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Research Article

Frequencies of Three Targeted HBB Gene Mutations Among Transfusion-Dependent B-Thalassemia Patients from Southern Punjab, Pakistan

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Abstract	Manuscript Information
Beta-thalassemia is a blood disorder characterised by the low production of β-globin chains in haemoglobin. The identification of the most frequent β-thalassemia mutation in Southern Punjab remains limited. We employed an allele-specific Polymerase Chain Reaction (AS-PCR) method to investigate three common mutations in 366 patients with β-thalassemia using peripheral blood samples. Three pairs of primers were used to detect the three distinct β-thalassemia mutations. The selected mutations were IVS-1-5 (G>C), frame-shift-codon 8/9 (+G), and codon 41/42 (-TCTT), referred to as mutations 1, 2, and 3, respectively. None of the three targeted mutations was detected in 172(47.0%) patients. The most frequently observed mutation detected in the population was mutation 1, found in 91 (24.9%) patients, followed by mutation 3 in 62 (16.9%) and mutation 2 in 41 (11.2%) patients. In the Southern Punjab population, the most common beta-thalassemia mutation among the targeted mutations was mutation 1, followed by mutations 3 and 2. Statistical analysis revealed that the frequencies were not equal among the targeted mutations, as $\chi^2(2) = 19.49$, and $P < 0.001$. The absence of these three mutations in the 172 patients points to the prevalence of mutations other than the three common mutations considered in this study.	▪ ISSN No: 2583-7397 ▪ Received: 11-08-2026 ▪ Accepted: 15-09-2026 ▪ Published: 18-09-2026 ▪ IJCRM:5(5); 2026: 300-306 ▪ ©2026, All Rights Reserved ▪ Plagiarism Checked: Yes ▪ Peer Review Process: Yes
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KEYWORDS: Allele-specific Polymerase Chain Reaction, β -thalassemia, Mutation, β -globin, genetics.

1. INTRODUCTION

Beta-thalassemia (β -thalassemia) is a blood disorder (an autosomal recessive trait), characterised by low production of β -globin chains for haemoglobin. (Dordevic et al., 2025). It arises due to point mutations or, less commonly, deletions within the beta-globin gene present on chromosome 11 (Kattamis et al., 2022; Khalid et al., 2020). β -thalassemia is a large-scale Public Health problem worldwide, with over 150 million carriers documented in over 60 countries (Taher et al., 2018). The distribution of thalassemia was previously limited to the traditional "thalassaemia belt," which ranges across the Mediterranean Basin, Middle East, and Southeast Asia. However, in recent years, this condition has become a worldwide health issue likely due to migration. This is particularly evident in Pakistan, with a carrier rate of 5-8%, corresponding to millions of genetically heterogeneous individuals who require rigorous surveillance programs. (Haq et al., 2025).

The global carrier rate of β -thalassemia is approximately 3%, and an estimated 60,000 significant cases of thalassemia are diagnosed annually. (Ali et al., 2021). Approximately 5,000 children are diagnosed annually, and the global population of carriers exceeds 10 million. Typical indications of β -thalassemia in children include slow growth, inadequate food intake, enlargement of the spleen, yellowing of the skin (jaundice), excessive growth of the upper jaw (maxillary hyperplasia), misalignment of teeth (malocclusion), and gallstone formation (cholelithiasis) (Iyevhobu et al., 2023).

β -Thalassemia is a monogenic disorder characterised by over 380 distinct mutations identified in Pakistan (El-Beshlawy et al., 2024). This shows the complexity of transmission and requires diagnosis through targeted genetic testing, treatment plans, and genetic counselling to improve the management of this condition in any population. (Biswas et al., 2022).

Clinically, β -thalassemia is categorised into three distinct types: major, minor, and intermediate forms. Among the diverse known mutations, three variants are the most frequently documented: IVS-1-5 (G>C) (mutation 1), frame shift codon 8/9 (+G) (mutation 2), and codon 41/42 (-TCTT) (mutation 3) (Siddique & Younas, 2026). These variants have been frequently reported among patients with β -thalassemia in South Asian populations and are therefore important targets for molecular screening. (Samorlu & Saleem, 2026; Sanchez-Villalobos et al., 2022).

Mutation 1 is a commonly occurring β -thalassemia mutation that occurs in intron 1 of the HBB gene, specifically at position 5, where guanine (G) is replaced with cytosine (C). This mutation disrupts the normal splicing of beta-globin mRNA, resulting in a decrease or no production of the beta-globin chain of haemoglobin (Haq et al., 2025). Mutation 2 is a frameshift mutation within the HBB gene, which involves the insertion of a single guanine (G) nucleotide at positions 27-28 of the gene. As a result, its reading frame shifts, leading to premature stop codons and nonfunctional beta-globin proteins (Muhammad et al., 2017).

Mutation 3 is a frameshift mutation found within the HBB gene that involves the deletion of four nucleotides from positions 126-129 of the genome. Consequently, the reading frame of the gene shifts, leading to premature stop codons and the production of nonfunctional beta-globin proteins. (Haq et al., 2025).

β -thalassemia is an autosomal recessive disorder inherited in a classical Mendelian manner. Individuals who inherit a faulty β -globin gene from both parents are homozygous for the mutation. At conception, the cross between two carriers of an autosomal recessive disorder has a 50% chance of producing an asymptomatic carrier, a 25% chance of producing a diseased child, and a 25% chance of producing a healthy child. (Dolai et al., 2026) (Figure 1).

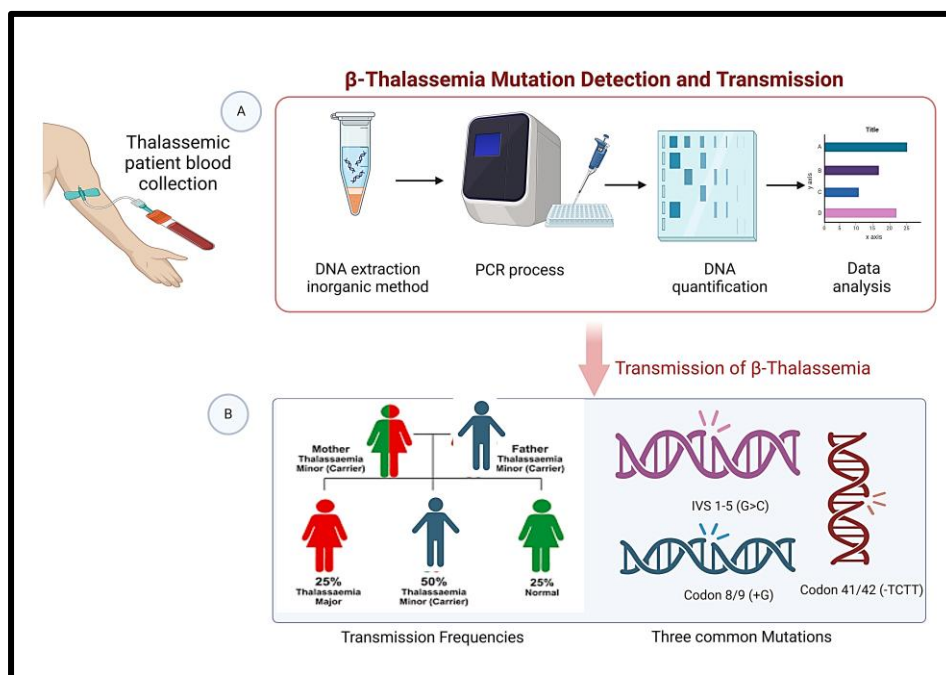


Figure 1: Workflow of β -Thalassemia Mutation Detection (A) and Genetic Pattern (B).

The process begins with the collection of peripheral blood from an affected individual, followed by DNA extraction, PCR amplification and quantification, and data analysis of the sequenced DNA. Figure B illustrates the transmission of β -thalassemia from parents to their offspring. The genetic profiles contain Mendelian inheritance ratios and specific mutation frequencies, such as those of mutations 2 and 3, respectively. Allele-specific PCR (AS-PCR) is a technique used to identify specific DNA alleles, or mutations. (Komijani et al., 2021). AS-PCR is particularly useful for detecting single nucleotide polymorphisms (SNPs) and other genetic variations. For β -thalassemia, AS-PCR is invaluable for diagnosis and genetic analysis because of its significant advantages. (Long et al., 2013). AS-PCR's high sensitivity and specificity allow for the precise detection of beta-globin gene mutations, even at low levels, which is crucial for identifying carriers and diagnosing affected individuals (Mahmud & Enni, 2025).

We employed Allele-Specific Polymerase Chain Reaction (AS-PCR) to identify the most frequently occurring β -thalassemia mutations in the population, specifically focusing on mutations 1, 2, and 3. Previous studies have primarily addressed broader genetic trends in β -thalassemia (Haq et al., 2025) without

systematically examining the specific mutations prevalent in Southern Punjab.

Hypothesis

H₀: The three investigated mutations have equal frequencies among patients in whom one of the three mutations was detected.

H₁: The frequencies of the three investigated mutations are not equal among patients in whom any of the three mutations are detected.

2. MATERIALS AND METHODS

Peripheral blood samples were collected from 366 transfusion-dependent β -thalassemia patients registered at the Mailsi Blood Bank & Thalassemia Centre in Punjab, Pakistan (Figure 2). All patients were initially diagnosed with β -thalassemia using standardised tests. These patients had different ethnic backgrounds from various regions in Southern Punjab. To gather individual data on each patient as well as epidemiological factors related to β -thalassemia, a questionnaire was administered and completed by both the patients and their family members. The questionnaire covered the age range and ethnicity of non-mutation-free individuals. Additional information (β -thalassemia status) was obtained from laboratory findings (Table 2).

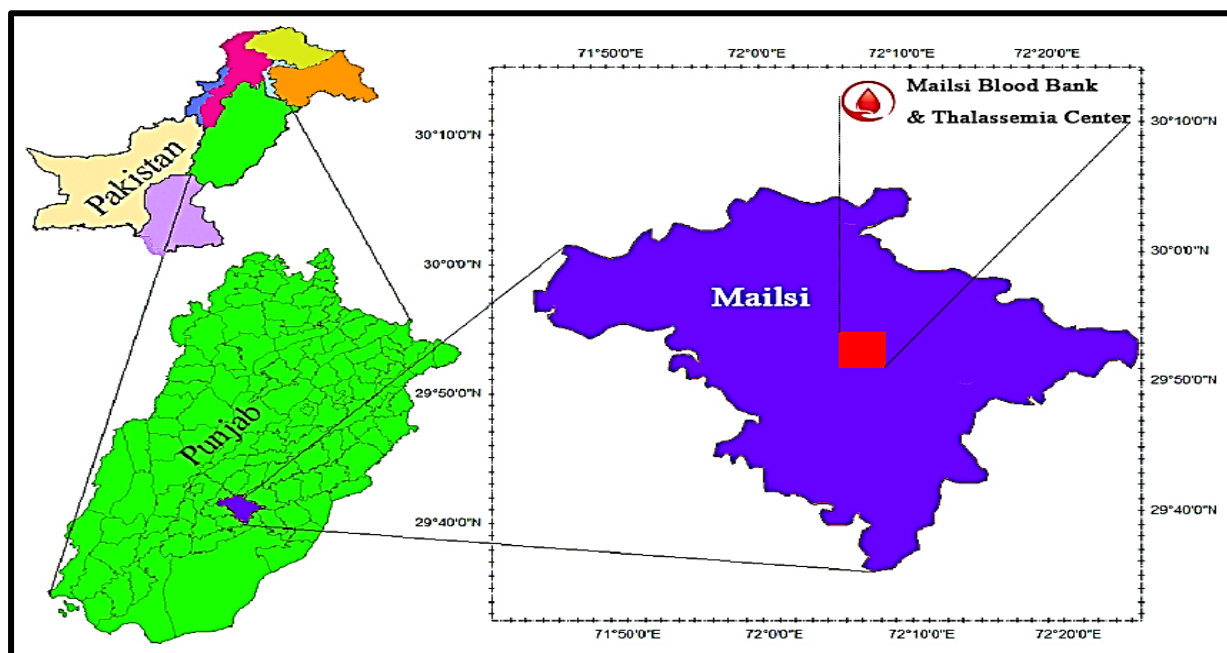


Figure 2: Geographic location of the study setting. Green map (Southern Punjab) extracted from Pakistan; purple map (a southern city) containing the blood bank (red).

Blood samples for DNA extraction were collected separately in EDTA-containing vacutainers and stored at 10°C. DNA was isolated using an inorganic extraction protocol and visualised on an ethidium bromide-stained agarose gel after exposure to ultraviolet (UV) light. The isolated DNA samples were stored at -20°C for AS-PCR. (Čanović et al., 2024).

Allele-specific PCR (AS-PCR) was used to detect point mutations based on the presence or absence of amplification products. Every DNA sample was examined for three common

mutations in Southern Punjab, Pakistan. (Siddique & Younas, 2026). AS-PCR was conducted in a 20 μ L reaction mixture containing 1X PCR buffer. Additionally, the mixture contained 50 pmol of each of the four primers, 0.5–1 μ g genomic DNA, 0.25 mM dNTPs, 1.5 mM MgCl₂, and 0.5–1 U Taq polymerase. Thermal cycling included 33 cycles of denaturation at 94°C for 45 s, followed by primer annealing and extension at 67°C for 1.5 min. In the final cycle, the extension time was increased to six minutes. The PCR product was mixed with 1X loading dye and

electrophoresed on a 1.8% agarose gel containing ethidium bromide for 20–60 min at 100 V. The gel was visualised and photographed using UV light.

A set of primers used in the PCR experiments was designed to amplify both mutation-free (patients without any of the three targeted HBB mutations) and mutation-specific sequences. Primers (MF1, MF2, and MF3) produced an 861 bp product,

representing mutation-free (MF) patients. In contrast, the mutation primers (1, 2, and 3) targeted specific genetic variations, yielding product sizes of 285, 215, and 439 bp, respectively. These primers were essential for identifying and analysing the different genetic variants in this study. (Rehman et al., 2021) (**Table 1**).

Table 1. Target gene primers and sequences

Primers	Sequences (5'>3')	Product Size (bp)
MF1	CAA TGT ATC ATG CCT CTT TGC ACC	861
MF2	GAC TCA AGG CTG AGA GAT GCA GGA	
MF3	TCA CTT AGA CCT CAC CCT GTG GAG CCA	
Mutation 1	CTC CTT AAA CCT GTC TTG TAA CCT TGT TAG	285
Mutation 2	CCT TGC CCC ACA GGG CAG TAA CGG CAC ACC	215
Mutation 3	GAG TGG ACA GAT CCC CAA AGG ACT CAA CCT	439

Statistical Analysis

Statistical analysis (frequencies) of the three investigated mutually exclusive mutations was performed among patients in whom one of the three mutations had been identified. The null hypothesis was tested using Pearson's chi-square goodness-of-fit test. The hypothesis established that the three mutations occurred at equal frequencies, with statistical significance set at $p < 0.05$ at 2 degrees of freedom, and the effect size was assessed using Cramér's V (Telford et al., 2020). Statistical Package for the Social Sciences (SPSS) version 27 was used for the analysis.

Ethical Statement

This study was reviewed and approved by the Ethics Committee for Human Patients at Nishtar Medical University, Multan (Pakistan). The study was approved under No.: 18186/NMU. All patients and their guardians were informed of the purpose of the study and provided written consent before commencing the study.

3. RESULTS

Each sample was tested separately for these three mutations 1, 2, and 3. Three PCRs were performed for each of the mutations. Each PCR reaction yielded an 861-bp control amplicon, confirming successful amplification within the 800-900 bp range on gel electrophoresis. PCR products for the internal typical mutation-free and mutant instances were observed for all types of mutations. Those without any of these three mutations exhibited a single band at 861 bp, whereas those with mutations displayed two bands simultaneously: one at 861 bp and the other in an altered region. Mutants with mutations 1, 2, and 3 displayed bands at 285, 215, and 439 bp, respectively (**Figure 3**). The study's findings showed that IVS-1-5 (mutation 1) had the highest frequency, accounting for 24.9% of the population studied. Mutations 3 and 2 had frequencies of 16.9% and 11.2%, respectively. No mutant bands were observed in the other samples (Table 2). This indicates that they may have alternative forms or no β -thalassemia mutations, as other β -globin defects were not investigated in this study.

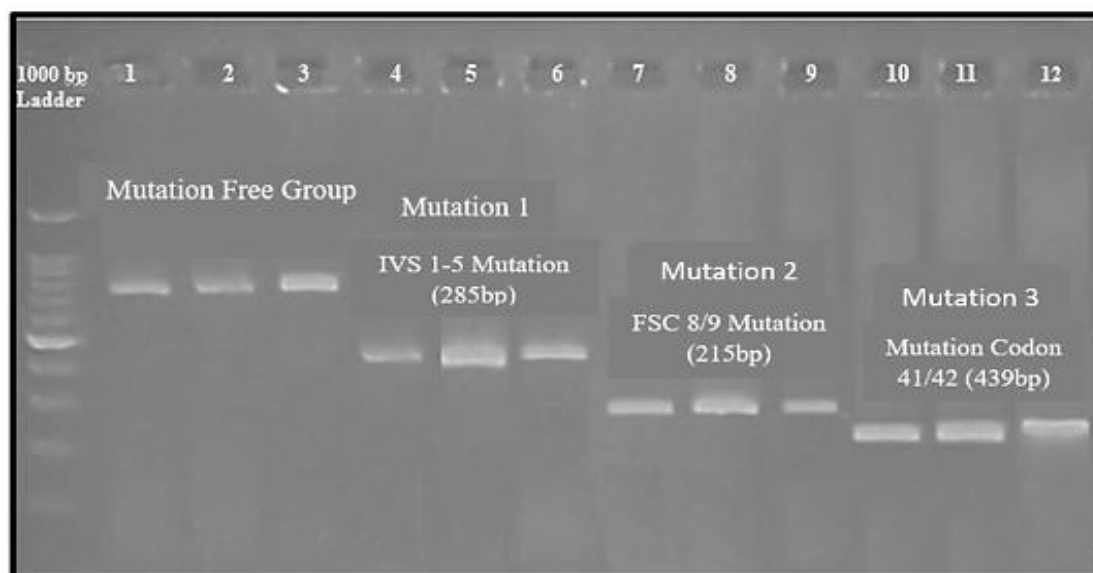


Figure 3: Distribution of three targeted genetic mutations 1, 2, and 3 among patients with β -thalassemia in Southern Punjab, Pakistan, highlighting mutation 1 as the most common mutation, followed by mutations 3 and 2.

The bar chart shows the distribution of mutations in this study. The portion labelled “mutation-free group” represents the

absence of the three investigated mutations. The remaining sections show the distributions of the three specific mutations.

Table 2. Mutation Spectrum of β -Thalassemia Patients

Mutation Types	n (%)	Age Group	n (%)	Ethnicity	n (%)
Mutation 1: IVS-1-5 (G>C)	91 (24.9)	< 2 years	234 (64.0)	Saraiki	146 (39.9)
Mutation 2: Fr 8/9 (+G)	41 (11.2)	2 – 5 years	88 (24.0)	Punjabi	78 (21.3)
Mutation 3: Codon 41/42 (-TCTT)	62 (16.9)	> 5 years	44 (12.0)	Urdu Speaking	55 (15.0)
Unknown Mutation	172 (47)				
Total Patients	366 (100)				

Table 2 displays the mutation spectrum, age distribution, and ethnic composition of the three targeted β -thalassemia patients in Punjab, Pakistan. Data are presented as the number of patients (n) with corresponding percentages (%). Mutation data are based on the actual 366 patients analysed. The ethnicity and age distribution show only the three main ethnic groups reported in the region (Saraiki, Punjabi, Urdu-Speaking), accounting for 279 patients (76.2%). The remaining 87 patients (23.8%) belong to other ethnic groups or have unknown ethnicity.

Among the 194 patients, the expected frequency for each mutation was 64.67, with $\chi^2(2) = 19.5$, $P < 0.001$, and Cramér's $V = 0.224$ at the 95% confidence level. In contrast, the actual frequencies of mutations 1, 2, and 3 were 46.91%, 21.13%, and 31.96%, respectively.

4. DISCUSSION

Mutation 1 was the most common in the Southern Punjab group. This finding is consistent with a study conducted in Pakistan that found five frequent mutations responsible for 99% of cases, with thalassemia mutation 1 having the highest frequency of 44.4% across all ethnic groups. (Hassan et al., 2017). This prevalence demonstrates the geographic and ethnic diversity of mutations, as evidenced by a study conducted in Bangkok, Thailand, where mutation 1 was found to be prevalent, and only approximately 8% of patients had this mutation, which is less frequent than other mutations. (Panichchob et al., 2021). The next highest identified mutation in our study was mutation 3, which corroborates the findings of a study conducted in Karachi, Pakistan, in which mutation 3 was reported at a frequency of 17.5% (Khaliq, 2022).

In contrast, the least common mutation among the three targeted mutations was mutation 2, which accounted for 11% of all patients. Understanding the distribution patterns of β -thalassemia mutations is crucial for establishing effective diagnostic strategies for parents. Interestingly, a study conducted at the Thalassemia Centre of the Pakistan Medical Institute (PIMS) in Islamabad reported that mutation 2 was the most frequently reported (28.7%). A study on HBB gene mutations in Mardan, Pakistan, demonstrated that mutation 1 was the most frequently reported mutation detected in Charsadah. (Shakeel et al., 2016). The most common mutations in the Pakistani population were Mutation 1 (36.48%), Mutation 2 (31.16%), and Mutation 3

(7.11%) (Black et al., 2010). Regional research has shown that Mutation 1 is more common in neighbouring countries such as India (56.31%), Sri Lanka (64.60%), the Iranian province of Baluchistan- Sistan (44.8%), the UAE (~50%), and Bangladesh

(56.25%). People living in the Mediterranean, Southeast Asia, and Africa are more affected by β -thalassaemia. (Hassan et al., 2017). The genetic prevalence of β -thalassemia in Southeast Asia is 2.5 to 15% (World Health Organization, 2021). These findings contrast sharply with our results, underscoring the variability in β -thalassemia mutations across different populations and provinces in Pakistan. Such differences in mutation prevalence highlight the need for advanced genetic sequencing techniques to uncover previously unidentified mutations and region-specific studies to inform public health strategies and genetic counselling efforts.

5. CONCLUSION

Numerous variants are responsible for the development of β -thalassemia. This study presents molecular data on three commonly reported HBB variants among transfusion-dependent β -thalassaemia patients in Southern Punjab, Pakistan. Three of the most frequent mutations were chosen, and their rates were examined in individuals from Southern Punjab, Pakistan. Among the three HBB variants investigated, IVS-1-5 (G>C) was the most frequently detected mutation in the study population, whereas none of the targeted variants was detected in 47% of the patients. The observed distribution of the three targeted mutations differed significantly from an equal distribution; therefore, the null hypothesis was rejected, suggesting that the frequencies of the three targeted mutations were not equal among the 194 patients included in this study. Detailed molecular characterisation is required to determine the variants responsible for these remaining cases.

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Data Availability Statement: Data available on request due to privacy/ethical restrictions.

Conflict of Interest: The authors declared no conflict of interest.

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