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Analysis of the impact of *FTO* (rs9939609) gene variants on anaerobic performance and body composition in active male adults: a preliminary study

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Abstract

Genetic variations can influence individual differences in sports performance by affecting energy metabolism, body composition, and adaptation to training. Among these genetic variants, *FTO* rs9939609 is particularly relevant due to its contribution to the regulation of body fat and appetite, with potential downstream effects on physical performance. This study examined whether the *FTO* rs9939609 variant was associated with aerobic endurance (Yo-Yo Intermittent Recovery Test Level 2) and anaerobic sprint performance (30 m sprint), and whether body mass index (BMI) was related to these outcomes. Sixty healthy, physically active males (18–22 years) underwent genotyping from buccal epithelial DNA samples and completed a six-week structured training program combining continuous running and whole-body muscular endurance circuit training. Performance was measured before and after training, and BMI was recorded. Genotype groups (A/A, A/T, T/T) were compared using ANOVA and ANCOVA controlling for BMI, height, and weight, and obesity risk was analyzed under dominant and recessive models using Fisher's exact test. No significant differences were found between genotypes for aerobic or anaerobic performance, and adjusting for anthropometric covariates did not alter this null result. Mean BMI was descriptively similar across genotype groups, and obesity odds did not differ significantly across genotypes; the T/T genotype was rare (n=4) and no T/T participants were classified as obese. No statistically significant associations were detected between *FTO* rs9939609 genotype and the assessed performance outcomes. in this sample. These findings should be considered preliminary given the small sample size and the limited number of T/T participants (n=4), which restrict the ability to detect modest genotype-related differences.

Keywords

BMI, physical performance, genotype, obesity, exercise genomics

Introduction

Exercise interventions, through regular and structured training loads, can produce meaningful changes in individuals' body composition, metabolic responses, and overall health status. However, the differences in physiological and morphological responses observed among individuals subjected to the same training protocol are closely related to individual variability in genetic makeup (Rankinen et al., 2006). Studies in sports genetics have increasingly identified associations between genetic variation and performance-related traits, suggesting that genetic information may contribute to research on individual differences in training response, body composition, and other performance-related characteristics. (Semenova et al., 2023).

Among the genetic markers involved in performance-related traits such as energy metabolism, adipose tissue distribution, and appetite regulation, the Fat Mass and Obesity-Associated Gene (*FTO*) stands out as an important candidate. The *FTO* rs9939609 polymorphism has been associated with various physiological parameters, including obesity susceptibility, energy balance, appetite control, fat oxidation, and exercise adaptation (Dina et al., 2007; Speakman et al., 2008). The *FTO* gene is located in the q12.2 region of chromosome 16 and is approximately 410,000 base pairs long (Vitti et al., 2013). The protein it encodes consists of approximately 505 amino acids and is classified as a 2-oxoglutarate (2-OG) and Fe(II)-dependent nucleic acid demethylase belonging to the AlkB family (Gerken et al., 2007).

The *FTO* rs9939609 polymorphism is an important genetic indicator of obesity risk. It's A/A, A/T, and T/T genotypes are reported to modulate responses to exercise type and intensity in different ways (Do Nascimento et al., 2019). Several studies report that A-allele carriers have higher energy intake, lower basal metabolic rate, and higher body fat percentage (Speakman et al., 2008; Li et al., 2010; Kaymak and Aral, 2014). T-allele carriers, in contrast, have been associated with lower fat mass and higher lean body mass and may possess body compositions more advantageous for anaerobic performance and strength sports (Frayling et al., 2007; Semenova et al., 2023). Regular physical activity has been shown to reduce the obesity risk of A-allele carriers by approximately 30% (Li et al., 2010).

FTO variants also appear to modulate responses to exercise type and intensity, and some studies indicate that dietary and aerobic-exercise interventions can partially offset the adverse body-composition effects in A-allele carriers (Do Nascimento et al., 2019). Work on elite swimmers and athletes from various disciplines suggests these genotypic differences may produce effects that vary by discipline, and that the gene's contribution to performance may operate at the behavioral as well as the physiological level (Heni et al., 2014). Together, these findings indicate that the variant may influence not only obesity but also exercise responses and performance, and that genotype should be considered in individualized training and nutrition, since lifestyle intervention remains important despite genetic predisposition.

The *FTO* rs9939609 polymorphism establishes multifaceted biological links among fat metabolism, muscle mass, energy balance, and exercise responses. These links offer a potential tool for genotype-based prediction of physical fitness and sports performance, and accounting for *FTO* genotype when designing personalized exercise and nutrition programs may be a valuable

strategy for both performance development and long-term metabolic health. Accordingly, this study aimed to evaluate the potential effects of the *FTO* rs9939609 variant on anaerobic/aerobic performance (30 m sprint test; Yo-Yo Intermittent Recovery Level 2 Test) and BMI in healthy, regularly physically active adult men aged 18–22 years.

Material and methods

Participants and study design

The study cohort consisted of healthy, physically active men aged 18–22 years who engaged in regular exercise. The study was approved by the Lokman Hekim University Scientific Research Ethics Committee (decision no. 2025/046). Genotype, sex, age, body weight, and body composition (BMI) were treated as independent variables, while the dependent variables were the physical-performance test results (30 m sprint; Yo-Yo IR2 test) obtained before and after a 6-week training program. A pre-test–post-test design was used, and performance changes were analyzed by comparing baseline and follow-up measurements (Hopkins, 2000; Thomas et al., 2023).

Sample size and instruments

A sample-size calculation using G*Power 3.1 for a one-way ANOVA with three groups, an assumed effect size of f=0.50, α=0.05, and a target power of 0.95 indicated a required total sample size of 66 participants. A total of 66 volunteers were initially enrolled. One participant withdrew during the study, and genetic samples from five participants could not be analyzed for technical reasons, leaving 60 men for the final analysis (A/A=26, A/T=30, T/T=4).

Participants were physically active men who regularly trained in various sports disciplines. All procedures were organized by appointment and around the participants' training schedules. Participants were informed both verbally and in writing about the measurement procedure and the study framework at least one week beforehand, and written informed consent was obtained using an "Informed Voluntary Consent Form." For genetic analysis, buccal epithelial swab samples were collected before testing using sterile, medium-free swabs designed to prevent bacterial growth, and were analyzed in collaboration with the Damagen Medical Genetics Laboratory (Damagen Genetic Diagnostic Center, Ankara). Height was measured barefoot, upright, with the head in the Frankfurt plane using a fixed stadiometer (SECA 213, Germany) to ±0.1 cm, and body weight in light clothing without shoes using a digital medical scale (SECA 877, Germany) to ±0.1 kg (Norton and Olds, 1996).

Training intervention

Throughout the study, participants underwent a structured training program three days per week for six consecutive weeks. Each session comprised aerobic and anaerobic threshold training (45 minutes of moderate- to low-intensity continuous running) to enhance cardiorespiratory endurance, combined with a whole-body circuit-training protocol targeting muscular endurance. The circuit consisted of 12 sequential exercises performed in three sets: squat jumps, push-ups, walking lunges, bent-over rows with dumbbells, plank hold, medicine-ball chest pass, step-ups, mountain climbers, overhead press with dumbbells, side planks, burpees, and Russian twists. Each exercise was performed for 30 seconds, with a 5-minute

rest between sets. Total session duration ranged from 75 to 90 minutes.

Yo-Yo Intermittent Recovery Test Level 2 (Yo-Yo IR2)

The Yo-Yo IR2 is a field-based test developed to assess aerobic and anaerobic energy-system capacity and, in particular, short-term recovery ability in sports requiring intermittent high-intensity effort. The protocol consisted of 20 m out-and-back shuttle runs with a 10 s active recovery between repetitions. Participants ran in synchrony with audio signals; testing was terminated when a participant could no longer keep pace with the signals, and the total distance covered was recorded as an indirect indicator of endurance. Although the test does not directly measure maximal oxygen uptake (VO_2max), the distances obtained correlate highly with VO_2max and are therefore accepted as an indirect measure of endurance (Krustrup et al., 2003; Bangsbo et al., 2008). The test was administered at the beginning and end of the training period, and the change in distance between the two measurements was used to evaluate endurance improvement.

30 m sprint test

The 30 m sprint test was used to assess maximal short-distance sprint performance, a field-based protocol widely used to evaluate anaerobic power and speed. To prevent environmental conditions (e.g., wind, temperature) from affecting results, testing was conducted in an indoor athletics facility. Times were recorded with a high-accuracy photocell-gate system (Witty Speed, Microgate, ITA), with gates placed at the start (0 m) and finish (30 m). Before testing, participants completed a 10-minute dynamic warm-up (light jogging, change of direction, dynamic stretching, and mobilization) followed by 3 minutes of passive rest. Each participant performed two trials separated by at least 5 minutes of rest, and the faster time was used in the analysis.

DNA extraction and genotyping of FTO rs9939609 polymorphism

Buccal epithelial samples were collected using sterile swabs, and molecular analyses were performed at the Damagen Genetic Diagnostic Center (Ankara, Türkiye). Genomic DNA was extracted using the Buccalyse DNA Extraction Kit (Isohelix, UK) according to the manufacturer's instructions. DNA concentration was measured using a NanoDrop spectrophotometer (Thermo Fisher Scientific, USA). The *FTO* rs9939609 polymorphism was genotyped using Kompetitive Allele-Specific PCR (KASP; LGC Genomics), following the KASP genotyping methodology described by He et al. (2014). The SNP-specific assay contained two allele-specific forward primers and one common reverse primer. Reactions were performed using KASP Master Mix on an Applied Biosystems 7500 Real-Time PCR System, and genotypes were assigned from allele-specific fluorescence signals. Participants were classified as A/A, A/T, or T/T.

Statistical analyses

All statistical procedures were performed in R version 4.5.1 (R Core Team, 2025). Statistical significance was set at $p < 0.05$ (two-tailed). Distributional properties were examined through visual inspection of Q–Q plots, and homogeneity of variances via Levene's test. When assumptions

of normality and homogeneity were met, between-group comparisons of the *FTO* genotype groups (A/A, A/T, T/T) for Yo-Yo IR2 and 30 m sprint were conducted using one-way analysis of variance (ANOVA). Within-group pre–post differences were evaluated with paired-samples t-tests, and between-genotype differences in change (Δ) using ANOVA on change scores. Where appropriate, BMI, stature, and body mass were entered as covariates in analyses of covariance (ANCOVA). When assumptions were violated, between-group comparisons used the Kruskal–Wallis test, within-group changes the Wilcoxon signed-rank test, and heteroscedastic data Welch's ANOVA. Dominant and recessive genetic models were constructed to evaluate associations between genotype and obesity status (BMI≥25 kg/m²). Categorical comparisons were performed using two-sided Fisher's exact tests. Odds ratios (ORs) were estimated using conditional maximum likelihood, with exact 95% confidence intervals.

Results and discussion

Descriptive statistics by FTO genotype (A/A, A/T, T/T) for Yo-Yo IR2 performance, 30 m sprint time, BMI, height, and body mass are presented in Table 1. While mean Yo-Yo IR2 and 30 m sprint values were similar across groups, the T/T genotype displayed a lower mean BMI than the A/T and A/A carriers. Between-genotype comparisons at pre-intervention, post-intervention, and as change (Δ) scores are shown in Table 2, and no statistically significant differences were observed among the genotype groups for either outcome ($p>0.05$). The ANCOVA results with height, body mass, and BMI as covariates are given in Table 3: no significant genotype effect emerged for either outcome ($p>0.05$), whereas body mass ($p<0.001$) and BMI ($p<0.05$) were significant at both time points. Obesity status across genotypes and under dominant and recessive models is summarized in Table 4; 30.8% of A/A, 43.3% of A/T, and 0% of T/T individuals were obese, and obesity odds did not differ across genotypes.

The principal finding of this study is that *FTO* genotypes (A/A, A/T, T/T) were not associated with Yo-Yo IR2 or 30 m sprint performance at baseline, post-intervention, or as change (Δ) scores (all $p>0.05$; Tables 2–3). In covariate-adjusted models, genotype effects remained non-significant, whereas body mass and BMI emerged as consistent predictors of pre- and post-test values (Yo-Yo IR2: body mass $p<0.001$, BMI $p<0.05$; 30 m sprint: body mass and BMI $p<0.001$); adjusting for these covariates did not alter the null genotype effect (Table 3), and genotype also did not predict change scores for either outcome (Table 2). Descriptively, the T/T group showed lower mean BMI and body mass and no cases of obesity; however, this subgroup was very small (n=4), and the corresponding obesity estimate (OR=0.00, based on a zero cell) is highly imprecise and should not be over-interpreted. Obesity odds did not differ across genotypes (A/T vs A/A OR=1.70, 95% CI 0.50–6.06, $p=0.412$; Table 4). In this sample, body mass and BMI were associated with performance in the adjusted models, whereas no statistically significant between-genotype differences in six-week performance changes were detected.

Consistent with this null finding, the broader literature indicates that *FTO* variation alone is unlikely to be a decisive determinant of aerobic fitness or recovery in active cohorts. While the A allele has been proposed to influence aerobic capacity indirectly via greater fat mass and lower lean mass (Loos and Yeo, 2014; Qi et al., 2012),

studies in trained athletes similarly report no meaningful associations between rs9939609 and endurance indices (Heffernan et al., 2017). In our cohort, BMI did not differ materially across genotypes, consistent with evidence that regular training and diet can attenuate genotype–phenotype expression in body composition (Huuskonen et al., 2012; Phillips et al., 2012), with the *FTO*–BMI link weakened in active individuals (Hubáček et al., 2008; Mier-Mota et al., 2023). Indeed, large-scale gene–environment work shows that habitual physical activity substantially attenuates the adiposity effect of the A allele (Li et al., 2010), potentially through lower visceral adiposity and metabolic adaptation (Do Nascimento et al., 2019).

Table 1. Descriptive statistics of Yo-Yo IR2, 30-m sprint, BMI, height, and body mass by *FTO* genotype (n = 60)

Variable	A/A (n=26)	A/T (n=30)	T/T (n=4)
Yo-Yo IR2 (m)	552±195 (200–840)	549±203 (160–920)	535±358 (280–1040)
30-m sprint (s)	5.32±0.56 (4.45–6.50)	5.44±0.64 (4.26–6.77)	5.39±0.77 (4.36–6.00)
BMI (kg/m²)	23.12±2.98 (16.60–28.40)	23.78±2.69 (17.70–28.70)	20.57±1.49 (19.10–22.00)
Height (m)	1.82±0.10 (1.67–2.03)	1.81±0.12 (1.60–2.08)	1.76±0.17 (1.65–2.01)
Body mass (kg)	76.5±12.1 (48–92)	78.5±16.4 (53–116)	64.8±17.2 (52–89)

Values are mean±SD (minimum–maximum). BMI-body mass index; SD-standard deviation; Yo-Yo IR2, Yo-Yo Intermittent Recovery Test Level 2.

Table 2. Between-genotype comparisons of Yo-Yo IR2 and 30-m sprint performance (one-way ANOVA)

Outcome / group	n	Pre-test mean	Pre-int. (F; p)	Post-int. (F; p)	Δ Post–Pre (F; p)
Yo-Yo IR2 (m)			0.011; 0.968	0.553; 0.578	1.335; 0.271
A/A	26	551.54			
A/T	30	549.33			
T/T	4	535.00			
30-m sprint (s)			0.272; 0.763	0.194; 0.824	2.569; 0.085
A/A	26	5.32			
A/T	30	5.44			
T/T	4	5.39			

Pre-int.-pre-intervention; Post-int.-post-intervention; Δ-change score (post–pre). F and p values represent between-genotype comparisons using one-way ANOVA (df=2, 57). Negative sprint change scores indicate improved performance; positive Yo-Yo IR2 change scores indicate improved performance.

Table 3. ANCOVA for Yo-Yo IR2 and 30-m sprint by *FTO* genotype, with height, body mass, and BMI as covariates

Model	FTO (F; p)	Height (F; p)	Body mass (F; p)	BMI (F; p)
Pre Yo-Yo IR2	0.13; 0.876	3.06; 0.086	19.92; <0.001***	5.01; 0.029*
Post Yo-Yo IR2	0.34; 0.712	1.91; 0.173	20.70; <0.001***	7.44; 0.009**
Pre 30-m sprint	0.68; 0.513	3.09; 0.085	35.97; <0.001***	13.01; <0.001***
Post 30-m sprint	0.39; 0.680	3.04; 0.087	25.53; <0.001***	12.56; <0.001***

ANCOVA-analysis of covariance; BMI-body mass index. Models included FTO genotype, height, body mass, and BMI. Degrees of freedom were 2, 54 for genotype and 1, 54 for each covariate. *p<0.05; **p<0.01; ***p<0.001.

Table 4. Obesity status (BMI ≥ 25 kg/m²) across FTO genotypes and genetic models (Fisher's exact test)

Model / group	Non-obese (n)	Obese (n)	Total (n)	Obese (%)	OR (95% CI)	p
Genotypes						
A/A	18	8	26	30.8%	Ref	
A/T	17	13	30	43.3%	1.70 (0.50–6.06)	0.412
T/T	4	0	4	0.0%	0.00 (0.00–4.24)	0.550
Dominant model (A/A + A/T vs T/T)						
A carriers (A/A+A/T)	35	21	56	37.5%	Ref	
T/T	4	0	4	0.0%	0.00 (0.00–2.78)	0.287
Recessive model (A/A vs A/T + T/T)						
A/A	18	8	26	30.8%	Ref	
T carriers (A/T+T/T)	21	13	34	38.2%	1.39 (0.42–4.80)	0.595

OR-odds ratio; CI-confidence interval; Ref-reference group. Each comparison uses the reference group indicated within its section. Odds ratios are conditional maximum likelihood estimates with exact 95% confidence intervals; p values are from two-sided Fisher's exact tests. Zero odds ratios reflect zero events in the corresponding group.

The 30 m sprint likewise showed no significant genotype differences at baseline or after training (Table 2). Heffernan et al. (2017) similarly emphasized that *FTO* polymorphisms do not exert a direct effect on anaerobic performance, and the wider evidence suggests that although *FTO* is linked to energy metabolism, its effect on sprint-type tests is limited (Frayling et al., 2007; Loos and Yeo, 2014). Descriptively, mean BMI was also similar across genotype groups (Table 1).

Although A-allele carriers have been reported to show higher adiposity and lower lean mass, and T/T carriers a more favorable body-composition and metabolic profile with stronger exercise adaptations (Do Nascimento et al., 2019; Zmijewski and Leńska-Duniec, 2021), these putative mechanisms did not translate into measurable performance differences in our sample. Proposed neurobiological and appetite-related pathways linked to the A allele (Tanofsky-Kraff et al., 2009; Heni et al., 2014) may influence long-term exercise adherence rather than acute performance, and structuring programs to support metabolic adaptation may still be relevant for A-allele carriers (Kilpeläinen et al., 2011; Loos and Yeo, 2014). These mechanisms were not directly

assessed in the present study, and their potential contribution to genotype-related differences in training responses remains uncertain.

Conversely, some studies contradict our findings: Cecil et al. (2008) reported that young sedentary A-allele carriers lagged in aerobic performance owing to higher energy intake. These conflicting results demonstrate how strongly the effect of *FTO* genotype interacts with lifestyle, with weaker effects expected in trained individuals and stronger effects in sedentary or high-obesity-risk groups (Qi et al., 2012). The absence of a clear BMI difference across genotypes may indicate that body composition was kept under control through training; regular physical activity and nutritional awareness in active men may stabilize BMI differences and make genotype-related differentiation harder to detect in athletic samples.

A further point concerns measurement reliability. The Yo-Yo IR2 is widely accepted as valid for assessing aerobic endurance–recovery capacity (Krustrup et al., 2003), and the 30 m sprint is a common field protocol for short-duration anaerobic performance (Haugen et al., 2013). Because test results can be affected by motivation, measurement surface, weather, and current psychological state, these potential limitations should be considered. Overall, no statistically significant associations between *FTO* rs9939609 genotype and the assessed performance outcomes were detected in this sample of physically active young men.

Limitations

Because all participants were active adult men with similar physical-activity levels, training histories, and lifestyles, the homogeneity of the group may have limited the observable variation in genetic effects. Only the *FTO* rs9939609 polymorphism was examined, so other performance-relevant variants were not considered. Environmental variables such as diet, sleep quality, and stress were not systematically controlled, which complicates isolation of genetic effects. The six-week follow-up period limited the assessment of longer-term associations between *FTO* rs9939609 genotype and training responses. Finally, the sample - and in particular the rare T/T genotype (n=4) - may have been too small to detect small between-genotype effects, so the results should be regarded as preliminary.

Conclusion

No statistically significant differences were detected among *FTO* rs9939609 genotype groups in Yo-Yo IR2 or 30-m sprint performance at baseline, after training, or in changes over the six-week intervention. These findings remain preliminary because of the small sample size, particularly the T/T subgroup (n=4), and do not exclude modest genotype-related associations. Genetic makeup is an important factor in performance capacity, but its effect is shaped by environmental factors and lifestyle habits; athletic performance therefore cannot be explained by genetic predisposition alone, and training history, motivation, nutrition, and psychosocial factors must also be considered.

To better understand the relationship between sports performance and genetic variants, future studies should use more heterogeneous samples spanning different ages, sexes, sports disciplines, and training levels, and should evaluate multiple polymorphisms simultaneously, since single-gene analyses have limited explanatory power.

Incorporating epigenetic, hormonal, and psychosocial variables would further support individualized performance planning.

Authors' contributions

M. Keskin:
Conceptualization; Investigation; Data curation; Methodology; Writing – original draft.

M. Cerit:
Conceptualization; Methodology; Supervision; Validation; Writing – review & editing.

A. Şamil Önlü:
Formal analysis; Visualization; Project administration; Supervision; Writing – review & editing.

Conflict of interest

Authors declare no conflict of interest.

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