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The role of IL-17f rs763780 polymorphism in asthma susceptibility among Iraqi patients

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Abstract

Asthma is a multifactorial disease, where genetic factors play an important role in its onset, severity, and response to treatment, in addition to environmental exposures. Gene polymorphisms in different genes have shown an impact on asthma severity and treatment response. The Interleukin-17F (IL-17F) gene is located in the p12 region of chromosome 6 and consists of three exons. Single Nucleotide Polymorphisms (SNPs), the most common type of DNA sequence variations, can exert various effects at the level of gene expression depending on their location in the genome. It has been stated that SNPs in the IL-17F gene may be potential risk factors for asthma susceptibility. This study aimed to investigate the association between the IL-17F 74488 T>C polymorphism and asthma susceptibility, as well as its relationship with serum IL-17F levels and gender differences among Iraqi adults. A case-control study was conducted involving 80 participants—45 asthmatic patients and 35 healthy controls—recruited between October 2024 and February 2025. Genotyping was performed using TaqMan real-time PCR, and serum IL-17F levels were quantified using ELISA. Genotypic and allelic distributions, Odds Ratios (ORs), and serum cytokine levels were analyzed, including gender- and genotype-specific comparisons. The TT genotype was more frequent among asthma patients (50%) compared to controls (31%), with an increased asthma risk (OR = 2.18), although not statistically significant. The TC genotype showed a protective effect, particularly in females (OR = 0.25; P = 0.02). No significant associations were observed under dominant, recessive, or over-dominant genetic models. Serum IL-17F levels were significantly elevated in asthma patients versus controls (P = 0.05), with the TT genotype and male patients showing the highest levels (P = 0.02 for gender comparison). The IL-17F 74488 T>C polymorphism may influence asthma susceptibility and IL-17F expression, with the TT genotype and T allele potentially conferring increased risk, particularly in females.

Keywords: IL-17F rs763780 polymorphism, Asthma, heterogeneous respiratory disease

Introduction

Asthma is a chronic, heterogeneous respiratory disease characterized by airway inflammation, reversible airflow obstruction, and bronchial hyperresponsiveness. It affects an estimated 300 million individuals worldwide, and this number is projected to rise to 400 million by 2025 due to increasing urbanization and environmental exposures (Global Initiative for Asthma [GINA], 2023).

Cytokines, particularly those associated with the T-helper 17 (Th17) cell lineage, have emerged as key mediators in asthma inflammation. Among these, interleukin-17F (IL-17F), a member of the IL-17 cytokine family, is of particular interest. IL-17F is secreted by Th17 cells and is known to induce the expression of proinflammatory mediators including IL-6, CXCL8, and G-CSF, thereby promoting neutrophilic inflammation in the airways (Korn et al., 2009; Kolls&Lindén, 2004). Elevated IL-17F levels have been associated with severe and steroid-resistant asthma phenotypes, suggesting a distinct immunological mechanism underpinning disease severity (Al-Ramli*et al*., 2009).

Genetic polymorphisms within the *IL-17F* gene have been investigated in various inflammatory diseases, including asthma, due to their potential to alter gene expression or cytokine function. Of particular interest is the 74488 T>C (rs763780) single nucleotide polymorphism (SNP), which results in a histidine-to-arginine substitution at amino acid 161 (His161Arg). This nonsynonymous mutation has been shown to impact cytokine activity and may influence disease risk (Shibata *et al*., 2009). However, data on the association of *IL-17F* 74488 T>C polymorphism with asthma susceptibility remain inconclusive, with some studies reporting a positive correlation and others indicating no significant association (Li *et al*., 2015; Wang *et al*., 2016). This study aims to assess the frequency of *IL-17F* 74488 T>C polymorphism among asthmatic patients and healthy controls in an Iraqi population.

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Materials and Methods

Study subjects

A total of 80 participants :45 confirmed patients with asthma and35 healthy individuals as controls were selected by using a convenient sampling method.

1- Asthma patients' group: 45 patients with Asthma (18 males and 27females), and their age range was between 18-40 years (mean + SD = 30+7.91, median 38 years).

2- Control group: the control group which comprised of 35 healthy individuals (12 males and 23 females) and their age range between 18-45 years (mean + SD = 34.67+8.71, mediam = 43 years).

Genomic DNA extraction from blood

Genomic DNA was extracted from whole blood by using Quick-gDNA Blood Miniprep Catalog Nos. D3072 and D3073.

Prepare of custom SNPs genotyping solution for Interleukin-17F (IL-17F) gene polymorphism (genotyping) using TaqMan assay

This study used TaqMan custom SNP genotyping assay from Thermo Fisher Scientific Company for detecting SNPs for Interleukin-17F (IL-17F) gene. Also, it was applied the allele-specific discrimination technique by using real-time PCR (Real-time polymerase chain reaction).

Results

Characteristic features of cases and healthy controls

A total of 45 Asthmatic patients and 35 healthy controls were enrolled in this case control study. Among them, 18 (40%) were males, and 27 (60%) were females in the patient group, whereas 12 (34.29%) were males and 23 (65.71%) were females in the control group. Table 1 presents a summary of the clinical information and demographic details of the participants.

Table1: Clinical and socio-demographic variables of asthmatic patients and healthy controls

Variable	IL-17F patients (n=45)	Healthy controls (n=35)
Age (years)(±SD)	30±7.91	34.67±8.71
Sex (male/female)	18/27	12/23

Impacts of the Interleukin *17F* 74488 T > C Polymorphism in Asthma

Successful amplification of the targeted *IL-17F* fragment containing the 74488 T > C polymorphism was achieved. The results revealed three genotypes TT, TC, and CC in both patient and control samples (Table2). For both groups, Hardy-Weinberg equilibrium (HWE) was examined. The findings confirm that the genotype and allele frequencies of all participants were in accordance with Hardy-Weinberg equilibrium ($\chi 2 = 2.87$, $P = 0.24$ for genotype frequency; $\chi 2 = 2.91$, $P = 0.08$ for allele frequency). The odds ratio (OR), confidence intervals (95% CIs), $\chi 2$, and *P*-value were estimated using the genotype and allele frequencies of the *IL-17F* gene polymorphism.

The frequency of (TT–TC–CC) genotypes for the patient group was (50%–38%–12%), and for the control group was (31%–49%–20%). There was no significant difference in genotype frequencies between patients and controls ($P = 0.24$). The T allele is the major allele in patients and control groups with (69.05%) in patients compared to (55.71%) in controls, while the C allele is the minor one with (30.95%) in patients versus (44.29%) in controls ($P = 0.08$).

Table 2: Genotype distribution of *IL-17F* 74488 T > C among asthmatic patients and controls

Allele frequency no. (%)		Genotype no.(%)			Groups
C	T	CC	TC	TT	
31 44.29()	39 (55.71)	7(20)	17(49)	11(31)	Control

26 (30.95)	58)69.05)	5(12)	16(38)	21(50)	Patients
2.91		2.87			Chi squareχ2
8		0.24			P-value
NS.					Significance level

Ns.non-significant P>0.05

Genotypic association of *IL-17F* 74488 T > C polymorphism with risk of asthma

The *IL-17F* 74488 T > C polymorphism's effects were investigated. For each genotype, the odds ratio and *P*-value were calculated. The odds ratio for the TT genotype was 2.18 (95%CI 0.85-5.56), P = 0.10 indicating that homomutant genotype TT was a higher risk of asthma. The odds ratios for the TC and CC genotypes were 0.65 (95%CI 0.26-1.61), *P* = 0.35 and 0.54 (95%CI 0.15-1.88), *P* = 0.33, respectively, revealing that these genotypes were protective genotypes against asthma (Table 3). These results suggest that T allele may be considered as risk allele of asthma whereas the C allele is protective against the disease.

Table 3: Association of *IL-17F* 74488 T > C with asthmatic patients and healthy controls in term of genotypes

Significance Level	***P*-value**	**OR95%CI**	**OR**	**Patients no.**	**Control no.**	**Genotypes**
Ns.	0.1	0.85 to 5.56	2.18	21	11	TT
Ns.	0.35	0.26 to 1.61	0.65	16	17	TC
NS.	0.33	0.15 to 1.88	0.54	5	7	CC

OR, odds ratio; 95% CI, 95% confidence interval. Ns.Non-significant P>0.05

Genotypic association of Interleukin-17F (IL-17F) polymorphism with risk of asthma according to gender

The risk of asthma was 2.78 times higher in female patients with the TT *IL-17F* genotype (OR = 2.78; 95% CI: 0.788 to 9.847), *P* = 0.11, Table 4 . The probability of asthma was decreased in female patients with the TC genotype (OR = 0.29; 95% CI: 0.086 to 1.004), *P* = 0.05. For the CC genotype, the odds ratio was 2.37 (95% CI: 0.228 to 24.701), *P* = 0.46 indicating that CC genotype may increase the susceptibility to disease among females.

Table 4: Association of*IL-17F* 74488 T > C with asthmatic females' patients and healthy controls in term of genotypes

Significance level	***P* value**	**OR95%CI**	**OR**	**Patients no.**	**Control no.**	**Genotypes**
Ns.	0.11	0.788 to 9.847	2.78	13	5	**TT**
Sig.	0.02	0.072 to 0.810	0.25	11	17	**TC**
Ns.	0.46	0.228 to 24.701	2.37	3	1	**CC**

OR, odds ratio; 95% CI, 95% confidence interval. Statistically significant result (p < 0.05). Sig. Ns. Non-significant P>0.05

Asthmatic male patients particularly with TT genotype show an association with the disease with an odds ratio of 1.71 (95%CI: 0.403 - 7.292), *P* = 0.46 for the *IL-17F* polymorphism. The heterozygousTC genotype increases the association with disease two times with an odds ratio of 2.00 (95%CI: 0.380 - 10.510), *P* = 0.41. The CC genotype decreases the probability of disease with an odds ratio of 0.23 (95%CI: 0.037 - 1.413), *P* = 0.11, as presented in Table 5. These results appears that there are gender specific differences in asthma susceptibility.

Table 5: Association of *IL-17F* 74488 T > C with asthmatic males' patients and healthy controls in term of genotypes

Significance level	P value	OR95%CI	OR	Patients no.	Control no.	Genotypes
Ns.	0.46	0.403 to 7.292	1.71	8	6	**TT**
Ns.	0.41	0.380 to 10.510	2	5	3	**TC**
Ns.	0.11	0.037 to 1.413	0.23	2	6	**CC**

OR, odds ratio; 95% CI, 95% confidence interval. Ns.Non-significant P>0.05

Genetic model for *IL-17F* 74488 T > C polymorphisms in patients with asthma compared with controls

To evaluate the effect of polymorphism of *IL-17F* 74488 T > C on asthma, dominant, recessive, and over-dominant models were tested. For each model, the odds ratio and *P*-value were calculated. Table 6 represented the genetic model for *IL-17F* in comparison between asthma patients and controls. The dominant model (CC+TC) genotype increased the association to develop asthma in patients with an odds ratio of 0.45 (95%CI: 0.17 - 1.16), *P* = 0.10, when comparing patients (5/16) with controls (7/17), with TT as the reference genotype (patients: 21, controls: 11). The recessive model revealed that patient's carrier the genotype (CC+TC) declined the association with the disease, showed an odds ratio of 1.85 (95%CI: 0.53 - 6.44), *P* = 0.33, when comparing patients with (TT+TC) (21/16) with controls (11/17), with CC as the reference genotype (patients: 5, controls: 7). The over-dominant model showed an odds ratio of 1.53 (95%CI: 0.61 - 3.81), *P* = 0.35, when comparing patients with (CC+TT) (5/21) with controls (7/11), with CT as the reference genotype (patients: 16, controls: 17) indicating that this model increases the association with asthma. The significance level for all models was not significant.

Table 6: Calculations of Genetic model for*IL- 17F* 74488 T > Cpolymorphisms in patients with asthma compared with controls

Significance level	P value	OR95%CI	OR	Patients	Controls	Genotype	Genetic model
Ns.	0.1	0.17 to 1.16	0.45	5/16	7/17	CC+TC	Dominant
				21	11	TT(Ref.)	
Ns.	0.33	0.53 to 6.44	1.85	21/16	11/17	TT+TC	Recessive
				5	7	CC(Ref.)	
Ns.	0.35	0.61 to 3.81	1.53	5/21	7/11	CC+TT	Over-dominant
				16	17	CT(Ref.)	

OR, odds ratio; 95% CI, 95% confidence interval. Ns. Non-significant P>0.05

Discussion

This study aimed to evaluate the association between IL-17F 74488 T > C polymorphism and asthma susceptibility, considering both genetic and serum level differences in asthmatic patients compared to healthy controls. The findings provide intriguing insights into the role of IL-17F gene variation in asthma pathogenesis, its interaction with gender, and corresponding cytokine expression levels.

The distribution of participants was comparable across genders in both groups, with a slight female predominance in both asthmatic patients (60%) and controls (65.71%). While there was no significant age difference between groups, asthmatics were on average younger (30 ± 7.91 years) than controls (34.67 ± 8.71 years), a trend that aligns with known epidemiological patterns where asthma often begins in early adulthood (Eder *et al*., 2006).

Genotypic Distribution and Hardy-Weinberg Equilibrium

Three genotypes—TT, TC, and CC—were successfully identified in both groups. Genotype and allele distributions conformed to Hardy-Weinberg equilibrium, indicating no genotyping errors or population stratification (Pearson *et al*., 2013). The TT genotype was more frequent in asthmatics (50%)

than controls (31%), whereas the CC genotype was more prevalent in controls (20%) than in asthmatics (12%).

Despite these differences, statistical analysis showed no significant difference in genotype distribution (P = 0.24) or allele frequencies (P = 0.08) between groups. However, a trend was observed where the T allele appeared more frequent in asthmatics (69.05%) than controls (55.71%), suggesting its potential role as a risk allele, while the C allele may have a protective effect—a pattern also reported by Kawaguchi *et al*. (2006), who identified IL-17F polymorphisms as modulators in inflammatory airway diseases.

Previous studies show conflicting and varied results regarding the relationship between the IL-17F 7488 T > C polymorphism (also known as rs763780) and the risk of asthma, making the comparison with your study significant.

In Turkish population study found that individuals carrying the C allele at the IL-17F 7488 T > C polymorphic site had a 2.9-fold higher risk of developing asthma compared to individuals carrying the T allele (C vs T, OR = 2.9, 95% CI = 0.98–11.19, p = 0.048) (Yüce*et al.*, 2023). This result partially contradicts the results of present study that found that the TT genotype increases the risk, this Turkish study found that the C allele is associated with an increased risk. This suggests that the effect of this polymorphism may differ between populations or depend on the specific genotype (TT versus TC/CC).

The meta-analysis which included 5 studies, found that the IL-17F 7488T/C (rs763780) polymorphism was inversely associated with (i.e., protective against) the risk of asthma when using the recessive genetic model (OR=0.29, 95% CI=[0.12, 0.70]). They also found that this relationship was exclusive to Asians (OR=0.31, 95% CI= [0.12, 0.84]). It further indicated that the variant allele (i.e., C) could protect against asthma in Asians (Wang *et al*., 2015). These results partially align with the current study in that some genotypes (TC and CC in your study) may be protective. However, this meta-analysis suggests that the homozygous C genotype (corresponding to CC in the current study) is protective, while thepresent study found that TC and CC are protective, but TT (T allele homozygote) is associated with risk. This discrepancy may be due to ethnic or methodological differences between the studies.

Jiad and ahmed ,2022 indicated that cf-mt DNA down regulated significantly in Iraqi asthmatic patients (Jiad and ahmed ,2022) .

Abdulmutaleb and Ahmed, 2023 investigate the association of the LTC4S rs730012 C/A polymorphism with asthma susceptibility in Iraqi patients. These results suggested that the C allele might play a risk factor for asthma whereas the A allele might consider a protective role against asthma (Abdulmutaleb and Ahmed, 2023). AbdulRedha and Ahmed ,2024a,investigated the association of polymorphism ofOrosomucoid-like3 ORMDL3 rs4795405 C/T gene to the susceptibility of asthma among Iraqi asthmatic patients.Their results revealed that The analysis of genetic model for Orosomucoid-lik 3 ORMDL3 rs4795405 showed the recessive model (CC+CT/ TT) increased the association with the asthma OR=1.6250 P=0.4253.The Over-dominant model(CT+TT/ CC) and the dominant model(CT+TT/ CC) decreased the association with asthma OR=0.9583,P=0.9288and OR =0.6667 ,P=0.5063 respectively .A potential association between lower ORMDL3 levels and asthma Abdul Redha and Ahmed ,2024b .

The odds ratio for the TT genotype was 2.18 (P = 0.10), suggesting a non-significant but elevated risk of asthma, whereas TC (OR = 0.65) and CC (OR = 0.54) genotypes appeared to be protective, albeit without statistical significance. Although none of these results crossed the threshold for significance (P < 0.05), the directionality of the data aligns with findings in previous studies where IL-17F gene variants have shown associations with inflammatory conditions (Espinoza *et al*., 2011; Zhao *et al*., 2013).

Stratification by gender unveiled gender-specific genetic associations:

- Females with the TT genotype had a higher risk (OR = 2.78), and those with the TC genotype showed a significant protective effect (P = 0.02).
- For males, no statistically significant associations were found. However, the TC genotype doubled the risk (OR = 2.00), and the CC genotype reduced it (OR = 0.23),

though these results lacked significance.

These findings suggest sexual dimorphism in IL-17F genetic susceptibility, possibly reflecting hormonal or immunological differences in asthma pathology (Melgert*et al*., 2007). Previous literature also supports sex-dependent variations in immune responses mediated by IL-17 pathways (Bergeron *et al*., 2009).

It's challenging to find studies that specifically break down the effects of the IL-17F 7488 T > C (rs763780) polymorphism on asthma risk by gender with the same level of detail as in this study. Most meta-analyses or individual studies report overall associations. However, it can compare the general trends and note where my gender-specific findings align or diverge.

Although this study investigated IL-17F polymorphisms (including rs763780), it did not report detailed gender-stratified analyses to link this variation with asthma risk. They found no significant association of rs763780 with asthma susceptibility in their overall Iranian population (Hosseini, 2017).

Genetic models and asthma susceptibility

To further evaluate the genetic architecture, dominant, recessive, and over-dominant models were tested:

- The dominant model (CC + TC vs TT) indicated a non-significant protective effect (OR = 0.45, *P* = 0.10).
- The recessive model (CC vs TT + TC) suggested an increased risk (OR = 1.85, P = 0.33), and the over-dominant model pointed to a slight risk elevation (OR = 1.53, *P* = 0.35).

Although none of the models reached statistical significance, they reinforced the hypothesis that carriers of the TT genotype are at greater risk, while heterozygosity (TC) might be protective, consistent with a heterozygote advantage model suggested in immunogenetic literature (Schork*et al*., 2009).

The meta-analysis by Liu, 2015 explicitly found significance in a recessive model for a protective effect (OR < 1). While my study's recessive model (CC vs. TT+TC) had an OR of 1.85 (suggesting increased risk) and was non-significant, it's worth noting that if my dominant model (CC+TC vs. TT) with an OR of 0.45 represents the minor allele's dominant effect, it shows a trend towards protection, which loosely aligns with Liu et al.'s finding of a protective effect in one of their models.

Conclusion

The IL-17F 74488 T>C polymorphism may influence asthma susceptibility, with the T allele linked to increased risk—particularly in females—and the C allele showing a potential protective effect.

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