/m^2), of whom 74% were women. No significant differences in age or BMI were observed between genotype groups ($p = 0.34$ and $p = 0.66$, respectively).

Genotype characteristics. Allele and genotype frequencies are presented in Tab. 1. Genotype distribution did not deviate significantly from the Hardy–Weinberg equilibrium ($\chi^2 = 1.96$; $p = 0.16$). This outcome validates the representativeness of the sample and the precision of the genotype determinations. The absence of substantial deviations from the Hardy-Weinberg equilibrium further underscores the suitability of treating the studied group as a random population in the context of association analyses.

Table 1. Frequencies of allele and genotypes of rs9939609 polymorphism in the FTO gene (n = 112)

Genotypes	n	%
Homozygous TT	29	25.9
Heterozygous TA	63	56.2
Homozygous AA	20	17.9
Alleles	n	%
A	103	46.0
T	121	54.0

$\chi^2 = 1.96$; $p = 0.16$ for Hardy–Weinberg equilibrium analysis

Genotype and nutritional status. Participants carrying at least one A allele (AA or TA genotypes) were more frequently classified as overweight or obese than TT homozygotes (78.3% vs. 58.6%). The odds ratio for overweight or obesity among AA/TA carriers compared with TT homozygotes was 2.55 (95% CI: 1.03–6.30). This association was statistically significant based on the chi-square test ($\chi^2 = 4.25$; $p = 0.04$), with a comparable result obtained using Fisher's exact test ($p = 0.04$) (Tab. 2). These findings indicate that carriage of the A allele was associated with higher odds of overweight or obesity in the examined cohort.

Table 2. Association of the genotype of the studied polymorphism with the risk of overweight and obesity

Genotype	No. of individuals with normal body mass	No. of overweight and obese individuals	Total	OR (95% CI)	p*
TT	12 (41.4%)	17 (58.6%)	29	2.55 (1.03–6.30)	0.04*
TA + AA	18 (21.7%)	65 (78.3%)	83		
Total	30	82	112		

OR – odds ratio (95%); CI – confidence intervals); * – Chi-square test.

Mean anthropometric and body composition values stratified by genotype are presented in Table 3. No statistically significant differences were observed for individual variables, likely due to substantial within-group variability. An additional reason for the lack of differences may be different group sizes, because with unequal group sizes, the sensitivity of statistical tests decreases. Nevertheless, AA homozygotes tended to show higher body weight, waist and hip circumferences, and fat mass, along with lower muscle mass and total body water percentages. These trends should be interpreted cautiously.

Table 3. Mean values of the anthropometric measurements and body composition of the individual genotypes

Parameter	Genotype TT n = 29 Mean ± SD	Genotype TA n = 63 Mean ± SD	Genotype AA n = 20 Mean ± SD	r variance analysis p
Height [cm]	172.5 ± 8.48	171.6 ±7.91	170.5 ± 9.32	NS*
Body weight [kg]	75.3 ± 20.75	84.2 ± 24.38	98.8 ± 22.47	NS
Hip circumference [cm]	98.0 ± 14.65	104.7 ± 14.95	116.0 ± 10.89	NS
Waist circumference [cm]	85.5 ± 17.08	90.6 ±19.65	104.0 ± 17.44	NS
WHR	0.87 ± 0.11	0.88 ± 0.12	0.88 ± 0.13	NS
WHtR	0.58 ± 0.09	0.56 ± 0.11	0.55 ± 0.10	NS
Body cell mass index	7.7 ± 3.25	8.4 ± 2.69	9.5 ± 2.81	NS
Fat mass [%]	29.3 ± 9.57	34.3 ± 10.04	38.1 ± 8.11	NS
Fat mass [kg]	23.5 ± 13.9	31.0 ± 17.64	38.7 ± 11.98	NS
Fat free mass [%]	70.7 ± 11.06	65.2 ± 10.57	61.9 ± 14.63	NS
Fat free mass [kg]	51.7 ± 9.40	53.2 ± 7.43	60.0 ± 8.21	NS
Cellular mass [%]	44.7 ± 13.74	46.3 ± 10.94	46.6 ± 12.51	NS
Cellular mass [kg]	23.0 ± 9.40	24.7 ± 7.43	28.0 ± 8.21	NS
Muscle mass [%]	39.8 ± 11.02	38.3 ± 10.75	36.1 ± 12.11	NS
Muscle mass [kg]	28.7 ± 10.75	30.8 ± 8.52	34.7 ± 9.58	NS
Total body water [%]	51.7 ± 6.58	47.9 ±7.26	45.3 ± 5.80	NS
Total body water [Lt.]	37.8 ± 7.94	39.0 ± 8.03	43.9 ± 10.54	NS
Extracellular water [%]	45.7 ± 5.09	45.2 ± 4.14	45.2 ±4.11	NS
Extracellular water [Lt.]	17.2 ± 3.37	17.5 ± 3.84	19.8 ± 4.67	NS
Intracellular water [%]	54.3 ± 5.09	54.2 ± 4.56	54.8 ± 4.11	NS
Intracellular water [Lt.]	20.6 ± 5.60	21.5 ± 4.90	24.1 ± 6.32	NS

WHR – waist-to-hip ratio; WHtR – waist-to-height ratio; * NS – non-significant

The studied polymorphism in the FTO gene explained a mere 7.82% of the overall variability of traits characterizing nutritional status. This finding indicates that the FTO rs9939609 polymorphism contributes modestly to the variability in nutritional status, supporting its role as a modulatory rather than a primary determinant.

Among the analyzed parameters, extracellular water content (ECW, litres) was associated with rs9939609 genotype in both genders, with the highest values in AA homozygotes and a significant gender difference (Fig. 1). No other genotype-related effects were detected for the remaining measures.

Genotype and familial patterns of overweight and obesity. Detailed data regarding the prevalence of excess body weight among relatives are shown in Table 4. In the TA/AA group, 61.11% of participants were classified as overweight or obese, compared with 18.06% among TT homozygotes. Among mothers of participants carrying the TA/AA genotypes, overweight or obesity was reported in 38.89%, whereas the corresponding value among mothers of TT participants was 8.33%. Similarly, overweight or obesity was reported in 20.84% of grandmothers of TA/AA carriers, compared with

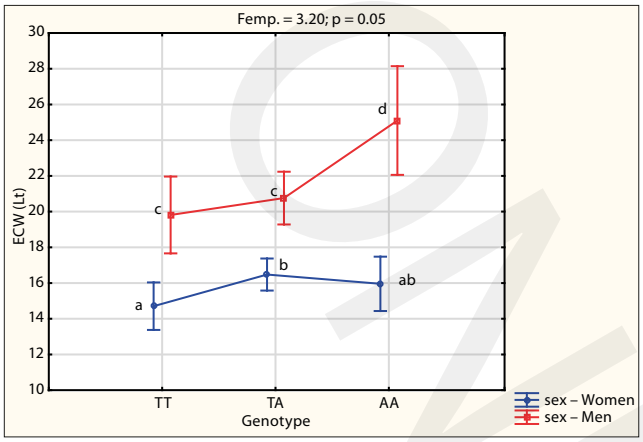

Figure 1. Relationship between extracellular water content (ECW in litres) and genotype (TT genotype, TA genotype, AA genotype) by gender

Table 4. Occurrence of excess body weight in relatives of the study participants (percentage of the total group / number of individuals)

Degree of kinship	Genotype of the studied individual	
	TT	TA & AA
Individual	18.06 / 14	61.11 / 44
Mother	8.33 / 7	38.89 / 28
Father	9.72 / 8	33.33 / 24
Brothers	1.39 / 1	27.77 / 20
Sisters	11.11 / 8	34.73 / 25
Sons	2.78 / 2	13.89 / 10
Daughters	2.78 / 2	16.66 / 12
Grandchildren	0.00 / 0	2.78 / 2
Father's brothers	2.78 / 2	9.72 / 7
Father's sisters	4.17 / 3	30.55 / 22
Father's father	1.39 / 1	1.39 / 1
Father's mother	4.17 / 3	12.50 / 9
Mother's brothers	2.78 / 2	15.28 / 11
Mother's sisters	0.00 / 0	19.45 / 14
Mother's father	1.39 / 1	5.56 / 4
Mother's mother	4.17 / 3	20.84 / 15

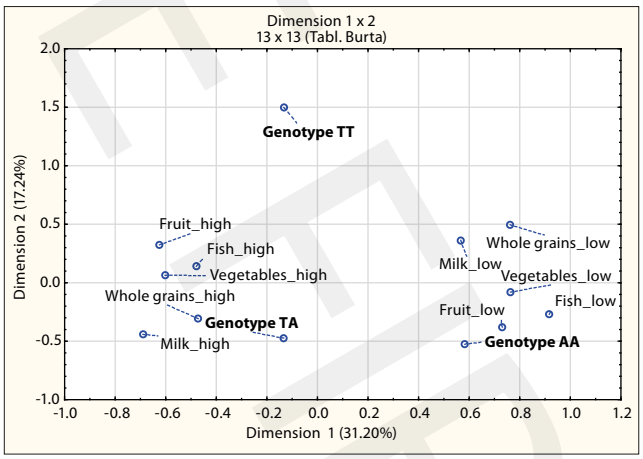

Figure 2. Correspondence analysis of genotypes and frequency of consumption of selected product groups: high – consumption at least once a day for fruit, vegetables, milk, and whole grains, or several times per month for fish; low – consumption less than once a day for fruit, vegetables, milk and whole grains, or less than several times per month for fish

4.17% among grandmothers of TT participants. Overall, the results indicate a tendency toward higher reporting of excess body weight along the maternal line among carriers of the A allele. Spearman's rank correlation analysis revealed weak but statistically significant associations between excess body weight in siblings and mothers and carriage of the risk genotype ($r = 0.24–0.27$; $p < 0.05$).

Genotype and dietary characteristics. Analysis of dietary intake did not reveal significant associations between the FTO rs9939609 polymorphism and total energy or nutrient intake. No statistically significant relationships were observed between genotype and total energy intake or macronutrient consumption when expressed per kilogram of body weight, as a percentage of total energy intake (Tab. 5), or after energy adjustment using the residual method (Tab. 6), nor for micronutrient intakes (vitamins and minerals) expressed as nutrient density per 1000 kcal of diet (Tab. 5).

AA homozygotes reported significantly lower consumption of refined small-grained groats ($p = 0.02$), tropical fruits (excluding kiwi and citrus fruits, $p = 0.05$), lean fish ($p = 0.05$), and yellow–orange vegetables ($p = 0.04$). In addition, AA homozygotes showed a tendency towards more frequent consumption of biscuits and cakes ($p = 0.09$), and less frequent consumption of vegetables such as cucumber ($p = 0.10$). Lower consumption frequencies of ice cream and pudding ($p = 0.07$), ready-to-eat breakfast cereals ($p = 0.09$), berries ($p = 0.05$), and avocado ($p = 0.07$), were also observed among carriers of the A allele compared with the remaining genotype groups.

The relationship between genotype and selected indicators of healthy dietary patterns was further explored using correspondence analysis (Fig. 2). The presence of the AA risk genotype corresponded to a lower frequency of consumption of nutrient-dense foods, including fruits, vegetables, whole grains, dairy products, and oily fish. In contrast, TA heterozygotes exhibited a distinct dietary pattern characterized by higher reported consumption of fruits, vegetables, oily fish, whole-grain cereals, milk, and natural dairy products, compared with AA homozygotes.

DISCUSSION

In the present study, the frequency of the A allele of the FTO rs9939609 polymorphism was 0.46, and genotype distribution conformed to the Hardy–Weinberg equilibrium, supporting genotyping quality and indicating no major population stratification. The observed allele and genotype frequencies were comparable to those reported in other European populations, including Polish and Italian cohorts, as well as in Asian populations [17, 18].

An association between the rs9939609 polymorphism and body mass status was observed, with carriers of at least one A allele (TA/AA genotypes) showing higher odds of overweight or obesity compared with TT homozygotes. This finding is consistent with large European studies demonstrating increased body mass and obesity risk among A allele carriers, as well as reports from Polish [19] and Asian populations [8, 20]. Collectively, these findings support a consistent association between the rs9939609 variant and obesity-related phenotypes across populations. Additionally, A allele carriers have been reported to exhibit a higher percentage of body fat [21].

Table 5. Average daily nutrient intake based on three-day food records for each individual genotype

Nutrient	Genotype TT (n = 18)				Genotype TA (n = 34)				Genotype AA (n = 12)				Kruskal-Wallis test (p)
	Mean	SD	Min	Max	Mean	SD	Min	Max	Mean	SD	Min	Max	
Energy [kcal/kg b.m.]	21.6	8.47	10.5	46.1	22.1	7.89	8.9	46.4	22.1	7.64	12.3	35.9	NS
Protein [g/kg b.m.]	1.0	0.36	0.5	1.7	1.0	0.43	0.4	2.0	0.9	0.38	0.2	1.7	NS
Animal prot. [g/kg b. m.]	0.7	0.29	0.2	1.2	0.6	0.27	0.3	1.4	0.7	0.24	0.4	1.2	NS
Plant prot. [g/kg b. m.]	0.3	0.09	0.2	0.5	0.3	0.09	0.1	0.7	0.3	0.14	0.0	0.6	NS
Fat [g/kg b. m.]	0.8	0.37	0.2	1.6	0.7	0.18	0.4	1.8	0.8	0.27	0.4	1.3	NS
SFA [g/kg b. m.]	0.3	0.15	0.1	0.7	0.3	0.05	0.2	0.7	0.3	0.15	0.2	0.7	NS
MUFA [g/kg b. m.]	0.3	0.16	0.1	0.6	0.3	0.06	0.2	0.7	0.3	0.09	0.2	0.5	NS
PUFA [g/kg b. m.s]	0.1	0.05	0.0	0.2	0.1	0.04	0.1	0.3	0.1	0.05	0.1	0.2	NS
Carbohydrates [g/kg b. m.]	2.5	1.17	1.1	6.1	2.8	0.72	1.3	5.8	2.6	1.02	1.3	4.7	NS
Fructose [g/kg b. m.]	0.1	0.09	0.0	0.3	0.1	0.01	0.1	0.6	0.1	0.11	0.0	0.3	NS
Glucose [g/kg b. m.]	0.1	0.07	0.0	0.3	0.1	0.01	0.1	0.3	0.1	0.09	0.0	0.3	NS
Lactose [g/kg b. m.]	0.1	0.05	0.0	0.2	0.1	0.01	0.1	0.2	0.1	0.05	0.0	0.2	NS
Starch [g/kg b. m.]	1.5	0.64	0.5	3.0	1.5	0.22	0.9	4.1	1.5	0.44	0.8	2.1	NS
Dietary fibre [g/1000 kcal]	11.7	4.02	6.7	21.6	11.7	5.71	3.6	20.2	11.3	3.33	6.3	15.2	NS
Cholesterol [mg/1000 kcal]	194.0	81.10	98.4	355.6	166.3	72.11	61.6	324.4	167.9	64.84	73.9	297.3	NS
Protein [% energy]	18.4	4.47	14.2	32.3	17.1	11.76	3.8	29.5	17.9	2.84	12.9	21.6	NS
Dietary fibre [% energy]	2.2	0.77	1.3	4.1	2.3	1.08	0.7	3.8	2.2	0.63	1.2	3.0	NS
Fat [% energy]	31.8	8.31	11.9	44.2	30.0	13.88	8.8	47.0	30.5	4.80	21.1	37.4	NS
Carbohydrates [% energy]	46.8	8.32	34.1	62.1	50.1	31.14	9.3	68.2	47.7	5.41	40.4	59.9	NS
Alcohol [% energy]	2.6	3.09	0.3	7.9	3.3	1.49	1.7	5.4	4.3	2.76	1.9	8.6	NS
Folate [mcg]	133.3	58.35	71.5	314.5	134.9	36.66	56.0	246.9	137.0	31.53	105.6	192.9	NS
Niacin [mg]	11.7	5.73	7.1	27.3	13.0	6.30	4.9	31.1	11.7	2.87	5.3	15.4	NS
Retinol [mcg]	210.4	80.74	83.9	381.7	189.9	86.60	65.4	408.8	176.3	79.52	67.5	313.6	NS
Riboflavin [mg]	0.8	0.23	0.6	1.6	0.8	0.20	0.5	1.3	0.8	0.16	0.6	1.2	NS
Thiamine [mg]	0.6	0.19	0.4	1.1	0.7	0.18	0.3	1.0	0.6	0.18	0.3	0.9	NS
Vitamin A [mcg]	729.5	481.20	202.8	1979.3	550.0	235.57	241.2	1241.8	477.3	139.08	200.2	734.0	NS
Vitamin B_{12} [mcg]	2.2	1.63	1.0	7.3	2.2	1.83	0.7	8.5	1.9	0.87	1.0	3.4	NS
Vitamin B_6 [mg]	1.0	0.40	0.6	2.2	1.0	0.31	0.5	1.8	1.0	0.23	0.5	1.3	NS
Vitamin C [mg]	45.9	48.81	3.8	172.7	74.3	199.74	9.7	1197.3	42.3	24.42	7.0	84.5	NS
Vitamin D [mcg]	2.8	2.65	0.8	9.7	2.7	2.95	0.4	11.6	1.8	1.12	0.6	4.5	NS
Vitamin E [mg]	4.9	1.63	2.3	7.8	4.7	1.87	2.0	8.8	4.5	1.36	2.2	6.6	NS
Zinc [mg]	5.5	1.40	3.8	8.5	5.0	1.04	2.9	7.6	5.4	1.10	4.1	7.8	NS
Phosphorus [mg]	695.7	192.82	545.6	1393.0	730.8	197.79	388.4	1191.7	712.2	120.17	502.3	926.6	NS
Iodine [mcg]	90.4	51.84	30.9	263.1	75.8	36.84	8.0	224.7	81.0	30.54	47.5	143.5	NS
Magnesium [mg]	168.4	58.09	113.7	374.0	169.6	47.36	89.7	310.1	180.6	37.16	128.5	237.9	NS
Manganese [mg]	2.9	1.53	1.6	6.9	2.7	1.04	1.3	4.9	2.7	0.91	1.5	4.5	NS
Copper [mg]	0.6	0.18	0.4	1.1	0.7	0.19	0.4	1.1	0.7	0.14	0.5	0.9	NS
Potassium [mg]	1736.8	498.18	891.2	3075.3	2010.0	792.66	854.3	4013.4	1839.7	457.09	1015.5	2587.4	NS
Sodium [mg]	2209.3	723.36	1258.7	4598.9	1810.9	429.73	925.6	2694.5	1948.6	721.70	955.0	3196.5	NS
Salt [g]	5.5	1.81	3.2	11.5	4.5	1.12	2.3	6.7	4.7	1.74	2.4	8.0	NS
Calcium [mg]	328.3	107.77	181.4	542.9	300.2	103.87	150.6	555.1	357.5	95.38	180.5	523.9	NS
Iron [mg]	6.1	1.64	4.3	11.6	6.3	1.56	4.1	11.5	6.4	0.99	4.9	8.1	NS

SD – standard deviation; Min – minimum; Max – maximum; b.m. – body mass; SFA – saturated fatty acids; MUFA – monounsaturated fatty acids; PUFA – polyunsaturated fatty acids

Table 6. Energy-adjusted macronutrient intake by genotype

Nutrient (g/day)*	Genotype TT (n = 18) Mean ± SD	Genotype TA (n = 34) Mean ± SD	Genotype AA (n = 12) Mean ± SD	Kruskal-Wallis test (p)
Protein	79.7 ± 11.99	78.6 ± 17.58	79.8 ± 11.11	NS
Fat	69.8 ± 14.62	63.0 ± 18.43	65.6 ± 10.20	NS
Carbohydrates	237.6 ± 34.12	256.4 ± 49.44	241.1 ± 32.02	NS

* Energy-adjusted using the residual method; SD – standard deviation

In contrast, no significant genotype-related differences were found in the current study for most anthropometric and body composition parameters. Although AA homozygotes tended to have higher body weight, waist and hip circumferences, and fat mass, these differences did not reach statistical significance, likely due to within-group variability and limited sample size. Extracellular water content was the only parameter associated with genotype, with higher values observed in AA homozygotes and a significant sex-related difference. As extracellular fluid volume has been linked to adiposity in obesity [22], this finding warrants cautious interpretation and confirmation in larger cohorts.

A novel aspect of this study was the analysis of familial patterns of overweight and obesity in relation to FTO genotype. A higher prevalence of self-reported excess body weight was observed among female relatives—mothers and grandmothers—of participants with TA/AA genotypes compared with TT homozygotes. Weak but significant correlations were also identified between excess body weight in siblings and mothers and carriage of the A allele. Despite reliance on self-reported data and modest effect sizes, these findings support familial clustering of obesity driven by shared genetic susceptibility and environmental exposures. The tendency toward higher prevalence along the maternal line suggests possible gene–environment interactions, although causal inferences cannot be drawn and further research is required.

Overall, the presence of the A allele may contribute to overweight and obesity risk, while maternal-line patterns indicate potential gene–environment interplay. Previous studies have reported associations between FTO risk alleles and dietary composition rather than total energy intake. A meta-analysis by Livingstone [23] showed a weak inverse relationship between the FTO risk allele and energy intake, as well as shifts in macronutrient distribution. Other studies observed higher fat intake [24], greater consumption of ultra-processed foods, adherence to Western dietary patterns [7], and stronger preferences for high-sugar foods among A allele carriers [9].

In line with earlier reports [8, 17], the present study found no associations between FTO rs9939609 genotype and total energy or nutrient intake. However, qualitative analyses revealed genotype-related differences in food choice, with AA homozygotes reporting lower consumption of nutrient-dense foods, such as fruits, vegetables, whole grains, dairy products, and fish. These findings partially align with reports of less favourable dietary behaviours among A allele carriers, although inconsistencies persist across populations.

The lack of associations with quantitative intake, alongside differences in dietary patterns, suggests that the influence of the rs9939609 polymorphism on obesity risk may extend beyond energy intake. Genotype-related differences in energy expenditure, metabolic efficiency, or nutrient utilization may play a role, as supported by studies reporting higher BMI or waist circumference among A allele carriers despite similar energy intake [17]. Consistently, principal component analysis indicated that the FTO rs9939609 polymorphism accounted for only a modest proportion (7.82%) of variability in nutritional status, supporting its role as a modulatory rather than dominant factor.

Despite the contribution of genetic susceptibility, genetic variation alone cannot explain the rapid rise in global obesity prevalence, as allele frequencies remain stable over time [25]. Obesity results primarily from long-term energy imbalance, and lifestyle factors—particularly diet and physical activity—remain central to prevention and management. Importantly, individuals with genetic predisposition can still achieve meaningful improvements through targeted lifestyle interventions.

Limitations and strengths of the study. The strengths of the study include the integrated assessment of genetic variation, dietary intake, nutritional status, and familial obesity patterns. It is among the first to examine the rs9939609 polymorphism in relation to both dietary behaviours and intergenerational overweight and obesity. Nevertheless, limitations include self-reported dietary data and the data on the occurrence of excess body weight in relatives, a predominantly female cohort with relatively high mean BMI due to purposive and snowball sampling, and limited sample size. Accordingly, the findings should be considered exploratory and interpreted with caution.

CONCLUSIONS

The study supports an association between the rs9939609 polymorphism of the FTO gene and increased susceptibility to overweight and obesity, particularly among A allele carriers. It also provides preliminary evidence that the prevalence of excessive body weight among relatives varies by FTO genotype, with higher occurrence among female relatives of A allele carriers, indicating the influence of shared genetic susceptibility and familial environment. Although no significant differences in quantitative dietary intake were observed between genotype groups, qualitative analyses revealed less favourable dietary patterns among AA homozygotes, characterized by lower intake of nutrient-dense foods.

Overall, the findings suggest that the FTO rs9939609 variant contributes modestly to obesity risk, likely through interactions with environmental and behavioural factors. Further studies in larger cohorts using objective measures of dietary intake and familial adiposity are warranted to clarify underlying mechanisms, and inform personalized obesity prevention strategies.

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