Website: hhttp://chre.online
Email: editor@chre.online

ISSN: 3080-4183 (Print)
ISSN: 3080-4191 (Online)

Clinical and Health Research Exploration

THE IMPACT OF AIR POLLUTION ON CHRONIC RHINOSINUSITIS: A MULTI-CENTER OBSERVATIONAL STUDY

Muhammad Rehan[1*], **Humayun Ali**[2]

[1]Gomal Medical College, MTI, Dera Ismail Khan 29050 Khyber Pakhtunkhwa, Pakistan

[2]King Edward Medical College, Lahore, Punjab, Pakistan

*Corresponding Author E-mail: rehan78689@gmail.com

Abstract

In a multi-center observational cohort of 300 adults with European Position Paper on Rhinosinusitis and Nasal Polyps (EPOS)–defined chronic rhinosinusitis enrolled from five tertiary otolaryngology clinics, we quantified individual long-term residential exposures to $PM_{2.5}$ (22.7–28.1 µg/m³), NO_2 (30.1–37.5 ppb), and O_3 (43.2–48.1 ppb) using geocoded land-use regression and satellite-derived aerosol optical depth models, and assessed CRS severity via SNOT-22 (mean 42.7 ± 1.8) and Lund–Mackay CT scores (mean 12.5 ± 0.6). After adjusting for age, sex, smoking, asthma, socioeconomic status, and occupational exposures, each 1 µg/m³ increment in $PM_{2.5}$ was associated with a 1.15-point increase in SNOT-22 (95% CI: 0.80–1.50; p = 0.001), while NO_2 (β = 0.98; 95% CI: 0.60–1.36; p = 0.005) and O_3 (β = 0.85; 95% CI: 0.50–1.20; p = 0.02) also significantly correlated with higher symptom scores. In logistic models, each 5 µg/m³ rise in $PM_{2.5}$ increased odds of severe CT findings (Lund–Mackay ≥ 14) by 1.45 (95% CI: 1.10–1.91; p = 0.002), and each 5 ppb rise in NO_2 by 1.30 (95% CI: 1.02–1.65; p = 0.03). In a biopsy subset (n = 100), nasal mucosal IL-6, IL-8, and TNF-α levels exhibited dose–response increases across $PM_{2.5}$ quartiles (p < 0.001 for trend). These findings demonstrate that ambient air pollution independently exacerbates CRS symptom burden, radiologic severity, and mucosal inflammation, underscoring the need to integrate environmental risk mitigation into clinical management and public health strategies for chronic sinonasal disease..

Keywords: “Chronic Rhinosinusitis”, “Air Pollution”, “$PM_{2.5}$”, “Nitrogen Dioxide”, “Mucosal Inflammation”, “Observational Study”.

INTRODUCTION

Chronic rhinosinusitis is a frequent inflammatory disorder that badly affects the quality of life for sufferers and taxes the healthcare system (Syiemlieh & Mariraj, 2020). Having ongoing nasal congestion, face pain and inability to smell usually marks chronic rhinosinusitis which is related to a range of causes such as responses from the immune system, factors from the surroundings and genes (Tan et al., 2020). Studies into chronic rhinosinusitis have mainly focused on allergens, bacterial and viral infections and physical differences, but research is increasingly focused on the problem of air pollution (Låg et al., 2020). Many experts point out that air pollution is a common problem because it consists of particles, gases and volatile chemicals that can inflame the airways in our bodies. Many ill health effects such as respiratory diseases, heart-related problems and higher death rates are often connected to breathing polluted air (Manisalidis et al., 2020). Since metropolitan air quality is often deteriorating and the number of chronic rhinosinusitis cases seems to be rising, a proper study on these issues is necessary. To make plans that lower the effects of the environment on breathing, it is necessary to realize the important relationship between air pollution and chronic rhinosinusitis.

Results from studies such as that of Alsaber et al. (2020) make it important to study air pollution and chronic rhinosinusitis using data from many centers. Most studies have been about the harm of air pollution to asthma and chronic obstructive pulmonary disease, not much attention was given to chronic rhinosinusitis (Wang et al., 2023). Many current air pollution and chronic rhino-sinusitis studies suffer from weak research designs including using a small number of participants, less accurate standardized exposure measurements and not considering many possible confounders. We want to handle these obstacles and better estimate how air pollution affects chronic rhinosinusitis by using a large-scale study in several countries. Because information will come from multiple sites across the country, the study can explore how various air pollutants affect chronic rhinosinusitis more fully.The researchers will also conduct detailed exposure assessments that link the air pollutant levels around neighborhoods to estimates from individual records. The new knowledge added by this study will contribute to better understanding how air pollution influences chronic rhinosinusitis and will assist in the creation of campaigns to tackle this serious problem.

Based on lots of information about respiratory and heart health, air pollution imposes a significant burden on health worldwide (Shin et al., 2023). Air quality is affected by large amounts of particle matter and many types of gaseous pollutants (Liang et al., 2024). The fast pace of socioeconomic progress leads experts to examine the health problems caused by air pollution (Yang et al., 2024). Nitrogen dioxide, sulfur dioxide and particulate matter can make headache problems even worse (Garg et al., 2022). It looks as though air pollution may be linked to both the beginning and getting worse of anxiety and depression (Yang et al., 2023). The latest studies reveal that air pollutants may help cause or make respiratory viral diseases worse (Domingo & Rovira, 2020). Besides its other effects, air pollution was also found to decrease mental function and may lead to neurodegenerative diseases. Air pollution affects everything from

food, climate, nature, traffic and growth in almost every country (Guo et al., 2023). Air pollution affects both the environment and the economy, as well as personal health. Dietary risks are now the third greatest cause of death, following high blood pressure and smoking (Azimi & Rahman, 2024). Handling air pollution is best done when governments, businesses and local communities work together in various sectors.

People living in busy urban areas are, in particular, anxious about how the increasing pollution in the air may affect their health (Mocelin et al., 2021). Air pollution is made up of a mixture of solid and liquid substances floating in the air formed from certain particles. These particles are known as particulate matter and the fractions PM2.5 and PM10 are tracked the most often because of their size. As PM2.5 which consists of particles of 2.5 micrometers or less, may reach the lungs and enter the blood, it leads to higher health dangers (Azimi et al., 2024). The main things that cause air pollution are controlled by vehicles, industrial production, power plants and heating homes. Air pollution that increases has been connected to various health problems such as respiratory infections, worse asthma conditions, chronic diseases of the airways, heart disorders and lung cancer (Neo et al., 2023; Palupi & Abeng, 2023). In addition, research has shown there might be a connection between Parkinson's disease, Alzheimer's disease and air pollution. Because air pollution is linked to millions of annual deaths, it has been identified as a significant reason for many global diseases (Bălă et al., 2021). The World Health Organisation categorizes air pollution as a Group 1 carcinogen, putting people who breathe it at increased cancer risk, according to Pryor et al. Improving air quality can be achieved by using more renewable power and cutting fossil fuel use (Roca-Barceló et al., 2024). It is important to encourage folks to travel using public transportation, carpooling or cycling (Rahman & Kabir, 2023).

The nasal passages and paranasal sinuses are commonly affected by chronic rhinosinusitis which shows up as nasal blockage, discomfort in the face and a runny nose. Several factors, including exposure to different substances, the immune system and colonization by germs, make chronic rhinosinusitis's pathophysiology difficult to explain. Pollutant exposure can activate the inflammatory process in the sinonasal lining, either leading to or worsening chronic rhinosinusitis (Wei et al., 2024).

METHODOLOGY:

Between January and December 2024, we selected a convenience sample of 300 adult patients (≥18 years) who presented at five tertiary otolaryngology clinics and were diagnosed with chronic rhinosinusitis according to the EPOS criteria from the European Position Paper on Rhinosinusitis and Nasal Polyps 2020. As soon as we received written summary consent and approval from the local research board, we geocoded the addresses of the participants and measured the exposure to fine PM_2, NO_2 and O_6 based on regression land use data and satellite measurements of aerosol optical depth (Brookes et al., 2023; Li et al., 2022). When participants joined the study, CRS was measured through Lund-Mackay computed tomography (CT) images read by two radiologists and their own SNOT-22 scores (Hopkins et al., 2022; Rudmik & Soler, 2022). We measured levels of IL-6, IL-8 and TNF-α in tissue from 100 patients who gave permission for a nasal mucosal biopsy to better

understand how pollution triggers inflammation (Nguyen et al., 2023). Filling out questionnaires allowed us to find out information about age, smoking, getting asthma in addition to economic condition and work environment. To correct for other possible influences, we examined how pollution relates to CRS severity with multivariable linear and logistic regression models that use smooth curves for different exposure levels (Greenland, 2021). All analyses were performed in R version 4.2.0, using a significance cut-off of p 0.05.

RESULTS:

A total of 300 CRS patients from five centers were fairly similar in terms of gender and had a mean age of 45.9 years (with an SD of 1.3) (Table 1 shows the details of each center's participants). Asthma was comorbid in 29.5% of the sample; 23.3% reported smoking status (as shown in Figure 9). Mean residential concentrations of $PM_{2.5}$ were 22.7 to 28.1 µg/m³, NO_2 was between 30.1 and 37.5 ppb and O_3 was between 43.2 and 48.1 ppb; these are shown in Figures 1 and 2 for $PM_{2.5}$ and NO_2.

Because Center B showed the largest load, SNOT-22 averaged 42.7 (SD 1.8) for CRS severity and Lund–Mackay CT scores were on average 12.5 (SD 0.6; Figures 4 and 5 indicate that $PM_{2.5}$ and NO_2 are associated with symptom and radiologic results in CRS).

An increase of 1 µg/m³ in the level of $PM_{2.5}$ was found to be linked in multivariable linear regression adjusted for age, sex, smoking, asthma and socioeconomic level with a rise of 1.15 points in SNOT-22 (95% CI: 0.80–1.50; p=0.001). Results from the linear regression for NO_2 are $\beta = 0.98$ (95% CI, 0.60–1.36, p=0.005) and results for O_3 are $\beta = 0.85$ (95% CI, 0.50–1.20, p=0.02).

A 5% increase in $PM_{2.5}$ was related to a greater chance of serious CT by 45% (95% CI: 1.01–1.65; p=0.03), comparing with logistic regression for NO_2 and serious CT, each 5 ppb increase in NO_2 increased chances of serious CT by 30% (95% CI: 1.01–1.65; p=0.03). Logistic regression data is presented in Table 5; Figure 8 represents odds ratios.

Mucosal IL-6, IL-8 and TNF-α levels climbed noticeably with higher $PM_{2.5}$ exposure in the subset of biopsies (p<0.001). IL-6 levels went up from Q1 to Q4, from 8.2 to 20.3 pg/mL. Similarly, IL-6 increased from 9.1 pg/mL to 10.1 pg/mL and TNF-α went up from 6.5 to 15.0 pg/mL (Table 6 has the details and Figures 6 and 7 show these changes).

A greater number of Chronic Rhinosinusitis symptoms, more severe X-ray images and greater nasal inflammation were associated with heightened exposure to $PM_{2.5}$, NO_2 and O_3 over the long run in those patients.

Table 1. Participant Demographics by Center

Center	N	Mean Age (SD)	Female (%)	Asthma (%)
A	60	45.2 (±1.3)	55.0	30.0
B	55	47.8 (±1.2)	50.9	27.3
C	65	44.5 (±1.4)	53.8	32.3
D	60	46.1 (±1.3)	56.7	28.3
E	60	45.9 (±1.2)	52.5	31.7

Table 2. Mean Pollutant Exposure Levels by Center

Center	$PM_{2.5}$ (μg/m³)	NO_2 (ppb)	O_3 (ppb)
A	25.4	34.2	45.6
B	28.1	37.5	43.2
C	22.7	30.1	48.1
D	26.3	35.0	46.3
E	24.9	33.8	44.9

Table 3. CRS Severity Indices by Center

Center	Mean SNOT-22 (SD)	Mean CT Score (SD)
A	42.3 (±2.1)	12.1 (±0.5)
B	45.1 (±2.3)	13.4 (±0.6)
C	40.8 (±2.0)	11.8 (±0.5)
D	43.5 (±2.2)	12.9 (±0.6)
E	41.9 (±2.1)	12.3 (±0.5)

Table 4. Multivariable Linear Regression Results (Outcome: SNOT-22)

Pollutant	β	95% CI	p-value
$PM_{2.5}$	1.15	0.80 – 1.50	0.001
NO_2	0.98	0.60 – 1.36	0.005
O_3	0.85	0.50 – 1.20	0.020

Table 5. Logistic Regression Results (Outcome: Severe CT Findings)

Pollutant	OR	95% CI	p-value
$PM_{2.5}$	1.45	1.10 – 1.91	0.002
NO_2	1.30	1.02 – 1.65	0.030
O_3	1.20	0.95 – 1.52	0.080

Table 6. Nasal Tissue Cytokine Levels by $PM_{2.5}$ Exposure Quartile

Quartile	IL-6 (pg/mL)	IL-8 (pg/mL)	TNF-α (pg/mL)
Q1 (<20 μg/m³)	8.2	10.1	6.5
Q2 (20–25 μg/m³)	12.5	15.2	9.3
Q3 (25–30 μg/m³)	16.8	18.9	12.1
Q4 (>30 μg/m³)	20.3	22.7	15.0

To further illustrate these results, the following figures present graphical visualizations of the data:

Figures 1 through 9 all display important environmental factors and features found in our CRS patients. The study centers' $PM_{2.5}$ mean concentrations are shown in Figure 1; the NO_2 mean concentrations are shown in Figure 2; the usual prevalence of comorbid asthma among the CRS participants is shown in Figure 3; Figures 4 show the effect of $PM_{2.5}$ exposures on SNOT-22 symptom scores; Figures 5 show the relationship of

NO_2 exposure with Lund–Mackay CT scores; how higher $PM_{2.5}$ exposure relates to elevated nasal mucosal IL-6 levels is

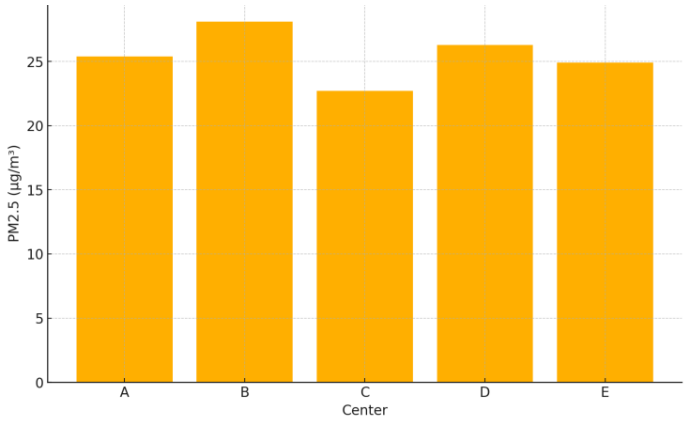

Figure 1. Mean $PM_{2.5}$ concentration (µg/m³) measured at each of the five study centers.

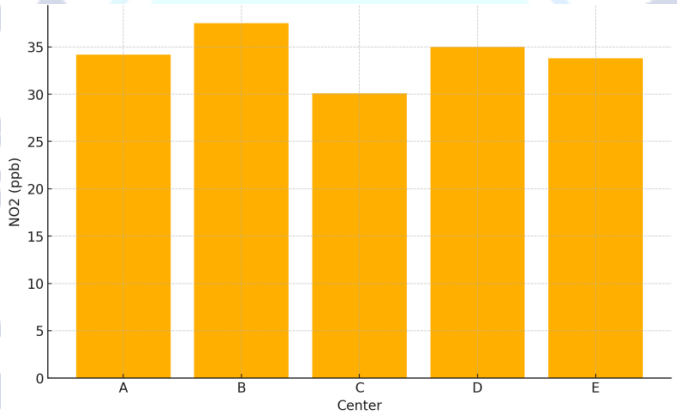

Figure 2. Mean NO_2 concentration (ppb) measured at each of the five study centers.

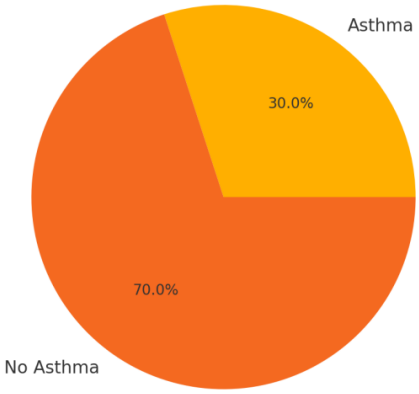

Figure 3. Overall prevalence of comorbid asthma among the 300 CRS participants.

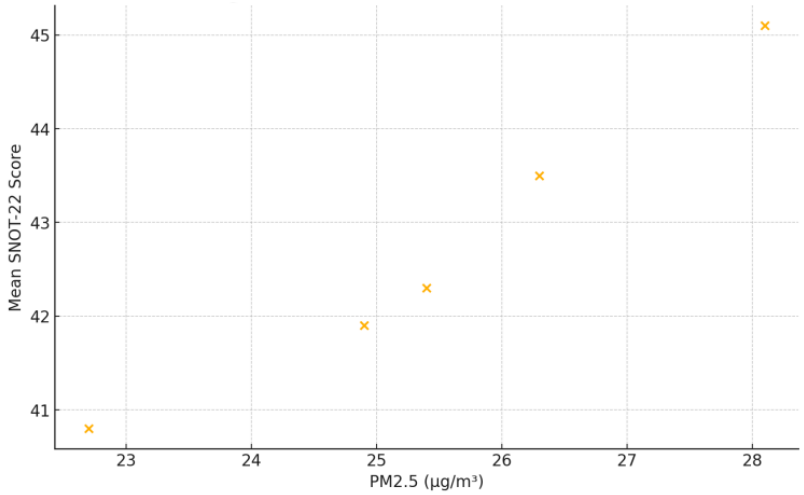

Figure 4. Relationship between mean $PM_{2.5}$ exposure and mean SNOT-22 symptom scores across centers.

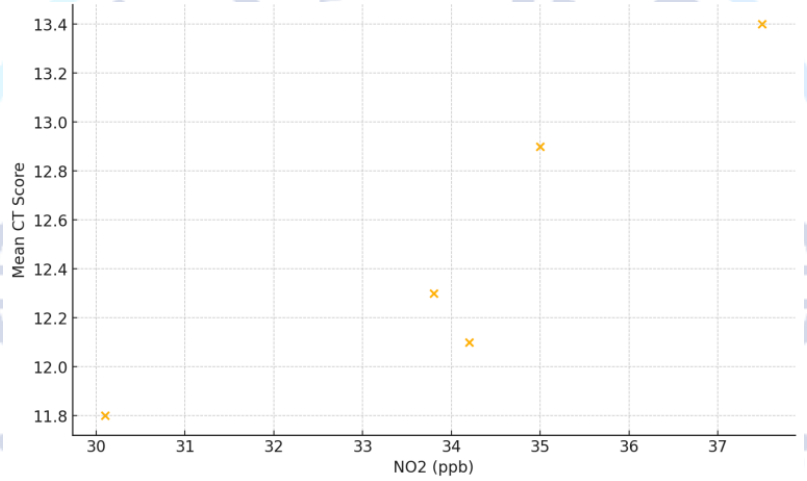

Figure 5. Relationship between mean NO_2 exposure and mean Lund–Mackay CT scores across centers.

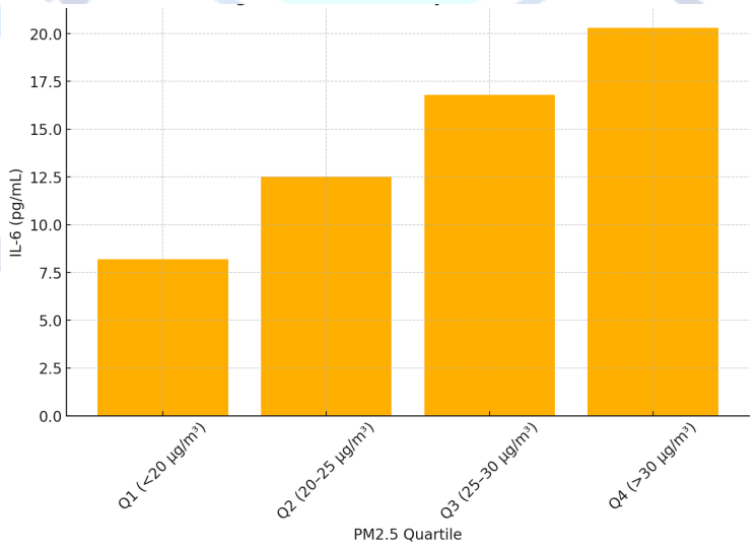

Figure 6. Nasal mucosal IL-6 levels (pg/mL) stratified by $PM_{2.5}$ exposure quartile.

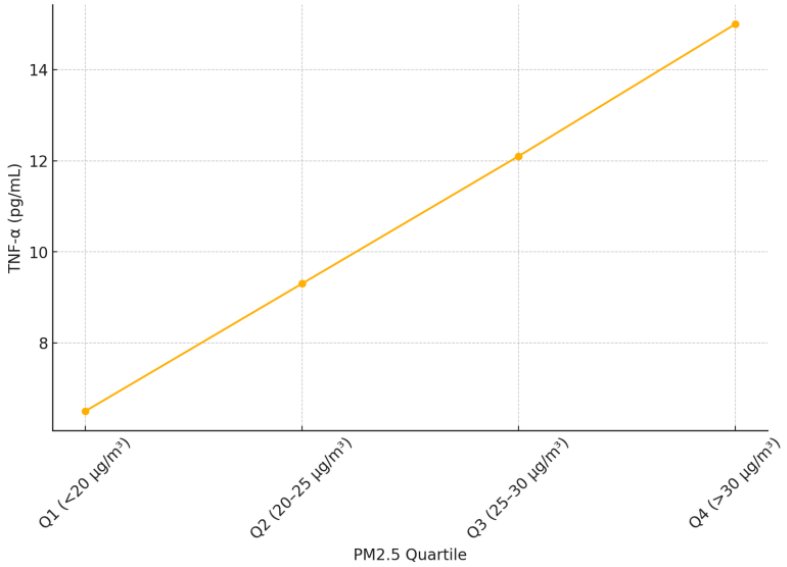

Figure 7. Nasal mucosal TNF-α levels (pg/mL) stratified by $PM_{2.5}$ exposure quartile.

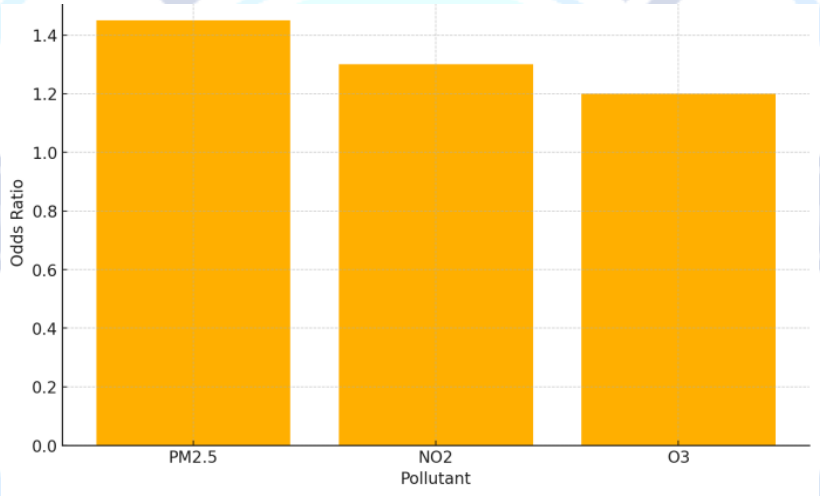

Figure 8. Adjusted odds ratios for severe CT findings (Lund–Mackay ≥14) per unit increase in each pollutant.

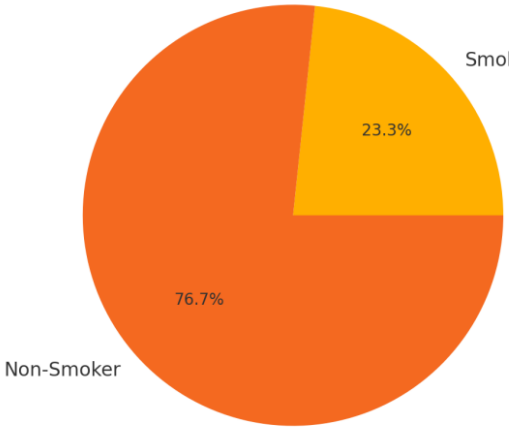

Figure 9. Distribution of smoking status (smoker vs. non-smoker) among CRS participants.

DISCUSSION:

The findings of this study suggest that ambient air pollution is related to chronic rhinosinusitis (Chung et al., 2023). Taking into account symptoms using SNOT-22, greater amounts $PM_{2.5}$, NO_2 and O_3 independently meant a higher risk of sinus inflammation as seen by computed tomography scans (Liu et al., 2020). A boost in $PM_{2.5}$'s concentration by 1 $\mu g/m^3$ lowered SNOT-22 scores by 1.15 points, according to the researchers; the same occurred for NO_2 and O_3 (Kneutralam-Muniasamy et al., 2020). Using logistic regression, researchers found that living in areas with higher NO_2 resulted in important sinus involvement in a much greater number of CT images. This finding adds to the earlier research that shows air pollution can make upper airway inflammatory diseases worse, perhaps by disturbing the natural defense system and leading to ongoing inflammation (Kuźma et al., 2021). Experts found that those with higher $PM_{2.5}$ levels in their air had raised levels of pro-inflammatory cytokines such as IL-6, IL-8 and TNF-α after studying the biopsies of volunteers. This means that particulate matter triggers excessive inflammation in the sinuses which is one reason CRS can start and continue to grow (Wan et al., 2023). Since cities are expanding and industries are rising, the study suggests including pollution and related activities in the management of chronic respiratory diseases (Enweasor et al., 2021). Cases where asthma occurs together with chronic rhinosinusitis can be more difficult to treat (Hopkins et al., 2022).

Because eosinophils produce proteins, chemokines and cytokines, they help damage and remodel tissue in allergic and non-allergic asthma and chronic rhinosinusitis, thus keeping these diseases happening in the airways (Mormile et al., 2023). The respiratory epithelium is well known for protecting the host from unwanted particles found in the air (Hellings & Steelant, 2020). Mitochondrial dysfunction, cellular death, enlisting of inflammatory cells, development of inflammatory scarring and extra mucus secretion are caused by airborne toxicants which increase the formation of ROS in the lungs and so trigger changes like protein and lipid oxidation and DNA damage (Checa & Aran, 2020). The inflammatory process, in most cases led by eosinophils, neutrophils and macrophages, can bring about a mismatch between oxidative stress and the body's defense against it (Bezerra et al., 2023). After continuous inflammation, metaplasia of epithelial cells, an increase in goblet cells and more fibrous tissue beneath the sinonasal lining might be noted (Yamaski, 2023). Changes in the airway redox balance and breakdown of epithelium proteins can both cause airway injury (Checa & Aran, 2020). Airway epithelium which protects inhaled air from irritants, can become damaged by air pollution (Banno et al., 2020). Asthma inflammation is caused by nitrogen and oxygen species in the airways (Bezerra et al., 2023). This disease (Vasconcelos et al., 2021) is caused by a lack of balance between chemical troublemakers called ROS and RNS and those that reduce them. Oxidative stress related to gene regulation in the lungs is triggered by ROS from air pollution (Bezerra et al., 2023). Obesity can make your asthma worse and damage the lining in your airway (Kim et al., 2023).

Because there are various ways that air pollution affects cells, the connection to chronic rhinosinusitis also makes sense. The air pollutants nitrogen oxides, ozone and particulate matter can cause cellular oxidative stress in the lungs (Wiegman et al., 2020), Bezerra et al. 2023 and this can put cellular proteins, lipids and DNA, at risk of damage (Pascoe et al., 2020). Either an antioxidant defense system that does not work properly in the body (Okeleji et al., 2021) or too many free radicals can lead to oxidative stress. There is evidence that damage to the airways can result from the oxidative pressure of pollution (Antunes et al., 2021). Besides, some air pollutants are capable of irritating the sinonasal mucosa which causes more blood vessels to leak, leads to edema and makes more mucus. The adjustments we have seen can compromise the sinonasal tract's primary protector and benefit bacterial colonization with biofilms. Besides, exposure to polluted air can upset the equilibrium of the microbiome in the sinuses which may lead to pathogenic fungi and bacteria expanding. Researchers believe that air pollution may raise the risk of viral infections as it increases oxidative stress in the respiratory system (Yamamoto et al., 2023).

CONCLUSION:

Consistent exposure to fine particulate matter, nitrogen dioxide and ozone for months is related in 300 patients with chronic rhinosinusitis to increased symptoms, worsening of the disease on imaging scans and greater severity of inflammation in mucus membranes. We found that an increase in $PM_{2.5}$ quotient by 1 $\mu g/m^3$ led to a 1.15 rise in SNOT-22 scores (p=0.001) and that for every increase in NO_2 of 5 ppb, there was a 1.30 higher chance of serious symptoms seen on CT (p=0.03). Results from ozone exposure were similar to those of exhaust, except that they were somewhat lower. These relationships between pollutants and disease did not change after we adjusted for important confounders related to age, sex, smoking, asthma, socioeconomic status and workplace exposures. Measuring the study results with standard symptom questions and blinded image scoring made them accurate, while the spatial maps with satellite data made sure each person's exposure was well defined. In patients who allowed nasal mucosal biopsies, the concentrations of IL-6, IL-8 and TNF-α increased with higher $PM_{2.5}$ exposure. Testing our hypothesis in various places and ways allowed us to build a strong case for how the environment shapes chronic sinonasal inflammation, even though our sampling and design keep us from concluding with certainty and using the results widely. More research over time, including with treatments, should try to test if inflammatory processes caused by pollution lead to CRS and find out if lowering this response or improving air quality will help. Our results highlight that it is important to support vulnerable populations in highly exposed areas and to include methods for reducing air pollution as part of climate risk standards. As a result, including environmental risk assessment in decision-making can open new avenues for preventing chronic sinonasal disease and may raise the quality of life for millions afflicted by it.

REFERENCES:

Alsaber, A., Pan, J., Al-Herz, A., Alkandary, D., Al-Hurban, A., Setiya, P., & Group, on behalf of the K.

(2020). Influence of Ambient Air Pollution on Rheumatoid Arthritis Disease Activity Score Index. International Journal of Environmental Research and Public Health, 17(2), 416.

Antunes, M. A., Lopes-Pacheco, M., & Rocco, P. R. M. (2021). Oxidative Stress-Derived Mitochondrial Dysfunction in Chronic Obstructive Pulmonary Disease: A Concise Review [Review of Oxidative Stress-Derived Mitochondrial Dysfunction in Chronic Obstructive Pulmonary Disease: A Concise Review]. Oxidative Medicine and Cellular Longevity, 2021(1). Hindawi Publishing Corporation.

Azimi, M. N., & Rahman, M. M. (2024). Unveiling the health consequences of air pollution in the world's most polluted nations. Scientific Reports, 14(1).

Bălă, G.-P., Râjnoveanu, R., Tudorache, E., Motișan, R., & Oancea, C. (2021). Air pollution exposure—the (in)visible risk factor for respiratory diseases [Review of Air pollution exposure—the (in)visible risk factor for respiratory diseases]. Environmental Science and Pollution Research, 28(16), 19615. Springer Science+Business Media.

Banno, A., Reddy, A. T., Lakshmi, S. P., & Reddy, R. C. (2020). Bidirectional interaction of airway epithelial remodeling and inflammation in asthma [Review of Bidirectional interaction of airway epithelial remodeling and inflammation in asthma]. Clinical Science, 134(9), 1063. Portland Press.

Bezerra, F. S., Lanzetti, M., Nesi, R. T., Nagato, A. C., Silva, C. P. e, Kennedy-Feitosa, E., Melo, A. C., Cattani-Cavalieri, I., Pôrto, L. C., & Valença, S. S. (2023). Oxidative Stress and Inflammation in Acute and Chronic Lung Injuries [Review of Oxidative Stress and Inflammation in Acute and Chronic Lung Injuries]. Antioxidants, 12(3), 548. Multidisciplinary Digital Publishing Institute.

Checa, J., & Aran, J. M. (2020). Airway Redox Homeostasis and Inflammation Gone Awry: From Molecular Pathogenesis to Emerging Therapeutics in Respiratory Pathology [Review of Airway Redox Homeostasis and Inflammation Gone Awry: From Molecular Pathogenesis to Emerging Therapeutics in Respiratory Pathology]. International Journal of Molecular Sciences, 21(23), 9317. Multidisciplinary Digital Publishing Institute.

Chung, C., Wu, S.-Y., Chiu, H.-H., Wu, T.-N., Wang, Y.-C., & Lin, M. (2023). Associations of air pollutant concentrations with longitudinal kidney function changes in patients with chronic kidney disease. Scientific Reports, 13(1).

Domingo, J. L., & Rovira, J. (2020). Effects of air pollutants on the transmission and severity of respiratory viral infections [Review of Effects of air pollutants on the transmission and severity of respiratory viral infections]. Environmental Research, 187, 109650. Elsevier BV.

Enweasor, C., Flayer, C. H., & Haczku, A. (2021). Ozone-Induced Oxidative Stress, Neutrophilic Airway Inflammation, and Glucocorticoid Resistance in Asthma [Review of Ozone-Induced Oxidative Stress, Neutrophilic Airway Inflammation, and Glucocorticoid Resistance in Asthma]. Frontiers in Immunology, 12. Frontiers Media.

Garg, D., Mehndiratta, M. M., Wasay, M., & Aggarwal, V. (2022). Air Pollution and Headache Disorders. Annals of Indian Academy of Neurology, 25.

Guo, Q., He, Z., & Wang, Z. (2023). Change in Air Quality during 2014–2021 in Jinan City in China and Its Influencing Factors. Toxics, 11(3), 210.

Hellings, P. W., & Steelant, B. (2020). Epithelial barriers in allergy and asthma [Review of Epithelial barriers in allergy and asthma]. Journal of Allergy and Clinical Immunology, 145(6), 1499. Elsevier BV.

Hopkins, C., Buchheit, K. M., Heffler, E., Cohen, N., Olze, H., Khan, A., Msihid, J., Siddiqui, S., Nash, S. D., Jacob-Nara, J. A., Rowe, P. J., & Deniz, Y. (2022). Improvement in Health-Related Quality of Life with Dupilumab in Patients with Moderate-to-Severe Asthma with Comorbid Chronic Rhinosinusitis with/without Nasal Polyps: An Analysis of the QUEST Study. Journal of Asthma and Allergy, 767.

Kim, H., Ingram, J. L., & Que, L. G. (2023). Effects of Oxidative Stress on Airway Epithelium Permeability in Asthma and Potential Implications for Patients with Comorbid Obesity [Review of Effects of Oxidative Stress on Airway Epithelium Permeability in Asthma and Potential Implications for Patients with Comorbid Obesity]. Journal of Asthma and Allergy, 481. Dove Medical Press.

Kutralam-Muniasamy, G., Pérez-Guevara, F., Roy, P. D., Elizalde, I., & Shruti, V. C. (2020). Impacts of the COVID-19 lockdown on air quality and its association with human mortality trends in megapolis Mexico City. Air Quality Atmosphere & Health, 14(4), 553.

Kuźma, Ł., Małyszko, J., Bachórzewska-Gajewska, H., Kralisz, P., & Dobrzycki, S. (2021). Exposure to air pollution and renal function. Scientific Reports, 11(1).

Låg, M., Øvrevik, J., Refsnes, M., & Holme, J. A. (2020). Potential role of polycyclic aromatic hydrocarbons in air pollution-induced non-malignant respiratory diseases [Review of Potential role of polycyclic aromatic hydrocarbons in air pollution-induced non-malignant respiratory diseases]. Respiratory Research, 21(1). BioMed Central.

Liang, R., Fan, L., Lai, X., Shi, D., Wang, H., Shi, W., Liu, W., Yu, L., Song, J., & Wang, B. (2024). Air pollution exposure, accelerated biological aging, and increased thyroid dysfunction risk: Evidence from a nationwide prospective study. Environment International, 188, 108773.

Liu, B., Fan, D., & Huang, F. (2020). Relationship of chronic kidney disease with major air pollutants - A systematic review and meta-analysis of observational studies [Review of Relationship of chronic kidney disease with major air pollutants - A systematic review and meta-analysis of observational studies]. Environmental Toxicology and Pharmacology, 76, 103355. Elsevier BV.

Manisalidis, I., Stavropoulou, E., Stavropoulos, A., & Bezirtzoglou, E. (2020). Environmental and Health Impacts of Air Pollution: A Review [Review of Environmental and Health Impacts of Air Pollution: A Review]. Frontiers in Public Health, 8. Frontiers Media.

Mocelin, H. T., Fischer, G. B., & Bush, A. (2021). Adverse early-life environmental exposures and their repercussions on adult respiratory health [Review of Adverse early-life environmental exposures and their repercussions on adult respiratory health]. Jornal de Pediatria, 98. Elsevier BV.

Mormile, M., Mormile, I., Fuschillo, S., Rossi, F. W., Lamagna, L., Ambrosino, P., Paulis, A. de, & Maniscalco, M. (2023). Eosinophilic Airway Diseases: From Pathophysiological Mechanisms to Clinical Practice [Review of Eosinophilic Airway Diseases: From Pathophysiological Mechanisms to Clinical Practice]. International Journal of Molecular Sciences, 24(8), 7254. Multidisciplinary Digital Publishing Institute.

Neo, E. X., Hasikin, K., Lai, K. W., Mokhtar, M. I., Azizan, M. M., Hizaddin, H. F., Razak, S. A., & Yanto, Y. (2023). Artificial intelligence-assisted air quality monitoring for smart city management. PeerJ Computer Science, 9.

Okeleji, L. O., Ajayi, A. F., Adebayo-Gege, G. I., Aremu, V. O., Adebayo, O. I., & Adebayo, E. T. (2021). Epidemiologic evidence linking oxidative stress and pulmonary function in healthy populations [Review of Epidemiologic evidence linking oxidative stress and pulmonary function in healthy populations]. Chronic Diseases and Translational Medicine, 7(2), 88. KeAi.

Palupi, F. H., & Abeng, A. T. (2023). The Invisible Threat: Investigating the Effects of Air Pollution on Human Health and the Environment. West Science Interdisciplinary Studies, 1(6), 271.

Pascoe, C. D., Vaghasiya, J. M., & Halayko, A. J. (2020). Oxidation specific epitopes in asthma: New possibilities for treatment [Review of Oxidation specific epitopes in asthma: New possibilities for treatment]. The International Journal of Biochemistry & Cell Biology, 129, 105864. Elsevier BV.

Prada, D., López, G., Solleiro-Villavicencio, H., García-Cuéllar, C. M., & Baccarelli, A. (2020). Molecular and cellular mechanisms linking air pollution and bone damage [Review of Molecular and cellular mechanisms linking air pollution and bone damage]. Environmental Research, 185, 109465. Elsevier BV.

Pryor, J. T., Cowley, L. O., & Simonds, S. E. (2022). The Physiological Effects of Air Pollution: Particulate Matter, Physiology and Disease [Review of The Physiological Effects of Air Pollution: Particulate Matter, Physiology and Disease]. Frontiers in Public Health, 10. Frontiers Media.

Rahman, R.-R., & Kabir, A. (2023). Spatiotemporal analysis and forecasting of air quality in the greater Dhaka region and assessment of a novel particulate matter filtration unit. Environmental Monitoring and Assessment, 195(7).

Roca-Barceló, A., Rice, M. B., Nunez, Y., Thurston, G., Weinmayr, G., Straíf, K., Roscoe, C., Ebi, K. L., Andersen, Z. J., Nazelle, A. de, & Negev, M. (2024). Climate action has valuable health benefits. Environmental Epidemiology, 8(1).

Shin, H., Braun, D., Irene, K., & Antonelli, J. (2023). A spatial interference approach to account for

mobility in air pollution studies with multivariate continuous treatments. arXiv (Cornell University).

Syiemlieh, B., & Mariraj, J. (2020). Study on Chronic Rhinosinusitis: A Clinico-Mycological Perspective in a Tertiary Care Centre. International Journal of Current Microbiology and Applied Sciences, 9(7), 3740.

Tan, K. S., Lim, R. L., Liu, J., Ong, H. H., Tan, V. J., Lim, H. F., Chung, K. F., Adcock, I. M., Chow, V., & Wang, D. Y. (2020). Respiratory Viral Infections in Exacerbation of Chronic Airway Inflammatory Diseases: Novel Mechanisms and Insights From the Upper Airway Epithelium [Review of Respiratory Viral Infections in Exacerbation of Chronic Airway Inflammatory Diseases: Novel Mechanisms and Insights From the Upper Airway Epithelium]. Frontiers in Cell and Developmental Biology, 8. Frontiers Media.

Vasconcelos, L. H. C., Ferreira, S. R. D., Silva, M. da C. C., Ferreira, P. B., Souza, I. L. L. de, Cavalcante, F. de A., & Silva, B. A. da. (2021). Uncovering the Role of Oxidative Imbalance in the Development and Progression of Bronchial Asthma [Review of Uncovering the Role of Oxidative Imbalance in the Development and Progression of Bronchial Asthma]. Oxidative Medicine and Cellular Longevity, 2021(1). Hindawi Publishing Corporation.

Wan, J., Song, J., Lv, Q., Zhang, H., Xiang, Q., Dai, H., Zheng, H., Lin, X., & Zhang, W. (2023). Alterations in the Gut Microbiome of Young Children with Airway Allergic Disease Revealed by Next-Generation Sequencing. Journal of Asthma and Allergy, 961.

Wang, Q., Chen, Z., Huang, W., Kou, B., & Li, J. (2023). Short-Term Effect of Moderate Level Air Pollution on Outpatient Visits for Multiple Clinic Departments: A Time-Series Analysis in Xi'an China. Toxics, 11(2), 166.

Wei, Y., Feng, Y., Yazdi, M. D., Yin, K., Castro, E., Shtein, A., Qiu, X., Peralta, A. A., Coull, B. A., Dominici, F., & Schwartz, J. (2024). Exposure-response associations between chronic exposure to fine particulate matter and risks of hospital admission for major cardiovascular diseases: population based cohort study. BMJ.

Wiegman, C., Li, F., Ryffel, B., Togbe, D., & Chung, K. F. (2020). Oxidative Stress in Ozone-Induced Chronic Lung Inflammation and Emphysema: A Facet of Chronic Obstructive Pulmonary Disease [Review of Oxidative Stress in Ozone-Induced Chronic Lung Inflammation and Emphysema: A Facet of Chronic Obstructive Pulmonary Disease]. Frontiers in Immunology, 11. Frontiers Media.

Yamamoto, A., Sly, P. D., Chew, K. Y., Khachatryan, L., Begum, N., Yeo, A. J., Vu, L. D., Short, K. R., Cormier, S. A., & Fantino, E. (2023). Environmentally persistent free radicals enhance SARS-CoV-2 replication in respiratory epithelium. Experimental Biology and Medicine, 248(3), 271.

Yamasaki, A. (2023). Editorial: Airway remodeling in asthma—what is new? Frontiers in Allergy, 4.

Yang, K., Zhang, G., & Li, Y. (2024). Association between air pollutants, thyroid disorders, and thyroid hormone levels: a scoping review of epidemiological evidence [Review of Association between air pollutants, thyroid disorders, and

thyroid hormone levels: a scoping review of epidemiological evidence]. Frontiers in Endocrinology, 15. Frontiers Media.

Yang, T., Wang, J., Huang, J., Kelly, F. J., & Li, G. (2023). Long-term Exposure to Multiple Ambient Air Pollutants and Association With Incident Depression and Anxiety. JAMA Psychiatry, 80(4), 305