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Atopic Dermatitis in Asians: A Review on Genetics, Clinical Presentation, and Therapeutic Implications

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Introduction

Atopic dermatitis (AD) is a common, multifactorial, and pruritic inflammatory skin condition with a chronic relapsing course. It typically manifests in infancy or early childhood and is often associated with other atopic conditions, such as asthma and allergic rhinoconjunctivitis. As the most prevalent chronic inflammatory skin disease worldwide, AD affects individuals across a wide range of racial and ethnic backgrounds. Recent studies suggest that Central Asia has the highest pediatric prevalence of AD in 2021, with a rate of 10.5%, surpassing the 5.4% prevalence observed in high-income North America.[1] According to the 2021 Census, individuals of Asian descent constitute the third-largest population group in Canada, following those of European and North American descent, with nearly half originating from East or Southeast Asia. Furthermore, Asians represent Canada's fastest-growing demographic. This review highlights the genetic factors, clinical presentations, and therapeutic

considerations for AD in Asian populations, aiming to improve understanding and inform tailored treatment approaches.

Epidemiology

Ethnic diversity across Asia is vast, encompassing East Asians (EAs), South Asians (SAs), and Southeast Asians (SEAs). A recent population study in Singapore estimated AD prevalence rates of 9.2% in EAs, 8.5% in SAs, and 8.4% in SEAs.[2] A 2025 study on Asian American and Pacific Islander children found an overall AD prevalence of 6.0%, with significantly higher rates among Filipino (12.8%), Chinese (12.0%), Vietnamese (11.7%), Native Hawaiian/Pacific Islander (9.5%), South Asian (8.4%), and Black (8.2%) children compared to non-Hispanic White and Hispanic children.[3] These disparities highlight the need for further research into the genetic and immune mechanisms contributing to increased AD risk in these populations. Notably, Canadian studies on this topic remain scarce.

Multi-ethnicity is also emerging as an important factor influencing the global prevalence of allergic diseases. A Korean study found that AD prevalence was significantly higher in non-multi-ethnic individuals compared to those from multi-ethnic backgrounds. Furthermore, within multi-ethnic groups, a parent's region of birth had a significant impact on the prevalence of allergic diseases.[4] These findings underscore the need for further investigation into the role of genetic and environmental factors in AD susceptibility across diverse populations, especially within Canada's growing multi-ethnic population.

Genetics

AD is a complex genetic disorder influenced by environmental factors. Among various ethnic groups, loss-of-function variants in the filaggrin (*FLG*) gene represent the strongest genetic risk factor for AD and play a crucial role in its pathogenesis.[5] Studies in Europe report that up to 50% of AD patients carry one or more *FLG* null mutations. In contrast, Asian populations exhibit lower *FLG* mutation frequencies but higher diversity. Reported prevalence rates of *FLG* mutations include 31.4% in Han Chinese, 27.0% in Japanese, 26.0% in Singaporean Chinese, and 15.7% in Koreans.[5,6] Besides the differences in prevalence, *FLG* mutations also show significant population specificity. For example, mutations common in European populations are rarely detected in Asian groups.[5,7] Moreover, *FLG* mutations vary considerably among different Asian subpopulations, including Chinese, Korean, Japanese, and Singaporean individuals.[6,8,9] Genetic diversity analyses further suggest that the geographic distribution of *FLG* mutations reflects prehistoric human migration patterns in East Asia, underscoring their potential as ancestryinformative markers.[10]

Genetic studies of *FLG* mutations have revealed unique associations across different Asian subpopulations. Unlike findings in European populations, *FLG* mutations do not appear to increase the risk of asthma in EAs with AD.[10] In the Han Chinese population, these mutations have been linked to a higher predisposition for food sensitization.[5] Among Korean patients, *FLG* mutations are significantly associated with elevated immunoglobulin E (IgE) levels, palmar hyperlinearity, and a family history of allergic diseases.[8] Among Indian populations, *FLG* mutations have been correlated with more severe hand eczema.[11] However, given the rising prevalence of allergic diseases in India over the past decade, further research on *FLG* mutations in this population is needed.[12]

Beyond *FLG* mutations, AD in East Asian populations has also been linked to loss-of-function mutations in the *SPINK5* gene, which encodes a serine protease inhibitor crucial for maintaining epidermal homeostasis.[13] Moreover, genomic and proteomic analyses suggest that East Asian AD exhibits fundamental differences from its European counterpart.[2] Additionally, studies have identified associations between AD and polymorphisms in immune-related genes such as interleukin *(IL)-4* and *IL-13/IL-13Ra1* in Chinese, Japanese, and Korean populations.[13] These findings highlight the need for a more nuanced understanding of genetic factors contributing to AD across diverse Asian subpopulations.

Immune Polarization

The AD phenotype observed in Asian populations exhibits a unique immunological profile, resembling a blend of both European AD and European psoriasis at the cellular and molecular levels.[13,14] While strong Th2 activation is a universal feature of AD, Asian AD patients also show significant upregulation of Th17 and Th22 pathways.[2,15] Increased Th17 activity is accompanied by a downregulation of the Th1 axis, with higher counts of IL-17–producing cells in Asian AD patients.[2,16] Notably, Th17-related mediators are elevated in the skin but not in peripheral blood, whereas IL-22 levels are increased in both compartments.[15] Histological analysis of lesional skin from Asian AD patients shows greater acanthosis, increased Ki67 expression, and more frequent parakeratosis compared to European AD.[16] These features may be attributed to elevated IL-22 levels, which promote keratinocyte proliferation, migration, and impaired differentiation by inhibiting proteins involved in terminal keratinocyte maturation.[2] Given these distinct immunological and histological differences, further research is needed to determine whether targeting Th2 alone is sufficient for managing all AD phenotypes, particularly those with a stronger Th17 component.

Clinical Presentation

In the United States, individuals identifying as Asian/Pacific Islander and Black are significantly more likely to seek medical care for AD despite generally lower overall healthcare utilization rates. Asian/Pacific Islanders, in particular, are nearly seven times more likely than White individuals to have an office visit resulting in an AD diagnosis.[17] One contributing factor may be the lack of familiarity with AD among Asian populations, compounded by the heterogeneity of clinical manifestations across diverse ethnic backgrounds.

Across all ethnic groups, the most universally prevalent AD features include pruritus, lichenification, and xerosis. However, in EA populations, AD lesions tend to be more sharply-demarcated due to the psoriasiform Th17/Th22 endotype (**Figure 1**).[15,18] Compared to White patients, Asian individuals with AD more frequently exhibit increased scaling and lichenification (**Figure 2**).[13] Perifollicular accentuation is a commonly observed feature, while papular eczema—characterized by small, flat-topped papules—can mimic lichen planus in appearance (**Figure 3**). In individuals with darker skin tones, such as those of SA populations, erythema may present as a violaceous hue or be subtle enough to go unnoticed altogether.[13] Detecting erythema in darker skin requires careful assessment of secondary signs, such as edema, increased skin warmth, and scaling.[13] Clinicians should compare affected areas to the patient's baseline skin tone, while pruritus, excoriations, and skin induration—best visualized with tangential lighting or palpation—serve as additional diagnostic clues. Moreover, patients with darker skin tones are at greater risk for post-inflammatory hyperpigmentation, which can be more distressing than the primary skin manifestations of AD itself.[19]

Beyond these general characteristics, AD phenotypes vary among Asian subpopulations. Studies from SEA populations report higher rates of exudative eczema, truncal involvement, lichenification, and prurigo nodularis.[2] In contrast, EA populations exhibit a higher prevalence of erythroderma, truncal, extensor (**Figure 4**), scalp, and auricular (**Figure 1**) involvement.[2] SA populations, particularly Indian patients, more commonly present with flexural involvement.[2] Environmental and cultural factors further shape the AD phenotype, both within Asia and among Asian immigrant communities worldwide. As a result, additional research is needed to determine whether these reported Asian AD phenotypes remain consistent among Asian Canadians, despite differences in environmental exposures, microbiome composition, and cultural practices. Understanding these variations will be essential for developing more tailored and effective treatment strategies.

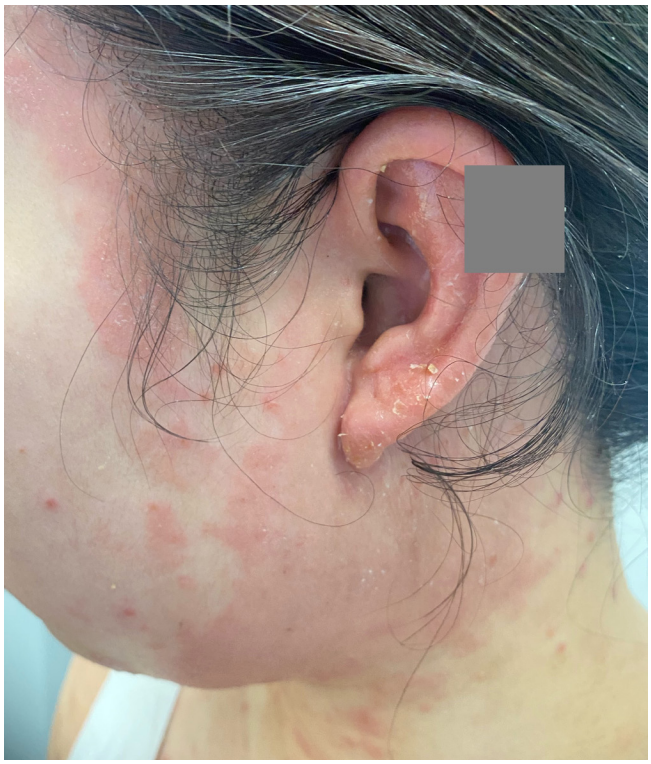

Figure 1. Biopsy-confirmed atopic dermatitis in a young East Asian female, presenting with well-demarcated psoriasiform plaques ranging from pink to erythematous during an acute flare; *courtesy of Harry Liu, MD, FRCPC, FAAD.*

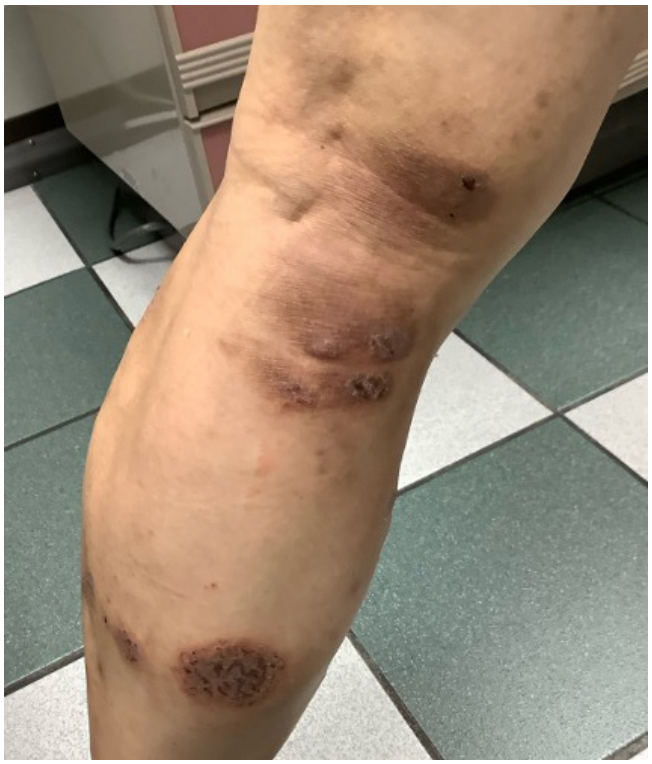

Figure 2. Marked lichenification with well-defined borders in a middle-aged Chinese male with atopic dermatitis; *courtesy of Harry Liu, MD, FRCPC, FAAD.*

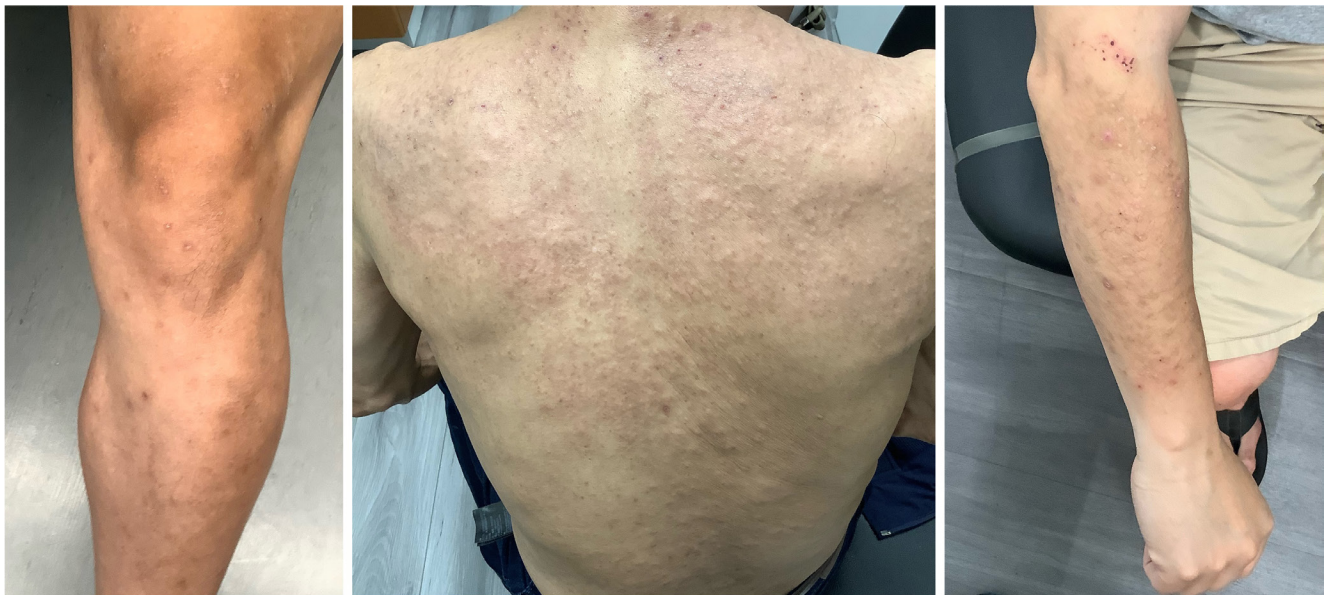

Figure 3. Distinct papular lesions in three different patients with atopic dermatitis (Indian patient on the left, and East Asian patients in the middle and on the right); *courtesy of Harry Liu, MD, FRCPC, FAAD.*

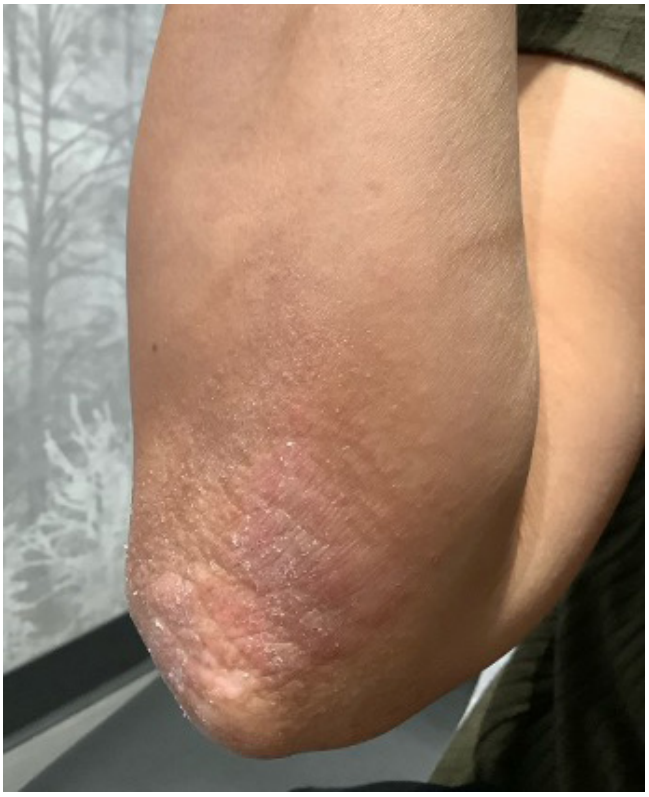

Figure 4. Notable extensor involvement in a young male with atopic dermatitis in a Chinese patient; *courtesy of Harry Liu, MD, FRCPC, FAAD*.

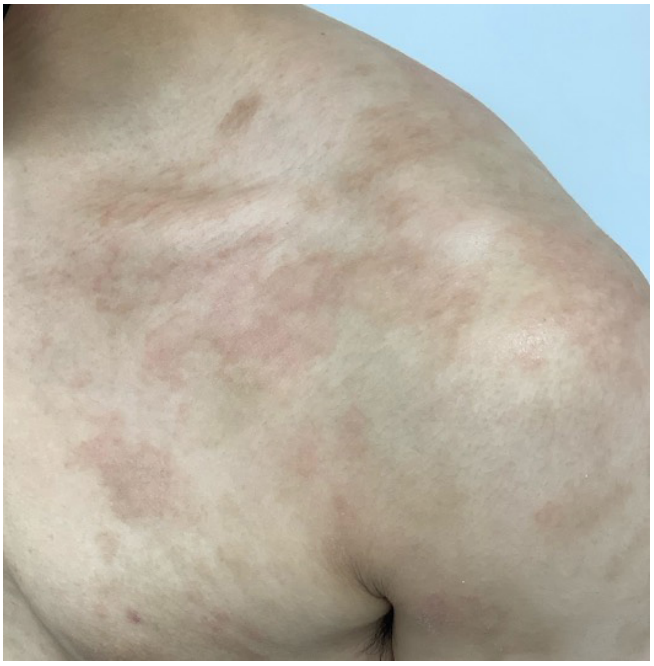

Figure 5. Active atopic dermatitis with significant post-inflammatory hyperpigmentation in a Filipino patient; *courtesy of Harry Liu, MD, FRCPC, FAAD*.

Therapeutic Implications

With the increasing burden of AD in Asia, the development of effective therapeutics is critical.[20] A major challenge contributing to this burden is the variable and often unpredictable response to treatment. While individuals from the same racial groups may share phenotypic and genetic traits, treatment efficacy can vary widely among different ethnic backgrounds. This highlights the complex and heterogeneous nature of AD, underscoring the necessity for personalized and region-specific therapeutic approaches.

In many Asian cultures, topical treatments, bath therapies, and oral herbal preparations are commonly used. These treatments can significantly impact the outcome of dermatological care and may have been used by patients prior to seeking professional treatment. Therefore, it is crucial for clinicians to inquire about these therapies before discussing the treatment plan. For prescribed topical therapies, prolonged use of high-potency topical corticosteroids can lead to hypopigmentation, a concern particularly relevant for patients with darker skin tones. Clinical studies have demonstrated the efficacy of pimecrolimus cream and tacrolimus ointment in treating AD in Asian patients.[13] Phototherapy, including narrow-band ultraviolet B (NB-UVB) and ultraviolet A (UVA), has shown effectiveness in managing moderate to severe AD in Asian populations.[13] Given the increased melanin content in more pigmented skin types, higher doses of NB-UVB may be required to achieve optimal results. Regarding systemic treatments, phase III trials of dupilumab that included 20–27% Asian participants demonstrated efficacy across diverse ethnic groups. A subsequent pooled sub-analysis further confirmed that treatment responses in Asian patients were comparable to those observed in other populations.[13]

Overall, studies have consistently highlighted the persistent underrepresentation of patients with skin of colour in global clinical trials for AD.[21] Despite the high prevalence of AD among non-White populations, data on the efficacy of common therapies in these groups remain limited. A significant barrier is the incomplete reporting of race and ethnicity in clinical trials. For instance,

among AD clinical trials published between 2000 and 2009, only 59.5% included race and ethnicity as part of baseline demographic data.[13] Furthermore, Asian participants accounted for just 6.9% of trial enrolment, underscoring the need for greater diversity in AD research.

Future studies focused on identifying specific and potentially unique molecular targets in Asian populations could pave the way for developing therapies that address the unmet needs of Asian patients with AD. However, the broader spectrum of *FLG* mutations observed in Asian populations may present challenges for developing targeted treatments. Additionally, the increased Th17 axis observed in Asian AD patients suggests they may be promising candidates for therapies targeting IL-17/IL-23.[22] Furthermore, the persistent elevation of IL-22 levels in both skin and peripheral blood among Asian populations presents another potential treatment target.[15,22]

Conclusion

AD presents a significant and growing challenge, particularly within Asian populations, due to its multifactorial nature, genetic diversity, and variable clinical manifestations. As AD prevalence continues to rise, it is essential to adopt more personalized and culturally competent approaches to diagnosis and treatment. Advancing our understanding of the genetic underpinnings, immune polarization, and environmental influences that contribute to the distinct AD phenotypes in Asian populations will be crucial for developing more targeted and effective therapies.

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