

Study of the effect of FTO gene polymorphisms on physical performance indicators and the response to weight loss programs in obese athletes after a period of cessation of training

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ABSTRACT

Introduction. Polymorphisms in the fat mass and obesity-associated (FTO) gene, particularly the A allele, are associated with obesity and may reduce responsiveness to exercise programs. Investigating their effect on physical performance and weight loss in obese athletes after training cessation is important for tailoring effective interventions. **Aim of Study.** To examine the influence of FTO gene polymorphisms on physical performance indicators and weight loss outcomes in obese athletes following an 8–12-week structured training program after a period of inactivity. **Material and Methods.** Thirty obese Iraqi athletes (20–50 years) were genotyped for the FTO rs9939609 variant (AA, AT, TT) using PCR. Baseline and post-program assessments included body weight, BMI, body fat percentage, VO_{2max} , muscle strength, flexibility, and cardiac recovery rate. The training combined aerobic, resistance, flexibility, and balance exercises. Data were analysed using ANOVA with post hoc tests ($p < .05$). **Results.** TT genotype carriers had the greatest reductions in weight (–4.5 kg) and body fat percentage (–4.8%), and the largest improvements in VO_{2max} (+5.3 ml/kg/min), muscle strength (+4.6 kg), flexibility (+3.5 cm), and recovery rate (–12 bpm). AA genotype carriers showed the least improvement across all measures. **Conclusions.** FTO gene polymorphisms were associated with differential responses to weight loss and performance improvements, with TT carriers exhibiting the most favourable adaptations. Inco (Hashim et al., 2024, pp. 684-692) prorating genetic testing into training design may support the development of more individualized and potentially more effective exercise programs, particularly for individuals carrying the A allele. In practical terms, coaches and clinicians might consider adjusting training intensity and nutrition plans for A-allele carriers, focusing on enhancing metabolic flexibility and satiety signalling. The study was approved by the Research Ethics Committee of the University of Samarra, and all participants gave informed consent.

Keywords: Sport medicine, Physical performance, Weight loss, Aerobic training, TO gene, Genetic profiling.

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INTRODUCTION

As Ahmed, a promising young athlete, stared at his reflection in the gym's mirror, he felt a surge of determination mixed with anxiety. After months away from the track due to an ankle injury, the weight he had gained during the downtime now felt like an insurmountable obstacle. The once easy laps around the field now seemed daunting. This struggle is not unique to Ahmed; many people around the world use terms such as obesity, chubby, puffy, fleshy, and overweight to describe bioaccumulation in the body. These terms differ scientifically in their descriptions of this phenomenon (Al Daghri et al., 2014). This phenomenon is not limited to humans; it can also be observed in animal societies (Arendas et al., 2008). Obesity is a biological condition characterized by excess fat accumulation. This fat is rarely used as a source of energy due to reduced activity and individual tendencies to avoid movement, work, and environmental interaction (Andreasen et al., 2008, Choi et al., 2005). The World Health Organization (WHO) considers obesity a major population health problem, describing it as a rapidly growing global epidemic of overweight people in many parts of the world (Clausnitzer et al., 2015).

A report by the Organization for Economic Co-operation and Development (OECD) indicates obesity rates ranging from 4% in Japan to 30% or more in the United States and Mexico (Farooqi, 2006). This phenomenon accounts for 1–3% of healthcare costs in most countries worldwide and 5–10% in the United States, amounting to approximately 25% of healthcare costs for a healthy individual. (The Economic Impact of Overweight & Obesity in 2020 and 2060 (2nd Edition).

In general, obesity has been associated with a medical condition requiring healthcare in most countries (Flynn et al., 2006). It is a social phenomenon resulting from overnutrition (Hiraike et al., 2021) and inactivity, as well as its association with the quality and quantity of food consumed. This differentiation can enhance the understanding of distinct drivers of obesity, paving the way for targeted interventions that address both genetic predispositions and lifestyle modifications.

Limiting the evaluation of this phenomenon to nutritional status is a weakness in scientific explanation, because the mechanisms of food (hunger and satiety) are subject to complex hormonal coordination and under the control of the central nervous system (Howard et al., 2008). These mechanisms operate under the influence of genetic factors in the normal state, and in many abnormal cases, obesity is unrelated to the nutritional process for various reasons, such as pathological, disease-related, medication-induced (Kim et al., 2016), or linked to psychological state and social prosperity (Leońska-Duniec et al., 2018). Obesity is a complex disease caused by a combination of genetic and environmental factors, and numerous studies have shown that genetic predisposition varies across ethnic groups (Manson et al., 1995).

The phenomenon of obesity has received widespread scientific attention since the late 20th century. Among the most important studies were those addressing the genetic basis of obesity, including the identification of specific genes and the mapping of the human genome (Mitchell et al., 2010, Morton et al., 2006). These studies looked at different groups of people like kids, women, and adults.

Besides the obvious environmental factors such as a fat-rich diet and sedentary lifestyle that cause obesity, genetic factors precipitate or make someone vulnerable to accumulating body fat. In this regard, one of the most scientifically validated is the Fat mass and obesity-associated (FTO) gene in its mutant forms like A allele which upregulates enzymes' expression among them alpha-ketoglutarate dependent dioxygenase changing energy metabolism into enhanced storage of fat (Neovius et al., 2009).

The FTO gene has recently turned out to become an area of increasing interest within the field of sports science, due to its potential implications on physical performance, body composition and response towards training and weight loss programs. Some researchers have identified that obese athletes may or may not benefit from training and nutritional programs based on their genetic makeup particularly genes related with metabolism and appetite regulation (Organization for Economic Co-operation and Development [OECD], 2012).

Athletes who return to training after a period of layoff due to any reason, health, injuries, or personal commitments provide an excellent model for genetic studies. Detraining can be considered as a 'stress test' for the effects of genotypes because it tests the ability of the body to maintain adaptations and performance in the absence of regular exercise stimuli.

Most commonly observed are losses in muscle mass accompanied by gains in fat mass together with decreases in aerobic capacity, strength, and endurance which effectively highlight the impacts of genetic predispositions. The knowledge about how much differences in the FTO gene affect these changes will guide more targeted interventions. Recent evidence shows that high-risk FTO variant carriers have reduced fat oxidation and energy conversion during exercise. This leads to worse training outcomes (Ponce-González et al., 2023). These individuals may also regain weight more easily after detraining, showing the need for individualized strategies.

Many studies have examined the effects of detraining on physiological variables. Based on these gaps, we aimed to analyse the effects of FTO gene polymorphisms on physical performance indicators and responsiveness to weight loss programs in obese athletes during a period of inactivity. The study seeks to aid genetically oriented sports and nutritional intervention program designs.

Aim of study

The present study has been designed with the following specific objectives: to determine the influence of FTO gene polymorphisms on physical performance parameters and effectiveness of weight loss programs in obese athletes after a period of training cessation, to find out the association between different variants of FTO gene with changes in body composition, aerobic capacity and muscle strength after a break in training.

1. Identify the connection between FTO gene variants and modifications in body composition, aerobic capacity, and muscle strength following a break in training.
2. Assess the influence of these genes on fat burning, energy efficiency, and predisposition for weight gain during and after organized training.
3. Establish scientifically proven personalized sports and nutritional strategies based on athletes' genotype to enhance weight-loss results as well as performance recovery.

This shall be the objective, therefore increasing knowledge on interactions between genes and exercise to support precision-based interventions concerning sports science as well as the management of obesity. This study aims to investigate the influence of FTO gene polymorphisms on physical.

MATERIALS AND METHODS

FTO gene changes in relation to differences in physical performance and weight loss among obese athletes after training cessation were comparatively studied between May 2024 and October 2024, in Tikrit and Samarra/Iraq. Participants were selected by the most standardized method of selection procedures

accompanied by baseline assessments before starting the actual training program or intervention (gene analysis). All assessments had been conducted at least two weeks prior to commencing any form of structured physical activity so as not to allow for acute effects resulting from previous exercises on measured parameters. The study is quasi-experimental because there has been no random assignment of subjects into different groups; hence it can also be classified under comparative studies.

Study participants

The research was carried out from May 2024 to October 2024. It covered both males and females with obese bodies who stopped training for not less than three months, collected from different sports centres in Tikrit and Samarra. Thirty participants were selected according to the WHO classification criteria of BMI for obesity. The comparisons made in the research design were only within the same sample between genotypes (AA, AT, TT), without any separate control group.

The inclusion criteria were:

- Age between 20–50 ± 2 years.
- No history of cardiovascular disease, diabetes, or genetic disorders unrelated to obesity.
- No use of weight-loss medications.

A structured questionnaire was used to collect demographic and training-related information from all participants.

The sample size ($n = 30$) was determined as the minimum required to detect genotype-related differences in physiological and anthropometric variables, with a statistical power of 0.80 and a significance level of $p \leq .05$, consistent with previous studies examining FTO polymorphisms in athletic populations. Effect sizes were calculated using eta squared (η^2) to determine the magnitude of genotype-related differences, with values interpreted according to established thresholds (small: 0.01, medium: 0.06, large: 0.14). Baseline and post-intervention anthropometric characteristics of participants according to FTO genotype are presented in Table 1.

Table 1. Baseline and post-intervention anthropometric characteristics of participants according to FTO genotype (mean ± SD).

Risk of developing health problems	BMI category (kg/m²)	Classification
Increased	18.5 and below	Slim
Least possible	18.5 – 24.9	Normal weight
Increased	25 – 29.9	Overweight
High	30 – 34.9	Class I obesity
Very high	35 – 39.9	Class II obesity
Excessive	40 and above	Class III obesity

Statistical analysis

Data were analysed using SPSS version 26.0. The normality of distribution was assessed using the Shapiro–Wilk test, and Levene’s test was applied to confirm the homogeneity of variances. Differences between the three genotypes (AA, AT, TT) were evaluated using one-way ANOVA followed by Tukey’s post hoc test. Pre- and post-training comparisons within each genotype group were determined using paired-sample t-tests.

Effect sizes were reported using partial eta-squared (η^2) to determine the magnitude of differences. Statistical significance was set at $p < .05$ for all tests.

Collection of personal and baseline data

A standardized questionnaire was used to gather:

- Age (years).
- Gender.
- Type of specialized sport prior to cessation.
- Duration of cessation from training (weeks/months).
- Duration of gradual return to training prior to the research program.

Baseline physiological and physical assessments

Before the training program, all participants underwent the following measurements:

- Anthropometric Measurements:
 - Weight (kg).
 - Height (cm).
 - Body Mass Index (BMI) is calculated as weight (kg) divided by height² (m²).
 - Body fat percentage using a body composition analyser (e.g., InBody).

Physical performance indicators

- Aerobic endurance: measured via maximum oxygen consumption (VO_{2max}) test.
- Muscular strength: assessed using handgrip dynamometry.
- Flexibility: assessed using the sit-and-reach test.
- Cardiac recovery rate: measured as heart rate recovery one minute after aerobic exercise.

Genetic analysis of FTO gene polymorphisms

Genotyping of the FTO rs9939609 polymorphism was performed using polymerase chain reaction (PCR) followed by allele-specific analysis. DNA was extracted from peripheral blood samples using a commercial extraction kit (Qiagen, Germany), and PCR amplification was conducted in a thermal cycler using the following conditions: initial denaturation at 95°C for 5 minutes, 35 cycles of 95°C for 30 seconds, 58°C for 30 seconds, and 72°C for 45 seconds, followed by a final extension at 72°C for 5 minutes.

Training program intervention (8–12 Weeks)

Grouped according to genotype, participants underwent a standardized training program as follows:

- Cardio workouts: brisk walking, stationary cycling, and light jogging.
- Strength training: free weights or resistance machines.
- Flexibility and balance training: dynamic and static stretching exercises.

The program ran three times weekly for a period between eight and twelve weeks with each session within sixty to seventy-five minutes. Participants, grouped according to genotype, underwent a standardized training program. In the first weeks, exercises were conducted at sixty to seventy-five percent of the maximum heart rate gradually increasing intensity in succeeding weeks as explicitly guided by individual enhancements.

Post-program measurements

Upon program completion, all baseline measurements were repeated:

- Anthropometric indicators (weight, BMI, body fat percentage).
- Physical performance indicators (VO_{2max} , strength, flexibility, recovery rate).

- Comparison of pre- and post-intervention values among different genotype groups.

Ethical considerations

The study was approved by the Research Ethics Committee, College of Physical Education and Sports Sciences, University of Samarra, Iraq (Form Number: 4; Decision Date: September 16, 2025), and conforms to institutional ethical standards and the principles laid down in the Declaration of Helsinki. Written informed consent was obtained from all participants before their enrolment in the research that took place from May 2024 until October 2024.

RESULTS

The results registered improvements in the values of all physical performance indicators after the training intervention, depending on the three FTO genotypes (AA, AT, TT). Genotype distribution Genetic analysis showed genotype distribution among participants for polymorphism FTO (rs9939609) as follows:

- AA genotype: 10 participants (33.3%).
- AT genotype: 12 participants (40%).
- TT genotype: 8 participants (26.7%).

Table 2. Arithmetic means for weight and fat percentage.

Genotype	Fat % Before	Fat % After	Change (%)	Weight Before (kg)	Weight After (kg)	Change (kg)
AA	33.5 ± 2.4	31.8 ± 2.6	-1.7	84.2 ± 3.1	81.5 ± 3.4	-2.7
AT	32.1 ± 2.0	28.5 ± 1.9	-3.6	82.6 ± 2.9	78.4 ± 2.5	-4.2
TT	30.8 ± 1.7	26.0 ± 1.5	-4.8	80.1 ± 3.0	75.6 ± 2.8	-4.5

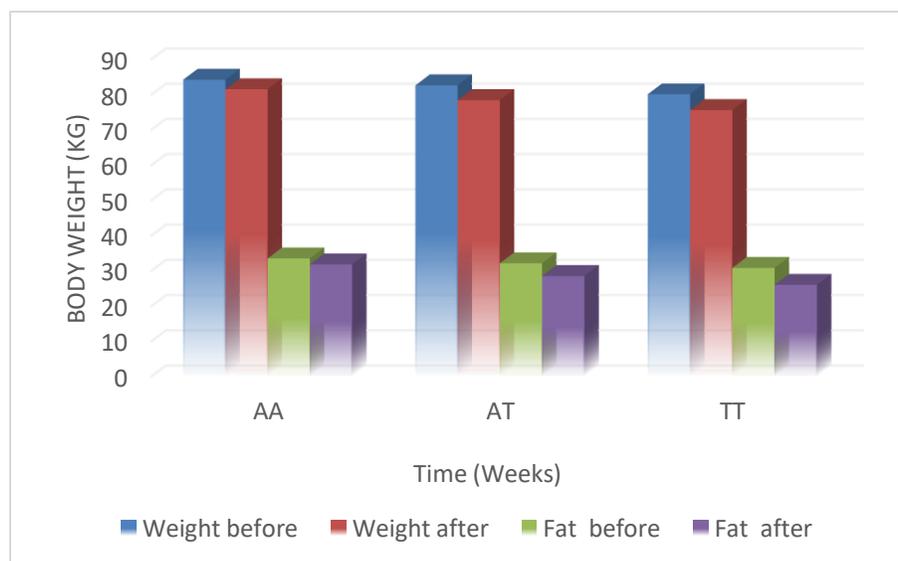


Figure 1. Changes in body weight and fat percentage across FTO genotypes before and after the intervention.

This pattern is consistent with reports in Arab populations where the A allele is relatively prevalent among individuals with obesity. In particular, studies involving Iraqi populations reveal a similar trend, highlighting the role of genetic predisposition in regional obesity rates. Such prevalence may reflect an underlying genetic predisposition to weight gain in the sampled population.

Changes in weight and fat percentage

Pre- and post-intervention values for weight and fat percentage are presented in Table 2 and illustrated in Figure 1.

- TT genotype participants achieved the greatest reductions, with decreases of -4.5 kg in body weight and -4.8% in body fat.
- AT genotype participants followed, with reductions of -4.2 kg and -3.6%, respectively.
- AA genotype participants showed the smallest changes (-2.7 kg and -1.7%).

The trend indicates that TT carriers exhibit the most favourable response to body composition interventions, whereas AA carriers display a comparatively resistant profile, potentially linked to allele-specific regulation of FTO expression and energy homeostasis (Rahimi & Symonds, 2025).

Physical performance enhancements

The effects of the intervention on VO_{2max} , muscle strength, flexibility, and recovery rate are presented in Figure 2 and summarized in Table 3.

Table 3. Post hoc comparisons.

Variables	Comparison	Mean Difference	SE	p-Value	Significance
Weight	AA vs AT	1.6	0.65	.023	$p < .05$
	AA vs TT	2.1	0.70	.010	$p < .05$
	AT vs TT	0.5	0.68	.450	Not significant
Fat Percentage	AA vs AT	1.4	0.55	.018	$p < .05$
	AA vs TT	2.3	0.60	.005	$p < .01$
	AT vs TT	0.9	0.58	.120	Not significant
VO_{2max}	AA vs AT	1.3	0.50	.030	$p < .05$
	AA vs TT	1.9	0.53	.008	$p < .01$
	AT vs TT	0.6	0.52	.380	Not significant
Muscle Strength	AA vs AT	1.1	0.58	.060	Not significant
	AA vs TT	2.4	0.62	.012	$p < .05$
	AT vs TT	1.3	0.60	.100	Not significant
Flexibility	AA vs AT	1.3	0.54	.080	Not significant
	AA vs TT	2.8	0.57	.005	$p < .01$
	AT vs TT	1.5	0.56	.090	Not significant
Recovery	AA vs AT	6.0	2.10	.040	$p < .05$
	AA vs TT	8.0	2.30	.020	$p < .05$
	AT vs TT	2.0	2.20	.300	Not significant

 VO_{2max}

- TT genotype: +5.3 ml/kg/min.
- AT genotype: +4.4 ml/kg/min.
- AA genotype: +2.4 ml/kg/min.

Muscle strength (hand grip)

- TT genotype: +4.6 kg.
- AT genotype: +3.7 kg.
- AA genotype: +2.3 kg.

Flexibility

- TT genotype: +3.5 cm.
- AT genotype: +2.7 cm.
- AA genotype: +1.9 cm.

Recovery Rate (decrease in heart rate one minute after exercise cessation)

- TT genotype: -12 bpm.
- AT genotype: -10 bpm.
- AA genotype: -8 bpm.

The results show a clear interaction between genotype and performance, with TT carriers showing greater improvements in both aerobic and strength training adaptations. AA carriers showed minimal improvements, mainly in aerobic endurance and cardiac recovery.

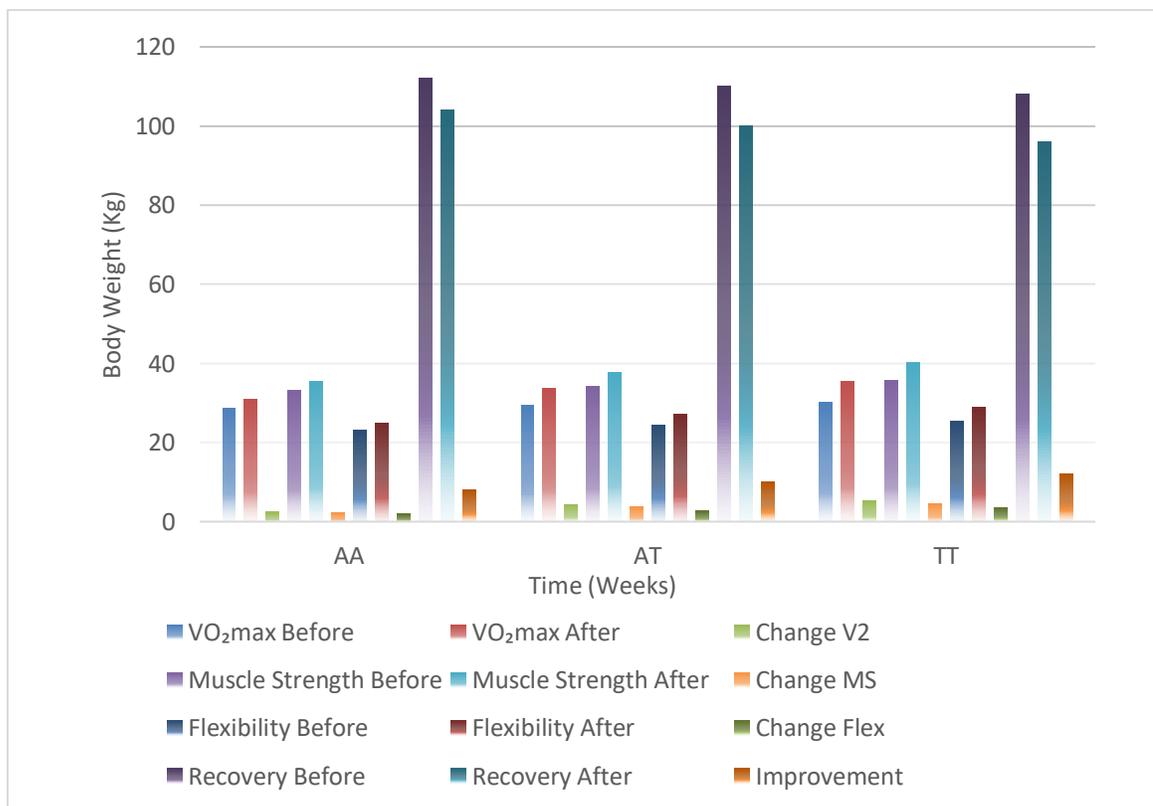


Figure 2. Pre- and post-intervention differences in VO₂max, muscle strength, flexibility and recovery rate across FTO genotypes.

Post hoc comparisons

Pairwise comparisons between genotypes were performed for all measured variables (Table 3).

Weight & fat percentage

Post-intervention weight in the AA group was observed to be significantly higher than both the AT ($p = .023$) and TT ($p = .010$) groups. Similarly, fat percentage remained significantly higher in AA participants compared

to AT ($p = .018$) and TT ($p = .005$). No significant differences were observed between AT and TT for these variables.

Aerobic capacity (VO_{2max})

The increase in VO_{2max} was much TT than AA ($p = .008$) and AT ($p = .030$). There was not AT and TT.

Muscle strength

TT carriers showed a significantly greater increase in muscle strength than AA ($p = .012$), while the differences between AA–AT and AT–TT were not statistically significant.

Flexibility

- TT carriers showed markedly greater improvements in flexibility than AA carriers ($p = .005$). No other genotype pairings reached statistical significance.
- Recovery Rate: Recovery rate improvement (lower heart rate after 1 minute) was significantly better in TT compared to AA ($p = .020$) and in AT compared to AA ($p = .040$). Differences between AT and TT were not significant.

Overall Interpretation: There were significant benefits to all genotypes but most TT, in a comparative analysis table of results on weight or fat lost, and VO_{2max} or muscle strength and flexibility gained or recovery rate improved. Least benefited was the AA genotype, thus making quite a strong case for the earlier supposed theory that probably it is the A allele which attenuates responsiveness to exercise- and diet-based interventions; possibly through its metabolic and appetite-regulating effects.

DISCUSSION

Variation in response to weight loss between genotypes

The results of this study showed that participants with the TT genotype recorded greater decreases in both body weight and body fat percentage followed by AT genotype while AA genotype carriers displayed outcomes which were least favourable hence suggesting a variability on responsiveness to intervention for loss of weight by genotypes where A allele may probably be associated with reduced signalling for satiety as well as poor regulation on energy (Morton et al., 2006 , Ponce-González et al., 2023).

Anthropometric indicators between AT and AA carriers were significantly different in favour of the TT group, consistently with much other literature, for example, a large meta-analysis review that included 30 studies comprising 46,976 subjects. Better weight loss results among people with the TT genotype involved in structured exercise and diet interventions were reported (Rankinen et al., 2010).

Improvements in physical performance indicators

There were increases in VO_{2max} , strength, flexibility and recovery time for all genotypes but the most TT carriers showed the highest magnitude of changes. This is partly consistent with results from the DREW trial which found genotypic differences related to weight loss but no significant differences in cardiorespiratory fitness improvements across groups (Mitchell et al., 2010 , Tsigos et al., 2008).

Exercise as a modifier of gene environment effects

Evidence from large-scale studies indicates that moderate physical activity can attenuate the negative effect of the FTO risk allele on body weight. For example, a Taiwanese cohort study ($n = 20,906$) reported that regular exercise significantly reduced the weight gain effect associated with FTO polymorphisms ($p \approx .03$)

(Hiraïke et al., 2021). These results align with our findings, which show that structured training programs reduced genetic disadvantage across all genotype groups.

Role of an allele in resistance to interventions

Previous research suggests that the A allele is associated with greater fat mass and reduced responsiveness to exercise-based interventions (Farooqi, 2006, Walley et al., 2009). For instance, individuals with the AA genotype often fail to exhibit significant fat loss after resistance training. The present findings are consistent with this evidence, as the AA group in our cohort demonstrated smaller improvements across most outcome measures.

Metabolic adaptations during exercise

Our results also align with studies indicating that TT carriers have a higher fat oxidation capacity during exercise compared to AT and AA carriers, as well as differences in post-exercise appetite regulation ($p = .004-.049$) (Ponce-González et al., 2023). This greater metabolic flexibility may explain the superior body composition and performance outcomes seen in the TT group.

Physical activity gene interaction

The broader literature supports the notion that moderate physical activity can reduce the negative metabolic effects of the A allele (Kim et al., 2016, Yanagiya et al., 2007). Consistent with this, our data showed meaningful improvements in all genotype groups, although the magnitude of improvement was attenuated in A-allele carriers.

Summary interpretation

The results demonstrated and confirmed the previous assumption of a major role that FTO gene polymorphisms play in the responsiveness of obese athletes to weight loss and physical performance enhancement programs, particularly the A allele. TT carriers expressed more favourable outcomes, structured exercise interventions reported benefits for all genotypes. How personalized should post-detraining programs be? Answering this question could help us bridge genetic evidence to practical application: how personalized interventions could optimize exercise benefits? This fact brings to light an important message for policymakers or practitioners who are hesitant about prescribing exercises as part of a weight management program due to someone's "unfavourable" genetic profile.

Limitations

The sample size is relatively small, involving only one population where results might be generalizable and the intervention period was short to allow any long-term evaluation of adaptations based on genotypes. Other genetic factors except for the FTO polymorphism were not assessed; dietary intake was reported rather than controlled. Studies recommended to further validate and explore these findings should have larger cohorts with controlled nutrition over an extended follow-up period.

CONCLUSIONS

The findings of the study demonstrated a relationship between FTO gene polymorphisms and reduced response to weight loss programs as well as improvements in physical performance, mainly detailing the effects observed among participants with AA and AT genotypes. Those having TT genotype showed results much positively compared to their counterparts with AA genotype who registered results mildly progressive thus associating an allelic form probably responsible for inefficient fat metabolism (apparent lack control over appetite).

Results support the possibility of genetic screening as an added component in the planning process for training and weight management programs, hence partly personalizing training interventions to optimize results based on individual genotype profile. Well-planned aerobic and resistance exercise seems to overcome some metabolic handicaps found more frequently among A allele carriers.

Despite the differences related to genotype, all of them showed improvements that were measurable at the end of the training program. This again emphasizes exercise as an effective intervention towards improved body composition and physical performance across genetic backgrounds.

Future studies should be conducted on a larger and more varied sample to confirm the associations between genotypes and exercises discovered in this study. Long interventions assessing adaptations over a longer term should also be conducted. Hormonal and inflammatory markers ought to be included for better understanding of the biological pathways connecting FTO variants with metabolic as well as performance outcomes.

These guidelines may further develop scientifically exact prescription training through the adjustment of training techniques in attaining maximum weight loss, and also improvement of performance gains among various genotypes. Genetic screening is recommended for coaches and policymakers at large to make their interventions more effective, hence making a step toward results that are more individualized as well as successful both athletes and patients.

AUTHOR CONTRIBUTIONS

Saif Rasheed Ghanim: conceptualization of the study, study design, data collection, supervision of experimental procedures, statistical analysis, and drafting of the original manuscript. Ammar Faris Atiyah: participation in data collection, implementation of the training program, and assistance in data interpretation. Ahmed Mahmood Mahdi: laboratory work related to genetic analysis, data validation, and contribution to the methodology section. AlSeddiq Oday Latof: scientific consultation, critical revision of the manuscript, methodological guidance, and final approval of the manuscript for submission. All authors have read and approved the final version of the manuscript and agree with its submission and publication.

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DISCLOSURE STATEMENT

No potential conflict of interest was reported by the authors.

ETHICAL APPROVAL

This study was reviewed and approved by the Research Ethics Committee of the College of Physical Education and Sports Sciences, University of Samarra, Iraq (Approval Form No. 4; Decision Date: 16 September 2025). All research procedures were conducted in accordance with institutional ethical standards and the principles of the Declaration of Helsinki. Written informed consent was obtained from all participants prior to their inclusion in the study. Data collection was carried out between May 2024 and October 2024.

ARTIFICIAL INTELLIGENCE AND SIMILARITY CHECK STATEMENT

The authors confirm that no generative artificial intelligence tools were used in the creation, fabrication, or manipulation of the scientific content, data, or results of this manuscript. Automated tools were used solely for language checking and proofreading purposes. A similarity check was conducted using Turnitin, and the overall similarity index was below 20%, in compliance with the journal's ethical and editorial requirements.

REFERENCES

- Al Daghri, N. M., Alkharfy, K. M., Al Attas, O. S., Krishnaswamy, S., Mohammed, A. K., Albagha, O. M., Alenad, A. M., Chrousos, G. P., & Alokail, M. S. (2014). Association between type 2 diabetes mellitus related SNP variants and obesity traits in a Saudi population. *Molecular Biology Reports*, 41(3), 1731-1740. <https://doi.org/10.1007/s11033-014-3022-z>
- Andreasen, C. H., Stender-Petersen, K. L., Mogensen, M. S., Torekov, S. S., Wegner, L., Andersen, G., Nielsen, A. L., Albrechtsen, A., Borch-Johnsen, K., Rasmussen, S. S., Clausen, J. O., Sandbæk, A., Lauritzen, T., Hansen, L., Jørgensen, T., Pedersen, O., & Hansen, T. (2008). Low physical activity accentuates the effect of the FTO rs9939609 polymorphism on body fat accumulation. *Diabetes*, 57(1), 95-101. <https://doi.org/10.2337/db07-0910>
- Arendas, K., Qiu, Q., & Gruslin, A. (2008). Obesity in pregnancy: Pre-conceptual to postpartum consequences. *Journal of Obstetrics and Gynaecology Canada*, 30(6), 477-488. [https://doi.org/10.1016/S1701-2163\(16\)32863-8](https://doi.org/10.1016/S1701-2163(16)32863-8)
- Choi, H. K., Atkinson, K., Karlson, E. W., & Curhan, G. (2005). Obesity, weight change, hypertension, diuretic use, and risk of gout in men. *Archives of Internal Medicine*, 165(7), 742-748. <https://doi.org/10.1001/archinte.165.7.742>
- Claussnitzer, M., Dankel, S. N., Kim, K. H., Quon, G., Meuleman, W., Haugen, C., Glunk, V., Sousa, I. S., Beaudry, J. L., Puvion, V., Abdennur, N. A., Liu, J., Svensson, P. A., Hsu, Y. H., Drucker, D. J., Mellgren, G., Hui, C. C., Hauner, H., & Kellis, M. (2015). FTO obesity variant circuitry and adipocyte browning in humans. *The New England Journal of Medicine*, 373(10), 895-907. <https://doi.org/10.1056/NEJMoa1502214>
- Farooqi, I. S. (2006). Genetics of obesity in humans. *Endocrine Reviews*, 27(7), 710-718. <https://doi.org/10.1210/er.2006-0040>
- Flynn, M. A., McNeil, D. A., Maloff, B., Mutasingwa, D., Wu, M., Ford, C., & Tough, S. C. (2006). Reducing obesity and related chronic disease risk in children and youth: A synthesis of evidence. *Obesity Reviews*, 7(Suppl. 1), 7-66. <https://doi.org/10.1111/j.1467-789X.2006.00242.x>
- Hiraike, Y., Yang, C. T., Liu, W. J., Yamada, T., & Lee, C. L. (2021). FTO obesity variant-exercise interaction on changes in body weight and BMI: The Taiwan Biobank study. *Journal of Clinical Endocrinology & Metabolism*, 106(9), e3673-e3681. <https://doi.org/10.1210/clinem/dgab295>
- Howard, M., Natasha, J., Taylor, A., Gill, T., & Chittleborough, C. (2008). Severe obesity: Socio-demographics within extremes of BMI. *Obesity Research & Clinical Practice*, 2(1), 51-59. <https://doi.org/10.1016/j.orcp.2008.01.001>
- Kim, J. Y., DeMenna, J. T., Puppala, S., Chittoor, G., Schneider, J., Duggirala, R., Mandarino, L. J., Shaibi, G. Q., & Coletta, D. K. (2016). Physical activity and FTO genotype influence on obesity. *BMC Genetics*, 17, Article 89. <https://doi.org/10.1186/s12863-016-0357-6>
- Leońska-Duniec, A., Jastrzębski, Z., Zarębska, A., Maciejewska, A., Ficek, K., & Cięszczyk, P. (2018). Interaction between FTO polymorphism and physical activity on obesity traits. *Journal of Sport and Health Science*, 7(4), 459-464. <https://doi.org/10.1016/j.jshs.2016.08.013>

- Manson, J. E., Willett, W. C., Stampfer, M. J., Colditz, G. A., Hunter, D. J., Hankinson, S. E., Hennekens, C. H., & Speizer, F. E. (1995). Body weight and mortality among women. *The New England Journal of Medicine*, 333(11), 677-685. <https://doi.org/10.1056/NEJM199509143331101>
- Mitchell, J. A., Church, T. S., Rankinen, T., Earnest, C. P., Sui, X., & Blair, S. N. (2010). FTO genotype and weight loss benefits of moderate exercise. *Obesity*, 18(3), 641-643. <https://doi.org/10.1038/oby.2009.311>
- Morton, G. J., Cummings, D. E., Baskin, D. G., Barsh, G. S., & Schwartz, M. W. (2006). Central nervous system control of food intake and body weight. *Nature*, 443(7109), 289-295. <https://doi.org/10.1038/nature05026>
- Neovius, K., Johansson, K., Kark, M., & Neovius, M. (2009). Obesity status and sick leave: A systematic review. *Obesity Reviews*, 10(1), 17-27. <https://doi.org/10.1111/j.1467-789X.2008.00521.x>
- Organisation for Economic Co-operation and Development. (2012). Obesity update 2012. Retrieved from [Accessed 2026, 10 February]: <https://www.oecd.org/health/fitnotfat>
- Ponce-González, J. G., Martínez-Ávila, Á., Velázquez-Díaz, D., Perez-Bey, A., Gómez-Gallego, F., Marín-Galindo, A., Corral-Pérez, J., & Casals, C. (2023). Impact of the FTO gene variation on appetite and fat oxidation. *Nutrients*, 15(9), 2037. <https://doi.org/10.3390/nu15092037>
- Rahimi, M. R., & Symonds, M. E. (2025). Effect of FTO genotype on training- and diet-induced weight loss. *Critical Reviews in Food Science and Nutrition*, 65(21), 4080-4096. <https://doi.org/10.1080/10408398.2024.2382346>
- Rankinen, T., Rice, T., Teran-Garcia, M., Rao, D. C., & Bouchard, C. (2010). FTO genotype and exercise-induced body composition changes. *Obesity*, 18, 322-326. <https://doi.org/10.1038/oby.2009.205>
- Tsigos, C., Hainer, V., Basdevant, A., Finer, N., Fried, M., Mathus-Vliegen, E., Micic, D., Maislos, M., Roman, G., Schutz, Y., Toplak, H., Zahorska-Markiewicz, B., & Obesity Management Task Force of the European Association for the Study of Obesity. (2008). Management of obesity in adults: European clinical practice guidelines. *Obesity Facts*, 1(2), 106-116. <https://doi.org/10.1159/000126822>
- Van Baal, P. H., Polder, J. J., de Wit, G. A., Hoogenveen, R. T., Feenstra, T. L., Boshuizen, H. C., Engelfriet, P. M., & Brouwer, W. B. (2008). Lifetime medical costs of obesity: Prevention no cure for increasing health expenditure. *PLoS Medicine*, 5(2), e29. <https://doi.org/10.1371/journal.pmed.0050029>
- Walley, A. J., Asher, J. E., & Froguel, P. (2009). The genetic contribution to non-syndromic human obesity. *Nature Reviews Genetics*, 10(7), 431-442. <https://doi.org/10.1038/nrg2594>
- World Health Organization. (2000). Obesity: Preventing and managing the global epidemic (WHO Technical Report Series No. 894). World Health Organization. Retrieved from [Accessed 2026, 10 February]: <https://iris.who.int/handle/10665/42330>
- Yanagiya, T., Tanabe, A., Iida, A., Saito, S., Sekine, A., Takahashi, A., Tsunoda, T., Kamohara, S., Nakata, Y., Kotani, K., Komatsu, R., Itoh, N., Mineo, I., Wada, J., Masuzaki, H., Yoneda, M., Nakajima, A., Miyazaki, S., Tokunaga, K., & Hotta, K. (2007). Association of single-nucleotide polymorphisms in MTMR9 gene with obesity. *Human Molecular Genetics*, 16(24), 3017-3026. <https://doi.org/10.1093/hmg/ddm260>

