Impact of air quality on tissue eosinophilia and clinical severity of chronic rhinosinusitis

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Dear Editor:

Particulate matter 2.5 ($PM_{2.5}$) exposure impairs nasal epithelial defenses, disrupts mucociliary clearance, increases oxidative stress, and exacerbates inflammatory pathways associated with chronic rhinosinusitis (CRS) [1,2]. While it impacts upper airway diseases, the relationship between $PM_{2.5}$ exposure and eosinophilic CRS (ECRS) with tissue eosinophil >10 cells/ HPF remains unclear [3,4]. A cross-sectional study was conducted in Thailand and Vietnam to investigate the impact of $PM_{2.5}$ exposure on tissue eosinophilia and the severity of CRS.

One hundred and twenty-nine patients with CRS were enrolled between 2024 and 2025 at four tertiary hospitals: Faculty of Medicine, Chulalongkorn University; Rajavithi Hospital; ENT Ho Chi Minh; and Hue UMP Hospital. Patients were excluded if they moved their residences from one city to another during the past two years. The study protocol received the institutional review boards approval. Sixty-five patients were Thai, whereas sixty-four were Vietnamese. The mean age was 49.3 ± 15.6 years (Table S1). The CRSwNP phenotype accounted for 72.9%. The study cohort demonstrated a high degree of homogeneity regarding disease severity, as reflected by symptoms score, SNOT-22, comparable rates of NSAID-exacerbated respiratory disease, revision sinus surgery, disease duration, treatment exposure, and comorbid conditions, including allergic rhinitis, asthma, and smoking. $PM_{2.5}$ exposure levels in Bangkok, Ho Chi Minh City, and Hue were collected from surface air quality monitoring stations (Figure S1). Hue UMP Hospital demonstrated the lowest pollution exposure, with a mean $AQI_{PM2.5}$ of 20.6 ± 15.1. In contrast, ENT Ho Chi Minh (mean $AQI_{PM2.5}$ = 96.4 ± 26.7), Chulalongkorn University (mean $AQI_{PM2.5}$ = 78.1 ± 28.6), and Rajavithi Hospital (mean $AQI_{PM2.5}$ = 78.1 ± 28.6) showed higher values (Figure S2). In the univariable negative binomial model, higher $AQI_{PM2.5}$ significantly associated with increased tissue eosinophil counts. Each 1-unit increase in $AQI_{PM2.5}$ was associated with a 1.4% increase in tissue eosinophils. This association remained significant after adjustment (adjusted prevalence rate ratio (PRR) = 1.0; 95% CI 1.0–1.0; $p < 0.01$). In univariable Gamma generalized linear models, $AQI_{PM2.5}$ associated with higher serum eosinophil levels. The association remained statistically significant after adjustment (adjusted mean ratio = 1.0; 95% CI 1.0–1.0; $p < 0.01$). Higher $AQI_{PM2.5}$ associated with increased odds of ECRS endotype. The association persisted after adjustment (adjusted odds ratio (OR) = 1.0; 95% CI 1.0–1.0; $p < 0.01$). Higher $AQI_{PM2.5}$ was significantly associated with CRSwNP phenotype. After adjustment, $AQI_{PM2.5}$ remained independently associated with CRSwNP (adjusted OR = 1.0, 95% CI 1.0–1.0; $p = 0.04$).

No statistically significant associations were identified between average daily $AQI_{PM2.5}$ and clinical symptoms severity. In linear regression analysis, the average daily $AQI_{PM2.5}$ was not associated with VAS total score. Using negative binomial regression models, no significant association was observed between $AQI_{PM2.5}$ and SNOT-22 total score and rhinologic subscale.

Higher $AQI_{PM2.5}$ significantly associated with increased Lund-–Mackay CT scores. In univariable negative binomial regression, each 1-unit increase in $AQI_{PM2.5}$ associated with a higher score. This association remained statistically significant after adjustment (PRR = 1.0, 95% CI 1.0–1.0, $p < 0.01$). In contrast, in univariable Poisson regression analysis, the average daily $AQI_{PM2.5}$

Table 1. Association between average daily AQIPM2.5, tissue eosinophil count, and severity of CRS symptoms (n = 129).

Outcome	Model	Univariable $AQI_{PM2.5}$ (95% CI)	p-value	Adjusted $AQI_{PM2.5}$ (95% CI)	p-value	Covariate included in adjusted model
Tissue eosinophil count (eosinophils/ HPF)	Negative binomial	PRR = 1.0 (1.0–1.0)	**< 0.01**	PRR = 1.0 (1.0–1.0)	**< 0.01**	Age
Serum eosinophil count (x10^9/L)	Gamma GLM	MR = 1.0 (1.0–1.0)	**0.02**	MR = 1.0 (1.0–1.0)	**< 0.01**	Country†, asthma†
CRS endotype (ECRS vs NECRS)	Logistic	OR = 1.0 (1.0–1.0)	**< 0.01**	OR = 1.0 (1.0–1.0)	**< 0.01**	Age
CRS phenotype (CRSwNP vs CRSsNP)	Logistic	OR = 1.0 (1.0–1.0)	**< 0.01**	OR = 1.0 (1.0–1.0)	**0.04**	Allergic rhinitis†
VAS score	Linear regression	β = –0.0 (–0.0–0.0)	0.84	β = 0.0 (–0.0–0.0)	0.81	Age, gender, country
SNOT-22 total score	Negative binomial	PRR = 1.0 (1.0–1.0)	0.14	PRR = 1.0 (1.0–1.0)	0.34	Allergic rhinitis
SNOT-22 rhinologic subscale	Negative binomial	PRR = 1.0 (1.0–1.0)	0.45	PRR = 1.0 (1.0–1.0)	0.41	Age, gender, allergic rhinitis
Modified Lund–Kennedy endoscopic score	Poisson	PRR = 1.0 (1.0–1.0)	0.10	PRR = 1.0 (1.0–1.0)	0.85	Gender†, allergic rhinitis†
Lund–Mackay CT score	Negative binomial	PRR = 1.0 (1.0–1.0)	**< 0.01**	PRR = 1.0 (1.0–1.0)	**< 0.01**	Age, gender, country†
Kennedy Osteitis score	Negative binomial	PRR = 1.0 (1.0–1.0)	0.26	PRR = 1.0 (1.0–1.0)	0.55	Gender†, prior ESS†, asthma

Effect estimates are per 1-unit increase in $AQI_{PM2.5}$. Bold values indicate statistical significance ($p < 0.05$). Abbreviations: †, significant covariate; β, beta coefficient; CI, confidence interval; Gamma GLM, Gamma generalized linear regression model; MR, mean ratio; OR, odds ratio; PRR, prevalence rate ratio; SNOT-22, 22-item Sino-Nasal Outcome Test; VAS, Visual Analogue Scale.

was not significantly associated with modified Lund–Kennedy endoscopic score (PRR = 1.0, 95% CI 1.0–1.0, p = 0.10). Negative binomial regression showed no statistically significant association between $AQI_{PM2.5}$ and Kennedy Osteitis score in the multivariable analysis (PRR = 1.0, 95% CI 1.0–1.0, p = 0.26). Data are shown in Table 1.

These findings provide epidemiologic evidence supporting the role of ambient air pollution in association with type 2 inflammatory endotypes of CRS, aligning with previously published articles [3,5-7] suggesting that particulate matter promotes epithelial dysfunction and eosinophil-dominant immune responses. Associations with patient-reported symptom burden or endoscopic severity were not demonstrated. These findings align with prior studies [3,8-9]. Potential explanations include chronic adaptation to long-term urban pollution and the modifying effects of medical therapy.

Conclusion

$PM_{2.5}$ exposure was linked to eosinophilic sinonasal inflammation. It was associated with greater tissue and serum eosinophilia, and CRSwNP phenotype with increased radiologic disease severity.

LIST OF ABBREVIATIONS

AQI: Air Quality Index; CI: Confidence interval; CRS: Chronic rhinosinusitis; CRSwNP: Chronic rhinosinusitis with nasal polyps; CRSsNP: Chronic rhinosinusitis without nasal polyps; ECRS: Eosinophilic chronic rhinosinusitis; ESS: Endoscopic sinus surgery; GLM: Generalized linear model; MR: Mean ratio; NECRS: Non-eosinophilic chronic rhinosinusitis; OR: Odds ratio; PM: Particulate matter; PRR: Prevalence rate ratio; SNOT-22: 22-item Sino-Nasal Outcome Test; VAS: Visual Analogue Scale.

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Authorship contribution

ALQ: study design, data collection, data analysis, and manuscript preparation. KSe: data collection, data analysis, and manuscript edits. WC: data collection, data analysis, and manuscript edits. TT: data collection and manuscript edits. JS: data collection and manuscript edits. MPH: data collection and manuscript edits. LVT: data collection and manuscript edits. MTQL: data collection and manuscript edits. SC: data collection and manuscript edits. JK: data collection and manuscript edits. PW: data collection and manuscript edits. KS: conception, study design, data analysis, manuscript edits, and final approval of the manuscript.

Conflict of interest

Kornkiat Snidvongs received honoraria for speaking at symposia from Menarini, Organon, GSK, and Abbott. The authors declare that they have no conflicts of interest.

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This manuscript contains online supplementary material

SUPPLEMENTARY MATERIAL

Table S1. Association between average daily $AQI_{PM2.5}$, tissue eosinophil count, and severity of CRS symptoms (n = 129).

Variable	Mean ± SD	n (%)
Study site distribution, n (%)		
KCMH		55 (42.6)
Rajavithi Hospital		10 (7.8)
ENT HCM Hospital		29 (22.5)
Hue UMP Hospital		35 (27.1)
Age (years)	49.3 ± 15.6	
Gender, n (%)		
Female		74 (57.4)
Male		55 (42.6)
Active smokers		36 (27.9)
Revision ESS		37 (28.7)
Allergic rhinitis		53 (41.1)
Asthma		28 (21.7)
NERD		2 (1.6)
ECRS		71 (55.0)
CRSwNP		94 (72.9)
VAS total score	5.0± 2.0	
SNOT-22 total score	43.5 ± 20.4	
SNOT-22 rhinologic subscale	20.0 ± 7.2	
Modified Lund–Kennedy endoscopic score	7.8 ± 2.6	
Lund–Mackay CT score	15.3 ± 5.3	
Kennedy Osteitis score	3.1 ± 3.2	
Tissue eosinophil count (cells/HPF)	29.9 ± 35.3	
Serum eosinophil count ($x10^9$/L)	0.4 ± 0.3	

SD, standard deviation; NERD, nonsteroidal anti-inflammatory drug exacerbated respiratory disease; VAS, Visual Analogue Scale; SNOT-22, 22-item Sino-Nasal Outcome Test; ESS, endoscopic sinus surgery; ECRS, eosinophilic chronic rhinosinusitis; HPF, high-powered field.

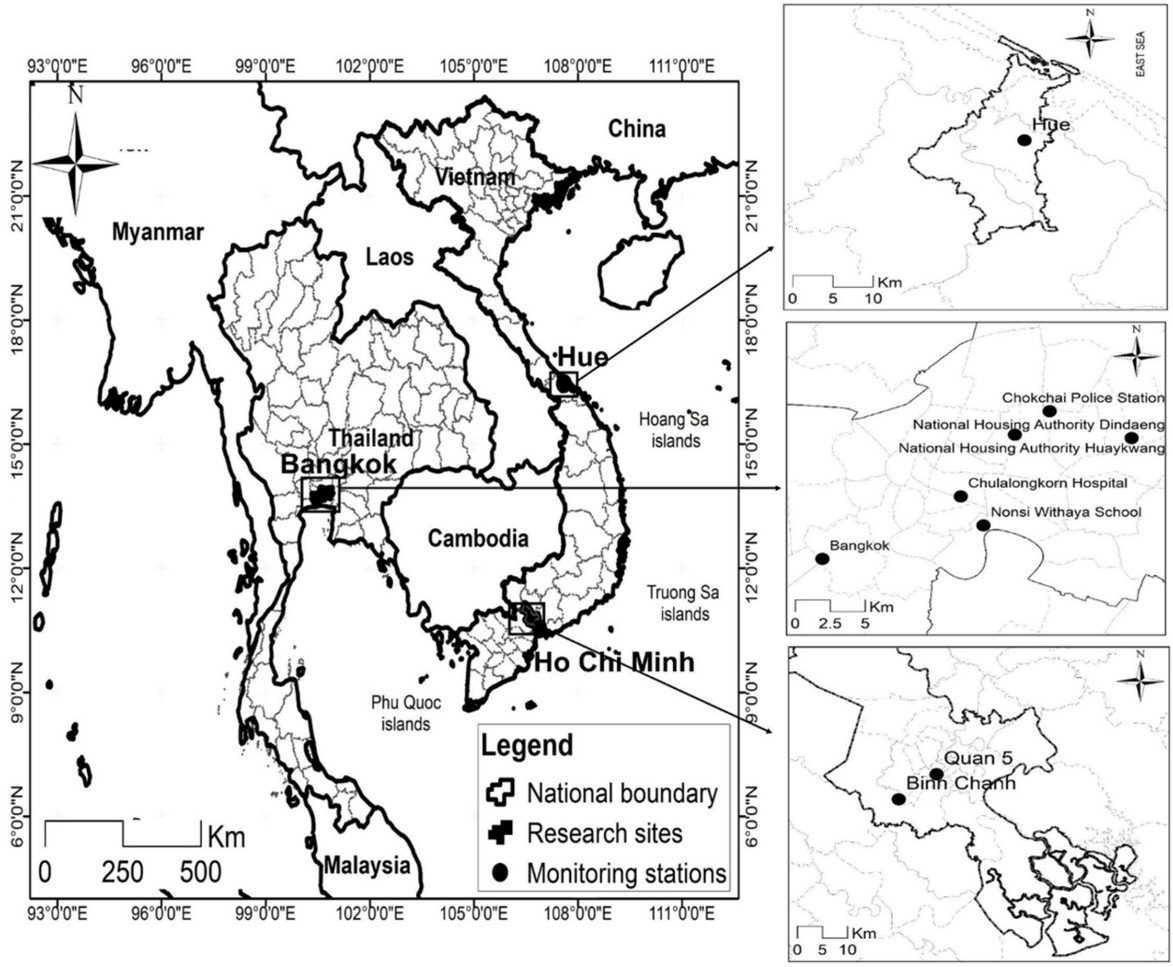

Figure S1. Research sites and monitoring station locations. The main map shows the study area, with insets indicating the locations of research sites and monitoring stations in Hue, Bangkok, and Ho Chi Minh City.

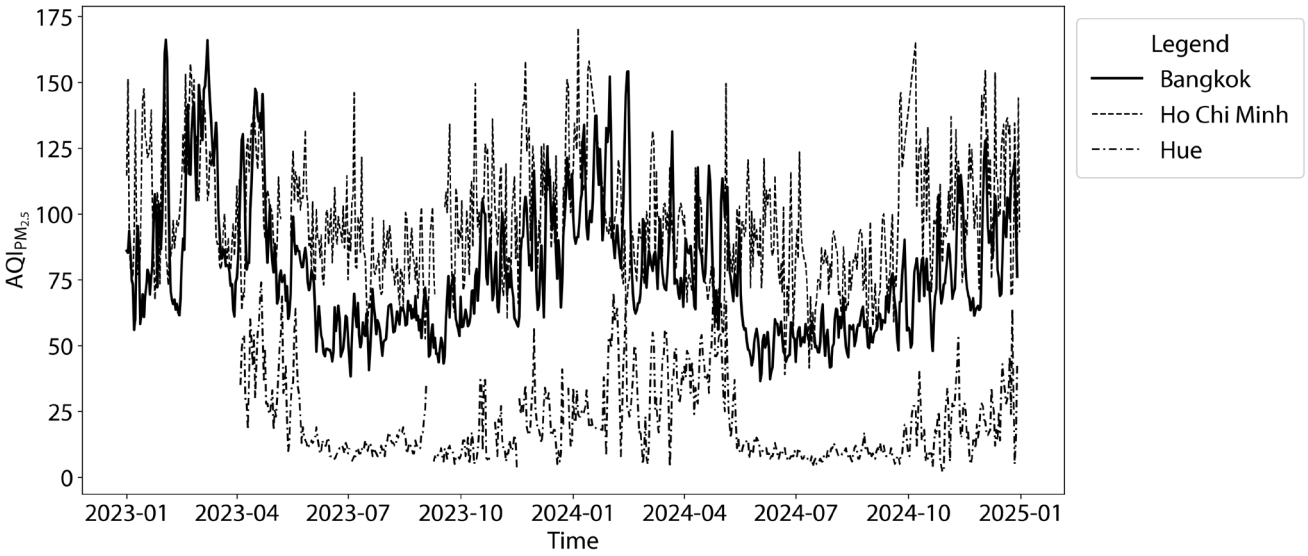

Figure S2. Daily mean $AQI_{PM2.5}$ in Bangkok, Ho Chi Minh City, and Hue (Jan 2023–Dec 2024). Bangkok is shown as a solid line, Ho Chi Minh City as a dashed line, and Hue as a dash-dot line.