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Research Article

Associations Between Ambient and Indoor Air Pollution Exposure and the Presence of Rhinitis Symptoms in the Past 12 Months: Income-Related Differences in a General Adult Population

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Previous studies on the associations between exposure to various indoor air pollutants, as well as coexposure to ambient and indoor air pollutants, and rhinitis are limited. Data from the Korea National Health and Nutrition Examination Survey (2020–2021) were analyzed ($n = 1812$). Ambient air pollutant concentrations were estimated using the Community Multiscale Air Quality model, while indoor air pollutant concentrations were measured in each household. Associations with the presence of rhinitis symptoms in the past 12 months (PRS) were examined using logistic regression models for individual ambient and indoor air pollutants, as well as for a composite exposure variable that combined ambient fine particulate matter ($PM_{2.5}$) and indoor total volatile organic compound (TVOC) concentrations (*low–low*, *low–high*, *high–low*, and *high–high*). Stratified analyses by household income were conducted using the same models. A doubling of ambient $PM_{2.5}$ concentrations over 1 year was associated with higher odds of PRS (odds ratio [OR] = 2.68, 95% confidence interval [CI]: 1.45, 4.78). A doubling of TVOC (OR = 1.08, 95% CI: 1.01, 1.16) and toluene (OR = 1.10, 95% CI: 1.02, 1.20) concentrations was associated with higher odds of PRS. Compared to the *low–low* group, other composite exposure groups had increased odds of PRS. These associations were more pronounced among individuals with lower income than among those with higher income. Ambient and indoor air pollution exposures were both individually and collectively associated with a higher risk of rhinitis symptoms among adults. Since this study used a cross-sectional design, further longitudinal studies are needed.

Keywords: ambient air pollution; coexposure; indoor air pollution; rhinitis

1. Introduction

Rhinitis is a common inflammatory disorder of the nasal mucosa, characterized by symptoms such as nasal congestion, rhinorrhea, sneezing, and nasal itching. Although it is often regarded as a minor condition, its chronic and recurrent nature imposes a considerable burden on both individuals and healthcare systems. In adults, rhinitis is associated with impaired quality of life and reduced daily productivity, as well as an increased risk of comorbidities such as asthma and chronic rhinosinusitis [1, 2]. The global prevalence of rhinitis ranges from 12.0% to 29.4%, with a reported prevalence of 17.1% in Korea [3–5]. Notably, the prevalence of rhinitis has been steadily increasing worldwide over recent

decades, particularly among adults [1, 4, 5], with environmental exposures possibly contributing to this trend.

Among the environmental factors which may potentially contribute to this increase in rhinitis prevalence, air pollution has been identified as one of the key risk factors. For example, long-term exposure to particulate matter with an aerodynamic diameter $\leq 2.5\,\mu$m ($PM_{2.5}$) was reported to be associated with increased rhinitis risk in French and Italian adults [6–8]. However, most studies have assessed ambient air pollutants or indoor air quality proxies such as solid fuel use [9–12], and studies using directly measured indoor air pollutants remain relatively limited. Furthermore, although individuals are simultaneously exposed to both ambient and indoor air pollution [13, 14], the combined effects of these exposures on rhinitis risk have not been thoroughly investigated and remain a critical gap in the literature.

Individuals with low incomes are more vulnerable to environmental hazards due to factors such as inadequate housing, limited access to healthcare, and higher baseline levels of exposure [15–19]. These vulnerabilities may heighten susceptibility to the respiratory effects of air pollution, including rhinitis. Although a stronger association between air pollution and rhinitis might be expected among low-income populations, no study to date has examined income-based differences in this association. This knowledge gap limits our understanding of environmental health disparities and impedes the development of effective, equity-focused interventions.

Therefore, we investigated the associations of individual ambient and indoor air pollutants, as well as their combined exposures, with the presence of rhinitis symptoms in the past 12 months (PRS), which is a widely used proxy indicator of current rhinitis [20–22], in a nationally representative sample of Korean adults. We also assessed whether these associations varied by household income levels.

2. Methods

2.1. Study Population. We obtained data from the Korea National Health and Nutrition Examination Survey (KNHANES), a nationwide cross-sectional study conducted by the Korea Disease Control and Prevention Agency (KDCA). Details of KNHANES are given elsewhere [23]. This survey selected participants using a stratified cluster sampling method, including health examinations with anthropometric measurements, biological sample collection, nutritional assessments, and health interviews. The survey was conducted periodically at 3-year intervals between 1998 and 2005 and then annually from 2007 onwards. We also used data from the Home Indoor Air Quality and Environmental Pollutant Biomarker Survey, which measured indoor air pollutant concentrations in the homes of a subset of participants in the Eighth KNHANES (2020–2021), selected using a stratified random sampling design that accounted for season, geographic region, and housing type [24]. After merging the indoor air pollution data with the Eighth KNHANES data to analyze individuals who underwent residential indoor air pollution measurements, a total of 1980 individuals aged 19 years or older were included in the initial sample. We excluded 89 individuals with missing data on PRS and 79 individuals with missing data on covariates (income levels [$n = 5$] and educational level [$n = 74$]), resulting in a final sample of 1812 individuals for analysis.

2.2. Estimation of Ambient Air Pollutant Concentrations. Detailed methods for estimating ambient air pollutant levels have been described elsewhere [25]. Briefly, concentrations of ambient air pollutants—including $PM_{2.5}$, particulate matter with an aerodynamic diameter $\leq 10\,\mu$m (PM_{10}), and nitrogen dioxide (NO_2)—were initially estimated at each participant's residential address using the Community Multiscale Air Quality (CMAQ) model (Version 4.7.1), a chemical transport model developed by the US Environmental Protection Agency. CMAQ model predictions were subsequently refined using ground-based monitoring data from Korea and China, employing Pun's interpolation method [26], to improve prediction accuracy. This approach yielded spatially resolved estimates of NO_2 concentrations at a 3×3 km resolution. For $PM_{2.5}$ and PM_{10}, additional multiple linear regression models were developed incorporating satellite-derived aerosol optical depth, temperature, humidity, and CMAQ model outputs as predictors, enabling prediction at a finer 1×1 km resolution [25]. Annual concentrations of $PM_{2.5}$, PM_{10}, and NO_2—calculated as 1-year moving averages of daily concentrations—were used as the exposures of interest for ambient air pollution.

2.3. Measurement of Indoor Air Pollutant Concentrations. Concentrations of indoor air pollutants—total volatile organic compounds (TVOCs), benzene, toluene, ethylbenzene, xylene, styrene, formaldehyde, $PM_{2.5}$, and carbon dioxide (CO_2)—were measured in participants' homes in accordance with the Indoor Air Quality Measurement Process Test Standard established by the Ministry of Environment of Korea [24] and were used as the exposures of interest for indoor air pollutants. On-site measurements were conducted by trained field personnel using standardized procedures and calibrated equipment. Briefly, concentrations of TVOC and its constituent compounds (benzene, toluene, ethylbenzene, xylene, and styrene) were measured twice for 30 min each using Tenax TA sorbent tubes, followed by thermal desorption and gas chromatography-mass spectrometry/flame ionization detection (TD-20/GC/MS-QP2020, Shimadzu). Formaldehyde concentrations were determined using 2,4-dinitrophenylhydrazine (2,4-DNPH) cartridges with ozone scrubbers, analyzed by liquid chromatography (LC2030-Plus, Shimadzu). $PM_{2.5}$ levels were measured over a 24-h period using the gravimetric method (KMS-4100, KEMIK), while CO_2 concentrations were assessed over a 1-h period using a nondispersive infrared method (IQ-610Xtra, GrayWolf Sensing Solutions). All indoor air pollutant measurements were conducted in the primary living area of each household (e.g., living room), following standardized sampling height and consistent distances from walls or windows.

2.4. Determination of the PRS. PRS was determined using the following question from the KNHANES questionnaire: "*In*

the past 12 months, have you had non-inflammatory symptoms such as sneezing, rhinorrhea, nasal congestion, or an itchy nose, not related to a cold (fever, sore throat)?" Individuals who answered "yes" to this question were classified as positive for PRS. Previous studies among adults and children have validated PRS as a proxy indicator of current rhinitis [20–22], with reported sensitivity and specificity exceeding 70% compared to physician-diagnosed rhinitis [27].

2.5. Information on Income Levels and Other Covariates. Based on previous studies examining the association between exposure to air pollution and rhinitis [28–30] and informed by expert opinion, we identified the following potential confounders or predictors for PRS: age (years), sex (male or female), income levels (total household income divided by the square root of the number of household members; quartiles), educational level (≤ middle school, high school, or ≥ college), marital or cohabitation status (no or yes), and tobacco smoking (never smoker or ever smoker).

2.6. Statistical Analysis. Concentrations of ambient ($PM_{2.5}$, PM_{10}, and NO_2) and indoor air pollutants (TVOC, benzene, toluene, ethylbenzene, xylene, styrene, formaldehyde, $PM_{2.5}$, and CO_2) were right-skewed and were log-transformed using base 2 to approximate normal distributions. These log-transformed values were used in all subsequent analyses. To account for potential nonlinear exposure–response relationships, we also used tertile-categorized variables for ambient and indoor air pollutants in our analyses.

Because KNHANES employed a complex survey design, we applied appropriate strata, cluster, and weight variables in all association analyses. To examine the associations between individual ambient and indoor air pollutants and PRS, we constructed logistic regression models—both unadjusted and adjusted for the aforementioned covariates—and estimated odds ratios (ORs) with corresponding 95% confidence intervals (CIs).

To evaluate the potential synergistic impact of ambient and indoor air pollution on PRS, we first selected $PM_{2.5}$ as an indicator of ambient air pollution and TVOC as an indicator of indoor air pollution, as these pollutants were most consistently associated with PRS in the analyses examining individual exposures. We then created a composite exposure variable by combining $PM_{2.5}$ levels categorized as < median (*low*) or ≥ median (*high*) with TVOC levels categorized in the same way, resulting in four categories: *low–low*, *low–high*, *high–low*, and *high–high*. This composite exposure variable was used as an independent variable in a similar logistic regression model to examine the association between coexposure to ambient and indoor air pollutants and PRS. The *low–low* category was used as the reference group for this analysis. We assessed the interaction between ambient $PM_{2.5}$ and indoor TVOC by formally testing the interaction term between the two pollutants categorized as < median and ≥ median, as well as the interaction term between continuous $PM_{2.5}$ and TVOC, using the Wald test.

We further evaluated a joint association between coexposure to ambient $PM_{2.5}$ and indoor TVOC and PRS using a quantile g-computation analysis with a binomial distribution for the outcome [31]. Ambient $PM_{2.5}$ and indoor TVOC were selected as exposures in this mixture analysis to ensure comparability with the analysis using the composite exposure variable and to enhance the interpretability of the findings. We adjusted for the same set of covariates and estimated the results using 300 bootstrap iterations. Because we categorized pollutant concentrations into tertiles in this analysis—in line with the analyses using individual pollutants as exposures—the result can be interpreted as the OR for PRS per simultaneous one-tertile increase in the included pollutants.

We assessed income-related heterogeneity in the associations between individual ambient $PM_{2.5}$ and indoor TVOC, as well as the composite exposure variable and PRS through stratified analyses by income level, categorized into quartiles: Q1 (low), Q2, Q3, and Q4 (high). We also conducted quantile g-computation analyses stratified by income level, categorized into quartiles.

All data management and statistical analyses, except for the quantile g-computation analysis, were performed using SAS Version 9.4 (SAS Institute Inc.). Quantile g-computation analysis and figure generation were conducted using R Version 4.4.3 (R Development Core Team).

2.7. Ethical Consideration. This study utilized data from KNHANES, which was approved by the Institutional Review Board (IRB) of the KDCA. All KNHANES participants provided written informed consent. The KNHANES data are publicly available, and all participants' information was anonymized to ensure confidentiality. Given the use of deidentified and publicly accessible data, this study was exempt from the IRB by the Yonsei University Health System (IRB No. 2025-1333-0001).

3. Results

The characteristics of the study participants are presented in Table 1 ($n = 1812$). The mean age was 53.4 ± 17.2 years, and 55.1% of the study participants were women. A total of 30.7% of participants were in the highest income quartile (Q4), and 40.4% had attained a college education or higher. Most participants were married or living with a partner (83.0%) and had never smoked (85.1%). Among the 1812 individuals, 780 reported PRS. Compared to those without PRS, participants with PRS had a higher proportion of college-educated individuals (43.5% vs. 38.1%).

The mean concentration of ambient $PM_{2.5}$ was 20.7 $\mu g/m^3$, and the mean concentration of indoor TVOC was 239.9 $\mu g/m^3$ in the total population. Compared to individuals without PRS, those with PRS exhibited higher concentrations of TVOC (257.5 vs. 225.1 $\mu g/m^3$) and formaldehyde (29.4 vs. 26.4 $\mu g/m^3$), whereas the levels of other pollutants were comparable between the two groups. In income-stratified analyses, concentrations of styrene, indoor $PM_{2.5}$, and CO_2 were generally higher in the lower-income groups (Q1 and Q2) than in the higher-income groups (Q3 and Q4) (Table S1).

In the analyses for the associations between ambient air pollution and PRS, a doubling of $PM_{2.5}$ concentrations was

TABLE 1: Characteristics of study participants.

Variables	Total (n = 1812)	Participants with PRS (n = 780)	Participants without PRS (n = 1032)
Age (year)	53.4 ± 17.2	54.2 ± 17.0	52.3 ± 17.4
Sex			
Male	814 (44.9)	354 (45.4)	460 (44.6)
Female	998 (55.1)	426 (54.6)	572 (55.4)
Income level			
Q1 (low)	341 (18.8)	144 (18.5)	197 (19.1)
Q2	449 (24.7)	193 (24.7)	256 (24.8)
Q3	465 (25.7)	202 (25.9)	263 (25.5)
Q4 (high)	557 (30.7)	241 (30.9)	316(30.6)
Educational level			
≤ Middle school	513 (28.3)	197 (25.3)	316 (30.6)
High school	567 (31.3)	244 (31.3)	323 (31.3)
≥ College	732 (40.4)	339 (43.5)	393 (38.1)
Marital or cohabitation status			
No	308 (17.0)	152 (19.5)	156 (15.1)
Yes	1504 (83.0)	628 (80.5)	876 (84.9)
Tobacco smoking			
Never smoker	1543 (85.1)	676 (86.7)	867 (84.0)
Ever smoker	269 (14.9)	104 (13.3)	165 (16.0)

Note: Values are presented as mean ± standard deviation for continuous variables and n (%) for categorical variables.
Abbreviation: PRS, the presence of rhinitis symptoms in the past 12 months.

associated with higher odds of PRS (OR = 2.68, 95% CI: 1.45, 4.78). Compared to the first tertile of $PM_{2.5}$, the third tertile was associated with higher odds of PRS (OR = 1.64, 95% CI: 1.21, 2.22). The associations were similar for PM_{10}, with an OR of 2.39 (95% CI: 1.36, 4.18) for doubling of concentration and an OR of 1.66 (95% CI: 1.25, 2.21) for the third tertile compared to the first tertile (Table 2).

In the analyses for the associations between indoor air pollution and PRS, TVOC concentrations were associated with higher odds of PRS, with an OR of 1.08 (95% CI: 1.01, 1.16) per doubling of concentration and an OR of 1.37 (95% CI: 1.02, 1.84) for the third tertile compared to the first tertile. Similarly, toluene showed an OR of 1.10 (95% CI: 1.02, 1.20) per doubling of concentration and an OR of 1.44 (95% CI: 1.04, 1.99) for the third tertile compared to the first tertile (Table 3).

Compared to the low $PM_{2.5}$ and low TVOC (*low–low*) group, the *low–high* (OR = 1.42, 95% CI: 0.99, 2.02), *high–low* (OR = 1.58, 95% CI: 1.11, 2.25), and *high–high* (OR = 1.80, 95% CI: 1.28, 2.54) groups had increased odds of PRS. However, the interaction between ambient $PM_{2.5}$ and indoor TVOC was not statistically significant, with p values for interaction of 0.35 when $PM_{2.5}$ and TVOC were treated as binary variables (< median and ≥ median) and 0.71 when treated as continuous variables (Table S2). In the quantile g-computation analyses, a simultaneous one-tertile increase in ambient $PM_{2.5}$ and indoor TVOC was associated with higher odds of PRS (OR = 1.27, 95% CI: 1.16, 1.40).

Income-stratified analyses revealed that the associations of ambient and indoor air pollution with PRS were not observed in the high-income group (Q4) and were generally stronger in the lower-income groups, especially for indoor air pollutants such as benzene (OR = 1.45, 95% CI: 1.11, 1.89 in Q1) and formaldehyde (OR = 1.41, 95% CI: 1.10, 1.81 in Q1) (Figure 1 and Table S3). The associations between the composite exposure variable of ambient $PM_{2.5}$ and indoor TVOC and PRS were also not observed in the high-income group (Q4) (OR = 1.25, 95% CI: 0.72, 2.16 for the *high–high* group compared to the *low–low* group) and were stronger in the lower-income groups (OR = 2.72, 95% CI: 1.40, 5.30 for the *high–high* group compared to the *low–low* group in Q2; OR = 3.49, 95% CI: 1.77, 6.89 in Q1) (Figure 2 and Table S4). Quantile g-computation analyses stratified by income levels yielded similar results, with more pronounced associations observed in the lower-income groups (OR = 1.31, 95% CI: 1.03, 1.66 in Q1; OR = 1.44, 95% CI: 1.21, 1.73 in Q2) and a weaker, imprecise association in the high-income group (OR = 1.15, 95% CI: 0.98, 1.35 in Q4) (Table S5).

4. Discussion

Exposure to ambient air pollutants, such as $PM_{2.5}$ and PM_{10}, and indoor air pollutants, such as TVOC and toluene, was associated with a higher risk of PRS in the general adult population. In the analyses using the composite exposure variable of ambient $PM_{2.5}$ and indoor TVOC, the *low–high*, *high–low*, and *high–high* groups exhibited increased odds of PRS compared to the *low–low* group. Income-stratified analyses revealed that the associations of ambient and indoor pollutants, as well as their combined exposure, with

TABLE 2: Associations between ambient air pollutant concentrations over 1 year and the presence of rhinitis symptoms in the past 12 months among adults.

Pollutants	Crude		Adjusted	
	OR	95% CI	OR	95% CI
$PM_{2.5}$				
Tertile 1	Ref.		Ref.	
Tertile 2	1.30	0.97, 1.74	1.30	0.97, 1.76
Tertile 3	1.65	1.22, 2.23	1.64	1.21, 2.22
Continuous	2.70	1.49, 4.87	2.68	1.45, 4.78
PM_{10}				
Tertile 1	Ref.		Ref.	
Tertile 2	1.22	0.91, 1.65	1.22	0.90, 1.64
Tertile 3	1.69	1.28, 2.24	1.66	1.25, 2.21
Continuous	2.41	1.37, 4.26	2.39	1.36, 4.18
NO_2				
Tertile 1	Ref.		Ref.	
Tertile 2	1.14	0.85, 1.53	1.15	0.86, 1.55
Tertile 3	1.24	0.94, 1.62	1.22	0.92, 1.62
Continuous	1.17	1.01, 1.35	1.16	0.99, 1.36

Note: Odds ratios and 95% confidence intervals were estimated using logistic regression models incorporating appropriate strata, cluster, and weight variables. The models were adjusted for age, sex, income level, educational level, marital or cohabitation status, and tobacco smoking. The results for continuous exposures are presented as association estimates per doubling of air pollutant concentrations.

Abbreviations: CI, confidence interval; NO_2, nitrogen dioxide; OR, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter $\leq 2.5\,\mu$m; PM_{10}, particulate matter with an aerodynamic diameter $\leq 10\,\mu$m; Ref., reference.

PRS were not evident in the high-income group but were generally stronger in lower-income groups.

Several previous studies have reported associations between exposure to various ambient air pollutants and rhinitis-related outcomes, which is consistent with the results of the present study. In a French adult cohort, annual concentrations of $PM_{2.5}$, black carbon, and NO_2 at the participants' residential addresses were associated with a higher risk of current rhinitis, defined similarly to PRS used in this study, as well as allergic and nonallergic rhinitis [32]. A previous multicenter study conducted among Italian adults reported that 3-year average concentrations of $PM_{2.5}$ and PM_{10} were associated with an increased risk of rhinitis, as well as both allergic and nonallergic rhinitis [33]. Among adult participants with rhinitis from two European cohorts, annual concentrations of $PM_{2.5}$, PM_{10}, and NO_2 were associated with greater rhinitis severity [7]. In a study of older adults in the United States, 3-year average concentrations of $PM_{2.5}$ were associated with the presence of rhinitis, with stronger associations observed among those with elevated interferon-gamma, interleukin-3, and interleukin-12 levels [34].

For indoor air pollution, relatively few studies have investigated associations with rhinitis-related outcomes, and many have used indoor air quality proxies rather than directly measured pollutant concentrations. For example, a Swedish study reported that adults living in buildings

TABLE 3: Associations between indoor air pollutant concentrations and the presence of rhinitis symptoms in the past 12 months among adults.

Pollutants	Crude		Adjusted	
	OR	95% CI	OR	95% CI
TVOC				
Tertile 1	Ref.		Ref.	
Tertile 2	1.22	0.92, 1.62	1.20	0.90, 1.60
Tertile 3	1.36	1.03, 1.81	1.37	1.02, 1.84
Continuous	1.08	1.01, 1.15	1.08	1.01, 1.16
Benzene				
Tertile 1	Ref.		Ref.	
Tertile 2	0.85	0.55, 1.32	0.86	0.54, 1.36
Tertile 3	1.04	0.68, 1.58	1.05	0.68, 1.62
Continuous	1.07	0.95, 1.20	1.07	0.95, 1.21
Toluene				
Tertile 1	Ref.		Ref.	
Tertile 2	1.20	0.91, 1.59	1.21	0.91, 1.60
Tertile 3	1.41	1.04, 1.92	1.44	1.04, 1.99
Continuous	1.10	1.01, 1.18	1.10	1.02, 1.20
Ethylbenzene				
Tertile 1	Ref.		Ref.	
Tertile 2	1.34	0.998, 1.80	1.34	0.99, 1.82
Tertile 3	1.22	0.91, 1.62	1.24	0.92, 1.68
Continuous	1.07	0.98, 1.18	1.08	0.98, 1.19
Xylene				
Tertile 1	Ref.		Ref.	
Tertile 2	1.07	0.79, 1.46	1.08	0.79, 1.48
Tertile 3	1.01	0.74, 1.37	1.03	0.74, 1.43
Continuous	0.997	0.93, 1.07	1.004	0.93, 1.08
Styrene				
Tertile 1	Ref.		Ref.	
Tertile 2	1.15	0.64, 1.72	1.15	0.75, 1.78
Tertile 3	1.06	0.71, 1.59	1.05	0.70, 1.58
Continuous	0.99	0.90, 1.10	0.98	0.89, 1.09
Formaldehyde				
Tertile 1	Ref.		Ref.	
Tertile 2	1.11	0.84, 1.47	1.13	0.85, 1.50
Tertile 3	1.30	0.99, 1.72	1.30	0.98, 1.71
Continuous	1.10	0.99, 1.23	1.10	0.99, 1.23
$PM_{2.5}$				
Tertile 1	Ref.		Ref.	
Tertile 2	0.99	0.77, 1.27	1.02	0.79, 1.31
Tertile 3	1.06	0.83, 1.35	1.10	0.86, 1.41
Continuous	1.08	0.97, 1.20	1.11	0.99, 1.24
CO_2				
Tertile 1	Ref.		Ref.	
Tertile 2	1.24	0.93, 1.64	1.22	0.91, 1.63
Tertile 3	1.30	0.995, 1.69	1.30	0.99, 1.71
Continuous	1.16	0.95, 1.41	1.15	0.94, 1.41

Note: Odds ratios and 95% confidence intervals were estimated using logistic regression models incorporating appropriate strata, cluster, and weight variables. The models were adjusted for age, sex, income level, educational level, marital or cohabitation status, and tobacco smoking. The results for continuous exposures are presented as association estimates per doubling of air pollutant concentrations.

Abbreviations: CI, confidence interval; CO_2, carbon dioxide; ORs, odds ratio; $PM_{2.5}$, particulate matter with an aerodynamic diameter $\leq 2.5\,\mu$m; Ref., reference; TVOC, total volatile organic compound.

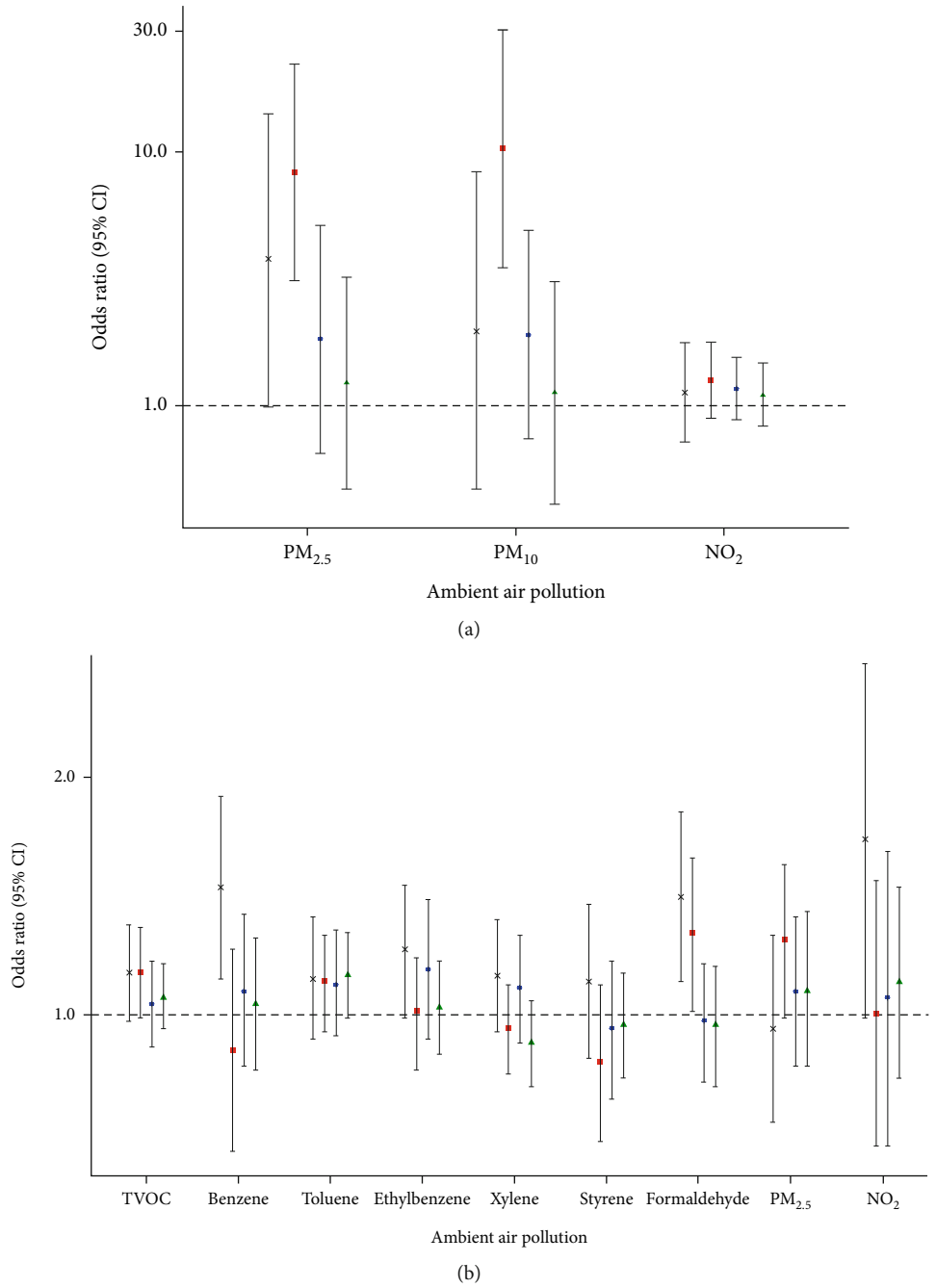

FIGURE 1: Associations between (a) ambient and (b) indoor air pollutant concentrations and the presence of rhinitis symptoms in the past 12 months, stratified by income levels. Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; PM_{10}, particulate matter with an aerodynamic diameter ≤ 10 μm; NO_2, nitrogen dioxide; TVOC, total volatile organic compound; CO_2, carbon dioxide. Odds ratios and 95% confidence intervals were estimated using logistic regression models incorporating appropriate strata, cluster, and weight variables. The models were adjusted for age, sex, income level, educational level, marital or cohabitation status, tobacco smoking. The results are presented as association estimates per doubling of air pollutant concentrations. The X mark indicates income level Q1 (low), the red rectangle indicates Q2, the blue circle indicates Q3, and the green triangle indicates Q4 (high).

constructed between 1976 and 1985, in cities with higher population density, and in rented apartments had an increased risk of current rhinitis [35]. In a study conducted in Poland, the use of solid-fuel stoves, bottled-gas stoves, and central heating systems was associated with an increased risk of rhinitis among adults [12]. A French study that directly measured 20 volatile organic compounds (VOCs) in participants' homes reported that a global VOC score—representing the number of VOCs with concentrations above the third quartile—was associated with a higher risk of rhinitis among adults, which partly aligns with the results of the present study [36].

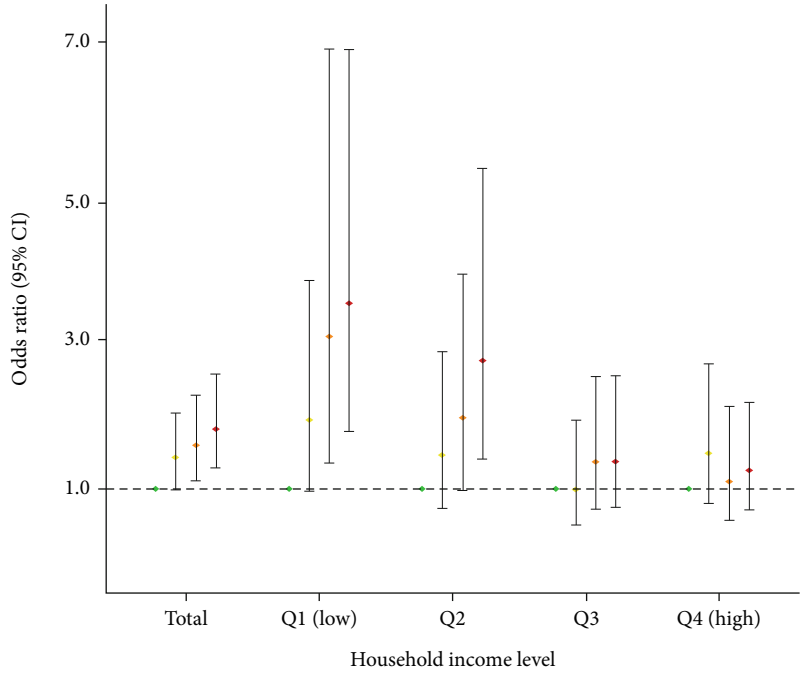

FIGURE 2: Associations of coexposure to ambient $PM_{2.5}$ and indoor TVOC with the presence of rhinitis symptoms in the past 12 months, stratified by income level. Abbreviations: CI, confidence interval; $PM_{2.5}$, particulate matter with an aerodynamic diameter ≤ 2.5 μm; TVOC, total volatile organic compound. Odds ratios and 95% confidence intervals were estimated using logistic regression models incorporating appropriate strata, cluster, and weight variables. The models were adjusted for age, sex, income level, educational level, marital or cohabitation status, and tobacco smoking. The analyses were conducted using a composite exposure variable of $PM_{2.5}$ and TVOC, categorized based on their median values (*low–low*, *low–high*, *high–low*, and *high–high*), with the *low–low* category used as a reference. Colored points represent the four exposure groups: green (*low–low*), yellow (*low–high*), orange (*high–low*), and red (*high–high*).

To our knowledge, only a limited number of studies have examined indoor and outdoor air pollution simultaneously in relation to rhinitis, and these have primarily focused on early-life exposures using proxy measures of indoor pollution (37, 38). These previous studies have shown that indoor sources, such as renovation- or new furniture–related VOCs and dampness-related allergens, together with traffic-related outdoor pollution, contribute to childhood allergic rhinitis, particularly during prebirth and early postnatal windows (37, 38). Although differences in age range, outcome definition, and exposure timing limit direct comparability, the collective evidence supports the view that coexposure to ambient and indoor air pollution may have additive effects on rhinitis risk.

Several studies have investigated the associations between coexposure to ambient and indoor air pollution and health outcomes other than rhinitis. In a study conducted among individuals aged 15–50 years in Western China, ambient ozone and indoor air pollution—defined as the primary use of solid fuels such as charcoal, coal, coke, wood, crop residues, or dung for cooking or heating—were associated with an increased risk of chronic obstructive pulmonary disease, and simultaneous high exposure to both ambient and indoor air pollution significantly strengthened these associations [37]. In another study conducted in China, ambient NO_2 levels and home environmental factors—defined as the presence of mold or damp stains in the homes—were individually and synergistically associated with a higher risk of preterm birth [38]. Although we did not find statistical evidence of interaction between ambient $PM_{2.5}$ and indoor TVOC in relation to PRS in this study, the risk was substantially increased when the participants were simultaneously exposed to both pollutants. This pattern suggests a cumulative exposure burden even in the absence of a statistically significant multiplicative interaction. The interaction test evaluates departures from multiplicativity and may not fully capture additive or cumulative effects. Moreover, the mismatch in exposure assessment windows (1-year $PM_{2.5}$ vs. single-time TVOC) and limited exposure variability likely attenuated the interaction estimate. The observed gradient across the composite exposure groups and the quantile g-computation findings support the interpretation that concurrent exposure may increase the overall inflammatory burden. As this is one of the first studies to address this potentially important issue and given that people are widely exposed to these common pollutants, further investigation is warranted—particularly regarding the interactive effects of ambient and indoor air pollution.

A recent review reported that US communities in poverty face higher levels of ambient and indoor air pollution and an increased risk of morbidity from asthma and other allergic diseases [39]. In another study conducted among Canadian children, the associations between traffic pollution parameters and respiratory symptoms were stronger in the lower-income groups than in the highest-income group [40]. A Chinese study demonstrated that low-income

families tend to live in substandard housing with inferior building materials and crowded conditions, which leads to elevated indoor air pollutant levels [41]. Although, to our knowledge, no study has investigated income-related heterogeneity in the associations between ambient and indoor air pollution and rhinitis-related outcomes, as observed in this study, the results of these studies help explain our findings, suggesting increased vulnerability to air pollution in relation to various health outcomes in lower-income groups. Vulnerability and coping capacity likely differ by income level, with low-income individuals having higher baseline risk factors (e.g., unfavorable lifestyle behaviors) and less access to preventive measures, thereby amplifying the health impact of a given pollution exposure. In contrast, high-income groups may be better able to mitigate exposure (e.g., by using air purifiers or residing in areas with cleaner ambient air), and their better access to medical care could help manage or mask rhinitis symptoms, resulting in a null association in that group.

Exposure to $PM_{2.5}$ and VOCs can induce rhinitis symptoms through overlapping pathways of oxidative injury, epithelial barrier disruption, inflammation, and neurogenic stimulation, which are relevant to both allergic and nonallergic rhinitis [42–44]. Specifically, inhalation of these pollutants generates excess reactive oxygen species in the respiratory tract, damages the nasal epithelial cells, and compromises epithelial barrier integrity, thereby facilitating the penetration of irritants and allergens. Activation of sensory neurons in the nasal mucosa by these irritants and allergens triggers the release of neuropeptides, resulting in reflex nasal symptoms such as vasodilation, plasma leakage, glandular secretion, and sneezing. Additionally, local inflammation contributes to nasal congestion and mucus hypersecretion [44–46].

There are several limitations to be noted in this study. First, ambient air pollutant concentrations were estimated based on the participants' residential addresses, without accounting for commuting to work or school. This approach results in exposure misclassification, which we assume to be nondifferential with respect to PRS and therefore likely biases the results toward the null hypothesis. Second, indoor air pollutant concentrations were measured only once over a relatively short period of time, which may not fully reflect real-life behavioral and temporal variations throughout the day or across multiple days. We also postulate that this exposure misclassification was nondifferential with respect to PRS. Third, although air pollutants such as VOCs can originate from both ambient and indoor sources, we could not account for these complex exposure scenarios due to data limitations, which may have led to exposure misclassification. Fourth, the outcome was defined based on self-reporting in response to the question, rather than a physician's diagnosis or objective clinical assessment, which may have led to misclassification of the outcome (e.g., participants may have had a cold but misattributed the symptoms to rhinitis), although the question used in this study has been widely used and accepted in numerous epidemiological studies [20–22]. Furthermore, although allergic and nonallergic rhinitis are distinct disease entities with different risk factors, pathophysiological mechanisms, and prognoses, classification between the two was not possible due to data limitations. Fifth, this study employed a cross-sectional study design, and the results should be interpreted cautiously, as they do not imply a causal relationship. In particular, reverse causality cannot be ruled out. Individuals with PRS may spend more time indoors and adjust ventilation differently, which could elevate indoor TVOC concentrations and alter the relative contribution of indoor versus ambient exposures. To better address this issue, longitudinal studies with repeated indoor and outdoor exposure measurements, combined with personal time–activity data, are needed to clarify temporal ordering and strengthen causal inference.

However, this study also has important strengths. First, this study is one of the few to examine the associations between coexposure to ambient and indoor air pollution and PRS, which is important from both scientific and public health perspectives. Second, this is the first study to examine income-related heterogeneity in the associations between ambient and indoor air pollution and PRS. Recognizing that air pollution–related health risks are modulated by social determinants such as income is crucial for designing equitable public health strategies, underscoring the need for environmental justice–oriented approaches. Third, this is one of the few studies that directly measure concentrations of indoor air pollutants, instead of using indoor air quality proxies, and link them to upper respiratory outcomes such as rhinitis, which increases the accuracy of exposure assessment.

5. Conclusion

Exposure to individual ambient and indoor air pollutants was associated with a higher risk of PRS in the general adult population. Compared to the low $PM_{2.5}$ and low TVOC group, the *low–high*, *high–low*, and *high–high* groups showed higher odds of PRS, with a gradually increasing trend. The associations of individual ambient and indoor air pollutants, as well as their coexposure, with PRS were more pronounced among individuals with lower income than among those with higher income. Incorporating both ambient and indoor air pollution data lends scientific novelty to our study and offers insights for public health strategies that reflect real-world exposure. The observed income-based disparities in the association between air pollution and PRS further underscore the need to prioritize vulnerable populations to promote health equity. Policies such as targeted subsidies for indoor air purifiers and enhanced ventilation standards in low-income housing may help reduce disparities in the burden of rhinitis. However, given the cross-sectional design and single-time indoor pollution measurement, longitudinal studies with repeated exposure assessments are warranted to validate these findings before informing policy.

Data Availability Statement

The data that support the findings of this study are available from the corresponding author upon reasonable request.

Ethics Statement

This study utilized data from KNHANES, which was approved by the Institutional Review Board (IRB) of the KDCA. All KNHANES participants provided written informed consent. The KNHANES data are publicly available, and all participants' information was anonymized to ensure confidentiality. Given the use of deidentified and publicly accessible data, this study was exempt from the IRB by the Yonsei University Health System (IRB No. 2025-1333-0001).

Conflicts of Interest

The authors declare no conflicts of interest.

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Supporting Information

Additional supporting information can be found online in the Supporting Information section. *(Supporting Information)* Table S1. Distribution of ambient and indoor air pollutants by presence of rhinitis symptoms in the past 12 months and household income quartiles. Table S2. Interaction between ambient $PM_{2.5}$ and indoor TVOC exposure on the presence of rhinitis symptoms in the past 12 months among adults. Table S3. Associations of ambient and indoor air pollution with symptoms of rhinitis in the past 12 months, stratified by income levels. Table S4. Association of combined effect of ambient and indoor air pollution exposure with symptoms of rhinitis and differences by household income levels. Table S5. Associations of ambient $PM_{2.5}$ and indoor TVOC with PRS and their weight contributions in quantile g-computation models and stratified by household income levels.

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