Assessing the Effect of Air Pollution on Respiratory health and Allergic Sensitization in Children: A Cross-Sectional Study in North Jakarta's Urban Slums

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Type of Fellowship : Short-term Research Fellowship
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Host Institution : Leiden University Medical Center (LUMC), Leiden, The Netherlands
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Duration : 3 months (May 5th - July 26th. 2025)

Reports of this Research Fellowships will answer these following questions:

o What questions were addressed and why?
o What was the nature of the research?
o What was the result?
o How will the findings impact future research?

1. Introduction

Jakarta is Indonesia's largest city, where the population is growing rapidly but unevenly, leaving many areas with an uneven distribution of infrastructure development and population growth, resulting in densely populated areas that lack adequate facilities. Out of 267 neighbourhoods, 118 are classified as urban slums, with the highest numbers in North Jakarta [1]. The dense population, coupled with high levels of vehicle emissions, industrial pollutants, and limited access to clean air, creates an environment with continued exposure to contaminants that could harm health quality. The Air Quality Index (AQI) in North Jakarta, which ranges from 112-172, indicated its unhealthy air condition [2]. The recent increase in air pollution in Jakarta, which is 9.1 times higher than the WHO global air quality guidelines, raises concerns about children's exposure and lung health risk, including respiratory infections, asthma and perennial allergies [3]. The high exposure to pollutants in North Jakarta, such as NO_2, O_3, PM10, and PM2.5 are associated with wheeze, and the exacerbation of asthma symptoms [4]. Pollutants such as PM2.5 and O_3 can enhance the number of allergen proteins processed by antigen-presenting cells in the respiratory tract. This process triggers type I hypersensitivity and inflammation, characterized by histamine release, increased mucus, and bronchospasm, a primary symptom of asthma [5,6]. One way to evaluate this inflammation is by measuring fractional exhaled nitric oxide (FeNO) level. During airway inflammation, epithelial cells release increased amounts of nitric oxide (NO), which appears in the exhaled breath. Measuring the fractional exhaled nitric oxide (FeNO) provides a simple, non-invasive indicator of airway inflammation, especially in asthma [7].

The American Thoracic Society recently reported that pollution contributes to the onset of asthma and increases allergic sensitization [8]. In urban slum areas like North Jakarta, poorly maintained homes provide a conducive environment for allergens such as house dust mites (HDMs). These mites are common indoor aeroallergens, and exposure to them is an independent risk factor for sensitization in children. The severity of asthma and rhinitis has been shown to correlate with higher mite densities in household dust, particularly *Dermatophagoides farinae* (Derf1) and *Dermatophagoides pteronyssinus* (Derp1). Environmental factors such as dampness, high humidity, coal-stove heating, PVC windows, the presence of pets, and sleeping on couches further increase mite abundance [9]. In Indonesia, detection rates for these HDM species are alarmingly high, with both Derf1 and

Derp1 identified in 97% of households (28 out of 29 homes sampled), and Derp1 levels generally exceeding those of Derf1 [10].

Pollutants and allergens can also cause airway cells to release type 2 alarmins such as TSLP, IL-25, and IL-33 as the initial defence mechanism against external stimuli [11]. Studies have shown that Derp1 increases the production of interleukin (IL)-6 and IL-8 in airway cells [12-15], while TNF-α levels are also reportedly elevated following NO_2 exposure [16,17]. On the other hand, the mechanism of respiratory inflammation triggered by pollutants like $PM_{2.5}$, similar to the reaction against allergens such as pollen in type I hypersensitivity, is mediated by IL-4, IL-5, IL-13, and elevated immunoglobulin E (IgE) [16-18]. Upon inhalation and entry into the lungs, $PM_{2.5}$ could also stimulate the production of pro-inflammatory cytokines, such as interleukin (IL)-1β, in pulmonary cells [18]. These cytokines can be collected and measured using a child-friendly, non-invasive technique that collects nasal fluid via a Synthetic Absorptive Matrix (SAM) strip placed against the nasal lining cavity of the inferior turbinate to capture relevant inflammatory markers. In this study, we will evaluate the upper respiratory health of children living in North Jakarta's urban slums in the context of elevated local air pollution. By establishing the presence of local cytokines and chemokines in the upper respiratory tract, skin prick test positivity to HDM, total serum IgE levels and respiratory complaints by questionnaires, we aim to gain insight how living circumstances including elevated air pollution may influence mucosal cytokine release, potentially triggering or worsening allergies in children from low socioeconomic areas in North Jakarta. The findings may support guiding public health interventions on pollution control and supporting healthcare providers in preventive care for children.

2. Objectives

This study aims to investigate the effect of living circumstances, including the elevated air pollution in North Jakarta (112–172 AQI+ during September–October 2024), on respiratory health by examining inflammatory responses to pollutants and their correlation with allergic sensitization in urban slum children through FeNO measurements, nasosorption, and skin prick tests (SPT).

3. Methods

This study is a collaboration between Leiden University Medical Center (LUMC) and Atma Jaya Catholic University of Indonesia (AJCUI), funded by LUMC and co-funded by AJCUI, with ethical clearance obtained from the Ethical Committee of the School of Medicine and Health Sciences, AJCUI (No. 17/05/KEP-FKIKUAJ/2024). LUMC provided all consumables, including FeNO device and mouthpieces, nasosorption materials, SPT allergens and prickers, and all the necessary reagents. AJCUI support the fieldwork activities, including FeNO measurement, nasosorption sampling demographic questionnaire and anthropometry. The sampling was conducted in a public elementary school in the area of Pejagalan Sub-district, Penjaringan District, North Jakarta, Indonesia. The samples and information for the analysis were obtained from students in grades 4 to 6 (age 11-13). Demographic data of students, including age, sex, body weight, and height, were collected to calculate their Body Mass Index (BMI). The nasosorption sampling, FeNO and SPT measurements were carried out from September to October 2024. Nasosorption sampling was conducted simultaneously with FeNO measurements, resulting in a total of 222 samples. Additionally, 216 samples were collected for SPT testing. Consequently, the paired data for this study consists of 216 samples.

As part of the collaboration, nasosorption sample processing and data analysis was conducted at Immunology Department, Leiden University Medical Center (LUMC) in Leiden, Netherlands.

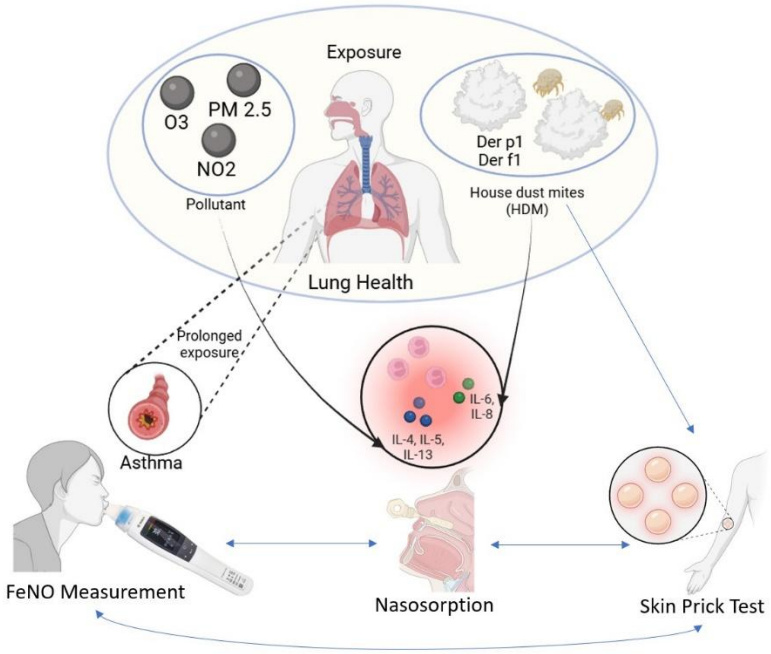

Figure 1. Exposure of Pollutant and HDM Affecting Lung Health. Pollutants like PM 2.5, O_3, and NO_2 stimulate pro-inflammatory cytokines, such as IL-4, IL-5, and IL-13, in response to aeroallergens. House dust mites (HDMs), prevalent aeroallergens, stimulate the secretion of cytokines such as IL-6 and IL-8. Prolonged exposure to both pollutants and HDM exacerbates hypersensitivity, frequently leading to asthma. The severity of Asthma can be assessed using fractional exhaled nitric oxide (FeNO), which indicates airway inflammation. Cytokine levels are measured using nasosorption sample of the nasal mucosal lining fluid and allergic sensitivity is evaluated using a Skin Prick Test (SPT). Increased exposure to pollutants and HDM is expected to elevate cytokine levels, increasing allergic sensitization and asthma onset likelihood. (*Figure created using BioRender Online Software*).

Activity During the Fellowship

Nasosorption samples were transported under cold chain conditions from Atma Jaya Catholic University of Indonesia (AJCUI) to the laboratory at LUMC and stored at −80°C until processed.

Nasosorption sample elution

Elution was performed using the optimized protocol developed by LUMC. There were 2 nasosorption samples that had missing labels. The total of 220 samples were randomized and divided into 18 elution batches with addition of 1 mock in every batch, resulting in 238 samples. A total of 100 µL of elution buffer was applied to the top of the nasosorption filter, ensuring even distribution across both sides. The filter was gently pressed against the inner wall of the collection tube, allowing each side to remain in contact with the buffer for at least 10 seconds. The filter was then placed on ice for over 1 minute before centrifugation. Initial centrifugation was done at 3,124 × g for 12 minutes at 4 °C. After centrifugation, the eluate was mixed and resuspended using a pipette, then transferred to a new Eppendorf tube and centrifuged again at 16,000 × g for 10 minutes at 4 °C to clear the sample. The resulting supernatant was divided into two Eppendorf tubes for inflammatory cytokine analysis [19,20]. The remaining volume, containing the pellet, was retained for pathogen detection which were not included in this fellowship period.

Cytokine measurement

The initial inflammatory cytokines selected for analysis included IL-1β, IL-4, IL-5, IL-6, IL-8, IL-13, IL-25, IL-33, TSLP, and TNF-α. These targets were subsequently refined based on the analyte availability in two LEGENDplex™ panels from BioLegend: the Human Inflammation Panel 1 (13-plex) with V-bottom Plate and the Human Allergy/Asthma Panel (11-plex) with V-bottom Plate. The Human Inflammation Panel 1 includes 13 human inflammatory cytokines and chemokines: IL-1β, IFN-α2, IFN-γ, TNF-α, MCP-1 (CCL2), IL-6, IL-8 (CXCL8), IL-10, IL-12p70, IL-17A, IL-18, IL-23, and IL-33. Meanwhile, the Allergy/Asthma Panel targets 11 analytes related to allergy and asthma: IL-4, Periostin, IL-5, IL-33, TSLP, RAGE, IL-13, YKL-40, MMP-9, IL-9, and CCL11 (Eotaxin). Both panels utilize a bead-based multiplex immunoassay platform with fluorescence-encoded beads, enabling the simultaneous quantification of multiple cytokines.

A total of 238 samples were distributed across three 96-well plates, and the assays were performed over three separate days, one plate per panel per day. All reagents from the kits were equilibrated to room temperature for at least 30 minutes prior to assay setup. Standards and samples were mixed with assay buffer and mixed capture beads provided in each kit, followed by shaking on a plate shaker at 800 rpm for 30 minutes at room temperature, and then incubated overnight in a cold room (4°C) on a shaker. On the following day, the plates were again shaken at 800 rpm for 30 minutes at room temperature, then centrifuged at 250 × g for 5 minutes. After centrifugation, the supernatants were discarded in one continuous and forceful motion. Plates were then washed twice using the provided wash buffer, with each washing step followed by the same centrifugation conditions as previously described. Detection antibodies were added to each well and incubated for 1 hour at 800 rpm. Without washing, Streptavidin-Phycoerythrin (SA-PE) as the fluorescent reporter was added and incubated for an additional 30 minutes with shaking (800 rpm). This was followed by a final wash step. Before acquisition, 65 µL of wash buffer was added to each well. All steps were conducted under light-protected conditions, using aluminum foil to prevent photobleaching [21,22]. Samples were measured using the BD FACSCanto™ Clinical Flow Cytometry System.

Data analysis

We first analyzed the flow cytometry data using BioLegend's LEGENDplex™ Data Analysis Software to gate signals for both the standard curve and all samples, as recommended in the assay instruction manual [21,22]. Cytokine concentrations were then organized in Microsoft Excel and log-transformed. We used GraphPad and R Studio to assess the distribution of cytokine levels and examine their correlations with FeNO and SPT results.

4. Results

Our study in an urban slum area in North Jakarta found that 26.4% of 222 children had elevated FeNO levels (20–35 ppb), while 9.3% showed high levels ranging from 37–75 ppb. Skin prick testing (SPT) for house dust mites (HDM), *D. farinae* and *D. pteronyssinus* revealed that 29% of 216 children reacted positively to at least one species. Specifically, 12.5% were reactive to *D. pteronyssinus,* 8.3% to *D. farinae* and 7.4% to both. Elevated FeNO was significantly associated with any HDM sensitivity (p = 0.014), suggesting that allergen sensitization contributes to airway inflammation (Figure 2).

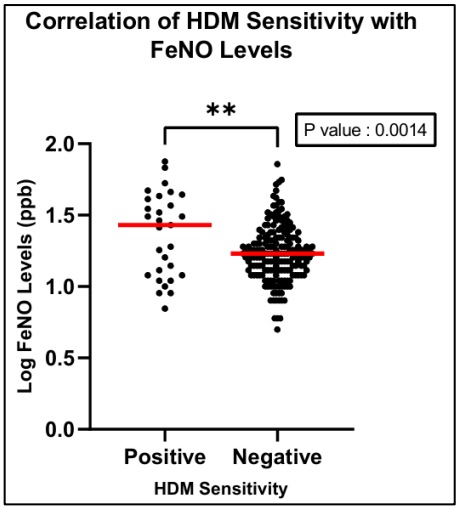

Figure 2. Correlation between HDM Sensitivity with log-transformed FeNO levels (ppb).

However, the positive HDM sensitivity group seems to be divided into two different clusters. Among children with positive HDM sensitivity results, larger wheal diameters to *D. pteronyssinus* were significantly correlated with higher FeNO levels (p = 0.0061), whereas no such correlation was observed for *D. farinae*. This suggests that stronger sensitization to *D. pteronyssinus* is more closely linked to increased airway inflammation (Figure 3), which may explain the clustering observed.

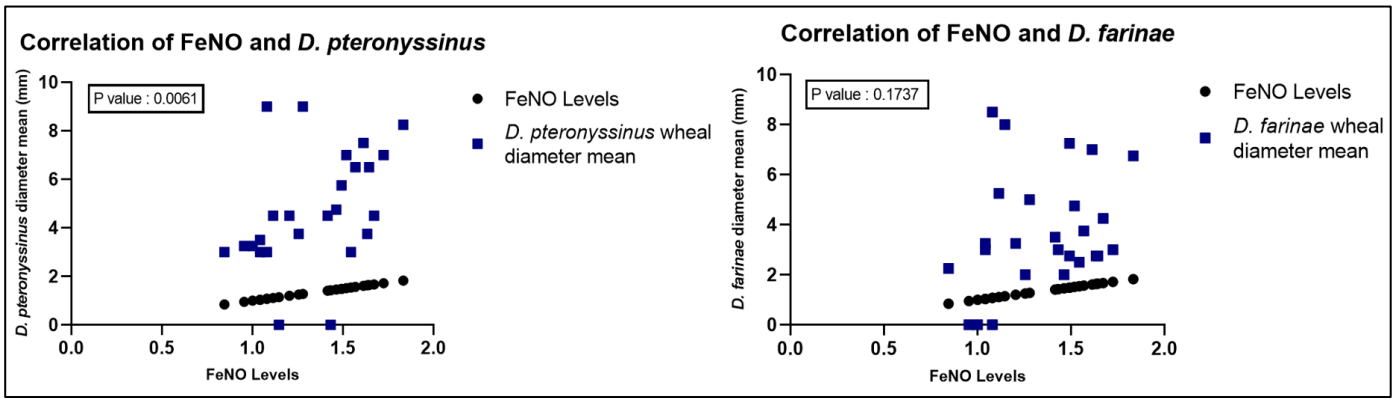

Figure 3. Correlation between FeNO levels (ppb) and Positive SPT Reactivity Species.

Multiplex analysis of the nasal lining fluid showed that most markers were expressed at low levels (less than 10 pg/ml), although considerable variation was observed between children (Figure 4). The variability suggesting heterogeneous inflammatory responses within the study population. A hierarchical clustering heatmap of log10-transformed analyte concentrations (Figure 5) identified four main expression patterns, labeled 1–4 above the dendrogram. The first cluster, which included IL-5, IL-9, RAGE, IL-4, IL-13, IL-33, CCL11, and TSLP, was consistently low, suggesting weak type-2 activity in this cohort. Intermediate expression patterns were observed in clusters 2 and 3, consisting of IFN-α2, IL-17A, IFN-γ, TNF-α, MMP-9, MCP-1, IL-6, YKL-40, IL-23, IL-10, and IL-12p70, with cluster 3 showing slightly higher concentrations compared to cluster 2. In contrast, a fourth cluster dominated by IL-1β, IL-18, IL-8, and periostin displayed much higher concentrations, highlighting a subgroup of children with stronger pro-inflammatory responses. Along the vertical axis, hierarchical clustering of participants revealed groups of children separated by their overall cytokine expression patterns. Each row represents one child, annotated by HDM sensitization status, gender, and school grade. Children with broadly higher pro-inflammatory cytokine levels clustered

together, while others displayed lower or intermediate profiles. These participant clusters did not align strictly with HDM sensitization status, gender, or grade, suggesting that the observed patterns likely reflect underlying biological heterogeneity.

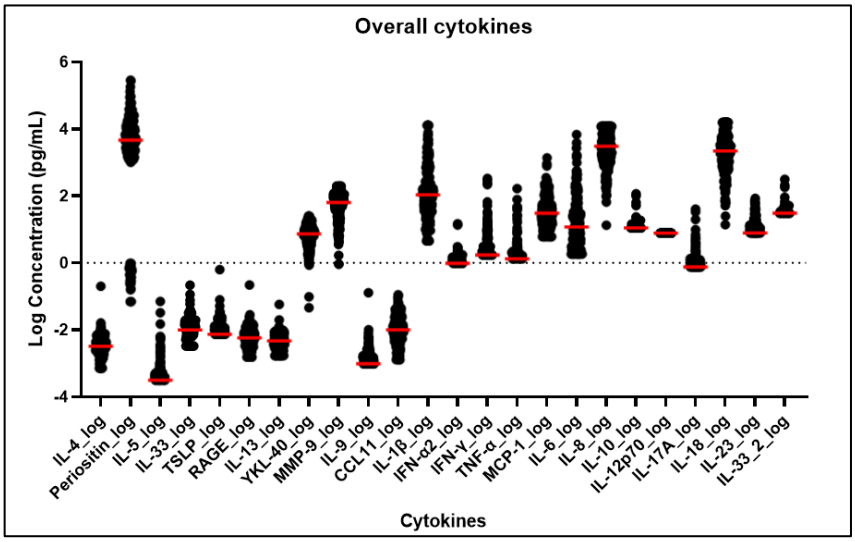

Figure 4. Scatter dot plot of log10-transformed cytokine concentrations distribution across all samples. Each black dot represents an individual data point, while the red horizontal bars indicate the median values.

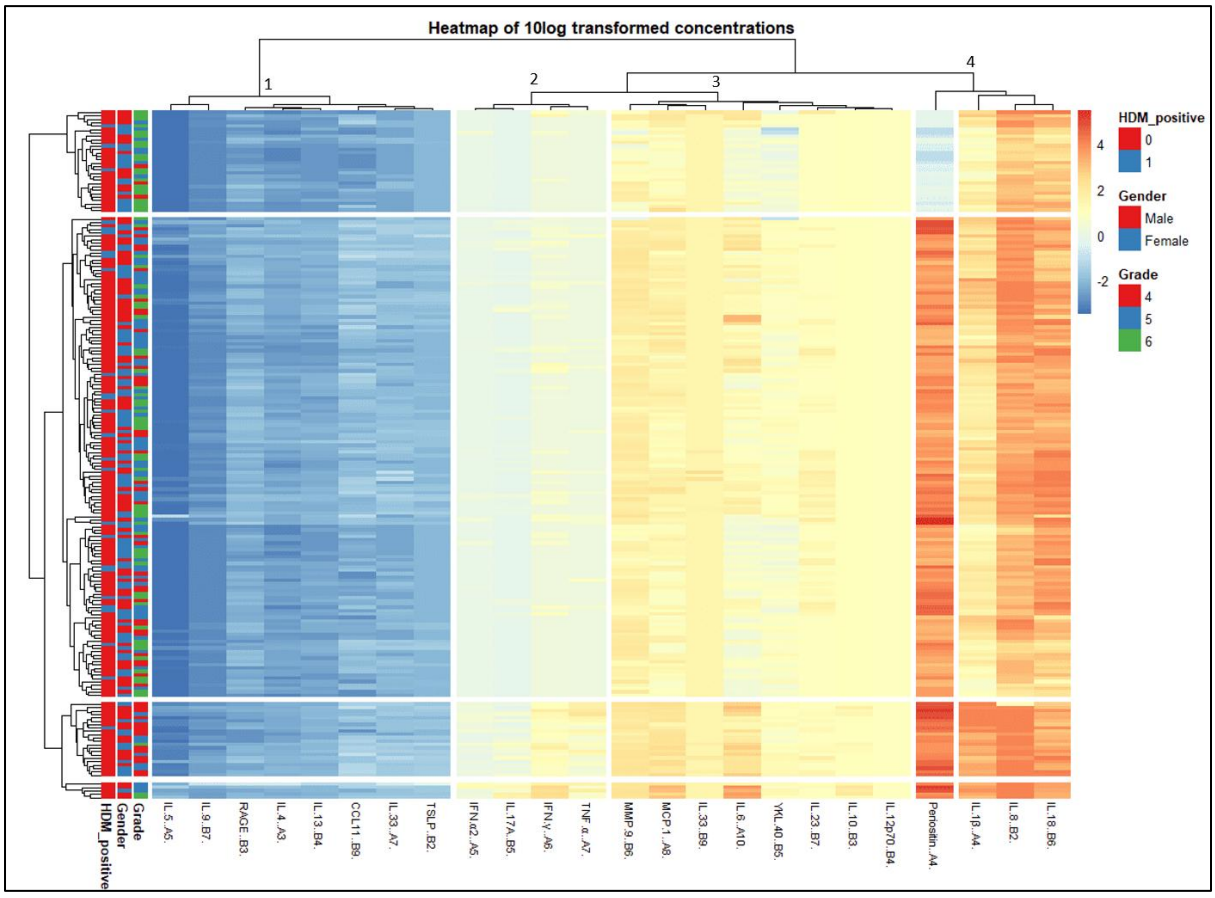

Figure 5. Heatmap of log10-transformed cytokine concentrations across subjects. Columns represent cytokines and rows represent individual subjects. Hierarchical clustering grouped the cytokines into four major clusters (labeled 1–4) based on similarities in their expression patterns.

The cytokine patterns observed are consistent with mechanisms known to be triggered by environmental air pollution. Pollutants such as particulate matter, nitrogen dioxide, and ozone activate epithelial and inflammasome pathways, resulting in increased secretion of IL-1β, IL-18, and IL-8, which mirrors the cytokine profile seen in the high-expression cluster. Clustering analysis further showed that HDM-sensitized children were more frequently represented in the third pro-inflammatory group, suggesting that allergen sensitization not only promotes type-2 cytokine activity but also strengthens epithelial and innate immune activation. Alternatively, it is possible that these children experience low-grade inflammation due to underlying allergy, which may amplify the effects of environmental exposures such as air pollution and poor living conditions. Conversely, chronic inflammatory responses induced by these environmental factors, including recurrent pathogen exposure, may increase susceptibility to the development of allergic sensitization. To further assess the relationship between airway inflammation and systemic markers, FeNO levels were categorized according to ATS guidelines for children (<20, 20–35, >35 ppb) and correlated with IL-1β, IL-18, IL-8, and periostin concentrations. No significant associations were observed between FeNO categories and these cytokines (data not shown). However, when FeNO was analyzed as a continuous variable, periostin showed a significant positive correlation with FeNO levels (p = 0.0003), indicating that children with higher periostin concentrations tended to have higher FeNO values (Figure 6). Interestingly, the distribution showed two clusters, a majority of high producers and a smaller group of low producers. The low producers did not exhibit reduced concentrations of other cytokines, suggesting that this finding is unlikely to be the result of technical failure. A second analysis confirmed that, even after excluding the low producers, the positive correlation between periostin and FeNO remained significant (p = 0.005; Figure 7). In contrast, IL-1β, IL-18, and IL-8 did not demonstrate significant associations with FeNO.

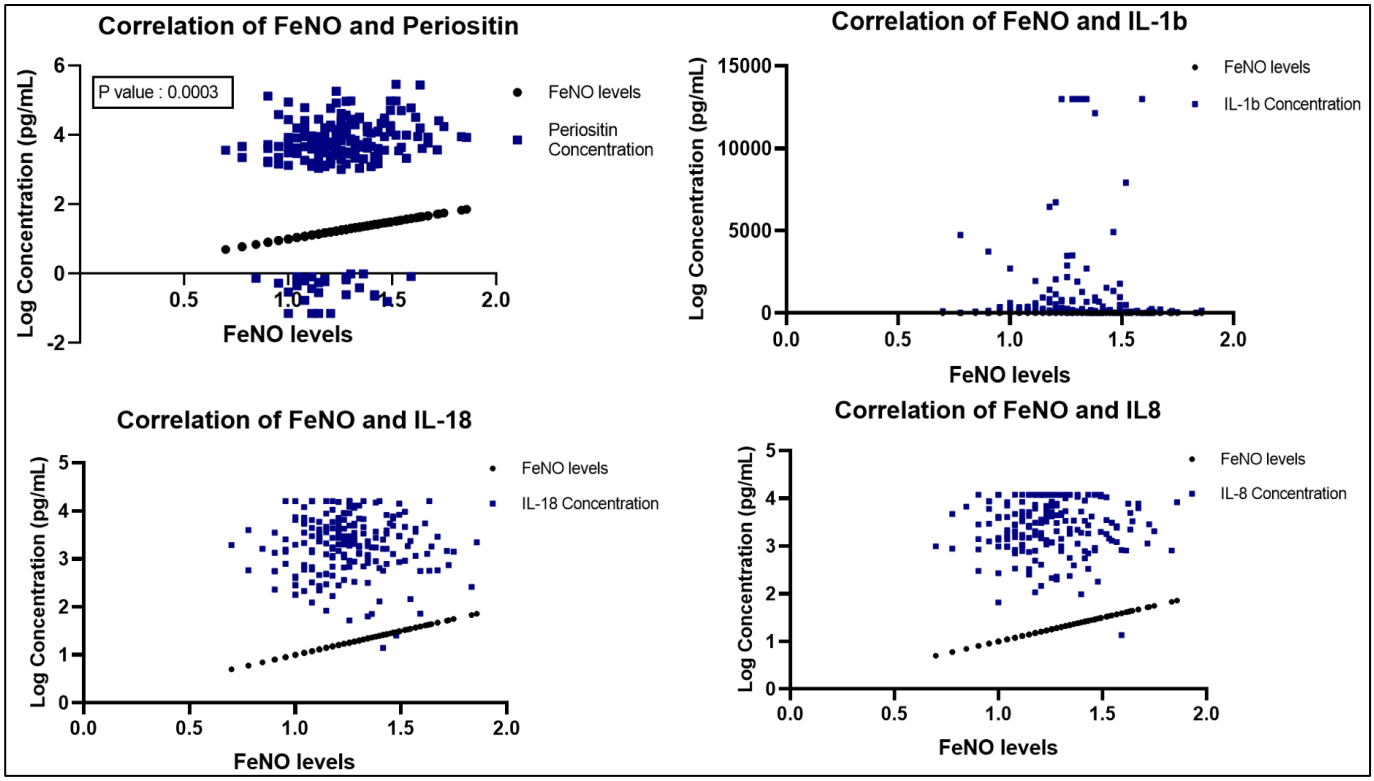

Figure 6. Correlation between FeNO Level with IL-1b, IL-18, IL-8 and Periositin Concentration.

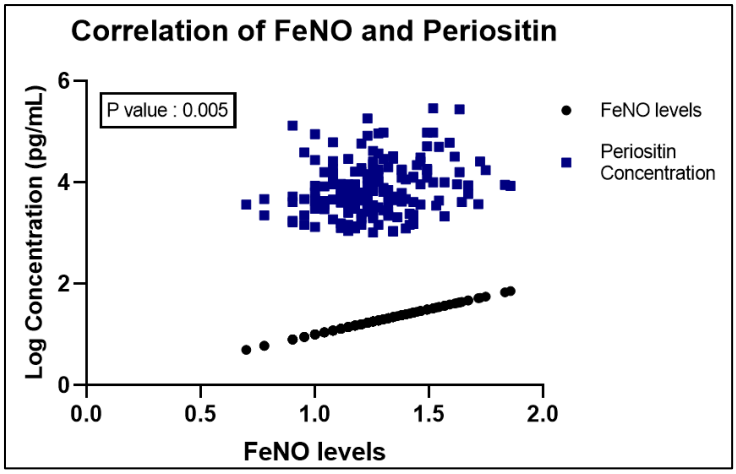

Figure 7. Correlation between FeNO levels and periostin concentrations after exclusion of the low-producer group. A significant positive correlation remained (p = 0.005).

These findings suggest that allergen sensitization and environmental exposures, such as air pollutants, may act together to influence mucosal immunity in the upper respiratory tract. While allergen sensitization amplifies type-2 immune pathways, pollutant exposure may drive epithelial and innate activation. The combined influence of these factors may predispose sensitized children to persistent airway inflammation and increase their vulnerability to asthma development and exacerbations.

5. How will the findings impact future research?

These findings highlight the importance of considering both allergen sensitization and environmental exposures when studying airway inflammation in children. The observed association between FeNO, SPT reactivity, and periostin suggests that future research should integrate clinical biomarkers with environmental exposure data to better understand asthma risk pathways. The lack of direct environmental measurements during sample collection, makes it challenging to link pollution exposure with airway inflammation. Further analyses could use correlation networks and PCA to group cytokines into functional immune axes, such as type 2 or pro-inflammatory patterns. In addition, IgE and ISAAC data are available but not yet analyzed. Integrating IgE with cytokine clusters may help define endotypes, separating allergy-driven from pollution-driven inflammation, while ISAAC data can provide symptom profiles that can be correlated with cytokine patterns. This work will be further developed in my master's project, which I will continue under the guidance of Prof. Hermelijn Smits and Maria Mardalena Martini Kaisar, PhD.

6. Personal reflections

This fellowship has given me an unforgettable and insightful research experience. It was my first time living abroad and working in a research environment surrounded by amazing and inspiring researchers. During this period, I learned new laboratory skills that I had never used or studied before, such as flow cytometry. I was especially grateful for the opportunity to join a training program before working independently with the flow cytometer. I also learned other methods from fellow PhD students in the research group and benefited greatly from participating in weekly journal clubs and project update meetings, which helped refresh my knowledge and exposed me to new research perspectives. In addition, I improved my statistical skills in R with guidance from experts in the team. Overall, this fellowship not only enhanced my research capabilities but also strengthened my foundation to continue my

academic journey, particularly as I prepare to pursue a master's degree. I hope to apply these skills to contribute more effectively to research projects at my home institution and in my future scientific endeavours.

7. Acknowledgements

First, I would like to express my sincere gratitude to my host supervisor, Prof. Hermelijn Smits, and her research team, especially Yvonne de Visser, for their continuous guidance and support throughout my fellowship. I am also thankful to Arifa Ozir-Fazalalikhan for providing training and assistance, from nasosorption elution to cytokine measurement using flow cytometry, and to Oscar van Hengel for his guidance in data analysis using R. My thanks also go to the research group members Willianne Hoepel, Margarida Viola, Yu Cui, and Samson Folami for their warm welcome, valuable advice, and support during my time at LUMC. I would also like to thank Dr. A.S. (Abena) Amoah for her valuable input and guidance in the study set-up and data collection. I am deeply grateful to my home supervisor, Maria Mardalena Martini Kaisar, PhD, for guiding me from the start of the project, giving me the opportunity to work on this research, and introducing me to such an inspiring team. Finally, I would like to thank the EAACI Research Fellowship Program Jury for selecting and supporting this project.

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