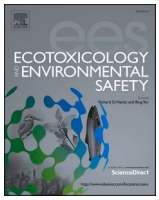
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Air pollution and risk of autoimmune thyroid diseases in children and adolescents: Findings from a nationwide cohort study in Korea

Jung Eun Choi [a,c,1], Jongmin Oh [b,c,d,1], Do Kyeong Song [e], Ji Hyen Lee [a,c], Kyung Hee Kim [a], Soontae Kim [f], Hae Soon Kim [a,c,*], Eunhee Ha [b,c,g,**]

[a] Department of Pediatrics, Ewha Womans University College of Medicine, Seoul, Republic of Korea
[b] Department of Occupational and Environmental Medicine, College of Medicine, Ewha Womans University, Seoul, Republic of Korea
[c] Institute of Ewha-SCL for Environmental Health (IESEH), College of Medicine, Ewha Womans University, Seoul, Republic of Korea
[d] Department of Human Systems Medicine, College of Medicine, Seoul National University, Seoul, Republic of Korea
[e] Department of Internal Medicine, Ewha Womans University College of Medicine, Seoul, Republic of Korea
[f] Department of Environmental and Safety Engineering, Ajou University, Suwon, Republic of Korea
[g] Graduate Program in System Health Science and Engineering, Ewha Womans University, Ewha Medical Research Institute, College of Medicine, Seoul, Republic of Korea

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ABSTRACT

Epidemiological evidence on the association between long-term air pollution exposure and the incidence of autoimmune thyroid diseases (AITD) in children and adolescents remains limited. This study aimed to examine the relationship between long-term exposure to air pollution and the onset of AITD in children and adolescents in South Korea. This nationwide cohort study included 1,400,433 children aged 6 years at baseline (2008–2010) from the National Health Information Database, with follow-up until 19 years of age. The onset of AITD was defined based on the 10th revision of the International Classification of Diseases (Hashimoto's thyroiditis: E063; Graves' disease: E050). Individuals with missing data on age, residence, income level, or underlying conditions such as congenital hypothyroidism were excluded. Exposure concentrations of particulate matter (PM_{10} and $PM_{2.5}$), nitrogen dioxide (NO_2), sulfur dioxide (SO_2), and ozone (O_3) were categorized into quartiles. Hazard ratios (HRs) with 95 % confidence intervals (CIs) were estimated using Cox proportional hazards models, comparing the lowest quartile with higher quartiles. Among the 1,400,433 children analyzed (boys: 729,367; girls: 671,066), 2344 cases (0.17 %) of Hashimoto's thyroiditis and 3424 cases (0.24 %) of Graves' disease were identified during follow-up. The total person-years for both diseases were approximately 19.6 million. Among girls, annual mean $PM_{2.5}$ exposure (HR_{3rd} quartile: 1.245, 95 % CI: 1.035–1.497; HR_{4th} quartile: 1.286, 95 % CI: 1.052–1.571) was significantly associated with the onset of Graves' disease, as was SO_2 exposure (HR_{2nd} quartile: 1.131, 95 % CI: 1.003–1.277). Among boys, a significant association was observed between higher annual NO_2 exposure (HR_{4th} quartile: 2.153, 95 % CI: 1.214–3.818) and the onset of Hashimoto's thyroiditis. These findings provide evidence that long-term exposure to air pollution, particularly $PM_{2.5}$, NO_2, and SO_2, is associated with the incidence of AITD in children and adolescents.

1. Introduction

1.1. Epidemiology of autoimmune thyroid disease

Autoimmune thyroid disease (AITD) is the most common acquired thyroid disorder in pediatric and adolescent populations, encompassing conditions such as Hashimoto's thyroiditis and Graves' disease. The prevalence of AITD in this age group is approximately 1 %–2 %. The pathogenesis of AITD involves the production of autoantibodies directed against thyroid tissue, along with genetic susceptibility related to immune regulation and environmental factors (Sperling, 2020). Key mechanisms underlying autoimmunity include epigenetic alterations,

* Correspondence to: Department of Pediatrics, Ewha Womans University College of Medicine, 260, Gonghang-daero, Gangseo-gu, Seoul 07804, Republic of Korea.
** Correspondence to: Department of Occupational and Environmental Medicine, Ewha Womans University College of Medicine, 260, Gonghang-daero, Gangseo-gu, Seoul 07804, Republic of Korea.
E-mail addresses: hyesk@ewha.ac.kr (H.S. Kim), eunheeha@ewha.ac.kr (E. Ha).
[1] Jung Eun Choi and Jongmin Oh contributed equally to this study.

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such as DNA methylation, histone post-translational modifications, and microRNA expression (Faustini et al., 2018). Furthermore, interactions between genetic factors and environmental exposures, such as cigarette smoking, alcohol consumption, and air pollution, are known to influence epigenetic states (Madrigano et al., 2011).

1.2. Biological mechanisms underlying autoimmunity in AITD

According to the 2019 Global Burden of Disease report, air pollution has become the fourth leading risk factor for human mortality. Air pollution consists of various gaseous substances, including sulfur dioxide (SO_2), nitrogen dioxide (NO_2), carbon monoxide (CO), and ozone (O_3), as well as particulate matter (PM). Fine particulate matter, defined as particles with diameters $\leq$ 2.5 μm ($PM_{2.5}$) and $\leq$ 10 μm (PM_{10}), is of particular concern because its small size, allows deep penetration into the lungs and subsequent entry into the bloodstream. This process can induce systemic inflammation and adversely affect multiple biological systems. PM also contains dust, soil, organic acids, and metals, which may act similarly to endocrine-disrupting chemicals (EDCs) (Darbre, 2018).

Long-term exposure to PM is known to increase oxidative stress, facilitate epigenetic changes, and stimulate the endocrine system, thereby mimicking the effects of environmental hormones. Experimental and population-based studies have demonstrated that exposure to air pollution can increase both the incidence and severity of autoimmune diseases (Zhao et al., 2019). Additionally, chronic inflammation induced by PM exposure has been suggested to disrupt immune regulation, potentially contributing to the development and progression of immune-related hypothyroidism (Sangkham et al., 2024). PM interacts with airway immune cells to generate excessive reactive oxygen and nitrogen species. These reactive species can modify the structure of self-antigens, producing novel epitopes that bypass immune tolerance and provoke autoreactive T-cell responses. The resulting immune cascade promotes B-cell activation and the production of thyroid-specific autoantibodies. Collectively, these mechanisms provide a biological basis supporting the link between long-term air pollution exposure and the pathogenesis of autoimmune thyroid diseases (Gawda et al., 2017)

1.3. Previous studies

Several studies have suggested a link between long-term exposure to air pollution and alterations in thyroid hormone levels. More recent, research has focused on the effects of short-term exposure to air pollution on thyroid function (Kim et al., 2024; Wang et al., 2024). Some studies indicate that exposure to air pollutants increases the risk of developing certain thyroid disorders, such as hypothyroidism and thyroid cancer (Karzai et al., 2022; Yang et al., 2024b). However, epidemiological evidence regarding the association between air pollution and the onset of AITD in children and adolescents remains limited. A cohort study investigated the association between air pollution and AITD in 174 individuals (Izic et al., 2022). In a more recent nationwide cross-sectional study conducted in China, involving 73,900 adults, long-term exposure to $PM_{2.5}$ was associated with an increased prevalence of thyroid disorders, including autoimmune thyroiditis, antithyroid antibody (TgAb) positivity and subclinical hypothyroidism (Yang et al., 2024a). Other studies have focused on thyroid hormone levels or abnormal thyroid states such as elevated thyroid stimulating hormone (TSH) levels and hypothyroxinemia. In addition, meta-analysis results have shown that exposure to air pollution during the first trimester of pregnancy significantly reduces free T4 levels, with more pronounced effects observed in areas with severe pollution (Liu et al., 2023).

In Korea, most previous studies have examined short-term exposure to air pollutants and changes in thyroid function, particularly among adults or pregnant women, rather than the incidence of AITD in children and adolescents. For example, short-term exposure to $PM_{2.5}$ was has been associated with increased TSH and decreased FT4 concentrations in Korean populations (Kim et al., 2024). However, no nationwide cohort study has evaluated the impact of long-term air pollution exposure on the onset of AITD in the pediatric population. Given the substantial regional variation in pollution sources across Korea, there is a clear need to investigate this association using large-scale, population-based data.

1.4. Aims

This study sought to evaluate the association between long-term exposure to air pollutants (PM_{10}, $PM_{2.5}$, NO_2, SO_2, and O_3) and the onset of AITD in children and adolescents in South Korea using a nationwide administrative cohort from 2008 to 2023. We further assessed whether these associations differed by gender through stratified analyses of Hashimoto's thyroiditis and Graves' disease.

2. Methods

2.1. Study population

This study was conducted using the National Health Information Database (NHID) for the years 2002–2023. In South Korea, enrollment in the National Health Insurance Service (NHIS) is mandatory for all citizens as part of the country's commitment to universal health coverage [NHIS-2025–08–1–007]. The NHID contains medical data for the entire population and is available for research purposes. Continuously updated since 2002, it provides a comprehensive and representative dataset that supports both retrospective and prospective studies (Seong et al., 2016). The NHID includes detailed information on individuals' sociodemographic characteristics, medical procedures, and underlying health conditions (Kim et al., 2022; Seong et al., 2016). From this dataset, we followed approximately 1.46 million children born between 2002 and 2004, starting at age 6 (2008–2010), and continued to follow them until they reached age 19 (2021–2023).

We excluded children with insufficient demographic information (gender, age, and residential area), non-Korean nationals, those diagnosed with autoimmune thyroiditis before age 6, and those with underlying conditions, including leukemia (C91), type 1 diabetes (E10), Addison's disease (E271, E272, and E274), congenital hypothyroidism (CH) without goiter (E031), polyglandular dysfunction (E31), celiac disease (K900), tuberous sclerosis (Q851), and congenital malformations (Q90–Q99), as identified using the International Classification of Diseases, 10th revision (ICD-10) (n = 64,138) (Fig. 1). This study was exempt from review by the Institutional Review Board of the Ewha Womans University Seoul Hospital (IRB No: SEUMC 2024–10–035).

2.2. AITD onset

The onset of the two types of AITD was defined based on ICD-10 codes (Hashimoto's thyroiditis: E063; Graves' disease: E050) and was identified as the first outpatient visit occurring between the ages of 6 and 19 years.

2.3. Exposure assessment

The association between long-term exposure to air pollution and the onset of pediatric AITD was examined in children aged 6 years during the period from 2008 to 2010. To assess exposure, we employed the modeling approach recommended by the U.S. Environmental Protection Agency, using the Community Multiscale Air Quality (CMAQ) model (version 4.7.1). This model was integrated with the Weather Research and Forecasting (WRF) model (version 3.6.1) and the Sparse Matrix Operator Kernel Emissions (SMOKE) model (version 2.7). The CMAQ model provides three-dimensional simulations of major air pollutants, including PM_{10}, $PM_{2.5}$, NO_2, SO_2, and O_3, by modeling processes such as

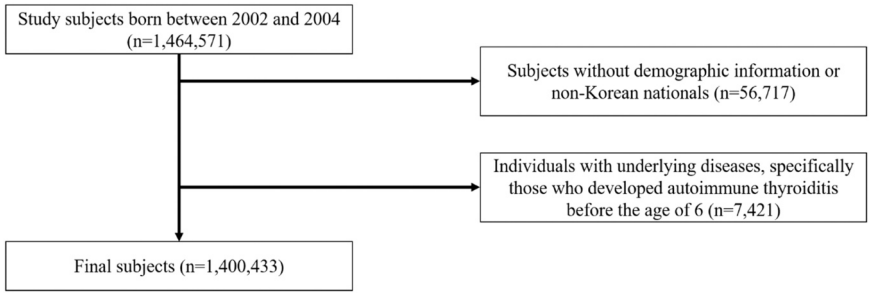


Fig. 1. Flow diagram of included study subjects.

emissions, advection, diffusion, chemical reactions, and deposition on an hourly basis for each grid cell (Bae et al., 2022, 2023; Byun and Schere, 2006). For atmospheric simulations, the model operated within a domain covering Northeast Asia with a 27-km resolution grid and within South Korea with a 9-km horizontal resolution grid. Meteorological input data were generated using the WRF model (version 3.4.1), with initial meteorological fields obtained from the National Center for Environmental Prediction-Final (NCEP-FNL) reanalysis dataset, provided by the National Oceanic and Atmospheric Administration (Bae et al., 2022, 2023; Byun and Schere, 2006). These exposure data have been extensively used in prior studies, with detailed methodology and validation described in the literature (Bae et al., 2020; Han et al., 2020; Kim et al., 2017; Lee et al., 2019; Oh et al., 2020).

The 1-year and 2-year average exposures prior to each baseline year (2008–2010) were calculated, and the exposure data were linked to each individual's residential area at the district level. These exposure measures represent postnatal exposure.

2.4. Statistical analysis

To evaluate the association between long-term exposure to air pollution and AITD, Cox proportional hazards models were applied to estimate hazard ratios (HRs) with 95 % confidence intervals (CIs). Time to disease onset (in months) was calculated from January 1 of 2008, 2009, and 2010, to the date of AITD onset or the end of follow-up (December 31 of 2021, 2022, or 2023), whichever occurred first. Deaths during the follow-up period were censored.

Covariates included insurance subscriber type (unknown, self-employed, or employee), residential area (metropolitan cities or provinces), birth year (2002, 2003, or 2004), average temperature (℃), relative humidity (%), district smoking rates, and the COVID-19 period (pre-pandemic or pandemic/post-pandemic). Metropolitan cities comprised Seoul, Busan, Daegu, Incheon, Gwangju, Daejeon, and Ulsan, while provinces comprised Gyeonggi, Gangwon, North Chungcheong, South Chungcheong, North Jeolla, South Jeolla, North Gyeongsang, South Gyeongsang, and Jeju.

Temperature and relative humidity data were obtained from the Korea Meteorological Administration (https://data.kma.go.kr), and district smoking rates were sourced from the Community Health Survey conducted by the Korea Disease Control and Prevention Agency. Because the NHID is a secondary administrative database created for health insurance purposes, data on children's exposure to second-hand smoke or parental smoking were not available. Therefore, district-level smoking rates were used as a proxy indicator for children's exposure to second-hand smoke. The COVID-19 pandemic period was categorized as pre-pandemic (before 2020) and pandemic/post-pandemic (from 2020 onward).

Four models were constructed: Model 1 (unadjusted), Model 2 (adjusted for insurance subscriber type and residential area), Model 3 (further adjusted for 1-year average temperature, relative humidity, and district smoking rate), and Model 4 (further adjusted for the COVID-19 period). Covariates were selected based on prior studies and the variables available in the dataset (Kim et al., 2024, 2022; Liu et al., 2023; Yang et al., 2024a, 2024b; Zeng et al., 2020). Among Models 1–4, Model 4 yielded the lowest Akaike Information Criterion and was therefore selected as the primary model. Given that sex differences are well documented in pediatric AITD, all analyses were stratified by gender. HRs and 95 % CIs were estimated for PM_{10} and $PM_{2.5}$ (per 1 μg/m^3 increase), as well as for NO_2, SO_2, and O_3 (per 1 ppb increase).

To assess potential nonlinearity, penalized splines were applied to evaluate exposure-response relationships. Additionally, exposure levels were categorized into quartiles, and HRs and 95 % CIs for each quartile were estimated relative to the lowest quartile.

Further analyses included stratified analyses by residential area (metropolitan cities vs. provinces), with metropolitan cities classified as urban areas and provinces as rural areas. We also implemented two-pollutant models after examining variance inflation factors (VIFs) and correlation coefficients among pollutants. Finally, we evaluated the association between exposure to 2-year average air pollution before baseline and the onset of AITD.

3. Results

3.1. Characteristics of study population

A total of 1400,433 children aged 6 years [729,367 boys (52.08 %) and 671,066 girls (47.92 %)] were included as study participants between 2008 and 2010. During the study period, 2344 cases (0.17 %) of Hashimoto's thyroiditis and 3424 cases (0.24 %) of Graves' disease were identified. Among these, 389 cases of Hashimoto's thyroiditis occurred in boys and 1955 cases in occurred girls. In contrast, 819 cases of Graves' disease were observed in boys, whereas 2605 cases were observed in girls. The incidence of Hashimoto's thyroiditis showed a slight increase in children born in 2003 (0.17 %) and 2004 (0.18 %) compared with those born in 2002 (0.16 %). The mean follow-up duration for both Hashimoto's thyroiditis and Graves' disease was 14 years. The mean (standard deviation, SD) survival times were 8.1 (12.6) years for Hashimoto's thyroiditis and 9.6 (11.5) years for Graves' disease. The total person-years for both conditions were approximately 19.6 million.

For the exposure assessment, average exposure data from the 1 year preceding baseline were used. Accordingly, exposure data from 2007 to 2009 are presented in Table 1. The mean (± SD) concentrations of PM_{10}, $PM_{2.5}$, NO_2, SO_2, and O_3 during this period were 55.5 (±7.8) μg/m^3, 28.6 (±4.0) μg/m^3, 25.4 (±9.3) ppb, 5.8 (±1.3) ppb, and 23.0 (±5.1) ppb, respectively. The average smoking rate, mean temperature, and mean relative humidity across the 250 districts were 25.2 (±2.8) %, 12.6 (±1.2) °C, and 65.7 (±4.3) %, respectively.

3.2. Association between long-term exposure to air pollution and AITD

As shown in Table S1, a 1 ppb increase in the 1-year average NO_2 exposure was associated with an increased risk of Hashimoto's thyroiditis in boys (HR: 1.037, 95 % CI: 1.010, 1.065). A similar association was observed for the 2-year average NO_2 exposure and the risk of

Table 1
Demographic characteristics of the study subjects.

Categories	Total	Hashimoto's thyroiditis			Graves' disease		
		Non-event	Event	P-value	Non-event	Event	P-value
Total	1,400,433	1,398,089 (99.83)	2344 (0.17)	< 0.001[a]	1,397,009 (99.76)	3424 (0.24)	< 0.001[a]
Gender							
Boys	729,367	728,978 (99.95)	389 (0.05)	< 0.001[a]	729,367 (99.89)	819 (0.11)	< 0.001[a]
Girls	671,066	669,111 (99.71)	1955 (0.29)		671,066 (99.61)	2605 (0.39)	
Residence							
Metropolitan cities	611,197	610,131 (99.83)	1066 (0.17)	0.076[a]	609,671 (99.75)	1526 (0.25)	0.283[a]
Others	789,236	787,958 (99.84)	1278 (0.16)		787,338 (99.76)	1898 (0.24)	
Insurance subscriber type							
Unknown	43,528	43,444 (99.81)	84 (0.19)	< 0.001[a]	43,391 (99.69)	137 (0.31)	0.048[a]
Local subscriber: low	288,087	287,657 (99.85)	430 (0.15)		287,395 (99.76)	692 (0.24)	
Local subscriber: high	127,035	126,857 (99.86)	178 (0.14)		126,718 (99.75)	317 (0.25)	
Employee subscriber: low	560,716	559,805 (99.84)	911 (0.16)		559,369 (99.76)	1347 (0.24)	
Employee subscriber: high	381,067	380,326 (99.81)	741 (0.19)		380,136 (99.76)	931 (0.24)	
Birth year							
2002	478,840	478,091 (99.84)	749 (0.16)	0.057[a]	477,666 (99.75)	1174 (0.25)	0.992[a]
2003	470,262	469,462 (99.83)	800 (0.17)		469,113 (99.76)	1149 (0.24)	
2004	451,331	450,536 (99.82)	795 (0.18)		450,230 (99.76)	1101 (0.24)	
COVID-19 pandemic period							
Pre-pandemic	3351	1658 (49.48)	1693 (55.52)	< 0.001[a]	1659 (45.79)	1964 (54.21)	< 0.001[a]
Pandemic/post-pandemic	1397,082	1396,431 (99.95)	651 (0.05)		1395,350 (99.90)	1460 (0.10)	
Continuous variables [Mean(±SD)]							
PM_{10} (μg/m^3)	55.49 (7.82)	55.49 (7.82)	55.40 (7.61)	0.567[b]	55.49 (7.82)	55.52 (7.75)	0.821[b]
$PM_{2.5}$ (μg/m^3)	28.57 (4.01)	28.57 (4.01)	28.53 (3.90)	0.620[b]	28.57 (4.01)	28.58 (3.98)	0.883[b]
NO_2 (ppb)	25.41 (9.32)	25.41 (9.32)	25.58 (9.26)	0.375[b]	25.41 (9.32)	25.40 (9.27)	0.950[b]
SO_2 (ppb)	5.76 (1.28)	5.76 (1.28)	5.74 (1.19)	0.416[b]	5.76 (1.28)	5.78 (1.28)	0.361[b]
O_3 (ppb)	22.97 (5.09)	22.97 (5.09)	22.65 (4.85)	0.001[b]	22.97 (5.09)	22.91 (5.05)	0.488[b]
District smoking rate (%)	25.17 (2.79)	25.17 (2.79)	25.03 (2.87)	0.018[b]	25.17 (2.79)	25.22 (2.79)	0.295[b]
Mean temperature (°C)	12.61 (1.23)	12.61 (1.21)	12.60 (1.23)	0.694[b]	12.61 (1.21)	12.59 (1.22)	0.338[b]
Mean relative humidity (%)	65.72 (4.26)	65.72 (4.26)	65.59 (4.30)	0.143[b]	65.72 (4.26)	65.56 (4.45)	0.036[b]

Abbreviations: SD, standard deviation; PM_{10}, particulate matter with a diameter ≤ 10 μm; $PM_{2.5}$, particulate matter with a diameter ≤ 2.5 μm; NO_2, nitrogen dioxides; SO_2, sulfur dioxide; O_3, ozone.
[a] Derived from the chi-squared test; [b] Derived from the two-sample *t*-test.

Hashimoto's thyroiditis (HR: 1.037, 95 % CI: 1.010, 1.066) (Table S2). In girls, no significant associations were observed between air pollutants and either Hashimoto's thyroiditis or Graves' disease in the continuous exposure analysis (Table S2).

Figs. S1–S4 illustrate the associations between 1-year average air pollution exposure and AITD. In boys, higher NO_2 exposure was

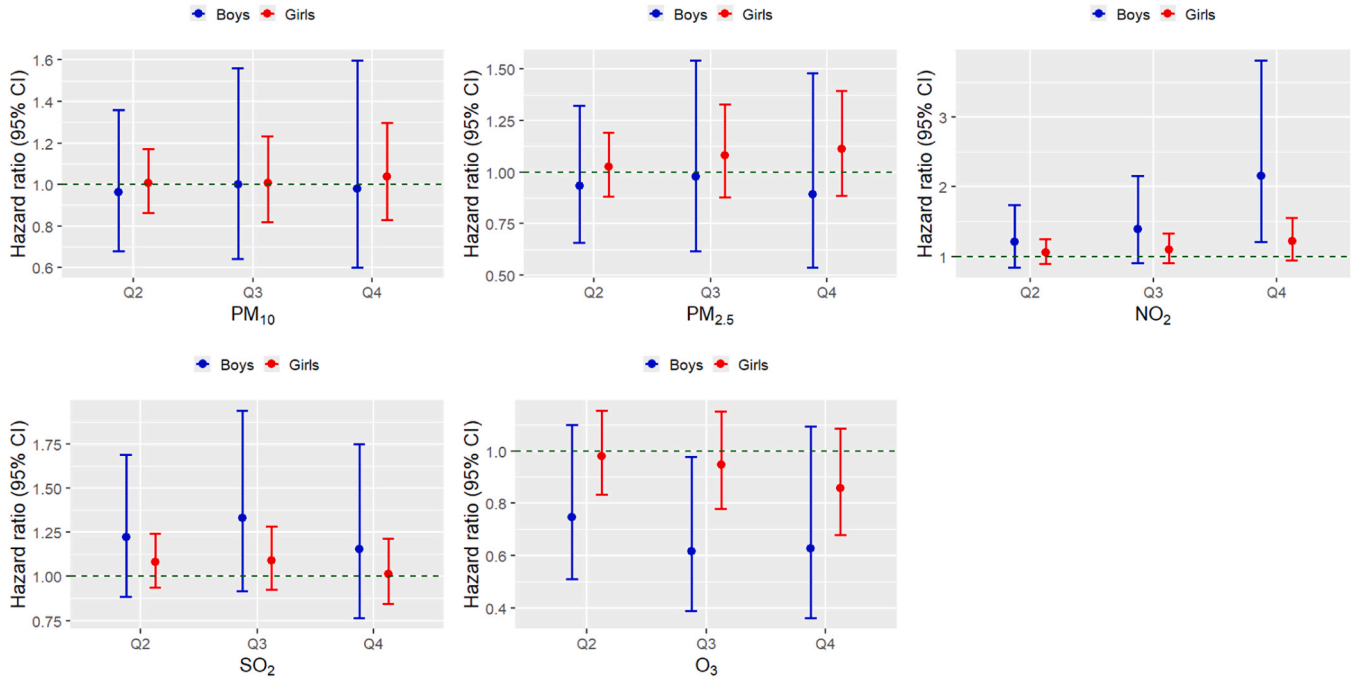


Fig. 2. Association between 1-year average air pollution exposure, categorized into quartiles, and Hashimoto's thyroiditis, expressed as hazard ratios (95 % confidence intervals). All models were adjusted for insurance subscriber type, residential area, 1-year average temperature, relative humidity, district smoking rate, and the COVID-19 period; all models were stratified by birth year. **Abbreviations:** PM_{10}, particulate matter with a diameter ≤ 10 μm; $PM_{2.5}$, particulate matter with a diameter ≤ 2.5 μm; NO_2, nitrogen dioxide; SO_2, sulfur dioxide; O_3, ozone; CI, confidence interval.

associated with an upward trend in HRs for Hashimoto's thyroiditis. In contrast, in girls, similar upward trends were observed for $PM_{2.5}$ and SO_2 exposures in relation to Graves' disease. For the remaining pollutants, neither linear nor nonlinear (inverted U-shaped) exposure–response relationships were identified.

The associations between AITD and air pollution exposure across quartiles are presented in Figs. 2 and 3. Compared with the 1st quartile (reference level) of NO_2 exposure, boys in the 4th quartile had a higher risk of Hashimoto's thyroiditis (HR: 2.153, 95 % CI: 1.214, 3.818) (Fig. 2). In girls, higher $PM_{2.5}$ exposure in the 3rd quartile (HR 1.245, 95 % CI: 1.035, 1.497) and 4th quartile (HR 1.286, 95 % CI: 1.052, 1.571) was associated with an increased incidence of Graves' disease. Additionally, 1-year average SO_2 exposure was significantly associated with the onset of Graves' disease, with an HR of 1.131 in the 2nd quartile (95 % CI: 1.003, 1.277). In boys, the association between 1-year average NO_2 exposure and Hashimoto's thyroiditis was more pronounced in rural areas than in urban areas. In girls, the association between 1-year average $PM_{2.5}$ exposure and Graves' disease was stronger in urban areas than in rural areas (Table S3).

3.3. Two pollutant model

Table S4 presents the Pearson correlation matrix of the 1-year average air pollutant concentrations. High correlations were observed between PM_{10} and $PM_{2.5}$, as well as between NO_2 and O_3. In the two-pollutant models, high variance inflation factors (VIFs $>$ 5) were observed for the combinations of PM_{10} and $PM_{2.5}$, $PM_{2.5}$ and NO_2, NO_2 and SO_2, and NO_2 and O_3. Although no statistically significant associations were identified in the two-pollutant models (Table 2), the overall trends were consistent with those observed in the single-pollutant models. When adjusting for additional pollutants, the effect estimates for $PM_{2.5}$ and PM_{10} tended to attenuate slightly.

4. Discussion

4.1. Main results

We examined the association between 1-year average exposure to air pollution (2008–2010) and autoimmune thyroiditis in children who were six years old at baseline and followed until age 19. A total of 1,400,433 children aged six years between 2008 and 2010 were included in the analysis. During the follow-up period, 2344 cases (0.17 %) of Hashimoto's thyroiditis and 3424 cases (0.24 %) of Graves' disease were diagnosed. Among boys, 1-year average NO_2 exposure in the highest quartile was associated with an increased risk of Hashimoto's thyroiditis (HR_{4th} quartile 2.153, 95 % CI: 1.214–3.818). Among girls, higher 1-year average exposures to $PM_{2.5}$ and SO_2 were associated with an increased incidence of Graves' disease. Specifically, average annual exposure to $PM_{2.5}$ (HR_{3rd} quartile 1.245, 95 % CI: 1.035–1.497; HR_{4th} quartile 1.286, 95 % CI: 1.052–1.571) and SO_2 (HR_{2nd} quartile 1.31, 95 % CI: 1.003–1.277) was associated with an increased risk of Graves' disease. Overall, our findings suggest that exposure to air pollutants, particularly NO_2, $PM_{2.5}$, and SO_2, may increase the risk of AITD in children. Similar trends were observed in the two-pollutant models, which were consistent with the single-pollutant models. In region-stratified analyses, $PM_{2.5}$ exposure was associated with Graves' disease in urban girls, whereas NO_2 exposure was associated with Hashimoto's thyroiditis in rural boys.

4.2. Previous studies

Numerous studies have investigated the relationship between air pollution and thyroid dysfunction. A South Korean study by Kim et al. (2024) reported that short-term exposure to pollutants including $PM_{2.5}$, NO_2, O_3, and CO, disrupted thyroid function, particularly affecting TSH and free thyroxine (FT_4) levels (Kim et al., 2024). These findings suggest that air pollution can subtly alter thyroid hormone levels in the general Korean population, including individuals aged 10 years and older.

Similarly, a large longitudinal study conducted in China involving

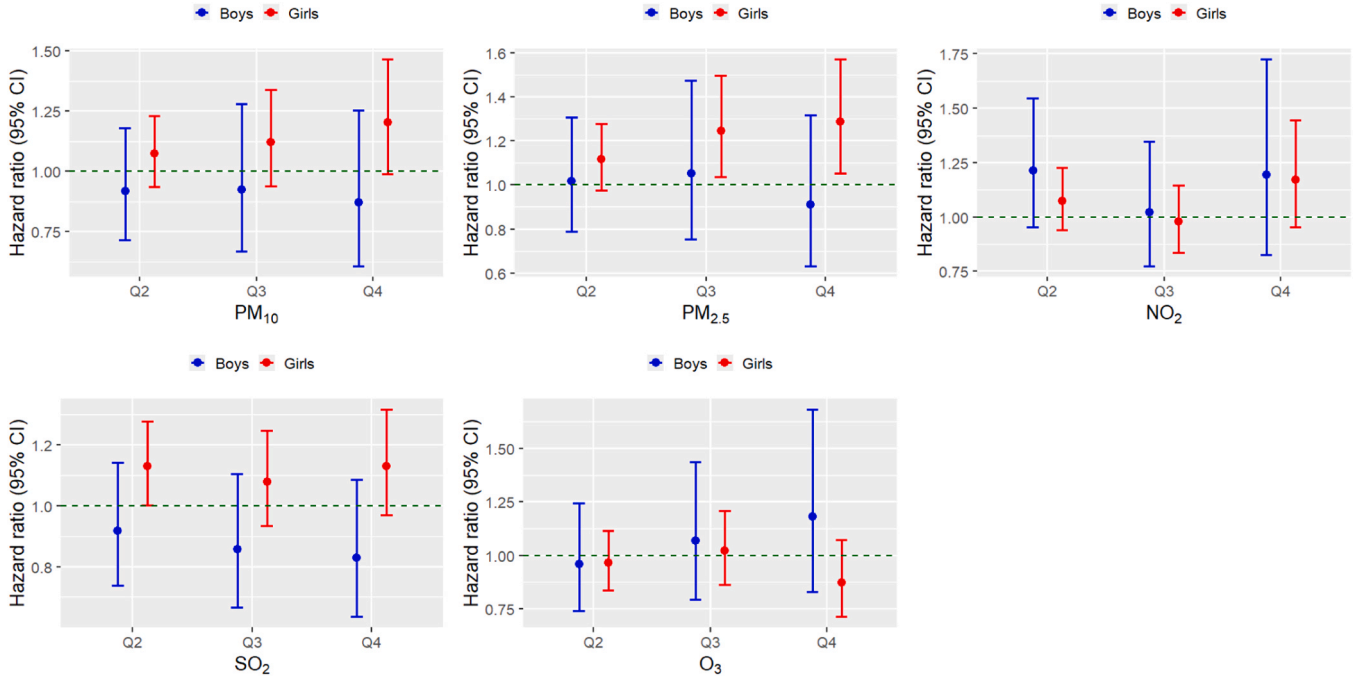


Fig. 3. Association between 1-year average air pollution exposure, categorized into quartiles, and Graves' disease, expressed as hazard ratios (95 % confidence intervals). All models were adjusted for insurance subscriber type, residential area, 1-year average temperature, relative humidity, district smoking rate, and the COVID-19 period; all models were stratified by birth year. **Abbreviations:** PM_{10}, particulate matter with a diameter $\leq$ 10 μm; $PM_{2.5}$, particulate matter with a diameter $\leq$ 2.5 μm; NO_2, nitrogen dioxide; O_3, ozone; CI, confidence interval.

Table 2
Association between 1-year average air pollution exposure and autoimmune thyroid diseases in the two-pollutant model.

Pollutant	Adjustment	Hashimoto's thyroiditis				Graves' disease			
		Boys		Girls		Boys		Girls	
		HR	95 % CI	HR	95 % CI	HR	95 % CI	HR	95 % CI
PM_{10}	Null	0.999	0.974, 1.025	1.004	0.992, 1.016	0.992	0.974, 1.011	1.009	0.999, 1.020
	$PM_{2.5}$	1.154*	0.632, 2.106	1.162*	0.880, 1.534	1.203*	0.775, 1.868	1.125*	0.887, 1.427
	NO_2	0.994*	0.969, 1.020	1.003*	0.991, 1.015	0.992*	0.973, 1.011	1.009*	0.999, 1.019
	SO_2	0.996	0.970, 1.022	1.004	0.992, 1.016	0.994	0.975, 1.012	1.009	0.998, 1.019
	O_3	0.989	0.962, 1.017	1.000	0.987, 1.013	0.993	0.974, 1.012	1.007	0.996, 1.018
$PM_{2.5}$	Null	0.998	0.950, 1.048	1.007	0.950, 1.030	0.984	0.949, 1.020	1.018	0.949, 1.038
	PM_{10}	0.756*	0.235, 2.434	0.753*	0.438, 1.292	0.688*	0.293, 1.614	0.810*	0.510, 1.286
	NO_2	0.989*	0.941, 1.040	1.005*	0.982, 1.029	0.984*	0.948, 1.020	1.017*	0.997, 1.038
	SO_2	0.992	0.943, 1.043	1.007	0.983, 1.030	0.987	0.951, 1.024	1.016	0.996, 1.037
	O_3	0.978	0.928, 1.032	0.999	0.975, 1.024	0.985	0.949, 1.024	1.014	0.993, 1.035
NO_2	Null	1.037	1.010, 1.065	1.009	0.998, 1.020	1.001	0.985, 1.017	1.005	0.995, 1.014
	PM_{10}	1.038*	1.010, 1.066	1.009*	0.998, 1.020	1.001*	0.985, 1.018	1.004*	0.994, 1.013
	$PM_{2.5}$	1.038*	1.010, 1.066	1.009*	0.998, 1.020	1.001*	0.985, 1.018	1.004*	0.994, 1.013
	SO_2	1.041*	1.009, 1.074	1.011*	0.999, 1.024	1.008*	0.989, 1.027	1.003*	0.992, 1.013
	O_3	1.039*	1.001, 1.078	1.003*	0.988, 1.019	1.008*	0.985, 1.032	0.996*	0.983, 1.010
SO_2	Null	1.062	0.944, 1.195	1.005	0.955, 1.058	0.954	0.882, 1.031	1.027	0.983, 1.072
	PM_{10}	1.067	0.945, 1.204	1.002	0.951, 1.055	0.957	0.885, 1.036	1.019	0.975, 1.065
	$PM_{2.5}$	1.067	0.945, 1.205	1.002	0.951, 1.056	0.957	0.885, 1.036	1.019	0.975, 1.065
	NO_2	0.966*	0.837, 1.114	0.979*	0.923, 1.039	0.935*	0.852, 1.025	1.021*	0.971, 1.073
	O_3	1.025	0.902, 1.165	0.985	0.932, 1.041	0.955	0.879, 1.038	1.014	0.967, 1.062
O_3	Null	0.962	0.921, 1.005	0.983	0.965, 1.001	1.007	0.980, 1.034	0.986	0.971, 1.001
	PM_{10}	0.956	0.913, 1.001	0.983	0.964, 1.002	1.004	0.976, 1.032	0.989	0.973, 1.005
	$PM_{2.5}$	0.956	0.913, 1.001	0.983	0.964, 1.001	1.004	0.976, 1.032	0.989	0.973, 1.005
	NO_2	1.004*	0.946, 1.066	0.987*	0.962, 1.011	1.017*	0.978, 1.058	0.982*	0.961, 1.003
	SO_2	0.965	0.921, 1.011	0.981	0.962, 1.000	1.001	0.973, 1.030	0.987	0.972, 1.004

All models were adjusted for insurance subscriber type, residential area, 1-year average temperature, relative humidity, district smoking rate, and COVID-19 period; all models were stratified by birth year.

Abbreviations: HR, hazard ratio; CI, confidence interval; PM_{10}, particulate matter with a diameter $\leq$ 10 μm; $PM_{2.5}$, particulate matter with a diameter $\leq$ 2.5 μm; NO_2, nitrogen dioxide; SO_2, sulfur dioxide; O_3, ozone.

* Note: The calculated variance inflation factor between the two air pollutants exceeded 5, indicating potential multicollinearity. HRs (95 % CI) were estimated per 1 μg/m^3 increase in $PM_{2.5}$ and PM_{10} and per 1 ppb increase in SO_2, NO_2, and O_3.

1.38 million women demonstrated positive correlations between exposure to air pollutants (PM_{10}, $PM_{2.5}$, and NO_2) and elevated TSH levels. The study indicated that recent air pollution exposure can significantly disrupt thyroid hormone homeostasis, particularly among women of reproductive age (20–49 years), who appear to be more susceptible. The high prevalence of thyroid disease and the increasing severity of air pollution in China have further driven research into the association between air pollution and thyroid dysfunction.

Other studies have focused on vulnerable populations, such as pregnant women and children. Two cross-sectional studies conducted in China investigated the association between air pollution and CH in children and identified significant positive relationships between maternal exposure to pollutants such as O_3, NO_2, and $PM_{2.5}$ and an increased risk of CH. No significant associations were observed for SO_2, CO, or PM_{10} (Qi et al., 2021; Shang et al., 2019). However, to date, no studies have examined the impact of postnatal particulate matter exposure on thyroid function during childhood or adolescence. A six-year retrospective study in the Lukavac region of Bosnia and Herzegovina reported elevated concentrations of several pollutants, including $PM_{2.5}$, NO_2, SO_2, CO, and O_3, contributing to an increase in AITD cases. Although the associations were not statistically significant, the study identified a numerical trend suggesting a positive relationship between higher pollutant levels and increased AITD incidence, highlighting the need for further investigation into effects of chronic exposure (Izic et al., 2022). Similarly, a cross-sectional study in China assessed the relationship between long-term $PM_{2.5}$ exposure and thyroid disorders in adults and identified a exposure–response pattern, demonstrating that higher $PM_{2.5}$ levels were associated with an increased prevalence of autoimmune thyroiditis and TgAb positivity (Yang et al., 2024a).

Our study, which focused on individuals aged 6–19 years, examined the incidence of AITD in relation to air pollutants and further clarifying these associations by analyzing differences across stratified concentration quartiles. In contrast to previous research, our study focused on a nationwide cohort of 1.4 million children and adolescents aged 6–19 years and evaluated the incidence of Hashimoto's thyroiditis and Graves' disease in relation to long-term exposure to major ambient air pollutants ($PM_{2.5}$, PM_{10}, NO_2, SO_2, and O_3). Additionally, we characterized exposure–response relationships by comparing risk estimates across stratified concentration quartiles, allowing us to identify pollutant-specific and dose-related patterns that have not been captured in earlier pediatric studies.

4.3. Biological mechanisms

Air pollutants induce systemic oxidative stress and inflammation, both of which are key factors in the development of autoimmune diseases. Zeng et al. demonstrated that thyroid function parameters, including TSH, FT4, FT3, and thyroid peroxidase antibody, are influenced by factors such as sex, age, climate, and seasonal variation (Zeng et al., 2021). Evidence from animal models and extensive epidemiological research indicates that air pollution can disrupt thyroid hormone levels, impair metabolic homeostasis, and contribute to thyroid dysfunction (Tang et al., 2022). Recent studies in female rats have shown that $PM_{2.5}$ exposure lowers circulating thyroid hormone levels by interfering with thyroid hormone biosynthesis and regulation via the hypothalamic-pituitary-thyroid (HPT) axis (Dong et al., 2021). Compared with PM_{10}, $PM_{2.5}$ shows stronger associations with thyroid hormone alterations and abnormal thyroid states, likely due to its smaller particle size, which allows it to penetrate deeper into the respiratory tract, reach the alveoli, and enter the circulatory system more efficiently (Nemmar et al., 2002).

Gaseous pollutants such as NO_2 and SO_2 can further intensify sensitization and inflammatory responses by activating cellular

signaling pathways, potentially disrupting the HPT axis (Xu et al., 2022). NO_2, which is primarily emitted from traffic sources, has been associated with elevated TSH levels, likely due to nitrite accumulation affecting thyroid regulation (Liu et al., 2022). In contrast, SO_2 exposure has been linked to decreased TSH levels, possibly through the formation of bisulfite and sulfite derivatives that interfere with thyroid hormone balance (Wang et al., 2024). In the present study, SO_2 exposure was associated with Graves' disease, which is characterized by hyperthyroidism and suppressed TSH levels.

4.4. Gender differences and ATID incidence

Our results reveal distinct gender-specific associations with long-term air pollution exposure. In boys, NO_2 exposure showed a stronger association with Hashimoto's thyroiditis, whereas in girls, $PM_{2.5}$ and SO_2 exposure were significantly associated with Graves' disease. AITD shows a clear female predominance, with a female-to-male ratio of approximately 2:1 (Calcaterra et al., 2020).

In pediatric populations, gender differences in AITD incidence are less evident before puberty but become more pronounced during, adolescence, reflecting the potential influence of hormonal changes on immune function. The association between human leukocyte antigen genes and autoimmune disorders also shows a female-biased pattern (Ngo et al., 2014; Simmonds and Gough, 2007). These gender differences may reflect hormonal and immunological variations that modulate susceptibility to pollutant-induced thyroid dysfunction. For example, estrogen has been implicated in immune regulation and may exacerbate the effects of air pollution in girls.

The heightened susceptibility observed in females may also be related to genes located on the X chromosome, as suggested by animal studies (Calcaterra et al., 2020). Furthermore, gender differences in exposure profiles, such as time spent outdoors or proximity to pollution sources, warrant further investigation. Taken together, these findings emphasize the importance of incorporating gender-specific analyses in studies examining the effects of air pollution on thyroid diseases.

4.5. Strengths

Our study has several notable strengths. It is among the first studies in Korea to assess the long-term association between air pollution and AITD in children and adolescents aged 6–19 years. We utilized a nationwide, population-representative health insurance database comprising more than 1.4 million children and adolescents and conducted long-term follow-up from 2002 to 2023 to investigate the association between air pollution exposure and AITD incidence beginning at age 6. This approach enabled an objective assessment of AITD incidence using ICD-10 codes, distinguishing our study from previous research that has primarily focused on thyroid hormone alterations.

The large-scale design ensures high representativeness and generalizability. We also identified sex-specific differences in AITD risk associated with long-term air pollution exposure, particularly the association between NO_2 and Hashimoto's thyroiditis in boys and the associations between $PM_{2.5}$ and SO_2 and Graves' disease in girls, findings that have not previously been reported in pediatric populations. Furthermore, we examined regional influences on environmental exposure by comparing urban and rural areas, thereby providing additional insight into spatial variability in AITD risk.

4.6. Limitations

Despite these strengths, several limitations should be acknowledged. First, exposure measurement errors and misclassification in exposure assignment may have led to underestimation of the observed associations. Second, we were unable to obtain direct individual-level information on important confounders, including iodine intake, body mass index, family history of thyroid autoimmunity, exposure to EDCs, indoor air pollution, and second-hand smoke (Dehghani et al., 2023; Wang et al., 2025). Although limited, district-level residence was used as a proxy for smoking rates in the area. Third, although particulate matter levels vary by season, region, emission source, mass concentration, chemical composition, and particle size, we were unable to assess health effects according to differences in PM composition (Heo et al., 2024; Nabizadeh et al., 2018; Yang et al., 2024b). Fourth, thyroid hormone test values (e.g., TSH and FT_4) were not available, and outcomes were defined based on diagnostic codes (ICD-10), which may have introduced outcome misclassification. Finally, our study did not account for changes in exposure over time. Cox proportional hazards analyses indicated that the associations of PM_{10} and $PM_{2.5}$ with Hashimoto's disease and Graves' disease violated the proportional hazards assumption in girls. This suggests that future studies examining the relationship between PM exposure and pediatric thyroiditis in girls should consider time-dependent Cox models. However, we were unable to implement this approach because exposure modeling data were not available beyond 2020 and multi-year moving averages were unavailable to support alternative exposure windows.

5. Conclusion

This study examined the association between 1-year average exposure to air pollution and the onset of AITD, including Hashimoto's thyroiditis and Graves' disease, in children aged 6 years between 2008 and 2010, with follow-up until age 19. Our findings indicate that 1-year average NO_2 exposure was associated with an increased risk of Hashimoto's thyroiditis in boys, whereas in girls, exposure to $PM_{2.5}$ and SO_2 showed positive, non-linear associations with the risk of Graves' disease. These pollutant-specific effects revealed clear sex-related susceptibility patterns, suggesting that distinct pollutants may influence different autoimmune pathways in pediatric populations. Although district-level exposure assignment and unmeasured individual confounders remain limitations, the consistency of the observed associations underscores their potential public health significance. Future studies incorporating multi-year exposure windows, clinical thyroid markers, and time-dependent modeling are warranted to clarify causal pathways and inform strategies to reduce harmful exposures among susceptible children.

CRediT authorship contribution statement

Jung Eun Choi: Writing – review & editing, Writing – original draft, Methodology, Investigation, Conceptualization. **Kyung Hee Kim:** Writing – review & editing, Investigation. **Ji Hyen Lee:** Writing – review & editing, Investigation. **Do Kyeong Song:** Writing – review & editing, Writing – original draft, Investigation. **Jongmin Oh:** Writing – review & editing, Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. **Eunhee Ha:** Writing – review & editing, Writing – original draft, Supervision, Conceptualization. **Hae Soon Kim:** Writing – review & editing, Writing – original draft, Supervision, Conceptualization. **Soontae Kim:** Resources.

Ethical statement

This study was exempt from review by the Institutional Review Board of Ewha Womans University Seoul Hospital (IRB No: SEUMC 2023–09–008).

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Declaration of Competing Interest

The authors declare that they have no known competing financial

interests or personal relationships that could have appeared to influence the work reported in this paper.

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Appendix A. Supporting information

Supplementary data associated with this article can be found in the online version at doi:10.1016/j.ecoenv.2025.119617.

Data availability

The authors do not have permission to share data.

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