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Association of ZNF521 rs7236481 Polymorphism and Hematological Markers with Susceptibility to Wound and Burn Infections in Iraqi Patients

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Abstract

Chronic wound and burn infections are associated with persistent inflammation, impaired tissue healing, and increased susceptibility to microbial colonization. Genetic polymorphisms within regulatory genes may influence host inflammatory responses and tissue repair mechanisms. ZNF521 is a transcriptional regulatory gene involved in cellular differentiation, regeneration, and immune modulation. A case–control study was conducted in Baghdad, Iraq, including 50 patients with wound and burn infections and 50 healthy controls. Hematological and inflammatory parameters, including complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP), were evaluated. Genomic DNA was extracted from peripheral blood samples, and the *ZNF521* rs7236481 polymorphism was analyzed using conventional PCR and Sanger sequencing. Genotype distribution, dominant and recessive genetic models, and Hardy–Weinberg equilibrium (HWE) were statistically assessed. Hematological analysis revealed significantly elevated white blood cell (WBC) counts (12.92 ± 4.55 vs. 7.52 ± 0.89 $\times 10^3/\mu L$, $p < 0.001$) and neutrophil counts (8.04 ± 2.67 vs. 4.87 ± 0.82 $\times 10^3/\mu L$, $p < 0.001$) in patients compared with controls. ESR and CRP levels were also significantly increased in patients (60.41 ± 39.74 mm/hr and 63.19 ± 59.91 mg/L, respectively) compared with controls (14.65 ± 3.12 mm/hr and 2.15 ± 0.41 mg/L, respectively; $p < 0.001$), indicating a pronounced inflammatory response. No significant differences were observed in lymphocyte, platelet, or RBC counts. Genotyping analysis demonstrated that the TT genotype was more frequent in patients (52.1%) than in controls (30.0%), whereas the GG genotype was less frequent in patients (8.3%) than in controls (28.0%). A significant difference in genotype distribution was observed between groups ($P = 0.02$). In the dominant model (GT+TT vs. GG), variant carriers exhibited increased susceptibility to wound and burn infections (OR = 4.28, $P = 0.01$). Similarly, the recessive model (TT vs. GG+GT) demonstrated a significant association with disease susceptibility (OR = 2.54, $P = 0.03$). HWE analysis showed no significant deviation in controls, whereas deviation was observed in patients. The *ZNF521* rs7236481 polymorphism may represent a potential genetic susceptibility marker for wound and burn infections among Iraqi patients. Elevated WBC, neutrophil, ESR, and CRP levels further reflect the significant inflammatory response associated with these infections. Further studies

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with larger sample sizes and functional validation are recommended to clarify the biological role of this polymorphism in wound and burn pathogenesis.

Key words: *ZNF521*, rs7236481, polymorphism, burn infections, chronic wounds, genetic susceptibility

Introduction

Chronic wound and burn infections with long-term inflammation, slow tissue repair, microbial colonization, and systemic complications, they remain key clinical challenges on a global level. Burn and wound injuries disrupt the protection of the skin barrier by enabling bacterial invasion of tissues, along with stimulation of more complicated inflammatory responses which can complicate the normal healing process.

Gram-negative bacteria, specifically *Pseudomonas aeruginosa* and *Acinetobacter baumannii*, are some of the most notorious pathogens in the chronic wound and burn infection epidemic, and frequently are associated with adverse clinical consequences and prolongation of hospital stay [1,2]. Wound healing is a highly regulated biological process which involves overlapping phases of hemostasis, inflammation, proliferation, and tissue remodeling. Coordinated interactions between inflammatory mediators with fibroblasts and components of extracellular matrix as well as signaling pathways become necessary for these processes to take place. On several different levels, genetic variations affecting governing pathways may lead to differences in individuals' ability to repair damaged tissue or their susceptibility to becoming infected [3,4].

Hematological and inflammatory biomarkers such as CBC parameters, ESR, and CRP are frequently utilized to assess systemic inflammatory responses and infection burden in patients with wound and burns. Such markers may offer helpful clinical information on immune activation and tissue damage during healing [1].

Persistent inflammation and impaired tissue regeneration are influenced not only by environmental and microbial factors but also by host genetic variations that may affect immune responses and healing efficiency.

Single nucleotide polymorphisms (SNPs) represent one of the most prevalent types of genomic variation in humans, and are associated with changes in gene expression, protein function, and immune responses. A growing body of evidence indicates that SNPs on regulatory or intronic regions may influence transcriptional activity, RNA splicing, chromatin accessibility, and cellular signalling systems, contributing to disease susceptibility and variability in clinical outcome [5].

ZNF521 is a zinc finger transcriptional regulator gene implicated in stem-cell maintenance, cell differentiation, and transcriptional regulation. Previous studies have demonstrated the role of *ZNF521* in regulating mesenchymal and hematopoietic differentiation, tissue regeneration, and inflammatory responses, all of which are essential processes involved in wound healing and host defense against infection [6,7].

Although wound and burn injuries differ in their etiology, both conditions share common pathological features, including microbial colonization, persistent inflammation, tissue damage, and impaired healing.

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Therefore, they were investigated together to evaluate common genetic factors that may influence susceptibility to infection and healing outcomes.

Despite the recognized biological importance of *ZNF521*, limited information is available regarding the association between *ZNF521* genetic polymorphisms and susceptibility to wound and burn infections. To the best of our knowledge, no previous Iraqi study has investigated the relationship between the *ZNF521* rs7236481 polymorphism and susceptibility to wound and burn infections. Therefore, this study was conducted to evaluate the association of the *ZNF521* rs7236481 polymorphism with susceptibility to wound and burn infections and to assess its potential value as a genetic marker in Iraqi patients.

Materials and Methods

Study design and subjects

A case–control study was conducted between August 2025 and March 2026 in Baghdad, Iraq. The study included 50 patients with wound and burn infections and 50 healthy controls. Peripheral blood samples were collected from all participants for hematological and molecular analyses.

Inclusion criteria

Patients aged 25–55 years with clinically diagnosed wound or burn infections were included. Both male and female participants were enrolled.

Exclusion criteria

Patients with uncontrolled diabetes mellitus, autoimmune diseases, malignancies, immunodeficiency disorders, or those receiving immunosuppressive therapy were excluded.

Hematological and inflammatory analysis:

The complete blood count (CBC), erythrocyte sedimentation rate (ESR), and C-reactive protein (CRP) tests were performed for all participants. Venous blood samples were collected and analyzed using standard laboratory procedures. CBC parameters, including red blood cells (RBCs), white blood cells (WBC), hemoglobin concentration, and platelets (PLTs), were measured using an automated hematology analyzer (DxH 500, Beckman Coulter, USA) according to the manufacturer's instructions. ESR was measured using the Westergren method, whereas CRP levels were determined using a quantitative immunoturbidimetric assay according to the manufacturer's instructions.

Molecular study:

DNA extraction

Genomic DNA was extracted from EDTA-treated blood samples using the EasyPure® Blood Genomic DNA Kit (TransGen Biotech, China; Cat. No. EE121) according to the manufacturer's instructions. DNA purity and concentration were evaluated using a NanoDrop spectrophotometer by measuring absorbance at 260 and 280 nm. DNA samples with an A260/A280 ratio ranging from 1.8 to 2.0 were considered acceptable for downstream molecular analyses.

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Agarose gel electrophoresis

PCR products were analyzed on 1% agarose gel prepared with 1× TAE buffer and stained with RedSafe dye. The amplified *ZNF521* rs7236481 fragment produced a PCR product of 390 bp. DNA fragments were separated by agarose gel electrophoresis and visualized under ultraviolet illumination using a gel documentation system. A 100 bp DNA ladder was used as a molecular size marker.

PCR amplification of genomic regions of *ZNF521* (rs7236481)

PCR was performed in a final reaction volume of 25 µL. The *ZNF521* rs7236481 region was amplified using specific primers: Forward primer (5′-GTCTCAAATTCCTGACCTCAG-3′) and Reverse primer (5′-CAACCTTCAAATTCTGCTTGG-3′). The expected amplicon size was 390 bp. Amplification was carried out using a MultiGene OptiMax Thermal Cycler (Labnet International, USA), and the optimal annealing temperature was determined by gradient PCR. The reaction components and optimized PCR conditions are presented in Tables (1) and (2), respectively. Amplification products were subsequently analyzed by agarose gel electrophoresis.

Table (1): Components of reaction mixture for amplification

Component	Volume (µl)	Concentration
Master Mix (2x)	12.5	2x
Forward Primer (10 pmol / µl)	1.25	10 pm
Reverse Primer (10 pmol / µl)	1.25	10 pm
DNA Template (50 ng / µl)	3	150 ng
D.W.	7	-
Final volume	25	-

Table (2): PCR amplification program

Step	Temperature (˚C)	Time	No. of Cycles
Initial	95	5 min	1
Denaturation	94	1 min	
Annealing	60	1 min	37
Extension	72	45 sec	
Final Extension	72	5 min	1

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DNA sequencing and genotype analysis

PCR products from 100 selected samples (50 patients and 50 healthy controls) were subjected to Sanger sequencing (Macrogen, Korea). Sequence chromatograms were analyzed using BioEdit and MEGA 11 software. Sequence quality and chromatogram peak clarity were assessed before genotype determination. Both forward and reverse sequence reads were aligned to generate a consensus sequence for each sample. Genotypes were identified by comparison with reference sequences obtained from the NCBI GenBank database. The obtained sequences were aligned with the reference *ZNF521* sequence, and the identified variants were verified using the corresponding reference SNP ID (rs7236481).

Bioinformatics analysis

Sequence quality and chromatogram peak clarity were assessed using BioEdit and MEGA 11 software. Forward and reverse sequences were aligned to generate a consensus sequence for each sample. Variants were identified by comparison with the reference *ZNF521* sequence obtained from the NCBI GenBank database using BLAST and verified using the reference SNP ID (rs7236481). Genotypes were classified as homozygous wild-type, heterozygous, or homozygous mutant.

Statistical analysis

Statistical analysis was performed using IBM SPSS Statistics version 28. Data normality was assessed using the Shapiro–Wilk test. Continuous variables were expressed as mean ± standard deviation and compared between patients and controls using the independent samples t-test. Genotype and allele frequencies were compared using the Pearson Chi-square test. Odds ratios (ORs) with 95% confidence intervals (CIs) were calculated to estimate the association between the *ZNF521* rs7236481 polymorphism and susceptibility to chronic wound and burn infections. Hardy–Weinberg equilibrium (HWE) was assessed in the control group using the Chi-square test. A P-value of less than 0.05 was considered statistically significant.

Results and Discussion

Hematology Analysis:

Complete blood count (CBC) and Erythrocyte Sedimentation Rate (ESR) and C-Reactive protein (CRP)

In this study, approximately 50 patients with wound and burn infections and 50 healthy controls were evaluated using hematological and inflammatory biomarkers, including WBC, LYM, NEUT, PLT, RBC, ESR, and CRP. Significant increases were observed in WBC, NEUT, ESR, and CRP levels in patients compared with controls ($p < 0.001$). In contrast, LYM, PLT, and RBC showed no statistically significant differences between the two groups. The hematological and inflammatory parameters of the study groups are presented in Table 3.

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Table (3): Comparison of Hematological and Inflammatory Parameters between Patient and Control groups.

Hematological and Inflammatory Parameter	Patient Group (Mean ± SD)	Control Group (Mean ± SD)	Normal Reference Range	p-value
WBC (×10³/µL)	12.92 ± 4.55	7.52 ± 0.89	4–11 ×10³/µL	p < 0.001
LYM (×10³/µL)	1.75 ± 0.65	2.19 ± 0.43	1.50–3.50 ×10³/µL	NS
NEUT (×10³/µL)	8.04 ± 2.67	4.87 ± 0.82	2.5–7.5 ×10³/µL	p < 0.001
PLT (×10³/µL)	437.09 ± 128.28	295.40 ± 31.62	150–450 ×10³/µL	NS
RBC (×10⁶/µL)	3.68 ± 0.81	4.62 ± 0.34	3.80–5.80 ×10⁶/µL	NS
ESR (mm/hr)	60.41 ± 39.74	14.65 ± 3.12	0–20	p < 0.001
CRP (mg/L)	63.19 ± 59.91	2.15 ± 0.41	0–5	P < 0.001

Clinical parameters were evaluated in patients with wounds and burns and compared to those of a control group. The results demonstrated Significant differences were observed for WBC, NEUT, ESR, and CRP ($p < 0.001$), whereas LYM, RBC, and PLT showed no significant differences as shown in figure 1.

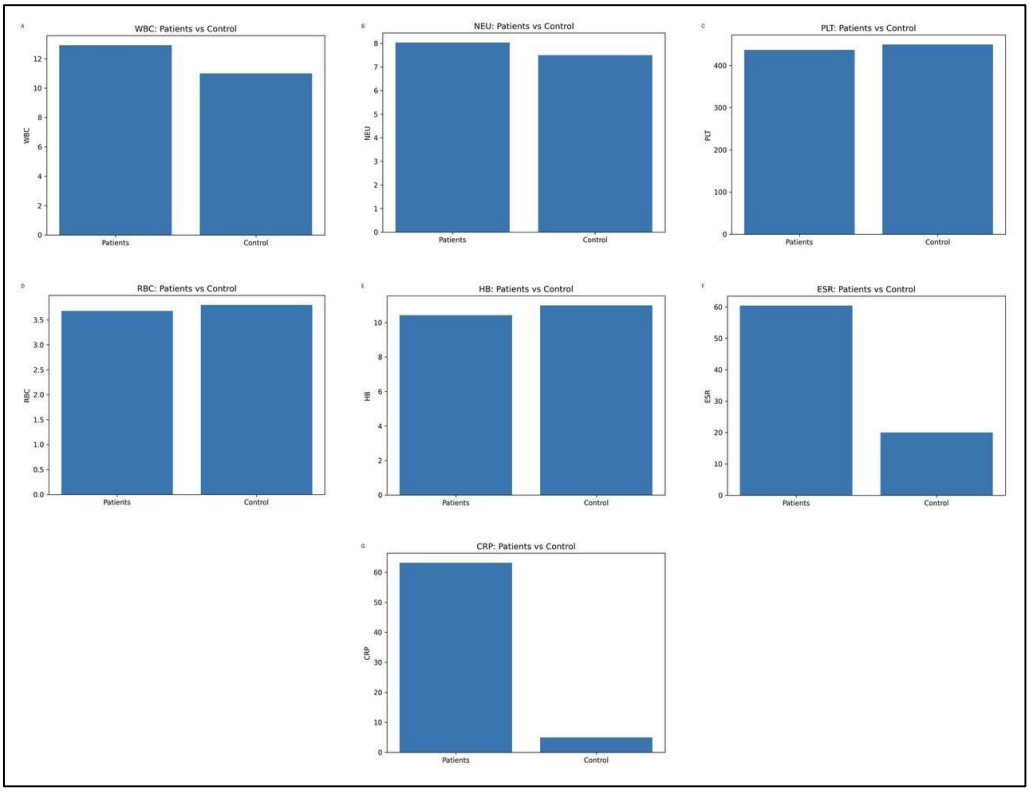

Figure 1: Comparison of hematological and inflammatory parameters between patients and healthy controls.

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The WBC count was markedly elevated in the patient group, reaching 12.92 × 10^3/µL, compared with 7.52× 10^3/µL in the control group. The LYM count was non significantly change in patients, measured at 1.75 ± 0.65 × 10^3/µL, while in the control group it was 2.19 ± 0.43 × 10^3/µL. Conversely, the NEUT count was highly increased in patients (8.04×10^3/µL) compared to controls 4.87 ± 0.82× 10^3/µL. The PLT level was non-significant change in the patient group, with a value of 437.09 × 10^3/µL, whereas the control group showed a value of 295.40 × 10^3/µL. The RBC level was non-significant change in the patient group, with a value of 3.68 × 10^6/µL, whereas the control group showed a value of 4.62 × 10^6/µL In addition, the ESR was elevated in patients 60.41 mm/hr) compared to controls (14.65 mm/hr). Similarly, the CRP was considerably higher in patients (63.19 mg/L) than in controls (2.15 mg/L).

These findings highlight the pronounced inflammatory and hematological responses observed in patients with wounds and burns. The findings of this study demonstrate that the inflammatory markers, particularly ESR, CRP, NEUT, and WBC, are significantly altered in patients compared to healthy individuals , while LYM, RBC and PLT are non-significantly changed .

Elevated WBC and neutrophil counts observed in burn patients indicate a strong systemic inflammatory and innate immune response following tissue injury. Neutrophils act as the first line of defense through phagocytosis, release of reactive oxygen species (ROS), antimicrobial enzymes, and formation of neutrophil extracellular traps (NETs), all of which contribute to pathogen clearance and inflammatory regulation during wound healing [8,9].

Lymphocytes also participate in later stages of tissue repair through cytokine production, immune regulation, and adaptive immune responses, supporting infection control and tissue regeneration [10,11]. These findings are consistent with previous studies reporting enhanced inflammatory and immune activation in burn injuries [12,13].

Platelet counts showed no statistically significant difference between patients and controls, despite their important role in hemostasis and tissue repair. Beyond coagulation, platelets release growth factors and inflammatory mediators that contribute to angiogenesis, immune-cell recruitment, and wound remodeling[14,15].

In contrast, RBC counts showed a non-significant reduction in patients compared with controls. The reduction of RBC may result from blood loss, inflammatory suppression of erythropoiesis, hemolysis, and nutritional deficiencies following injury. Although the reduction in RBC count was not statistically significant, lower RBC levels may contribute to impaired oxygen delivery to damaged tissues, potentially affecting tissue regeneration and wound healing [16].

Since RBCs contribute to oxygen transport and regulation of extracellular matrix remodeling, their reduction may negatively affect the healing process [17].

In this study, ESR and CRP levels were evaluated in patients with wound and burn infections. The results showed a significant increase in both markers ($p \leq 0.05$) compared with the control group.

The increased ESR and CRP in the current study may reflect a more pronounced inflammatory response toward wound and burn events. ESR is a nonspecific inflammatory marker that indicates tissue damage and ongoing inflammation [16]; its diagnostic specificity concerning infection is also limited on its own. A systemic inflammatory response associated with burn injury includes the upregulation of inflammatory cytokines and acute-phase proteins, including CRP. CRP is an acute-phase reactant produced

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by the liver in response to inflammation and tissue injury. Increased CRP levels were related to more severe burns and systemic inflammatory responses, and potential complications following injury [12].

The current study's alarming increase in CRP levels confirms its role as a major inflammatory marker in wound infection and burn. However, long-term elevated CRP may also be the result of burn-related inflammation, and not infection itself, so that CRP results need to be cautiously interpreted in a clinical setting [18].

Molecular study:

Analysis of *ZNF521* genetic polymorphism (rs7236481)

Conventional PCR amplification was successfully performed for *ZNF521* gene in DNA samples obtained from patients and controls. Agarose gel electrophoresis demonstrated clear and distinct amplified DNA bands corresponding to the expected target sizes, indicating successful amplification and good primer specificity.

For the *ZNF521* gene, electrophoretic analysis revealed a distinct amplification band with an approximate product size of 390 bp in the analyzed samples. The amplified fragments appeared as sharp and well-defined bands with minimal nonspecific amplification, confirming the efficiency of the PCR conditions and primer design as shown in Figure (2). DNA ladder markers were used to estimate the molecular size of the amplified products.

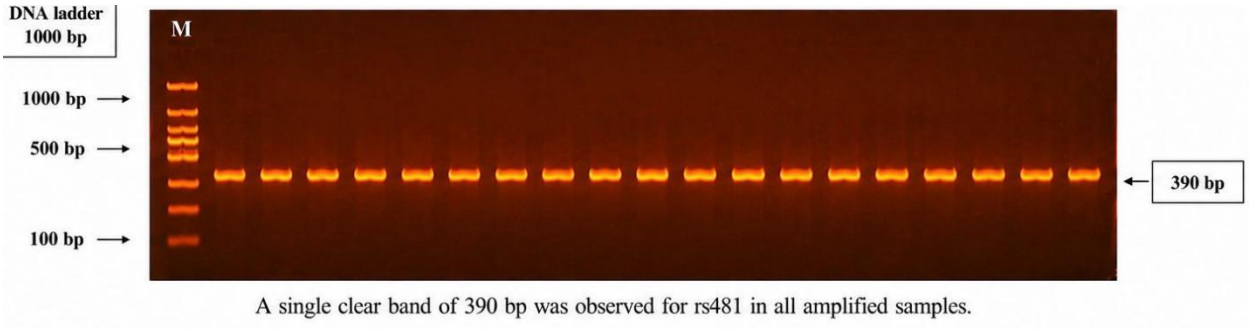

Figure (2): Agarose gel electrophoresis of PCR products amplified from the *ZNF521* rs7236481 region. M: 100 bp DNA ladder; lanes represent representative patient and control DNA samples. A single clear band of approximately 390 bp was observed in all amplified samples.

Genotype and allele frequency of *ZNF521* (rs7236481)

The present analysis investigated the intronic single nucleotide polymorphism rs7236481 within the *ZNF521* gene in patients with wounds and burn injuries as shown in Figure 3.

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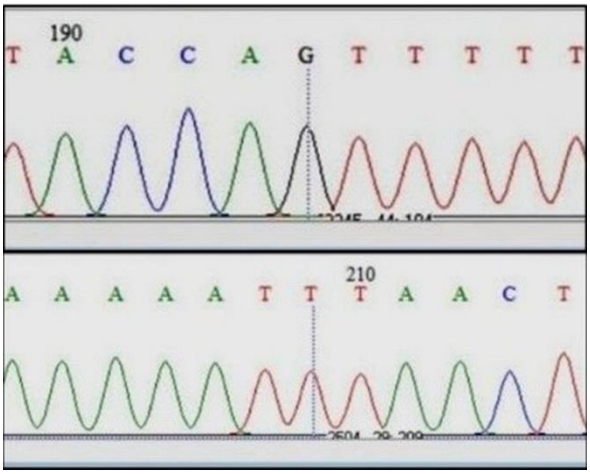

Figure (3): Sanger sequencing chromatograms of the *ZNF521* rs7236481 polymorphism demonstrating different genotypes (GG and TT).

Dominant Model (GT + TT vs GG)

The distribution of variant carriers (GT + TT) compared to the wild-type genotype (GG) is shown in Table 4.

The frequency of variant carriers was higher in patients compared to controls. The odds ratio (OR) was 4.28 with a P-value of 0.01.

Table (4): Dominant Model (GT+TT vs GG)

Group	Patients	Controls	OR (95% CI)	P-value
GG (WT)	4	14	Reference	0.01
GT+TT	44	36	4.28	

Recessive Model (TT vs GG + GT)

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The distribution of the TT genotype compared to (GG + GT) is presented in Table 5.

The TT genotype was more frequent in patients than controls. The odds ratio (OR) was 2.54 with a P-value of 0.03.

Table (5): Recessive Model (TT vs GG+GT)

Group	Patients	Controls	OR (95% CI)	P-value
GG+GT	23	35	Reference	0.03
TT	25	15	2.54	

Hardy–Weinberg Equilibrium (HWE)

Hardy–Weinberg equilibrium analysis was performed for both patients and controls Table 6.

In the control group, genotype frequencies did not deviate from HWE ($P > 0.05$). In contrast, a significant deviation from HWE was observed in the patient group ($P < 0.05$).

Table (6): Hardy–Weinberg Equilibrium

Controls

Genotype	Observed	Expected
GG	14	12.25
GT	21	25.5
TT	15	12.25

- $\chi^2 \approx 2.5$ $P > 0.05$ should be non-significant

Patients

Genotype	Observed	Expected
GG	4	7.4
GT	19	22.3
TT	25	18.3

- $\chi^2 \approx 5.2$, $P < 0.05$ normal significant

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Results indicated a significant association between *ZNF521* rs7236481 and susceptibility to wound and burn infections in the present study. The higher frequency of the homozygous variant genotype (TT) among patients suggests that this polymorphism may be associated with disease susceptibility.

This association was observed in both dominant and recessive genetic models, providing additional evidence that the variant allele may contribute to susceptibility to wound and burn infections. Moreover, individuals carrying at least one variant allele, particularly those with the homozygous variant genotype, showed higher odds of developing the disease compared with individuals carrying the wild-type genotype.

The predominance of the TT genotype in the patient group may indicate a possible genetic predisposition influencing inflammatory regulation and tissue repair responses following wound and burn injury. Wound healing is a complex biological process involving inflammatory signaling, fibroblast activation, extracellular matrix remodeling, angiogenesis, and tissue regeneration. Genetic variations affecting regulatory pathways may therefore influence healing efficiency and susceptibility to infection [19].

The observed association may be related to the regulatory effects of intronic variants. Although rs7236481 does not alter the protein-coding sequence, it may influence gene expression through mechanisms involving transcriptional regulation or RNA splicing. Variations in these regulatory processes may affect cellular pathways involved in inflammatory responses, immune regulation, and tissue repair [20,21].

Previous studies have suggested that *ZNF521* functions as an important transcriptional regulator involved in stem-cell maintenance and cellular differentiation pathways [22]. *ZNF521* has been reported to participate in mesenchymal and hematopoietic differentiation through several developmental signaling pathways. These biological functions may indirectly influence tissue regeneration and repair following injury.

Similarly, Bond et al. [6] reported that *ZNF521* is involved in the regulation of progenitor-cell proliferation and differentiation. Because wound and burn healing depends on the coordinated activity of progenitor cells, fibroblasts, and immune-related signaling pathways, alterations in pathways associated with *ZNF521* may potentially influence tissue remodeling and inflammatory responses during healing. Maurano et al. [23] reported that intronic SNPs may influence transcription-factor binding, enhancer activity, chromatin accessibility, and RNA-processing mechanisms, thereby affecting gene expression and downstream cellular responses.

Hardy–Weinberg equilibrium analysis showed no significant deviation in the control group, supporting the genetic stability of the studied population. In contrast, deviation from HWE was observed among patients.

According to Nielsen et al. [24], deviation from HWE in affected individuals may occur when specific genotypes are associated with disease susceptibility rather than random population variation. Therefore, the observed deviation may support the potential association between rs7236481 and susceptibility to wound and burn infections.

Inflammation and impaired healing are critical factors influencing the outcome of wound and burn injuries. Martin and Nunan [25] highlighted chronic inflammation and impaired cellular remodeling as major contributors to delayed wound healing and increased susceptibility to infection. Consequently, genetic

variations affecting pathways involved in cellular differentiation and inflammatory regulation may be associated with disease pathogenesis.

In conclusion, the findings of the present study suggest that the *ZNF521* rs7236481 polymorphism may be associated with genetic susceptibility to wound and burn infections. However, the absence of functional validation, gene expression analysis, protein analysis, and functional assays limits the interpretation of the biological significance of this association.

Therefore, larger multicenter studies combined with functional investigations are required to clarify the molecular mechanisms underlying the observed relationship. *ZNF521* acts as a transcriptional regulator involved in several biological pathways related to cell differentiation and immune modulation. Consequently, genetic variation within this gene may influence host responses to infection and tissue injury and may contribute to inter-individual variability in clinical outcomes [26].

Conclusion

The present study demonstrated a significant association between the *ZNF521* rs7236481 polymorphism and susceptibility to chronic wound and burn infections among Iraqi patients. The higher frequency of the TT genotype among patients, together with the observed associations in dominant and recessive genetic models, suggests that this variant may be associated with an increased risk of developing wound and burn infections.The findings indicate that *ZNF521* rs7236481 may serve as a potential genetic marker of susceptibility to wound and burn infections. However, the current results demonstrate an association rather than a causal relationship, and therefore the biological role of this polymorphism cannot be conclusively established based on the present study alone. Further investigations involving larger sample sizes, multicenter populations, and functional studies are required to validate these findings and clarify the molecular mechanisms through which *ZNF521* genetic variation may influence host responses to tissue injury, inflammation, and wound healing. Such studies may contribute to a better understanding of the genetic factors associated with wound and burn infection susceptibility.

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Authors Declaration

Consent for publication: Not applicable.

Data availability: Available from the corresponding author upon reasonable request.

Competing interests: None declared.

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Authors' Contribution Statement

Dina A. Aziz performed sample collection, molecular analysis, statistical analysis, and manuscript writing. Dr. Sahar M. Hussein supervised the study and revised the manuscript.

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