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A Novel Strategy for Diabetes in the Armed Forces: Policy Justification for Integrating *FTO* Gene Screening into Nutritional Protocols to Manage Metabolic Risks

Sobhan Mohammadi [1], Karim Parastouei [2*]

[1] Student Research Committee, Baqiyatallah University of Medical Sciences, Tehran, Iran
[2] Department of Nutrition and Food Hygiene, Faculty of Health, Baqiyatallah University of Medical Sciences, Tehran, Iran

***Corresponding Author:** Karim Parastouei, Ph.D., Department of Nutrition and Food Hygiene, Faculty of Health, Baqiyatallah University of Medical Sciences, Tehran, Iran. Tel: +989125906270, Email: parastouei@gmail.com

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Abstract

Type 2 diabetes is a growing global health issue, and military personnel are at increased risk due to unique stressors such as chronic occupational stress, disrupted sleep, and intense physical demands. These factors contribute to metabolic strain and raise the likelihood of developing the disease. Recent genetic studies highlight the role of the *FTO* gene, particularly the rs9939609 AA genotype, in increasing susceptibility to obesity and metabolic dysfunction. In military populations, individuals with this genotype show higher BMI, elevated fasting glucose, and increased LDL cholesterol, along with higher calorie and fat intake. These findings suggest the need for personalized health strategies that consider genetic predispositions. A one-size-fits-all approach is no longer sufficient. Instead, healthcare providers should tailor nutrition, exercise, and lifestyle recommendations based on individual genetic profiles to better manage metabolic risks. From a policy standpoint, military health systems should consider implementing genetic screening to identify high-risk personnel early. Targeted interventions, such as customized nutrition and fitness programs, should be prioritized for those with the AA genotype. Incorporating genomic data into long-term health monitoring can help track individual health trends and allow for early, proactive management of diabetes risk. Adopting these precision health strategies can reduce the prevalence of diabetes and related complications, improve force readiness, and lower long-term healthcare costs. Embracing genetic insights in military health policy can lead to more effective, individualized care and a stronger, healthier force.

Keywords: *FTO* gene, Type 2 Diabetes, Military Personnel, Nutrigenomics, Lipid Profile, Personalized Nutrition

1. Background

Type 2 diabetes (T2D) is recognized as one of the fastest-growing metabolic disorders worldwide, posing a major public health concern that continues to escalate at an alarming rate. Epidemiological projections indicate that the global prevalence of T2D will rise dramatically, increasing from approximately 382 million cases in 2013 to more than 593 million cases by the year 2035.[1] This sharp upward trend underscores the urgent need for preventive strategies and highlights the immense burden that diabetes places on healthcare systems, economies, and societies across both developed and developing nations.

The increasing prevalence of T2D is closely and almost inseparably linked to the rising rates of obesity observed globally. Obesity has been identified as the single most important modifiable risk factor for T2D, with estimates suggesting that nearly 90% of all cases can be attributed directly to excess body weight.[2] This strong correlation reflects the complex interplay between adiposity and metabolic dysfunction. The pathophysiological mechanisms underlying this association are primarily driven by insulin resistance, where the body's tissues fail to respond adequately to insulin, and insulin deficiency, which occurs when pancreatic β-cells are unable to produce sufficient insulin to meet metabolic demands.[3] Together, these processes create a vicious cycle that accelerates the onset and progression of diabetes in obese individuals.

Genetic studies have provided further insight into the biological underpinnings of obesity and diabetes. Genome-wide association studies (GWAS) have consistently demonstrated that the Fat Mass and Obesity-Associated (*FTO*) gene exhibits one of the strongest and most reproducible associations with both obesity and T2D.[4-5] The *FTO* gene is highly polymorphic, meaning it contains numerous genetic variations that can influence individual susceptibility to metabolic disorders. Case-control studies have revealed that several of these polymorphisms are significantly associated with obesity or elevated body mass index (BMI), reinforcing the gene's central role in metabolic regulation.[6]

Strong associations between *FTO* polymorphisms and

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BMI, obesity, and T2D have been reported across diverse populations, particularly in adults and children of European descent.[7] While much of the observed link between *FTO* variants and T2D has been attributed to their impact on BMI, several studies conducted in European cohorts have demonstrated that certain *FTO* polymorphisms may exert an independent effect on diabetes risk, beyond their influence on body weight.[6] This suggests that the gene may contribute to metabolic dysfunction through multiple pathways, including direct effects on glucose metabolism and lipid regulation.

However, findings from Asian populations have been less consistent. Some studies have failed to replicate the strong associations observed in European groups, while others have reported weaker or variable results. These discrepancies are likely attributable to differences in study design, relatively small sample sizes, and the considerable ethnic diversity present within Asian populations.[8,9] Such variability highlights the importance of conducting large-scale, ethnically diverse studies to fully elucidate the role of *FTO* polymorphisms in diabetes susceptibility across different genetic backgrounds.

2. Methods

This case–control study included 70 military personnel with T2D and 70 healthy individuals as controls. Dietary intake was assessed using a validated Food Frequency Questionnaire (FFQ). Genotyping of *FTO* (rs9939609) was performed using standard PCR methods. Anthropometric indices, fasting blood glucose, and serum lipid profiles (total cholesterol, LDL-C, HDL-C, triglycerides) were measured according to American Diabetes Association (ADA) guidelines.

Participants were screened for additional metabolic disorders, medications affecting lipid or glucose levels, and comorbidities that could confound genetic–metabolic associations.

3. Results

The genetic analysis revealed a critical finding in the management of T2D within the armed forces: the frequency of the high-risk genotype (AA) was significantly higher in the T2D patient group compared to the healthy group. This confirms the central role of this genetic marker in the onset and progression of the disease in this institutional population. More importantly, individuals carrying the AA genotype, regardless of their current diabetes status, systematically exhibited a more adverse metabolic risk profile. These individuals in both groups had higher Fasting Blood Glucose (FBG), higher Body Mass Index (BMI), and elevated levels of Total Cholesterol (TC) and LDL-C (bad cholesterol). This triple combination of risk (glycemic, anthropometric, and lipid) indicates a profound metabolic vulnerability rooted in the genetic structure. Behaviorally, the AA genotype was associated with higher caloric intake, particularly with a greater proportion of high-fat foods in the diet. These results are fully consistent with international genomic studies demonstrating that the *FTO* risk allele enhances metabolic vulnerability through abnormal appetite regulation, reduced satiety, and altered energy balance. This key insight underscores the necessity of moving away from general dietary recommendations toward personalized, gene-based nutritional interventions for the effective management of diabetes and the maintenance of operational readiness among armed forces personnel.

4. Discussion and Conclusion

The findings of this study show that the prevalence of the high-risk *FTO* allele (AA genotype) is significantly greater among individuals with T2D than in healthy controls, suggesting that the high-risk *FTO* genotype is a contributing risk factor for T2D. Fasting blood glucose levels were significantly higher in individuals with the AA genotype compared to those with the TT genotype. These findings support previous studies showing the role of *FTO* in glucose metabolism and diabetes risk.[10]

Animal and cellular studies have demonstrated that reduced *FTO* activity leads to lower body fat, improved glucose tolerance, and increased insulin sensitivity, whereas elevated *FTO* expression contributes to fat accumulation, weight gain, and metabolic dysfunction.[11,12] Additionally, hepatic *FTO* regulates gluconeogenesis by modulating the expression of genes such as *PCK1* and *G6PC*, responding to metabolic states including fasting, feeding, and insulin levels.[13]

Consistent with other human and animal studies, individuals with the AA genotype in this study had higher BMI, supporting the role of *FTO* in regulating energy intake, body composition, and central appetite pathways.[14] Regarding lipid metabolism, carriers of the AA genotype showed higher triglycerides, total cholesterol, and LDL-C levels, highlighting the involvement of *FTO* in lipid metabolic pathways through mechanisms such as altering m6A methylation of lipogenic gene transcripts and regulating *ATF4* expression.[15,16]

Variability in findings across populations may be due to differences in ethnicity, specific *FTO* polymorphisms studied, environmental factors, and sample characteristics.[17] Overall, evidence suggests that *FTO* plays a central role in regulating body weight, glucose metabolism, and lipid homeostasis, and its high-risk polymorphisms may predispose individuals to T2D and related metabolic disorders.

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Author Contributions

Authors contributed equally to this work.

Conflict of Interest Disclosures

All authors declared that they have no conflict of interest.

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