

Research Articles**Open Access**

Environmental Exposures, Clinical Allergy Symptoms, and Serum IgE in Young Adults: A Cross-Sectional Study from Makassar, Indonesia

Andi Khairul Anaam¹, Liong Boy Kurniawan^{1,2}, Sitti Rafiah^{1,3}, Andi Alfian Zainuddin^{1,4}, Uleng Bahrun^{1,2*}

¹Master of Biomedical Sciences, Postgraduate Program, Hasanuddin University, Makassar, South Sulawesi, Indonesia

²Department of Clinical Pathology Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia

³Department of Anatomy, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, 90245, Indonesia

⁴Department of Public Health Sciences, Faculty of Medicine, Hasanuddin University, Makassar, South Sulawesi, Indonesia

***Corresponding Author:** Email: ulengbahrun2024@gmail.com

ARTICLE INFO

Manuscript Received: 05 Jun, 2025

Revised: 10 Oct, 2025

Accepted: 15 Oct, 2025

Date of Publication: 04 Nov, 2025

Volume: 8

Issue: 11

DOI: [10.56338/mppki.v8i11.8279](https://doi.org/10.56338/mppki.v8i11.8279)

KEYWORDS

Immunoglobulin E;
Allergy;
Environmental Exposure;
Young Adults

ABSTRACT

Introduction: The global prevalence of allergic diseases has increased significantly, particularly among young adults. Immunoglobulin E (IgE) plays a central role in the pathophysiology of allergic responses and is considered a crucial biomarker in evaluating allergic sensitivity. However, the influence of environmental exposures and clinical complaints on serum IgE levels in adult populations remains understudied, especially in tropical developing countries.

Methods: This study aimed to analyze the relationship between environmental factors and clinical complaints with serum IgE levels in a population of healthy young adults in Makassar, Indonesia. A cross-sectional study was conducted involving 80 healthy adults aged 18–40 years. Environmental exposures and clinical symptoms were analyzed against serum IgE levels. Non-parametric tests (Mann-Whitney U and Kruskal-Wallis) showed no significant associations. However, in the multivariate regression model, humidity was the only environmental factor significantly associated with serum IgE concentration.

Results: The mean serum IgE level among participants was 176.8 IU/mL ($SD \pm 196.9$), with a wide distribution. No statistically significant associations were found between serum IgE levels and clinical complaints such as asthma, allergic rhinitis, and atopic dermatitis ($p > 0.05$). Similarly, environmental exposures including cigarette smoke, dust, pet dander, and poor ventilation were not significantly related to IgE levels. Notably, high environmental humidity was negatively associated with serum IgE levels ($p = 0.047$). Most environmental factors and clinical complaints assessed in this study were not significantly associated with serum IgE concentrations in healthy young adults.

Conclusion: The observed inverse association with high humidity suggests a potential modulatory role that warrants further investigation. Future longitudinal studies with larger samples are recommended to confirm these findings and provide stronger evidence to inform public health strategies in allergy prevention.

Publisher: Fakultas Kesehatan Masyarakat Universitas Muhammadiyah Palu

INTRODUCTION

The global incidence of allergic diseases has significantly increased over the past few decades and is now recognized as a growing public health concern. One of the primary environmental contributors to this trend is air pollution, particularly exposure to fine particulate matter (PM2.5) (1, 2). Recent studies have demonstrated that such pollutants can exacerbate allergic symptoms including asthma by enhancing immune responses mediated by Immunoglobulin E (IgE) (3). These findings emphasize the direct role of environmental conditions in modulating immune activity and allergic sensitization. Immunoglobulin E (IgE) is a key antibody involved in type I hypersensitivity reactions and plays a central role in the body's immune defense against allergens and pathogens. Elevated serum IgE levels are strongly associated with various allergic conditions, including asthma, allergic rhinitis, atopic dermatitis, food allergies, and even life-threatening anaphylactic reactions (4). The clinical manifestations of allergies can involve multiple organ systems, including the skin, respiratory tract, and gastrointestinal system, making IgE a widely accepted biomarker for allergic activity. Over time, the prevalence of allergic diseases in adults has shown a marked upward trend. This rise is not solely attributed to genetic predisposition but is also closely linked to environmental and lifestyle changes (5). The rapid pace of industrialization and urbanization has intensified human exposure to allergens and pollutants, thus increasing the population's susceptibility to allergic reactions. According to the World Allergy Organization (WAO), over 300 million people suffer from asthma globally, and approximately 250 million more are affected by other allergic disorders. These figures are expected to rise, with asthma alone projected to affect 400 million individuals by 2025 (5-7). In Indonesia, the 2018 National Health Survey (Riskesdas) reported an asthma prevalence of 2.4%, largely influenced by environmental and behavioral determinants (8). Environmental conditions such as exposure to airborne pollutants, domestic allergens (e.g., dust mites, mold, pet dander), smoking, high humidity, and poor ventilation are well-documented factors contributing to increased serum IgE levels (1). These factors facilitate prolonged contact with allergens, especially in enclosed spaces, thereby triggering exaggerated immune responses in sensitized individuals. Additionally, clinical complaints such as respiratory distress, chronic coughing, skin rashes, and gastrointestinal discomfort are commonly linked with elevated IgE levels (9), reinforcing its relevance in allergic disease diagnostics and monitoring (4, 10).

Among young adults, immune responses remain robust and relatively stable compared to older age groups. However, this demographic is frequently subjected to diverse and dynamic environmental exposures due to increased mobility and social activity. As such, serum IgE measurements in young adults provide valuable insights into the subclinical effects of environmental factors on immune function (4, 11). In addition, the immune system in this age group is still highly responsive, making it suitable for detecting early immunological shifts that may not yet present clinically.

Furthermore, the hygiene hypothesis provides an additional framework to understand the rise in allergic sensitization. It postulates that reduced microbial exposure in early life, due to improved sanitation and widespread antibiotic use, may impair immune tolerance and promote Th2-dominant immune profiles that favor increased IgE production (12-14). In adulthood, persistent environmental exposure to allergens may continue to modulate immune responses and maintain elevated IgE levels, even in the absence of active symptoms. Considering the complex interactions between environmental exposures, clinical manifestations, and immunological mechanisms, it is crucial to investigate their combined impact on serum IgE concentrations (15-17). Understanding these relationships will not only aid in the early detection of allergic risk but also inform preventive strategies aimed at reducing the overall disease burden in the community. Based on this rationale, the present study aims to examine the association between environmental exposures and clinical allergic complaints with serum IgE levels among healthy young adults.

METHOD

This study utilized a cross-sectional analytical design to evaluate the association between environmental exposures and clinical allergic complaints with serum Immunoglobulin E (IgE) levels. The cross-sectional approach enabled the simultaneous assessment of exposure variables and immunological outcomes without manipulating the study environment, allowing for the identification of correlations at a single point in time.

Research Type

The research is categorized as quantitative observational analytic research, specifically employing a cross-sectional design. This design is appropriate for identifying potential associations between variables in a defined population at a specific time.

Population and Sample/Informants

The target population comprised adults aged 18 to 40 years residing in Makassar, South Sulawesi, Indonesia. This age range was selected to represent physiologically active individuals likely to be exposed to various environmental allergens. A total of 80 respondents were recruited using a non-probability purposive sampling technique, with inclusion criteria being: aged 18–40 years, clinically healthy, and willing to provide informed consent. Exclusion criteria included pregnancy, breastfeeding, autoimmune disease, malignancy, or ongoing immunosuppressive therapy (18).

Research Location

The study was conducted in Makassar, South Sulawesi, with collaboration from Hasanuddin University Hospital (RSUH) and Dr. Wahidin Sudirohusodo Hospital (RSWS). Laboratory analyses were carried out in a certified clinical laboratory to ensure standardization and analytical accuracy.

Instrumentation or Tools

A structured questionnaire designed to assess exposure to environmental risk factors such as cigarette smoke, household dust, pet dander, and indoor air quality, as well as self-reported allergic complaints (asthma, allergic rhinitis, atopic dermatitis). Serum IgE measurements, obtained through the Electrochemiluminescence Immunoassay (ECLIA) method. All biochemical analyses were conducted in certified laboratories in accordance with standard operating procedures to ensure result validity and reliability.

Data Collection Procedures

Data collection was conducted within a predefined period and followed a standardized protocol to ensure consistency and reliability. Prior to data collection, participants were informed about the study objectives, procedures, and potential risks through both oral and written explanations, and written informed consent was obtained from each respondent before participation. Participants who met the inclusion and exclusion criteria were recruited using a purposive sampling technique. While purposive sampling was deemed appropriate to capture individuals meeting the study objectives, it inherently limits the external validity and generalizability of the findings. This limitation is explicitly addressed in the discussion section to provide a balanced interpretation of the study outcomes.

Data Analysis

Data analysis was performed using the Statistical Package for the Social Sciences (SPSS) version 26. Descriptive statistics, including range, median, mean, standard deviation, and data distribution, were employed to summarize the characteristics of serum IgE levels. The normality of the data distribution was assessed using the Kolmogorov Smirnov test, with $p > 0.05$ interpreted as normally distributed and $p \leq 0.05$ as non-normally distributed. For comparative analyses, the independent *t*-test was applied when data met parametric assumptions (normal distribution and homogeneity of variances), whereas the Mann–Whitney U test was used for data that violated these assumptions. Similarly, comparisons between groups exposed and unexposed to environmental risk factors were analyzed using either the independent *t*-test or Mann Whitney U test according to the distributional characteristics. Differences in serum IgE levels across varying numbers of environmental exposures were assessed using the Kruskal–Wallis test, as the data did not meet parametric assumptions. To evaluate the partial and simultaneous influence of independent variables (environmental factors and clinical complaints) on the dependent variable (serum IgE level), multiple linear regression analysis was employed, contingent on residuals meeting assumptions of linearity, normality, and homoscedasticity. Statistical significance was determined at a *p*-value < 0.05 .

Ethical Approval

Ethical clearance was granted by the Health Research Ethics Committee (KEPK) of the Faculty of Medicine, Hasanuddin University, under reference number 14/UN4.6.4.5.31/PP36/2025. All participants provided written informed consent after receiving comprehensive explanations of the study's aims, procedures, and potential risks.

RESULTS

Participant Characteristics

A total of 80 participants were enrolled in this study. The majority were female (56.3%), while males comprised 43.8% of the study population. Most participants were within the age group of 18–30 years (72.5%), with the remaining 27.5% aged between 31–40 years. In terms of workplace environment, a significant proportion of the participants worked indoors (83.8%), whereas only 16.3% were employed in outdoor settings. These demographic and occupational characteristics were considered in subsequent analyses to evaluate their potential association with environmental exposures and immunological outcomes.

Table 1. Characteristics of Study Participants

Characteristic (N=80)	Category	n	%
Sex	Male	35	43.8%
	Female	45	56.3%
Age Group (years)	18–30	58	72.5%
	31–40	22	27.5%
Workplace Location	Indoor	67	83.8%
	Outdoor	13	16.3%

Source: Primary Data, 2025

Based on the demographic profile presented in Table 1, the majority of participants were female, aged 18–30 years, and predominantly employed in indoor occupational settings.

Environmental Exposure and Clinical Complaints

Based on the data presented in Table 2, dust exposure and poor ventilation were the most commonly reported environmental factors among participants, while exposure to cigarette smoke, high humidity, and pet dander was less prevalent. Although the majority of respondents did not report asthma or allergic rhinitis, atopic dermatitis was more frequently observed.

Table 2. Distribution of Participants by Environmental Exposure and Clinical Complaints

Variable (N=80)	Yes (n/%)	No (n/%)
Environmental Exposure		
Cigarette smoke	29 (36.3%)	51 (63.7%)
Dust	49 (61.3%)	31 (38.7%)
Pet dander	6 (7.5%)	74 (92.5%)
High humidity	44 (55.0%)	36 (45.0%)
Poor ventilation	27 (33.7%)	53 (66.3%)
Clinical Complaints		
Asthma	5 (6.3%)	75 (93.7%)
Allergic rhinitis	14 (17.5%)	66 (82.5%)
Atopic dermatitis	17 (21.3%)	63 (78.7%)

The analysis of Table 2 indicates that dust exposure and inadequate ventilation were the predominant environmental factors among participants, whereas exposure to tobacco smoke, elevated humidity, and animal dander occurred less frequently. Despite varying levels of environmental exposure, asthma and allergic rhinitis were relatively uncommon, whereas atopic dermatitis appeared more prevalent, suggesting a distinct pattern of atopic manifestation within the population. Environmental factors such as dust, inadequate ventilation, humidity, and

cigarette smoke are well-documented contributors to elevated serum IgE levels, which play a central role in allergic sensitization and immune system dysregulation (19) emphasized that exposure to indoor dust and poor air circulation increases the risk of respiratory disorders, while (20) found a direct association between cigarette smoke and elevated IgE concentrations. (21) further highlighted the relationship between house dust mite density and allergic manifestations, and Cachon et al. (2014) (22) demonstrated that indoor air pollution exacerbates airway inflammation. The higher prevalence of atopic dermatitis in this study may be linked to elevated indoor humidity, which has been shown to worsen immune responses and IgE production (23).

Analysis of Serum IgE Levels

As shown in Table 3, serum IgE levels among participants exhibited a wide distribution, with a range of 848.10 IU/mL. The median IgE concentration was 82.36 IU/mL, while the mean value reached 176.82 IU/mL.

Table 3. Descriptive Analysis of Serum IgE Levels

Variable (N=80)	Range (IU/mL)	Median (IU/mL)	Mean (IU/mL)	Standard Deviation (IU/mL)
IgE	848.10	82.36	176.82	196.89

Serum IgE Levels Based on Clinical Symptoms and Environmental Exposure

Based on the Mann Whitney U test, no statistically significant differences were observed in serum IgE levels between participants with and without a history of allergy ($p = 0.233$), despite higher mean IgE concentrations in the allergic group (195.57 IU/mL). Although no statistically significant differences were observed in serum IgE levels across groups with varying numbers of environmental exposures, the trend of higher mean IgE concentrations in participants exposed to multiple factors suggests a possible cumulative effect. This finding is consistent with the hypothesis that repeated or combined exposure to allergens and environmental risk factors may enhance immune sensitization, leading to elevated IgE production over time. However, the absence of statistical significance in this study may be related to sample size limitations or heterogeneity of individual responses. Future studies with larger cohorts and longitudinal designs are warranted to further explore the cumulative exposure hypothesis and its implications for allergic disease development.

Table 4. Serum IgE Levels Based on Clinical Symptoms and Environmental Exposure

Variable (N=80)	Serum IgE Level (IU/mL)			
	n (%)	Mean±SD	Median (min-max)	p-value
Number of exposure				
- Non exposure	4 (5,00%)	39,95 33,02±	33,59 (9,96-82,65)	
- One exposure	24 (30,00%)	201,38 182,79±	159,1 (7,56-559,80)	
- Two exposure	28 (35,00%)	173,90 217,79±	69,39 (3,90-852,00)	
- Three exposure	21 (26,25%)	154,97 156,29±	112,90 (11,74-536,40)	0,348 ^b
- Four exposures	3 (3,75%)	343,00 411,75±	201,70 (20,50-806,80)	
Environmental Factors				
Cigarette smoke				
Exposed	29 (36,25%)	219,32 250,05±	82,06 (3,90-852,00)	
Non exposed	51 (63,75%)	152,65 156,83±	82,65 (7,56-572,10)	0,545 ^a

- Dust				
Exposed	49 (61,25%)	204,86 218,55±	112,90 (3,90-852,00)	0,162 ^a
Non exposed	31 (38,75%)	132,50 149,49±	73,93 (4,05-559,80)	
- Animal fur				
Exposed	6 (7,5%)	192,92 309,23±	54,01 (12,72-806,80)	0,635 ^a
Non exposed	74 (92,5%)	175,51 188,10±	87,29 (3,90-852,00)	
- High humidity				
Exposed	44 (55,0%)	143,75 151,27±	86,99 (3,90-852,00)	0,262 ^a
Non exposed	36 (45,0%)	217,23 237,35±	81,68 (4,05-572,10)	
- Poor ventilation				
Exposed	27 (33,75%)	169,26 184,03±	134,20 (9,10-806,80)	0,939 ^a
Non exposed	53 (66,25%)	180,67 204,74±	80,71 (3,90-852,00)	
Workplace Location				
- Indoor	67 (83,75%)	178,70 204,72±	82,65 (3,90-852,00)	0,871 ^a
- Outdoor	13 (16,25%)	167,10 156,81±	75,28 (9,10-419,80)	
Notes:	^a Mann-Whitney	U	test;	^b Kruskal-Wallis
				test

Multiple Linear Regression Model

The results of the multiple linear regression analysis indicated that the overall model was not statistically significant, with an F-value of 1.095 and a p-value of 0.379. The F-test was conducted to assess whether the set of independent variables collectively exerted a significant effect on the dependent variable, namely serum IgE concentration (IU/mL). Given that the p-value exceeds the conventional threshold of 0.05, it can be concluded that the model does not explain a significant proportion of the variance in IgE levels. In other words, the independent variables included in the model do not significantly predict serum IgE concentrations when evaluated simultaneously.

Table 5. Summary of Multiple Linear Regression Model

Source	Sum of Squares	df	Mean Square	F-value	p-value
Regression	460,697.814	11	41,881.619	1.095	0.379
Residual	2,601,927.249	68	38,263.636		
Total	3,062,625.064	79			

Note: Simultaneous F-test of multiple linear regression

Associated with Serum IgE Levels

As shown in Table 6, most independent variables including allergy status, asthma, allergic rhinitis, atopic dermatitis, cigarette smoke exposure, pet dander, poor ventilation, and dust had p-values greater than 0.05, indicating no statistically significant association with serum IgE levels (9, 24). Although allergen exposure exhibited a positive trend ($p = 0.115$), the effect was not statistically significant. Notably, high humidity demonstrated a significant negative association with IgE levels ($p = 0.047$), suggesting that increased ambient moisture may suppress IgE

elevation. The workplace variable also showed a negative tendency ($p = 0.092$), yet did not reach statistical significance at the 0.05 level.

Table 6. Factors Associated with Serum IgE Levels

Variable (N=80)	B	95% CI (Lower–Upper)	t-value	p-value
Allergy history	0.044	-230.761 to 266.121	0.142	0.887
Asthma	0.017	-241.960 to 269.095	0.106	0.916
Allergic rhinitis	0.135	-152.893 to 292.064	0.624	0.535
Atopic dermatitis	-0.048	-231.024 to 185.487	-0.218	0.828
Allergen exposure	0.216	-48.559 to 436.562	1.596	0.115
Cigarette smoke	0.086	-69.123 to 138.816	0.669	0.506
Pet dander	-0.005	-174.575 to 167.056	-0.044	0.965
High humidity	-0.287	-223.899 to -1.504	-2.022	0.047
Poor ventilation	-0.033	-116.645 to 89.032	-0.268	0.790
Dust exposure	0.117	-59.620 to 153.671	0.880	0.382
Workplace (indoor/outdoor)	-0.239	-275.086 to 21.161	-1.710	0.092

Note: Multiple linear regression using partial t-test. Significant p-values (< 0.05) are bolded.

Based on the Mann Whitney U test, no statistically significant differences were observed in serum IgE levels between participants with and without a history of allergy ($p = 0.233$), despite higher mean IgE concentrations in the allergic group (195.57 IU/mL). Similarly, clinical complaints such as asthma, allergic rhinitis, and atopic dermatitis showed non-significant p-values of 0.713, 0.188, and 0.995, respectively. Although IgE levels tended to be higher in participants exposed to allergens compared to unexposed individuals, the difference did not reach statistical significance ($p = 0.098$). In addition, exposure to specific environmental factors including cigarette smoke, dust, pet dander, high humidity, and poor ventilation also yielded non-significant associations with IgE levels ($p > 0.05$). The Kruskal–Wallis test for the number of allergen exposures showed no significant differences among groups ($p = 0.348$). However, descriptively, the highest median IgE level (201.70 IU/mL) was observed in participants exposed to four types of allergens, while the lowest median level (33.59 IU/mL) was recorded in those with no allergen exposure. Table 4 indicates that although there was a trend toward elevated serum IgE levels in individuals exposed to allergens and environmental factors, these differences were not statistically significant. This outcome may be attributed to uncontrolled variables such as genetic predisposition, unmeasured environmental exposures, and individual immunologic variability. Prior studies by (15) (17, 23) have similarly emphasized that while IgE elevation is often associated with allergen exposure, immune responses are multifactorial and can be influenced by a complex interplay of biological and environmental determinants.

DISCUSSION

The study involved 80 young adult participants aged between 18 and 40 years. All respondents underwent a series of preliminary assessments prior to blood sample collection. Participant recruitment was conducted in November 2024 at the Teaching Hospital of Hasanuddin University (RSPTN).

Interpretation of Key Finding

This study examined the relationship between environmental exposures, clinical allergy symptoms, and serum Immunoglobulin E (IgE) levels in a cohort of 80 young adults. While descriptive analyses indicated a tendency for increased IgE levels among individuals exposed to allergens and with allergic symptoms, inferential statistical tests (Mann–Whitney U and multiple regression analysis) revealed no significant associations for most variables. Interestingly, only high humidity demonstrated a statistically significant negative association with IgE levels ($p = 0.047$). This inverse relationship may be explained by mucosal immunology mechanisms, where elevated humidity supports better hydration of the airway epithelium, enhancing mucosal barrier integrity and reducing allergen penetration. Additionally, high moisture levels can influence allergen transport dynamics by diluting airborne allergen particles, thereby decreasing antigen load and subsequent immune sensitization. The median serum IgE concentration was 82.36 IU/mL, with substantial inter-individual variability (range: 848.10 IU/mL, SD: 196.89 IU/mL). This

variability highlights the heterogeneity in immune responses and suggests the influence of unmeasured or multifactorial determinants. Despite higher mean IgE levels in those with a history of allergy and environmental exposures, these associations were not statistically significant. The multiple linear regression model further confirmed the lack of predictive value of the combined independent variables on serum IgE concentration ($F = 1.095$; $p = 0.379$).

Comparison with Previous Studies

Our findings partially align with previous literature. The absence of significant associations between IgE levels and common environmental exposures (e.g., dust, cigarette smoke, poor ventilation) (25, 26), which reported positive correlations. However, the high variability observed in this study mirrors that of (27, 28), who attributed differences in IgE response to genetic predisposition and individual immune profiles. Interestingly, the significant inverse relationship between high humidity and IgE levels diverges from earlier suggestions that humidity exacerbates allergen proliferation and allergic symptoms (19, 23). This discrepancy may reflect a complex threshold effect where moderate humidity reduces airborne allergen dispersion, or differences in environmental context, such as regional climate or housing conditions. Consistent with Abbafati et al. (2020) and Gilles et al. (2018), this study supports the concept that young adults, despite having optimal immunological function, remain susceptible to immunomodulatory effects from indoor exposures. However, the absence of statistical significance in most variables suggests that environmental and immunologic interactions are likely moderated by additional unaccounted factors, including psychological stress, diet, genetic polymorphisms, or co-existing health conditions (4, 17).

Limitations and Cautions

Several limitations must be acknowledged: Sample Size: The relatively small sample size ($N = 80$) may have reduced the statistical power to detect subtle associations. Cross-sectional Design: The study's observational and cross-sectional nature limits causal inference. Exposure Assessment: Environmental exposure data were self-reported and not validated with environmental monitoring, which may introduce recall or misclassification. Confounding Variables: Unmeasured confounders such as stress levels, nutritional status, genetic background, or past medical history could have influenced IgE levels. IgE as a Single Biomarker: Relying solely on serum IgE may overlook broader immune responses or non-IgE mediated allergies.

Recommendations for Future Research

Future studies should consider the following directions: Larger, multi-center cohorts are recommended to improve generalizability and statistical power. Longitudinal designs could help establish causal relationships between environmental exposures and immunologic outcomes. Objective environmental monitoring (e.g., PM2.5 levels, humidity sensors, VOC measurements) should supplement self-reported data. Incorporating genetic and epigenetic profiling could elucidate individual susceptibility to allergen sensitization. Expanding immunological profiling to include cytokines, eosinophils, and other immunoglobulin subclasses may offer a more comprehensive understanding of allergic pathophysiology.

CONCLUSION

The present study found that the mean serum IgE concentration among adults in Makassar was 176.8 IU/mL, reflecting a wide inter-individual variation. No statistically significant associations were observed between environmental factors and serum IgE levels. Similarly, clinical allergic symptoms, including asthma, allergic rhinitis, and atopic dermatitis, were not significantly correlated with IgE concentrations. These findings suggest that additional underlying factors such as genetic predisposition or unmeasured environmental exposures—may play a more prominent role in influencing IgE-mediated immune responses in this population.

AUTHOR'S CONTRIBUTION STATEMENT

Conceptualization, Methodology, Software, Data curation, Writing - Original draft preparation; Funding acquisition, Project administration, A.K.A; Software, Validation, Supervision.L.B.K.: Supervision.S.R. A.A.Z; Software, Validation, Conceptualization, Writing – Review & Editing, Methodology, Project administration; Supervision. U.B.

CONFLICTS OF INTEREST

Confirms that the authors have declared any potential conflicts that could influence the impartiality of the research. The authors explicitly state that they have no financial or personal relationships with entities that might unduly affect their objectivity. This declaration ensures the integrity of the study by transparently addressing any possible influences on the research outcomes, contributing to the credibility and trustworthiness of the article.

DECLARATION OF GENERATIVE AI AND AI-ASSISTED TECHNOLOGIES IN THE WRITING PROCESS

Authors are required to transparently disclose any use of generative artificial intelligence (AI) tools or AI-assisted technologies—such as ChatGPT, Grammarly, or DeepL—during the manuscript preparation process. This policy aims to uphold academic integrity, promote responsible authorship practices, and ensure compliance with ethical publication standards. If AI tools have been employed to support language refinement, enhance clarity, or improve the overall readability and structure of the manuscript.

SOURCE OF FUNDING STATEMENTS

This study was supported by the Ministry of Education, Culture, Research, and Technology of Indonesia under grant number 123/UNHAS/2025. The funding agency had no involvement in the design of the study, data collection, data analysis, data interpretation, or writing of the manuscript. This declaration ensures the independence and objectivity of the research.

ACKNOWLEDGMENTS

Health Research Ethics Committee (Komisi Etik Penelitian Kesehatan, KEPK) of the Faculty of Medicine, Hasanuddin University, in collaboration with Hasanuddin University Hospital (RSUH) and Dr. Wahidin Sudirohusodo Hospital (RSWS), Makassar

BIBLIOGRAPHY

1. Zhong J, Li W, Yang S, Shen Y, Li X. Causal association between air pollution and allergic rhinitis, asthma: a Mendelian randomization study. *Front Public Health*. 2024;12:1386341.
2. Zhang L, Akdis CA. Environmental exposures drive the development of allergic diseases. *Allergy*. 2024;79(5):1081-4.
3. Guarnieri M, Balmes JR. Outdoor air pollution and asthma. *Lancet*. 2014;383(9928):1581-92.
4. Wollenberg A, Thomsen SF, Lacour JP, Jaumont X, Lazarewicz S. Targeting immunoglobulin E in atopic dermatitis: A review of the existing evidence. *World Allergy Organ J*. 2021;14(3):100519.
5. Ansotegui IJ, Melioli G, Canonica GW, Caraballo L, Villa E, Ebisawa M, et al. IgE allergy diagnostics and other relevant tests in allergy, a World Allergy Organization position paper. *World Allergy Organ J*. 2020;13(2):100080.
6. Wu YZLZDWY. Potential Provoking Effects of Environmental Pollutants on Food Allergy: An Issue That Is Gaining Increasing Attention. *China CDC Wkly*. 2024;vo.6 no.24.
7. Calvani M, Anania C, Caffarelli C, Martelli A, Miraglia Del Giudice M, Cravidi C, et al. Food allergy: an updated review on pathogenesis, diagnosis, prevention and management. *Acta Biomed*. 2020;91(11-S):e2020012.
8. Agache I, Annesi-Maesano I, Cecchi L, Biagioli B, Chung KF, Clot B, et al. EAACI guidelines on environmental science for allergy and asthma: The impact of short-term exposure to outdoor air pollutants on asthma-related outcomes and recommendations for mitigation measures. *Allergy*. 2024;79(7):1656-86.
9. Cachon BF, Firmin S, Verdin A, Ayi-Fanou L, Billet S, Cazier F, et al. Proinflammatory effects and oxidative stress within human bronchial epithelial cells exposed to atmospheric particulate matter (PM(2.5) and PM(>2.5)) collected from Cotonou, Benin. *Environ Pollut*. 2014;185:340-51.
10. Perkin MR, Strachan DP. The hygiene hypothesis for allergy - conception and evolution. *Front Allergy*. 2022;3:1051368.
11. Pfefferle PI, Keber CU, Cohen RM, Garn H. The Hygiene Hypothesis - Learning From but Not Living in the

Past. Front Immunol. 2021;12:635935.

- 12. Grandjean P, Poulsen LK, Heilmann C, Steuerwald U, Weihe P. Allergy and sensitization during childhood associated with prenatal and lactational exposure to marine pollutants. Environ Health Perspect. 2010;118(10):1429-33.
- 13. Papapostolou N, Makris M. Allergic Asthma in the Era of Personalized Medicine. J Pers Med. 2022;12(7).
- 14. Hu J, Chen J, Ye L, Cai Z, Sun J, Ji K. Anti-IgE therapy for IgE-mediated allergic diseases: from neutralizing IgE antibodies to eliminating IgE(+) B cells. Clin Transl Allergy. 2018;8:27.
- 15. Humbert M, Bousquet J, Bachert C, Palomares O, Pfister P, Kottakis I, et al. IgE-Mediated Multimorbidities in Allergic Asthma and the Potential for Omalizumab Therapy. J Allergy Clin Immunol Pract. 2019;7(5):1418-29.
- 16. Internasional I. Enzyme immunoassay (microtiter strips) for the quantitative determination of immunoglobulin E (IgE) in human serum and plasma. In: H IBLINTERNATIONALGMB, editor. (RE59061) 2014.
- 17. Xu-De Z, Bei-Bei G, Xi-Juan W, Hai-Bo L, Li-Li Z, Feng-Xia L. Serum IgE Predicts Difference of Population and Allergens in Allergic Diseases: Data from Weifang City, China. Mediators Inflamm. 2021;2021:6627087.
- 18. Rasha El-Bushra Abdulhamid1 AHSaSAO. Association of smoking and IgE levels among smoker women in Khartoum. American Journal of Research Communication. 2015;3(11):48-54.
- 19. Shobrina Insan Sakina Armunanto1 DPD, Dodik Pramono3, Moh. Syarofil Anam4. correlation between house dust mite density with healthy house criteria and asthma status in pediatric patients : study among asthma pediatric patient in semarang. diponegoro medical journal. 2021;Volume 10.
- 20. Sikorska-Szaflik H, Sozanska B. Primary Prevention of Food Allergy-Environmental Protection beyond Diet. Nutrients. 2021;13(6).
- 21. Dewi Inderiati CAA, Lita Puspitasari. Analisis Kadar IgE Atopy Tungau Debu Rumah Pada Pasien Dermatitis Atopik. Jurnal Kesehatan Perintis. 2024;11:10-8.
- 22. Song X, Ding X, Niu P, Chen T, Yan T. The Associations between Exposure to Multiple Heavy Metals and Total Immunoglobulin E in U.S. Adults. Toxics. 2024;12(2).
- 23. Sutoh Y, Hachiya T, Otsuka-Yamasaki Y, Komaki S, Minabe S, Ohmomo H, et al. Genetic predisposition for immunoglobulin E production explains atopic risk in children: Tohoku Medical Megabank cohort study. The American Journal of Human Genetics. 2025;112(8):1852-63.
- 24. Melen E, Standl M, Gehring U, Altug H, Anto JM, Berdel D, et al. Air pollution and IgE sensitization in 4 European birth cohorts-the MeDALL project. J Allergy Clin Immunol. 2021;147(2):713-22.
- 25. Paciencia I, Sharma N, Hugg TT, Rantala AK, Heibati B, Al-Delaimy WK, et al. The Role of Biodiversity in the Development of Asthma and Allergic Sensitization: A State-of-the-Science Review. Environ Health Perspect. 2024;132(6):66001.
- 26. Goel S, Sahu S, Minz RW, Singh S, Suri D, Oh YM, et al. STAT3-Mediated Transcriptional Regulation of Osteopontin in STAT3 Loss-of-Function Related Hyper IgE Syndrome. Front Immunol. 2018;9:1080.
- 27. Fundora-Hernandez H, Venero-Fernandez SJ, Suarez-Medina R, Mora-Faife Ede L, Garcia-Garcia G, del Valle-Infante I, et al. What are the main environmental exposures associated with elevated IgE in Cuban infants? A population-based study. Trop Med Int Health. 2014;19(5):545-54.