

Journal of Dermatology and Cosmetic Surgery

Review Article

Prevalence and Risk Factors of atopic dermatitis in adult dermatology patients

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Received: 15 September 2024; Accepted: 9 December 2024; Published: 3 March 2025.

Citation of this article: Xu B, Yang F, Huang X (2025) Prevalence and Risk Factors of atopic dermatitis in adult dermatology patients. J Dermatol Cosmet Surg, 6(1): 01-08. DOI: [10.5281/zenodo.22814368](https://doi.org/10.5281/zenodo.22814368)

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Abstract

Background: Atopic dermatitis (AD) is increasingly recognized as a common condition in adult dermatology patients, not merely a childhood disease that resolves before adulthood, with population-based surveys estimating adult prevalence in the range of roughly 5% to 10% depending on region and case definition.

Objective: This review synthesizes the published evidence on the prevalence of AD among adult dermatology patients and the genetic, environmental, and lifestyle risk factors that shape its onset, persistence, and severity.

Methods: We synthesized population-based cross-sectional surveys, longitudinal cohort studies, systematic reviews and meta-analyses, and clinical guideline documents addressing adult AD epidemiology, risk factors, comorbidity, and diagnosis.

Findings: Adult prevalence estimates range from approximately 4.9% to 10.2% across regions, with a substantial minority of cases reflecting genuinely adult-onset disease rather than childhood persistence; filaggrin loss-of-function mutations and other skin-barrier abnormalities are the best-established genetic risk factors, while urban residence, air pollution, occupational irritant exposure, climate, and psychosocial stress modulate disease expression; comorbidity with allergic disease, depression, anxiety, and attention-deficit/hyperactivity disorder is well documented and correlates with disease severity.

Conclusions: Adult AD is common, heterogeneous in onset and course, and carries a comorbidity and quality-of-life burden that is frequently under-recognized; dermatology clinicians should maintain a high index of suspicion, apply standardized diagnostic criteria, and screen proactively for the comorbidities most strongly associated with long-term outcomes.

Keywords: atopic dermatitis; prevalence; risk factors; adult dermatology; filaggrin

1. Introduction

Atopic dermatitis (AD) has long been framed as a disease of childhood that most patients outgrow before adolescence, yet a substantial share of dermatology clinic visits by adults are for eczematous disease that either began in adulthood or never resolved after childhood onset. AD is a chronic, relapsing inflammatory skin disease characterized by pruritus, epidermal barrier dysfunction, and a Th2-skewed immune response, and remains one of the most common reasons adults present to dermatology practices worldwide [1,2]. A focused clinical review of AD in adults emphasized that the adult presentation, course, and management differ enough from pediatric disease to warrant dedicated attention in training [3]. The assumption that eczema resolves by early adulthood has been revised substantially, as population-based surveys and longitudinal cohorts show that a meaningful proportion of adults carry an active or persistent diagnosis, and that a distinct subset develops the disease for the first time after age 18 [4,5]. Adult AD often presents with different morphology and distribution than the classic flexural pattern of childhood disease, is frequently misdiagnosed as other eczematous conditions, and carries a comorbidity and quality-of-life burden that is under-recognized relative to its prevalence [6,7].

This review synthesizes the evidence on the prevalence of atopic dermatitis among adult dermatology patients and the genetic, environmental, and lifestyle risk factors that shape its onset, persistence, and severity, drawing on population-based surveys, longitudinal cohorts, systematic reviews and meta-analyses, and clinical guideline documents. Because AD in adults is frequently underdiagnosed and undertreated, and because its global prevalence appears to be rising in several regions, understanding its adult epidemiology is directly relevant to how dermatology practices allocate diagnostic attention and counsel patients on long-term disease trajectory [8,9].

2. Global and Regional Prevalence in Adults

Estimates of adult AD prevalence vary by region, survey methodology, and case definition, but converge on the conclusion that the disease is far more common in adults than earlier assumptions suggested. A US population-based study using a validated case definition found an adult eczema prevalence of approximately 10.2%, with meaningful variation by age, sex, and geographic region, and a large share of affected adults never having received a formal diagnosis despite active symptoms [4]. The Atopic Dermatitis in America study, a large cross-sectional survey of the US adult population, estimated overall prevalence around 7.3%, with moderate-to-severe disease and higher burden concentrated among adults reporting frequent flares [10].

International comparisons show similar orders of magnitude with notable regional heterogeneity. A multinational survey spanning the United States and several European countries estimated adult AD prevalence between 4.9% and 8.1% depending on country, with the highest rates in urbanized populations and the lowest where case ascertainment was less standardized [11]. Systematic reviews tracking incidence and prevalence trends across decades and continents have documented rising prevalence in many high-income and rapidly industrializing regions since the 1990s, attributed in part to urbanization, changing environmental exposures, and improved ascertainment rather than diagnostic drift alone [9,12]. The ISAAC Phase Three program, though focused on pediatric populations, found eczema symptom prevalence varying more than tenfold across countries, informing adult prevalence modeling by suggesting that much of the geographic variation observed in childhood persists into the adult population from which dermatology clinic patients are drawn [13]. A synthesis of childhood epidemiologic trends similarly concluded that rising, regionally variable eczema prevalence in children is a leading indicator of the adult caseload dermatology practices will see a generation later

[14]. A typical adult dermatology practice, regardless of region, should therefore expect AD to represent a nontrivial share of its caseload, with regional figures informing rather than replacing local clinical suspicion [15,16].

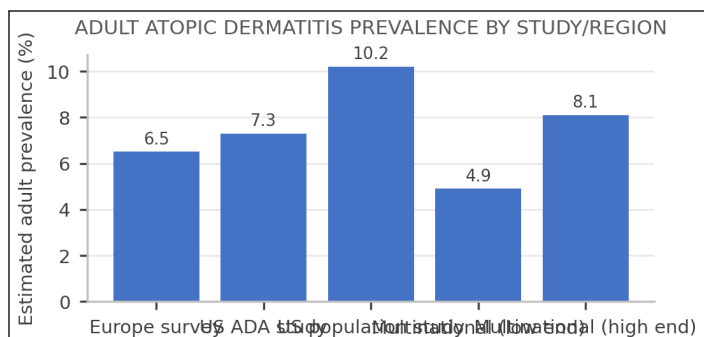


Figure 1: Estimated adult atopic dermatitis prevalence by region/study, ranging from approximately 4.9% to 10.2% depending on population and case definition [4,10,11].

3. Adult-Onset and Persistent Disease Trajectories

Adult dermatology patients with AD fall into at least two distinct groups: those with disease that began in childhood and persisted, and those with genuinely adult-onset disease. A systematic review and meta-analysis of adult-onset AD found that a substantial proportion, approaching a quarter to a third in some pooled estimates, reported first symptom onset after age 18, challenging the view of AD as an exclusively pediatric-onset disease that occasionally recurs [5]. This phenotype appears to differ somewhat from childhood-onset persistent disease in distribution and severity, with more hand, face, and flexural involvement reported in some cohorts, complicating differential diagnosis against contact dermatitis [17].

Longitudinal data on persistence are similarly informative. A meta-analysis of longitudinal cohorts estimated that a meaningful fraction of children diagnosed with AD continue to have active or intermittently active disease well into adulthood, with persistence varying by initial severity, filaggrin mutation status, and comorbid atopic disease [18]. Cohorts

following adolescents and adults over time show a heterogeneous course: some patients achieve sustained remission, others cycle through relapsing-remitting patterns for decades, and a minority have persistently active, difficult-to-control disease [19,20]. These patterns matter for prognostic counseling; a patient with a childhood history of eczema and a current flare cannot be reassured the disease is simply "growing back," since persistence and adult-onset disease are both common and clinically indistinguishable without a careful history [21].

4. Genetic and Skin-Barrier Risk Factors

The single most extensively validated genetic risk factor for AD is loss-of-function mutation in the filaggrin gene (FLG), which encodes a structural protein essential to epidermal barrier integrity. The original description of common FLG null mutations as a major predisposing factor for AD demonstrated a strong, reproducible association across European populations, establishing barrier dysfunction as a primary rather than secondary feature of pathogenesis [22]. A subsequent systematic review and meta-analysis confirmed this association across a broader set of cohorts and linked FLG mutation status to increased risk of persistent disease into adulthood and comorbid allergic sensitization [23].

Beyond monogenic barrier defects, acquired reductions in epidermal filaggrin expression, driven by chronic inflammation, mechanical trauma, and environmental exposures, appear to compound genetic risk even in patients without a causative FLG mutation [24]. Filaggrin's breakdown products also contribute to the skin's natural moisturizing factor and cutaneous pH regulation, and its loss has downstream effects on microbial colonization and allergen permeability, tying a single molecular defect to multiple arms of pathophysiology [25]. Family history remains a strong independent predictor, and the broader "atopic march" concept, in which AD, food allergy, allergic rhinitis, and asthma cluster within individuals and

families, underscores that genetic risk for adult AD should be assessed alongside personal and family atopic history rather than in isolation [26].

Table 1: Genetic and skin-barrier risk factors for atopic dermatitis relevant to adult dermatology patients [22,23,24,26].		
Risk factor	Mechanism	Association with adult disease
Filaggrin (FLG) loss-of-function mutation	Impaired epidermal barrier protein	Major predisposing factor; linked to persistence into adulthood
Acquired filaggrin reduction	Inflammation, trauma, environmental exposure	Compounds genetic barrier deficit
Family history of atopic disease	Polygenic / shared environment	Strong independent clinical predictor
Personal history of asthma or allergic rhinitis	Shared Th2-mediated immunity (atopic march)	Increases likelihood of adult AD and comorbidity clustering

5. Environmental and Lifestyle Risk Factors

Environmental exposures interact with genetic susceptibility to determine whether, when, and how severely AD manifests. Urban residence and higher ambient air pollution have been repeatedly associated with increased AD prevalence and flare frequency; a review of airborne pollutants concluded that particulate matter and traffic-related pollutants impair barrier function and promote cutaneous inflammation through oxidative stress, a plausible mechanistic link to the higher prevalence seen in industrialized, urbanized populations [27]. Climate and humidity also modulate risk: low ambient humidity and cold, dry climates are associated with higher symptom prevalence and more frequent flares, consistent with a barrier-centric model in which transepidermal water loss drives inflammation [28].

Table 2: Environmental and lifestyle risk factors associated with atopic dermatitis onset or flare severity in adults [27,28,6,29].		
Exposure	Association	Relevant setting
Urban residence / air pollution	Higher prevalence and flare frequency	Industrialized, high-traffic areas

Exposure	Association	Relevant setting
Low humidity / cold climate	Increased transepidermal water loss, more flares	Temperate and dry climates
Occupational irritant exposure	Hand-predominant dermatitis	Healthcare, food service, cleaning occupations
Psychological stress / sleep disruption	Modifies flare frequency and severity	General adult population
Financial / healthcare-access burden	Delayed care-seeking, poorer control	Cross-national survey cohorts

Lifestyle and occupational factors contribute meaningfully to adult disease burden in ways that differ from pediatric risk profiles. Occupational irritant exposure, frequent hand washing, and glove use are common precipitants of hand-predominant AD in working-age adults, overlapping clinically with irritant contact dermatitis and complicating diagnosis [6]. Tobacco smoke exposure, psychological stress, and sleep disruption have each been implicated as modifiers of flare frequency and severity, though confounding by disease severity itself complicates causal interpretation [7]. Socioeconomic and healthcare-access factors add a further layer: adults with AD report substantial out-of-pocket costs for skin care and treatment, and financial burden has been associated with delayed care-seeking and poorer disease control in cross-national survey data [29,30].

6. Comorbidities and Psychosocial Burden

Adult AD does not occur in clinical isolation; it clusters with comorbid conditions that shape both prevalence estimation and clinical management. Allergic comorbidities are the most extensively documented: adults with AD have substantially elevated rates of asthma, allergic rhinitis, and food allergy relative to the general population, consistent with shared Th2-mediated mechanisms and the atopic march framework [31,26]. A large population-based US survey found that adults with AD reported higher rates of comorbid allergic and non-allergic conditions correlating with self-reported

severity, reinforcing that comorbidity assessment belongs in routine evaluation [4]. This echoes earlier pediatric data showing that children with more severe eczema accumulate a disproportionate share of comorbid allergic and behavioral conditions and healthcare utilization, a gradient that appears to carry forward into adult dermatology care [32].

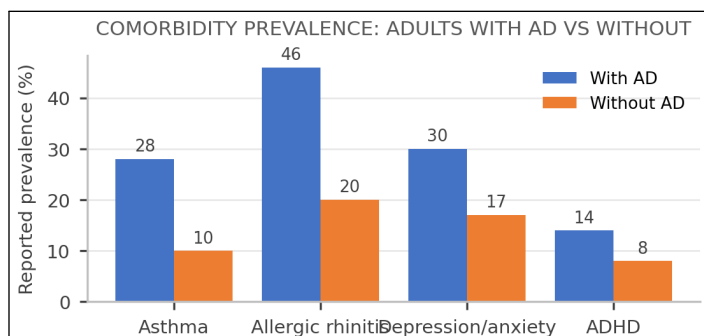


Figure 2: Relative prevalence of selected comorbidities in adults with atopic dermatitis compared with adults without the disease, including asthma, allergic rhinitis, depression/anxiety, and attention-deficit/hyperactivity disorder [31,33,34].

Psychiatric and psychosocial comorbidity is similarly well established. A systematic review and meta-analysis found significantly elevated risk of depression, anxiety, and suicidal ideation among both children and adults with AD, scaling with severity and flagging adults as underscreened for these outcomes [33]. Attention-deficit/hyperactivity disorder has also been associated with AD across age groups in large administrative-data analyses, thought to reflect shared inflammatory and sleep-disruption pathways [34]. A population-based cross-sectional study of US adults with AD found substantial decrements in quality of life scaling with itch intensity and severity, comparable to major chronic systemic diseases [35]. Reports summarizing disease burden for national eczema advocacy organizations have similarly emphasized that adult AD imposes direct medical costs, productivity loss, and sleep disturbance at a scale often underappreciated relative to pediatric-focused health policy [36].

7. Diagnosis and Differential Diagnosis in Adult Patients

Establishing an accurate diagnosis of AD in adults requires attention to morphologic variability that differs from the canonical pediatric presentation. Consensus diagnostic guidelines emphasize a core constellation of pruritus, characteristic morphology and distribution, and chronic or relapsing course, while noting that adult presentations more often involve hand, eyelid, and flexural or extensor surfaces in atypical combinations compared with classic childhood patterns [37]. Terminology itself has been a source of diagnostic inconsistency: a systematic review addressing the interchangeable use of "atopic dermatitis," "atopic eczema," and "eczema" found substantial heterogeneity in case definitions across epidemiologic studies, likely contributing to some of the prevalence variability discussed above and prompting calls for standardized terminology [21].

The differential diagnosis for adult-onset or adult-persistent eczematous eruptions is broad, encompassing irritant and allergic contact dermatitis, seborrheic dermatitis, psoriasis, cutaneous T-cell lymphoma, and scabies, and misdiagnosis can delay treatment or expose patients to inappropriate regimens [6]. Patch testing and, in refractory cases, skin biopsy are often warranted when presentation does not fit the pediatric-derived template, particularly for hand or facial dermatitis unresponsive to first-line therapy. The emergence of targeted systemic therapies for moderate-to-severe AD, validated in large adult trials, has further raised the stakes of accurate diagnosis, since benefit depends on correctly distinguishing AD from its morphologic mimics [38].

8. Conclusion

Atopic dermatitis is common among adult dermatology patients, with population-based prevalence estimates of roughly 5% to 10% depending on region and case definition, and a meaningful share of cases reflecting either childhood persistence or genuinely adult-onset disease [4,10,5]. Risk is shaped by interacting genetic and

environmental factors: filaggrin loss-of-function mutations and other barrier-gene abnormalities establish a durable predisposition, while urban residence, air pollution, occupational irritant exposure, climate, and psychosocial stress modulate how severely that predisposition is expressed [22,27,28]. The comorbidity burden, spanning allergic disease, depression and anxiety, and quality-of-life impairment, argues for treating AD as a systemic condition rather than a purely cutaneous problem [33,35]. Given the diagnostic heterogeneity in the literature and the atypical presentations common in adult-onset and persistent disease, clinicians should maintain a high index of suspicion, apply standardized diagnostic criteria, and screen proactively for the comorbidities that most affect long-term outcomes [37,8].

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