



Frequency of the IL12B rs3212227 polymorphism and associated clinical features in Guatemalan patients with plaque psoriasis: a cross-sectional molecular study

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ABSTRACT

Background: Psoriasis is a chronic inflammatory disease in which the IL-12/IL-23 pathway plays a central pathogenic role. Genetic variation within IL12B, which encodes the shared p40 subunit of IL-12 and IL-23, has been associated with psoriasis and psoriatic arthritis in several populations; however, data from Central American populations remain limited. **Methods:** A cross-sectional observational study was conducted in adult patients with plaque psoriasis attending a tertiary dermatology and rheumatology center in Guatemala. Clinical data were collected using standardized instruments, including the Psoriasis Area and Severity Index (PASI). Psoriatic arthritis was classified using CASPAR criteria, incorporating musculoskeletal ultrasound when indicated. Genomic DNA was extracted from peripheral blood, and the IL12B rs3212227 polymorphism was analyzed using polymerase chain reaction (PCR) followed by Sanger sequencing. Exploratory analyses between genotype and clinical variables were explored using descriptive statistics and appropriate statistical tests. **Results:** Thirty-five patients were enrolled. Valid genotyping results were obtained for nine samples. Genotype frequencies were 88.9% for TT and 11.1% for GG, with a G allele frequency of 11.1%. The mean PASI score was 13.3 ± 8.4 , and 34.3% of patients met CASPAR criteria for psoriatic arthritis. No statistically significant associations were observed between rs3212227 genotype and disease severity, age of onset, or presence of psoriatic arthritis. **Conclusions:** In this Guatemalan cohort, the IL12B rs3212227 polymorphism was infrequent. Although no clinical associations were identified, this study provides the first molecular data describing this variant in a Central American population. These findings contribute to the understanding of genetic diversity in psoriasis and highlight the need for larger, population-based studies to clarify the role of IL12B variation in disease expression and therapeutic response.

Keywords: *Psoriasis, IL12B, rs3212227, Genetic polymorphism, Psoriatic arthritis, Guatemalan population.*

INTRODUCTION

Psoriasis is a chronic immune-mediated inflammatory disease with systemic manifestations that extend beyond the skin, including a substantial risk of psoriatic arthritis and cardiometabolic comorbidities (1,2).

Although plaque psoriasis is the most common clinical subtype, affecting approximately 85-90% of patients worldwide, the disease burden is increasingly recognized as multisystemic and heterogeneous, with significant implications for rheumatologic care (2,3). Dysregulation of the IL-12/IL-23 axis is central to pathogenesis of psoriasis, which promotes differentiation and activation of Th1 and Th17 lymphocyte subsets (2). These pathways drive sustained production of pro-inflammatory cytokines such as interferon- γ , IL-17, and IL-22, resulting in keratinocyte hyperproliferation, chronic synovial inflammation, and enthesitis (2). The clinical relevance of this pathway is underscored by the efficacy of biologic agents targeting IL-12/23, which are widely used in rheumatologic practice (2).

The IL12B gene encodes the p40 subunit shared by IL-12 and IL-23 (4,5). Genetic variation within IL12B has been associated with susceptibility to psoriasis and psoriatic arthritis in several populations, particularly through the single-nucleotide polymorphism rs3212227 located in the 3' untranslated region. Meta-analyses and population-based studies in European and Asian cohorts suggest that this variant may influence disease risk and an inflammatory phenotype; however, results have been inconsistent across ethnic groups (5-7). Notably, studies in Latin American populations remain scarce and have produced mixed results, suggesting that genetic susceptibility to psoriasis may vary substantially by ancestry (6).

Guatemala represents a genetically underrepresented population in immunogenetic research, despite its ethnically diverse background and growing use of targeted biologic therapies. To date, no molecular studies have characterized IL12B polymorphisms in Guatemalan patients with psoriasis, limiting the extrapolation of genetic risk models derived from other populations. This knowledge gap is particularly relevant for rheumatology, given the role of IL-12/23 signaling in psoriatic arthritis pathogenesis and treatment response.

The present study aimed to describe the frequency of the IL12B rs3212227 polymorphism in Guatemalan patients with plaque psoriasis and to explore its relationship with clinically relevant features, including disease severity, age of onset, and presence of psoriatic arthritis. By providing the first molecular data on this variant in a Central American cohort, this study contributes foundational evidence for future population-based and translational research in psoriasis and psoriatic arthritis.

Methods

Study design and setting

A cross-sectional observational study was conducted at the outpatient Rheumatology Clinic of the Instituto de Dermatología y Cirugía de Piel (INDERMA), a tertiary referral center in Guatemala City. Patients were recruited consecutively during scheduled clinic visits between April and October 2023.

Participants

Adult patients (> 18 years) with a clinical diagnosis of plaque psoriasis were eligible for inclusion. Exclusion criteria included contraindications to venipuncture, inability to provide informed consent, or refusal to participate. No control group was included, as the primary objective of the study was descriptive and exploratory.

Clinical assessment

Demographic and clinical data were collected using a standardized electronic data capture form (REDCap). Sex was recorded as female or male as assigned at birth. Disease severity was assessed using the Psoriasis Area and Severity Index (PASI), calculated with the PsoriasisCalc application. Age at disease onset, disease course, family history of psoriasis, and presence of nail involvement were recorded. Psoriatic arthritis was classified according to the CASPAR criteria (8). Patients with clinical suspicion of inflammatory joint involvement underwent musculoskeletal ultrasound of the hands performed by an experienced radiologist. Ultrasound evaluation included assessment of synovitis, tenosynovitis, erosions, and Doppler signal using a high-frequency linear transducer (9). Rheumatoid factor testing was performed when clinically indicated. The radiographic criterion of new bone formation was not applied due to its low sensitivity in early disease.

Sample collection and DNA extraction

Peripheral blood samples (3 mL) were obtained by venipuncture and collected in tubes without anticoagulant. Samples were transported under refrigerated conditions (<10 °C) and stored at 4 °C until processing. Genomic DNA was extracted using the ReliaPrep™ Blood gDNA Miniprep System (Promega®) following the manufacturer's protocol and stored at -20 °C until analysis.

Genotyping of IL12B rs3212227

The rs3212227 polymorphism in the IL12B gene was amplified by polymerase chain reaction (PCR) using specific primers (forward: 5'-GGCAACTTGAGAGCTGGAAAATC- 3'; reverse: 5'-ACCCTCAAGAAAGCCTCTGG-3'). PCR conditions included an initial denaturation at 95 °C for 2 minutes, followed by 30 cycles of denaturation (95 °C for 40 seconds), annealing (60 °C for 45 seconds), and extension (72 °C for 30 seconds), with a final extension at 72 °C for 10 minutes. Primer specificity was verified in silico using Primer-BLAST (NCBI). PCR products were submitted for bidirectional Sanger sequencing (Macrogen, South Korea). Sequence quality was assessed using Chromas v2.6.6, and alignment was performed against the IL12B reference sequence (RefSeq NG_011493.1). Genotypes were determined based on nucleotide identity at the rs3212227 locus.

Statistical analysis

Descriptive statistics were used to summarize demographic and clinical variables. Genotype and allele frequencies were calculated for samples with valid sequencing results. Exploratory analyses between the rs3212227 genotype and categorical clinical variables were evaluated using Fisher's exact test or chi-square tests as appropriate. Differences in age at disease onset were assessed using Student's t-test after visual assessment of normality (histograms and Q-Q plots) and confirmation with the Shapiro-Wilk test. Statistical analyses were performed using Stata BE, with a significance threshold of $p < 0.05$.

Results

Study population

Between April and October 2023, a total of 35 adult patients with plaque psoriasis were enrolled (Figure 1 and 2). Baseline demographic characteristics are summarized in Table 1. The mean age was 44.9 ± 14.4 years, and 57.1% of participants were female. Most patients were born in Guatemala City, while the remainder originated from other departments (Escuintla, Chimaltenango, Jalapa, Quetzaltenango, Retalhuleu, Izabal and Quiché).

Clinical characteristics

Clinical features are presented in Table 2. Most patients had chronic disease (94.3%). Disease course was classified as progressive in 42.9%, intermittent in 34.3%, and stationary in 22.9%. A family history of psoriasis was reported by 28.6% of participants. The mean PASI score was 13.3 ± 8.4 , corresponding predominantly to moderate disease severity (Graph 1). The most frequently affected anatomical region was the lumbosacral area (Graph 2, Figure 3). Nail involvement was observed in 37.1% of patients, most commonly presenting as nail pitting and oil-drop changes (Graph 3, Figure 4).

Evaluation of psoriatic arthritis

Musculoskeletal ultrasound of the hands *demonstrated* inflammatory changes compatible with arthritis in 18 patients (51.4%), while 3 patients (8.6%) showed no ultrasonographic evidence of arthritis (Figure 5-7); ultrasound data were unavailable for 14 patients (Table 3). Rheumatoid factor testing was negative in 48.6% of patients, positive in 17.1%, and unavailable in 34.3%. Based on CASPAR criteria, 12 patients (34.3%) met classification criteria for psoriatic arthritis.

Genotyping results

Of the 35 samples collected, 10 were excluded from molecular analysis due to insufficient DNA quality. Of the 25 sequenced samples, 9 showed optimal sequencing quality, allowing for the interpretation of the rs3212227 SNP in the IL12B gene (Figure 8). In these samples, the SNP was located between base pairs 300 and 330. The remaining samples exhibited low-quality signals, preventing genotype determination (Supplementary Files).

Among the samples with valid sequences, only one showed the variant corresponding to the polymorphic allele. In that sample (Figure 8A), the flanking motif TAGTT was identified, followed by a C at the SNP position. This C, observed in the reverse strand, complements the G allele on the forward strand, indicating the A → G substitution (variant allele). The other eight samples (Figure 8B) presented the motif TAGTT followed by a T at the SNP position, observed as an A in the reverse sequence. This A complements the T allele on the forward strand, corresponding to the non-polymorphic allele. Genotypic frequencies (TT and GG) and allelic frequencies (T and G) for the rs3212227 SNP are presented in Table 4.

Association between rs3212227 and clinical variables

No statistically significant association was observed between rs3212227 genotype and psoriasis severity as categorized by PASI score (Fisher's exact test, $p = 1.000$; Table 5). Among patients with valid genotyping, 4 had psoriatic arthritis, including the single individual carrying the GG genotype. Genotype distribution did not differ significantly according to the presence of psoriatic arthritis ($\chi^2 = 0.41$, $p = 0.52$; Table 6). Age at disease onset did not differ significantly between genotype groups (Student's t-test, $p = 0.81$; Table 7). Normality assumptions for age and age at onset were assessed with histograms and Q-Q plots and confirmed with the Shapiro–Wilk test.

Discussion

The clinical characterization of the cohort in this study provides relevant insight into the phenotype of psoriasis in a Guatemalan population. The predominantly chronic and progressive disease course observed is consistent with the long-standing inflammatory nature of psoriasis described in other populations. Most patients presented with moderate disease severity (mean PASI 13.3), indicating a substantial cutaneous burden. This finding may reflect barriers to access to specialized care, delayed consultation, or referral bias toward more complex cases at tertiary centers such as INDERMA.

Although women predominated in this cohort, psoriasis has been reported to affect men and women at similar rates globally (2,3,5). Therefore, the higher proportion of female patients in this study is more likely attributable to differences in health-seeking behavior rather than true biological variation. This observation highlights the importance of future studies aimed at improving early identification and engagement of male patients with psoriasis in Guatemala.

Nail psoriasis was identified in 37.1% of patients, a frequency comparable to that reported in the literature, where nail involvement ranges from 32% to 50% among adults with plaque psoriasis (3,5). The most common findings (nail pitting and oil-drop changes) are consistent with internationally described patterns (3,5). Clinically, nail involvement is relevant because it has been associated with more extensive skin disease and an increased risk of psoriatic arthritis (3,5). The nail manifestations observed in this cohort therefore align with global data and may serve as early clinical indicators of increased disease activity or severity in this population.

The prevalence of psoriatic arthritis in this cohort was relatively high (34.3%). This exceeds estimates based solely on clinical criteria, which typically identify psoriatic arthritis in 10–15% of patients with psoriasis (3,5). However, studies incorporating musculoskeletal ultrasound, which is an imaging modality with greater sensitivity for detecting subclinical synovitis and enthesitis, have reported prevalences closer to 25–30% (8). In this context, the frequency observed in the present study is consistent with ultrasound-based investigations and may reflect improved detection of inflammatory joint involvement or a higher

inflammatory burden in this population. The frequency of the G allele of IL12B rs3212227 was low (11.1%) in this cohort, lower than that reported in European and Asian populations, where frequencies between 20% and 25% have been described (4,7,11). This difference likely reflects genetic variability within the Guatemalan population. A low allele frequency may also suggest a reduced contribution of this variant to psoriasis susceptibility in this setting, although the limited sample size precludes definitive conclusions. Larger studies will be necessary to determine whether this observation represents a true population-specific pattern.

An interesting finding was the numerical equivalence between genotype and allele frequencies for rs3212227, an uncommon observation in population genetic studies. This pattern is primarily explained by the absence of heterozygous genotypes among samples with valid sequencing results and by the small number of successfully genotyped individuals. In such circumstances, when most individuals are homozygous for one allele and only a single individual carries the variant allele, allele frequencies closely mirror genotype frequencies. This should not be interpreted as evidence of a particular biological equilibrium, but rather because of sample size and limited genetic variability detected in this cohort. Studies with larger sample sizes may reveal a more complex distribution.

In other populations, the G allele of IL12B rs3212227 has been associated with increased susceptibility to psoriasis and psoriatic arthritis through modulation of expression of the shared p40 subunit of IL-12 and IL-23. These cytokines promote Th1 and Th17 differentiation and amplify production of IFN- γ , IL-17, and IL-22, thereby contributing to epidermal hyperproliferation and chronic inflammation characteristic of psoriasis (2,4). In the present study, however, no clear association was identified between rs3212227 and disease severity, age of onset, or presence of psoriatic arthritis. It is possible that this variant does not play a major role in determining the clinical phenotype of psoriasis in the Guatemalan population, or that its effect is modulated by other genetic or environmental factors. Nevertheless, the limited number of genotyped samples does not allow for the complete exclusion of a biological association.

Consistent with these findings, Sandoval-Talamantes et al. reported no association between rs3212227 and plaque psoriasis susceptibility in a Mexican mestizo population (6). Together, these observations suggest that rs3212227 may contribute to psoriasis risk only in the presence of specific haplotypes or in interaction with other immunoregulatory genes, such as IL23R, TNFAIP3, or HLA-C, whose risk variants may differ across Latin American populations (2,10).

From a clinical perspective, these findings are relevant because IL12B encodes the p40 subunit targeted by biologic therapies such as ustekinumab and guselkumab. Although treatment response was not evaluated in this study, future research could explore whether rs3212227 or related variants influence therapeutic efficacy or tolerability, thereby contributing to the development of personalized treatment strategies in psoriasis.

Several limitations should be acknowledged. The primary limitation was the small number of samples with valid sequencing results, which reduced statistical power. Additionally, the absence of a non-psoriatic control group limited the ability to estimate population-level allele frequencies. Technical issues, including low-quality chromatograms, likely resulted from DNA degradation related to prolonged storage times and potential disruptions in the cold chain. Such degradation can impair PCR efficiency and increase nonspecific amplification, leading to background noise in sequencing reads (12).

Secondary technical factors may also have contributed, including possible cross-contamination during sample handling or partial primer non-specificity identified during *in silico* analysis, which revealed two potential amplification sites. Although PCR conditions were optimized to minimize nonspecific amplification, residual effects may have influenced sequencing quality (12).

Despite these limitations, this study demonstrates the feasibility of applying molecular biology techniques to the study of psoriasis genetics in Guatemala and establishes a methodological foundation for future investigations. Larger studies with stricter preanalytical control, improved sample preservation, and higher-throughput genotyping methods are warranted. Evaluation of multiple variants within the IL-23/Th17 pathway may further clarify the genetic and immunologic landscape of psoriasis in this population.

Conclusions

In this Guatemalan cohort, the IL12B rs3212227 polymorphism was infrequent. Due to the limited number of samples with valid sequencing results, no reliable associations could be established with age of disease onset, psoriasis severity, or presence of psoriatic arthritis. Nonetheless, detection of the G allele confirms its presence in individuals from this population. DNA integrity was identified as a key limiting factor affecting sequencing quality, underscoring the importance of optimized sample handling and storage in future molecular studies. Overall, these findings highlight the need for continued genetic research on psoriasis in Guatemala using integrative approaches that combine genetics, immunology, and environmental factors. Expanding sample size, including appropriate control groups, and strengthening preanalytical logistics will be essential to advance understanding of psoriasis pathogenesis and to support the development of population-specific, personalized therapeutic strategies.

Declarations

Ethics approval and consent to participate

The study was conducted in accordance with the Declaration of Helsinki and was approved by the Research Department and Ethics Committee of the Faculty of Medicine, Universidad Francisco Marroquín, Guatemala. Authorization was obtained from the Instituto de Dermatología y Cirugía de Piel (INDERMA). Written informed consent was obtained from all participants before enrollment.

Consent for publication

Written informed consent for publication of clinical data and images was obtained from all participants.

Availability of data and materials

The dataset generated and analyzed during the current study is available from the corresponding author on reasonable request. Sequencing chromatograms are included in the supplementary files.

Competing interests

The authors declare that they have no competing interests.

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Authors' contributions

MIBO and ACM conceived and designed the study. MIBO collected clinical data, performed statistical analysis, interpreted results, and drafted the manuscript. MAT contributed to rheumatologic interpretation and critical revision of the manuscript. All authors read and approved the final manuscript.

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References

1. Villarreal-Martínez A, Gallardo-Blanco H, Cerda-Flores R, Torres-Muñoz I, Gómez-Flores M, Salas-Alanís J, et al. Candidate gene polymorphisms and risk of psoriasis: a pilot study. *Exp Ther Med*. 2016;11(4):1217–22. doi:10.3892/etm.2016.3066.
2. Van de Kerkhof PCM, Nestlé FO. Psoriasis. In: Bologna JL, Schaffer JV, Cerroni L, editors. *Dermatology*. 4th ed. Philadelphia: Elsevier; 2018. p. 138–60.
3. Kaufman BP, Alexis AF. Psoriasis in skin of color: insights into the epidemiology, clinical presentation, genetics, quality-of-life impact, and treatment of psoriasis in non-white racial/ethnic groups. *Am J Clin Dermatol*. 2018;19:405–23. doi:10.1007/s40257-017-0332-7.
4. Filer C, Ho P, Smith RL, Griffiths C, Young HS, Worthington J, et al. Investigation of association of the IL12B and IL23R genes with psoriatic arthritis. *Arthritis Rheum*. 2008;58(12):3705–9. doi:10.1002/art.24128.
5. Zhu KJ, Zhu CY, Shi G, Fan YM. Meta-analysis of IL12B polymorphisms (rs3212227, rs6887695) with psoriasis and psoriatic arthritis. *Rheumatol Int*. 2013;33(7):1785–90. doi:10.1007/s00296-012-2637-4.
6. Sandoval-Talamantes AK, Brito-Luna MJ, Fafutis-Morris M, Villanueva-Quintero DG, Graciano-Machuca O, Ramírez-Dueñas MG, et al. The 3'UTR 1188A/C polymorphism of IL12B is not associated with susceptibility to plaque psoriasis in a mestizo population from western Mexico. *Immunol Lett*. 2015;163(2):221–6. doi:10.1016/j.imlet.2014.10.004.
7. Li XL, Wu CF, Wu GS. Genetic variations of cytokines and cytokine receptors in psoriasis patients from China. *Int J Genomics*. 2014;2014:870597. doi:10.1155/2014/870597.
8. Chang EY, Chen KC, Huang BK, Kavanaugh A. Adult inflammatory arthritides: what the radiologist should know. *Radiographics*. 2016;36(6):1849–70. doi:10.1148/rg.2016160011.
9. Taljanovic MS, Melville DM, Gimber LH, Scalcione LR, Miller MD, Kwok CK, et al. High-resolution US of rheumatologic diseases. *Radiographics*. 2015;35(7):2026–48. doi:10.1148/rg.2015140250.
10. Tsoi LC, Spain SL, Knight J, Ellinghaus E, Stuart PE, Capon F, et al. Identification of 15 new psoriasis susceptibility loci highlights the role of innate immunity. *Nat Genet*. 2012;44(12):1341–8. doi:10.1038/ng.2467.
11. Bustin SA, Mueller R, Nolan T. Parameters for successful PCR primer design. *Methods Mol Biol*. 2020;2065:5–22. doi:10.1007/978-1-4939-9833-3_2.
12. Elbrecht V, Hebert PDN, Steinke D. Slippage of degenerate primers can cause variation in amplicon length. *Sci Rep*. 2018;8(1):10999. doi:10.1038/s41598-018-29364-z.

Figures, tables and additional files

Table 1: Sociodemographic characteristics of the study population treated at INDERMA (n = 35)

Variable	Mean \pm SD / n (%)
Age (years)	44.9 \pm 14.4
Age disease at onset (years)	32.2 \pm 13.4
Sex	
Female	20 (57.1%)
Male	15 (42.9%)
Occupation	
Household work (homemaker)	9 (25.7%)
Teaching (teacher, professor)	5 (14.3%)
Students	3 (8.6%)
Self-employed or technical workers	12 (34.3%)
Retired	2 (5.7%)
Office and service workers	2 (5.7%)
Professionals (architect, engineer)	2 (5.7%)
Place of birth	
Guatemala City	25 (71.4%)
Other departments	10 (28.6%)

Table 2: Clinical characteristics of patients with plaque psoriasis treated at INDERMA (n = 35)

Variable	n (%)
Disease duration	
Acute (< 2 weeks)	1 (2.9%)
Subacute (2–4 weeks)	1 (2.9%)
Chronic (> 4 weeks)	33 (94.3%)
Clinical course of psoriasis	
Progressive	15 (42.9%)
Intermittent	12 (34.3%)
Stable	8 (22.9%)
Family history of psoriasis	
Yes	10 (28.6%)
No	25 (71.4%)

Table 3: Evaluation of psoriatic arthritis in patients with plaque psoriasis treated at INDERMA (n = 35)

Variable	n (%)
Presence of arthritis on ultrasound	
Yes	18 (51.4%)
No	3 (8.6%)
Unknown	14 (40.0%)
Rheumatoid factor	
Positive	6 (17.1%)
Negative	17 (48.6%)
Unknown	12 (34.3%)

Table 4: Genotype and allele frequencies in patients with plaque psoriasis treated at INDERMA (n = 9)

Genotype	Frequency n (%)
TT (normal homozygous)	8 (88.9)
GG (variant homozygous)	1 (11.1)
TG (variant heterozygous)	0 (0.0)
Allele	
T	16 (88.9)
G	2 (11.1)

Table 5: Association between psoriasis severity and SNP rs3212227 of the IL12B gene in patients with plaque psoriasis treated at INDERMA (n = 9)

Severity (PASI)	Genotype TT	Genotype GG	Total
Mild (PASI < 10)	2	0	2
Moderate (PASI 10–20)	5	1	6
Severe (PASI > 20)	1	0	1

Fisher's exact test: p = 1.000.

Table 6: Association between the presence of psoriatic arthritis and SNP rs3212227 of the IL12B gene in patients with plaque psoriasis treated at INDERMA (n = 9)

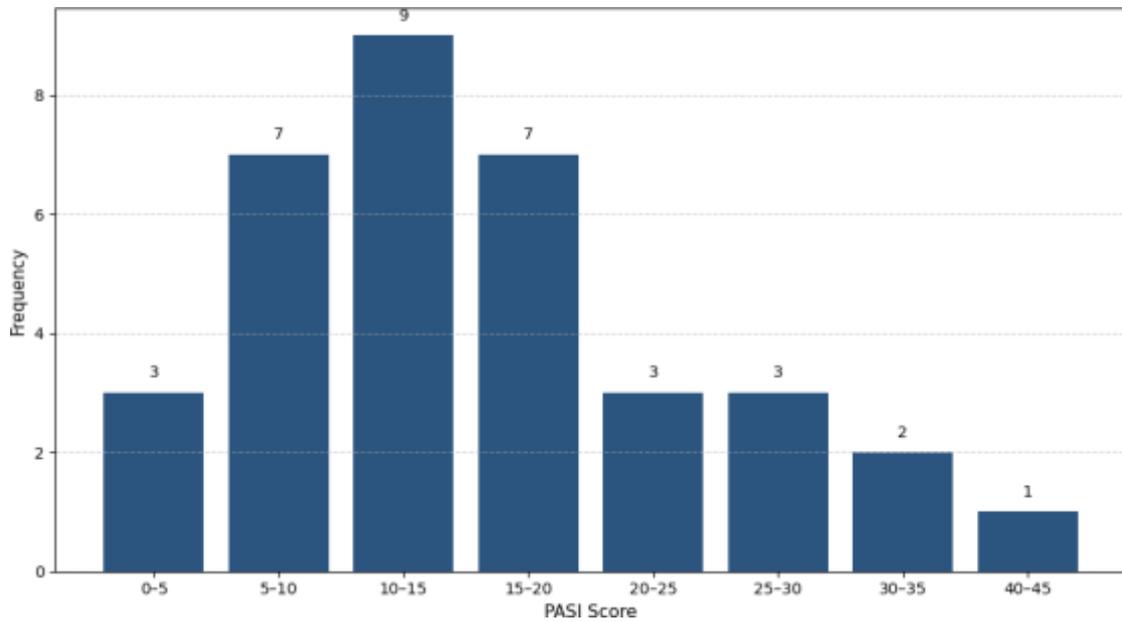
Psoriatic arthritis	Genotype TT	Genotype GG	Total
Present	3	1	4
Absent	5	0	5
Total	8	1	9

Chi-square test of independence: $\chi^2 = 0.41$, p = 0.52.

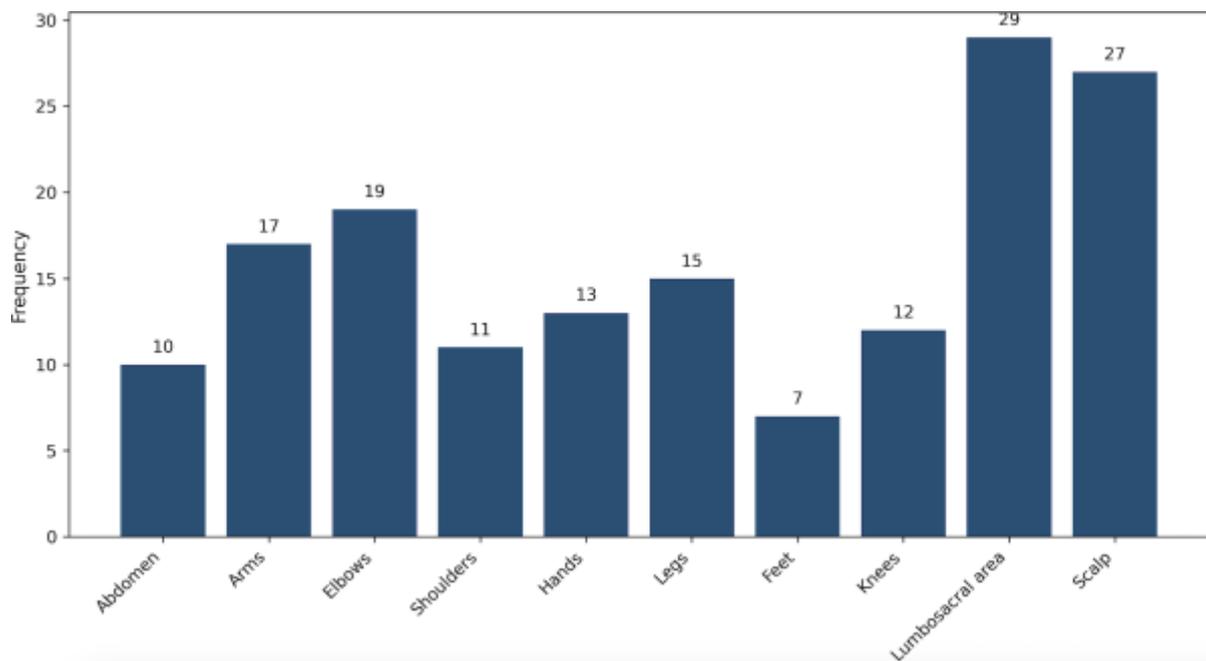
Table 7: Comparison of age of onset of psoriasis according to SNP rs3212227 of the IL12B gene in patients with plaque psoriasis treated at INDERMA (n = 9)

Genotype	n	Mean (years)	SD
TT	8	32.5	12.8
GG	1	30.0	–
Total	9	32.2	13.4

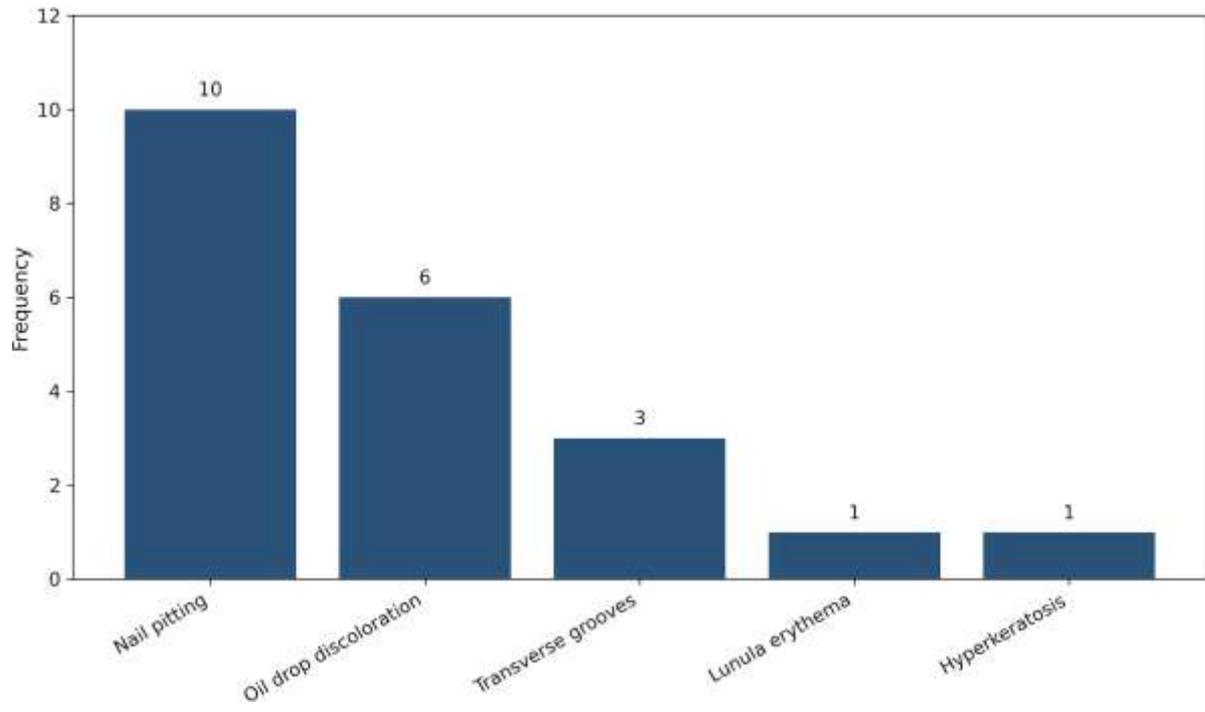
Student's t-test: t = 0.25, p = 0.81.



Graph 1: PASI score distribution among patients with plaque psoriasis at INDERMA (n = 35)



Graph 2: Body areas affected in patients with plaque psoriasis treated at INDERMA (n = 35)



Graph 3: Subtypes of nail psoriasis in patients with plaque psoriasis treated at INDERMA (n = 13)

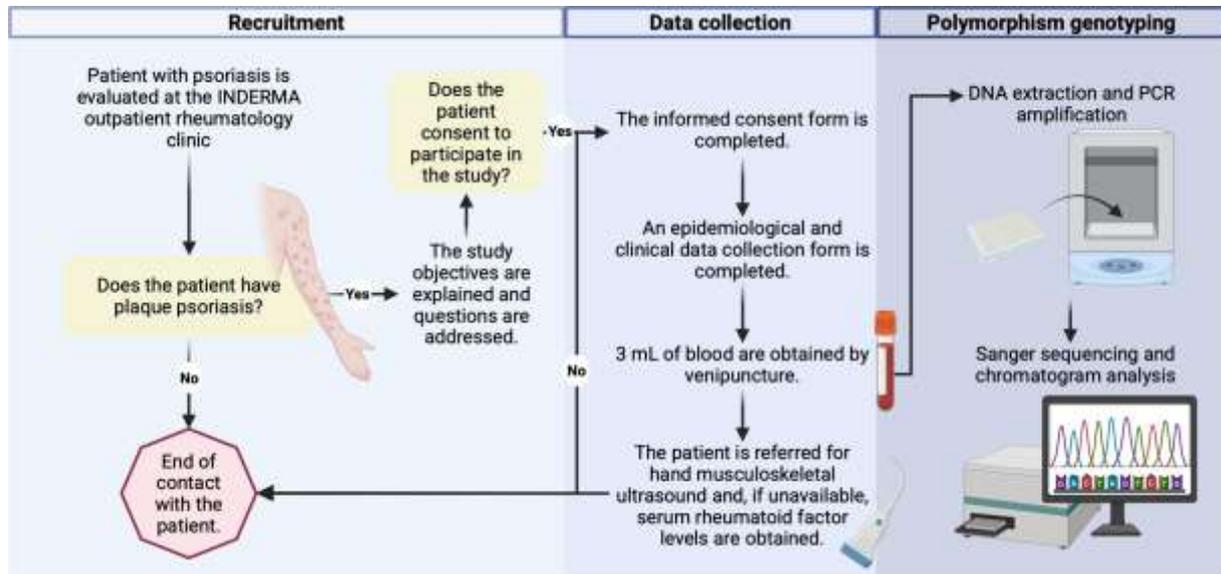


Figure 1: Study workflow

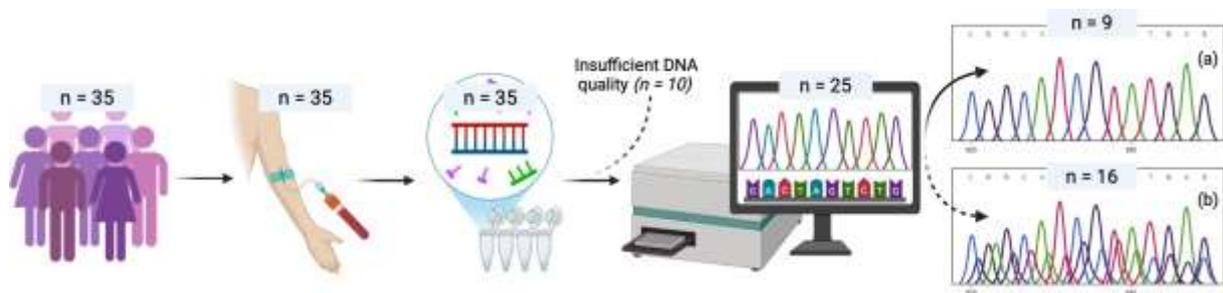


Figure 2: Inclusion and exclusion of samples for molecular analysis

Of the 35 initial samples, 10 were excluded because the DNA was not suitable for Sanger sequencing. Among the 25 processed samples, 9 yielded sequencing of sufficient quality to allow genotyping, whereas 16 exhibited low-quality reads with overlapping peaks and high background noise in the chromatograms.



Figure 3: Clinical manifestations in patients with plaque psoriasis treated at INDERMA
(A–C) Calves: (A) Hyperpigmented, lichenified lesions with fine scaling; (B) Erythematous plaques with thick scales and areas of central remission; (C) Active erythematous plaques with intense scaling and a positive Auspitz sign. **(D–E) Dorsum of the hand:** (D) Plaques with marked erythema, scaling, and periungual involvement; (E) Scaly plaques with palpable synovial thickening of the distal interphalangeal (DIP) joint of the third finger, and fixation of the DIP joints of the second, fourth, and fifth fingers. **(F–G) Anterior chest and upper limbs:** Confluent erythematous plaques with well-defined borders and fine surface scaling.



Figure 4: Nail psoriasis manifestations in patients with plaque psoriasis treated at INDERMA (n = 13) (A) Nail pitting, (B) nail pitting and lunula erythema, (C) nail pitting, oil spots, and onycholysis, (D) nail pitting and active erythematous-scaly psoriasis plaques on the fingers, (E) nail pitting, oil spot, subungual hyperkeratosis, onycholysis, and periungual scaling.

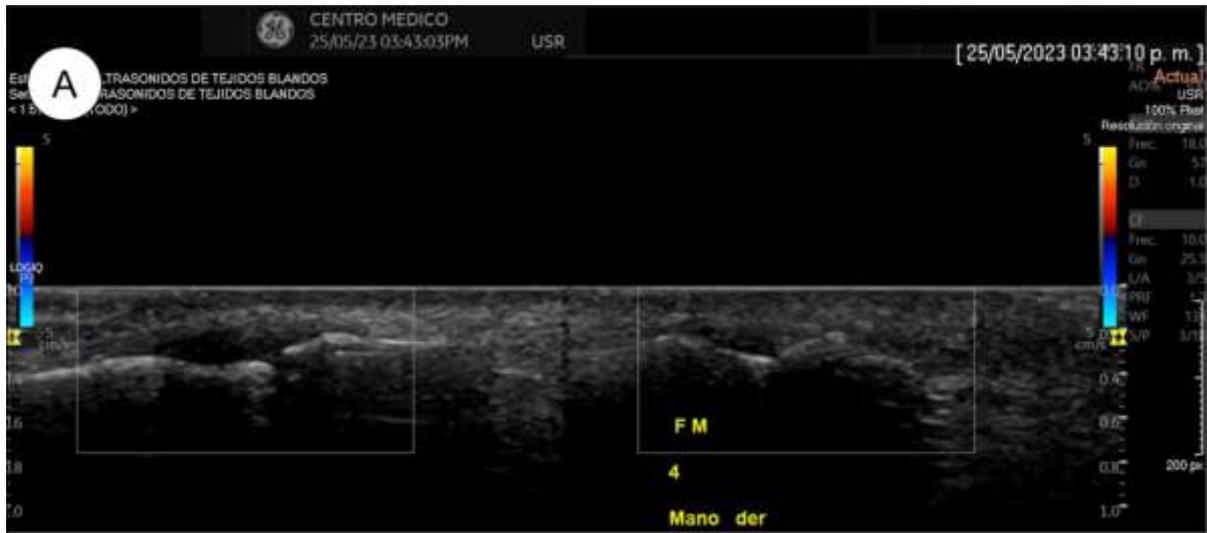


Figure 5: **Ultrasonographic findings in patients with plaque psoriasis at INDERMA** A) Longitudinal B-mode and color Doppler images of the right hand showing synovial thickening in the fourth metacarpophalangeal joint, with mild cortical irregularity.

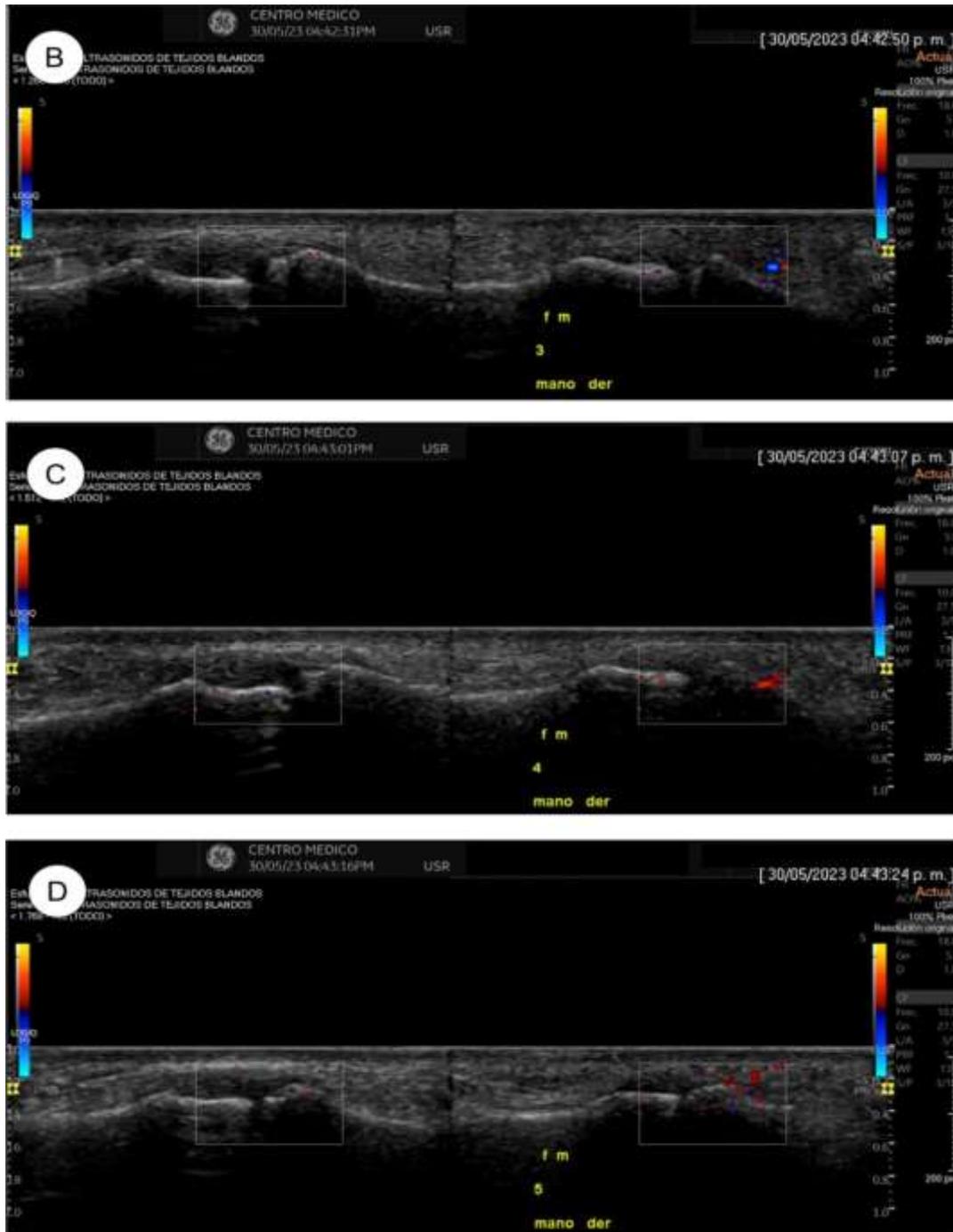


Figure 6: **Ultrasonographic findings in patients with plaque psoriasis at INDERMA (B–D)** Longitudinal B- mode and color Doppler images of the right hand: synovial thickening and increased color Doppler signal, indicative of active synovitis in the third (B), fourth (C), and fifth (D) fingers.

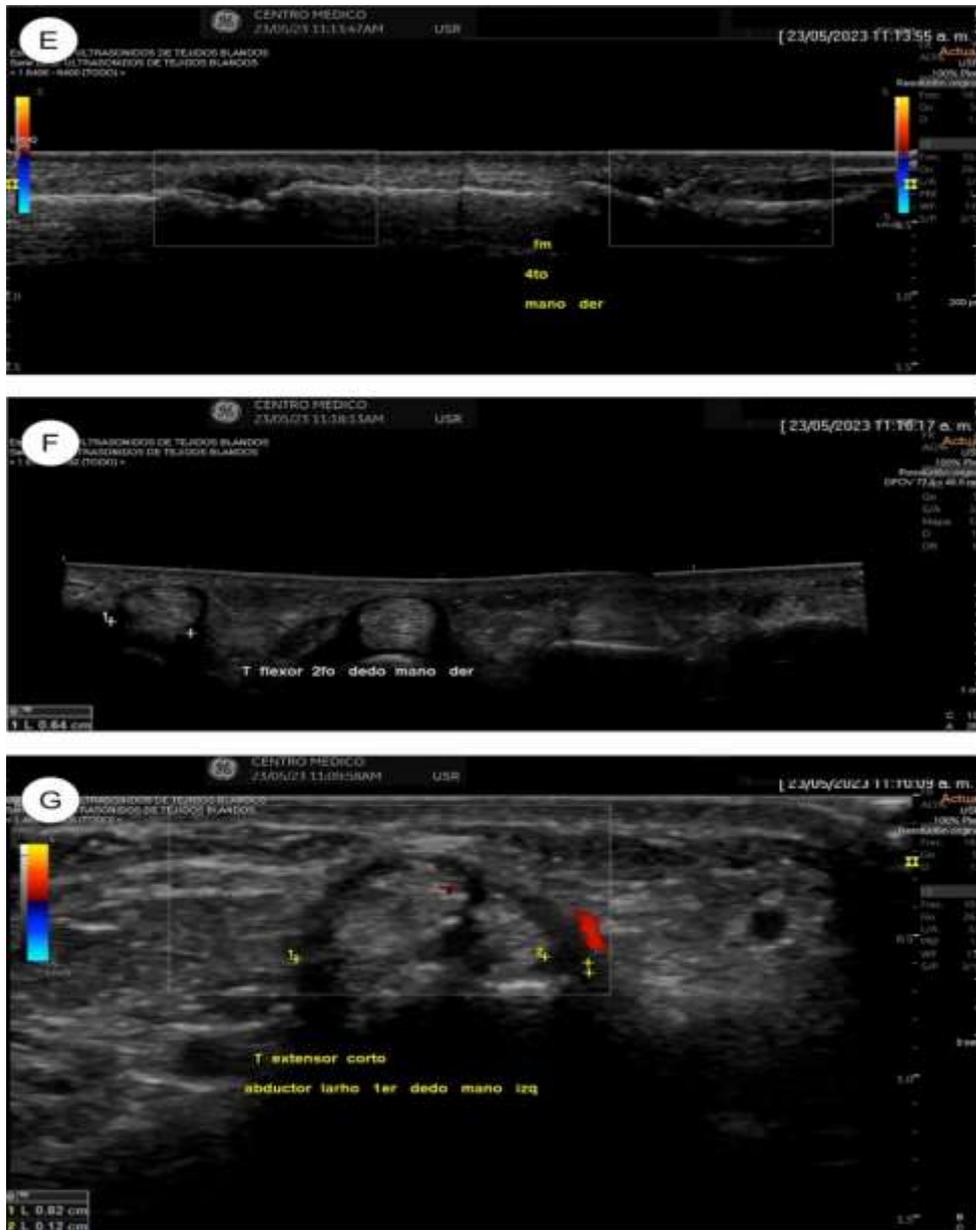


Figure 7: **Ultrasonographic findings in patients with plaque psoriasis at INDERMA** (E–G) B-mode and color Doppler ultrasound images: (E–F) Right hand: synovial thickening in the interphalangeal joint of the fourth finger and a hypoechoic peritendinous collection with surrounding thickening in the flexor tendon of the second finger, indicative of synovitis (E) and tenosynovitis (F). (G) Left hand: thickening of the sheath of the extensor pollicis brevis and abductor pollicis longus tendons, with increased Doppler signal, indicative of tenosynovitis.

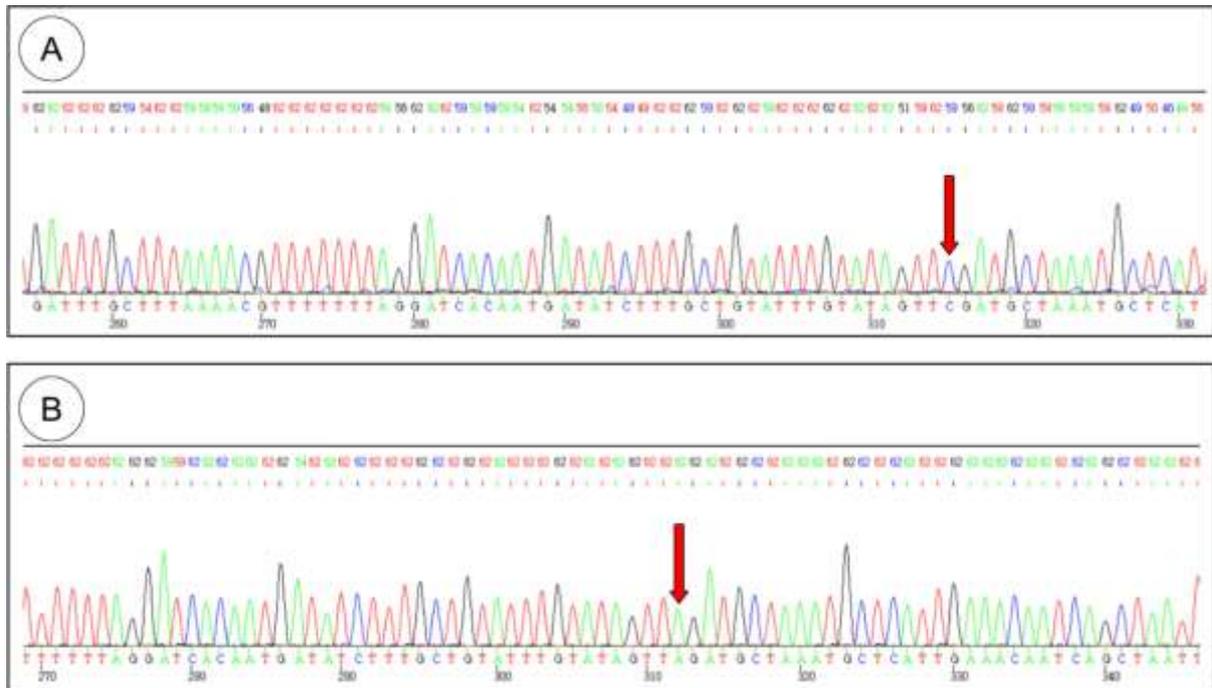


Figure 8: Sanger sequencing chromatograms of the IL12B gene (SNP rs3212227) in high-quality samples from patients with plaque psoriasis treated at INDERMA (n = 9)

(A–B) Reverse strand readings showing the flanking motif TAGTT. The high-resolution peaks and absence of background noise indicate good sequencing quality. The presence of a single peak confirms a homozygous genotype. (A) Homozygous genotype CC (G allele): TAGTT motif followed by a C base at position 315 of the SNP rs3212227. The C base observed in the chromatogram complements the G allele on the forward strand, confirming the presence of the variant allele. (B) Homozygous genotype AA (T allele): TAGTT motif followed by an A base at position 312 of the SNP rs3212227. The A base complements the T allele on the forward strand, confirming the presence of the normal (non-polymorphic) allele.