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Behind the Itch: Mapping the Interplay Between Genetics, Immunity, and Lifestyle in Skin Irritation with Rash

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Abstract. Eczema, or atopic dermatitis, affects nearly 10% of adults worldwide and imposes a heavy burden through chronic itch and impaired quality of life. While many genetic, immune, and lifestyle factors have been proposed, their relative contributions and interactions remain unclear, particularly across different age groups. To address this gap, we designed a survey-based study that systematically investigated demographic, behavioral, and environmental contributions to eczema among teenagers and adults. Survey items were developed based on an extensive literature review and targeted factors such as age, gender, stress, sleep, moisturization, and public awareness of skin conditions. Responses were analyzed using statistical method to identify significant associations and exclude spurious correlations, Among the numerous factors examined, age, gender, and moisturization habits consistently emerged as significant predictors of eczema. These findings refine our understanding of the condition by emphasizing significant correlates and clarifying misconceptions surrounding eczema. By distinguishing between meaningful associations and less relevant factors, this research contributes both to the scientific knowledge of eczema and to public health practice in managing one of the world's most common skin conditions.

Keywords: Atopic Dermatitis, Eczema, Allergies

1. Introduction

The skin is the body's largest organ, providing essential barrier protection, sensory perception, and immune defense [1]. When these functions are disrupted, inflammatory skin diseases such as atopic dermatitis (AD) — the most common form of eczema — can arise. AD affects about 10% of adults and up to 20% of children worldwide, causing chronic itch, recurrent rashes, and reduced quality of life [2,3].

AD pathogenesis reflects an interplay of genetic, immune, and environmental factors. Loss-of-function mutations in the filaggrin (FLG) gene impair the epidermal barrier, increasing allergen penetration and water loss [4,5]. A skewed Th2 immune response characterized by elevated IL-4 and IL-13 drives chronic inflammation [6], while neuroimmune signals such as IL-31 further amplify itch and hypersensitivity [1]. Although these mechanisms are well established, fewer studies have explored how they intersect with everyday behaviors—such as stress, sleep, and moisturization

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habits—across age groups. Understanding which demographic and lifestyle factors most strongly correlate with eczema can refine prevention and self-management strategies.

To address this gap, our study surveyed 104 individuals to examine how genetic background, immune imbalance, and daily habits jointly influence skin irritation and rash. Drawing on recent findings on FLG mutations [7] and environmental triggers [8], we aimed to identify significant predictors and clarify misconceptions surrounding eczema by distinguishing meaningful associations from less relevant factors.

2. Methods

This study analyzed a cross-sectional, anonymous online survey of adults (N = 104). Respondents self-classified eczema status as A: no history, B: unsure with eczema-like symptoms, or C: personal diagnosis or family history. For analysis, categories B and C were combined as the eczema group (n = 66) and compared with the healthy group (n = 38). Missing data were handled by pairwise deletion.

Demographic and behavioral variables were cleaned and standardized. Age categories were rebinned as <25, 25–40, and 41+ to ensure stable cell counts. Gender labels were harmonized as female or male. Moisturizing frequency (Q5) was analyzed in both its six original levels for descriptive plots and as collapsed contrasts—"Daily or more," "Less often," and "Never"—for inference. Multi-select questions (e.g., sleep problems, diet, family diseases, symptom locations, and triggers) were tokenized and converted to binary variables, merging close textual variants (e.g., two phrasings of "sleep deprivation" unified).

The primary outcome was eczema status (B/C = 1; A = 0). Secondary outcomes included individual multi-select options and ordinal ratings for stress (Q8) and self-rated skin condition (Q6).

Group differences in categorical variables were tested using Pearson chi-square, and ordinal outcomes by Mann–Whitney U or Kruskal–Wallis tests. Predictors of eczema status were estimated with binomial logistic regression, using <25 years and "no family skin condition" as reference levels. Penalized ridge regression was applied when separation or sparse data occurred, and odds ratios with 95% confidence intervals were computed for planned contrasts (e.g., "Never" vs. "Daily or more" moisturization). All tests were two-sided and unadjusted given the study's exploratory aim.

3. Results

3.1. Survey captured a balanced mix of healthy and eczema respondents

A total of 104 individuals completed the survey. Of these, 38 (37%) reported no history of eczema, 23 (22%) were unsure but described eczema-like symptoms, and 43 (41%) reported either a personal diagnosis or a family history of eczema. For analysis, categories B (Unsure with eczema-like symptoms) and C (Diagnosed or family history) were combined into an eczema group (B/C; n = 66) and contrasted with the healthy group (n = 38).

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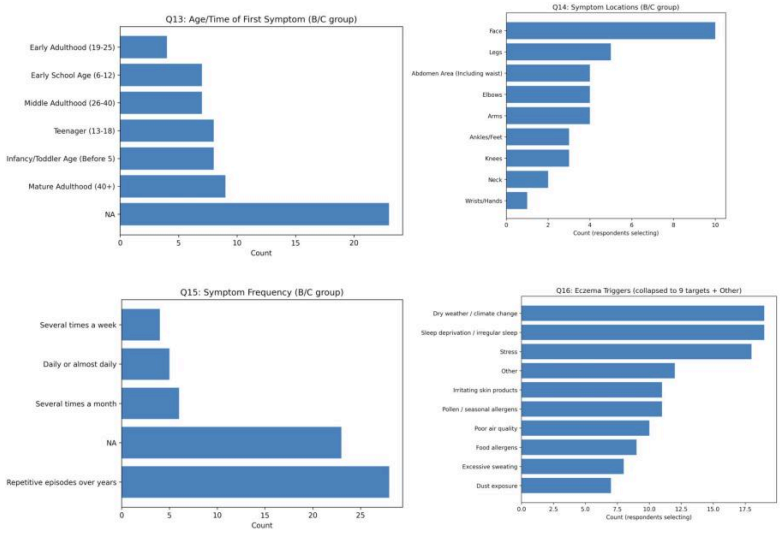


Figure 1. Descriptive profile of respondents with eczema or possible eczema (n = 66)

(A) Q13 – Onset timing. Number of respondents selecting each age/time of first symptom; "NA" indicates not reported. (B) Q14 – Symptom locations. Number of respondents selecting each location (multi-select). (C) Q15 – Symptom frequency. Number of respondents selecting each frequency option. (D) Q16 – Triggers (collapsed categories). Number of respondents selecting each trigger after collapsing to nine targets plus "Other" (multi-select). B/C = B 'Unsure with eczema-like symptoms' + C 'Diagnosed or family history' (Q12).

3.2. Descriptive profile of respondents with eczema

Among respondents with eczema or possible eczema (n = 66), symptom onset spanned childhood through adulthood, and about one third did not recall the timing (Fig.1A). The face was the most frequently reported site, followed by legs; abdomen/waist, elbows, and arms were next, with ankles or feet, knees, neck, and hands less common (Fig.1B). The course was usually recurrent over years rather than daily or weekly (Fig.1C). Reported triggers centered on environment and lifestyle: dry weather or climate change and sleep deprivation or irregular sleep were most common, with stress close behind; irritating skin products, pollen or seasonal allergens, and poor air quality were mid-tier; food allergens, excessive sweating, and dust exposure were less frequently endorsed (Fig.1D).

3.3. Age and family history strongly predicted eczema risk

Age was significantly associated with eczema status ($\chi^2 = 8.7$, $p = 0.013$). Compared with respondents under 25 years, those aged 25–40 had 4.37-fold higher odds (95% CI 1.25–15.25, $p = 0.021$) and those aged 41+ had 3.36-fold higher odds (95% CI 1.19–9.50, $p = 0.022$) of belonging to the eczema group. Reporting a family skin condition was also linked to nearly three-fold higher odds of eczema (OR 2.96, 95% CI 1.16–7.55, $p = 0.023$), underscoring the importance of genetic predisposition. Consistent with this, family allergic rhinitis (hay fever) was substantially more common in the eczema groups ($\chi^2 = 13.90$, $p = 0.00096$; Fig. 3B). These effects are visualized in the odds-ratio forest plot (Fig. 2B).

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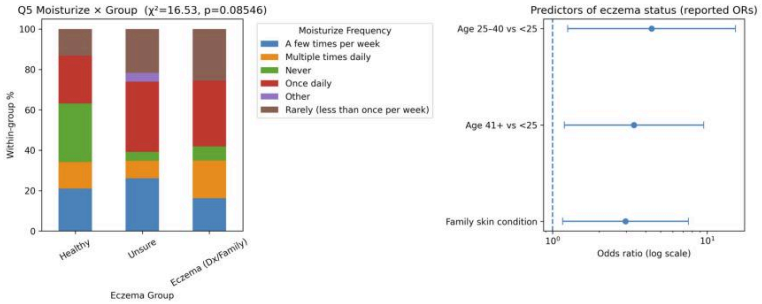


Figure 2. Moisturizing behavior by group and predictors of eczema status

Q5 Moisturize × Group. Stacked bars show within-group percentages of moisturizing frequency for Healthy, Unsure, and Eczema (Dx/Family). Pearson χ^2 for the full 3×6 table is shown in the title (χ^2 = 16.53, p = 0.085). (B) Predictors of eczema status (forest plot). Points are odds ratios and horizontal lines are 95% CIs from the reported logistic model; vertical dashed line marks OR = 1. Reference levels are <25 years and no family skin condition. Reported estimates: Age 25–40 vs <25 OR 4.37 (95% CI 1.25–15.25), Age 41+ vs <25 OR 3.36 (1.19–9.50), Family skin condition OR 2.96 (1.16–7.55).

3.4. Frequent moisturizing reflected disease management rather than protection

As shown in Figure 2A, the eczema group clusters in the daily or multiple-daily categories, “never” is relatively common among healthy respondents, and the unsure group is intermediate. The six-category omnibus comparison trended but was not significant (χ^2 = 16.53, p = 0.085; Figure 2A). In a prespecified collapsed contrast, moisturizing frequency did differ between groups (χ^2 = 11.6, p = 0.040). Participants with eczema were more likely to moisturize daily or multiple times per day, whereas those who never moisturized had markedly lower odds of eczema compared with daily moisturizers (OR 0.14, 95% CI 0.03–0.58, p = 0.007). Exercise frequency did not differ between groups (χ^2 = 2.23, p = 0.53).

3.5. Stress, sleep, and awareness showed minimal differences between groups

Stress ratings were nearly identical between the healthy and eczema groups (mean = 3.14 vs. 3.18 on a 1–5 scale; Mann–Whitney U = 1283.0, p = 0.84), and self-rated skin condition also showed minor, non-significant differences (mean = 2.97 vs. 3.21; U = 1435.0, p = 0.18). Most sleep problems showed no group differences. 'Waking up too early and unable to fall back asleep’ trended higher in the eczema group but was not statistically significant (χ^2=5.43, p=0.066, Fig.3A). Healthy respondents described preventive routines (regular moisturizers, balanced diet, sunscreen), while eczema respondents listed treatments and medications (steroids, antihistamines, herbal remedies). Awareness of immune contributions and dermatological terms was higher among eczema groups, but willingness to use advanced therapies did not differ (mean scores 2.71 vs. 2.77; U = 1230.5, p = 0.87). Social media and healthcare providers were the most common sources of health information across all groups.

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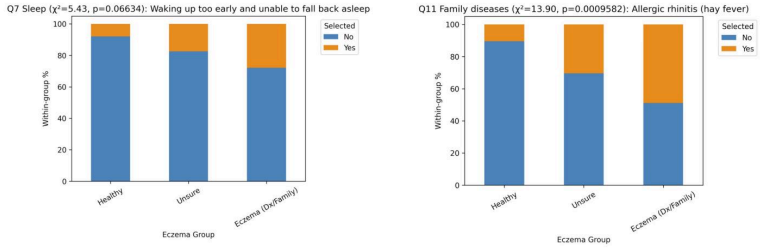


Figure 3. Item-level associations with eczema status

(A) Sleep: "Waking up too early and unable to fall back asleep" by group. Bars show within-group percentages selecting the item (orange = Yes; blue = No). Pearson χ^2 (3 groups) = 5.43, p = 0.066 (trend). (B) Family diseases: "Allergic rhinitis (hay fever)" by group. Bars show within-group percentages (orange = Yes; blue = No). Pearson χ^2 (3 groups) = 13.90, p = 0.00096 (significant). Axes: x = Eczema group (Healthy, Unsure, Eczema [Dx/Family]); y = within-group %. Titles display the test statistic and p-value.

4. Discussion

This study reveals that eczema risk is primarily shaped by genetic predisposition and age-related physiology, with behavioral factors playing a secondary, adaptive role. The strong association between family history and eczema supports prior evidence that filaggrin (FLG) mutations compromise the skin barrier and promote chronic inflammation [4,5,7]. The enrichment of allergic rhinitis in affected individuals further reflects a shared atopic pathway driven by Th2 cytokines such as IL-4 and IL-13 [6]. Both are driven by a type-2–skewed inflammatory program in which IL-4 and IL-13 promote class-switching to IgE, enhance eosinophil recruitment, and downregulate filaggrin and other barrier-related genes. This cytokine milieu facilitates heightened sensitivity to aeroallergens and cutaneous irritants, providing a mechanistic explanation for why eczema and allergic rhinitis frequently co-occur [4,5,7].

Age also emerged as a significant predictor: adults over 25 showed greater eczema prevalence, likely reflecting cumulative environmental exposure and progressive immune imbalance [2]. In contrast, frequent moisturization was more common among eczema respondents, suggesting reverse causality—symptomatic individuals engage more in management routines rather than prevention.

Contrary to common belief, stress and sleep problems were not significantly associated with eczema status. These null findings align with emerging evidence that neuroimmune crosstalk, rather than stress alone, maintains chronic pruritus [1].

Overall, the results emphasize that eczema arises from interacting genetic, immune, and behavioral factors. Genetic susceptibility sets the foundation, while lifestyle and environment modify symptom expression. Effective management should therefore prioritize personalized education and barrier-repair strategies over one-size-fits-all prevention.

5. Conclusion

This study provides an integrated view of how genetic predisposition, age, and everyday behaviors interact to influence eczema risk and management. Family history and age emerged as the most consistent predictors, highlighting the central role of heritable barrier dysfunctions—such as filaggrin (FLG) mutations—in shaping disease susceptibility. In contrast, lifestyle variables like stress, sleep, and moisturization primarily reflected adaptive responses rather than direct causes. By

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distinguishing genetic foundations from behavioral adaptations, our findings clarify why eczema management must go beyond generic lifestyle advice. Personalized interventions that address individual risk background, disease stage, and skin-barrier maintenance may offer more effective prevention and control. Future research should adopt longitudinal and mechanistic designs to disentangle causality, examine neuroimmune pathways, and test targeted interventions such as stress reduction or barrier-repair therapies. Together, such efforts can advance more precise, biology-informed strategies for managing one of the world's most common chronic skin conditions.

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