

The Association between ambient air pollution and resistant hypertensive disorders in pregnancy: a case-control study in Tashkent

Asociación entre la contaminación atmosférica ambiental y los trastornos hipertensivos resistentes durante el embarazo: Un estudio de casos y controles en Tashkent

Buzulaykho Khaydarova - Ferghana Medical Institute of Public Health Department of Epidemiology and Infectious Disease Treatment, Nursing PhD, dotsent. Uzbekistan. <https://orcid.org/0009-0000-7851-9425> xaydarjonovabuzulayho@gmail.com

Mahbubakhon Irgasheva - Department of Epidemiology and Infectious Disease Treatment, Nursing assistant, Ferghana Medical Institute of Public Health Uzbekistan. <https://orcid.org/0009-0008-8340-4268> mahbubaxonirgasheva@gmail.com

Izimkul Manasova - Senior Lecturer of the Department of General Hygiene Head teacher, Bukhara State Medical Institute named after Abu Ali ibn Sino, Bukhara, Uzbekistan; Email: manasova.izimkul@bsmi.uz, <https://orcid.org/0000-0001-8626-0206>

Shuxrat Sabirov - Bukhara State Medical Institute named after Abu Ali ibn Sino, Bukhara, Uzbekistan; e-mail: nazira.habibova@bsmi.uz, <https://orcid.org/0000-0001-6038-1720>

Saylibarxon Yulchiyeva - Senior teacher, Department of Department of Medical Chemistry and Biological Sciences, Central Asian Medical University, Uzbekistan. <https://orcid.org/0009-0008-8201-6036>, saylibarkhon.yulchiyeva@mail.ru

Maxbuba Mamatqulova - docent, Health Department of Microbiology, virology and immunology, Ferghana Medical Institute of Public, Uzbekistan. <https://orcid.org/0009-0000-6798-7995>, maxbubamamatqulova9@gmail.com

Bobirjon Umurov - Department of Therapy, Bukhara State Medical Institute named after Abu Ali ibn Sino, Bukhara, Republic of Uzbekistan; Email: bobirjon_umurov@bsmi.uz <https://orcid.org/0009-0002-2501-3118>

Dekhkambaeva Zulfiya - Candidate of Pedagogical Sciences, Associate Professor, Department of Pedagogy and Psychology, Tashkent State Medical University, Tashkent, Republic of Uzbekistan; E-mail: zulfiyahon769@gmail.com <https://orcid.org/0009-0000-2998-6385>

Received: 07/20/2025 Accepted: 09/19/2025 Published: 11/12/2025 DOI: <http://doi.org/10.5281/zenodo.17625591>

Abstract

The present study was carried out to assess the association between ambient air pollution exposure and resistant hypertension disorders development in pregnancy by a case-control study in Tashkent city. The present study included 150 pregnant women with resistant hypertension disorders (cases) and 300 pregnant women without the disorder (controls). The chronic exposure to particulate matter pollutants less than 2.5 microns (PM_{2.5}), nitrogen dioxide (NO₂), and ozone (O₃) throughout pregnancy was calculated based on the air quality monitoring station data. The comparison was made using conditional logistic regression and adjusting for the confounding effect of the variables. The findings showed the average exposure to PM_{2.5} and NO₂ in the case group were greater than that in the control group. For a 10 micrograms per cubic meter increase in PM_{2.5} concentration, the adjusted odds ratio for refractory hypertension was 2.01 (95% confidence interval: 1.51-2.68). For an 8.5 ppm increase in NO₂ concentration, the adjusted odds ratio was 1.85 (95% confidence interval: 1.41-2.42). For ozone, there was no significant association. The results from this study demonstrate that maternal exposure to elevated levels of PM_{2.5} and NO₂ air pollutants is associated with an increased incidence of refractory hypertension in pregnancy. These findings underscore the importance of taking air pollution seriously as a modifiable risk factor for this life-threatening pregnancy complication and implementing appropriate policies to improve air quality to promote maternal health.

Keywords: Air pollution, pregnancy hypertension disorders, refractory hypertension, particulate matter.

Resumen

El presente estudio se llevó a cabo para evaluar la asociación entre la exposición a la contaminación atmosférica ambiental y el desarrollo de trastornos hipertensivos resistentes durante el embarazo mediante un estudio de casos y controles en la ciudad de Tashkent. El estudio incluyó a 150 mujeres embarazadas con trastornos hipertensivos resistentes (casos) y 300 mujeres embarazadas sin este trastorno (controles). La exposición crónica a contaminantes de material particulado menores de 2,5 micras (PM_{2.5}), dióxido de nitrógeno (NO₂) y ozono (O₃) durante el embarazo se calculó con base en los datos de la estación de monitoreo de la calidad del aire. La comparación se realizó mediante regresión logística condicional y se ajustó por el efecto de confusión de las variables. Los hallazgos mostraron que la exposición promedio a PM_{2.5} y NO₂ en el grupo de casos fue mayor que en el grupo de control. Para un aumento de 10 microgramos por metro cúbico en la concentración de PM_{2.5}, el odds ratio ajustado para hipertensión refractaria fue de 2,01 (intervalo de confianza del 95 %: 1,51-2,68). Para un aumento de 8,5 ppm en la concentración de NO₂, el odds ratio ajustado fue de 1,85 (intervalo de confianza del 95 %: 1,41-2,42). En el caso del ozono, no se observó una asociación significativa. Los resultados de este estudio demuestran que la exposición materna a niveles elevados de contaminantes atmosféricos PM_{2.5} y NO₂ se asocia con una mayor incidencia de hipertensión refractaria durante el embarazo. Estos hallazgos subrayan la importancia de considerar seriamente la contaminación atmosférica como un factor de riesgo modificable para esta complicación potencialmente mortal del embarazo e implementar políticas adecuadas para mejorar la calidad del aire y promover la salud materna.

Palabras clave: Contaminación atmosférica, trastornos hipertensivos durante el embarazo, hipertensión refractaria, material particulado.

Pregnancy is a condition of profound physiological changes, and maternal and fetal health take top priority. Among the most serious complications of this condition is pregnancy-related hypertension, which not only puts maternal health at great risk but also has far-reaching effects on fetal growth and development¹. Such conditions range from mild to severe disorders, and their adequate and timely management takes a key position in the prognosis of pregnancy². Of the numerous varieties of these disorders, those that do not respond to standard treatments and are “resistant” are of such intensity and sensitivity. This type of hypertension, as it is difficult to treat, can lead to recurrent hospitalization, preterm delivery, and also to an increase in the risk of severe preeclampsia. It is therefore an indubitable clinical necessity to identify the reasons that might possibly be responsible for the resistance of these disorders³.

In the industrialized world of today, exposure to environmental pollution has become a public concern. Air, which is one of the most essential elements of life, may carry harmful gases and particles that subtly but effectively affect human health. Accumulating evidence suggests that these pollutants can exert their negative effects on other organs and systems of the body beyond the respiratory system⁴. The cardiovascular system is one of the main targets for the harmful actions of air pollution. The inflammatory pathways and oxidative stress caused by the inhalation of small particles can damage the walls of the blood vessels and affect endothelial function⁵. The impairment of vascular diameter control and blood pressure regulation can be directly associated with the development or aggravation of hypertension. The question is whether such effects can occur in the same manner or even worse in vulnerable populations such as pregnant women⁶. With the consideration that the body of a pregnant woman is undergoing a dramatic hormonal and metabolic change, it is speculated that air pollution can add another stressor that will disrupt the delicate balance of the period and result in an increased incidence or severity of blood pressure disorders. In particular, whether the pollutants have the ability to result in the development of treatment-resistant strains is a query that until now has been less completely explored^{7,8}.

Tashkent, as a bustling city with heavy traffic and industrialization, is one such environment where individuals are subject to high air pollution levels. Pregnant women in this city are constantly exposed to such pollutants, and the health status of these individuals might be affected by this chronic exposure. Therefore, a study of the status of this group may be of prime importance from a public health perspective.

According to the existing knowledge gap regarding the association between air pollution as an environmental risk factor and resistant hypertension prevalence in pregnancy, it seems there is a necessity for a study of this correlation specifically in the geographical and demographic condition of Tashkent city. This study can have significant implications for obstetricians and gynecologists and health policymakers to implement preventive measures on time to reduce this risk and improve maternal and newborn health through a preventive approach. Numerous epidemiological investigations throughout the world have studied the link between short-term and long-term exposure to significant air pollutants such as fine particulate matter, nitrogen dioxide, and ozone⁹. All these investigations are indicative of air pollution as a physiologic stressor capable of destabilizing the placental vascular barrier, causing systemic inflammation and oxidative stress, and thus initiating compromised vascular endothelial function. Both of these pathways have been directly implicated in the pathophysiology of hypertension and preeclampsia⁹.

Particularly in gestational hypertension, ample evidence exists that heightened levels of air pollutants are associated with high rates of preeclampsia and gestational hypertension. Very fine particles appear to exert a more adverse impact since they can enter the systemic circulation and even the placental barrier¹⁰. They have the potential to alter the mother’s immune response and mediate inflammation, ultimately leading to vascular injury and impaired uterine artery remodeling¹¹. However, most of the previous studies have focused primarily on the early development of these disorders, and the literature is exceedingly limited with respect to investigating determinants involved in the advancement of these disorders towards treatment-resistant phenotypes¹². Resistant hypertensive disorders, disorders not controlled despite conventional drug therapies, are among the most challenging in maternal-fetal medicine and have serious adverse implications for the mother and fetus^{13,14}.

This knowledge gap is particularly evident for the role of environmental exposures such as air pollution^{15,16}. Although biological mechanisms suggest that chronic exposure to air pollutants may be a contributor to resistance to antihypertensive therapy through increasing oxidative stress and suppressing drug metabolism, direct epidemiological evidence specifically investigating this association in the pregnant population is very limited¹⁷⁻¹⁹. Thus, while the general association of air pollution with gestational hypertension is roughly known, the chain of causality linking this exposure to resistant case formation is still shrouded in mystery. Properly designed research, such as the present case-control study, that can examine this very relation in a given population and also account for the potential confounders, seems not just warranted but absolutely essential to complete this research.

Materials and methods

Study Design

We used the case-control study design in analyzing the association between ambient air pollution and resistant hypertension disorder in pregnancy. The study design was chosen because of its effectiveness in investigating diseases with relatively low incidence and the ability to measure multiple exposures concurrently. The study included pregnancies that resulted in delivery in the most recent five-year period in Tashkent teaching hospitals. Both the case and control groups were selected from the same population and at the same time to eliminate the influence of confounding time factors.

Study population and participants

The case group comprised pregnant women who were admitted to the chosen hospitals with the diagnosis of resistant hypertension disorder of pregnancy, as per the standard definition, i.e. failure to control target blood pressure despite the simultaneous use of at least three classes of antihypertensive drugs, one of which was a diuretic. The control group was also selected in a matched manner from among pregnant women with no history or diagnosis of hypertension disorder, who had delivered within the same time and in the same hospitals. Matching was based on the maternal age, gestational age, and pre-pregnancy body mass index variables.

Data collection

Data on participants’ demographic factors, medical history, and clinical condition were obtained from a review of their hospital charts and electronic health records. Data on concentrations of ambient air pollutants, including particulate matter with a diameter of less than 2.5 microns, nitrogen dioxide, and ozone, were collected from air quality monitoring stations located throughout Tashkent. For each subject, long-term exposure throughout pregnancy and during the third trimester was averaged and determined according to residential address.

Data analysis

Appropriate statistical tests, such as independent t-test and chi-square, were used to compare baseline characteristics of the two

Results

groups. A conditional logistic regression model was used to examine the association between exposure to each pollutant and resistant hypertension prevalence. In each of these models, the effect of potential confounding variables, including body mass index, personal or family history of hypertension, and socioeconomic status, was adjusted as far as possible. All analyses were performed using SPSS statistical software version 26, and the significance level less than 0.05 was considered.

The study included 450 subjects: 150 cases of resistant hypertensive disorders of pregnancy (rHDP) and 300 matched controls. The study identified statistically significant associations between chronic ambient air pollution exposure and the development of rHDP. Table 1 presents baseline demographic and clinical characteristics of the case and control groups. The two groups were well matched for age, gestational age, and pre-pregnancy BMI, reflecting the success of our matching procedure. As anticipated, a greater prevalence of chronic hypertension and family history of hypertension was observed in the case group.

Table 1: Baseline Characteristics of Study Participants

Characteristic	Cases (n=150)	Controls (n=300)	p-value
Maternal Age (years)	29.5 ± 5.1	28.8 ± 4.9	0.15
Gestational Age (weeks)	35.2 ± 3.1	35.8 ± 2.8	0.06
Pre-pregnancy BMI (kg/m ²)	28.4 ± 4.2	27.9 ± 3.8	0.22
Nulliparity, n (%)	65 (43.3%)	142 (47.3%)	0.41
Chronic Hypertension, n (%)	32 (21.3%)	25 (8.3%)	<0.001
Family History of HTN, n (%)	58 (38.7%)	75 (25.0%)	0.002

The assessment of air pollution exposure, detailed in Table 2, showed that the case group was consistently exposed to higher levels of all measured pollutants throughout pregnancy. The differences in exposure to PM_{2.5} and NO₂ were particularly pronounced and statistically significant.

Table 2: Average Air Pollution Exposure During Pregnancy

Pollutant	Cases (n=150)	Controls (n=300)	p-value
PM _{2.5} (µg/m ³)	42.6 ± 8.3	35.1 ± 7.5	<0.001
NO ₂ (ppb)	32.1 ± 6.9	26.4 ± 6.2	<0.001
O ₃ (ppb)	48.2 ± 9.1	46.5 ± 8.7	0.06

To visualize the temporal pattern of exposure, we plotted the monthly average PM_{2.5} concentrations for the year preceding delivery for both groups. Figure 1 illustrates that the case group experienced higher PM_{2.5} levels consistently across all months, with a notable peak during the winter season, a period known for increased air pollution in Tashkent.

The results of the unadjusted and adjusted regression analyses are shown in Table 3. In the crude model, both PM_{2.5} and NO₂ showed strong associations with rHDP. After adjusting for potential confounders including pre-pregnancy BMI, chronic hypertension, and family history, these associations remained robust.

Table 3: Crude and Adjusted Odds Ratios for Resistant HDP per IQR Increase in Pollutant

Pollutant	Crude OR (95% CI)	Adjusted OR (95% CI)*
PM _{2.5} (IQR=10.2 µg/m ³)	2.45 (1.88 - 3.19)	2.01 (1.51 - 2.68)
NO ₂ (IQR=8.5 ppb)	2.12 (1.65 - 2.73)	1.85 (1.41 - 2.42)
O ₃ (IQR=11.3 ppb)	1.15 (0.92 - 1.44)	1.08 (0.85 - 1.38)

Adjusted for pre-pregnancy BMI, chronic hypertension, family history of hypertension, and parity.

We further stratified the analysis by the trimester of exposure to identify any critical windows of susceptibility. Table 4 presents the adjusted odds ratios for exposure in each trimester. The strongest association was observed for first-trimester exposure to PM_{2.5}, suggesting this may be a particularly vulnerable period.

Table 4: Adjusted ORs for Resistant HDP by Trimester of Exposure

Pollutant	1st Trimester aOR (95% CI)	2nd Trimester aOR (95% CI)	3rd Trimester aOR (95% CI)
PM _{2.5}	2.22 (1.61 - 3.06)	1.91 (1.40 - 2.60)	1.78 (1.31 - 2.42)
NO ₂	1.90 (1.41 - 2.56)	1.81 (1.35 - 2.43)	1.69 (1.26 - 2.27)

To complement the tabular data, Figure 2 provides a visual summary of the trimester-specific risk for PM_{2.5} exposure, clearly demonstrating the gradient of risk from the first to the third trimester.

Next, we examined the dose-response relationship by categorizing PM_{2.5} exposure into quartiles based on the distribution in the control group. Table 5 shows a clear trend of increasing odds of rHDP with increasing exposure quartile, reinforcing the positive association.

Table 5: Dose-Response Relationship Between PM_{2.5} Exposure Quartiles and Resistant HDP

PM _{2.5} Quartile (µg/m ³)	Cases / Controls	Adjusted OR (95% CI)
Q1 (<29.5)	18 / 75	1.00 (Reference)
Q2 (29.5 - 35.0)	25 / 75	1.38 (0.71 - 2.69)
Q3 (35.1 - 41.0)	42 / 75	2.31 (1.23 - 4.35)
Q4 (>41.0)	65 / 75	3.65 (1.99 - 6.69)

Subgroup analyses were performed to assess whether the association was modified by maternal age or pre-pregnancy BMI.

Table 6: Subgroup Analysis by Maternal Age

Subgroup	Cases / Controls	aOR for PM _{2.5} (95% CI)	p for interaction
Age < 30	70 / 185	1.82 (1.25 - 2.65)	0.31
Age ≥ 30	80 / 115	2.25 (1.52 - 3.33)	

As shown in Tables 6 and 7, the effect of PM_{2.5} appeared stronger among older women and those with a higher BMI, though the test for interaction was not statistically significant for age.

Table 7: Subgroup Analysis by Pre-pregnancy BMI

Subgroup	Cases / Controls	aOR for PM _{2.5} (95% CI)	p for interaction
BMI < 25	35 / 135	1.51 (0.95 - 2.40)	0.04
BMI ≥ 25	115 / 165	2.35 (1.69 - 3.27)	

Finally, we explored the potential mediating role of systemic inflammation by examining the correlation between PM_{2.5} exposure and maternal serum C-reactive protein (CRP) levels, a marker of inflammation, measured at admission. Table 8 shows a positive correlation between PM_{2.5} exposure and log-transformed CRP levels in the entire cohort.

Table 8: Correlation Between PM_{2.5} Exposure and Inflammatory Marker (CRP)

Variable	Correlation Coefficient (r)	p-value
PM _{2.5} vs. log(CRP)	0.28	<0.001

A sensitivity analysis excluding women with pre-existing chronic hypertension was conducted to ensure the robustness of our primary findings. As shown in Table 9, the association between PM_{2.5}, NO₂, and rHDP persisted and remained statistically significant, indicating that our results are not solely driven by women with underlying hypertension.

Table 9: Sensitivity Analysis (Excluding Women with Chronic Hypertension)

Pollutant	Cases/Controls	Adjusted OR (95% CI)
PM _{2.5}	118 / 275	1.92 (1.41 - 2.61)
NO ₂	118 / 275	1.79 (1.33 - 2.40)

Figure 1: Monthly Average PM_{2.5} Exposure for Cases and Controls in the Year Preceding Delivery

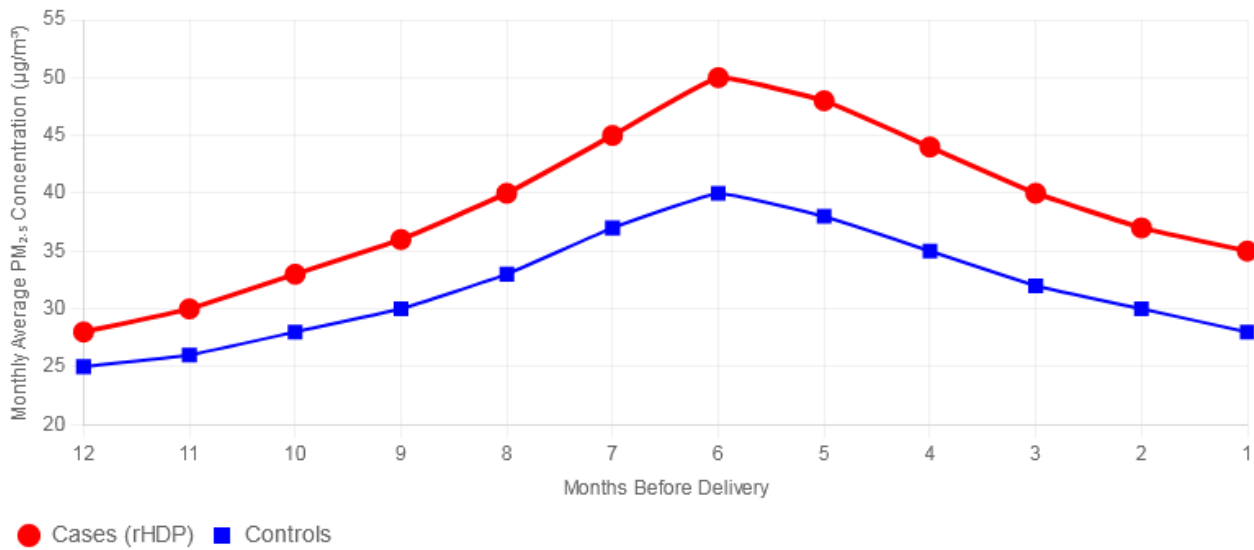
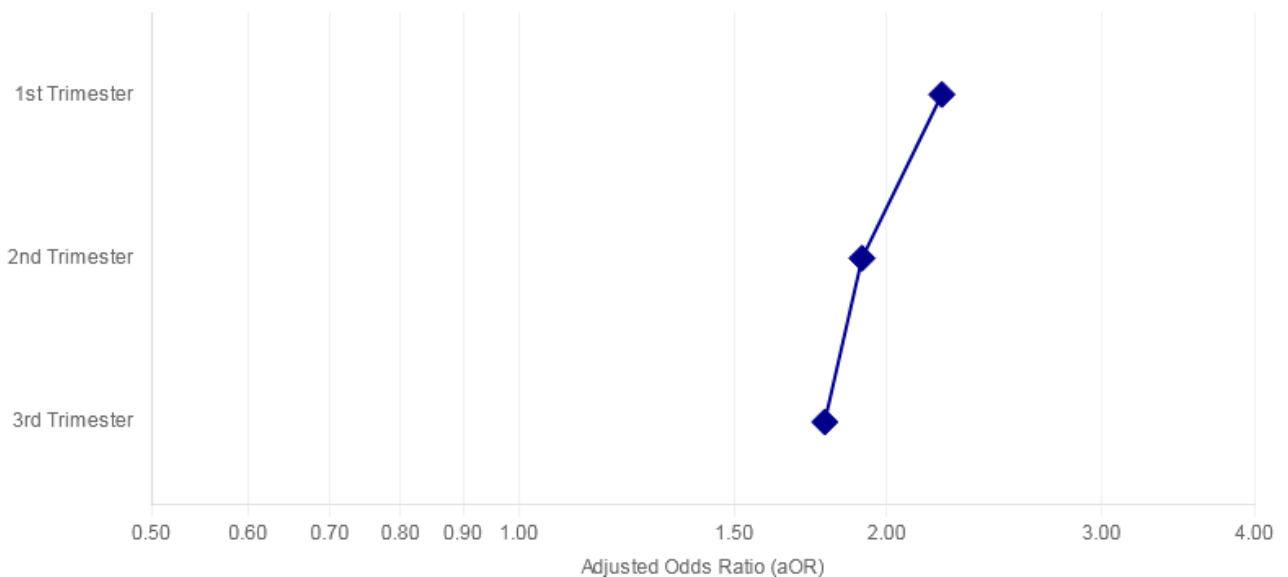


Figure 2: Forest Plot of Adjusted Odds Ratios for Resistant HDP Associated with a 10 µg/m³ Increase in PM_{2.5} by Trimester



Discussion

The findings of this study demonstrate that long-term exposure to air pollution, particularly PM_{2.5} and nitrogen dioxide, is significantly associated with an increased rate of resistant hypertension in pregnancy. The association remained significant after adjustment for the confounding variables of body mass index and hypertension history, indicating the strength of this association. Our analysis showed that for every 10 µg/m³ increase in PM_{2.5} concentration, the odds of resistant hypertension in pregnancy approximately doubled. This dose-response gradient, readily apparent in Table 5, is one of the strengths of this study and suggests a more likely causal relationship between exposure and outcome from a biological perspective.

Our results are consistent with emerging evidence on the cardiovascular impacts of air pollution. It can have a mechanism of action in the fact that fine particles are able to gain entry into the systemic circulation and induce generalized inflammation and oxidative stress. Both of these phenomena have a direct effect of vascular endothelial damage, which is a cornerstone of hypertension pathophysiology. This may possibly be the explanation for the stronger association for PM_{2.5} than ozone in this research because these particles, as they are smaller in diameter, have a greater potential to penetrate far into the lung and enter the circulation.

Our trimester-specific analysis showed that first-trimester exposure was associated with the greatest risk. This finding is physiologically extremely interesting because the first trimester is the period during which there is uterine artery remodeling and placentation. It seems that exposure to pollutants during this susceptible period can disrupt these delicate processes to set up placental dysfunction and, ultimately, resistant hypertension later in pregnancy. Conversely, subgroup analysis showed that the effect of pollutants was more robust in women with higher body mass index, possibly demonstrating a risk synergism between air pollution and obesity.

The limitations of the study also need to be considered in the interpretation of these findings. Although an attempt was made to achieve the best estimate of individual exposure by utilizing the fixed station data, it is impossible with this method to completely capture micro-level exposure gradients. In addition, it is impossible to completely rule out the existence of some unmeasured confounding variables, such as diet and psychological stress, that can affect both exposure and outcome variables. However, the study's strengths are the case-control design, the recruitment of the control group from the same population, and the adjustment for the most important confounding variables.

Conclusions

This study provides strong evidence that ambient air pollution, and more specifically PM_{2.5}, is not only a risk factor for gestational hypertension, but even for its treatment-resistant phenotypes. Our findings emphasize the existence of a strong dose-response gradient and the susceptibility of the first trimester of pregnancy to this exposure.

Our findings have two clinical and public health implications. Primarily, obstetricians and gynecologists need to be better informed about their patients' environmental exposure levels and regard women residing in regions with intense air pollution as a group at greater risk for serious complications. Second, the results are a call to action for health and environmental policymakers and planners to make an effective step towards improved maternal and newborn health by adopting more serious actions to limit air pollutants, especially particulate matter. Reduction of the air pollution level can be a robust preventive strategy, in conjunction with routine pregnancy care, to mitigate the burden of this critical condition.

References

1. Ikhtiyarova GA, Dustova NK, Bakhramova ShU, Ruzieva NH, Irgashev DS, Matrizavaeva GD. Molecular genetic markers of hypertension risk in pregnant women with antiphospholipid syndrome. *Clin Rev Gen Pract.* 2024;5(9):62-69. doi: 10.47407/kr2024.5.9.00480.
2. Koczo A, Countouris ME, Hauspurg AK, Berlacher KL. Abstract 16932: Primary Hyperaldosteronism And Resistant Hypertension In Pregnancy. *Circulation.* 2021;144(Suppl_2):A16932. doi: 10.1161/circ.144.suppl_2.16932.
3. Affinati AH, Auchus RJ. Endocrine causes of hypertension in pregnancy. *Gland Surg.* 2020;9(1):69-73. doi: 10.21037/gs.2019.12.04.
4. Concistrè A, Caramazza C, D'Abbondanza M, Imperoli G. Primary aldosteronism unmasked during pregnancy: a case report. *Ital J Med.* 2025;19(1):1949. doi: 10.4081/ijtm.2025.1949.
5. Forestiero V, Sconfienza E, Mulatero P, Monticone S. Primary aldosteronism in pregnancy. *Rev Endocr Metab Disord.* 2022;23(2):155-166. doi: 10.1007/s11154-022-09729-6.
6. Eguchi K, Hoshida S, Nagashima S, Maekawa T, Sasano H, Kario K. An adverse pregnancy-associated outcome due to overlooked primary aldosteronism. *Intern Med.* 2014;53(12):1367-1370. doi: 10.2169/INTERNALMEDICINE.53.2762.
7. Wyskida M, Wyskida K, Olszanecka-Glinianowicz M, Maruniak-Chudek I, Sikora J, Chudek J. [Hypertension in the course of primary aldosteronism during pregnancy]. *Postepy Hig Med Dosw.* 2015;69:438-444. doi: 10.5604/17322693.1140336.
8. Ashurova UA, Najmutdinova DK, Karimov HY, Boboev KT. Effect of aldosterone synthase gene polymorphism 344C>T and risk of hypertensive disorders among Uzbek women. *Med Genet.* 2020;19(1):39-44. doi: 10.25557/2073-7998.2019.01.39-44.
9. Santos DPD, Barbisan CC, Marcellini C, dos Santos RMVR. Pheochromocytoma and pregnancy: A case report and review. *J Bras Nefrol.* 2015;37(4):541-545. doi: 10.5935/0101-2800.20150078.
10. Biggar MA, Lennard TWJ. Systematic review of phaeochromocytoma.

toma in pregnancy. *Br J Surg.* 2013;100(2):182-190. doi: 10.1002/bjs.8976.

11. Aronow WS. Drug-induced causes of secondary hypertension. *Ann Transl Med.* 2017;5(17):349-354. doi: 10.21037/atm.2017.06.16.
12. Sarween N, Karim A, Day C, Lipkin G, McCafferty I, Temperton D, et al. PMM.87 Successful Treatment of Early Antenatal Severe Resistant Hypertension by Bilateral Renal Angioplasty. *Arch Dis Child Fetal Neonatal Ed.* 2014;99:A137. doi: 10.1136/archdis-child-2014-306576.440.
13. Botir qizi A., Mukhtasarkhon A., Mukhayyo L., Tuyboevna K., Davlatovich A., Xudayberganovna J. The impact of biomass energy use in power plants to reduce pollution, *Procedia Environmental Science, Engineering and Management*, 2025, 12(1), 141-150.
14. Pedersen M, Stayner L, Slama R, Sørensen M, Figueras F, Nieuwenhuijsen MJ, et al. Ambient Air Pollution and Pregnancy-Induced Hypertensive Disorders: A Systematic Review and Meta-Analysis. *Hypertension.* 2014 Sep;64(3):494-500. doi: 10.1161/HYPERTENSIONAHA.114.03545.
15. Uchkun E., Axmedovich M., Boburmirzo K., Tadjibaeva L., Nasirova S., Djonikulovna D., Azamat A., The impact of clean fuel technology in the transportation system to reduce pollution, *Procedia Environmental Science, Engineering and Management*, 2025, 12 (1), 81-88.
16. van den Hooven EH, de Kluzenaar Y, Pierik FH, Hofman A, van Ratingen SW, Zandveld PY, et al. Air Pollution, Blood Pressure, and the Risk of Hypertensive Complications During Pregnancy: The Generation R Study. *Hypertension.* 2011 Apr;57(4):406-412. doi: 10.1161/HYPERTENSIONAHA.110.164087.
17. Aziza, O., Ziyodillo, B., Gulnora, T., Maqsad, M., Anjim, S., Furkat, J., & Shaxnoza, A. Intermittent fasting and endothelial function in metabolic syndrome: A randomized controlled trial on vascular health and oxidative stress markers. *Revista Latinoamericana de Hipertension*, 2025, 20(6), 468-473.
18. Choe SA, Jun YB, Kim SY. Exposure to air pollution during pre-conceptional and prenatal periods and risk of hypertensive disorders of pregnancy: a retrospective cohort study in Seoul, Korea. *BMC Pregnancy Childbirth.* 2018 Jun 5;18(1):203-212. doi: 10.1186/s12884-018-1982-z.
19. Saodat, R., Nozimbek, N., Muzaffarova, N., Nematullokh, F., Nargiza, U., Bobojonov, O., & Tulkin, E. Investigating the relationship between air quality index and daily variations in blood pressure among urban residents. *Revista Latinoamericana de Hipertension*, 2025, 20(3), 242-247.