

Evaluation of FTO Gene Polymorphism and Leptin Receptor Gene Polymorphism in Patients with Type 2 Diabetes Mellitus

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ABSTRACT

Background: Type 2 diabetes mellitus (T2DM) is a major global health burden strongly linked to obesity and genetic factors. Polymorphisms in the fat mass and obesity-associated (FTO) gene and leptin receptor (LEPR) gene have been implicated in adiposity, insulin resistance, and T2DM susceptibility, yet data from the Egyptian population remain limited.

Aim: To evaluate (FTO) (rs17817449) gene and leptin receptor (LEPR) (rs1137101) gene polymorphism in type 2 diabetes mellitus patients with and without obesity.

Patients and methods: This study was a case control study, carried out at clinical chemistry laboratories in specialized Internal Medicine Hospital (routine laboratory tests) and immunology unit Clinical pathology department (molecular study), Mansoura, Egypt, from April 2023 to April 2025, conducted on: 100 patients diagnosed with Type 2 diabetes mellitus obese and non-obese. 100 healthy subjects as a control group.

Results: LEPR AG and GG genotypes significantly increased T2DM risk (OR = 1.84 and 3.75, respectively); the G allele conferred higher risk (OR = 2.10). FTO TG and GG genotypes also elevated risk (OR = 1.67 and 3.10); the G allele was more frequent in patients (OR = 1.91). Both polymorphisms were associated with obesity and dyslipidemia. Multivariate analysis confirmed LEPR (and FTO GG) as independent predictors.

Conclusion: FTO (rs17817449) and LEPR (rs1137101) polymorphisms served as genetic risk predictors for T2DM in Egyptians. AG/GG (LEPR) and TG/GG (FTO) genotypes, along with G alleles, significantly increased T2DM risk (ORs 1.67–3.75), were associated with obesity and dyslipidemia, and may have contributed independently of obesity.

Keywords: Type 2 diabetes mellitus, FTO gene, LEPR gene, polymorphism, obesity

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INTRODUCTION

Diabetes mellitus affects 529 million people worldwide. With a worsening diet and rising BMI in the population, it is projected to affect 1.31 billion people by 2050.(1) .

The global prevalence of T2DM showed increase over the last decades and it is supposed to reach 12.2 % in 2045 (2). The 2025 International Diabetes Federation (IDF) guidelines recognise global disparities in healthcare access, with 80% of people with type 2 diabetes mellitus (T2DM) living in low to middle income countries (LMICs) (3).

Adipose tissue plays a central role in systemic metabolic regulation and represents one of the most dynamic endocrine organs involved in the pathophysiology of T2DM (4).

Beyond its function as an energy storage depot, adipose tissue regulates glucose and lipid metabolism through the secretion of adipokines, inflammatory mediators, and metabolic signaling molecules (5).

Consequently, alterations in adipocyte differentiation and adipose tissue expansion significantly contribute to the

development of insulin resistance and metabolic dysfunction (Hade et al., 2025).

Leptin is an important member of adipokines. It is often referred to as the “satiety hormone” because it helps signal the brain to reduce appetite and increase energy expenditure when fat stores are sufficient. The gene encoding leptin is called LEP and is located on chromosome 7 in humans (6). Leptin exerts its physiological function by combining with the leptin receptor (LEPR), which is primarily located in the hypothalamus of the brain but is also found in other tissues throughout the body (7).

Moreover, there is a gene called Fat mass and obesity-associated (FTO) that has been previously associated with both childhood and adult obesity (8).

FTO is ubiquitously expressed in human tissues, with its highest levels found in the hypothalamic nuclei, which regulate energy balance. Variants within the FTO gene have been linked not only to increased adiposity in humans, but also to the development of metabolic syndrome and cardiovascular disease (9).

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The link between polymorphisms at fat mass and obesity-correlated protein (FTO), leptin (LEP), and leptin receptor (LEPR) genes and DM still needs further illustration (10). Our study aimed to evaluate (FTO) (rs17817449) gene and leptin receptor (LEPR) (rs1137101) gene polymorphism in type 2 diabetes mellitus patients with and without obesity.

SUBJECTS AND METHODS

This study was a case control study, carried out at clinical chemistry laboratories in specialized Internal Medicine Hospital (routine laboratory tests) and immunology unit Clinical pathology department (molecular study), Mansoura, Egypt, from April 2023 to April 2025. This study was conducted on: 100 patients diagnosed with Type 2 diabetes mellitus obese and non-obese. 100 healthy subjects as a control group.

Inclusion criteria:

Patients with Type 2 Diabetes diagnosed by diabetologists according to definitions of the International Diabetes Federation (IDF) (11).

Exclusion criteria:

All the cases with probable diagnosis of type 1 diabetes or other rare forms of diabetes as MODY (maturity onset diabetes of young), severe respiratory disease, acute and chronic Liver diseases, acute and chronic Kidney diseases, and inflammatory diseases (infections, autoimmune disorders and malignancy).

METHODS

All patients in this study were subjected to: **History taking.** **Physical examination:** Weight and height were measured without shoes using digital scales (to 0.1 kg) and a stadiometer (to 0.1 cm) (12). BMI was calculated as weight (kg)/height (m²) and classified per WHO criteria (Ashraf et al., (13), (Organization, (14)) <18.5 underweight; 18.5–24.99 normal; 25–29.99 preobese; 30–34.99 obese I; 35–39.99 obese II; ≥40 morbidly obese. **Specimen collection:** After overnight fasting, 7 ml venous blood was collected: 3 ml for serum biochemistry (Cobas c 311, Roche), 2 ml EDTA for HbA1c, and 2 ml EDTA for DNA PCR-RFLP of FTO and LEPR polymorphisms. **Routine Laboratory investigations:** Fasting glucose (hexokinase), HbA1c (turbidimetric immunoassay), lipids (enzymatic TC/TG/HDL-C/LDL-C), albumin (BCG), bilirubin (dialysis), creatinine (Jaffé), and AST/ALT (IFCC) were measured on the cobas c311 analyzer. **Specific laboratory investigations:** FTO (rs17817449) and leptin receptor (rs1137101) gene polymorphisms were determined by DNA extraction, polymerase chain reaction (PCR), restriction

fragment length polymorphism (RFLP), and electrophoresis. **DNA extraction:** DNA was isolated from EDTA-blood samples using the GeneJET Whole Blood Genomic DNA Purification Mini Kit (QIAGEN, cat. no. 51104, Germany). Samples were digested with proteinase K, bound to a silica membrane, washed to remove impurities, and eluted under low-ionic-strength conditions according to the manufacturer's instructions. Purified DNA was stored at –20 °C for subsequent PCR-RFLP analysis. **Assessment of DNA quality:** DNA quality and concentration were assessed using a NanoDrop spectrophotometer. It is suitable for molecular biology techniques when its ratio at OD 260/280 ranges between 1.8 and 2.0. **Polymerase chain reaction (PCR):** FTO (rs17817449) polymorphism was amplified according to Scott et al. (2010) using primers F: 5'-CGGTGAAGAGGAGGAGATTG-3' and R: 5'-CATCTCTGCCCCAGTTTCTC-3'. Leptin receptor (rs1137101) was amplified per Ashraf et al. (13), with primers F: 5' ACCTCTGGTTCCCCAAAAAG-3' and R: 5'-TCATCATTTTGTAGTCATAACTTACCC-3'. PCR used DreamTaq Green Master Mix (2X, cat. no. K1081) with 12.5 µL mix, 11.3 µL water, 0.1 µL each primer, and 1 µL DNA on a Bio-Rad PTC-100 thermal cycler. **Restriction fragment length polymorphism (RFLP) using restriction enzymes:** PCR products were digested with AlwNI (FTO rs17817449) or MspI (LEPR rs1137101) (Thermo Fisher Scientific) using 10 µL PCR product, 1 µL enzyme, 2 µL buffer, and 17 µL nuclease-free water, incubated at 37 °C. **Electrophoresis:** Restriction products were visualized on 2% agarose gel with a 50 bp ladder under UV. FTO (AlwNI) genotypes: TT (100+123 bp), GG (223 bp), GT (100+123+223 bp). LEPR (MspI) genotypes: AA (212 bp), GG (151+61 bp), AG (212+151+61 bp).

Ethical consideration:

Study informed consent was obtained from patients before their inclusion in the study. The study protocol was submitted for approval from Institutional Review Board of Faculty of Medicine – Mansoura University prior to initiation of the study. All data will be kept confidential.

Statistical analysis of the data:

Data were analyzed using IBM SPSS Statistics Version 25.0 and Peacock (15). Normality was tested by Kolmogorov Smirnov. Descriptive and analytical statistics (Chi-square, t-test, Mann Whitney, ANOVA, Kruskal Wallis, logistic regression) were applied, with p < 0.05 considered significant.

RESULTS

Table 1.: LEPR (rs1137101) gene polymorphism among T2DM patients and the control group.

LEPR (rs1137101)		T2DM patients n = 100		Control® n = 100		p value	OR (95 % CI)
		N ₀	%	N ₀	%		
Genotypes	AA	24	24.0	55	55.0		Reference
	AG	42	42.0	36	36.0	0.003*	1.84(1.23–2.75)
	GG	34	34.0	9	9.0	<0.001*	3.75(2.25–6.27)
Dominant model	AA	24	24.0	55	55.0		Reference
	AG+GG	76	76.0	45	45.0	<0.001*	2.32(1.60–3.35)
Recessive model	AA+AG	66	66.0	91	91.0		Reference
	GG	34	34.0	9	9.0	<0.001*	2.74(1.72–4.38)
Alleles	A	90	45.0	146	73.0		Reference
	G	110	55.0	54	27.0	<0.001*	2.10(1.63–2.72)

®: Reference group; A, adenine; G, Guanine; reference genotype and allele based on NCBI database; N₀, number; OR, odds ratio; CI, confidence interval; OR<1, protective; OR>1, risky; *: Significant when p value <0.05.

Table 1 showed a statistically significant link was discovered between the LEPR (rs1137101) polymorphism and T2DM. Patients with the AG and GG genotypes had significantly higher odds of developing T2DM than controls (OR = 1.84 and 3.75, respectively). Both dominant

and recessive models revealed significant association with T2DM ($p < 0.001$), with the G allele being significantly more frequent among patients (OR = 2.10).

Table 2:. FTO (rs17817449) gene polymorphism among T2DM patients and the control group.

FTO (rs17817449)		T2DM patients n = 100		Control® n = 100		p value	OR (95 % CI)
		N _o	%	N _o	%		
Genotypes	TT	35	35.0	62	62.0		Reference
	TG	40	40.0	31	31.0	0.009*	1.67(1.14–2.47)
	GG	25	25.0	7	7.0	<0.001*	3.10(1.79–5.37)
Dominant model	TT	35	35.0	62	62.0		Reference
	TG+GG	65	65.0	38	38.0	<0.001*	2.0(1.40–2.85)
Recessive model	TT+TG	75	75.0	93	93.0		Reference
	GG	25	25.0	7	7.0	0.001*	2.49(1.48–4.19)
Alleles	T	110	55.0	155	77.5		Reference
	G	90	45.0	45	22.5	<0.001*	1.91(1.46–2.49)

®: Reference group; T, thymine; G, Guanine; reference genotype and allele based on NCBI database; N_o, number; OR, odds ratio; CI, confidence interval; OR<1, protective; OR>1, risky; *: Significant when p value <0.05.

Table 2 showed a significant link was noticed between the FTO (rs17817449) polymorphism and T2DM. Both the TG and GG genotypes were significantly related with increased T2DM risk as compared to the TT genotype (OR = 1.67,

3.10 respectively). The dominant and recessive models were also significant. Furthermore, the G allele was more frequent among T2DM patients (OR = 1.91), indicating its role as a risk allele for T2DM.

Table 3. Association between LEPR (rs1137101) gene polymorphism and obesity among T2DM patient

	LEPR (rs1137101) gene polymorphism						Test	p1	Pairwise		
	AA n = 24		AG n = 42		GG n = 34				p2	p3	p4
	N _o	%	N _o	%	N _o	%					
Obesity											
Non-obese	19	79.2	23	54.8	8	23.5	$\chi^2=$ 18.077	<0.001*	0.047*	<0.001*	0.006*
Obese	5	20.8	19	45.2	26	76.5					
BMI (kg/cm ²)											
Mean ± SD.	27.26 ± 3.70		29.79 ± 5.48		31.61 ± 5.75		H= 9.842	0.007*	0.064	0.002*	0.116
Median	26.73		29.32		32.88						
Min. – Max.	21.41 – 34.88		20.81 – 42.19		21.19 – 46.52						

N_o, number; SD, Standard deviation, Min.: Minimum, Max.: Maximum, χ^2 : Chi-Square test, H: Kruskal Wallis test, p1: Comparing different categories, p2: Comparing AA and AG, p3: Comparing AA and GG, p4: Comparing AG and GG, *: Significant when p value <0.05.

Table 3 showed a significant connection was detected between the LEPR (rs1137101) polymorphism and obesity among T2DM patients ($p < 0.001$), with the GG genotype

being much more frequent in obese subjects. GG carriers had significantly higher BMI levels than AA persons ($p = 0.002$).

Table 4. Association between FTO (rs17817449) gene polymorphism and obesity among T2DM patients.

	FTO (rs17817449) gene polymorphism						Test	p1	Pairwise		
	TT n = 35		TG n = 40		GG n = 25				p2	p3	p4
	N _o	%	N _o	%	N _o	%					
Obesity											
Non-obese	24	68.6	19	47.5	7	28.0	$\chi^2=$ 9.769	0.008*	0.066	0.002*	0.118
Obese	11	31.4	21	52.5	18	72.0					
BMI (kg/cm ²)											
Mean ± SD.	28.01 ± 4.85		30.34 ± 5.40		31.46 ± 5.69		H= 7.151	0.028*	0.049*	0.012*	0.423
Median	27.04		30.40		31.56						
Min. – Max.	20.81 – 40.76		21.08 – 42.19		21.36 – 46.52						

N_o, number; SD, Standard deviation, Min.: Minimum, Max.: Maximum, χ^2 : Chi-Square test, H: Kruskal Wallis test, p1: Comparing different categories, p2: Comparing TT and TG, p3: Comparing TT and GG, p4: Comparing TG and GG, *: Significant when p value <0.05.

Table 4 showed a significant association was noticed between the FTO (rs17817449) polymorphism and obesity among T2DM patients ($p = 0.008$), with the GG genotype being more common among obese subjects. BMI levels

were also significantly higher in GG and TG carriers than in TT subjects, especially in the TT and GG genotypes ($p = 0.012$).

Table 5: Logistic regression analysis for prediction of T2DM susceptibility.

		Univariate		Multivariate	
		p	OR (95% CI)	p	OR (95% CI)
Sex		0.384	1.171(0.820–1.673)	-	-
Age		0.276	0.989(0.971–1.009)	-	-
Obesity		0.156	1.500(0.857-2.626)	-	-
LEPR (rs1137101)	AA	-	Reference	-	Reference
	AG	0.003*	1.84(1.23–2.75)	0.022*	1.748(1.084-2.819)
	GG	<0.001*	3.75(2.25–6.27)	0.004*	3.129(1.455-6.729)
FTO (rs17817449)	TT	-	Reference	-	Reference
	TG	0.009*	1.67(1.14–2.47)	0.166	1.077(0.661-1.756)
	GG	<0.001*	3.10(1.79–5.37)	0.020*	1.305(1.180-2.936)

OR: Odd Ratio; CI, confidence interval. *: Significant when p value <0.05.

Table 5 showed that in the entire cohort, univariate analysis revealed that both the LEPR (rs1137101) and FTO (rs17817449) polymorphisms were significantly related with higher susceptibility to T2DM. After multivariate analysis, the link with LEPR (AG and GG genotypes) remained statistically significant, indicating its role as an independent predictor of T2DM risk. Notably, FTO remained significant for the GG genotype in the adjusted model, indicating partial attenuation of the effect after correcting for confounders. Meanwhile, sex, age, and obesity did not have significant independent relationships. Overall, these findings indicate that both LEPR and FTO polymorphisms influence T2DM susceptibility, with LEPR having a greater and more persistent independent effect.

DISCUSSION

In the current study, there is a statistically significant link reported between the LEPR (rs1137101) polymorphism and T2DM. Patients with the AG and GG genotypes had significantly higher odds of developing T2DM than controls (OR = 1.84 and 3.75, respectively). Both dominant and recessive models revealed significant association with T2DM ($p < 0.001$), with the G allele being significantly more frequent among patients (OR = 2.10).

This agreed with Parmar et al.(16), who showed that the GG genotype ($p < 0.01$) and mutant allele 'G' ($p < 0.025$) of LEPR rs1137101 A/G were associated with increased risk for T2DM with an Odds Ratio (OR) of 1.66 and 1.24, respectively, with modest effect size.

In our study, a significant link was noticed between the FTO (rs17817449) polymorphism and T2DM. Both the TG and GG genotypes were significantly related with increased T2DM risk as compared to the TT genotype (OR = 1.67, 3.10 respectively).

This agreed with Maghraby et al.(17), who showed that the minor G allele of FTO rs17817449 polymorphism was significantly higher in diabetics than controls. The association with T2DM was observed under additive, and dominant models of inheritance but the highest was observed with the recessive mode of inheritance.

In the current study, a significant connection was detected between the LEPR (rs1137101) polymorphism and obesity among T2DM patients ($p < 0.001$), with the GG genotype being much more frequent in obese subjects. GG carriers had significantly higher BMI levels than AA persons ($p = 0.002$).

This agreed with the study by Murugan et al.(18), that included 300 participants selected based on their responses

to the questionnaire and an Ayurvedic consultation. Blood samples were collected from these individuals, and DNA was extracted and analyzed for the rs1137101 variation using the PCR–RFLP method. The prevalence of the risk allele G was 50% in the obese group, compared to 38% in the normal group.

However, opposite to our findings, studies in Turkish and Brazilian populations showed no association between LEPR rs1137101 A/G polymorphism and BMI (18), (19). In the current study, a significant association was noticed between the FTO (rs17817449) polymorphism and obesity among T2DM patients ($p = 0.008$), with the GG genotype being more common among obese subjects. BMI levels were also significantly higher in GG and TG carriers than in TT subjects ($p = 0.012$).

This was also within the same line with Barseem et al.(20), who assessed the genotyping FTO rs17817449 polymorphism (SNP) in 120 obese children and 120 controls. FTO rs17817449G allele was positively associated with WC (Waist Circumference) (Mean $\pm$ SD 84.1 ± 9.3) and raised BMI (Body Mass Index) (32.7 ± 3.5). A significant link was found between the LEPR (rs1137101) polymorphism and lipid profile parameters. GG genotype carriers had significantly higher TG, total cholesterol, and LDL-C levels, but lower HDL-C levels compared to AA subjects ($p < 0.05$). Pairwise comparisons indicated that the most significant differences occurred between the AA and GG genotypes.

This agreed with Parmar et al. (16) who reported that the GG genotype was strongly correlated with elevated FBG and TC levels.

CONCLUSIONS

The study concluded that FTO (rs17817449) and LEPR (rs1137101) polymorphisms served as genetic risk predictors for T2DM in Egyptians. AG/GG (LEPR) and TG/GG (FTO) genotypes, along with G alleles, significantly increased T2DM risk (ORs 1.67–3.75), were associated with obesity and dyslipidemia, and may have contributed independently of obesity.

RECOMMENDATIONS

Further large-scale population-based studies were recommended to clarify the association of FTO (rs17817449) and LEPR (rs1137101) polymorphisms with T2DM. Multi-center studies with larger samples, especially in obese subjects, novel diagnostic polymorphisms, and

investigation of gene environment interactions in T2DM patients were also advised.

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