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Original Article

Seasonal Oxidative Stress and Airway Reactivity in Rhinitis: Distinct Patterns in Allergic vs. Non-allergic Individuals

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ABSTRACT

OBJECTIVE: Rhinitis is a common upper airway disorder, classified as either allergic rhinitis (AR) or non-allergic rhinitis (NAR). While the association between air pollution and AR airway diseases has been well documented, its specific effects on NAR remain poorly understood. This study aimed to evaluate the seasonal impact of air pollution on pulmonary function, oxidative stress biomarkers, and bronchial hyperresponsiveness in patients with AR, patients with NAR, and healthy controls.

MATERIAL AND METHODS: In this prospective case-control study, 58 participants (23 AR, 22 NAR, 13 controls) were evaluated during periods of low pollution (summer) and high pollution (winter). Assessments included symptom questionnaires, pulmonary function tests, bronchial provocation tests (BPT), serum total antioxidant status (TAS), and total oxidative status.

RESULTS: In the high pollution period, the NAR group exhibited significantly lower TAS levels compared to summer (1.51±0.15, 1.60±0.2, $P = 0.041$), indicating an increased oxidative stress. A significant decrease in post-bronchodilator forced expiratory volume in 1 second (FEV_1) was also observed in the NAR group, suggesting heightened airway reactivity. The AR group demonstrated a higher frequency of BPT reactivity. Pulmonary function declined across all groups in winter, with the greatest reduction observed in AR patients. Within-group analyses revealed seasonal reductions in both FEV_1 and post-BPT FEV_1 in AR and NAR groups.

CONCLUSION: Seasonal air pollution exerts phenotype-specific effects on oxidative stress and airway reactivity in rhinitis. AR patients exhibited increased bronchial hyperresponsiveness, whereas NAR individuals showed a marked decline in antioxidant capacity. These findings highlight the importance of phenotype-based monitoring and management during periods of high environmental exposure.

KEYWORDS: Rhinitis, allergic rhinitis, non-allergic rhinitis, asthma, oxidative stress, air pollution, allergy

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INTRODUCTION

Rhinitis is a common inflammatory disorder of the upper airways and is classified as allergic rhinitis (AR) or non-allergic rhinitis (NAR) depending on the presence or absence of allergen-specific immunoglobulin E (IgE).[1] AR frequently coexists with asthma and other atopic diseases, forming part of a broader allergic spectrum.[2] Both AR and NAR phenotypes are increasingly influenced by environmental factors, particularly air pollution.[3] Although genetic predisposition contributes to susceptibility, the rapid increase in rhinitis prevalence underscores the importance of environmental exposures, especially urbanization and air pollutants such as particulate matter (PM), nitrogen dioxide (NO_2), and ozone.[4]

Fine $PM_{2.5}$ is of particular concern because it disrupts epithelial barrier integrity and promotes inflammatory responses through the excessive generation of reactive oxygen species (ROS).[5,6] Oxidative stress, defined as an imbalance between

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ROS production and antioxidant defense systems, is considered a key mechanism underlying pollutant-induced airway damage. Biomarkers such as total antioxidant status (TAS) and total oxidant status (TOS) provide integrative measures of systemic redox homeostasis.[7] While these have been studied in asthma and AR,[8] data regarding their relevance in NAR are sparse. Furthermore, limited research has examined how these biomarkers vary seasonally among different rhinitis phenotypes.

Recent evidence suggests that AR patients readily generate ROS in response to environmental triggers,[9] whereas the oxidative profile and airway reactivity of NAR patients under pollution stress remain poorly understood. Some studies suggest a potential link between air pollution and increased bronchial hyperresponsiveness, even in non-asthmatic individuals.[10] This study investigates the seasonal impact of air pollution on oxidative stress and bronchial responsiveness in patients with AR and NAR, and in healthy controls. By evaluating TAS, TOS, and pulmonary parameters during both low-pollution (summer) and high-pollution (winter) seasons, we aim to delineate phenotype-specific responses to environmental oxidative burden.

Study Design

This prospective, observational, case-control study was conducted at a tertiary allergy and immunology clinic in Kırıkkale, Türkiye between January 2022 and June 2023. A total of 58 participants (23 AR, 22 NAR, 13 controls) were enrolled. Each participant was evaluated at two time points corresponding to seasonal variations in air pollution: summer (low-pollution season; June–August) and winter (high-pollution season; December–February). All procedures were carried out in accordance with the Declaration of Helsinki. The study protocol was approved by the Kırıkkale University Clinical Research Ethics Committee (date/approval no: 12.09.2022/07-02), and supported by a Kırıkkale University Scientific Research Grant (BAP project no: 2022/108). Written informed consent was obtained from all participants prior to enrollment.

Kırıkkale is a mid-sized industrial city characterized by substantial seasonal fluctuations in ambient air quality. During winter, average concentrations of sulfur dioxide and PM_{10} exceeded World Health Organization thresholds, reaching 340 $\mu g/m^3$ and 220 $\mu g/m^3$, respectively.[11] These natural seasonal variations provided a real-world context to explore pollution-associated respiratory effects.[12]

Main Points

- Air pollution affects allergic rhinitis and non-allergic rhinitis (NAR) through different pathways.
- Winter pollution causes a significant antioxidant drop, specifically in NAR.
- Both phenotypes show higher bronchial hyperresponsiveness in winter.
- Monitoring total antioxidant status levels may help manage rhinitis during high pollution.

The sample size for the study group was determined using G*Power 3.1.9.7. The mean TAS and TOS values and standard deviations of the patient and control groups in the study by Pekince and Baccioglu[13] were used to determine the sample size. As a result of these analyzes, it was determined that a power of 95% and a type 1 error level of 5% could be achieved with at least 56 cases.

Participants

Eligibility criteria included adults aged 18–60 years who were lifelong non-smokers and long-term residents of Kırıkkale. This age range was selected to minimize age-related comorbidities, polypharmacy and age-dependent variability in lung function that might confound spirometric and bronchial responsiveness measurements. Prior to enrollment, a detailed survey was conducted to assess residence, education level, home heating methods, proximity to industrial zones, and daily outdoor exposure. Based on these data, participants were selected to represent the average environmental exposure profile of the region. Healthy controls were recruited from the same catchment area and with the same characteristics as the patients (hospital staff and hospital visitors without respiratory or allergic disease). To ensure comparability, frequency matching based on age and sex was employed rather than individual matching. Specifically, only non-smokers were included in all three groups. Recruitment was aimed at achieving an age range and female predominance similar to those in the rhinitis groups. All participants resided in the same urban area.

AR was diagnosed based on symptoms and a positive skin prick test (SPT) to common aeroallergens. NAR was defined as chronic rhinitis symptoms with negative SPT results and non-IgE-mediated triggers. Controls had no respiratory disease, no history of allergy, atopy, or chronic illness. Exclusion criteria included pregnancy, a diagnosis of asthma, chronic systemic diseases, recent use of corticosteroids or immunosuppressives, and occupational exposure to inhaled pollutants.

Clinical and Environmental Assessments

Participants underwent comprehensive evaluations during both visits. They completed standardized questionnaires that captured rhinitis symptom severity (total nasal symptom score, TNSS), environmental exposure history, indoor irritant sources, heating type, and duration of outdoor exposure. Physical examinations and pulmonary assessments were performed under standardized clinical conditions during both seasonal visits.

SPT was performed during the initial visit using a panel of standardized aeroallergens. Pulmonary function testing (PFT) included spirometry measurements of forced expiratory volume in 1 second (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio in accordance with American Thoracic Society/European Respiratory Society guidelines.[14] Bronchial hyperresponsiveness was evaluated using a methacholine challenge test, with PD_{20} calculated when feasible.[15]

Blood samples were obtained at both visits for complete blood count, C-reactive protein (CRP), total IgE, and oxidative stress markers. Serum TAS and TOS were quantified using a fully automated spectrophotometric method described by Erel.[7] TAS values were expressed as mmol Trolox equivalents/L and

TOS as μmol H_2O_2 equivalents/L. The oxidative stress index (OSI) was calculated as the TOS/TAS ratio. All measurements were performed in a single central laboratory using the same analyzer, calibrators, and internal controls. Replicate values with discrepancies greater than 10% were retested, and outliers were compared with the original sample and clinical data.

Environmental exposure was further assessed through air quality data from the Turkish National Monitoring Network.[12] Variables such as outdoor exposure duration, home heating type, and passive smoke exposure were also recorded. These data were matched to the corresponding seasonal visits for each participant.

Statistical Analysis

Statistical analyses were performed using SPSS version 23. Data distribution was assessed using the Shapiro–Wilk test. Depending on the normality assumptions, two-group comparisons were performed using t-tests or the Mann–Whitney U test. Additionally, three groups were analyzed using, ANOVA and Kruskal–Wallis test, and post-hoc adjustments. In post-hoc analyses, significant differences were confirmed using Bonferroni and Tamhane's T2 tests, as appropriate for their distributions; for comparisons involving three variables, the alpha value was set at 0.0167. Seasonal within-group changes were assessed using paired t-tests or Wilcoxon signed-rank tests. Categorical variables were analyzed via chi-square or Fisher's exact tests. Correlation analyses were performed using Pearson or Spearman methods, depending on the data distribution. The *P* value <0.05 was considered statistically significant.

RESULTS

Participant Characteristics

A total of 58 participants were included in the study: 23 with AR, 22 with NAR, and 13 healthy controls. The mean age of participants was 30.4±9.4 years, and the majority were female (79.7%). Educational level was generally high across all groups, and the majority of participants (93.2%) resided in urban apartment settings in the city center. Evaluation of environmental factors showed a high rate of indoor irritant exposure: approximately 50% of cases reported exposure to detergents, air fresheners, and cigarette smoke. Similarly, approximately 50% of the patients lived on main streets or in areas of heavy traffic. No statistically significant differences were detected between the AR and NAR groups with respect to these exposure characteristics.

Environmental Exposures and Trigger Factors

Environmental and exposure characteristics were similar between the AR and NAR groups. The majority of patients with rhinitis (77%) reported identifiable symptom triggers, most commonly air pollution, dust, and cold air. Only one-third of symptomatic participants reported symptom control during the study period. Indoor irritants, particularly detergents and air fresheners, were frequently reported and did not differ significantly across groups.

Pulmonary Function and Seasonal Variation

Baseline pulmonary function values, including FEV_1, were similar among the three groups during both seasonal visits. A general decline in FEV_1 was observed during the winter period, although differences were not statistically significant in intergroup comparisons. Mean winter declines in FEV_1 were -3.2% in the NAR group, -2.8% in the AR group, and -1.9% in the control group. This seasonal decrease in pulmonary function is illustrated in Figure 1.

Oxidative Stress Markers

TAS, TOS, and OSI were evaluated in all participants. Intergroup comparisons revealed no significant seasonal differences in oxidative stress parameters (Table 1). However, within-group analysis in the NAR group showed a significant reduction in TAS levels during winter ($P < 0.05$), suggesting an increased oxidative burden during periods of high air pollution. This decline is depicted in Figure 2.

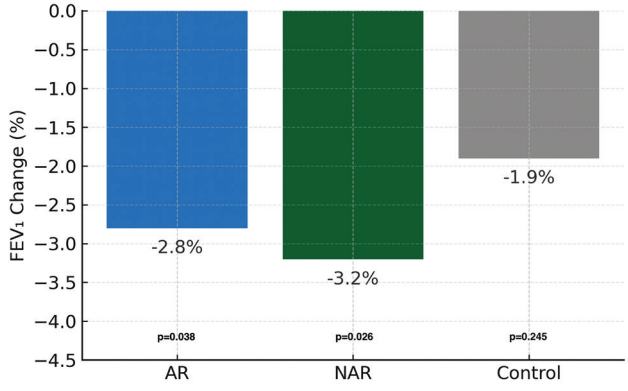

Figure 1. Seasonal changes in FEV_1 values across allergic rhinitis (AR), non-allergic rhinitis (NAR) and control groups. FEV_1: forced expiratory volume in 1 second

Table 1. Baseline demographic characteristics and oxidative stress of study groups

Parameter	AR (n = 23)	NAR (n = 22)	Control (n = 13)	*P* value
Age	28.5±9.3	34±12.2	29.3±5.8	0.105
Sex, female	16 (69.6%)	19 (86.4%)	24 (82.8%)	0.327
BMI (kg/m²)	24.2±3.1	24.6±3.5	23.9±2.8	0.753
TAS (mmol Trolox equiv./L)	1.59±0.22	1.58±0.17	1.56±0.15	0.819
TOS (μmol H_2O_2 equiv./L)	8.4±4.8	9.2±5.6	10.1±6.5	0.570
OSI (arbitrary units)	0.52±0.30	0.58±0.33	0.69±0.49	0.295

Control participants were presented with the average of both summer and winter period data
The ANOVA test and the Kruskal–Wallis test were used according to distributions
AR: allergic rhinitis, NAR: non-allergic rhinitis, BMI: body mass index, TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, FEV_1: forced expiratory volume in 1 second

TOS and OSI values did not exhibit significant changes in any group (Tables 2 and 3). Collectively, these findings suggest that NAR patients may be more susceptible to pollutant-associated oxidative imbalance, whereas AR participants may exhibit relatively stable systemic oxidative profiles.

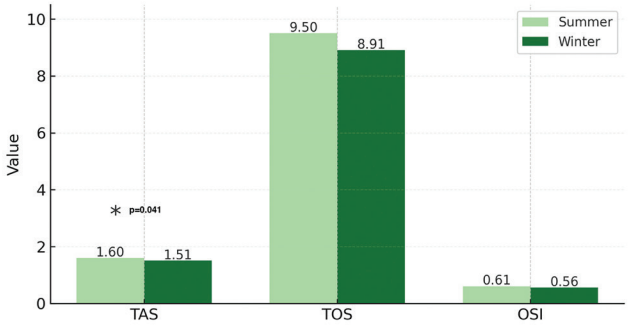

Figure 2. Seasonal comparison of TAS, TOS, and OSI levels in the NAR group

TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, NAR: non-allergic rhinitis

Bronchial Hyperresponsiveness

Bronchial provocation testing (BPT) results indicated seasonal changes in airway responsiveness. In winter, the NAR group showed a significant reduction in post-BPT FEV_1 (% change from baseline),indicating increased bronchial hyperresponsiveness ($P = 0.02$). The AR group also showed seasonal reductions in both FEV_1 ($P = 0.038$) and post-BPT FEV_1 ($P = 0.009$),though to a lesser extent. These findings are visualized in Figure 3 and detailed in Table 3.

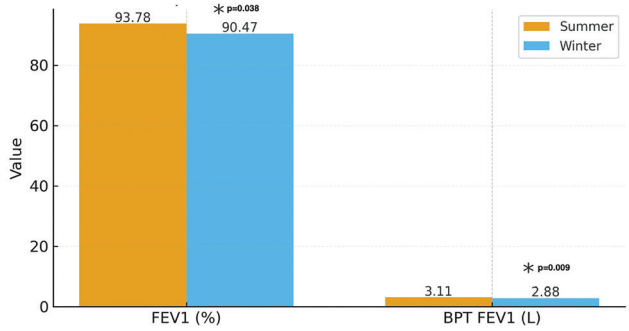

Figure 3. Seasonal changes in forced expiratory volume in 1 second (FEV_1) and post-bronchial provocation test (BPT) FEV_1 values in the allergic rhinitis (AR) group

Table 2. Comparison of the variables examined during the summer visit of the study patients

Parameter	AR (n = 23)	NAR (n = 22)	Control (n = 13)	*P* value
TAS (mmol Trolox equiv./L)	1.6±0.23	1.6±0.2	1.55±0.19	0.737
TOS (μmol H_2O_2 equiv./L)	9.6±8.3	9.5±7.2	11.8±8.2	0.668
OSI (arbitrary units)	0.581±0.51	0.61±0.48	0.803±0.6	0.455
TNSS	6.7±3.1	6.1±2.5	0.2±0.8	<0.001*
FEV_1 (L)	3.51±0.75	2.93±0.5	3.19±0.45	0.009
FEV_1 (%)	93.7±8.5	93.9±10.4	98.7±9.4	0.270
BPT FEV_1 (L)	3.11±0.55	2.6±0.57	3.01±0.58	0.011
BPT FEV_1 change	-9.3±7.6	-9.8±9.06	-5.94±8.7	0.395

*A significant difference was observed between the control and AR/NAR groups. The ANOVA test and the Kruskal–Wallis test were used according to distributions

AR: allergic rhinitis, NAR: non-allergic rhinitis, TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, TNSS: total nasal symptom score, BPT: bronchial provocation test, FEV_1: forced expiratory volume in 1 second

Table 3. Comparison of the variables examined during the winter visit of the study patients

Parameter	AR (n = 23)	NAR (n = 22)	Control (n = 13)	*P* value
TAS (mmol Trolox equiv./L)	1.58±0.22	1.51±0.15	1.45±0.17	0.123
TOS (μmol H_2O_2 equiv./L)	7.27±3.58	8.91±7.83	8.04±8.20	0.658
OSI (arbitrary units)	0.47±0.25	0.57±0.41	0.56±0.41	0.594
TNSS	6.5±3.2	6.6±2.7	0.8±1.6	<0.001*
FEV_1 (L)	3.40±0.71	2.87±0.54	3.24±0.80	0.036**
FEV_1 (% predicted)	90.4±9.7	90.6±11.3	93.4±9.3	0.629
BPT FEV_1 (L)	2.88±0.69	2.42±0.56	2.90±0.72	0.036**
BPT FEV_1 change (%)	-11.6±11.2	-12.9±11.7	-7.1±9.3	0.254

*A significant difference was observed between the control and AR/NAR groups.

**A significant difference was observed between the NAR and AR/control groups.

The ANOVA test and the Kruskal-Wallis test were used according to distributions

AR: allergic rhinitis, NAR: non-allergic rhinitis, TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, BPT: bronchial provocation test, TNSS: total nasal symptom score, FEV_1: forced expiratory volume in 1 second

Notably, the AR group maintained stable TAS levels despite the functional decline in FEV_1. The control group showed no significant seasonal variation in oxidative or pulmonary markers. These findings suggest that while both rhinitis phenotypes experience functional impairment in winter, the underlying mechanisms may differ, with oxidative stress being more pronounced in NAR and airway hyperresponsiveness (AHR) being more evident in AR.

Intergroup and Seasonal Comparisons

During the summer and winter visits, no statistically significant differences were observed in TAS, TOS, or body mass index among the three groups. However, TNSS was significantly higher in both rhinitis groups compared to controls ($P < 0.001$). FEV_1 values were lower in the NAR group than in the AR group and the controls ($P = 0.009$). These results are summarized in Tables 2, 3.

A more detailed comparison of the groups during winter is presented in Table 4a. While OSI and TOS remained stable across phenotypes, TAS showed a significantly greater reduction in the NAR group than in AR ($P = 0.041$). No significant seasonal differences were noted in OSI or post-BPT FEV_1 between AR and NAR. These comparisons are shown in Table 4a, 4b.

Correlation Analyses

A moderate inverse correlation was observed between seasonal changes in TAS and TNSS (r: -0.337, $P = 0.012$), suggesting that reduced antioxidant capacity is associated with increased nasal symptom severity. No significant correlations were found between OSI or TOS and symptom scores.

Inflammatory markers, such as CRP and white blood cell counts, showed no significant variation between groups. However, neutrophil counts were significantly higher in the AR group than in the NAR group during winter ($P < 0.05$), potentially indicating differing inflammatory responses to pollution exposure.

Further correlation analyzes demonstrated associations between environmental pollutant levels and clinical parameters. Elevated $PM_{2.5}$, NO_2, and CO levels were significantly correlated with higher TOS and OSI values, and with lower FEV_1 (change from baseline) ($P < 0.05$). These findings support a link between ambient air pollution and systemic oxidative stress and pulmonary impairment, particularly among patients with rhinitis.

Table 4a. Comparison of changes in respiratory function test and laboratory parameters of NAR group patients in winter (W) and summer (S)

Variables	Mean (W)	Mean (S)	*P* value
TAS	1.51±0.1576	1.60±0.20	0.041
TOS	8.91±7.8345	9.50±7.29	0.782
OSI	0.56±0.4112	0.61±0.48	0.747
TNSS	6.63±2.73	6.18±2.59	0.116
FEV_1 (L)	2.87±0.54	2.93±0.50	0.314
FEV_1 (%)	90.63±11.31	93.95±10.40	0.026
BPT FEV_1 (L)	2.42±0.56	2.6005±0.5708	0.110
FEV_1 change (%)	-12.99±11.76	-9.8273±9.0653	0.234

Paired t-tests or Wilcoxon signed-rank tests were used according to distributions. The *P* value <0.05 is considered statistically significant

NAR: non-allergic rhinitis, TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, BPT: bronchial provocation test, TNSS: total nasal symptom score, FEV_1: forced expiratory volume in 1 second

Table 4b. Comparison of changes in respiratory function test and laboratory parameters of AR group patients in winter (W) and summer (S)

Variables	Mean (W)	Mean (S)	*P* value
TAS	1.58±0.22	1.60±0.23	0.559
TOS	7.27±3.58	9.60±8.36	0.201
OSI	0.46±0.25	0.58±0.51	0.322
TNSS	6.52±3.21	6.78±3.19	0.299
FEV_1 (L)	3.40±0.72	3.51±0.75	0.115
FEV_1 (%)	90.47±9.76	93.78±8.58	0.038
BPT FEV_1 (L)	2.88±0.69	3.11±0.55	0.009
FEV_1 change (%)	-11.60±11.22	-9.308±7.60	0.174

Paired t-tests or Wilcoxon signed-rank tests were used according to distributions. The *P* value <0.05 is considered statistically significant

AR: allergic rhinitis, TAS: total antioxidant status, TOS: total oxidant status, OSI: oxidative stress index, BPT: bronchial provocation test, TNSS: total nasal symptom score, FEV_1: forced expiratory volume in 1 second

DISCUSSION

This study investigated the seasonal effects of air pollution on oxidative stress and airway responsiveness in patients with AR and NAR, revealing distinct pathophysiological responses. In winter, when pollutant concentrations were elevated, the NAR group exhibited a significant decline in TAS and an increase in bronchial hyperresponsiveness, whereas the AR group showed greater functional impairment on spirometry but maintained stable antioxidant profiles.

These findings suggest phenotype-specific vulnerability to environmental stressors. Previous studies have shown that PM and gaseous pollutants such as NO_2 and CO contribute to epithelial barrier dysfunction and oxidative stress via excessive ROS production.[5,6] Oxidative imbalance has been well documented in asthma and AR,[8] but data regarding NAR remain limited. Our results demonstrate that NAR patients, despite lacking IgE-mediated inflammation, may exhibit impaired redox balance during high-exposure periods, consistent with emerging studies on non-atopic airway diseases.[16]

Interestingly, TAS levels declined significantly only in the NAR group, while TOS and OSI remained stable across all phenotypes. This may suggest that antioxidant depletion, rather than oxidant excess, is a key driver of seasonal redox shifts in NAR. Li et al.[17] reported similar patterns in asthmatic children exposed to air pollution, noting stronger oxidative biomarker responses in non-allergic subtypes. Pekince and Baccioglu[13] also demonstrated that oxidative impairment due to pollution was more prominent in non-allergic asthma patients, paralleling our observations in NAR. However, the wide standard deviation observed in TOS values likely reflects inter-individual biological heterogeneity, potentially driven by genetic *polymorphisms* in antioxidant enzyme systems (e.g. GSTM1, GSTP1), which are known to modulate individual responses to oxidative triggers[18] rather than methodological limitations. This suggests that antioxidant capacity (TAS) might be a more sensitive marker for detecting pollution-induced redox shifts in this cohort.

AHR, classically associated with asthma and AR[15] was also significantly increased in NAR during winter. Crucially, although seasonal AHR was observed in both groups, the underlying mechanisms appear to diverge. The specific reduction in TAS observed in NAR suggests that airway reactivity in this phenotype may be driven by failure of antioxidant defenses, whereas in AR it likely results from aggravation of established allergic inflammation, independent of systemic redox shifts. This raises the possibility that NAR patients may have heightened reactivity to non-allergic stimuli, such as air pollutants, through non-IgE pathways involving neurogenic inflammation or epithelial-derived mediators.[19,20] The modest inverse correlation between TAS and TNSS further supports the hypothesis that oxidative stress contributes to symptom exacerbation.

Environmental pollutant levels ($PM_{2.5}$, NO_2, CO) were significantly associated with reduced pulmonary function and elevated TOS/OSI values, corroborating previous research linking air pollution to systemic inflammation and respiratory morbidity.[4,5] These findings emphasize that ambient exposures can impact both upper and lower airway physiology, even in individuals without classic allergic sensitization.

This study contributes to the growing recognition that NAR is not a benign or "residual" diagnosis, but rather may represent a distinct inflammatory endotype with environmental susceptibility. The observed functional and biochemical impairments highlight the need for individualized clinical management, including environmental exposure counseling and possibly antioxidant strategies.

While this study has several strengths, including seasonal design and the use of validated clinical and biochemical markers, some limitations must be acknowledged. The sample size was modest, and individual exposure levels were not directly measured. The methacholine challenge, although standardized, may not capture all dimensions of AHR in rhinitis patients. TAS and TOS are systemic markers and may not fully reflect local nasal or bronchial oxidative status. Air pollution exposure was estimated using city-level data from fixed monitoring stations, without personal exposure monitoring or time-activity diaries. Therefore, individual exposure misclassification is possible. Indoor air pollutant levels and detailed home microenvironment characteristics were not systematically measured. The study was conducted in a single center with a modest sample size, which limits generalizability to populations with different genetic, dietary, or pollution profiles. Multicenter studies in diverse settings are needed to confirm these phenotype-specific patterns. These limitations may introduce bias and should be considered when interpreting our findings.

In summary, our findings support a model in which seasonal air pollution induces distinct oxidative and functional responses in AR and NAR phenotypes. The disproportionate impact on NAR patients suggests that environmental monitoring, phenotype-specific evaluation, and tailored interventions are critical for optimal disease management in polluted settings.

CONCLUSION

Seasonal air pollution exerts differential effects on patients with AR and NAR. While AR patients exhibited greater bronchial hyperresponsiveness, individuals with NAR demonstrated significant reductions in antioxidant capacity during the winter months, highlighting a potential vulnerability to an oxidative imbalance. These findings underscore the need for phenotype-specific management strategies that incorporate environmental exposure assessment. Monitoring redox biomarkers and implementing protective interventions during high-pollution periods may improve clinical outcomes in susceptible populations.

Ethics

Ethics Committee Approval: The study protocol was approved by the Kırıkkale University Clinical Research Ethics Committee (date/approval no: 12.09.2022/07-02).

Informed Consent: Written informed consent was obtained from all participants prior to enrollment.

Footnotes

Authorship Contributions

Concept: S.A.Y., A.F.K., A.B., Design: S.A.Y., A.F.K., A.B., Data Collection or Processing: S.A.Y., A.B., Analysis or Interpretation: S.A.Y., A.B., Literature Search: S.A.Y., A.B., Writing: S.A.Y.

Conflict of Interest: No conflict of interest was declared by the authors.

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