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Association of IKZF1 Polymorphisms rs4132601 and rs11978267 with Non-Hodgkin Lymphoma: A Case-Control Study

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Abstract

Background: Non-Hodgkin Lymphoma (NHL) is a malignancy with a significant genetic component. This study aimed to evaluate the association between the IKZF1 rs4132601 T>G and rs11978267 A>G polymorphisms and NHL risk in a Southeast Iranian population.

***Method*:** This case-control study included 195 NHL patients and 200 age- and sex-matched healthy controls. Genotyping was performed using the polymerase chain reaction-restriction fragment length polymorphism (PCR-RFLP) method. Odds ratios (ORs) and 95% confidence intervals (CIs) were calculated, and haplotype analysis was also conducted.

***Results*:** The results showed that the rs4132601variant reduced the risk of NHL in codominant and recessive (OR = 0.384, 95% CI = 0.205–0.721, p =0.003 and OR= 0.417, 95% CI = 0.235–0.740, P = 0.002, respectively). The G allele was associated with a significantly reduced risk of NHL (OR = 0.678, 95% CI = 0.509–0.902; p = 0.007). Conversely, the rs11978267 variant increased the risk of NHL in codominant (OR= 1.806, 95% CI =1.196–2.728, p=0.005, AG vs AA; and OR= 3.287, 95%CI = 1.119–9.653, p=0.030, GG vs AA) and dominant (OR= 1.906, 95% CI= 1.276- 2.849, P= 0.001) and allelic (OR= 1.699, 95% CI= 1.223- 2.360, P= 0.001 G vs A) models.

***Conclusion*:** Our findings suggest that IKZF1 polymorphisms significantly influence NHL susceptibility.

Keywords: Alleles, Genotype, Haplotype, Non-Hodgkin Lymphoma, Single Nucleotide Polymorphism.

Introduction

Non-Hodgkin Lymphoma (NHL) represents a heterogeneous group of malignancies originating from lymphocytes, constituting a major global health burden with an estimated 544,000 new cases and 260,000 deaths annually(1). The etiology of NHL is complex and multifactorial, involving environmental exposures, infectious agents, immune suppression, and a significant heritable genetic component(2, 3). Over the past decade, Genome-Wide Association Studies (GWAS) have substantially advanced our understanding, identifying numerous susceptibility loci that underscore the polygenic nature of NHL risk(4, 5). A pivotal gene located within several GWAS-identified loci is IKAROS family zinc finger 1 (IKZF1) (NM_006060.6) on chromosome 7p12.2. IKZF1 encodes a master transcription factor that is indispensable for hematopoietic stem cell maintenance and differentiation, particularly in the lymphoid lineage(6, 7). It

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functions as a tumor suppressor by regulating cell cycle exit, chromatin remodeling, and cytokine receptor signaling during B-cell and T-cell development(8, 9). Somatic alterations of IKZF1, such as deletions and dominant-negative mutations, are well-established drivers of high-risk acute lymphoblastic leukemia (ALL)(10, 11). Beyond somatic mutations, common germline variation in IKZF1 is a key area of investigation. Specific single-nucleotide polymorphisms (SNPs) can modulate disease risk by acting as expression quantitative trait loci (eQTLs), influencing IKZF1 expression levels and potentially altering lymphocyte development and immune surveillance(12, 13). Two such polymorphisms, rs4132601 and rs11978267, have been extensively studied in ALL, with the rs4132601 G allele consistently identified as a risk factor across diverse populations(14, 15). More recent research has explored their roles in other hematological conditions and autoimmune diseases, suggesting broader immunogenetic pleiotropy(16, 17). However, the association of these IKZF1 polymorphisms with NHL risk remains less clear, with limited and sometimes conflicting data from different ethnic groups(18, 19). Given the fundamental role of IKZF1 in lymphoid homeostasis and the shared cellular origins of leukemias and lymphomas, it is plausible that germline variants perturbing its function could also modulate NHL susceptibility(20, 21). There are some investigations about gene polymorphisms and gene expression in Lymphoma(22-24), but to the best of our knowledge, there has yet been no study investigating the effect of *IKZF1* variants on NHL risk in the Iranian population. Therefore, this case-control study was designed to evaluate the association of IKZF1 rs4132601 T>G and rs11978267 A>G polymorphisms, both individually and in haplotype combinations, with the risk of NHL in a cohort from Southeast Iran.

Materials and Methods

Patients

This case- control study has been prepared on 195 Non- Hodgkin Lymphoma patients and 200 age and sex matched healthy subjects in Zahedan, Southeast Iran. The study design and enrolment process have been defined previously(25-27). The local ethics committee of Zahedan University of Medical Sciences approved the project (IR.ZAUMS.REC.1404.135), and informed consent was obtained from cases and controls. DNA was extracted from peripheral whole blood using the salting-out method as described previously(28).

Genotyping

Genotyping of IKZF1 rs4132601and rs11978267 polymorphisms was done using PCR-RFLP. The primers were synthesized by SinaClon, Iran and scheduled in Table 1. PCR-RFLP method was utilized to genotype IKZF1 variants. In each 0.20-ml PCR reaction tube, 1 µl of genomic DNA (~100 ng/ml), 1 µl of each primer, 10 µl of 2× Prime Taq Premix (Genet Bio, Korea), and 7 µl of ddH2O were added. The PCR conditions for all SNPs were set as follows: 95 °C for 5 min, 30 cycles of 95 °C for 30 s, 60 °C for 30 s, 72 °C for 30 s and a final extension step of 72 °C for 10 min. Then, 10 µl of PCR product is digested with the proper restriction enzyme (Table 1) and electrophoresed onto 2.5% agarose gels containing 0.5 µg/ml ethidium bromide and detected under UV light (Figs. 1and 2).

Table 1. The primers used for detection of *IKZF1* polymorphisms using PCR-RFLP methods.

***IKZF1* polymorphisms**	**Primer sequence (5′->3′)**	**Restriction enzyme**	**Fragment (bp)**
rs4132601 T >G	F: AACCAATCAAATGTCTGGGAAGC R: TGGCTATAAAAGGCAGTGAGAGG	*MboI*	T allele: 321 + 280; G allele: 321 + 223 + 57
rs11978267 A>G	F: GATGGGAAGGAATTATCCATGCAATGAC R: TCAAAGCACATGAGCCCATCC	*CseI*	A allele: 208; G allele: 169+ 38

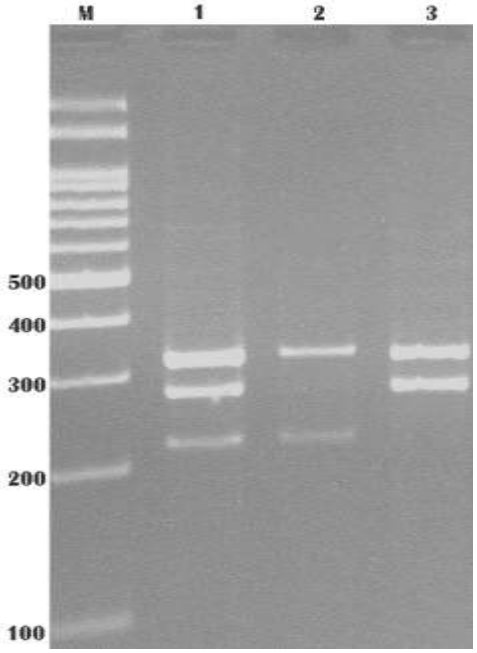

Fig. 1. Photograph of *IKZF1* rs4132601 polymorphism. G allele was digested by the *MboI* restriction enzyme and produced 321, 223 and 57 bp fragments, while the T allele was undigested (321 and 280 bp fragments). M: DNA marker; Lanes 1: TG; Lane 2: GG; Lanes 3: TT.

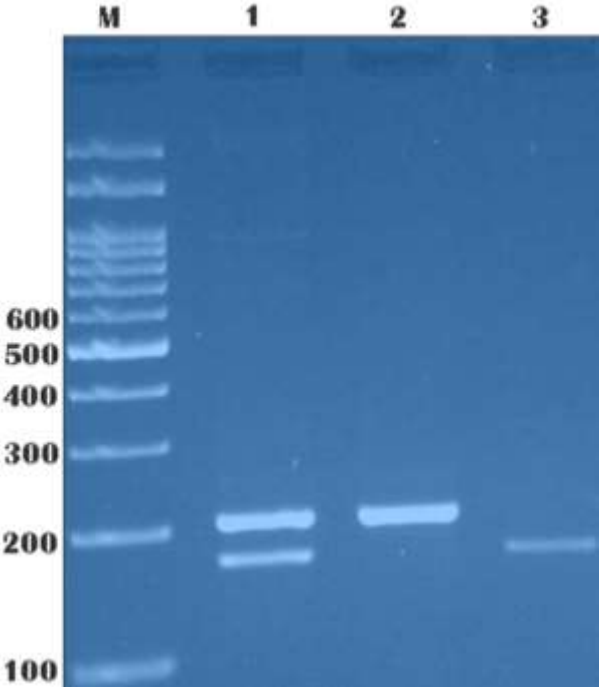

Fig. 2. Photograph of *IKZF1* rs11978267 polymorphism. G allele was digested by the *CseI* restriction enzyme and produced 169 and 57 bp fragments, while the A allele was undigested (208 bp). M: DNA marker; Lane 1: AG; Lane 2: AA; Lane 3: GG.

Statistical analysis

All statistical computations were carried out using IBM SPSS Statistics for Windows, Version 22.0. Descriptive statistics are presented as means ± standard deviations for continuous data and frequencies for categorical data. Inter-group differences for continuous variables were analyzed using independent samples t-tests, and associations between categorical variables were examined with Pearson's chi-square test. The strength of the

association between specific genotypes and non-Hodgkin lymphoma (NHL) was estimated using logistic regression, with findings reported as odds ratios (OR) and 95% confidence intervals (95% CI). A two-sided p-value of less than 0.05 was considered statistically significant.

Results

Our study involved 195 NHL patients (124 male,71 female; age 45.49 ± 15.12 years) and 200 healthy subjects (113 male, 87 Female; age 44.60 ± 23.95 years). The study groups were well-matched for the demographic variables of age and sex (P= 0.182 and 0.661, respectively).

The genotype and allelic frequencies of IKZF1 gene polymorphisms in patients and controls are shown in Table 2. The finding showed that rs4132601 T>G polymorphism decreased the risk of NHL in the codominant (OR = 0.384, 95% CI = 0.205–0.721, p =0.003, GG vs TT) and recessive (OR= 0.417, 95% CI = 0.235–0.740, P = 0.002, GG vs TT+ TG) inheritance models. The rs4132601 G allele decreased the risk of NHL (OR = 0.678, 95% CI = 0.509 –0.902; p =0.007) compared with the T allele. Concerning rs11978267 A>G polymorphism, our finding showed that this polymorphism increased the risk of NHL in codominant (OR= 1.806, 95% CI =1.196–2.728, p=0.005, AG vs AA; and OR= 3.287, 95%CI = 1.119–9.653, p=0.030, GG vs AA), dominant (OR= 1.906, 95% CI= 1.276-2.849, P= 0.001) inheritance models. G allele in rs11978267 increased the risk of NHL (OR= 1.699, 95% CI= 1.223- 2.360, P= 0.001) compared with A allele.

Table 2. Genotypes and Allele frequencies of IKZF1 gene polymorphisms in NHL patients and Healthy subjects

***IKZF1* polymorphism**	**Case n (%)**	**Control n (%)**	**OR (95% CI)**	**P-value**
rs4132601 T >G				
Codominant				
TT	75 (38.5)	62 (31.0)	1	--
TG	100 (51.3)	95 (47.5)	0.870 (0.561- 1.349)	0.534
GG	20 (10.2)	43 (21.5)	0.384 (0.205- 0.721)	**0.003**
Dominant				
TT	75 (38.5)	62 (31.0)	1	--
TG+GG	120 (61.5)	138 (69.0)	0.719 (0.474- 1.089)	0.119
Recessive				
TT+TG	175 (89.8)	157 (78.5)	1	-
GG	20 (10.2)	43 (21.5)	0.417 (0.235- 0.740)	**0.002**
Allele				
T	250 (64.1)	219 (54.8)	1	-
G	140 (35.9)	181 (45.2)	0.678 (0.509- 0.902)	**0.007**
rs11978267 A>G				
Codominant				
AA	92 (47.2)	126 (63.0)	1	-
AG	91 (46.7)	69 (34.5)	1.806 (1.196- 2.728)	**0.005**
GG	12 (6.1)	5 (2.5)	3.287 (1.119- 9.653)	**0.030**
Dominant				
AA	92 (47.2)	126 (63.0)	1	-
AG+ GG	103 (52.8)	74 (37.0)	1.906 (1.276- 2.849)	**0.001**
Recessive				
AA+AG	183 (93.9)	195 (97.5)	1	--
GG	12 (6.1)	5 (2.5)	2.557 (0.884- 7.401)	0.074
Allele				
A	275 (70.5)	321 (80.2)	1	--
G	115 (29.5)	79 (19.8)	1.699 (1.223- 2.360)	**0.001**

OR: Odds Ratio, **CI:** Confidence Interval, **Significant p-values** are shown in **bold** ($p < 0.05$).

A haplotype analysis was performed, and the finding showed that GA significantly decreased the risk of NHL compared with TA (Table 3). The association between the two IKZF1 polymorphisms (rs4132601 and rs11978267) and various clinicopathological characteristics of NHL patients summarizes on the Table 4, including age, histological subtype, disease stage, treatment regimen, radiotherapy status, and recurrence type. The distribution of genotypes across these clinical subgroups did not reveal any statistically significant associations (all p-values > 0.05). The distribution of the rs4132601 and rs11978267 genotypes showed no significant association with clinical parameters, including patient age (≤50 vs. >50 years; p = 0.740 and p = 0.539, respectively), lymphoma subtype (DLBCL vs. others), disease stage, treatment regimen (R-CHOP vs. others), or relapse status. These findings suggest that these polymorphisms are unlikely to be determinants of disease progression or treatment response.

Table 3. Haplotype association of *IKZF1* polymorphisms with NHL risk.

rs4132601	rs11978267	Controls (frequency)	NHL (frequency)	OR (95% CI)	P- value
T	A	0.4116	0.4535	1	---
G	A	0.3909	0.2516	0.59 (0.40- 0.86)	**0.0069**
T	G	0.1359	0.1875	1.49 (0.89- 2.50)	0.13
G	G	0.0616	0.1073	1.55 (0.79- 3.05	0.21

OR: Odds Ratio, **CI:** Confidence Interval, **Significant p-values** are shown in **bold** ($p < 0.05$).

Table 4. Association of IKZF (rs4132601 and rs11978267) Polymorphisms with Clinicopathological Characteristics of NHL Patients.

Characteristic of Patients	rs4132601			P-value	rs11978267			P-value
Age	TT	TG	GG	**0.740**	AA	AG	GG	0.539
≤50	46	67	13		61	59	6	
>50	29	33	7		31	32	6	
Histology				**0.715**				0.796
DLBCL	36	50	8		42	46	6	
Others	39	50	12		50	45	6	
Stage				**0.369**				0.821
I	41	51	13		48	51	6	
II	12	11	3		15	10	1	
III	5	9	3		10	6	1	
IV	17	28	1		19	24	3	
Treatment				**0.698**				0.983
R-CHOP	40	50	12		48	48	6	
Others	35	50	8		44	43	6	
Radio Therapy				**0.989**				0.815
No	64	86	17		78	78	11	
Yes	11	14	3		14	13	1	
Kind				**0.302**				0.958
Primary	67	92	20		85	83	11	
Recurrent	8	8	0		7	8	1	

Discussion

This case-control study demonstrates a significant and contrasting association of two IKZF1 polymorphisms with the risk of Non-Hodgkin Lymphoma in Southeast Iranian people. We found that the rs4132601 G allele was protective, while the rs11978267 G allele was a risk factor. Furthermore, our haplotype analysis provided a more nuanced view, revealing that the specific combination of alleles across these two loci, the GA haplotype, conferred significant protection against NHL.

The protective association of the rs4132601 G allele is particularly intriguing because this same variant is a well-documented risk allele for childhood and adult B-cell precursor ALL(14, 23, 29, 30). This discrepancy highlights the disease-specific nature of genetic risk variants. The mechanism of rs4132601 is believed to involve altered transcriptional regulation, potentially affecting the binding of transcription factors and the expression of IKZF1 or neighboring genes in a cell-context-dependent manner(12, 30, 31). A plausible explanation is that this polymorphism exerts divergent effects on different stages of lymphocyte development; a genetic change that predisposes to the developmental arrest characteristic of ALL may, conversely, protect against the malignant transformation and proliferation of mature lymphocytes in NHL. This is supported by recent functional studies showing that IKZF1 modulates transcriptional programs and chromatin accessibility differently in progenitor versus mature B-cell populations(9, 32). Furthermore, population-specific genetic backgrounds and linkage disequilibrium patterns with other causal variants could also explain these divergent associations(30, 33). In contrast, the risk association of the rs11978267 G allele aligns with its proposed biological function. This allele has been linked to lower IKZF1 expression levels, and IKZF1 haploinsufficiency is known to disrupt normal B-cell differentiation, leading to aberrant proliferation and a pre-malignant state(17, 34). Our finding that both the heterozygous (AG) and homozygous (GG) states significantly increase NHL risk suggests a dose-dependent effect, further supporting its functional relevance. While direct evidence for this SNP in NHL is scarce, its association with other B-cell pathologies, including systemic lupus erythematosus and chronic lymphocytic leukemia (CLL), reinforces its role in immune dysregulation and B-cell malignancy(16, 35). The haplotype analysis provides critical additional insight. The significant protective effect of the GA haplotype (rs4132601 G - rs11978267 A) indicates that the protective effect of the rs4132601 G allele is most pronounced in the presence of the rs11978267 A allele. This epistatic interaction suggests that the net effect on NHL risk is determined by the combined genotype at both loci, rather than either SNP in isolation. This underscores the limitation of single-locus analyses and emphasizes the importance of considering haplotype structures and gene-gene interactions in genetic association studies(36, 37). The non-significant risk trends observed for the TG and GG haplotypes may be due to limited statistical power for these less frequent combinations. The strengths of our study include a well-matched case-control design, the use of a robust PCR-RFLP methodology consistent with our previous work(25-27) and the inclusion of haplotype analysis. However, several limitations must be acknowledged. First, the sample size, while sufficient to detect the reported strong genetic effects, limits the power for subgroup analyses by NHL histological subtypes, which are known to have distinct genetic etiologies(5, 38). Second, the single-population design may affect the generalizability of our findings, and replication in independent, ethnically diverse cohorts is necessary(39). Third, the lack of functional data, such as IKZF1 expression levels from patient samples, means the biological mechanisms remain inferred.

In conclusion, our data provide compelling evidence that germline variation in the IKZF1 gene affects susceptibility to NHL. We identified rs4132601 as a potential protective

factor and rs11978267 as a risk factor, with their combined haplotype providing further discriminatory power. These findings contribute to the growing understanding of the genetic architecture of NHL and highlight the IKZF1 pathway as a key area for future research(40, 41). Future studies should focus on large-scale, multi-ethnic validation and functional genomics to elucidate the precise mechanisms by which these polymorphisms and their haplotypes influence lymphomagenesis.

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Conflict of interest
The authors declare that there is no conflict of interest regarding the content of this article.

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Author's Contributions
Conceptualization, G.B. and M.T.; Data curation. and G.B.; Formal analysis, M.T. and G.B.; Funding acquisition, S.M.H. and G.B.; Investigation, H.D., M.T. and G.B.; Methodology, G.B., M.T. and S.M.H.; Project administration, G.B.; Writing original draft, H.D., G.B, and M.T; All authors have read and agreed to the published version of the manuscript.

Ethical approval
The study received ethical approval from the local Ethical Committee of Zahedan University of Medical Sciences (IR.ZAUMS.REC. 1404.135).

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