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Original Research Article

Association of Fat Mass and Obesity-associated (FTO) Gene Polymorphism with BMI dependent Obesity in Central Indian Cardiovascular disease Patients

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Abstract

Background: several study revealed that the FTO gene polymorphisms rs9939609 have been linked to obesity. It's essential to note that genetic variations exist between different populations. This study aims to assess the correlation between FTO gene polymorphisms rs9939609 and the BMI dependent obesity in CVD patients in central Indian population.

Methods: This cross sectional, observational study carried out from November-2024 to October-2025 in the department of Anatomy and the OPD of cardiology unit of department of Medicine in central Indian population at PCMS & RC, Bhopal (M.P.). Data were collected and managed using Microsoft Excel spread sheets. Gender and Obesity parameters were tested using Kruskal-Wallis and Pearson's chi^2 test. Significance between distribution of genotypes and alleles were tested by using Fisher exact test.

Results: Total 110 patients with cardiovascular disease were enrolled; of these 68.18% were males and 31.81% female patients. The age range of 55–73 years old accounted for the biggest percentage of patients. Among these 70.91% had a BMI of 25 kg/m² or above (obese) and only 29.09% had a BMI of less than 25 kg/m² (non-obese). There is three FTO rs9939609 genotypes AA, AT and TT were found, the most common genotype was AA (63.64%) and associated alleles were 'A' accounted for 76.82% of all alleles followed by T allele (23.18%). In this research neither AA nor AT genotypes and the allele-level comparison were linked to obesity as determined by BMI.

Conclusion: The results highlight that FTO rs9939609 ought to be regarded as a possible modifying genetic element instead than a standalone cause of obesity in CVD patients.

Keywords: FTO, Gene Polymorphism, BMI, Obesity, Cardiovascular Disease (CVD).

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Introduction

FTO gene is a nuclear protein belongs to member of AlkB (alpha-ketoglutarate-dependent dioxygenase) is a class of direct reversal DNA repair enzymes. [1,2] The FTO protein is greatly articulated in hypothalamus, as well as in many other tissues such as mesenteric fat, adipose tissue, pancreas, and liver. [3] It helps in regulating the metabolic rate, expenditure of energy, homeostasis of energy, size of the body and accumulation of body fat. [4]

The fat mass and obesity–associated (FTO) gene was reported as closely associated with body mass index (BMI) and increases the risk of obesity. It is located onchromosome16q12. FTO gene is strongly associated with body mass index, body composition, waist circumference, and metabolism. [5,6] It is well established that there is an association between FTO and obesity risk, however obesity risk related to FTO may be modulated by lifestyle. [7] Body fatness mediated by FTO may be attenuated in physically active individuals has been suggested by numerous studies.

Those individuals who carry the risk allele of FTO gene have been reported to have more weight, BMI, and Waist Circumference (WC), respectively. [8,9,10] The common variation in the Fat Mass and

Obesity Associated (FTO) gene is strongly associated with higher body mass index (BMI) and obesity in several populations across the world.11 Obesity is a global issue that affects 2 billion people worldwide and poses a threat to public health. By 2030, projections suggest that approximately 20 % of women and 14 % of men will be affected by obesity, which is equivalent to over 1 billion individual's worldwide.1 In 2019, over 160 million years of healthy life were lost worldwide as a result of high body mass index (BMI). [12,13] Obesity is one of the most prevalent civilization diseases, defined by a body mass index (BMI) (weight in kg/height in m2) ≥ 30kgm2. [14] Risk of obesity can be also estimated on the basis of body fat percentage — BF% (> 25% for men, > 35% for women) and waist circumference (> 104 for men, > 88 cm for women.15 By this study we can more clarify about the co-relation of FTO Gene polymorphism to obesity on basis of BMI. Hence, by this study we are trying to evaluate the "Correlation of FTO Gene Polymorphism with BMI and obesity in CVD patients.

Material and Method

This cross sectional, observational study carried out from November-2024 to October-2025 in the department of Anatomy and the OPD of cardiology unit of department of Medicine in central Indian population at PCMS & RC, Bhopal (M.P.). Total 110 known CVD patients were enrolled who attended the OPD of Cardiology unit of department of Medicine during the study duration. After written informed consent the blood samples of these patients were collected, and anthropometric measurements were taken to calculate BMI and body weight.

All known CVD patients of age 18-80 years who agree to participate with information that the study results will only be used for the purpose of research and the reports of the study shall not be provided to them, however their identity shall not be revealed at any level were included in study were enrolled. The patients who was not willing to give blood sample, having psychiatric illness, pregnant and breast feeding women, patients with history of previous surgery on one or more heart valves, patients with serious illness such as cancer, end-stage renal disease (ESRD) and severe heart failure (New York Heart Association Class IV) were excluded from the study.

Genetic assessments was done by collecting 3ml of the venous blood sample from the antecubital vein from each patient in EDTA tubes, by using disposable syringe with needle with all aseptic precautions. These EDTA tube were preserved at -20°C in deep freezer. DNA extraction procedure is a complex process which includes several steps including RBC lysis, WBC cell lysis, protein precipitation (salting out), DNA precipitation, DNA pelleting, washing, DNA resuspension, Agarose gel electrophoresis, reconstitution and dilution of primers and PCR amplification**.**

Anthropometric measurements were taken. Body weight (kg) was measured (by using portable weighing machine), Height (cm) was measured by using flexible steel tape) using standardized procedures**,** Waist circumference (WC) was measured midway between the lowest rib and the superior border of the iliac crest in standing position, using an inelastic measuring tape at the end of normal expiration. BMI was calculated as weight (kg) divided by the square of the height (m2). BMI and body fat % was calculated by using the calculator. The risk of obesity can be estimated on the basis of body fat percentage (BF % >24% for men, >33% for women), [16] waist circumference (>90 cm for men, >80 cm for women) [17] and neck circumference **(**≥34 cm for male and≥30.5 cm female) [18].

Statistical Analyses: Data were collected and managed using Microsoft Excel spread sheets. All qualitative variables were expressed as numbers and percentages, while quantitative data were presented as median**.**

Statistical analysis was performed using appropriate statistical tests. Gender and Obesity parameters were tested using Kruskal-Wallis and Pearson's chi^2 test. Significance between distribution of genotypes and alleles were tested by using Fisher exact test. The strength of association between FTO polymorphism and BMI dependent obesity was estimated by calculating pooled odd ratio with 95%CI. A p-value of ≤ 0.05 was considered statistically significant.

Observations and Results

Table 1: Age and Gender wise distribution of CVD patients.

Age Group (in years)	Males(n=75)		Females (n=35)		Grand Total (n=110)	
	No	%	No	%	No	%
18-35	12	16.0	6	17.14	18	16.36
36-54	19	25.33	6	17.14	25	22.73
55-73	34	45.33	21	60.0	55	50.0
>= 74	10	13.33	2	5.71	12	10.91
Total	75	100.0	35	100.0	110	100.0

Table 2: BMI classification for Asian Indians wise distribution of CVD Patients

BMI	Males(n=75)		Females (n=35)		Grand Total (n=110)	
	No	%	No	%	No	%
Non-obese (BMI <25)	26	34.67	6	17.14	32	29.09
Obese (BMI >=25)	49	65.33	29	82.86	78	70.91
Total	75	100.0	35	100.0	110	100.0

Table 3: FTO rs9939609 genotypes and Allele frequencies wise distribution of CVD patients

Section	Genotype / Allele	Frequency of Genotypes and Alleles distribution n=110	
		Number	Percentage (%)
Genotype	AA	70	63.64
	AT	29	26.36
	TT	11	10.0
Allele	A	169	76.82
	T	51	23.18

Table 4: Comparison of Anthropometric parameters across different genotypes of FTO rs9939609 in CVD Patients

Section	variable	AA	AT	TT	summary	test	static	P-value
	Age in years	58.50 (40.25-65.00)	59.00 (52.00-69.00)	59.00 (51.00-67.00)	Median (IQR)	Kruskal-Wallis	1.122	0.571
Gender	Male	49 (70.00%)	19 (65.52%)	7 (63.64%)	n (%)	Chi-square	0.306	0.858
	Female	21 (30.00%)	10 (34.48%)	4 (36.36%)	n (%)	Chi-square	0.306	0.858
Obesity parameter	Weight	75.50 (60.50-84.00)	80.00 (68.00-85.00)	75.00 (75.00-81.50)	Median (IQR)	Kruskal-Wallis	1.152	0.562
	Height	165.00 (155.25-170.00)	168.00 (155.00-170.00)	168.00 (160.00-168.00)	Median (IQR)	Kruskal-Wallis	1.193	0.551
	BMI	28.00 (23.73-30.88)	28.30 (24.20-30.40)	27.10 (26.25-31.20)	Median (IQR)	Kruskal-Wallis	0.569	0.752
	NC	38.00 (34.00-40.00)	38.00 (34.00-42.00)	40.00 (38.00-43.50)	Median (IQR)	Kruskal-Wallis	2.631	0.268
	WC	95.00 (85.75-100.00)	98.00 (89.00-105.00)	94.00 (91.00-105.00)	Median (IQR)	Kruskal-Wallis	0.675	0.714
	BF %	29.75 (26.45-34.22)	32.10 (26.60-36.70)	34.90 (24.55-39.85)	Median (IQR)	Kruskal-Wallis	1.368	0.505

Table 5: Genotype and allele frequency of FTO rs9939609 gene polymorphism in obese and non- obese Patients

Section	Genotype / allele group	Obese frequency (n=78)		Non-obese frequency (n=32)		OR (95% CI)	Test used	P-value
		No.	%	No.	%			
Genotype	AA	48	61.53	22	68.75	1.00 (Ref.)	Reference	
	AT	19	24.35	10	31.25	0.87 (0.35-2.18)	Fisher exact	0.815
	TT	11	14.10	0	0.0	10.67 (0.60-189.18)	Fisher exact	0.030
Allele	A	115	73.71	54	84.37	1.00 (Ref.)	Reference	
	T	41	26.28	10	15.62	1.93 (0.90-4.13)	Fisher exact	0.113

Results

Total 110 patients with cardiovascular disease were enrolled; of these 68.18% were males and 31.81% female patients. The age range of 55–73 years old accounted for the biggest percentage of patients. This age group had 55 patients in total, making up precisely 50% of the overall study population. In which 45.33% were males and 60% were females.

The second most prevalent age group was 36–54 years with 22.73% patients (25.33% males and 17.14% females). (Table1). Among 110 CVD patients, 70.91% had a BMI of 25 kg/m² or above (obese) and only 29.09% had a BMI of less than 25 kg/m² (non-obese). Of these obese patients, 65.33% were males and 82.86% were females. This suggests that over two-thirds of the study's male CVD patients fell into the obese BMI group. (Table2). The distribution of FTO rs9939609 genotypes and associated allele frequencies across the 110 patients with cardiovascular diseases, three genotypes—AA, AT, and TT—were found. The most common genotype found was AA (63.64%), it was present in almost two-thirds of all patients followed by heterozygous AT (26.36%) genotype. Only 10% patients had the TT genotype, which was the least common. A total of 220 alleles were assessed since each of the 110 individuals had two alleles, the A allele accounted for 76.82% of all alleles followed by T allele (23.18%). (Table3).

Patients with the AA genotype, median age was 58.5 years and age of patients with the AT genotype was 59 years. Similarly, the median age of patients with the TT genotype was 59 years. There was no statistically significant difference in age between the three genotype groups, (p=0.571). The distributions of genders were similar as well. Of the patients with the AA genotype, 30% were females and 70% males. In the AT group 34.48% were females and 65.52% were males and in the TT group 63.64% and 36.36% were females and male respectively. Therefore, there was no discernible difference in the gender distribution across the three FTO genotype groups (p=0.858). The median weight of AA group patients was 75.5 kg, AT was 80 kg, and the TT was 75 kg. The median body weight of patients with the AT genotype was numerically larger, but this difference was not statistically significant (p=0.562).

AA carriers had a median height of 165.0 cm, whereas both AT and TT genotype carriers had a median height of 168 cm. there was no statistically significant difference (p=0.551). The median BMI was 28 kg/m² in AA genotype group, followed by 28.3 kg/m² in AT and 27.1 kg/m² in the TT group patients. Despite of these little numerical variations, the FTO rs9939609 genotype did not significantly affect BMI. (p=0.752). The AA and AT groups' median neck circumferences were 38.0, whereas the TT group's was 40.0. The median neck circumference was somewhat greater in TT carriers, but the difference was not statistically significant (p=0.268). Patients with AA, AT and TT had median waist circumferences of 95 cm, 98 cm and 94 cm respectively, the waist circumferences in different genotype groups did not statistically significantly (p=0.714). Patients with the AA genotype had a median body fat percentage of 29.75%, AT had 32.1%, and TT genotype had 34.9%. Although, the difference did not reach statistical significance, (p=0.505). (Table4). Among total patients, 78% were obese 32% non-obese. In the AA genotype 61.53% patients were obese and 68.75% non-obese. The AA genotype was given an odds ratio of 1.00 as it was the most suitable reference group. 31.25% non-obese and 24.35% obese patients had the AT genotype. The odds ratio for AT genotype in relation to the AA genotype was 0.87, with a 95% confidence interval, this was statistically not significant correlation between the AT genotype and BMI. (p=0.815). 14% obese patients had TT genotype with no patients in non-obese group, this shown clinically significant association of TT genotype and obesity. (p=0.030). However, as there were no TT individuals in the non-obese group, this result needs to be interpreted cautiously. In allele's correlation, allele A had 73.71% obese and 84.37% non-obese patients. Similarly allele T had 26.28% obese and 15.62% non-obese patients respectively. The T allele showed an odds ratio of 1.93 with a 95% confidence range of 0.90–4.13 when the A allele was used as the reference. There was no statistical significance difference in T allele frequency between obese and non-obese (p=0.113). (Table5).

Discussion

In present study a significant prevalence of obesity was noted 76.36% of patients met the research's body-fat percentage criteria for obesity, and 70.91% of patients were placed in the BMI defined obese category using the cut-off used in the study.

The primary investigations concerning central obesity in this research contrasted genotype and allele frequencies among 70.91% obese individuals defined by BMI and 29.09% non-obese patients with cardiovascular disease. In order to investigate the relationship between the FTO rs9939609 polymorphism and body mass index and other measures of adiposity among cardiovascular disease patients receiving tertiary care in Central India. At the genetic level, the most common genotype of FTO rs9939609 was AA (63.64%), followed by AT (26.36%) and TT (10%); the A allele made up 76.82% of all alleles. The distribution of the TT genotype varied across BMI-defined obese and non-obese groups, with all TT carriers detected in the obese group and none in the non-obese group. This was the most significant finding related obesity. On the other hand, neither AT nor the allele-level comparison were linked to obesity as determined by BMI. Additionally, neither AT nor TT demonstrated a statistically significant correlation with adiposity, categorized by using waist circumference, neck circumference, or body-fat percentage. There were no significant differences in continuous anthropometric traits between the AA, AT, and TT groups. Overall,

rather having a single consistent impact across all evaluated outcomes, the data show a complicated and phenotype-dependent connection between FTO rs9939609, and obesity.

Association of FTO rs9939609 with BMI-Defined Obesity and Other Measures of Adiposity: AA was seen in 61.53% obese and 68.75% non-obese patients, while AT was noted in 24.35% obese and 31.25% non-obese patients, respectively. The AT genotype showed no correlation with BMI-defined obesity as compared to AA (OR 0.87, 95% CI 0.35-2.18; p=0.815). Conversely, all 10% TT carriers belonged to the obese category, while none were classified as non-obese. The documented Fisher exact p-value was 0.030. The odds-ratio estimation for TT was 10.67; however, the confidence range was very broad (0.60-189.18), indicative of the limited size of the TT group and the absence of data among non-obese individuals. At the allele level, the T allele exhibited an odds ratio of 1.93 compared to A, although the connection lacked statistical significance (p=0.113).

In several studies, the 'A' allele is considered the allele linked to obesity risk, with AA homozygosity being correlated with elevated BMI or increased obesity risk. Nindrea and Thongwichian et al. [19] documented substantial correlations of rs9939609 with obesity across several genetic frameworks in both Western and Asian cohorts. Nilupher et al. [20] identified elevated adiposity indicators in risk-genotype carriers among adults in North Delhi, whereas Parthasarthy et al. [21] noted an increased prevalence of AA in obese Indian children. Shabana et al. [22] also reported a progressive elevation in obesity risk correlated with the rising quantity of FTO risk alleles in a Pakistani cohort. Consequently, the trend in the current dataset, where TT was only identified in the BMI >=25 cohort, does not reflect the often documented association with rs9939609 risk.

Population diversity offers a plausible explanation for this noticeable inconsistency. Allele frequencies and linkage configurations differ across ethnicities, and Yu et al. [23] highlighted that the association between FTO and obesity traits is variable between cultures. Putri et al. [24] noted a genotype prevalence characterized by TA and TT in young Sundanese women with elevated body fat, but Hussain et al. [25] identified TT as the predominant genotype in their T2DM/CVD cohort [150]. Such results indicate that the incidence of genotypes and their correlation with phenotypes may vary across Asian populations. Nevertheless, demographic variability should not be used to excessively rationalize a statistically unreliable finding. The limited quantity of TT carriers and the total lack of TT in the non-obese cohort significantly heighten ambiguity. Additional understanding is derived from the examination of waist circumference. Individuals with elevated waist circumference included 71.4% AA, 79.31% AT, and 90.9% TT carriers, whereas those with normal waist circumference consisted of 28.57% AA, 20.68% AT, and 1.1% TT. The odds ratios for AT (1.53) and TT (4.00) exceeded 1, although neither comparison attained statistical significance (p=0.464 and p=0.273). The examination of body fat % revealed an alternative trend. The high body fat percentage group included 75.71% AA, 79.31% AT, and 72.72% TT patients, whereas the normal body fat percentage group consisted of 24.28% AA, 20.68% AT, and 27.27% TT patients. Neither AT nor TT exhibited a significant correlation with raised BF%, and the TT estimate was not even directionally heightened (OR 0.86; p=1.000). This disparity among BMI, waist circumference, and body-fat percentage suggests a lack of a single, strong genetic influence on adiposity in the current population. It rather implies that the perceived correlation is contingent upon the criteria used to define obesity. Nilupher et al. [20] indicated that the predictive association of rs9939609 varied across different adiposity indicators. Kolackov et al. [26] discovered that FTO haplotypes did not significantly affect body-fat distribution in non-obese people, even with the existence of obesity-related polymorphisms. Poosri et al. [27] also found no notable anthropometric correlation, even though there was evidence that FTO risk alleles affected eating habits. These investigations indicate that FTO could function at an upstream level via hunger, dietary choices, or metabolic control; however the ultimate anthropometric manifestation is influenced by environmental and behavioral factors. The analysis conducted by Loos and Yeo28 highlights that obesity associated with FTO is significantly affected by appetite and gene-environment interactions, rather than being governed by a deterministic genetic framework.

Conclusion

The current findings on obesity provide limited but perhaps significant evidence of genetic variation. The statistically significant comparison of TT in BMI-defined obesity is remarkable since it was not replicated with waist circumference, neck circumference, body-fat percentage, or continuous anthropometric metrics. Consequently, the whole of information from this research does not substantiate a sweeping assertion that a single gene uniformly predicts obesity across all metrics. The results are better understood as indicating that rs9939609 might be linked to certain obesity phenotypes within this Central Indian CVD group, with the BMI related TT signal necessitating validation in a larger and ideally independently gathered sample.

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