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ORIGINAL RESEARCH

Breastfeeding, Family Stress, and Climate as Key Determinants of Atopic Dermatitis Severity in Children: A Cross-Sectional Study

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Background: Atopic dermatitis (AD) severity in children is influenced by biological, environmental, and psychosocial factors, yet severity-focused, multicenter evidence from Middle Eastern populations remains limited.

Objective: To identify early-life, familial, and climatic determinants of clinical severity in pediatric AD.

Methods: This multicenter cross-sectional study included 600 children diagnosed with atopic dermatitis according to the Hanifin and Rajka criteria. Disease severity was measured using the SCORAD index and categorized as mild, moderate, or severe. Independent variables included breastfeeding history, parental marital status, residential climate, animal exposure, and family atopy. Ordinal logistic regression models were used to estimate adjusted odds ratios (aORs) controlling for age, sex, and nationality.

Results: Compared with bottle-feeding, breastfeeding <6 months (aOR = 0.02; 95% CI 0.007–0.079) and ≥6 months (aOR = 0.29; 95% CI 0.17–0.50) were strongly protective against greater AD severity. Children from separated/divorced or widowed households and those living in mountain regions had significantly higher severity than peers from married families and coastal areas. Cat exposure was an independent determinant of greater severity (aOR = 6.48; 95% CI 3.33–12.64) and higher SCORAD, whereas general animal exposure, consanguinity, sex, and family atopy were not significant.

Conclusion: Breastfeeding, family structure, residential climate, and cat exposure are key, potentially modifiable determinants of pediatric AD severity and should inform risk-stratified, context-aware management.

Keywords: atopic dermatitis, disease severity, breastfeeding, psychosocial stress, climate factors, pediatric dermatology

Introduction

Atopic dermatitis (AD) is a common chronic inflammatory skin disease characterized by recurrent episodes of pruritus, erythematous papules or vesicles, and immune dysregulation that compromise skin barrier function.[1] It affects millions of children worldwide and imposes substantial psychosocial and economic burdens on patients and families through persistent itching, sleep disturbance, and reduced quality of life.[2] Recent global estimates suggest an increasing prevalence, particularly in developing regions, likely driven by changing environmental exposures and urban lifestyles.[3]

The pathogenesis of AD is multifactorial, involving complex interactions among genetic, immunologic, and environmental determinants.[4] Several studies have highlighted the contribution of genetic predisposition and family history of atopy—including asthma and allergic rhinitis—as key risk factors for both the onset and severity of AD.[5] Environmental factors such as humidity, temperature variability, and climate influence barrier integrity, while early-life exposures including breastfeeding patterns, diet, and household stressors shape immune and inflammatory responses.[6] Exclusive breastfeeding transfers immunomodulatory components such as cytokines and secretory IgA,[7] although evidence for its role in modifying AD severity remains mixed across populations.[8]

Psychosocial stress and family dynamics also play crucial roles. Children in single-parent or high-stress households may experience elevated stress mediators that worsen inflammation and exacerbate flares.[9] Moreover, climatic conditions

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exert additional influence: low humidity and colder weather increase transepidermal water loss and barrier dysfunction.[10] Whereas warmer or humid climates may reduce dryness but introduce greater exposure to environmental allergens and microbial colonization.[11]

Quality of life is a central concern in pediatric AD. Symptoms such as severe itching, visible lesions, and sleep loss affect both children and caregivers, leading to emotional distress and disrupted family functioning.[12] Several studies have shown that more severe AD is associated with poorer health-related quality of life in affected children and their families.[13] Chronic skin diseases significantly impair mental well-being and highlight the need for psychosocial support in dermatologic care.[14] Pediatric AD also places a substantial burden on caregivers' daily activities and healthcare utilization.[15]

Despite extensive global research, there remains a paucity of region-specific data addressing environmental, familial, and psychosocial correlates of AD severity in Saudi Arabia. To our knowledge, no multi-center study has comprehensively evaluated how breastfeeding, marital structure, climate, animal exposure, and atopic heredity jointly influence AD severity in children in Saudi Arabia. Although international models have integrated multiple determinants of AD severity,[16] such multidimensional studies remain scarce in Middle Eastern populations where climatic and cultural factors differ markedly from Western settings.

Most regional studies have focused on AD prevalence rather than validated severity outcomes, and few have jointly evaluated early-life feeding, family structure, climate, and species-specific pet exposure using SCORAD.

This multi-center cross-sectional study aimed to evaluate the associations between biological, environmental, and familial determinants and the clinical severity of AD using the SCORAD index. By examining the independent contributions of breastfeeding duration, parental marital status, climatic region, animal exposure, and atopic background, our objective was to generate region-specific evidence to inform preventive strategies and tailored management for pediatric AD in Saudi Arabia.

Materials and Methods

Study Design and Sample

This multicenter cross-sectional study was conducted over six months beginning April 2025 across outpatient dermatology clinics in governmental and private hospitals in Saudi Arabia. A total of 600 pediatric patients newly diagnosed with AD and with no history of systemic treatment, confirmed by board-certified dermatologists using the Hanifin and Rajka criteria, were enrolled. The sample size was calculated assuming a medium effect size (Cohen's $w = 0.3$), 80% power, and $\alpha = 0.05$ for models including multiple predictors with a three-level outcome (mild, moderate, severe). A minimum of 540 participants was required; to enhance power and accommodate exclusions, 600 were recruited.

AD severity was evaluated using the SCORAD index and analyzed in relation to feeding practices, marital status, climatic conditions, animal exposure, and family history of atopy. Children were excluded if they had neurodevelopmental disorders, chronic systemic illnesses, or dermatologic conditions that could mimic AD (eg, psoriasis, lichen planus, ichthyosis). Participants unable to complete questionnaires due to cognitive or linguistic limitations were also excluded.

Study Variables

Independent Variables

Breastfeeding History

Infant feeding practices were obtained from parent or primary caregiver report at the study visit and categorized as breastfeeding for more than 6 months, breastfeeding for less than 6 months, bottle-feeding, or mixed feeding. Breastfeeding duration was recorded according to the caregiver's recall of the predominant feeding pattern during the first year of life.

Family History of Atopy

To assess genetic predisposition, data were collected regarding the presence of atopic conditions among first-degree relatives. This included any family history of bronchial asthma, allergic conjunctivitis, allergic rhinitis, or atopic dermatitis. The presence of any of these conditions was considered a positive family history, which could contribute to a heightened risk or greater severity of AD in children.

Parental Marital Status

Participants were categorized based on the current marital status of their parents, including whether they were married, separated, divorced, or widowed. This variable was explored in relation to the psychological and environmental stress experienced by the child, which may potentially exacerbate AD symptoms.

Animal Exposure

Information regarding household animal exposure was obtained from parent or primary caregiver report. Participants were classified as exposed if they had household pet exposure for at least 6 months at the time of AD diagnosis. The type of animal was recorded as cats, dogs, birds, reptiles, fish, or other mammals. However, the available records did not capture more detailed information on the exact intensity, frequency, indoor versus outdoor exposure, or allergen burden; therefore, animal exposure was analyzed as a categorical household exposure variable rather than a quantitative measure.

Climatic Conditions

Children were categorized according to their region of residence based on the city reported by the parent or caregiver and classified into three predefined geographic-climatic categories: mountain cities (eg, Abha, Taif), inland arid cities (eg, Riyadh, Madinah, Qassim), and coastal humid cities (eg, Jeddah, Dammam) This classification was based on the general climatic characteristics of these regions in Saudi Arabia, where mountain areas are typically cooler with lower oxygen pressure, inland areas are hotter and drier, and coastal areas are warmer and more humid Detailed patient-level environmental measurements, such as household humidity, ambient temperature, seasonal variation, or pollution exposure, were not available in the dataset.

Dependent Variable (Atopic Dermatitis Severity)

The SCORAD (SCORing Atopic Dermatitis) index was utilized to evaluate the severity of AD in each participant. This standardized tool assesses disease extent, lesion intensity, and subjective symptoms including pruritus and sleep disturbance. Scores range from 0 to 103 and are categorized as mild (<25), moderate (25–50), and severe (>50). This classification was used as the outcome measure in the analysis of all influencing variables.[17] SCORAD assessments were performed during routine clinical evaluation by board-certified dermatologists at the participating centers. The full SCORAD index was used. Inter-observer reliability was not formally assessed, which should be considered when interpreting the consistency of severity measurement across centers.

Covariates

Demographic covariates included gender, nationality, and age. Gender was categorized as either male or female. Nationality was classified as either Saudi or non-Saudi; due to the small number and heterogeneity of non-Saudi participants, they were grouped together for analysis. Age, although all participants were children, was stratified into four distinct groups to allow better understanding of disease patterns across developmental stages: infants and toddlers (0–2 years), preschool-aged children (3–5 years), school-aged children (6–11 years), and early adolescents (12–18 years). These age brackets align with common paediatric developmental stages and are widely used in pediatric research.

Data Presentation and Statistical Analysis

All analyses were conducted using R (R Foundation for Statistical Computing). Descriptive statistics were summarized as means (SD) for continuous variables and frequencies with percentages for categorical variables. Group differences were examined using Chi-square tests for categorical variables and one-way ANOVA for continuous variables.

The primary analysis used a proportional-odds ordinal logistic regression model to estimate adjusted odds ratios (aORs) with 95% confidence intervals for factors associated with AD severity. Model fit was assessed using likelihood-ratio tests and pseudo-R^2 measures. The proportional-odds assumption was evaluated using Brant tests; minor deviations were judged acceptable given stability of effect estimates.

Diagnostic checks included assessment of residual trends, influence measures, and variance inflation factors, all of which indicated no concerning multicollinearity.

A sensitivity analysis modeled SCORAD as a continuous count-like variable. Due to overdispersion, a negative binomial regression was selected over Poisson models based on likelihood criteria. Results were reported as adjusted rate ratios (aRRs) with 95% CIs. All statistical tests were two-sided with $\alpha = 0.05$.

Ethical Considerations

Ethical approval was obtained from the Institutional Review Board of the authors' institution on April 5, 2025 (TU-25-033). Written informed consent was obtained from parents or legal guardians, with child assent when appropriate. The study adhered to the Declaration of Helsinki and relevant human research standards. Confidentiality was maintained, no identifying data were collected, and participation was voluntary with the option to withdraw at any time.

Results

Participant Characteristics

A total of 600 children and adolescents with atopic dermatitis (AD) were included. Girls represented 55% (n = 332) and boys 45% (n = 268), with a mean age of 6.7 ± 4.7 years. Age distribution was 21% aged 0–2 years, 28% aged 3–5 years, 34% aged 6–11 years, and 17% aged 12–18 years. Most participants were Saudi nationals (85%).

Overall, 79% had married parents, 11% were separated, 8% divorced, and 2% widowed. Infant feeding in the first year included breastfeeding ≥ 6 months (41%), breastfeeding < 6 months (28%), mixed feeding (17%), and bottle-feeding (14%). Regarding residence, 43% lived in inland cities, 39% in coastal cities, and 18% in mountain regions. Animal exposure was reported in 20%, and parental consanguinity in 33%.

A parental or family history of atopy was present in 36%. The mean SCORAD score was 16.7 ± 15.2, with a median (IQR) of 11 (13). Key demographic and clinical characteristics by severity category are summarized in Table 1.

Bivariate Associations with AD Severity

Infant feeding pattern ($p < 0.001$), parental marital status ($p < 0.001$), residential climate ($p < 0.001$), and nationality ($p = 0.038$) showed significant unadjusted associations with AD severity (Table 1). Children breastfed < 6 months or ≥ 6

Table 1 Distribution of Participants' Characteristics and Their Association with AD Severity

Characteristics	Category	Total	AD severity			*P*-value
			Mild	Moderate	Severe	
Gender	Female	332 (55%)	260 (78.3%)	51 (15.4%)	21 (6.3%)	0.534
	Male	268 (45%)	200 (74.6%)	50 (18.7%)	18 (6.7%)	
Age group	Early adolescents	103 (17%)	85 (82.5%)	14 (13.6%)	4 (3.9%)	0.417
	School-aged	205 (34%)	149 (72.7%)	39 (19.0%)	17 (8.3%)	
	Preschool-aged	165 (28%)	132 (80.0%)	23 (13.9%)	10 (6.1%)	
	Infants & toddlers	127 (21%)	94 (74.0%)	25 (19.7%)	8 (6.3%)	
Nationality	Saudi	508 (85%)	390 (76.8%)	90 (17.7%)	28 (5.5%)	0.038[a]
	Other	92 (15%)	70 (76.1%)	11 (12.0%)	11 (12.0%)	
Parent marital status	Widow	11 (2%)	4 (36.4%)	6 (54.5%)	1 (9.1%)	<0.001[a]
	Separated	67 (11%)	41 (61.2%)	8 (11.9%)	18 (26.9%)	
	Divorced	47 (8%)	23 (48.9%)	11 (23.4%)	13 (27.7%)	
	Married	474 (79%)	392 (82.7%)	76 (16.0%)	6 (1.3%)	

(*Continued*)

Table 1 (Continued).

Characteristics	Category	Total	AD severity			*P*-value
			Mild	Moderate	Severe	
Feeding in 1st year of life	Breastfeeding > 6 months	245 (41%)	195 (79.6%)	44 (18.0%)	6 (2.4%)	<0.001[a]
	Breastfeeding < 6 months	170 (28%)	167 (98.2%)	2 (1.2%)	1 (0.6%)	
	Mixed	100 (17%)	54 (54.0%)	24 (24.0%)	22 (22.0%)	
	Bottle-feeding	85 (14%)	44 (51.8%)	31 (36.5%)	10 (11.8%)	
Climate conditions	Inland city	256 (43%)	193 (75.4%)	46 (18.0%)	17 (6.6%)	<0.001[a]
	Mountain City	109 (18%)	68 (62.4%)	29 (26.6%)	12 (11.0%)	
	Coastal city	235 (39%)	199 (84.7%)	26 (11.1%)	10 (4.3%)	
Parental consanguinity	1st cousin	75 (12%)	65 (86.7%)	7 (9.3%)	3 (4.0%)	0.340
	2nd cousin	44 (7%)	31 (70.5%)	8 (18.2%)	5 (11.4%)	
	Distant cousin or third cousin	81 (14%)	62 (76.5%)	13 (16.0%)	6 (7.4%)	
	No consanguinity	400 (67%)	302 (75.5%)	73 (18.2%)	25 (6.2%)	
Parental history of atopy	Allergic Conjunctivitis	49 (8%)	42 (85.7%)	4 (8.2%)	3 (6.1%)	0.661
	Allergic Rhinitis	93 (16%)	71 (76.3%)	18 (19.4%)	4 (4.3%)	
	Bronchial Asthma	182 (30%)	134 (73.6%)	36 (19.8%)	12 (6.6%)	
	Atopic Dermatitis	170 (28%)	132 (77.6%)	27 (15.9%)	11 (6.5%)	
	No History	106 (18%)	81 (76.4%)	16 (15.1%)	9 (8.5%)	
Family history of atopy	Allergic Conjunctivitis	40 (7%)	31 (77.5%)	6 (15.0%)	3 (7.5%)	0.846
	Allergic Rhinitis	40 (7%)	29 (72.5%)	10 (25.0%)	1 (2.5%)	
	Bronchial Asthma	89 (15%)	67 (75.3%)	17 (19.1%)	5 (5.6%)	
	Atopic Dermatitis	46 (8%)	36 (78.3%)	6 (13.0%)	4 (8.7%)	
	No	385 (64%)	297 (77.1%)	62 (16.1%)	26 (6.8%)	
Any animal exposure	Yes	117 (20%)	83 (70.9%)	21 (17.9%)	13 (11.1%)	0.064
	No	483 (80%)	377 (78.1%)	80 (16.6%)	26 (5.4%)	
Cat		41 (7%)	7 (17.1%)	21 (51.2%)	13 (31.7%)	<0.001[a]
Dog		17 (3%)	17 (100%)	0 (0.0%)	0 (0.0%)	0.070
Reptile		6 (1%)	6 (100.0%)	0 (0.0%)	0 (0.0%)	0.398
Other Mammals		12 (2%)	12 (100.0%)	0 (0.0%)	0 (0.0%)	0.155
Birds		29 (5%)	29 (100.0%)	0 (0.0%)	0 (0.0%)	0.010[a]
Fish		16 (3%)	16 (100.0%)	0 (0.0%)	0 (0.0%)	0.082
Age (years)			6.7 ± 4.7	6.4 ± 4.4	6.2 ± 3.7	0.659
SCORAD score (Mean ±SD)		16.7±15.2	10.5 ± 7.8	28.1 ± 6.4	59.7 ± 7.5	
SCORAD score Median (IQR)		11 (13)	10 (6)	25 (9)	60 (10)	

Note: [a]significance was tested using chi-square for categorical variables and ANOVA for numerical variables.

months had very low proportions of severe AD (0.6% and 2.4%, respectively), compared with mixed feeding (22.0% severe) and bottle-feeding (11.8% severe).

Among children of married parents, 82.7% had mild AD and 1.3% had severe disease, whereas those of divorced and separated parents had a markedly higher proportion of severe AD (27.7% and 26.9%, respectively). Coastal residents had the most favorable severity distribution (84.7% mild; 4.3% severe), whereas mountain residents had the highest proportion of severe AD (11.0%). Non-Saudi children more often had severe AD than Saudi children (12.0% vs 5.5%).

No significant crude associations were observed for gender, age group, parental consanguinity, or parental and family history of atopy. Overall animal exposure (any pet versus none) was not significantly associated with AD severity ($p = 0.064$), although exposure to specific pets such as cats and birds showed significant unadjusted associations (Table 1).

Multivariable Ordinal Logistic Regression

In the proportional-odds ordinal logistic regression model (Table 2), breastfeeding remained strongly protective. Compared with bottle-feeding, breastfeeding for < 6 months was associated with markedly lower odds of higher AD severity (aOR = 0.02; 95% CI 0.007–0.079; $p < 0.001$), and breastfeeding for ≥ 6 months also reduced the odds (aOR = 0.29; 95% CI 0.17–0.50; $p < 0.001$).

Parental marital status was the strongest social determinant. Children from divorced or separated families had more than fourfold higher odds of greater severity compared with those from married households (aOR = 4.29; 95% CI 2.61–7.04; $p < 0.001$). Those with widowed parents also had increased odds (aOR = 4.42; 95% CI 1.41–13.87; $p = 0.011$).

Residential climate showed a significant environmental effect. Children living in mountain cities had higher odds of greater severity than those in coastal areas (aOR = 2.47; 95% CI 1.37–4.44; $p = 0.003$). Inland versus coastal residence showed a trend toward higher severity (aOR = 1.63; 95% CI 0.98–2.71; $p = 0.060$).

Table 2 Predictors of the Atopic Dermatitis Severity Among Children and Adolescents (Adjusted Odds Ratios Using Proportional-Odds Ordinal Logistic Regression)

Term	Adjusted OR [b]	95% CI Lower Limit	95% CI Upper Limit	P-value
Feeding (1st yr): Mixed vs Bottle	0.990	0.552	1.778	0.950
Feeding (1st yr): Breastfeeding < 6 months vs Bottle	0.023	0.007	0.079	<0.001[a]
Feeding (1st yr): Breastfeeding > 6 months vs Bottle	0.292	0.171	0.498	<0.001[a]
Marital status: Divorced /Separated vs Married	4.289	2.613	7.041	<0.001[a]
Marital status: Widow vs Married	4.417	1.406	13.873	0.01[a]
Climate: Mountain City vs Coastal	2.466	1.368	4.442	0.003[a]
Climate: Inland city vs Coastal	1.631	0.980	2.714	0.060
Animals in childhood: Yes vs No	1.084	0.662	1.777	0.748
Cat exposure: Yes vs No	6.48	3.33	12.64	<0.001[a]
Consanguinity (any): Yes vs No	0.709	0.444	1.133	0.151
Parental atopy (any): Yes vs No	0.765	0.431	1.357	0.360
Family atopy (any): Yes vs No	0.980	0.618	1.554	0.931
Gender: female vs male	0.726	0.472	1.116	0.144
Nationality: Other vs Saudi	1.775	0.966	3.261	0.064

Note: [a]Significant association. [b]Ordinal logistic model using marginal standardization with odds ratio adjusted for age group, gender, nationality, family history, animal exposure.

Importantly, when specific cat exposure was included in the adjusted model alongside general animal exposure, cat ownership remained an independent predictor of greater AD severity (aOR = 6.48; 95% CI 3.33–12.64; $p < 0.001$), whereas general animal exposure was not significantly associated with severity.

Other predictors—including parental consanguinity, parental or family atopy, sex, and nationality—were not significantly associated after adjustment. The model showed good overall fit (likelihood-ratio $\chi^2 = 175.1$, df = 14, $p < 0.001$) with moderate explanatory power (McFadden $R^2 = 0.216$; Nagelkerke $R^2 = 0.342$), and proportional-odds assumptions were acceptable (Figure 1).

Sensitivity Analysis Using Negative Binomial Model

When SCORAD was modeled as a count-like outcome using negative binomial regression (Table 3), the main findings were consistent. Compared with bottle-feeding, breastfeeding for < 6 months reduced the expected SCORAD score by 41% (aRR = 0.59; 95% CI 0.49–0.72; $p < 0.001$), and breastfeeding for ≥ 6 months reduced it by 25% (aRR = 0.75; 95% CI 0.63–0.89; $p = 0.001$).

Children from non-intact households had significantly higher expected SCORAD scores than those from married households. For separated/divorced versus married parents, the adjusted rate ratio was 1.59 (95% CI 1.37–1.83; $p < 0.001$), and for widowed versus married parents it was 1.63 ($p = 0.020$).

Cat exposure remained independently associated with higher SCORAD scores, with children exposed to cats showing a 71% higher expected SCORAD than unexposed children (aRR = 1.71; 95% CI 1.22–2.40; $p = 0.002$).

Climate, consanguinity, parental or family atopy, and sex were not statistically significant in this model (all $p > 0.05$). Dispersion parameters (Pearson $\varphi = 1.19$; deviance $\varphi = 1.06$) indicated good model fit and supported the robustness of the negative binomial specification.

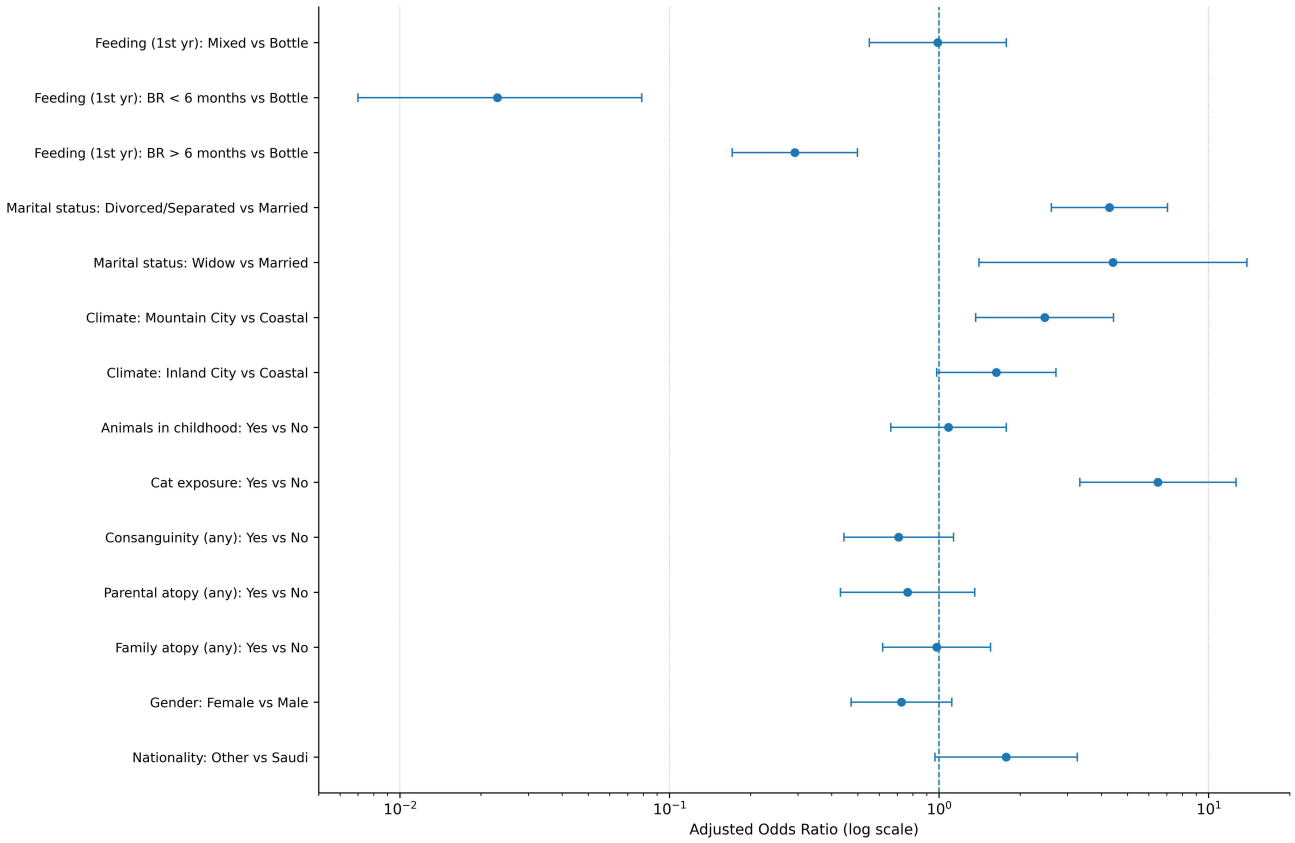

Figure 1 Adjusted odds ratios (aORs) and 95% confidence intervals for determinants of atopic dermatitis severity from the proportional-odds ordinal logistic regression model.

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Table 3 Adjusted Rate Ratios (aRR) for Predictors of SCORAD Scores According to Negative Binomial (NB) Model

Predictor	aRR [b]	95% CI	*P*-value
Infant feeding (1st year): *Breastfeeding < 6 months vs Bottle*	0.593	0.492–0.716	<0.001[a]
Infant feeding (1st year): *Breastfeeding > 6 months vs Bottle*	0.749	0.630–0.890	0.001[a]
Infant feeding (1st year): *Mixed vs Bottle*	1.155	0.944–1.412	0.161
Parental marital status: *Separated/Divorced vs Married*	1.587	1.373–1.834	<0.001[a]
Parental marital status: *Widow vs Married*	1.629	1.079–2.462	0.020[a]
Residence climate: *Mountain vs Coastal*	1.002	0.849–1.182	0.984
Residence climate: *Inland vs Coastal*	0.981	0.865–1.113	0.764
Cat exposure: *Yes vs No*	1.71	1.22–2.40	0.002[a]
Parental consanguinity: *Yes vs No*	0.951	0.842–1.074	0.415
Parental atopy: *Yes vs No*	0.925	0.794–1.077	0.316
Family history of atopy: *Yes vs No*	1.030	0.912–1.163	0.634
Sex: *Female vs Male*	1.089	0.972–1.220	0.141

Notes: [a] Significant association. [b] Negative binomial (NB) model with risk ratio adjusted (aRR) for age group, gender, nationality, family history, animal exposure.

Discussion

AD is among the most common chronic inflammatory skin diseases of childhood and substantially affects quality of life, psychosocial well-being, and healthcare utilization.[18,19] Its pathogenesis reflects complex interactions between genetic susceptibility and environmental modifiers. Loss-of-function filaggrin variants compromise barrier function and promote allergen penetration, while environmental and psychosocial factors influence disease expression and control.[20–22] In this context, our study examined how early-life feeding, family structure, climate, animal exposure, and atopic heredity relate to AD severity in a Middle Eastern pediatric population. Breastfeeding and coastal residence were associated with milder disease, whereas non-intact households and mountain residence predicted greater severity. Other factors, including sex, consanguinity, and parental/family atopy, showed no independent associations.

The main contribution of this work is the integrated, severity-focused evaluation of modifiable determinants in a multicenter Middle Eastern pediatric cohort using SCORAD with adjusted effect estimates, highlighting a species-specific pet signal (cats) that is not captured by composite "any animal" exposure.

Breast-Feeding Practices and AD Risk

The protective association of breastfeeding against greater AD severity in our cohort is consistent with several prospective studies and meta-analyses linking exclusive or prolonged breastfeeding to a reduced risk of AD.[23–25] A meta-analysis of 18 prospective cohorts reported that exclusive breastfeeding for at least three months lowered the odds of childhood AD (OR = 0.68, 95% CI 0.52–0.88).[23] Swedish cohort data also showed a protective effect on eczema outcomes.[24] A study in Qatar found a lower prevalence of AD among breastfed infants,[25] and an Iranian study reported fewer early-childhood eczema symptoms with longer breastfeeding.[26]

However, other studies have reported inconsistent findings. A cluster-randomized trial of prolonged exclusive breastfeeding showed no significant reduction in eczema risk,[27] and analyses from ISAAC Phase II found a protective effect of ever-breastfeeding on severe eczema but no clear benefit of exclusive breastfeeding for ≥4 months on overall eczema risk.[27] Likewise, a German birth cohort suggested that breastfeeding did not prevent eczema in genetically predisposed children.[28] Some studies have even reported higher AD risk among children breastfed for ≥ 6 months in New Zealand,[29] and greater eczema

prevalence among exclusively breastfed Japanese adolescents.[30] These discrepancies likely reflect differences in study design, outcome definitions, and the possibility of reverse causation. In this context, our study adds to the literature by focusing on disease severity rather than disease occurrence, suggesting that even if breastfeeding does not uniformly prevent AD, it may still attenuate its clinical expression through immunomodulatory and microbiota-shaping effects.[31]

Psychosocial and Environmental Factors

Children from non-intact families in our study had markedly higher odds of greater AD severity, and this association remained robust after adjustment. The link between psychosocial stress and AD is biologically plausible given the influence of neuroimmune pathways and stress-mediated barrier dysfunction.[32] Survey data from Europe showed that single parents of children with AD report heightened stress and helplessness related to scratching behavior, which may worsen disease control and adherence.[33] Our findings support the view that family disruption and caregiver strain contribute to worse disease expression and align with evidence that minority or socioeconomically disadvantaged children bear a higher and more persistent AD burden.[34] Clinically, these results argue for integrating caregiver support, stress management, and psychosocial assessment into care plans for children with persistent or severe AD.

Environmental context further shaped severity in our cohort. The greater severity in mountain compared with coastal regions is consistent with ecological work linking drier, cooler, low-humidity conditions with higher eczema prevalence and poorer control.[35] Data from the United States similarly show that warmer temperatures, higher humidity, and greater ultraviolet exposure are associated with lower eczema prevalence, whereas colder and drier climates correlate with higher rates.[35] Additional registry data indicate that warm, humid, high-sunlight regions may still be associated with poor control in children already diagnosed with AD, likely due to heat- and sweat-induced flares.[36] In our sensitivity analysis, climate did not significantly affect continuous SCORAD, suggesting an influence on severity category thresholds rather than incremental score changes. The consistent disadvantage in mountain climates underscores the need for hydration-focused skin care and anticipatory counseling.[37] However, our climatic categories were region-based rather than derived from patient-level meteorologic measurements, so these findings should be interpreted as reflecting broad environmental context rather than precise humidity or temperature exposure.

Other Determinants

Parental and extended-family atopy were not associated with higher AD severity after adjustment, suggesting that once disease is established, environmental and psychosocial modifiers may outweigh genetic background. Because all participants in this cohort had a confirmed diagnosis of AD, the underlying level of genetic susceptibility was likely already enriched, which may have reduced the discriminatory value of familial atopic history for predicting severity within this selected population. In this context, range restriction may partly explain why parental or extended-family atopy did not remain associated with greater severity after adjustment. The lack of association with consanguinity is consistent with AD's polygenic nature[20] and with observations from related regional dermatologic literature.[38,39] Unlike classical autosomal recessive disorders, AD has a complex polygenic architecture with multifactorial environmental and immunologic influences; therefore, consanguinity would not necessarily be expected to increase disease severity in a predictable manner. Sex differences were not observed—matching mixed findings from pediatric studies—though adult AD often shows female predominance.[40] In contrast, animal exposure showed a more nuanced pattern: "any pet" exposure was not associated with severity in adjusted models, in line with earlier systematic review data indicating that pet ownership does not consistently increase or reduce eczema risk,[41] whereas specific cat ownership emerged as an independent determinant of greater severity in both ordinal and negative binomial models in our cohort. This finding should be interpreted cautiously, as pet-keeping practices may also reflect reverse causation or avoidance behavior; for example, some families may avoid keeping dogs because of pre-existing allergic or atopic concerns, while cats are generally more commonly accepted as household pets in the local cultural context. This is broadly compatible with meta-analytic findings that early exposure to dogs or pets overall may modestly reduce the risk of incident AD, with no clear harmful effect of cats at the population level,[42] but also with cohort and school-based studies showing that cat ownership in cat-sensitized children markedly increases eczema risk while dog ownership appears protective.[43,44] More recent birth-cohort data

also report generally weak overall associations between early-life animal exposure and childhood AD, with a tendency toward lower risk among dog-exposed children.[45] Taken together, these data and our results support a species-specific and phenotype-specific interpretation: in children with established AD, ongoing cat exposure may sustain higher allergen load and inflammatory activity, contributing to more severe clinical expression, whereas the apparently milder pattern among bird-exposed children in our sample likely reflects small numbers and residual confounding rather than a true protective effect. The borderline association with nationality may further indicate unmeasured socioeconomic or access-to-care differences.[34] The higher proportion of severe AD among non-Saudi children may also reflect differences in health-care access, care-seeking patterns, treatment continuity, living conditions, or other unmeasured social determinants rather than nationality itself as a biologic factor.

Clinical Implications, Strengths, and Limitations

Strengths of this study include a large, geographically diverse pediatric sample, the use of a validated clinical severity measure (SCORAD), and complementary ordinal and negative binomial models that yielded generally consistent findings. However, several limitations should be acknowledged. First, the cross-sectional design precludes causal inference and limits interpretation of temporal relationships between exposures and AD severity. Second, several exposure variables—including household animal exposure, and residential information—were based on caregiver report and may therefore be subject to recall bias or non-differential misclassification. Although household animal exposure was defined as pet exposure for at least 6 months at the time of diagnosis, more detailed data on exposure intensity, frequency, indoor versus outdoor exposure, and allergen burden were not available. Third, the climatic categories were based on broad geographic classification of the reported city of residence rather than direct patient-level environmental measurements; thus, we lacked objective data on ambient temperature, humidity, seasonal variation, indoor environmental exposures such as mold or household humidity, and pollution levels. Fourth, important contextual variables such as socioeconomic status, parental education, urban versus rural residence, skin care practices, and treatment use were not systematically measured, and these unmeasured factors may have contributed to residual confounding. Fifth, we did not assess allergic sensitization or specific IgE reactivity, including sensitization to cat dander, which limits mechanistic interpretation of the observed association between cat exposure and greater AD severity. Finally, although SCORAD assessments were performed by board-certified dermatologists, inter-observer reliability was not formally evaluated across centers. These limitations should be considered when interpreting the associations observed in this study.

Conclusion

This cross-sectional study of 600 children with atopic dermatitis identified three key determinants of clinical severity: infant feeding practices, parental marital status, and residential climate. Compared with bottle-feeding, breastfeeding was consistently associated with milder disease, and this protective effect was also evident when SCORAD was analyzed as a continuous outcome. Children from non-intact households (separated, divorced, or widowed) exhibited higher severity, likely reflecting the impact of psychosocial stress and care complexity. Residence in mountain regions was also associated with greater disease severity than coastal areas, underscoring the role of low humidity and cooler climates in aggravating barrier dysfunction.

In contrast, sex, parental consanguinity, family or parental atopy, and general animal exposure were not significantly associated with severity after adjustment, although cat exposure specifically emerged as an independent determinant of higher AD severity. Together, these findings support a holistic, context-aware approach to managing pediatric AD—one that promotes breastfeeding where feasible, incorporates family stress assessment and psychosocial support into eczema care, and tailors' skincare regimens to local environmental and climatic conditions.

Statement of Ethics

Ethical approval for this study was obtained from the Institutional Review Board (IRB) of the College of Medicine at Taibah University on April 5, 2025 (ID: TU-25-033). Written informed consent was obtained from parents or legal guardians, with child assent when appropriate. The study adhered to the principles of confidentiality and data protection throughout all stages

of data collection, storage, and analysis. No personally identifiable information was recorded, and participation was entirely voluntary, with all participants informed of the study's aims and their right to withdraw at any time without consequence.

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Author Contributions

Both authors made a significant contribution to the work reported, whether that is in the conception, study design, execution, acquisition of data, analysis and interpretation, or in all these areas; took part in drafting, revising or critically reviewing the article; gave final approval of the version to be published; have agreed on the journal to which the article has been submitted; and agree to be accountable for all aspects of the work. The data that support the findings of this research are available from the corresponding author upon reasonable request. Due to legal and ethical considerations, the data cannot be made publicly available. Requests for data access should be directed to the corresponding author.

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Disclosure

The authors declare no conflicts of interest in this work.

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