CURRENT EVIDENCE ON *FLG*, *IL4Rα* AND *CLDN-1* GENETIC VARIANTS IN ATOPIC DERMATITIS IN THE VIETNAMESE POPULATION: A REVIEW STUDY

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This narrative review was conducted to summarize the currently available evidence on FLG, IL4Rα and CLDN1 genetic variants in atopic dermatitis in the Vietnamese population, based on studies published between 2019 and 2025. The review focused on genotype distribution, allele frequencies and reported associations between genetic variants and clinical characteristics. Available evidence suggests that skin barrier-related genes, particularly FLG and CLDN1, may be associated with epidermal barrier dysfunction, disease severity and family history. The spectrum of FLG variants in the Vietnamese population also appears to be genetically diverse. For IL4Rα, the variants rs1801275 and rs1805010 have not shown a clear association with disease susceptibility, but may be related to disease severity as measured by the SCORAD index and to age of onset. Overall, current findings provide preliminary evidence that genetic factors may contribute to the clinical heterogeneity of atopic dermatitis in the Vietnamese population.

Keywords: Atopic dermatitis, eczema, Vietnamese, FLG, filaggrin.

I. INTRODUCTION

Atopic dermatitis is a chronic, pruritic, relapsing inflammatory skin disorder that typically has an early onset but may persist into or present in adulthood. The disease is heterogeneous, characterized by epidermal barrier dysfunction, activation of type 2 inflammatory pathways and complex interactions between host susceptibility and environmental exposures.[1,2] It is among the most common chronic dermatologic conditions worldwide, with a higher prevalence in children but a substantial burden in adults.[1-3] Disease burden varies by age, sex and geographic region and increasing trends have been reported across multiple populations, with prevalence in individuals aged ≥16 years approaching 10% in certain regions.[3,4] In Vietnam, available surveys also suggest a meaningful epidemiologic impact, with reported prevalence of approximately 15.3% in young children and 6.7% in adults.[5]

The pathogenesis of atopic dermatitis cannot be fully explained by environmental factors alone. Current evidence supports a central role for genetic determinants, interacting with allergens, microbial factors, pollution and climate in the initiation and persistence of disease.[1] From a mechanistic perspective, genes involved in epidermal barrier integrity and immune regulation represent two major axes. The *FLG* gene encodes filaggrin, a key protein in epidermal differentiation and stratum corneum integrity. Loss-of-function variants in *FLG* lead to increased transepidermal water loss, altered skin surface pH, reduced natural moisturizing factors and enhanced susceptibility to inflammation and allergen penetration.[1,6] In addition, the *CLDN1* gene encodes claudin-1,

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a critical component of tight junctions. Reduced expression or genetic variation in *CLDN1* may compromise intercellular barrier function, thereby promoting chronic inflammation and increasing cutaneous sensitivity.[7-9] Meanwhile, *IL4Rα* is a key mediator within the IL-4/IL-13 signaling axis, influencing the magnitude of Th2 responses and IgE production. Variants in *IL4Rα* may contribute to phenotypic heterogeneity by modulating disease severity, age at onset and associated atopic manifestations.[1,5,10]

Globally, genetic studies of atopic dermatitis have expanded from candidate gene investigations to multi-ethnic genome-wide association studies and integrative multi-omics approaches, providing a broader view of disease susceptibility and biological heterogeneity.[11,12] However, evidence from Southeast Asia remains limited, and data from Vietnam are still sparse. Available Vietnamese studies on *FLG, IL4Rα* and *CLDN1* are few, mostly small in scale and methodologically heterogeneous, which limits cross-study comparison and weakens conclusions in the local setting. Accordingly, this narrative review aims to synthesize published Vietnamese evidence on these genetic variants and to identify key gaps for future research.

II. OVERVIEW

Study design and methods

This narrative review was conducted to summarize the published evidence on *FLG, IL4Rα* and *CLDN1* genetic variants in Vietnamese patients with atopic dermatitis. Studies published between 2019 and 2025 were considered for inclusion.

Databases and search strategy

A structured literature search was performed using PubMed, Google Scholar and ResearchGate to identify relevant studies. The search strategy combined controlled vocabulary and free-text terms, including: "atopic dermatitis", "eczema", "Vietnamese", "*FLG*", "filaggrin", "*IL4R*", "IL4RA", "rs1801275", "rs1805010", "*CLDN1*", "rs17501010", "rs9290927", "rs893051", "rs9290929", "genetics" and "skin barrier". The search focused on domestic studies in order to summarize the currently available evidence and to characterize the genetic and clinical features of atopic dermatitis in the Vietnamese population.

Eligibility criteria

Studies were considered eligible if they met the following criteria:

- Participants were Vietnamese individuals or the study provided background data on *FLG, IL4Rα* and *CLDN1* variants in the Vietnamese population.

- The study content was directly related to atopic dermatitis or to molecular and laboratory methodologies applicable to atopic dermatitis research.

- Study designs included descriptive studies, case–control studies, cross-sectional studies, population genetic analyses, or methodological studies focused on assay development.

- At least one outcome related to genotype, allele frequency, variant spectrum, or associations between genetic variants and clinical characteristics was reported.

- The study clearly described the population, sample size, investigated genes or variants and main findings.

Exclusion criteria

Studies were excluded if they met any of the following:

- Not focused on atopic dermatitis or not related to its genetic or molecular basis.

- Did not report genotype data, allele frequencies, variant distributions, or relevant genetic findings.

- Lacked a clear description of the study population or essential methodological details.

- Review articles, expert opinions, editorials, or conference abstracts without original data.

Selection process

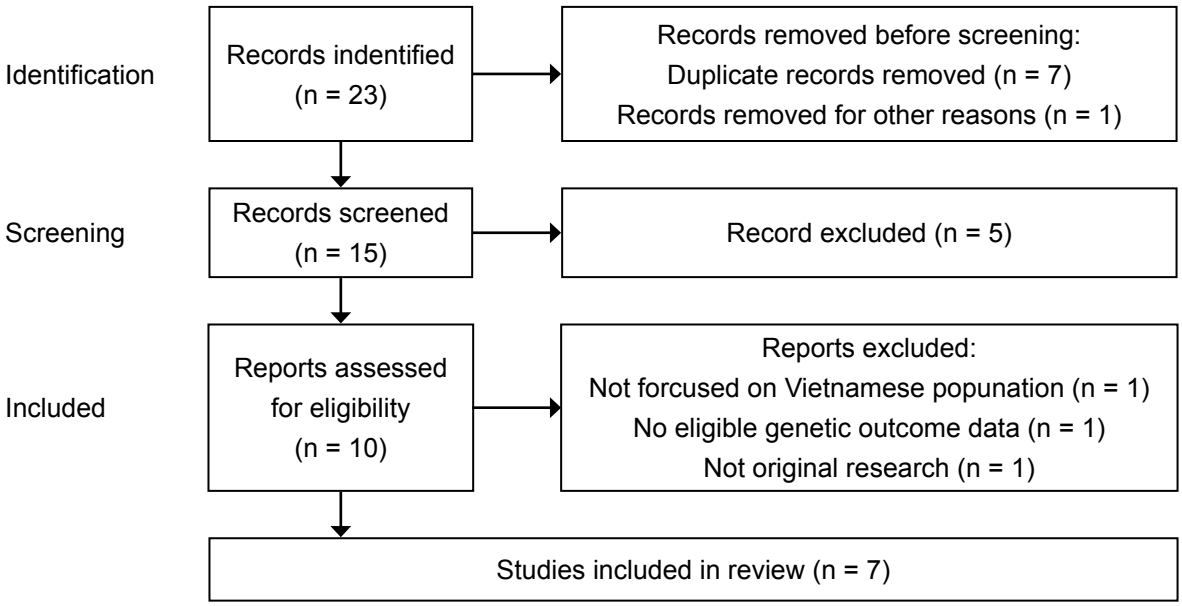

Figure 1. Flowchart of study selection process

Study selection was conducted in three stages: identification, screening and full-text assessment. A total of 23 records were identified through PubMed, Google Scholar and ResearchGate. After removal of 7 duplicate records and 1 record excluded for other reasons, 15 records underwent title and abstract screening, of which 5 were excluded. The remaining 10 reports were assessed in full text. Three reports were excluded after full-text review because they were not focused on the Vietnamese population, did not report eligible genetic outcome data or were not original research. Finally, 7 studies were included in the review. Given the limited number of eligible domestic studies, the available evidence should be interpreted as an initial synthesis rather than a comprehensive estimate of all possible genetic associations.

Data extraction

From each included study, the following data were extracted:

- Characteristics of the study population
- Genes or genetic variants investigated
- Genotyping techniques or laboratory methods used
- Reported clinical parameters
- Main findings regarding associations between genetic variants and disease characteristics

Quality appraisal

As this study was designed as a narrative review with a structured literature search, a formal risk-of-bias assessment using standardized appraisal tools was not performed. However, the included studies were reviewed descriptively with attention to sample size, study design, population characteristics, genetic methods and clarity of reported outcomes. These methodological features were taken into account when interpreting the strength and consistency of the available evidence.

II. RESULTS

Table 1. Studies included in the review

Author (year)	Population	Objective	Results
Dang Thi Hong Phuong, Le Thai Van Thanh (2019)	16 patients with atopic dermatitis at Ho Chi Minh City Hospital of Dermato-Venereology	To identify *FLG* mutation patterns and evaluate associated factors in patients with atopic dermatitis	The prevalence of *FLG* mutations was 31.25% (5/16). Five loss-of-function variants were identified: c.1217C>G, c.6950del8, c.7189C>T, c.7249C>T and c.7264G>T. Mutation frequency was significantly higher in patients with xerosis (80% vs 9%, p = 0.013), palmar hyperlinearity (66.7% vs 10%, p = 0.036) and hand involvement (66.7% vs 10%, p = 0.036). No significant differences were observed by age of onset, atopic history, or SCORAD score.
Vu PN, Nguyen HG, Nguyen THN, et al. (2022)	244 Vietnamese individuals in a whole-exome sequencing study.	To construct the *FLG* variant spectrum in the Vietnamese population using whole-exome sequencing.	A total of 126 missense variants, 6 nonsense variants, 6 frameshift indels and 1 in-frame indel were identified. Eleven novel variants were reported. Potentially pathogenic variants were predominantly nonsense mutations, mainly located in exon 3, indicating high diversity of *FLG* variants in the Vietnamese population.
Nguyen THN, Le DHH, Huynh TTM, et al. (2025)	113 patients with atopic dermatitis and 213 healthy controls in Ho Chi Minh City	To evaluate the frequency and clinical significance of *IL4Rα* rs1801275 in Vietnamese patients with atopic dermatitis.	Allele frequencies in cases were A: 82.74%, G: 17.26% and in controls A: 79.58%, G: 20.42%, with no significant difference (p = 0.315), suggesting no association with disease susceptibility. The G allele was associated with higher SCORAD severity under a dominant model (AG+GG vs AA: median 40 vs 30.5, p = 0.010; OR = 4.67, p = 0.005). The additive model was also significant (p = 0.023), indicating a gene-dose effect (AA = 30.5; AG = 39; GG = 49.65).

Author (year)	Population	Objective	Results
Nguyen Huu Ngoc Tuan, Huynh Thi Mai Thi, Le Duong Hoang Huy, Chau Van Tro (2025)	113 Vietnamese patients with atopic dermatitis	To investigate the association between *IL4Rα* rs1805010 polymorphism and age of onset and disease severity	More than 50% of participants were <12 years, with a female-to-male ratio of 1.26:1. Genotype frequencies were AA 20.35%, AG 52.21%, GG 27.44%, consistent with Hardy–Weinberg equilibrium (p = 0.94). The rs1805010 variant was associated with age of onset under codominant (p = 0.037) and dominant models (p = 0.019). The GG genotype was predominant in early-onset cases (<2 years), whereas AG was more frequent in late-onset (>12 years). Disease severity (SCORAD) also differed significantly (p = 0.018), particularly in the AG genotype.
Nguyen Huu Ngoc Tuan, Le Duong Hoang Huy, Tran Le Hoang (2024)	82 adult patients with atopic dermatitis	To determine the frequency of four *CLDN1* polymorphisms in adult patients with atopic dermatitis	Median age was 36 years, with 59.76% males. Allele and genotype frequencies were reported as follows: rs17501010 (G: 95.73%, T: 4.27%; GG: 91.46%, GT: 8.54%), rs9290927 (A: 50.61%, T: 49.39%; AA: 26.83%, AT: 47.56%, TT: 25.61%), rs893051 (C: 71.95%, G: 28.05%; CC: 54.88%, CG: 34.15%, GG: 10.98%) and rs9290929 (A: 91.46%, G: 8.54%; AA: 82.93%, AG: 17.07%). This study provides initial baseline data on these single nucleotide polymorphisms (SNPs) in Vietnamese adults.
Nguyen Huu Ngoc Tuan, Huynh Thi Mai Thi, Chau Van Tro (2024)	82 adult patients with atopic dermatitis	To assess the association between *CLDN1* rs17501010 and clinical characteristics in adult atopic dermatitis	Personal history of atopic dermatitis, asthma and allergic rhinitis was 84.15%, 8.54% and 15.85%, respectively; family history was 41.46%, 4.88% and 15.85%. SCORAD severity distribution was mild 13.41%, moderate 50.00% and severe 36.59%. Allele frequencies were G: 95.73%, T: 4.27%; genotype GG: 91.46%, GT: 8.54%, with no TT observed. The variant was significantly associated with disease severity when comparing mild–moderate versus severe groups (p = 0.045).

Author (year)	Population	Objective	Results
Nguyen Huu Ngoc Tuan, Le Duong Hoang Huy, Chau Quoc Khanh, Chau Van Tro (2024)	82 adult patients with atopic dermatitis	To describe clinical features and genotype distribution of *CLDN1* rs9290927 in atopic dermatitis	Median age was 36 years, with 40.24% females. Clinical characteristics were similar to related cohorts. Allele frequencies were A: 50.61%, T: 49.39%; genotype AA: 26.83%, AT: 47.56%, TT: 25.61%. The rs9290927 variant was significantly associated with family history of atopic dermatitis under codominant, recessive and overdominant models (p = 0.002, 0.016 and 0.0004, respectively). Under the overdominant model, individuals with genotype AT had a 5.28-fold higher likelihood of having a family history compared with other genotypes (OR = 5.28; 95% CI: 2.0–13.75).

Current Vietnamese studies have focused on three main gene groups: *FLG, IL4Rα* and *CLDN1*. The earliest Vietnamese study on *FLG* was a case series by Dang Thi Hong Phuong and colleagues at the Ho Chi Minh City Hospital of Dermato-Venereology, including 16 patients with atopic dermatitis. The prevalence of *FLG* mutations was 31.25% and five loss-of-function variants were identified: c.1217C>G, c.6950del8, c.7189C>T, c.7249C>T and c.7264G>T. These variants were significantly associated with palmar hyperlinearity, xerosis and hand involvement.[6] In parallel, a whole-exome sequencing study in 244 Vietnamese individuals established the *FLG* variant spectrum in the population and identified 126 missense variants, 6 nonsense variants, 6 frameshift indels and 1 in-frame indel, including 11 novel variants. Notably, the potentially pathogenic variants were predominantly nonsense variants, most of which were located in exon 3.[13]

With regard to *IL4Rα*, a case-control study by Nguyen Huu Ngoc Tuan and colleagues in 2025, including 113 patients with atopic dermatitis and 213 healthy controls in Ho Chi Minh City, showed that rs1801275 was not significantly associated with disease susceptibility, as the A/G allele frequencies were 82.74%/17.26% in cases and 79.58%/20.42% in controls (p = 0.315).[5] However, the G allele was associated with greater disease severity as assessed by the SCORAD index under both the dominant and additive models, with evidence of a gene-dose trend across the AA, AG and GG genotypes. Another study in 113 Vietnamese patients evaluating rs1805010 reported genotype frequencies of 20.35% for AA, 52.21% for AG and 27.44% for GG, with Hardy-Weinberg equilibrium maintained (p = 0.94). This variant was significantly associated with both age at onset and disease severity

according to SCORAD. The GG genotype was more frequent in patients with onset before 2 years of age, whereas AG was more common among those with onset after 12 years.[10]

For *CLDN1*, a 2024 study by Nguyen Huu Ngoc Tuan and colleagues in 82 adults with atopic dermatitis determined the frequencies of four SNPs: rs17501010, rs9290927, rs893051 and rs9290929. Allele frequencies were G/T = 95.73%/4.27% for rs17501010, A/T = 50.61%/49.39% for rs9290927, C/G = 71.95%/28.05% for rs893051 and A/G = 91.46%/8.54% for rs9290929, all of which were reported to conform to Hardy-Weinberg equilibrium.[8] In the variant-specific analyses, rs17501010 in 82 adults was significantly associated with disease severity according to SCORAD when the mild-to-moderate group was compared with the severe group ($p = 0.045$).[14] The rs9290927 variant in the same population was significantly associated with family history of atopic dermatitis across multiple genetic models.[7]

Overall, the currently available Vietnamese studies suggest two relatively consistent patterns. First, skin barrier genes, particularly *FLG* and *CLDN1*, appear to be more strongly associated with barrier-related phenotypic features, family history, or disease severity.[6-8,14] Second, the immune-related gene *IL4Rα* appears to be more closely associated with disease severity and age at onset than with overall disease susceptibility.[5,10]

III. DISCUSSION

Based on the available findings, current evidence suggests that the pathogenesis of atopic dermatitis in the Vietnamese population reflects the classical dual-axis model of the disease, comprising the epidermal barrier axis and the type 2 immune axis. Along the barrier axis, studies on *FLG* and *CLDN1* indicate that epidermal structural dysfunction is not merely a secondary consequence of inflammation, but likely represents a fundamental component of disease phenotype in specific patient subsets.[1,6-8,14] This is consistent with current understanding that filaggrin contributes to skin hydration, pH regulation and stratum corneum integrity, whereas claudin-1 is essential for maintaining tight junction function. Disruption of these elements increases cutaneous permeability to allergens, microbes and danger signals, thereby triggering inflammation and perpetuating a pathogenic feedback loop.[1] Such barrier impairment may also interact with cutaneous microbial dysbiosis, particularly through greater susceptibility to Staphylococcus aureus colonization, further aggravating inflammation and disease chronicity. Moreover, recent international evidence indicates that genetic susceptibility in atopic dermatitis may be influenced not only by structural and immune pathways, but also by broader multi-omics and epigenetic mechanisms, including transcriptomic, microbiome and gene-environment interactions.[11,12]

Findings on *FLG* in Vietnamese populations carry two important implications. First, the observed mutation rate of 31.25% in patients, although derived from a small sample, falls within the range reported in other populations.[6] Second, the mutation spectrum differs from the canonical variants described in European populations, indicating substantial ethnic variation in *FLG* variants.[1,6,13] This distinction is critical, as extrapolation from Western datasets may overlook variants relevant to the Vietnamese population. The whole-exome sequencing study in 244 Vietnamese individuals further supports this observation, demonstrating a highly diverse *FLG* variant spectrum, including multiple rare and novel variants.[13] This observation is in line with the

study by Chen et al. in Singaporean Chinese patients, which also showed a broad spectrum of *FLG* null variants distinct from the recurrent European mutations, further supporting marked ethnic heterogeneity in *FLG*-associated atopic dermatitis.[15] Compared with data from European populations, in which a limited number of recurrent *FLG* loss-of-function variants account for a substantial proportion of genetic risk, the Vietnamese data appear to suggest greater allelic heterogeneity. This pattern is consistent with broader international evidence showing that both the frequency and spectrum of *FLG* variants vary markedly across ethnic groups.[11-13] However, the Vietnamese evidence remains preliminary, because the clinical study included only 16 patients and the population sequencing study was not designed specifically to estimate disease risk.

For *IL4Rα*, the pattern of results is more nuanced. The rs1801275 study demonstrated no significant difference between cases and controls in allele or genotype frequencies, but did show an association with disease severity.[5] This suggests that the variant may act as a phenotype modifier rather than a primary susceptibility factor. Similarly, rs1805010 was associated with age at onset and disease severity.[10] From a mechanistic perspective, this is consistent with the biological role of *IL4Rα*. Unlike *FLG* or *CLDN1*, it does not directly contribute to the physical barrier, but instead functions as a key regulator of IL-4/IL-13 signaling, influencing IgE production, eosinophil activation, Th2 differentiation and chronic inflammation.[1] This observation is also compatible with the broader concept that immune-related variants may contribute more strongly to phenotypic expression and endotype variation than to disease occurrence alone. In contemporary multi-omics models of atopic dermatitis, such immune signaling pathways are increasingly interpreted within a network that includes barrier injury, microbial colonization and downstream transcriptional reprogramming, rather than as isolated drivers.[11,12] Nevertheless, both Vietnamese studies on IL4Rα were conducted in relatively small samples and have not yet been independently replicated, so the apparent associations with severity and age at onset should be interpreted as suggestive rather than definitive.

Studies on *CLDN1* suggest that not all polymorphisms confer equivalent biological significance. Aggregate frequency data for four SNPs provide a baseline genetic profile.[8] However, variant-specific analyses reveal that rs17501010 is associated with disease severity, whereas rs9290927 is associated with family history of atopic dermatitis.[7,14] This heterogeneity may reflect differential functional effects among variants within the same gene. Certain variants may alter gene expression or tight junction integrity sufficiently to influence clinical severity, whereas others may represent population stratification or linkage disequilibrium with causal loci. International data similarly demonstrate substantial ethnic variation in both frequency and effect size of *CLDN1* polymorphisms.[7,8,12] The Vietnamese findings are broadly consistent with this international pattern, but the current evidence remains limited by the fact that these analyses were derived from closely related single-center cohorts of 82 adult patients, without external validation or functional confirmation. As a result, the observed associations are better viewed as preliminary signals than as robust evidence of causality.

Variability across Vietnamese studies may be explained by three main factors. First, differences in study populations are notable. Some studies included only adults, whereas studies on IL4Rα included both pediatric and adult patients or had a high proportion

of early-onset cases. Age at onset is closely linked to genetic background and variant effects may therefore differ across age groups. Second, study design differences contribute to heterogeneity. Most domestic studies are single-arm analyses and only the rs1801275 study included a control group.[5] Consequently, the ability to assess disease susceptibility is limited, whereas analyses of disease severity and clinical features are more robust. Third, differences in genotyping strategies are relevant. The *FLG* study employed targeted exon sequencing to identify loss-of-function variants, whereas *IL4Rα* and *CLDN1* studies primarily examined preselected polymorphisms. This approach may fail to detect rare variants with larger effects. Taken together, these differences also explain why formal meta-analysis was not feasible in the present review. The included studies differed substantially in design, target variants, study populations and reported outcomes, precluding direct pooling of effect estimates. However, a structured narrative synthesis suggests two recurring patterns across the Vietnamese literature: barrier-related genes, particularly *FLG* and *CLDN1*, are more often linked to disease severity, family history, or barrier-associated phenotypes, whereas *IL4Rα* appears more closely related to severity modulation and age at onset than to overall disease susceptibility.

Current studies are predominantly single-center, with modest sample sizes, limited phenotypic stratification and no integration of immunologic biomarkers or environmental data. Multigene models evaluating both barrier and immune axes simultaneously are also lacking. In contrast, recent international studies have employed large-scale, multi-ethnic genome-wide approaches, identifying numerous additional susceptibility loci and potential therapeutic targets.[11,12] They have also shown that genetic associations may differ substantially across ancestral groups, underscoring the importance of population-specific data when interpreting candidate variants. In this context, the Vietnamese literature remains at an early stage and is still insufficient to determine whether the observed variant-phenotype relationships are reproducible across regions, age groups, or clinical subtypes. Future research in Vietnam should therefore prioritize multicenter studies with larger sample sizes, inclusion of healthy controls, standardized phenotyping and integration of genetic data with clinical indices such as SCORAD, age at onset, atopic history, serum IgE levels, eosinophil counts, skin microbiome profiles and response to targeted therapies such as dupilumab. This narrative review has several limitations. First, only seven studies met the eligibility criteria, reflecting the still limited volume of published genetic research on atopic dermatitis in the Vietnamese population. Although multiple sources were searched, the small number of included studies raises the possibility of incomplete capture of relevant evidence and selection bias. Second, a formal risk-of-bias assessment was not performed, as this work was designed as a narrative review rather than a systematic review. Third, most associations have been reported only once and lack replication in independent Vietnamese cohorts, which substantially limits confidence in the consistency of the observed signals. Fourth, because the available studies reported heterogeneous outcomes and rarely provided directly comparable effect estimates, the present review could not perform a quantitative meta-analysis and instead relied on structured narrative and semi-quantitative synthesis. Nevertheless, this review provides a structured summary of the currently available Vietnamese evidence and highlights important gaps for future research.

III. CONCLUSION

Current evidence provides preliminary support for a role of genetic factors in the clinical heterogeneity of atopic dermatitis in the Vietnamese population, particularly for variants in *FLG, CLDN1* and *IL4Rα*. However, the available data remain limited and heterogeneous, and the reported associations should be interpreted with caution. Further well-designed multicenter studies are needed to confirm these findings and to better characterize the genetic background of atopic dermatitis in Vietnam.

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