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Ambient particulate matter and surrounding greenness in relation to sleep quality among pregnant women: A nationwide cohort study

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ABSTRACT

Background: Particulate air pollution and residential greenness are associated with sleep quality in the general population; however, their influence on maternal sleep quality during pregnancy has not been assessed.

Objective: This cross-sectional study investigated the individual and interactive effects of exposure to particulate matter (PM) air pollution and residential greenness on sleep quality in pregnant women.

Methods: Pregnant women (n = 4933) enrolled in the Korean Children's Environmental Health Study with sleep quality information and residential address were included. Sleep quality was assessed using the Pittsburgh Sleep Quality Index (PSQI). The average concentrations of PM (PM_{2.5} and PM₁₀) during pregnancy were estimated through land use regression, and residential greenness in a 1000 m buffer area around participants' residences was estimated using the Normalized Difference Vegetation Index (NDVI_{1000-m}). Modified Poisson regression models were used to estimate the associations between PM and NDVI and poor sleep quality (PSQI >5) after controlling for a range of covariates. A four-way mediation analysis was conducted to examine the mediating effects of PM.

Results: After adjusting for confounders, each $10 \ \mu g/m^3$ increase in PM_{2.5} and PM₁₀ exposure was associated with a higher risk of poor sleep quality (relative risk [RR]: 1.06; 95% confidence interval [CI]: 1.01, 1.11; and RR: 1.09; 95% CI: 1.06, 1.13, respectively), and each 0.1-unit increase in NDVI_{1000-m} was associated with a lower risk of poor sleep quality (RR: 0.97; 95% CI: 0.95, 0.99). Mediation analysis showed that PM mediated approximately 37%–56% of the association between residential greenness and poor sleep quality.

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Conclusions: This study identified a positive association between residential greenness and sleep quality. Furthermore, these associations are mediated by a reduction in exposure to particulate air pollution and highlight the link between green areas, air pollution control, and human health.

Abbreviations

BMI	body mass index
CDE	controlled direct effect
CES-D:	center for epidemiologic studies-depression
INTREF	reference interaction
IQR	interquartile range
INTMED	mediated interaction
Ko-CHEN	S Korean children's environmental health study
LUR	land-use regression
NDVI	normalized difference vegetation index
NIR	near-infrared radiation
$PM_{2.5}$	particulate matter with an aerodynamic diameter ${\leq}2.5~\mu\text{m}$
PM_{10}	particulate matter with an aerodynamic diameter ${\leq}10~\mu m$
PIE	pure indirect effect
PSG	polysomnography
PSQI	Pittsburgh sleep quality index
RERI	relative excess risk due to interaction
SD	standard deviation

1. Introduction

Poor sleep is a common complaint during pregnancy [1], and approximately 46% of pregnant women experience poor sleep quality, as measured by the Pittsburgh Sleep Quality Index (PSQI) [2]. Moreover, poor sleep quality is associated with adverse outcomes in mothers and fetuses, such as prolonged labor [3], gestational diabetes [4], preterm birth [5], and low birth weight [3]. Furthermore, poor sleep quality can affect memory and the ability to concentrate in pregnant women [6], leading to poor quality of life [7] and mental health problems [8]. In South Korea, the estimated prevalence of poor sleep quality in pregnant women is 72.5% [9], whereas another study reported that 80.7% of pregnant women experienced poor sleep [10]. To provide potential prevention and therapy, modifiable risk factors for poor sleep quality must be identified.

Previous studies have found that poor socioeconomic status, an unhealthy lifestyle, heavy work pressure, and poor health are associated with poor sleep quality [11–13]. Additionally, recent studies have suggested that environmental factors, such as air pollution and greenness, could also be associated with poor sleep quality [14,15]. According to a multicenter cohort study conducted in the United States, an interquartile range (IQR) increase in ambient particulate matter (PM) with aerodynamic diameters of \leq 10 µm (PM₁₀) was significantly associated with decreased sleep efficiency in adults [16]. In addition, a study conducted on rural residents aged 18–79 years in China reported that long-term exposures to PM₁₀ and PM with aerodynamic diameters \leq 2.5 µm (PM_{2.5}) were associated with poor sleep quality, as defined by a global PSQI score of >5 [17]. Similar results were observed in a community-level longitudinal study in South Korea, which found that higher levels of PM₁₀ were associated with shorter sleep duration in adults aged \geq 19 years [18]. Recent reviews have concluded that exposure to air pollution is generally associated with poor sleep quality in a wide range of study populations, including young children and older adults [14,19].

A growing number of reports have suggested that living in greener areas is associated with a lower risk of poor sleep quality [20], but results vary. A study among adults in rural China reported that higher residential greenness (as assessed by the Normalized Difference Vegetation Index [NDVI] and Enhanced Vegetation Index) was associated with better sleep quality [15], whereas no convincing evidence of a link between greenspace exposure and sleep quality has been found among children and adolescents in Australia and Germany [21]. Unlike literature on air pollution-sleep association, overall findings from literature on greenness-sleep are less generalizable due to a small number of studies and exposure assessment, study design and population, and geographic scale differences.

One of the potential mechanisms responsible for greenness-sleep association is that greenness is associated with reductions in exposure to air pollution [22], which is regarded as a risk factor for poor sleep quality [23]. Greenness can interact with exposure to air pollutants [24], and previous studies have shown that substantial air quality improvements can be achieved through green infrastructure [25,26]. However, studies on the interaction between air pollution and greenness and the mediating effects of air pollution on the association between greenness and sleep quality in pregnant women are lacking. Here, we conducted a cross-sectional study to investigate the association between residential environment (residential NDVI and PM) and sleep quality among pregnant women, who are particularly vulnerable to air pollution due to their high oxygen requirements for developing fetuses and decreased oxygen-binding capacity [27]. Previous studies indicated that air pollution, psychological stress, and physical activity influence the relationship between greenness and sleep [22,28,29]. Additionally, we explored the PM-greenness interaction and mediating effect of PM on the association between residential greenness and sleep quality.

2. Methods

2.1. Study population

We used data from the Korean Children's Environmental Health Study (Ko-CHENS), designed to investigate environmental influences on health outcomes. The details of the study design have been reported elsewhere [30]. Briefly, participants were selected from 12 regional centers in South Korea. The distribution of participants' residences is shown in Fig. 1; most (90.6%) of the participants

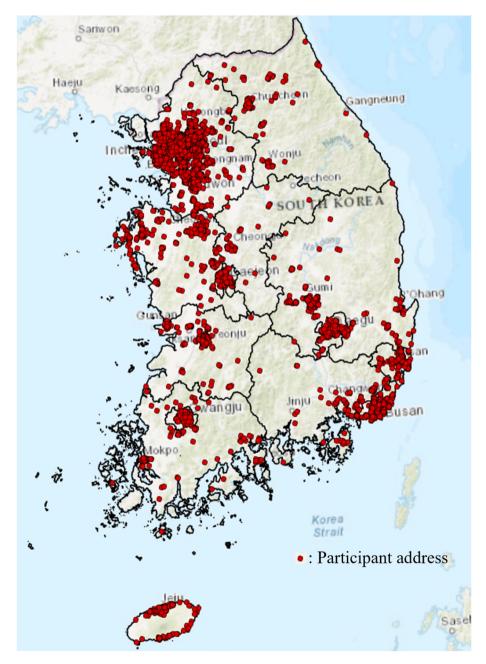


Fig. 1. Location of the study participants in Ko-CHENS.

resided in urban areas. Trained personnel collected data on sociodemographic information, health behaviors, disease history, mental health status, and sleep quality. Initially, 5458 pregnant women were recruited between 2015 and 2019. After excluding women with a lack of exposure (NDVI and PM) information and sleep quality data, data of 4933 women were analyzed (Fig. S1). Characteristics of the excluded participants did not differ significantly from those of the study population, except for parity, smoking, chronic diseases, and sociodemographic variables (education, income, and occupation) (Table S1). All study participants provided written informed consent before enrollment. The Institutional Review Boards (IRB) of the National Institute of Environmental Research (NIER) provided consent for the Ko-CHENS (approval no. NIER-2015-BR-005-01, 15 May 2015), and the present study was approved by the IRB of the Inha University Hospital (approval no. 2021-10-023).

2.2. Outcome assessment

Sleep quality during pregnancy was assessed using the PSQI scale [31], which has been widely used to assess sleep quality and patterns in various patient and research populations, including pregnant women [10,32]. The timing of PSQI administration during pregnancy varied across participants (1–30 weeks of gestation). The PSQI contains 19 items in seven subscales. Each subscale is scored between 0 and 3, and the seven subscales are summed to produce total scores ranging between 0 and 21, with a higher score representing poorer sleep quality. The Korean version of the PSQI has been validated and shown to have high specificity and sensitivity [33]. Previous studies of the Korean version of the PSQI have used a cut-point of 5 to define good (\leq 5) and poor (>5) sleep quality [34, 35], and we also applied this definition. Cronbach's alpha for the Korean version of the PSQI was 0.69 [36]. In this study, the internal consistency of the PSQI was adequate, with a Cronbach's alpha of 0.64.

2.3. PM and residential greenness assessment

A land use regression (LUR) model was used to estimate $PM_{2.5}$ and PM_{10} concentrations during pregnancy using maternal residential addresses at delivery. The standard LUR modeling procedure has been described elsewhere [37]. Briefly, we obtained $PM_{2.5}$ and PM_{10} concentrations from the AirKorea National Air quality Monitoring Station between 2015 and 2020 (472 stations in 2020). Daily 24-h average $PM_{2.5}$ and PM_{10} concentrations were measured at each monitoring station. The LUR models were developed for individual pollutants, and it included several potential predictors, including variables related to land use, industry-related variables, demographic characteristics, traffic-related variables, and amounts discharged to the atmosphere. Details of the data sources are provided in the Supplementary Material (Table S2). The LUR models were applied to each geocoded address where the participants resided to estimate the pollutant concentrations. We included all potential predictor variables as independent variables after assigning the direction of the effect to PM concentration. Model construction started with the variable with the expected regression slope and the highest explained variance by univariate analysis. The remaining variables were only added to this model if they satisfied all of the following criteria: (a) the gain of adjusted R² was no less than 1%; (b) the p-value was <0.1; (c) variables already included in the model retained the same direction of effect; and (d) the variance inflation factor was <3. This procedure was repeated until none of the variables meet the criteria. Cross-validation indicated that the R² values and root mean squared errors for PM_{2.5} and PM₁₀ were 73.4% and 0.39 µg/m³ and 51.6% and 1.7 µg/m³, respectively (Table S3).

To assess long-term exposure for each participant during pregnancy, we determined $PM_{2.5}$ and PM_{10} concentrations for three trimesters of pregnancy. Hourly pollutant concentrations were aggregated to daily levels, and daily concentrations were averaged across trimesters (first trimester: 0–12 weeks, second trimester: 12–27 weeks, and third trimester: 27–40 weeks of gestational age). Gestational age in weeks was estimated using prenatal ultrasonography.

Residential greenness levels of the participants were determined using the NDVI, which has been widely used to estimate the effects of green vegetation on mental health including sleep quality [15,38]. The NDVI values were derived from the Moderate Resolution Imaging Spectroradiometer of the National Aeronautics and Space Administration's Terra satellite images, which were produced at 16-day intervals with a spatial resolution of up to 500 m [39]. Normalized Difference Vegetation Index is defined as the ratio of the difference between near-infrared radiation (NIR) and visible red (RED) and the sum of these two measures, according to the formula: NDVI = (NIR – RED)/(NIR + RED); NDVI values range from -1 to +1. Negative NDVI values close to -1 represent blue space or water and area without vegetation, whereas positive values close to 1 indicate dense green vegetation [40]. Negative NDVI values were coerced as zero in the analysis. Residential greenness was estimated as the mean of the NDVI values in zones at 100 m (NDVI_{100-m}), 200 m (NDVI_{200-m}), 500 m (NDVI_{500-m}), 1000 m (NDVI_{1000-m}), and 2000 m (NDVI_{2000-m}) around the participant residences. In the present study, we used NDVI_{1000-m} values in the main analysis to indicate a broader scale of the neighborhood, which has also been applied in previous studies on mental health and sleep quality [15,41,42].

2.4. Covariates

We used the directed acyclic graph (DAG) to select the potential confounders (Fig. S2) and also included the covariates that only influence the exposure or outcome that were not identified by DAG [15,43]. A range of demographic, behavioral, and socioeconomic covariates was selected, namely, age (<30, 30–34, \geq 35), education (high school, college, and graduate school), occupation during pregnancy (yes vs. no), parity (multiparity vs. primiparity), pre-pregnancy body mass index (BMI) (kg/m²), frequency of physical activity (none, 1–2/week, 3–4/week, 5–6/week, and everyday), smoking history (>5 packs in a lifetime), exposure to secondhand smoke (yes vs. no), household income (<2,000, 2000–3,999, and \geq 4000 USD/month), center for epidemiologic studies-depression (CES-D) scores, chronic diseases (yes vs. no), and season. Age was calculated from delivery and self-reported birth dates, which

were verified using identification cards. We divided pre-pregnancy BMI into four categories based on the World Health Organization Asia-specific standards: underweight (BMI <18.5 kg/m²), normal ($18.5 \le BMI < 23 \text{ kg/m}^2$), overweight ($23 \le BMI < 25 \text{ kg/m}^2$), and obese ($\ge 25 \text{ kg/m}^2$) [44]. Data on gestational age (in weeks) were obtained from medical records at delivery. We assessed prenatal depression using a 10-item CES-D scale [45]: a score of ≥ 12 was used to define depressive symptoms [46]. The internal consistency of the CES-D indicated excellent reliability in our study (Cronbach's alpha = 0.82). Chronic diseases were assessed by considering a history of nine diseases: stroke, arthritis, osteoporosis, malignant tumors, asthma, chronic bronchitis, chronic hepatitis, diabetes, and chronic nephritis. The season in which the PSQI information was collected was used to address seasonal variations in sleep quality during pregnancy. Years were divided into four seasons according to weather patterns in Korea: spring (March to May), summer (June to August), autumn (September to November), and winter (December to February).

2.5. Statistical analysis

Frequency distributions, means, and standard deviations (SD) were used to describe the sample, and the chi-square test was used to compare the population characteristics. Correlations between the exposures were evaluated using Pearson's correlation analysis.

To estimate the relative risk (RR) and 95% confidence interval (CI) of poor sleep quality associated with the residential environment, we used the modified Poisson regression with a robust error variance approach [47]. Separate models were created to examine the independent effects of the residential NDVI, PM, and their interactions. Model 1 was used to determine the main effect of each 0.1-unit increase in NDVI on poor sleep quality, whereas model 2 was constructed to show the main effect of each 10 μ g/m³ increase in PM on poor sleep quality. Model 3 was a two-exposure model in which the NDVI and PM were regressed on poor sleep quality. In Model 4, we added the product term of centered NDVI and PM to assess multiplicative interactions between 0.1-unit increases in NDVI and 10 μ g/m³ increases in PM on poor sleep quality. Additionally, we assessed additive interactions by calculating relative excess risks due to interactions (RERIs) [48]. All the models were adjusted for age, education level, smoking history, exposure to secondhand smoke, gestational age, parity, occupation, family income, and season. Pre-pregnancy BMI, physical activity, chronic diseases, and depression were considered potential mediators of the association between residential environment and health outcomes [15,49]; thus, they were not adjusted for in the main analysis. Considering the mediating role of PM between greenness and mental health [50], we also evaluated whether the greenness-sleep quality association was mediated by PM. The four-way counterfactual approach was used to evaluate the mediation effects [51]. The user-written med4way command in Stata was used to test the mediation and interaction of the total effect of residential greenness on sleep quality [52]. This command generated five models to decompose the total effect of residential greenness on sleep quality: (1) total excess RR (TE: the overall effect of residential greenness on sleep quality); (2) excess RR due to controlled direct effects (CDE: the direct effect of residential greenness on sleep quality that was not explained by PM); (3) excess RR due to reference interaction (INTREF: the effect of residential greenness due to the interaction with PM); (4) excess RR due to mediated interaction (INTMED: the effect of residential greenness due to both mediation and interaction with PM); and (5) excess RR due to pure indirect effects (PIE: the effect of residential greenness that was transferred through PM). The statistical significance of the PIE indicates a potential mediating effect of the pathway variable. A description of this counterfactual concept is provided in the Supplementary Table S4.

The exposure-response relationship between the residential environment and poor sleep quality was investigated using restricted cubic spline models with three knots positioned according to the Harrell's recommended percentiles [53]. Nonlinearity was determined by testing the regression coefficient of the second spline variable equal to zero [54]. Additionally, we plotted exposure-response curves for poor sleep quality using the NDVI and PM quantiles.

For sensitivity analyses, we 1) examined associations between poor sleep quality and PM and NDVI_{1000-m} individually and jointly using different PSQI cut-offs (PSQI >7 and PSQI \geq 8.5) [33,55]; 2) considered sleep quality as a continuous variable rather than a binary measure; 3) performed analysis using PM_{2.5} and PM₁₀ exposures during different time windows (the three trimesters of pregnancy); 4) included NDVI within the 100-, 200-, 500-, and 2000-m buffer in the models to assess the potential effect of buffer size; and 5) used logistic regression to compute odds ratios (ORs) and compare the ORs of main results with RRs computed using modified Poisson regression models. Stratified analyses were conducted to investigate the effect modification by selected maternal characteristics (age, BMI, education, income, smoking, physical activity, depression, chronic diseases, season, and timing of sleep assessment). The significance of effect modifications was evaluated using two-sample z-tests [56].

Stata software (version 17.0) was used to conduct statistical analysis, and ArcGIS version 10.8 was used for mapping and developing the LUR models. Statistical significance was defined as p < 0.05.

3. Results

Table 1 summarizes the characteristics of the study population. Mean participant age was 32.9 years (SD = 3.8), and 1467 (29.7%) of the participants were \geq 35 years old. Most of the participants had a college or higher degree (88.5%), were primiparous (70.3%), and had not smoked during pregnancy (88.7%). The mean PSQI score of all the participants was 6.3 (SD = 2.2), and 58.4% of them had poor sleep quality (PSQI >5). The mean NDVI_{1000-m} was 0.4 (SD = 0.1), and PM_{2.5} and PM₁₀ were 26.0 (SD = 5.4) and 47.2 (SD = 7.5) µg/m³, respectively (Table 2). A negative correlation was observed between NDVI_{1000-m} and PM (Table 2). Women with poor sleep quality were more likely to be primiparous, have a lower income (<2000 USD/month), be obese (BMI \geq 25 kg/m²), smoke, be depressed (CES-D \geq 12), and more likely to be exposed to secondhand smoke than women with good sleep quality (PSQI \leq 5).

Table 3 shows the association between residential environment and poor sleep quality. According to the adjusted model, $NDVI_{1000}$.

Table 1

PSQI >5

m exposure was significantly associated with poor sleep quality, with an RR of 0.97 (95% CI: 0.95, 0.99) per 0.1-unit increase in NDVI_{1000-m} (model 1). PM_{2.5} and PM₁₀ were significantly associated with poor sleep quality (RR: 1.06; 95% CI: 1.01, 1.11; and RR: 1.09; 95% CI: 1.06, 1.13, respectively, per 10 μ g/m³ increase in PM_{2.5} or PM₁₀) (model 2). The results of the single-exposure model (models 1 and 2) also persisted in the two-exposure model (model 3). Multiplicative interactions between PM_{2.5} and NDVI_{1000-m} were observed for poor sleep quality (RR: 0.94; 95% CI: 0.89, 0.98; p = 0.011 (model 4). The RERI estimates indicated no additive interactions between PM and NDVI_{1000-m} (Table S6). The results of the crude models were consistent with those of the adjusted models (Table 3). Exposure-response curves of the associations between sleep quality and PM and NDVI_{1000-m} did not suggest a nonlinear relationship (PM_{2.5}: p-overall = 0.035, p-nonlinear = 0.935; PM₁₀: p-overall = <0.001, p-nonlinear = 0.791; NDVI_{1000-m}: p-overall = 0.015, p-nonlinear = 0.052; Fig. S3).

We compared the greenness-sleep quality association in different quartiles of the PM; the association was significant only among

Characteristics	Mean (SD)	n (%)
Age (years)	32.9 (3.8)	
Age group		
< 30		1055 (21.4)
30–34		2411 (48.9)
≥ 35		1467 (29.7)
Education		
High school		561 (11.4)
College		3753 (76.1)
Graduate school		619 (12.6)
Parity		
Primiparity		3477 (70.5)
Multiparity		1456 (29.5)
Gestational age (weeks)	38.8 (1.8)	
Family income (US\$/month)		
<2000		314 (6.4)
2000–3999		2447 (49.6)
≥ 4000		2172 (44.0)
Occupation		
No		1666 (33.8)
Yes		3267 (66.2)
Physical activity		
None		4226 (85.7)
1–2/week		253 (5.1)
3–4/week		313 (6.4)
5–6/week		100 (2.0)
Everyday		41 (0.8)
Body mass index (kg/m ²)		
< 18.5		564 (11.4)
18.5–22.9		2936 (59.5)
23–24.9		663 (13.4)
≥ 25		770 (15.6)
Smoking status		
No		4304 (87.2)
Yes		629 (12.8)
SHS exposure		(100 (00 5)
No		4130 (83.7)
Yes		803 (16.3)
Chronic diseases		1506 (00.0)
No		4596 (93.2)
Yes		337 (6.8)
Season		10(7 (05 7)
Spring		1267 (25.7)
Summer		1126 (22.8)
Autumn Winter		1227 (24.9)
CES-D scores	4.6 (4.3)	1313 (26.6)
	4.0 (4.3)	
CES-D group CES-D < 12		45,068 (92.
$CES-D \ge 12$ PSQI scores	6.3 (2.2)	365 (7.4)
PSQI scores	0.3 (2.2)	
PSQI ≤5		2053 (41.6)
1 0 Q1 _0		2000 (41.0)

Abbreviations: Standard deviation: SD; secondhand smoke: SHS; Center for Epidemiologic Studies-Depression Scale: CES-D; Pittsburgh Sleep Quality Index: PSQI.

2880 (58.4)

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Table 2

Summary of residential greenness and particulate matter levels.

	Mean	SD	SD Min	Quantiles		Max	Pearson's correlation coefficients			
				Q25	Q50	Q75		NDVI _{1000-m}	PM _{2.5}	PM_{10}
NDVI _{1000-m}	0.4	0.1	0.0	0.3	0.3	0.4	0.8	1.0		
PM _{2.5}	26.0	5.4	11.8	22.3	25.4	28.7	47.3	-0.2	1.0	
PM ₁₀	47.2	7.5	24.5	41.8	45.7	51.5	91.6	-0.2	0.7	1.0

Abbreviations: Standard deviation: SD; normalized difference vegetation index: NDVI; particulate matter with aerodynamic diameters of \leq 2.5 µm: PM_{2.5}; particulate matter with aerodynamic diameters of \leq 10 µm: PM₁₀.

participants exposed to $PM_{2.5}$ in the fourth quantile (Table 4). We did not find a monotonic trend in the association between greenness and sleep quality within the different PM quartiles. Additionally, we compared the association between PM and poor sleep quality within the different quartiles of greenness (Table S7). We found that greater greenness attenuated the risk of poor sleep quality; however, we did not observe a monotonic trend in this association. This finding was also apparent in the spline analysis (Fig. 2). The spline model results showed that in the NDVI_{1000-m} quartiles, the risk of PM_{2.5}-related poor sleep quality increased in the middle quartiles (Q2 and Q3), whereas in the upper quartile (Q4), greater greenness substantially flattened the risk (Fig. 2A). Additionally, for the PM_{2.5} quartiles, RRs of NDVI_{1000-m}-related poor sleep quality were substantially lower in the upper quartile (Q4), whereas in the lower quartile (Q1), higher PM_{2.5} substantially increased the risk (Fig. 2B).

The four-way mediation analysis showed that the pure indirect effects of $PM_{2.5}$ and PM_{10} on the $NDVI_{1000-m}$ -sleep quality relationship for the adjusted model were significant, and the mediated proportions were 36.9% and 55.7%, respectively (Table 5). The mediated interactions (effects due to mediation and interaction) were significant for $PM_{2.5}$, but not for PM_{10} . Furthermore, we found no evidence of mediation and interaction by physical activity, pre-pregnancy BMI, chronic diseases, or depression on the association between residential environment and sleep quality (Table S8).

Sensitivity analyses showed that when the association between residential environment and poor sleep quality was examined by changing the PSQI cut-off scores; significant associations for NDVI_{1000-m}, PM_{2.5}, and PM₁₀ were observed at a cut-off of >7, and the association for PM₁₀ was significant at a higher cut-off score (\geq 8.5) (Table S9). For continuous outcomes, the relationships between residential environment and PSQI scores were similar to the main results (Table S10). The results did not change substantially when examining the association using trimester-specific PM concentrations, and significant interaction terms were found between NDVI_{1000-m} and PM_{2.5} exposure during the second and third trimesters (Table S11). Additionally, we found significant positive associations of PM_{2.5} during the first and third trimesters and PM₁₀ in all three trimesters with poor sleep quality. Furthermore, sensitivity analysis using NDVI_{2000-m} showed independent and interactive effects of PM_{2.5} and residential greenness on poor sleep quality was similar to

Table 3

Associations between sleep quality and residential greenness and particulate matter levels.

	Model 1	Model 2	Model 3	Model 4	
	RR (95% CI)	RR (95% CI)	RR (95% CI)	RR (95% CI)	p value
NDVI _{1000-m}					
Crude model	0.97 (0.95, 0.99)				
Adjusted model	0.97 (0.95, 0.99)				
PM _{2.5}					
Crude model		1.05 (1.00, 1.09)			
Adjusted model		1.06 (1.01, 1.11)			
PM10					
Crude model		1.07 (1.04, 1.11)			
Adjusted model		1.09 (1.06, 1.13)			
PM _{2.5} and NDVI _{1000-m}					
NDVI _{1000-m}			0.98 (0.95, 1.00)	0.97 (0.95, 0.99)	
PM _{2.5}			1.05 (1.01, 1.10)	1.05 (1.01, 1.10)	
$NDVI_{1000-m} \times PM_{2.5}$				0.94 (0.89, 0.98)	0.011
PM ₁₀ and NDVI _{1000-m}					
NDVI _{1000-m}			0.99 (0.96, 1.01)	0.99 (0.96, 1.01)	
PM10			1.09 (1.05, 1.12)	1.09 (1.05, 1.12)	
$NDVI_{1000-m} \times PM_{10}$				0.99 (0.96, 1.02)	0.568

Abbreviations: Relative risk: RR; normalized difference vegetation index: NDVI; particulate matter with aerodynamic diameters \leq 2.5 µm: PM_{2.5}; particulate matter with aerodynamic diameters \leq 10 µm: PM₁₀.

Note: Model 1 showed the main effect of 0.1-unit increases in NDVI on poor sleep quality; model 2 showed the main effect of $10 \ \mu g/m^3$ increases in PM_{2.5} or PM₁₀ on poor sleep quality; model 3 showed the main effects of 0.1-unit increases in NDVI and $10 \ \mu g/m^3$ increases in PM_{2.5} or PM₁₀ on poor sleep quality; and model 4 showed the interaction between 0.1-unit increases in NDVI and $10 \ \mu g/m^3$ increases in PM_{2.5} or PM₁₀ on poor sleep quality. The adjusted models and Models 3 and 4 were controlled for maternal age, history of smoking, exposure to secondhand smoke, maternal education, gestational age, parity, occupation, family income, and season..

p values are for the interaction between 0.1-unit increases in NDVI and 10 μ g/m³ increases in PM_{2.5} or PM₁₀.. Bold values denote statistical significance at p < 0.05.

Table 4

Associations between residential greenness (0.1-unit increases in $NDVI_{1000-m}$) and poor sleep quality, modified by particulate matter.

	RR (95% CI)
PM _{2.5} (μg/m ³)	
1st quartile (range 11.8–22.3)	1.04 (0.99, 1.09)
2nd quartile (range 22.3–25.4)	0.97 (0.92, 1.01)
3rd quartile (range 25.4–28.7)	0.98 (0.94, 1.03)
4th quartile (range 28.7–47.3)	0.93 (0.88, 0.98)
PM ₁₀ (μg/m ³)	
1st quartile (range 24.5–41.8)	0.99 (0.95, 1.04)
2nd quartile (range 41.8–45.7)	0.97 (0.92, 1.02)
3rd quartile (range 45.7–51.5)	1.01 (0.96, 1.06)
4th quartile (range 51.5–91.6)	0.98 (0.94, 1.03)

Abbreviations: Relative risk: RR; normalized difference vegetation index: NDVI; particulate matter with aerodynamic diameters of \leq 2.5 µm: PM_{2.5}; particulate matter with aerodynamic diameters of \leq 10 µm: PM₁₀.

Note: Adjusted for maternal age, history of smoking, exposure to secondhand smoke, maternal education, gestational age, parity, occupation, family income, and season.

Bold values denote statistical significance at p < 0.05.

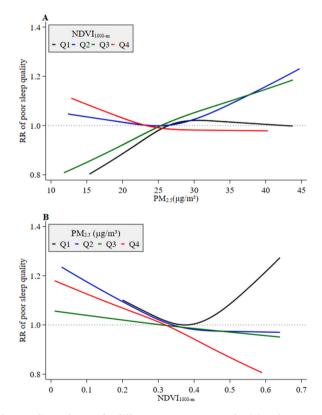


Fig. 2. Association between poor sleep quality and $PM_{2.5}$ for different $NDVI_{1000-m}$ quantiles (A) and $NDVI_{1000-m}$ for different $PM_{2.5}$ quantiles (B). **Abbreviations:** Relative risk: RR; normalized difference vegetation Index: NDVI; particulate matter with aerodynamic diameters of $\leq 2.5 \mu$ m: $PM_{2.5}$; particulate matter with aerodynamic diameters of $\leq 10 \mu$ m: PM_{10} . **Note:** All the models were adjusted for maternal age, history of smoking, exposure to secondhand smoke, maternal education, gestational age, parity, occupation, family income, and season.

the main results (Table S12). Interactive effects of $PM_{2.5}$ and $NDVI_{500-m}$ on poor sleep quality were also found; however, these results were inconsistent with the $NDVI_{100-m}$ and $NDVI_{200-m}$ analyses. Furthermore, sensitivity analysis using logistic regression conformed to the Poisson regression results (Table S13). Finally, we did not observe statistically significant subgroup differences according to maternal characteristics, including age, education, income, smoking, physical activity, depression, chronic diseases, and season, for the association between residential environment and poor sleep quality (p for subgroup difference >0.05) (Fig. 3, S4, and S5). Additionally, the associations between $PM_{2.5}$ and PM_{10} with sleep quality were significant in both subgroups stratified by the timing of

Table 5

Four-way mediation analysis of residential greenness (NDVI1000-m), particulate matter, and poor sleep quality.

Mediator/	TE	CDE	INTREF	INTMED	PIE	Mediation	
moderator	β (95% CI)	β (95% CI)	β (95% CI)	β (95% CI)		proportion	
PM _{2.5}	-0.090 (-0.179, -0.001)	-0.077 (-0.147, -0.006)	-0.008 (-0.072, 0.055)	0.028 (0.007, 0.049)	-0.033 (-0.053, -0.013)	36.9%	
PM10	-0.074 (-0.145, -0.002)	-0.042 (-0.115, 0.032)	0.001 (-0.019, 0.021)	0.008 (-0.010, 0.026)	-0.041 (-0.059, -0.023)	55.7%	

Abbreviations: Total effect: TE; controlled direct effect: CDE; reference interaction: INTREF; mediated interaction: INTMED; pure indirect effect: PIE. **Note:** Logistic regression models were used with poor sleep quality as the outcome and residential greenness as the main exposure. Linear regression models for each mediator/moderator (PM_{2.5} and PM₁₀) were used, and the mediator was allowed to interact with the main exposure. All models were adjusted for maternal age, smoking history, exposure to secondhand smoke, maternal education, gestational age, parity, family income, occupation, and season.

Bold values denote statistical significance at p < 0.05.

PSQI administration during pregnancy (weeks of gestation), and we found no statistically significant differences (Table S14).

4. Discussion

In this nationwide cohort of pregnant women, higher $PM_{2.5}$ and PM_{10} levels were significantly associated with poor sleep quality (PSQI >5), whereas higher residential greenness was associated with better sleep quality. Furthermore, this association remained consistent across the subgroups, with no evidence of effect modification by participant characteristics. This study showed that PM mediated 37–56% of the effects on the association between residential greenness and sleep quality. Additionally, ambient $PM_{2.5}$ potentially interacts with greenness and mediates the relationship between greenness and sleep quality.

Growing evidence suggests that air pollution is associated with sleep quality [14,16,17]. A multicenter cohort study of 3030 US

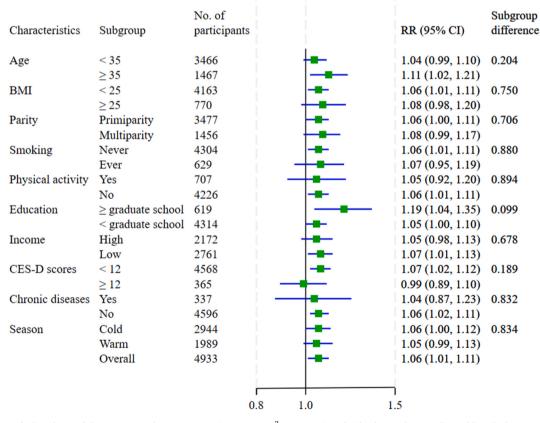


Fig. 3. Stratified analyses of the association between $PM_{2.5}$ (per 10 μ g/m³ increment) and risk of poor sleep quality. **Abbreviations:** Relative risk: RR; body mass index: BMI; Center for Epidemiologic Studies-Depression: CES-D. **Note:** The RRs were adjusted for maternal age, history of smoking, exposure to secondhand smoke, maternal education, gestational age, parity, occupation, family income, and season. For each stratified analysis, the stratification variable was omitted from the model.

adults aged >39 years (mean age, 63 years), with objectively measured sleep quality assessed by polysomnography (PSG), reported that an IQR increase in short-term PM_{10} in the summer was positively associated with sleep disorders [16]. Another study conducted in the US consisting of 1974 adults (mean age, 68 years), revealed an association between $PM_{2.5}$ and sleep disorder, as assessed by PSG [57]. A Mexican study of 397 mother-child pairs reported that $PM_{2.5}$ exposure during pregnancy was associated with a reduction in sleep efficiency (assessed objectively using actigraphy) in children [58]. We retrieved only three studies that investigated the association between PM and sleep quality using PSQI in adults [17,59,60]. A Chinese study of 27,417 adults (aged 18–79 years) found that long-term exposure to $PM_{2.5}$ and PM_{10} (per IQR increase) was associated with poor sleep quality (PSQI >5) (OR: 1.15; 95% CI: 1.03, 1.29; and OR: 1.11; 95% CI: 1.02, 1.21, respectively) [17]. Another Chinese study reported that $PM_{2.5}$ and PM_{10} were associated with reduced sleep duration [60] and prolonged sleep latency [59]. It is difficult to compare the effect sizes of the current evidence regarding the associations between PM and sleep quality with those of previous studies due to differences in study populations, air pollutants, and sleep measure. Nevertheless, consistent with other studies [17], we observed a significant association between PM and poor sleep quality (PSOI score >5) in pregnant women.

However, there is insufficient evidence linking residential greenness to sleep quality. Only five relevant studies have examined the association between objective greenspace measurements and self-reported sleep quality [15,23,61-63], and only one of these studies used the PSQI scale to measure poor sleep quality [15]. For example, a recent prospective study of 21,878 participants (aged \geq 45 years) in China showed that each 0.1 increase in NDVI was significantly associated with a 9% (OR: 0.91; 95% CI: 0.86, 0.96) decrease in the odds of poor sleep quality, which was defined as the combination of short sleep duration (<7 h per night) and a feeling of unrest for >3 nights per week [23]. Another study based on 27,654 rural residents in the Henan Province reported that higher residential greenness was associated with better sleep quality, as assessed using the PSQI [15]. Additionally, a systematic review concluded that greenspace exposure is positively associated with sleep quality [20].

In the present study, we did not find monotonic trends in the association between PM and sleep quality at different levels of greenness exposure. Our restricted cubic spline models showed a pattern of decreasing risks of poor sleep quality associated with exposure to elevated PM_{2.5} among women living in the greenest quantile. This indicates that a certain level of residential greenness is required to reduce air pollution. We also noted that the distribution of PM_{2.5} was fairly similar across NDVI_{1000-m} quintiles (e.g., ranging between 15.4 and 45.8 μ g/m³ in the lowest quintile and between 12.5 and 44.7 μ g/m³ in the highest). These results indicate that greenness may modify the relationship between exposure to PM_{2.5} and sleep quality. In our interaction model, the RR of a 0.1-unit increase in NDVI_{1000-m} and 10 μ g/m³ increase in PM_{2.5} was 0.94 (p = 0.011), which suggests that decreasing air pollution and increasing residential greenness have a synergistic effect on sleep quality. Previous studies have provided evidence on the effects of greenness and air pollution interactions on other health outcomes [24,64], supporting our findings to some extent. A Chinese Longitudinal Healthy Longevity Survey found that elderly individuals (\geq 65 years) living in areas with higher greenness levels were associated with lower risks of air pollution-related mortality [24]. One plausible explanation for the interaction between exposure to PM_{2.5} and residential greenness is that greenspace filters out PM aerodynamically [65], thereby reducing the oxidative stress caused by PM_{2.5} and residential greenness is the adverse effects of exposure to PM_{2.5} on sleep.

Several potential mechanisms have been proposed to explain the association between greenness and sleep quality, including reducing the harmful effects of environmental exposure, providing spaces for outdoor and social activities, and reducing psychological stress [22,28,29]. We found that greenness affects sleep quality in pregnant women through the mediating effect of particulate air pollution. A Spanish cross-sectional study in adults (n = 958) reported that PM_{2.5} mediated 14% of the association between surrounding greenness and poor mental health [67], and a prospective cohort study in China (n = 31,176) reported that the mediation proportions of PM_{2.5} and PM₁₀ on the association between greenness (NDVI_{250-m}) and poor mental health were approximately 53% and 39%, respectively [50]. However, no studies have reported the mediating effect of air pollutants on the association between greenness and sleep quality. Greenness may attenuate the adverse effects of air pollution exposure and remove PM [68], and residential greenness may improve mental health [29] and help to improve sleep quality. Our findings suggest that particulate air pollution may explain the association between greenness and sleep quality. A previous study suggested that several pathways, including physical activity and mental stress, explained the association between greenness and health [22]. However, the present study did not support the mediating effects of physical activity and depression on the association between greenness and sleep quality. Further studies are needed to investigate the potential mechanisms, preferably through the inclusion of multiple mediators, of greenness-sleep quality association among pregnant women.

The strengths of our study are that it examined associations at a national level with a relatively large sample size, and included multiple sensitivity analyses. We included detailed demographic and psychosocial behavioral information, which enabled us to conduct a stratified analysis of these risk factors. To our knowledge, this is the first study to investigate exposure to air pollution, greenness, and sleep quality among pregnant women.

This study has several limitations. First, exposure to particulate air pollution during pregnancy was based on maternal residential addresses at delivery, as predicted by the LUR model, and this model did not consider exposure at the work address or during transportation, which may have led to exposure misclassification. However, studies have shown that maternal mobility during pregnancy is generally restricted to short distances, and exposure levels remain homogenous within a community [69,70]. Hence, we believe that this shortcoming had a minor impact on our exposure estimates and is unlikely to have changed our findings. Second, although several studies have used the NDVI to assess individual residential greenness [15,71], it does not provide information on specific types of vegetation. Third, sleep quality was assessed only once during pregnancy using the PSQI scale, and we did not confirm it by clinical examinations, such as PSG, which is the gold standard for diagnosing sleep disorders [72]. Fourth, although our analyses controlled several confounders, residual confounding remains possible. For example, we did not consider the use of a partial-body heating system while sleeping in a cold indoor environment, which may be related to changes in sleep quality over time [73]. The

effects of PM on sleep quality may also be confounded by ambient temperature, relative humidity, artificial light at night, and noise [74,75], which were not measured in this study and may be estimated in future studies. Fifth, we did not consider multiple pollutants that may have had confounding or modifying effects on our analysis. Finally, longitudinal studies are required to validate the causal relationships between air pollution and greenness and sleep quality in pregnant women.

5. Conclusions

Our study provides evidence that increases in $PM_{2.5}$ and PM_{10} concentrations are related to the risk of poor sleep quality (PSQI >5) in pregnant women. Furthermore, a higher level of residential greenness is associated with better sleep quality, and greenness and $PM_{2.5}$ interact with sleep quality. Additionally, PM mediated approximately 37–56% of the association between greenness and poor sleep quality. These findings suggest that greenness may be significant in reducing PM concentrations, and is subsequently linked to a lower risk of poor sleep quality. Our findings provide evidence for urban planners, as improvements in residential greenness could be a protective measure in areas with high air pollution. The protection and restoration of greenspace may decrease PM concentrations and help improve maternal and fetal health. Future studies are required to investigate the role of mental stress on the relationship between the residential environment and sleep quality, and to explore the underlying biological mechanisms.

Data availability statement

Data will be made available on request.

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The funders of the study had no role in the study design, data collection, data analysis, data interpretation, or writing of the report.

CRediT authorship contribution statement

Dirga Kumar Lamichhane: Writing – original draft, Visualization, Methodology, Formal analysis, Data curation, Conceptualization. Eunhee Ha: Supervision, Investigation, Conceptualization. Yun-Chul Hong: Supervision, Investigation, Conceptualization. Dong-Wook Lee: Writing – review & editing. Myung-Sook Park: Writing – review & editing. Sanghwan Song: Writing – review & editing. Suejin Kim: Writing – review & editing. Woo Jin Kim: Writing – review & editing. Jisuk Bae: Writing – review & editing. Hwan-Cheol Kim: Writing – review & editing, Supervision, Methodology, Investigation, Conceptualization.

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. Supplementary data

Supplementary data to this article can be found online at https://doi.org/10.1016/j.heliyon.2024.e26742.

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