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Original Research Article

Polymorphisms of the Interleukin-17 Gene and their Association with Physiological Variables in Leukemia Patients

Noor Rassam Kamil[1]*, Riham Firas Faris Abed[1], Haneen Mohanad Maher[1]

[1]Biology Department, College of Science, Tikrit University, Iraq

***Corresponding Author:** Noor Rassam Kamil
Biology Department, College of Science, Tikrit University, Iraq

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Abstract: ***Background*:** Interleukin-17 (IL-17) gene polymorphisms have been implicated in modulating immune responses and inflammation, which are critical factors in leukemia pathogenesis. ***Objective*:** This study aimed to investigate the association between the IL-17 rs3819024 polymorphism and physiological markers—IRF8 and TGF-β—in leukemia patients compared to healthy controls. ***Methods*:** A total of 90 participants aged 16 to 45 years were enrolled, including leukemia patients and healthy controls. Genotyping of IL-17 rs3819024 was performed using the Tetra-ARMS PCR technique. Serum levels of IRF8 and TGF-β were measured by ELISA. Statistical analyses assessed differences in marker levels between genotypes and groups, alongside allele and genotype frequency comparisons. ***Results*:** The AG genotype carriers among leukemia patients displayed slightly elevated IRF8 (42.24 ng/mL) and TGF-β (45.66 ng/mL) mean levels but the confidence intervals overlapped. The AA genotype carriers among healthy controls showed elevated IRF8 levels but AG carriers displayed significantly higher TGF-β levels (32.88 ng/mL vs. 15.29 ng/mL). The G allele and AG genotype appeared more frequently in healthy controls than in leukemia patients according to allelic analysis which indicated a protective effect against leukemia. The correlation between IRF8 and TGF-β showed weak results that failed to reach statistical significance. ***Conclusion*:** The IL-17 rs3819024 polymorphism produces different effects on IRF8 and TGF-β expression between leukemia patients and healthy individuals which could influence their immune regulation and disease susceptibility. Further research is needed to understand the role of IL-17 in leukemia pathophysiology based on these findings.

Keywords: IL-17 polymorphism, leukemia, IRF8, TGF-β, genetic association.

1. INTRODUCTION

Leukemia represents a wide range of hematological malignancies which develop when abnormal leukocytes multiply uncontrollably inside bone marrow and blood vessels. The abnormal growth pattern disrupts normal hematopoiesis leading to anemia and infections and bleeding disorders according to Chennamadhavuni *et al*., (2023). The leukemia family consists of acute and chronic subtypes that show different pathophysiological and clinical characteristics (Leszczenko *et al*., 2021). Despite the recent improvements in leukemia biology research and therapeutic methods the disease continues to be one of the main causes of cancer deaths throughout the world. There is an immediate requirement for ongoing research about the molecular pathways that drive leukemia development and evolution (Looi *et al*., 2021).

Leukemia biology heavily depends on the relationship between cancerous cells and the immune system. Cytokines function as crucial signaling proteins between immune cells to establish communication and play a vital role in this interaction (Zenobia and Hajishengallis, 2015). Among these cytokines, Interleukin-17 (IL-17) stands out as an essential cytokine which regulates both inflammatory and immune system responses. The main source of IL-17 production occurs in Th17 cells among T-helper cells which subsequently activates neutrophil recruitment and inflammation (Zhang *et al*., 2025). The protective role of IL-17 in fighting pathogens remains important yet its abnormal expression leads to autoimmune diseases and chronic inflammation together with cancer development (Li *et al*., 2019).

IL-17 functions in cancer through different mechanisms which sometimes work in contradictory ways. IL-17 functions to promote tumor development through enhanced angiogenesis and improved immune evasion and the creation of pro-tumorigenic inflammation in specific contexts (Zhao *et al*., 2020). IL-17 acts as a dual agent which stimulates both anti-tumor immunity through T cell and NK cell activation. Research on IL-17 becomes increasingly important for cancer studies because it functions in leukemia and other hematological malignancies where immune system dysfunction is prevalent (Kuen *et al*., 2020).

Multiple genetic variations found in the IL-17 gene including single nucleotide polymorphisms (SNPs) determine how much IL-17 is produced and how it functions (Baindara, 2024). The polymorphisms adjust the inflammatory environment which determines how people react to diseases and their progression and therapy outcomes (McGeachy *et al*., 2019). IL-17 gene polymorphisms have been associated with autoimmune conditions and chronic inflammatory diseases and cancers but their relationship to leukemia is not well established (Qian *et al*., 2017; Zhang *et al*., 2020; Chandra *et al*., 2024).

Analyzing how IL-17 gene polymorphisms affect cytokine levels and regulatory protein expression in leukemia patients could produce essential insights about disease mechanisms. The immune response regulators IRF8 and Transforming Growth Factor Beta (TGF-β) play essential roles in immune regulation and have been shown to influence leukemia progression (Huangfu *et al*., 2023). IRF8 functions as a transcription factor which regulates immune cell differentiation together with function and TGF-β functions as a multifunctional cytokine that controls immune suppression and tissue remodeling. IL-17 polymorphisms affect the expression levels or activity of these regulatory molecules leading to modified disease progression (Eshwar *et al*., 2022).

Research into the relationship between IL-17 gene polymorphisms and physiological measurements in leukemia could help find new markers for disease outcomes as well as therapeutic possibilities. The practice of personalized medicine uses genetic profiling to create customized treatments for individual patients which results in better treatment outcomes and decreased adverse effects (Han *et al*., 2022).

The research investigates IL-17 gene polymorphisms with a focus on rs3819024 SNP along with their effects on IRF8 and TGF-β levels in leukemia patients. This research analyzes leukemia pathogenesis and immune regulation by studying genotype-phenotype correlations through comparison between leukemia patients and healthy controls.

2. MATERIALS AND METHOD

2.1 Sample Collection

The research collected 90 samples from participants between 16 and 45 years old throughout June 1, 2024 to September 1, 2024. The collected samples were separated into two distinct groups which included patients and healthy visitors at the Specialized Hospital for Leukemia Patients in 2024. Specialist physicians performed biochemical analyses to confirm or exclude leukemia diagnosis in all participants.

2.2 Serum Preparation

Five milliliters of venous blood were drawn from each individual, whether diagnosed with leukemia or not. The collected blood samples were divided into two portions. The first portion consisted of 2 mL of blood placed in EDTA tubes to prevent coagulation and stored for later molecular analysis. The second portion consisted of 3 mL of blood placed in silicone gel tubes. The samples were then centrifuged at 3500 rpm to separate the serum. The serum was transferred into Eppendorf tubes and stored at −20°C. All relevant sample data were recorded and documented in preparation for the biochemical analyses. In this study, the concentrations of TGF and TRF8 were measured using the ELISA technique with commercially prepared reagent kits, following the manufacturer's instructions for each assay.

2.3 Molecular Study

Genomic DNA was extracted from the blood samples of all participants (both patients and controls) using a commercially available extraction kit from QIAamp DNA Blood Mini Kit. The concentration and purity of the extracted DNA were measured. The Tetra-ARMS PCR technique was employed to genotype the single nucleotide polymorphism (SNP rs3819024) in the IL-17 gene. Four specific primers were designed to amplify both alleles in a single reaction as shown in Table 1.

Table 1: Primer Sequences Used for Genotyping SNP rs3819024 in the IL-17 Gene Using Tetra-ARMS PCR

Primer Name	Sequence	No.
IF	GGCCAAGGAATCTGTGATGA	1
IR	TTGATTTTCCATTTGATCTTTCTGTC	2
OF	ATCTCCATCACCTTTGTCCAGTC	3
OR	GGAAGGGCAGAAATTCATGTTCCTA	4

PCR was performed in a final volume of 25 µL using a ready-to-use Master Mix containing Taq DNA Polymerase and the necessary components for amplification. Each primer was added at a concentration of 10 pmol/µL (1 µL per primer), along with 2 µL of template DNA at a concentration of 50 ng/µL. The total volume was adjusted with DNase/RNase-free water.

Thermal cycling was carried out using a Thermal Cycler under the following conditions:

- Initial denaturation at 94°C for 5 minutes
- Followed by 35 cycles of:
  - Denaturation at 94°C for 30 seconds
  - Primer annealing at 57°C for 30 seconds
  - Extension at 72°C for 45 seconds
- Final extension at 72°C for 5 minutes

The PCR products were analyzed by electrophoresis on a 1.5% agarose gel stained with RedSafe dye. The gel was visualized using a gel documentation system. The PCR products yielded bands of 350 bp for the outer band, 226 bp for the G allele, and 173 bp for the A allele. The presence of both allele-specific bands indicates a heterozygous GA genotype, while the presence of only one band corresponds to a homozygous genotype for either allele.

3. RESULTS

3.1 Genotyping and PCR Amplification of IL-17 SNP rs3819024 Visualized by Agarose Gel Electrophoresis

The present study focused on the Interleukin-17 gene polymorphism, specifically the single nucleotide polymorphism (SNP rs3819024). This genetic variant was investigated using the Tetra-ARMS PCR technique to differentiate between the A and G alleles. During PCR amplification, an annealing temperature of 59°C was used to ensure specific primer binding. The expected amplicon sizes for each allele were distinct and easily identifiable by gel electrophoresis: the A allele produced a band of 249 base pairs, while the G allele produced a band of 321 base pairs. Additionally, a 524 base pair fragment amplified by the two outer primers served as a control band to confirm successful amplification. This setup allowed for accurate genotyping of individuals as homozygous AA, homozygous GG, or heterozygous AG based on the presence of one or both allele-specific bands along with the outer product band.

Image 1 demonstrated the genotyping outcomes obtained through 1% agarose gel electrophoresis following PCR amplification using the Tetra-ARMS (Amplification Refractory Mutation System) technique, targeting the Interleukin-17 (IL-17) gene, specifically the SNP rs3819024. To accurately estimate the fragment sizes, a DNA ladder with 100 base pair increments was employed as a molecular size marker. The analysis shows three distinct banding patterns, each corresponding to a different genotype. A control band of approximately 524 base pairs, produced by the outer primers, appears in all samples and serves as an internal control to validate the success of the PCR reaction. Samples showing three bands—the control band along with a 249 bp band (specific to the A allele) and a 321 bp band (specific to the G allele)—are interpreted as heterozygous (AG). In contrast, samples with only the control band and the 249 bp A-specific band are considered homozygous for the A allele (AA), while those showing the control band along with the 321 bp G-specific band are identified as homozygous for the G allele (GG). These gel patterns were derived from DNA samples of leukemia patients, and the genotyping results contribute to investigating the potential relationship between IL-17 polymorphisms and leukemia susceptibility, in combination with the patients' clinical and physiological data.

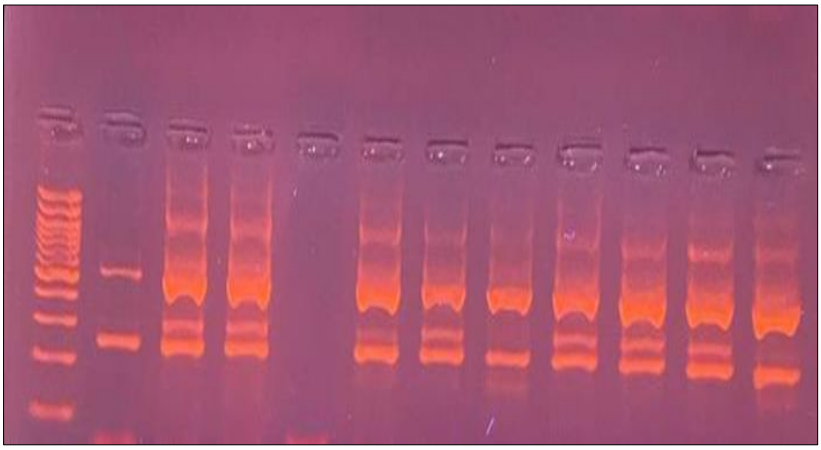

Image 1: Genotyping of IL-17 SNP rs3819024 by Tetra-ARMS PCR and Agarose Gel Electrophoresis

Image 2 showed the result of polymerase chain reaction (PCR) analysis followed by separation of the products using agarose gel electrophoresis. It illustrates the gene amplification outcomes, likely for the IL-17 gene or another immune-related gene, based on the observed banding patterns.

At the top of the gel, a molecular weight marker (DNA ladder) is visible, containing several graduated bands used as a reference to estimate the size of the PCR products in base pairs. Comparing the bands in the other wells to the ladder, most samples display a single clear and well-defined band, indicating successful and specific gene amplification without secondary products or contamination.

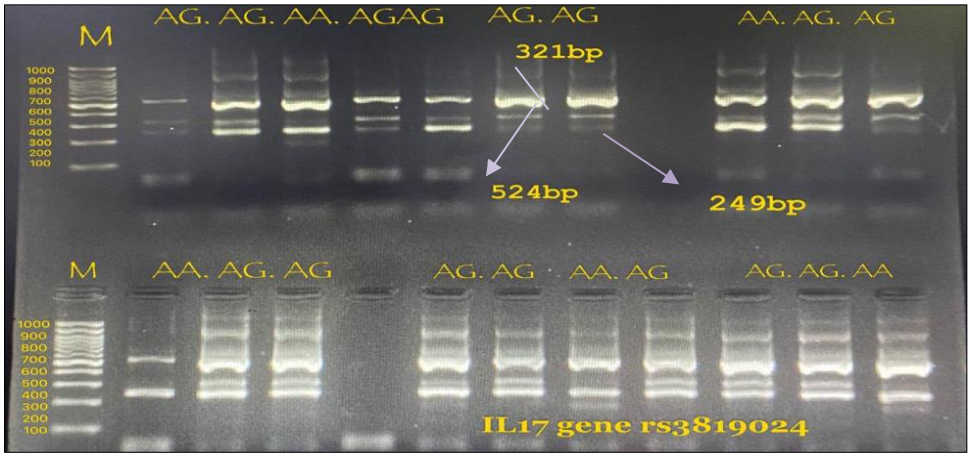

Image 2: Agarose Gel Electrophoresis Image Showing IL-17 Gene Polymorphism in Leukemia Patients Using PCR Technique

Image 3 showed the results of PCR amplification visualized by agarose gel electrophoresis stained with a fluorescent dye under UV light. A DNA ladder is loaded in the first lane for size estimation. Each lane displays a single, sharp band of uniform size, indicating successful and specific amplification of the target gene across all samples. The absence of smearing or additional bands suggests high reaction specificity with no contamination or non-specific products. This confirms the efficiency and reliability of the PCR protocol applied."

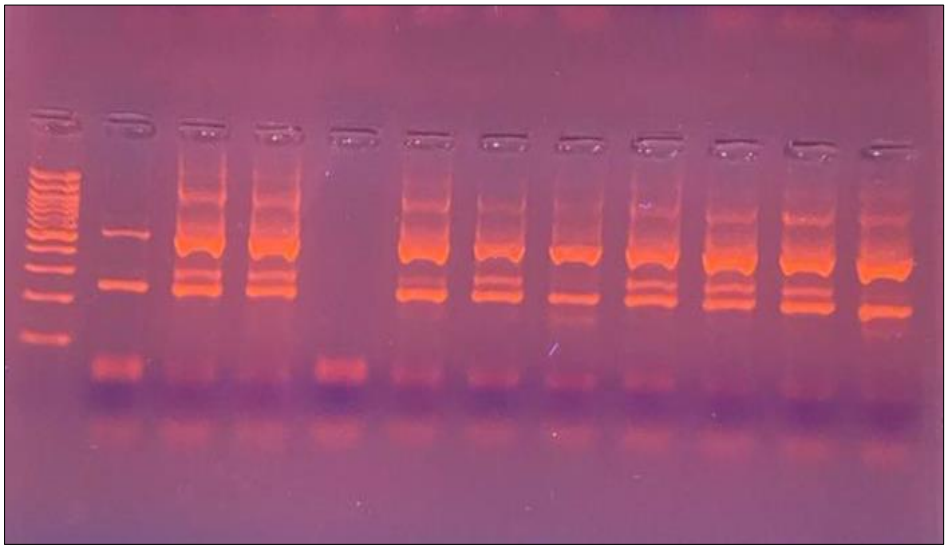

Image 3: Gel Electrophoresis Results of IL-17 Gene Amplification by PCR"

3.2 IRF8 Expression Differences by IL-17 rs3819024 Genotype in Patients and Controls

Table 2 showed leukemia patients, IRF8 protein levels were analyzed based on IL-17 rs3819024 genotypes (AA vs. AG). The results show that individuals with the AG genotype had a higher mean IRF8 level (42.24 ng/mL) compared to those with the AA genotype (39.51 ng/mL). The median values also support this trend (41.36 for AG vs. 39.95 for AA). However, the AG group demonstrated greater variability, with a standard deviation of 11.60 compared to 8.103 in the AA group, and a wider range (65.83 vs. 34.90). The coefficient of variation further emphasizes this difference in consistency, being 27.45% in AG compared to 20.51% in AA, suggesting a more heterogeneous expression of IRF8 among AG

individuals. The 95% confidence intervals for the mean values overlap (AA: 36.16–42.85; AG: 38.26–46.23), indicating that while the mean is higher in AG carriers, the difference may not be statistically significant without further inferential testing. Overall, the data suggest a possible genotype-related modulation of IRF8 expression in leukemia patients, with a tendency toward elevated levels in those carrying the G allele (Figure 1).

Table 2: IRF8 Levels in Leukemia Patients by IL-17 rs3819024 Genotype

IRF 8 IN patients	AA	AG
Number of values	25	35
Minimum	19.74	21.40
25% Percentile	35.27	38.04
Median	39.95	41.36
75% Percentile	42.47	47.08
Maximum	54.64	87.23
Range	34.90	65.83
Mean	39.51	42.24
Std. Deviation	8.103	11.60
Std. Error of Mean	1.621	1.960
Lower 95% CI of mean	36.16	38.26
Upper 95% CI of mean	42.85	46.23
Coefficient of variation	20.51%	27.45%

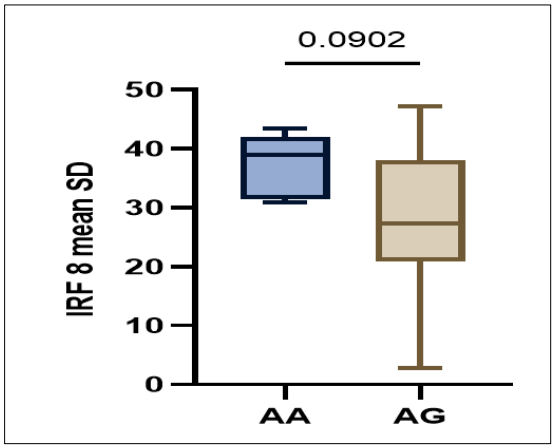

Figure 1: Comparison of Mean IRF8 Levels in Leukemia Patients with AA and AG Genotypes of IL-17 rs3819024

Table 3 demonstrated healthy control group, IRF8 levels varied significantly between individuals with the AA and AG genotypes of the IL-17 rs3819024 polymorphism. Individuals with the AA genotype showed a notably higher mean IRF8 concentration (37.10 ng/mL) compared to those with the AG genotype (27.70 ng/mL). The median value also reflects this trend (38.86 vs. 27.37, respectively). Additionally, the AG group displayed substantially higher variability, with a standard deviation of 11.60 and a coefficient of variation of 41.87%, compared to just 14.72% in the AA group. The wide range observed in AG individuals (from 2.851 to 47.16) (Figure 2) suggests a heterogeneous expression pattern of IRF8 among G allele carriers. Although the sample size for the AA group is small (n=5), the data indicate a potential genotype-dependent downregulation of IRF8 in AG individuals, which may play a role in immune regulation in non-diseased individuals. This finding contrasts with the pattern seen in leukemia patients and may reflect distinct biological effects of the IL-17 polymorphism under healthy versus pathological conditions.

Table 3: IRF8 Levels in Healthy Samples by IL-17 rs3819024 Genotype

IRF 8 IN CONTROL	**AA**	**AG**
Number of values	5	25
Minimum	30.81	2.851
25% Percentile	31.41	20.87
Median	38.86	27.37
75% Percentile	41.92	38.11
Maximum	43.37	47.16
Range	12.56	44.31
Mean	37.10	27.70

IRF 8 IN CONTROL	AA	AG
Std. Deviation	5.463	11.60
Std. Error of Mean	2.443	2.320
Lower 95% CI of mean	30.32	22.91
Upper 95% CI of mean	43.89	32.49
Coefficient of variation	14.72%	41.87%

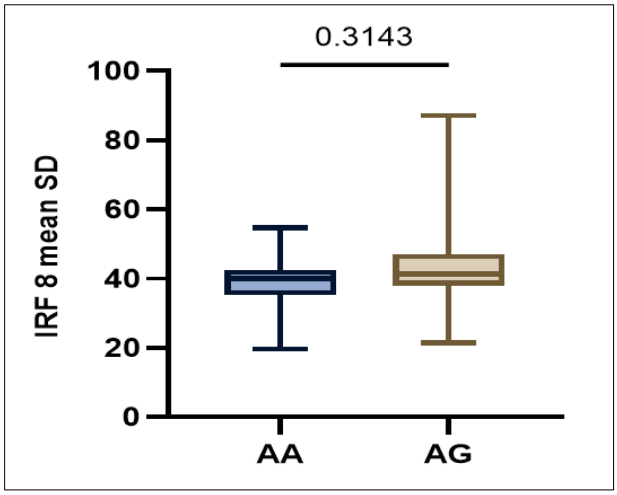

Figure 2: Comparison of Mean IRF8 Levels in Healthy Controls with AA and AG Genotypes of IL-17 rs3819024

3.3 TGF Expression Differences by IL-17 rs3819024 Genotype among Leukemia Patients and Controls

In Table 4 leukemia patients, TGF levels were assessed based on IL-17 rs3819024 genotypes (AA vs. AG). The analysis revealed that individuals with the AG genotype had a slightly higher mean TGF level (45.66 ng/mL) compared to those with the AA genotype (43.37 ng/mL). The median values also followed this trend (45.27 for AG vs. 41.48 for AA), indicating that AG individuals generally exhibited elevated TGF levels. Despite these differences in central tendency, both groups showed considerable overlap in their interquartile ranges and overall distributions. The AG group exhibited a slightly higher variability, with a standard deviation of 13.29 and a coefficient of variation of 29.11%, compared to 11.92 and 27.48% in the AA group, respectively. The 95% confidence intervals for the mean TGF levels overlapped as well (AA: 38.45–48.29; AG: 41.10–50.23) (Figure 3), suggesting that the observed differences may not be statistically significant without further testing. Nonetheless, these findings suggest a potential trend toward increased TGF expression in AG genotype carriers, which could reflect genotype-related modulation of inflammatory or immunosuppressive pathways in leukemia.

Table 4: TGF Levels in Leukemia Patients by IL-17 rs3819024 Genotype

TGF IN PATIENTS	AA	AG
Number of values	25	35
Minimum	27.35	19.27
25% Percentile	33.18	35.17
Median	41.48	45.27
75% Percentile	55.00	54.19
Maximum	69.28	69.23
Range	41.93	49.96
Mean	43.37	45.66
Std. Deviation	11.92	13.29
Std. Error of Mean	2.384	2.247
Lower 95% CI of mean	38.45	41.10
Upper 95% CI of mean	48.29	50.23
Coefficient of variation	27.48%	29.11%

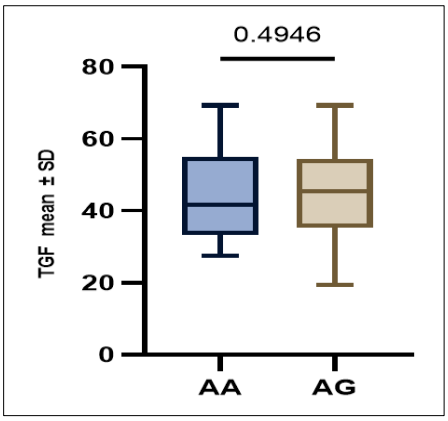

Figure 3: Comparison of Mean TGF Levels in Leukemia Patients with AA and AG Genotypes of IL-17 rs3819024

In Table 5 the healthy control group, TGF levels varied markedly between individuals with the AA and AG genotypes of the IL-17 rs3819024 polymorphism. Participants with the AG genotype had a substantially higher mean TGF level (32.88 ng/mL) compared to those with the AA genotype (15.29 ng/mL), more than doubling the average concentration. This trend is reflected in both the median values (34.83 for AG vs. 14.90 for AA) and the interquartile range, suggesting a consistently elevated TGF profile among AG carriers. Additionally, the AG group displayed a much broader range (60.16 ng/mL) and higher variability, with a standard deviation of 17.07 and a coefficient of variation of 51.91%, compared to 41.19% in the AA group. The wide 95% confidence interval for AG individuals (25.83–39.92) (Figure 4) further emphasizes this heterogeneity. Despite the small sample size in the AA group (n = 5), the data suggest a potential genotype-dependent upregulation of TGF in healthy individuals carrying the G allele, which may play a role in immune tolerance or inflammation regulation. These observations highlight a contrast with patterns observed in leukemia patients and warrant further investigation into the biological implications of the IL-17 polymorphism in immune homeostasis.

Table 5: TGF Levels in Healthy Controls by IL-17 rs3819024 Genotype

TGF CONTROL	AA	AG
Number of values	5	25
Minimum	8.396	5.648
25% Percentile	9.888	16.36
Median	14.90	34.83
75% Percentile	20.88	46.34
Maximum	24.95	65.80
Range	16.56	60.16
Mean	15.29	32.88
Std. Deviation	6.298	17.07
Std. Error of Mean	2.816	3.413
Lower 95% CI of mean	7.469	25.83
Upper 95% CI of mean	23.11	39.92
Coefficient of variation	41.19%	51.91%

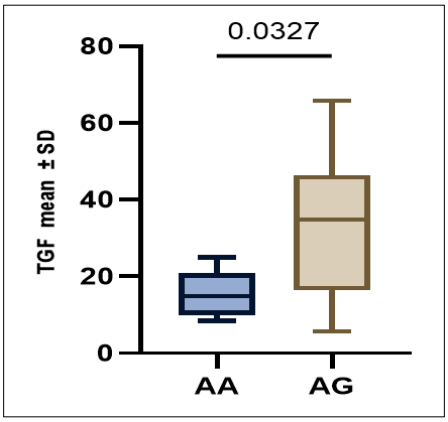

Figure 4: Comparison of Mean TGF Levels in Healthy Controls with AA and AG Genotypes of IL-17 rs3819024

Table 6 presented the distribution of IL-17 rs3819024 alleles (A and G) among leukemia patients and healthy controls. The A allele was more prevalent in both groups, serving as the reference. However, the G allele appeared significantly more frequently in the healthy control group (25 out of 120 alleles) compared to the patient group (5 out of 60 alleles). The calculated odds ratio (OR) of 2.895, with a 95% confidence interval of 1.100 to 7.250 and a p-value of 0.0339, indicates that individuals carrying the G allele.

Table 6: Allele Frequencies of IL-17 rs3819024 in Leukemia Patients and Healthy Controls

Alleles	**Study participants**					
	Patients No.	**Healthy control No.**	**OR**	**95% CI**		**p-value**
				Lower	**Upper**	
A (reference)	55	95	1			
G	5	25	2.895	1.100	7.250	0.0339
Total	**60**	**120**				

Table 7 presented a comparison of genotype frequencies (AA vs. AG) of the IL-17 rs3819024 polymorphism between leukemia patients and healthy controls. The AA genotype was used as the reference. While both groups showed a presence of the AG genotype, it was notably more frequent among healthy controls (25 individuals) compared to patients (5 individuals). The calculated odds ratio (OR) was 3.571, with a 95% confidence interval ranging from 1.258 to 9.395 and a p-value of 0.0177, indicating a statistically significant association ($p < 0.05$). These results suggest that individuals with the AG genotype were approximately 3.6 times more likely to be healthy than to have leukemia, relative to those with the AA genotype. This finding implies a potential protective role of the heterozygous AG genotype against leukemia in the study population, possibly mediated through functional differences in IL-17 pathway activity.

Table 7: Genotype Frequencies of IL-17 rs3819024 in Leukemia Patients and Healthy Controls

Genotype	**Study population**					
	Patients No.	**Healthy control No.**	**OR**	**95% CI**		**p-value**
				Lower	**Upper**	
AA (reference)	25	35	1			
AG	5	25	3.571	1.258	9.395	0.0177

3.4 Correlation Between IRF8 and TGF-β Levels in Study Subjects

Figure 5 scatter plot demonstrated the relationship between IRF8 (Interferon Regulatory Factor 8) levels and TGF (Transforming Growth Factor) levels among the study subjects. Each blue dot represents an individual sample, showing the distribution of TGF relative to IRF8 expression. The calculated Pearson correlation coefficient (r = 0.1603) indicates a weak positive correlation, and the associated p-value (P = 0.2212) suggests that this relationship is not statistically significant. These findings imply that there is no meaningful or strong association between IRF8 and TGF levels within the analyzed cohort.

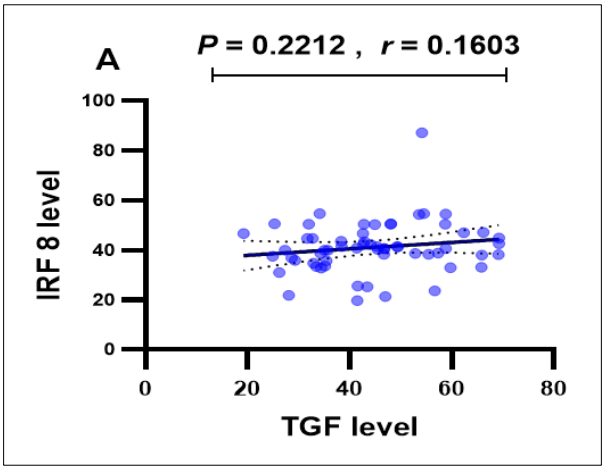

Figure 5: IRF8 vs. TGF-β Levels Correlation

4. DISCUSSION

These findings parallel observations in other IL-17 gene polymorphism studies. For instance, Elsissy *et al*., (2019) reported that IL-17F rs763780 did not predispose to acute myeloid leukemia (AML) in Egyptians, but suggested that certain polymorphisms might even be protective. Similarly, Alnagar *et al*., (2020) found that although IL-17A polymorphism had

limited prognostic value, elevated serum IL-17 correlated with poor treatment response in AML patients, implying functional effects of cytokine deregulation even in the absence of genotype significance.

By contrast, Wróbel *et al*., (2014), Ismail, (2021) and Zayed (2020) indicated that IL-17F variants were associated with AML susceptibility in Polish and Egyptian populations, respectively. These conflicting data likely reflect ethnic and genetic heterogeneity, reinforcing the need to investigate alternative variants like rs3819024, which may operate via distinct mechanisms.

In healthy controls, AG genotype carriers showed significantly lower IRF8 but markedly elevated TGF-β levels compared to AA carriers, suggesting a genotype-specific baseline anti-inflammatory phenotype. The pronounced coefficient of variation in TGF-β among AG carriers (~52%) highlights substantial inter-individual heterogeneity. Given TGF-β's role in immune tolerance and tumor suppression, this profile may underlie a protective genotype effect against leukemogenesis, consistent with Mayer *et al.,* (2021) and Mikkola, (2022)'s findings of IL-17 gene variants being potentially protective.

The allelic and genotypic frequency comparisons further support this view. The G allele and AG genotype were significantly more prevalent among healthy controls, yielding odds ratios (~2.9 and ~3.6, respectively) indicative of reduced leukemia risk. These associations contrast with Elsissy et al.'s lack of risk association for IL-17F, but align with Wróbel et al.'s report that IL-17F G variant conferred AML susceptibility in other cohorts. This dichotomy underscores allele-specific effects, where rs3819024 G might confer immunomodulatory advantage in our population, while other SNPs exert different or opposite influences.

Notably, no strong correlation was found between IRF8 and TGF-β in our study ($r = 0.16$, $p = 0.22$), implying these markers may operate independently in their response to IL-17 genotype variation. This independence echoes results from other cytokine studies where multiple immune mediators act via distinct pathways (e.g. IFN-γ and IL-10 polymorphisms, as reviewed by Pravica *et al*., 2000 and Tripathi *et al*., 2022).

Together, our findings suggest that the rs3819024 G allele may play a dual role: enhancing TGF-β expression while dampening IRF8 in healthy states—thereby promoting immune homeostasis—and potentially contributing to elevated IRF8/TGF-β in leukemia, amplifying disease-related immune dysregulation.

5. CONCLUSION

The research examined how IL-17 gene polymorphism (SNP rs3819024) affects leukemia by analyzing its genetic patterns and its impact on IRF8 and TGF-β expression levels. The Tetra-ARMS PCR method enabled researchers to detect AA, AG and GG genotypes while showing that AG genotype and G allele appeared more often in healthy controls. The statistical results demonstrated a significant relationship which indicates the AG genotype might protect against leukemia.

The leukemia patients who carried the AG genotype displayed elevated IRF8 and TGF-β levels when compared to AA genotype carriers. The AG genotype in healthy individuals resulted in decreased IRF8 expression while TGF-β levels increased substantially indicating different immune response patterns based on genotype. The relationship between IRF8 and TGF-β expression levels was weak and failed to reach statistical significance.

The research demonstrates IL-17 rs3819024 polymorphism plays a role in leukemia development and immune system control which requires additional investigation to understand its disease and health-related functional effects. The research needs to confirm these findings through studies involving bigger groups of people from different ethnic backgrounds while investigating rs3819024's functional effects through gene expression analysis and cytokine production tests and cell signaling experiments. Our research reveals new understanding about IL 17 variation effects on phenotype while establishing its dual potential as both a leukemia susceptibility marker and immune therapy target.

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