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ORIGINAL ARTICLE

Prospective Analysis of Environmental Triggers in Atopic Dermatitis Severity

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ABSTRACT:

Aim: This study aims to evaluate the impact of environmental triggers on the severity of atopic dermatitis (AD) through a prospective observational analysis conducted over 12 months. **Materials and Methods:** A total of 100 patients with AD, aged 18 to 65 years, were recruited based on the American Academy of Dermatology criteria. Baseline assessments included detailed medical histories and questionnaires identifying environmental exposures, such as allergens, pollutants, and climatic factors. The severity of AD was measured using the Eczema Area and Severity Index (EASI) score. Monthly follow-ups documented changes in EASI scores and flare-ups. Data analysis involved correlation and multivariable regression to identify significant environmental predictors of AD severity. **Results:** The study population comprised 50 males and 50 females, with a mean age of 38.5 years (±12.7). Most participants (72%) reported a family history of atopy. Dust mites (68%) and air pollution (81%) were the most frequently reported triggers, both showing significant associations with AD severity ($p = 0.02$ and $p < 0.01$, respectively). Over 12 months, participants exposed to dust mites experienced smaller reductions in EASI scores (-2.8 ± 1.6) compared to those with stable environmental conditions (-2.9 ± 1.8). Correlation analysis identified PM2.5 levels ($r = 0.48$, $p < 0.001$) and dust mite exposure ($r = 0.41$, $p < 0.001$) as significant contributors to disease severity. Multivariable regression confirmed PM2.5 levels as the strongest predictor (beta = 0.39, $p < 0.001$). **Conclusion:** Environmental triggers such as air pollution, allergens, and climatic variations significantly influence AD severity. Mitigating these exposures through individualized care and public health interventions can reduce disease burden and improve outcomes for individuals with AD.

Keywords: Atopic dermatitis, environmental triggers, air pollution, allergens, EASI score.

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INTRODUCTION

Atopic dermatitis (AD) is a chronic inflammatory skin condition characterized by intense itching, recurrent flares, and a significant impact on the quality of life. It affects individuals of all ages, with an increasing prevalence in both developed and developing nations. The pathophysiology of AD is multifactorial, involving a complex interplay of genetic predisposition, immune dysregulation, and environmental factors. Among these, environmental triggers play a crucial role in influencing disease severity, acting as initiators or exacerbators of flares in susceptible individuals.[1]Environmental factors are ubiquitous and diverse, encompassing allergens, climatic variations, pollution, and lifestyle-related exposures. These triggers often interact with an impaired skin barrier and an overactive immune system, common features in individuals with AD. The compromised epidermal barrier, characterized by reduced levels of filaggrin and other structural proteins, allows environmental irritants and allergens to penetrate the skin more easily, initiating or worsening inflammation. Understanding these interactions is pivotal for managing AD effectively and reducing the burden of this condition.[2]One of the most common environmental triggers for AD is exposure to allergens, particularly house dust mites, pet dander, molds, and pollens. These allergens can directly irritate the skin or provoke an immune response, leading to inflammation and worsening of symptoms. Dust mites, for instance, are known to release proteolytic enzymes that can degrade skin proteins, exacerbating barrier dysfunction and triggering inflammatory cascades. Similarly, exposure to pet dander or molds in indoor environments can contribute to persistent or recurrent flares, particularly in patients sensitized to these allergens. Identifying and minimizing exposure to specific allergens is therefore a key component of personalized AD management.[3]Climatic factors such as temperature fluctuations, humidity levels, and seasonal variations also significantly influence AD severity. Extreme temperatures, whether hot or cold, can disrupt the skin's homeostasis by altering its hydration and lipid content. High humidity levels may exacerbate sweat-related irritation, while low humidity can lead to increased dryness and barrier dysfunction. Moreover, seasonal transitions, such as the shift from winter to spring or summer to fall, are often associated with increased environmental allergen loads, further aggravating AD symptoms. These climatic influences underscore the importance of adapting skincare routines and environmental controls to seasonal changes.[4]Air pollution, particularly exposure to

particulate matter (PM2.5 and PM10), is another critical environmental factor linked to AD severity. Fine particulate matter can penetrate the skin barrier, induce oxidative stress, and provoke an inflammatory response. Urban environments with high levels of air pollution are frequently associated with higher rates of AD exacerbations, highlighting the impact of external pollutants on skin health. Additionally, exposure to volatile organic compounds (VOCs) and tobacco smoke can further aggravate symptoms by disrupting skin integrity and enhancing immune activation.[5]Lifestyle and behavioral factors also contribute to the environmental burden on individuals with AD. Frequent use of harsh soaps or detergents, excessive bathing, and wearing irritant fabrics such as wool can exacerbate skin barrier dysfunction. Stress, often overlooked as an environmental trigger, has a bidirectional relationship with AD, where psychological stress can worsen symptoms, and severe symptoms can, in turn, elevate stress levels. These lifestyle-related triggers emphasize the need for comprehensive patient education on maintaining a supportive environment for their skin.[6]In addition to physical environmental factors, microbial exposure plays a role in the course of AD. Colonization or infection with Staphylococcus aureus, a common finding in individuals with AD, exacerbates inflammation and delays healing. The interaction between microbial exposure and the host immune system is a crucial aspect of environmental influences on AD severity. Strategies to manage or modify microbial exposure, such as the use of antimicrobial treatments or probiotics, are emerging areas of interest in AD management.[7]Understanding the role of environmental triggers in AD severity is essential not only for tailoring management strategies but also for guiding public health interventions. Environmental control measures, such as allergen avoidance, improving indoor air quality, and using appropriate skincare products, have the potential to significantly reduce disease burden. Moreover, public policies aimed at reducing air pollution and mitigating climate change could have far-reaching benefits for individuals with AD and other chronic conditions exacerbated by environmental factors.[8]Despite advances in understanding the impact of environmental triggers on AD, challenges remain. The heterogeneity of triggers across individuals, variations in geographic and cultural contexts, and the dynamic nature of environmental exposures make it difficult to establish universal recommendations. Nevertheless, identifying and addressing modifiable environmental factors offers a tangible opportunity to improve outcomes for patients with AD. By integrating environmental considerations into the broader framework of AD care, clinicians and researchers can enhance the effectiveness of treatment plans and contribute to a better understanding of this complex condition.

MATERIALS AND METHODS

This study was designed as a prospective observational analysis to evaluate the impact of environmental triggers on the severity of atopic dermatitis (AD). It was conducted over a 12-month period at a tertiary care dermatology center.

A total of 100 patients diagnosed with atopic dermatitis, based on the American Academy of Dermatology criteria, were recruited. The study population included 50 males and 50 females, aged between 18 and 65 years. Patients with other chronic dermatological or systemic conditions, those receiving immunosuppressive therapy, or those unable to provide informed consent were excluded.

The study was approved by the institutional ethics committee, and written informed consent was obtained from all participants before enrollment.

For data collection, a baseline assessment was performed, including a detailed medical history that captured the duration and previous treatment of AD. Participants completed a comprehensive questionnaire to identify potential environmental exposures, including allergens, pollutants, climatic factors, and lifestyle habits. The severity of AD was evaluated using the Eczema Area and Severity Index (EASI) score.

Environmental triggers were assessed based on self-reported exposure to irritants such as dust mites, pet dander, mold, pollen, and climatic variations, including temperature and humidity changes. Real-time environmental data on temperature, humidity, and air pollution (PM2.5, PM10, and AQI) were obtained from local meteorological and pollution monitoring stations to complement self-reported data.

Participants were followed up monthly for 12 months. At each follow-up visit, changes in EASI scores and reports of flare-ups were documented. To ensure comprehensive data, participants maintained a daily symptom diary, recording their exposure to environmental triggers, symptom severity, and use of medications or topical treatments.

The data were analyzed using SPSS 25.0. Continuous variables were expressed as mean ± standard deviation, and categorical variables as percentages. The correlation between environmental factors and changes in AD severity was evaluated using Pearson or Spearman correlation coefficients. Multivariable regression analysis was conducted to identify independent environmental predictors of AD severity. A p-value of <0.05 was considered statistically significant.

RESULTS

Table 1: Demographic and Baseline Characteristics of Study Population

The mean age of the participants was 38.5 years (±12.7), reflecting a broad distribution across adulthood. The study had an equal gender distribution (50% male, 50% female), and there was no significant gender-based difference in the study outcomes (p =

0.75). The mean duration of atopic dermatitis (AD) was 8.2 years (±5.4), indicating that the study cohort largely consisted of patients with chronic disease. A significant majority of participants (72%) had a family history of atopy ($p < 0.01$), underscoring the genetic predisposition in AD. Regarding prior treatment, 76% had used topical steroids, while 24% had received systemic therapy, with a statistically significant difference ($p = 0.04$). The baseline Eczema Area and Severity Index (EASI) score was 14.6 (±5.8), reflecting moderate disease severity on average.

Table 2: Environmental Trigger Exposure Frequency

Exposure to environmental triggers varied among participants. Dust mites were the most common trigger, reported by 68% of patients, with a statistically significant association with AD ($p = 0.02$). Other allergens, such as pet dander (45%) and mold (37%), showed weaker associations ($p = 0.10$ and $p = 0.15$, respectively). Pollen exposure was reported by 53% of participants, but the p-value (0.08) suggested a trend rather than a significant association. Climate-related triggers, including temperature fluctuations (72%, $p = 0.01$) and humidity changes (60%, $p = 0.03$), were significantly linked to AD severity. Air pollution (PM2.5/PM10) had the highest frequency of exposure (81%) and demonstrated a strong association with AD severity ($p < 0.01$), highlighting its role as a prominent environmental factor.

Table 3: EASI Score Changes Over 12 Months by Environmental Trigger Exposure

Patients exposed to dust mites had a higher mean baseline EASI score (15.2 ± 5.3) compared to those not exposed (13.1 ± 6.2). Over 12 months, the EASI scores decreased by an average of 2.8 ± 1.6 points in those with dust mite exposure and by 2.6 ± 1.9 points in those without, suggesting a slightly greater reduction among those exposed. Temperature fluctuations were associated with smaller improvements in EASI scores (-1.8 ± 1.5) compared to patients in stable temperature conditions (-2.9 ± 1.8). These findings suggest that exposure to environmental triggers might modulate the rate of improvement in AD severity over time.

Table 4: Correlation Between Environmental Factors and AD Severity

This table highlights the strength of association between environmental factors and AD severity. PM2.5 levels showed the strongest positive correlation with AD severity ($r = 0.48$, $p < 0.001$), indicating that higher pollution levels were associated with worse disease outcomes. Dust mite exposure also had a strong correlation ($r = 0.41$, $p < 0.001$), followed by temperature fluctuations ($r = 0.35$, $p < 0.01$) and humidity changes ($r = 0.28$, $p = 0.02$). These results confirm that both allergen exposure and environmental conditions are significant contributors to AD severity.

Table 5: Multivariable Regression Analysis: Predictors of AD Severity

In the regression analysis, PM2.5 levels emerged as the strongest independent predictor of AD severity, with a beta coefficient of 0.39 ($p < 0.001$). Dust mite exposure also significantly predicted severity (beta = 0.25, $p < 0.01$), followed by temperature fluctuations (beta = 0.21, $p = 0.02$) and humidity changes (beta = 0.15, $p = 0.05$). These findings underscore the multifactorial nature of AD, where both indoor and outdoor environmental factors contribute to disease outcomes.

Table 1: Demographic and Baseline Characteristics of Study Population

Characteristic	Number (N=100)	Percentage (%)	p-value
Mean Age (years) (Mean ± SD)	38.5 ± 12.7	-	-
Gender			0.75
Male	50	50%	
Female	50	50%	
Duration of AD (Mean ± SD) (years)	8.2 ± 5.4	-	-
Family history of atopy			<0.01
Yes	72	72%	
No	28	28%	
Previous treatment for AD			0.04
Topical steroids	76	76%	
Systemic therapy	24	24%	
Baseline EASI score (Mean ± SD)	14.6 ± 5.8	-	-

Table 2: Environmental Trigger Exposure Frequency

Trigger	Number (N=100)	Percentage (%)	p-value
Dust mites	68	68%	0.02
Pet dander	45	45%	0.10
Mold	37	37%	0.15

Pollen	53	53%	0.08
Temperature fluctuations	72	72%	0.01
Humidity changes	60	60%	0.03
Air pollution (PM2.5/PM10)	81	81%	<0.01

Table 3: EASI Score Changes Over 12 Months by Environmental Trigger Exposure

Trigger	Mean Baseline EASI ± SD	Mean 12-Month EASI ± SD	Mean Change ± SD
Dust mites exposure	15.2 ± 5.3	12.4 ± 4.7	-2.8 ± 1.6
No dust mites exposure	13.1 ± 6.2	10.5 ± 5.1	-2.6 ± 1.9
Temperature fluctuation	15.0 ± 4.9	13.2 ± 4.3	-1.8 ± 1.5
Stable temperature	14.2 ± 6.1	11.3 ± 5.0	-2.9 ± 1.8

Table 4: Correlation Between Environmental Factors and AD Severity

Environmental Factor	Correlation Coefficient (r)	p-value
PM2.5 Levels	0.48	<0.001
Temperature fluctuations	0.35	<0.01
Humidity changes	0.28	0.02
Dust mite exposure	0.41	<0.001

Table 5: Multivariable Regression Analysis: Predictors of AD Severity

Predictor	Beta Coefficient	p-value
PM2.5 levels	0.39	<0.001
Dust mite exposure	0.25	<0.01
Temperature fluctuations	0.21	0.02
Humidity changes	0.15	0.05

DISCUSSION

The demographic characteristics of this study, including the mean age of 38.5 years and an equal gender distribution, align with findings from Simpson et al. (2012), who reported similar age and gender distributions in adult AD cohorts. Simpson's study also highlighted the chronic nature of AD, with a mean disease duration of 10 years, corroborating the findings of this study's mean disease duration of 8.2 years. The significant association between family history of atopy and AD severity observed here (72%, $p < 0.01$) is consistent with Simpson's conclusion that genetic predisposition plays a key role in AD pathogenesis, underscoring the hereditary nature of atopy.[9]

Exposure to environmental triggers like dust mites, which were reported by 68% of participants in this study, supports the findings of Arlian et al. (2014). Their study identified house dust mites as a prevalent allergen and a significant contributor to AD severity. However, while Arlian et al. emphasized pet dander as another critical factor, our study found a weaker association (45%, $p = 0.10$). The strong association between PM2.5 exposure and AD severity (81%, $p < 0.01$) in this study is consistent with the findings of this study, reinforcing the growing concern over air pollution as a driver of AD exacerbations.[10]

The results showing smaller improvements in EASI scores in participants exposed to temperature fluctuations align with findings by Thyssen et al. (2015). Thyssen's study found that extreme temperatures and abrupt changes in climate exacerbated AD symptoms by disrupting skin barrier function. The greater EASI reduction in participants under stable temperature conditions (-2.9 ± 1.8) observed in this study corroborates the benefit of consistent environmental conditions in mitigating AD symptoms. These findings underscore the importance of managing environmental conditions to improve long-term disease outcomes.[11]

The significant correlations between PM2.5 levels ($r = 0.48$, $p < 0.001$) and dust mite exposure ($r = 0.41$, $p < 0.001$) with AD severity parallel the results of Lee et al. (2013), who also found that particulate matter pollution was a key factor aggravating AD. Lee's study identified that PM2.5 not only induces inflammation but also impairs skin barrier function, similar to this study's observations. The association between temperature fluctuations and AD severity ($r = 0.35$, $p < 0.01$) also supports Lee's findings that climate variability exacerbates skin conditions.[12]

Finally, the multivariable regression analysis highlights PM2.5 levels as the strongest predictor of AD severity, a finding supported by Kim et al. (2015). Their study demonstrated that higher exposure to air pollution is independently associated with worse AD outcomes, emphasizing the need for preventive measures in areas with high pollution levels. The association between dust mites (beta = 0.25, $p < 0.01$) and AD severity is consistent with their findings, which suggest allergen mitigation as a key intervention strategy for managing AD. The relatively weaker associations of temperature and humidity changes with AD severity (beta = 0.21 and 0.15,

respectively) indicate that while these factors are relevant, they are less impactful compared to air pollution and allergen exposure.[13]

CONCLUSION

In conclusion, environmental triggers play a significant role in the severity and progression of atopic dermatitis by interacting with genetic predisposition and immune dysregulation. Factors such as allergens, climatic variations, air pollution, and lifestyle choices exacerbate symptoms and impact disease management. Identifying and mitigating these triggers through individualized care, environmental control measures, and public health interventions can significantly reduce disease burden. Further research into the mechanisms of these interactions will enhance targeted treatment strategies and improve the quality of life for individuals with AD.

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