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ORIGINAL ARTICLE

Single-Nucleotide Polymorphism in Iraqi patients of TNF-α-308G/A(rs1800629) Susceptibility to Asthma

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Abstract

Objectives: The study examined the effects of genetic variations in TNF-α-308G/A, as well as TNF-α gene variants, using PCR-SSP to analyze individual characteristics.

Method: Researchers used molecular and immunological methods to investigate genotypes and alleles associated with asthma incidence, including 50 asthma patients and 40 healthy individuals.

Results: It was demonstrated that polymorphisms and the risk of developing asthma were correlated among asthma patients. The findings showed that, in comparison to the control group, asthma patients had considerably ($P<0.05$) greater levels of TNF-α-308G/A alleles than GA heterozygotes (TNF-α-308G/A).

Conclusion: Gene promoter polymorphisms may affect asthma sensitivity to TNF-α-308G/A, as evidenced by the strong association between the GG and AG gene genotypes and cytokine levels and disease development.

The development of asthma and immunological markers (TNF-α) are closely related. One perspective holds that there is a correlation between allergic rhinitis and the development and risk of asthma. It has been successful to use HDM immunotherapy to assist patients in establishing long-term clinical and immunological tolerance.

Keywords: asthma, Iraqi patients, TNF-α-308G/A, susceptibility, polymorphism SSP-PCR technique

INTRODUCTION

Asthma is characterized by complex pathophysiology involving airway inflammation, intermittent airflow obstruction, and bronchial hyper responsiveness. The condition presents various signs and symptoms, such as wheezing, coughing, shortness of breath, and chest tightness [1]. The symptoms of this illness include dyspnea, wheezing, chest tightness, and recurrent episodes of coughing that might vary in severity over time [2]. A major global health concern, asthma affects at least 3.5-20% of any country's population [3]. In 2020 asthma and chronic lung disease together were the third most important reason of death [4-5]. The promoter regions of several cytokine genes contain polymorphisms that can directly impact the transcription or expression of the cytokine, leading to either high or low production levels of that particular cytokine [6]. The hereditary pro- and anti-inflammatory cytokines are influenced by a variety of factors [7]. A change in DNA sequence that occurs in individuals belonging to the same species is called polymorphism, and it is considered less common if it is present in at least 1% of the population being studied [8]. Several studies have shown that cytokines and cytokine gene polymorphisms are associated with asthma susceptibility in different ethnic groups [9]. There are a number of genetic differences that influence severity of asthma development, patient response to treatment, and degree of heritability of the condition [10,11]. Because cytokines contribute to the inflammatory process linked to the disease, and are coordinated and controlled by a complex network, cytokines are thought to be important participants in the pathogenesis. Variations in the genes of these cytokines have been linked to changes in their plasma concentrations [12]. T-cells, NK cells, and macrophages release the inflammatory cytokine TNF-α in response to allergens. Asthma risk has previously been linked to this area; genetic studies have linked polymorphisms in the TNF-α gene to asthma risk.

The A allele is connected with higher levels of TNF-α in bronchoalveolar fluid and the fluid used to clean asthmatics' airways [10]. A recent meta-analysis found a correlation between a upper occurrence of asthma in children and adults [13-14]. This region has previously been associated with asthma risk; genetic studies have connected mutations in the TNF-α gene to asthma risk.

The present study aimed to explore the role of genetic variants in the TNF-α gene as well as the relevance of genotypes and alleles associated with sickness occurrence. Differences in the two genes may affect the levels of TNF in the airways. The TNFA nucleotide -308G is the promoter region. A polymorphism that substitutes guanine (G) for adenine (A) has been associated with higher levels of secretion and promoter activity. The polymorphism LTA 252A>G, found in the first intron of LTA, is thought to be associated with elevated LT-α production [15]. By looking at individual traits including age and gender, the present study aimed to discover the role of alleles and genotypes related with the advance of asthma as well as genetic variations of the TNF-α gene.

MATERIALS AND METHODS

Collection of samples

From October 2018 to the end of July 2020, fifty asthma patients from the Al-Anbar Teaching Hospital's Allergy Centre participated in the study. Thirty-one of these patients were men, and 19 were women. Ethical approval was given by the University of Anbar University's Scientific Study Ethics Committee, which is overseen by the

Ministry of Higher Education and Scientific Research (Number 114, dated 3-5-2018). An anonymous questionnaire containing the patients' medical history and other relevant data was used for in-person interviews. Each participant gave their verbal agreement in compliance with the ethical protocols of Al-Anbar Hospital. Twenty men and twenty women who were all in good health and did not have asthma were chosen at random to be in the control group. Both ELISA and traditional PCR were used to analyze the control group. A questionnaire was presented to each member of the control group, none of whom had an acute or chronic disease. Asthma sufferers and healthy controls sat while three milliliters of venous blood were drawn using disposable syringes.

Extraction of human genomic DNA

DNA was extracted from blood samples in accordance with the instructions supplied by the GeneiusTM Micro-DNA Extraction Kit (Geneaid, USA).

Determination of DNA concentration and purity

A NanoDrop Spectrophotometer (THERMO, USA) was used to test and determine the purity of the extracted genomic DNA.

Preparation of Primers Suspension

The stock solution containing 100 mol/L of the required concentration was made by dissolving the lyophilized primers in deionized D.W.

Polymerase chain reaction protocol

An SNP that is known can be found using the SSP-PCR method. It is composed of two complimentary reactions: one includes a primer specific to the normal DNA sequence and amplifies mutant DNA at a particular locus without amplifying normal DNA [16]. A cool micro centrifuge was used to combine the PCR components in a PCR tube for 10 seconds at 50 rpm, as shown in Table 1.

Table (1): The specific primers and their sequences

Gene	Sequences (5́-3́)	Size bp
TNF-α-308G/A	F: CTG CAT CCC CGT CTT TCT CC R1: ATA GGT TTT GAG GGG CAT CG R2: ATA GGT TTT GAG GGG CAT CA	836

Mixture PCR

DNA polymerase, free nucleotides known as ddNTPs, DNA primers, and a DNA sample were among the many ingredients needed for PCR, as seen in Table 2.

Table (2): The mixture of PCR

PCR master mix reaction		Volume / µl
GoTaq®Promega Green Master Mix 2X		12.5
DNA template		5
Primers	Forward	2
	Reverse	2
Nuclease Free water		3.5
Total volume		25

Thermocycling condition

The PCR thermocycler settings for each sample were carried out using a standard PCR thermocycler equipment [16].

Preparation of Agarose Gel

For DNA profiling, 1% of an agarose gel was created in line with [17]. A final concentration of 1X and PH 8 was achieved by dissolving one gram of agarose powder in 100 milliliters of TBE buffer (90 milliliters of D.W. were added to ten milliliters of TBE buffer 10X). To create an agarose gel, this procedure was repeated, after a minute in the microwave at 90 degrees Celsius. The agarose-ethidium bromide solution was put into the gel tray of the electrophoresis apparatus with the combs, and fixed combs were used to close the two ends. The agarose was allowed to crystallize at room temperature for half an hour. The combs and seals were carefully removed from the tray. The purpose of the combs was to serve as wells for the insertion of DNA samples. Five microliters of the amplified PCR produce were added to each agarose gel well, and one well was then loaded with a ladder, a DNA marker.

RESULTS

According to the study's findings, 38% of people in the 41–50 age group and 28% of those in the 31–40 age group had the condition (table 3).

Table 3 Patients with asthma distributed according to age groups and gender

Age (years) Group	Males N.	Females N.	Total N.(%)
10-20	2	4	6(12)
21-30	3	8	11(22)
31-40	7	7	14(28)
41-50	7	12	19(38)
Total	19(38%)	31(62%)	50(100%)

The results revealed that asthmatic patients (20%) had a greater homozygous genotype AA frequency than the healthy controls (7.5%) (Figures 1, 2, and 3). As shown in Table (4), there was a significant difference in the prevalence of homozygous genotype GG between the control and patient groups, and the distribution of homozygous genotype AA was statistically different between the two groups. In the former, 50% of patients had the GA genotype. About 35% of healthy controls and 24% of asthmatic patients had this genotype. There was a substantial association between the two, with asthmatic patients having a greater prevalence of the A allele (48%), compared to controls (32.5%). The relative frequency of the "G" allele was 67.5% in the control group and 52% in asthmatic patients.

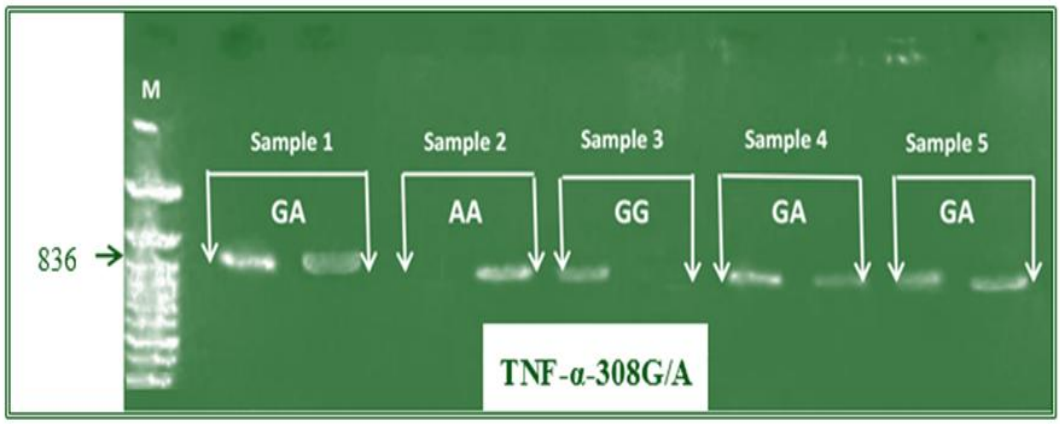

Figure 1 : PCR-amplified products (836 bp) demonstrating five sample genotypes for the TNFα -308 G/A gene (rs1800629).Samples 1 through 5 are GA, AA, GG, GA, and GA, respectively. DNA Ladder -M:-(100 bp).

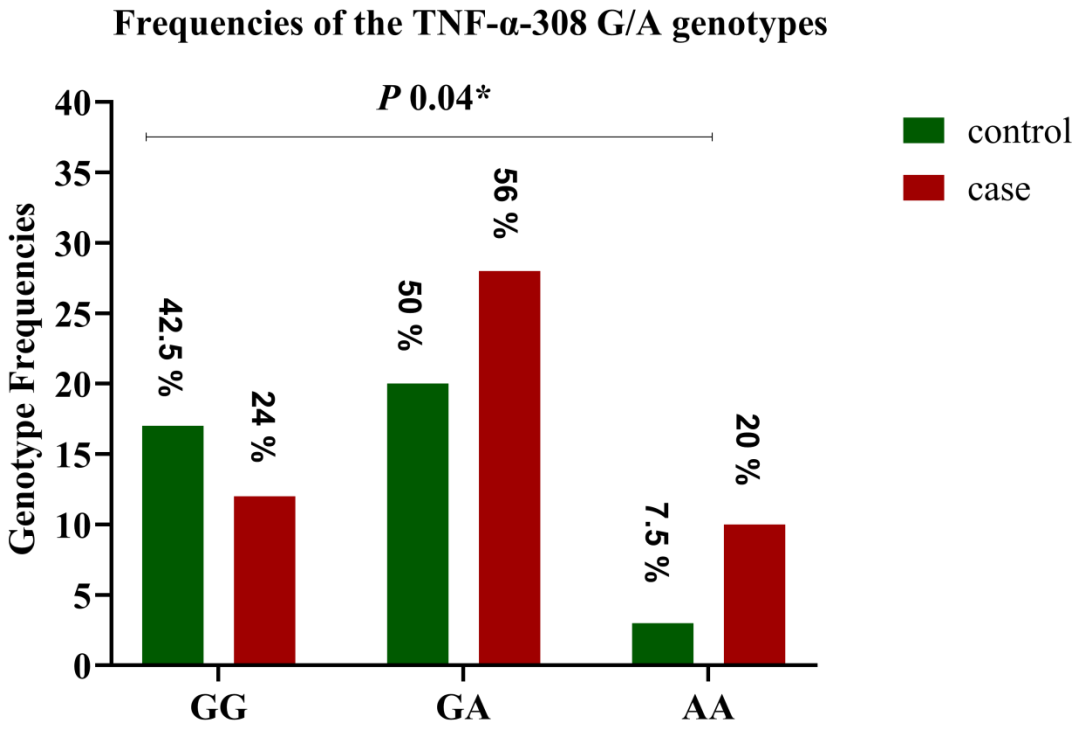

Figure 2: Comparison of the relative frequencies of the three chosen TNF-α-308 G/A genotypes between patients and controls

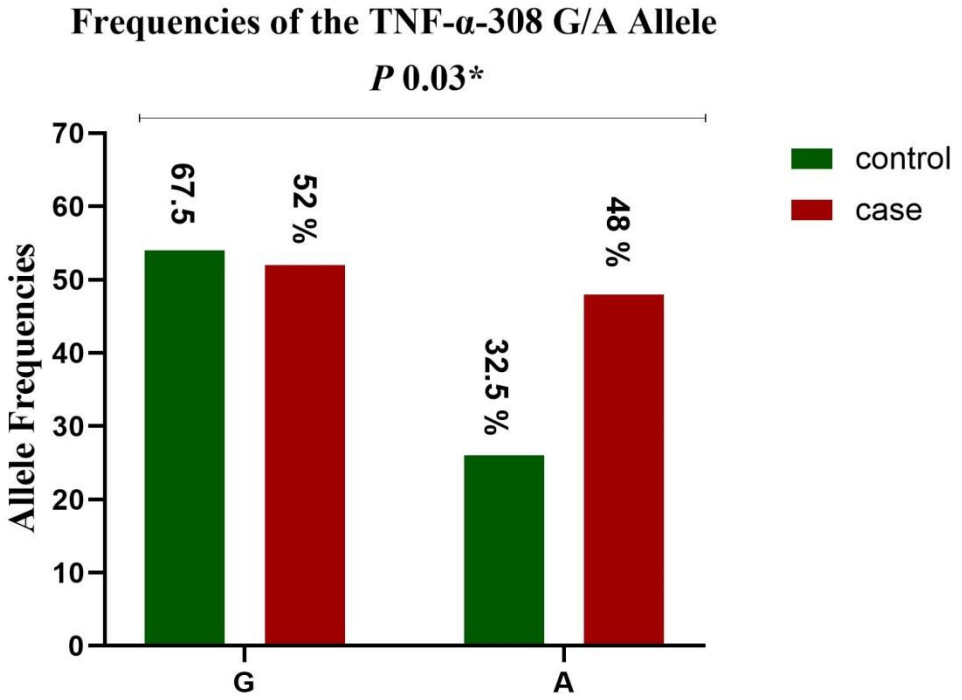

Figure 3: Comparison of Relative Frequency of TNFα-308 G/A Gene Alleles in Patients and Controls.

Table 4: TNF-α-308 G/A genotype and allele frequencies, including incidence and distribution, in asthmatic patients and controls

Genotype	Control (No.= 40)		Cases (No.= 50)		P-Value	OR (95% CI)
	Fr.	%	Fr.	%		
GG	17	42.5	12	24	0.04*	1.8 (0.81 to 5.18)
GA	20	50	28	56		
AA	3	7.5	10	20		
Allele						
G	54	67.5	52	52	0.03*	1.9 (1.103 – 3.507)
A	26	32.5	48	48		

OR=Odd ratio, P(alpha<0.05)

DISCUSSION

shows that the average ages of the patients in the control group were 35.1±11.8 and 30.8±10.2, respectively (P = 0.177). shows that the average ages of the patients and the control group were 35.1±11.8 and 30.8±10.2, respectively (P = 0.177). This is consistent with 397 people with severe asthma, whose mean age was 48.95 years (standard deviation 13.55), were included in the information collection. selected participants from the 1958 British Birth Cohort who were in the same age range and had a balanced sex ratio to establish the control group [18]. This

study investigated the association between 333 adult Pakistani patients with a diagnosis of asthma and 16 SNPs in 10 putative genes. The control group consisted of two hundred healthy, non-asthmatic individuals who were matched for race and gender [19].

Distribution of Asthma Patients Depending on Age.

According to a local study by Al-Shamma et al. adults aged 50–59 years, represent cases of the uppermost prevalence of asthma, followed by those aged 40–49 years. As people age, their pulmonary function decreases, making them more susceptible to respiratory diseases [20]. Wan et al. conducted a similar study to ours, examining the age distribution of asthma patients amongst people aged 45 and older. This study highlighted that asthma prevalence increased with age, particularly among mid-aged and ageing populaces. According to the study, the odds of having asthma are significantly higher for individuals aged 60-69 and those over 70 compared to younger age groups [21]. Another relevant study is one that evaluated the incidence of asthma diagnosis across different age groups. This study provided insights into how asthma incidence varies with age and identifies differences between asthma diagnosed in childhood versus adulthood [22]. Additionally, the US National Health and Nutrition Examination Survey (NHANES), reported that those 60 years of age and older had the greatest prevalence of asthma. This study also emphasized that lifestyle choices, gender, and race might affect asthma prevalence in a variety of age groups [23,24]. The Swiss Cohort Study on Air Pollution and Lung and Heart Disease in Adults investigated asthma incidence across the adult lifespan, with a focus on gender differences. The study found that asthma incidence is generally higher in women than in men, with a reported incidence of 4.6 per 1000 person-years in women compared to 3.6 per 1000 person-years in men. The study also noted that asthma incidence tended to increase with age, although sex-specific trends are less conclusive [24]. According to Hernandez et al. women with asthma, particularly those aged 18-24, experienced a larger deviation from general population-based health-related quality of life (HRQoL) norms compared to men, indicating that asthma had a more significant impact on the quality of life for younger women[25].

Molecular and Immunological Study

One important potential gene linked to the genesis of the complex polygenic disease known as asthma is the TNFα gene. Numerous studies have examined the relationship between TNFα rs1800629 polymorphism (rs1800629 G identified as TNF1) and asthma susceptibility in different populations (rs1800629 A designated as TNF2). There is proof that TNF2 and asthma are positively correlated. There has been evidence of a positive correlation between TNF1 and asthma in one research, but a negative correlation in other studies [26,27].

TNF-α has a significant role in the pathogenesis of autoimmune diseases. The development of autoimmune illnesses and polymorphisms in the TNF-α gene promoter that impact their severity or susceptibility seem to be significantly correlated [28] [29]. Khudhair et al found associations between TNF-α gene polymorphisms and asthma risk [30]. They concentrated on the TNFα gene polymorphism at position -308G/A since it is associated with higher plasma TNF-α levels and larger amounts of TNF-α following inspiration in vivo and in vitro. This site is crucial since it has been proposed as a

potential candidate gene for asthma [31, 32]. According to Louis et al, a correlation exists between elevated incidence of asthma and TNF. Their findings demonstrated a significant correlation between Iraqi patients' chronic asthma and the TNF-a-308 AA polymorphism. The A allele was found to be around twice as predominant in asthmatic patients as in the control group, indicating a clear correlation between the AA genotype and the allele. The TNFA-308G allele-containing genotype was significantly less prevalent in asthmatics than in normal controls [34]. Children with TNF–308 GG had a lower lifetime risk of wheezing and asthma [14]. Nonetheless, as the GG genotype and "G" allele primarily serve preventative purposes, the "G" allele could offer protection. The TNF-α-308G>A gene's promoter region has many polymorphisms, one of which is the SNP at position _308G/A. The TNF-α A allele was shown to be more common in asthmatics than in controls, and it was linked to increased TNF-α production [35]. An important connotation between TNF-α polymorphisms and asthma was reported, in addition to a high association between TNF-a-308 AA polymorphism and chronic asthma. According to this finding, those who have the GG homozygote have a lower risk of developing asthma than people who have the AA or AG genotype. comparing the carriers of the variation A allele with the homozygote GG [14]. Similar findings were published by [34], which showed that Asian people with the A allele had a much higher chance of developing asthma and that there was a strong correlation between TNF-α-308 AA and asthma. The TNF-α-308 GA genotypic polymorphism may be linked to the level of asthma susceptibility in Egyptian children, according to [36]. Previous association studies indicated that Punjabi patients [37, 38] were more likely to be from North India. In Korea and Iran, a substantial association between the TNF-a-308 GA promoter polymorphism and asthma was discovered [10,39,40]. Other studies found a significant association between one SNP (TNF-α-308G>A) and the incidence of asthma [27,41]. The incidence of asthma was shown to be significantly correlated with the single nucleotide polymorphism TNFA-308G>A, according to statistical analysis. Genetic differences in TNF, linked to the most common haplotype of the TNF gene, can cause asthma. The TNF-α-308G>A promoter polymorphism has been linked to an increased risk of asthma in a number of independent investigations [2].

CONCLUSIONS

Variations in blood TNF levels can explain different connections with diseases in different groups and may also mark expression divergence, fitness, and evolution. The pathogenesis of asthma in the Iraqi population is significantly influenced by TNF-α cytokines. Determine the connection between the TNFA-308G>A gene polymorphism and asthma risk. The G allele of TNF-α-308 may operate as a protective factor against the sickness, whereas the A allele and the AA genotype are most linked to the start of the illness.

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تعدد أشكال النوكليوتيدات المفردة لدى المرضى العراقيين الذين لديهم قابلية للإصابة بالربو من خلال عامل نخر الورم ألفا−308(rs1800629)G/A

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الملخص

الخلفية والأهداف : تشمل التأثيرات الفسيولوجية المهمة لعامل نخر الورم ألفا (TNF−α) الالتهاب وإعادة تشكيل مجرى الهواء. يُنتج عامل نخر الورم ألفا (TNF−α) بكميات أكبر بواسطة الخلايا الوحيدة والبلعمية في الدم المحيطي لدى مرضى الربو عند تعرضهم لمسببات الحساسية. ويُعزى تنشيط العضلات الملساء، وفرط استجابة مجرى الهواء في المرحلة المتأخرة، والتهاب مجرى الهواء لدى مرضى الربو إلى عامل نخر الورم ألفا

منهجية الدراسة : تدرس الدراسة آثار الاختلافات الجينية في عامل نخر الورم ألفا (TNF−α−308G/A)، بالإضافة إلى متغيرات جين عامل نخر الورم ألفا، باستخدام تفاعل البوليميراز المتسلسل−التسلسل الجزيئي (PCR−SSP) لتحليل الخصائص الفردية. استخدم الباحثون أساليب جزيئية ومناعية لدراسة الأنماط الجينية والأليلات المرتبطة بحدوث الربو، بما في ذلك 50 مريضًا بالربو (31 أنثى و19 ذكرًا) و40 فردًا سليمًا (20 أنثى و20 ذكرًا).

النتائج : ثبت أن تعدد الأشكال الجينية وخطر الإصابة بالربو مرتبطان، وأن تعدد أشكال النوكليوتيدات المفردة لعامل نخر الورم ألفا−PCR308) SNP) G/A) كان له انتشار أعلى بكثير ($P < 0.05$) بين مرضى الربو. تُظهر النتائج أنه بالمقارنة مع المجموعة الضابطة، كان لدى مرضى الربو مستويات أعلى بكثير ($P < 0.05$) من أليلات عامل نخر الورم ألفا−G/A308 مقارنةً بمتغايري الزيجوت (TNF−α−308G/A) GA). قد تؤثر تعدد أشكال محفز الجينات على حساسية الربو لعامل نخر الورم ألفا−G/A308، كما يتضح من الارتباط القوي بين النمط الجيني لجينات GG وAG ومستويات السيتوكينات وتطور المرض.

الاستنتاجات : يرتبط تطور الربو والعلامات المناعية (TNF−α) ارتباطًا وثيقًا. يرى أحد الآراء وجود علاقة بين التهاب الأنف التحسسي وتطور الربو وخطر الإصابة به. لقد كان من الناجح

الكلمات الدالة: الربو، المرضى العراقيون، ((TNF−α−308G/A، القابلية للإصابة، تعدد الأشكال الجيني، تقنية تفاعل البوليميراز المتسلسل باستخدام بادئات خاصة بالتسلسل (SSP−PCR)