# Spatial analysis of congenital heart disease in São Paulo State, Brazil 2012-2022: associations with air pollution, maternal factors and social vulnerability

Amarílis Bahia Bezerra, Ligia Vizeu Barrozo, Alfredo Pereira de Queiroz

University of São Paulo, Faculty of Philosophy, Languages and Human Sciences, Department of Geography, São Paulo, SP, Brazil

Correspondence: Amarílis Bahia Bezerra, University of São Paulo, Faculty of Philosophy, Languages and Human Sciences, Department of Geography, São Paulo, SP, Brazil.
Tel.: +55.6198145-8851
E-mail: amarilis.bezerra@usp.br

Key words: congenital heart disease; air pollution; social vulnerability; maternal and child health; spatial analysis; Brazil.

Conflict of interest: the authors declare that they have no conflict of interest.

Funding: Alfredo Pereira de Queiroz was supported by the National Council for Scientific and Technological Development (CNPq), process 307438/2023-6.

Contributions: ABB conceptualized the study, conducted data analysis, interpreted the results, drafted, and critically revised the manuscript. LVB and APdQ contributed to the study design and critically revised the manuscript. All authors approved the final version and are fully responsible for all aspects of the work, including its accuracy and integrity.

Ethical approval: this study relied exclusively on secondary, anonymised, and aggregated data, publicly available through the Brazilian Ministry of Health. No individual-level or identifiable information was accessed or used. Therefore, this research does not involve human subjects directly and is exempt from ethical approval, in accordance with internationally recognized standards for research involving anonymised public data.

Availability of data and material: the database and R script used in this study are publicly available for access and reproducibility through the Zenodo repository (Bezerra, 2025).

Acknowledgements: the authors would like to thank the National Council for Scientific and Technological Development (CNPq) for its support.

Received: 28 May 2025.
Accepted: 11 August 2025.


*Geospatial Health 2025; 20:1407*
*doi:10.4081/gh.2025.1407*



## Abstract

Congenital Heart Disease (CHD) is a major cause of neonatal and infant morbidity and mortality and it has a multifactorial aetiology. This study aimed to analyse the spatial association between exposure to air pollutants during the first trimester of pregnancy, social vulnerability, and maternal factors with the occurrence of CHD between 2012 and 2022 in the state of São Paulo, Brazil. Data were obtained from the live birth information system for maternal outcomes and characteristics, the São Paulo social vulnerability index as a contextual indicator, and concentrations of fine particulate matter ($PM_{2.5}$), Carbon Monoxide (CO) and ozone, estimated using the Copernicus Atmosphere Monitoring Service (CAMS-EAC4) reanalysis dataset of environmental exposure. A Bayesian hierarchical spatial model with a Besag–York–Mollié 2 (BYM2) specification was applied using the INLA approach. The results showed that exposure to $PM_{2.5}$ was significantly associated with an increased risk of CHD (RR = 1.022; 95% CrI: 1.005-1.040), as were advanced maternal age (>35 years) (RR = 1.649; 95% CrI: 1.587-1.715) and inadequate prenatal care (RR = 1.112; 95% CrI: 1.070-1.155). Conversely, municipalities classified as having medium (RR = 0.757; 95% CrI: 0.641-0.894) and high social vulnerability (RR = 0.643; 95% CrI: 0.492-0.844) showed a significantly lower adjusted risk compared to those with low vulnerability. No significant associations were identified for CO or ozone. Spatial analysis revealed persistently high risks in municipalities within the São Paulo Metropolitan Region, even after adjusting for environmental and socio-demographic variables, highlighting population profiles and priority areas for public health surveillance and targeted interventions.

## Introduction

Congenital malformations are a significant cause of neonatal and infant morbidity and mortality worldwide, accounting for approximately 295,000 deaths annually, according to estimates from the World Health Organization (WHO, 2025). Among these conditions, Congenital Heart Defects (CHD) are particularly notable due to their frequency and severity, representing around one-third of all congenital malformations (van der Linde *et al.*, 2011). While genetic factors play a central role in the aetiology of CHD, an increasing body of evidence highlights the contribution of environmental and social exposures during pregnancy (Gelb & Chung, 2014; Patel & Burns, 2013). Air pollution, particularly in densely populated urban areas, has been recognised as a significant environmental risk factor for numerous health conditions (Cohen *et al.*, 2017; Zhan *et al.*, 2023). Pollutants such as fine particulate matter ($PM_{2.5}$), Carbon Monoxide (CO) and Ozone ($O_3$) are well documented for their detrimental effects on the cardiovascular and respiratory systems (Guo *et al.*, 2022; Choi *et al.*, 2024). Evidence suggests that maternal exposure to these pollutants during pregnancy may disrupt embryonic development, thereby increasing the risk of congenital malformations, including CHD (Buteau *et al.*, 2023; Sun *et al.*, 2023). Major urban centres characterised by high population density, heavy traffic, intensive land use and limited green spaces tend to exhibit elevated concentrations of atmospheric pollutants, amplifying the risk of harmful exposure during pregnancy (Abellan *et al.*, 2024). Within this context, socially marginalised populations are disproportionately affected, facing greater vulnerability to pollution due to residence in highly contaminated areas, coupled with persistent inequalities in access to prenatal care, diagnostic services, and appropriate treatment (Mathiarasan & Hüls, 2021). In Brazil, there is a scarcity of studies examining the influence of environmental and socioeconomic factors on the occurrence of CHD. The state of São Paulo, the most populous federal unit in the country, is characterised by pronounced socioeconomic disparities among its municipalities, intense urbanisation and high emissions of atmospheric pollutants (Barrozo *et al.*, 2020; Nogueira *et al.*, 2021; Silva *et al.*, 2021). This convergence of factors makes the region particularly pertinent for investigating the combined effects of environmental and social determinants on CHD. Furthermore, the incorporation of spatial risk modelling to address this issue remains limited in existing literature. Bayesian spatial models have been increasingly employed in public health research due to their capacity to account for geographical variability, spatial dependence among neighbouring areas, and uncertainties inherent in observational data (Wang, 2015; Ribeiro *et al*., 2017). These approaches are particularly valuable for elucidating territorial patterns of risk and for formulating and validating aetiological hypotheses related to health outcomes (Kang *et al.*, 2016). Within this context, the present study aims to examine the association between exposure to air pollutants during the first trimester of pregnancy, municipal-level social vulnerability, and maternal factors with the occurrence of congenital heart defects in live births in the state of São Paulo, Brazil, from 2012 to 2022.

## Materials and Methods

### Study area

The study was conducted in the state of São Paulo, located in the south-eastern region of Brazil (Figure 1). The Brazilian Institute of Geography and Statistics (IBGE) tells us that the state is currently home for more than 44 million inhabitants distributed across 645 municipalities (IBGE, 2025). It is the most populous and urbanised area in the country, characterised by pronounced socio-economic and environmental disparities. High population density, industrial concentration, and intense urban traffic contribute substantially to the region's air pollution burden, particularly in metropolitan areas (Girotti *et al.*, 2024).

### Study design and population

This ecological study employed spatial analysis, with municipalities of maternal residence serving as the units of analysis. The study population comprised all live births recorded between 2012 and 2022, according to data from the live birth information System (SINASC), accessed from anonymised public databases provided by the Ministry of Health (MoH) in Brazil (DATASUS, 2025). The outcome of interest was the occurrence of congenital heart defects (CHD), identified using codes Q20–Q28 of the International Classification of Diseases, 10th Revision (ICD-10), which encompasses congenital malformations of the circulatory system.

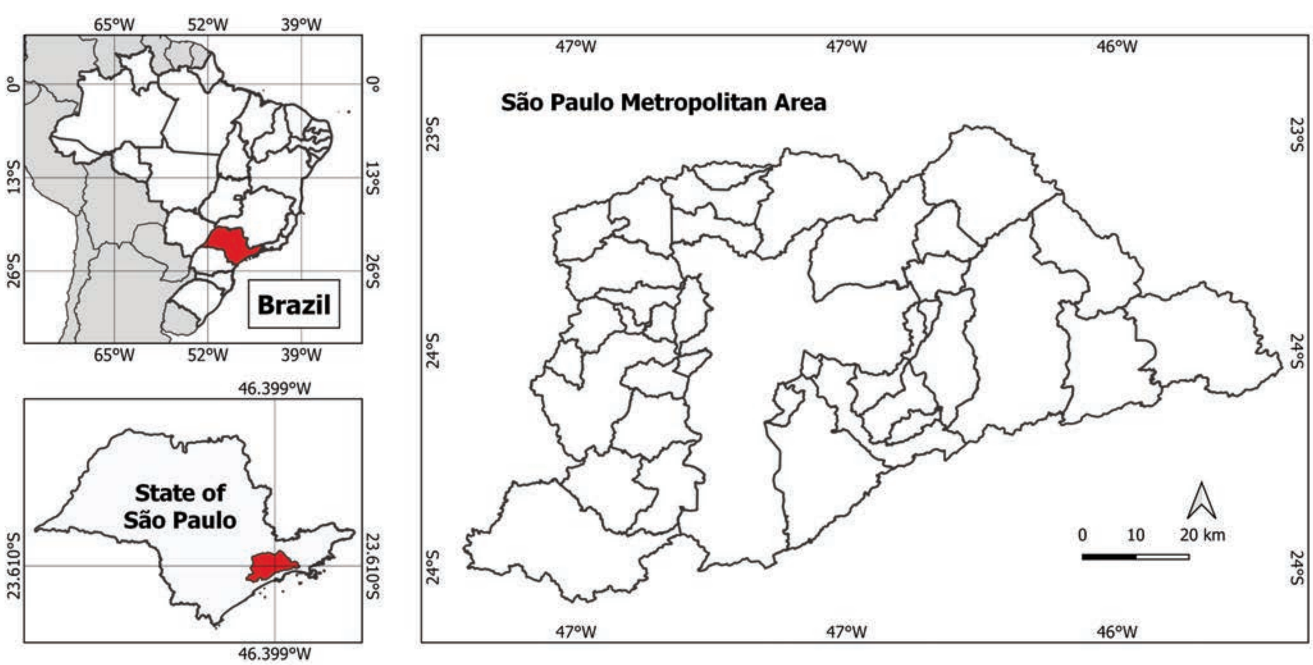


**Figure 1.** Location of the study area in South America.

## Exposure

Environmental exposures included mean air concentrations of $PM_{2.5}$, CO, and ozone $O_3$ estimated for the first trimester of pregnancy, covering the period from 2012 to 2022. Daily pollutant data were obtained from the Copernicus Atmosphere Monitoring Service (CAMS-EAC4) reanalysis dataset, accessed via the Climate Data Store (CDS) Application (API), in network common data form (NetCDF) format. The pollutant concentrations were extracted in raster format, representing a regular spatial grid where each cell contains the estimated concentration for a given date. $PM_{2.5}$ concentrations were extracted at the surface level, with CO and $O_3$ concentrations were obtained at the 1,000 hPa pressure level. For each pollutant, daily time series were generated based on the mean of the eight hourly records available per day. These data were subsequently aggregated to the municipality of maternal residence using zonal statistics, with municipal boundaries serving as the spatial unit. Concentrations were converted to µg/m³ using a factor of $1\times10^9$ for $PM_{2.5}$ and $O_3$ (given as kg/m³) and CO (kg/kg) assuming an air density of 1 kg/m³. To estimate exposure during the first trimester, the gestational period for each birth was calculated based on the date of birth and gestational age as recorded in SINASC. Exposure was defined as the mean daily concentration of each pollutant over the first 90 days of pregnancy, assigned to the municipality of maternal residence. The mean exposure of pregnant women residing in each municipality was then computed for the period 2012-2022

## Socio-demographic data

Three socio-demographic variables were included at the municipal level: i) the proportion of mothers aged over 35 years (advanced maternal age); ii) the proportion of births with an inadequate number of prenatal visits (fewer than seven), both derived from SINASC; and iii) the São Paulo Social Vulnerability Index (IPVS), based on the 2010 census and provided by the SEADE foundation. The IPVS was classified into three levels (low, medium, and high vulnerability), considering the most frequent category observed per municipality during the study period. Prior to model fitting, univariate analyses were performed to explore associations between each covariate and the outcome. Additionally, collinearity between the explanatory variables was assessed using the Variance Inflation Factor (VIF), retaining only variables with VIF ≤3

## Statistical modelling

A Bayesian hierarchical spatial model was fitted using the Besag–York–Mollié 2 (BYM2) specification (Riebler *et al.,* 2016), a 're-parameterisation' of the classical BYM model (Besag *et al.*, 1991) that addresses identifiability issues between spatially structured and unstructured components, while enabling an interpretable specification of hyperpriors. In this formulation, a single marginal precision parameter controls the variance of the combined spatial random effect, and a mixing parameter specifies the proportion of variance attributable to the structured effect. The BYM2 model can be expressed as:

$$b = \frac{1}{\sqrt{\tau_b}}\left(\sqrt{1-\phi}\,v + \sqrt{\phi}\,u*\right) \qquad \text{Eq. 1}$$

where $v$ is the unstructured random effect, independent and identically distributed (i.i.d.) as normal with mean 0 and variance 1; term$u*$ the scaled spatially structured effect, which follows an intrinsic conditional autoregressive (ICAR) distribution with marginal variance = 1; $\tau_b$ the overall precision; and $\phi$ $(0 \le \phi \le 1)$ the mixing parameter controlling the proportion of variance attributed to the structured component. The combined effect $b$ enters the linear predictor of the log-risk under the BYM2 specification and the outcome variable $Y_i$ representing the number of live births with CHD cases in municipality $i$, was assumed to follow a Poisson distribution:

$$\begin{aligned} &Y_i \sim \text{Poisson}(\mu_i), \\ &\log(RR_i) = \alpha + \textstyle\sum_k \beta_k x_{ki} + b_i \end{aligned} \qquad \text{Eq. 2}$$

where $n_i$ is the total number of live births in municipality $i$, α is the intercept, and $\beta_k$ are the regression coefficients for covariates $x_{ki}$, and $b$ *is* the combined spatial effect from the BYM2 specification.

Model estimation was performed using the Integrated Nested Laplace Approximation (INLA) method implemented via the R-

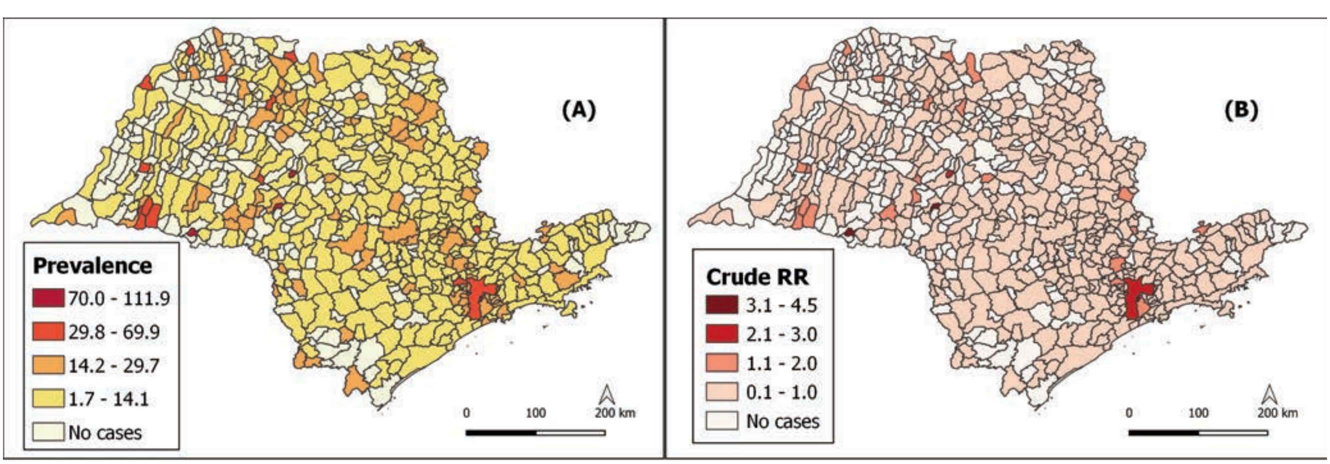


**Figure 2.** Prevalence of congenital heart disease per 10,000 live births by municipality in São Paulo State, Brazil 2012–2022. RR, relative risk.

INLA package (Lindgren & Rue, 2015). Penalised complexity (PC) priors were assigned to $\tau_b$($U$=1, α=0.01) and to the mixing parameter $\phi$ (U=0.5, α=2/3) as recommended by Riebler *et al.* (2016). Non-informative normal (0, 0.106) priors were used for fixed effects. Posterior estimates for the fixed effects were expressed as Relative Risks (RR), obtained by 'exponentiating' the corresponding regression coefficients and presented with their respective 95% Credible Intervals (CrIs). All data processing, spatial analyses, and statistical modelling were conducted in R version 4.3.2 and QGIS version 3.34 (R Core Team, 2022; QGIS Development Team, 2025).

## Results

Between 2012 and 2022, a total of 16,720 cases of Congenital Heart Defects (CHD) were recorded among live births in the state of São Paulo. The spatial distribution revealed a higher prevalence of cases in more populous regions, particularly within the São Paulo Metropolitan Region (SPMR) (Figure 2A). However, when considering the crude RR (Figure 2B), several municipalities across other regions of the state also exhibited elevated risk.

The spatial distribution of air pollutants displayed distinct patterns (Figure 3). Mean concentrations of $PM_{2.5}$ during the first trimester of pregnancy were highest in the eastern part of the state, particularly in the SPMR and adjacent municipalities (Figure 3A), while the CO and $O_3$ concentrations were predominantly higher in the northern regions, with CO also higher in and the Northeast (Figure 3B,C). The two latter concentrations exhibited a more diffuse distribution, with the highest levels observed in the northern areas (Figure 3C). The distribution of the IPVS showed a greater concentration of municipalities with high vulnerability in the extreme South of the state, while low vulnerability was concentrated in the centre-east regions of the state (Figure 3D).

The IPVS indicated that municipalities with high vulnerability were mainly located in the far South of the state, while areas with low vulnerability were concentrated in the central-eastern regions (Figure 3D). Table 1 presents the characterisation of municipalities according to IPVS and maternal factors. Municipalities classified as highly vulnerable exhibited higher proportions of births with inadequate prenatal care (mean 19.8%, with Standard Deviation,

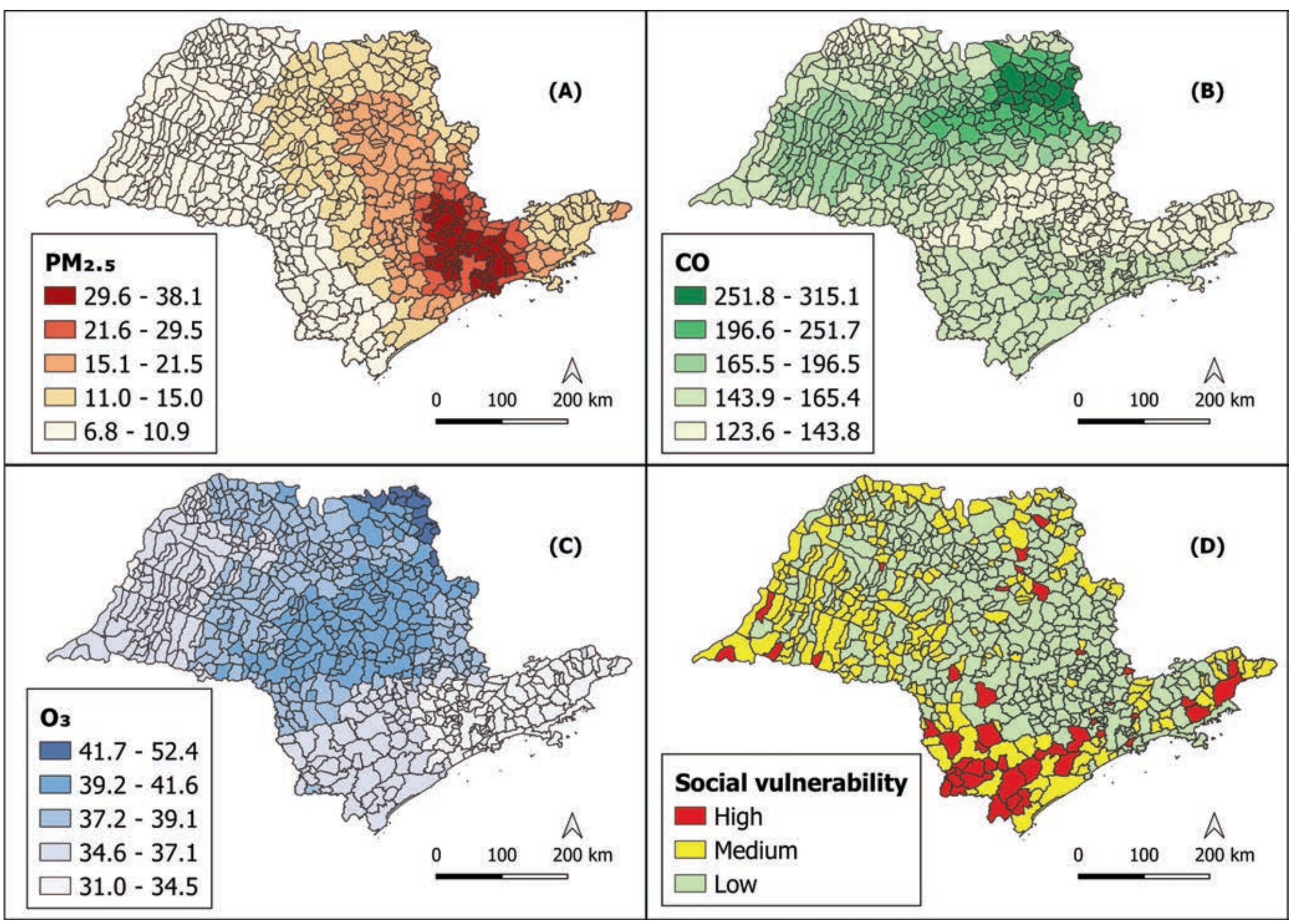


**Figure 3.** Spatial distribution of the average atmospheric pollutants in São Paulo State, Brazil 2012-2022. (**A**) $PM_{2.5}$; (**B**) CO; (**C**) $O_3$; (**D**) São Paulo Social Vulnerability Index (IPVS).

SD, of 8.2) compared to those with low vulnerability (mean 17.4%, SD 5.4). Conversely, the proportion of mothers aged over 35 years was lower in highly vulnerable municipalities (10.3%, SD 1.8) than in those with low vulnerability (11.9%, SD 2.2).

This study identified a statistically significant spatial association between the average concentrations of $PM_{2.5}$ during the first trimester of pregnancy and the occurrence of CHD in live births in São Paulo State, Brazil between 2012 and 2022, an association that remained after adjusting for socio-demographic variables. Table 2 summarises the posterior estimates of the RRs associated with the occurrence of CHD. A statistically significant association was observed between the average concentration of $PM_{2.5}$ during the first trimester of pregnancy and CHD (RR=1.022; 95% CrI: 1.005–1.040). No significant associations were identified for CO (RR=1.001; 95% CrI: 0.997-1.004) or $O_3$ (RR=0.991; 95% CrI: 0.944-1.042). Among maternal factors, advanced maternal age (>35 years) was strongly associated with an increased risk of CHD (RR=1.649; 95% CrI: 1.587-1.715), followed by inadequate antenatal care (<7 consultations) (RR=1.112; 95% CrI: 1.070-1.155). Interestingly, municipalities classified as having medium (RR=0.757; 95% CrI: 0.641-0.894) and high vulnerability (RR=0.644; 95% CrI: 0.492-0.844) showed significantly lower risks of CHD compared to those with low vulnerability. Figure 4A displays the spatial distribution of the posterior RR adjusted for environmental exposures, maternal socio-demographic characteristics, and contextual factors. Persistent areas of elevated risk were evident in certain municipalities within the SPMR. Figure 4B shows the posterior SD, indicating high precision in the model estimates (SD ranging from 0.000 to 0.004).

**Table 1.** Distribution of vulnerability by category in São Paulo State 2012-2022.

| Social vulnerability | Municipality (no.) | Proportion of total (%) | Age >35 years* (%) | SD** | Inadequate natal care* (%) | SD** |
|---|---|---|---|---|---|---|
| Low | 369 | 57.2 | 11.9 | 2.2 | 17.4 | 5.4 |
| Medium | 232 | 36.0 | 10.4 | 1.9 | 19.3 | 6.9 |
| High | 44 | 6.8 | 10.3 | 1.8 | 19.8 | 8.2 |

*Mean proportion of mothers; **standard deviation.

**Table 2.** Variables associated with congenital heart disease in live births in São Paulo State, Brazil 2012-2022.

| Variable | Posterior RR (95% CrI) | Posterior SD (log) |
|---|---|---|
| Exposure in 1st trimester | | |
| $PM_{2.5}$ | 1.022 (1.005-1.040) | 0.009 |
| CO | 1.001 (0.997-1.004) | 0.002 |
| Ozone | 0.991 (0.944-1.042) | 0.025 |
| Maternal and obstetric factor | | |
| Maternal age >35 years | 1.649 (1.587-1.715) | 0.020 |
| Inadequate prenatal care | 1.112 (1.070-1.155) | 0.019 |
| Social Vulnerability Index (SVI) | | |
| Medium vulnerability | 0.757 (0.641-0.894) | 0.085 |
| High vulnerability | 0.644 (0.492-0.844) | 0.137 |

CrI, credible interval; SD, standard deviation; SVI, São Paulo Social Vulnerability Index (IPVS).

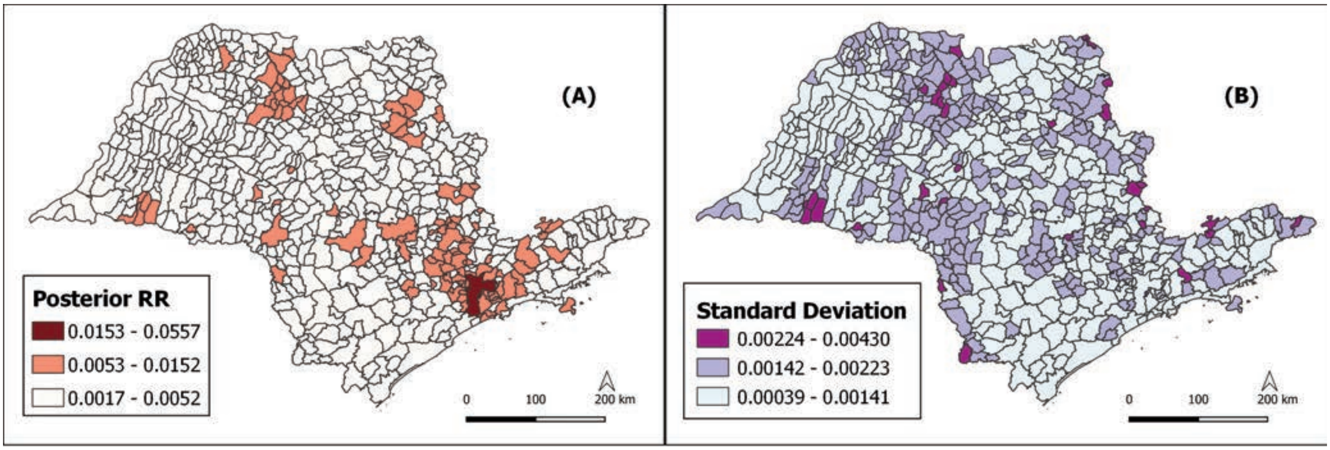


**Figure 4.** The relative risk for congenital heart disease by municipality in São Paulo State, Brazil 2012–2022. RR, relative risk.

## Discussion

The spatial association found between the average concentrations of $PM_{2.5}$ during the first trimester of pregnancy and the occurrence of CHD in live births in São Paulo State, Brazil between 2012 and 2022 aligns with previous research indicating $PM_{2.5}$ as a relevant environmental factor in the aetiology of CHD, particularly when exposure occurs during early pregnancy (Buteau *et al.*, 2023). Conversely, average concentrations of CO and $O_3$ during the same gestational period were not significantly associated with CHD occurrence. The literature on these pollutants presents heterogeneous results, with some studies reporting associations with specific cardiac anomalies, while others demonstrate less consistent effects (Dadvand *et al.*, 2011; Agay-Shay *et al.*, 2013; Ma *et al.*, 2021). Factors such as lower spatial variability of these compounds in our study or limitations related to exposure assessment resolution may have contributed to the absence of significant associations.

Advanced maternal age (>35 years) was strongly associated with increased CHD risk, corroborating well-established epidemiological evidence that highlights reproductive ageing as a key risk factor for congenital anomalies, including cardiovascular malformations (Miller *et al.*, 2011; Mamasoula *et al.*, 2023). Similarly, the proportion of births with inadequate prenatal care was positively associated with CHD risk. Although inadequate antenatal care is not a direct aetiological factor for congenital anomalies, it serves as a proxy for structural inequalities in access to healthcare and maternal support. These inequalities are often linked to environmental risks, adverse socioeconomic conditions, and poorer maternal health behaviours (Davtyan *et al.*, 2023). A lower risk of CHD was observed in municipalities classified as medium and high social vulnerability compared with those classified as low vulnerability. This pattern may reflect structural inequalities in access to diagnostic services and surveillance capacity. In less vulnerable municipalities, improved availability of healthcare, better screening, and more robust diagnostic infrastructure may lead to higher detection rates, thereby increasing the epidemiological visibility of CHD (Pinto Júnior *et al.*, 2015; Perez *et al.*, 2022). Conversely, more vulnerable municipalities may experience underreporting, resulting from limited access to diagnostic resources and weaker healthcare systems.

The application of a spatial Bayesian hierarchical model enabled stabilisation of estimates in areas with low case counts, reducing variance inflation and mitigating the small number problem. The inclusion of environmental and socio-demographic variables substantially reduced residual spatial variance, suggesting that these factors explain an important proportion of the observed geographical heterogeneity. Nonetheless, the persistence of elevated risk in certain municipalities indicates the influence of unmeasured contextual determinants, warranting further investigation.

This study presents several limitations inherent to its ecological design, including the inability to assess individual-level exposures or establish causal relationships. Additionally, key factors such as genetic predisposition, maternal health conditions, medication use, lifestyle factors, and other environmental exposures were not included due to the lack of individual-level data. Another important limitation pertains to the spatial resolution of the atmospheric data. While the CAMS-EAC4 dataset offers consistent and comprehensive spatial coverage, it lacks the granularity needed to capture intra-urban exposure variations or proximity to specific emission sources. In a highly heterogeneous region such as São Paulo, this may result in exposure misclassification, potentially biasing risk estimates towards the null. Future research should prioritise the integration of higher-resolution spatial and temporal data, including local monitoring networks, geostatistical interpolation, and models capable of capturing intra-urban pollution gradients. Incorporating spatio-temporal modelling frameworks would also enable assessment of seasonal variations, temporal trends, and the impact of public policies. Furthermore, enhancing datasets with clinical, environmental, and behavioural variables would strengthen causal inference and reduce ecological bias.

Despite these limitations, the findings provide important insights into the spatial distribution of CHD and its environmental and social determinants within the state of São Paulo. By highlighting territorial inequalities in CHD occurrence, this study underscores the importance of implementing public health policies aimed at reducing environmental exposures, strengthening prenatal care, enhancing diagnostic capacity, and addressing the needs of socially vulnerable populations.

## Conclusions

This study highlights that the occurrence of congenital heart defects in the state of São Paulo is spatially patterned and influenced by the interplay between environmental exposures, maternal characteristics, and municipal-level structural conditions. The significant association between $PM_{2.5}$ exposure during early pregnancy and this risk reinforces the importance of environmental health measures aimed at protecting maternal and child health. The inverse association observed with social vulnerability likely reflects diagnostic inequalities and underreporting in more deprived areas, underscoring the need to strengthen health surveillance and improve access to specialised care. These findings emphasise that CHD prevention requires comprehensive public policies that integrate environmental control, enhancement of prenatal care, and improved diagnostic capacity, particularly in vulnerable territories. Moreover, the use of spatial modelling emerges as a valuable tool for public health surveillance, enabling the identification of risk clusters and informing targeted interventions. Future research should further explore unmeasured contextual factors, such as healthcare quality and specific local environmental exposures, to better understand the residual spatial risk identified in this study.

## References

Abellan A, Warembourg C, Mensink-Bout SM, Ambros A, de Castro M, Fossati S, Guxens M, Jaddoe VW, Nieuwenhuijsen MJ, Vrijheid M, Santos S, Casas M, Duijts L, 2024. Urban environment during pregnancy and lung function, wheezing, and asthma in school-age children. The Generation R study. Environ Pollut 344:123345.

Agay-Shay K, Friger M, Linn S, Peled A, Amitai Y, Peretz C, 2013. Air pollution and congenital heart defects. Environ Res 124:28-34.

Barrozo LV, Fornaciali M, de André CDS, Morais GAZ, Mansur G, Cabral-Miranda W, de Miranda MJ, Sato JR, Amaro Júnior E, 2020. GeoSES: A socioeconomic index for health and social research in Brazil. PLoS One 15:e0232074.

Besag J, York J, Mollié A, 1991. Bayesian image restoration, with two applications in spatial statistics. Ann Inst Stat Math 43:1-20.

Bezerra AB, 2025. R script and data used for the analysis of factors associated with congenital heart disease in the state of São Paulo, Brazil (2012–2022). Zenodo. https://doi.org/10.5281/zenodo.16897066

Buteau S, Veira P, Bilodeau-Bertrand M, Auger N, 2023. Association between first trimester exposure to ambient PM2.5 and NO2 and congenital heart defects. Environ Health Perspect 131:067009.

Choi J, Henze DK, Nawaz MO, Malley CS, 2024. Source attribution of health burdens from ambient PM2.5, O3, and NO2 exposure for assessment of South Korean national emission control scenarios by 2050. Geohealth 8:e2024GH001042.

Cohen AJ, Brauer M, Burnett R, Anderson HR, Frostad J, Estep K, Balakrishnan K, Brunekreef B, Dandona L, Dandona R, Feigin V, Freedman G, Hubbell B, Jobling A, Kan H, Knibbs L, Liu Y, Martin R, Morawska L, Pope CA 3rd, Shin H, Straif K, Shaddick G, Thomas M, van Dingenen R, van Donkelaar A, Vos T, Murray CJL, Forouzanfar MH, 2017. Estimates and 25-year trends of the global burden of disease attributable to ambient air pollution. Lancet 389:1907-18.

Dadvand P, Rankin J, Rushton S, Pless-Mulloli T, 2011. Ambient air pollution and congenital heart disease. Environ Res 111:435-41.

DATASUS, 2025. Transferência de Arquivos. Available from: https://datasus.saude.gov.br/transferencia-de-arquivos/.

Davtyan A, Ostler H, Golding IF, Sun HY, 2023. Prenatal diagnosis rate of critical congenital heart disease remains inadequate with significant racial/ethnic and socioeconomic disparities and technical barriers. Pediatr Cardiol 45:1713-23.

Gelb BD, Chung WK, 2014. Complex genetics and the etiology of human congenital heart disease. Cold Spring Harb Perspect Med 4:a013953.

Girotti C, Oliveira MCQD, Sato AE, Chiquetto JB, Santos AP, de Miranda RM, Mülfarth RCK, Shimomura ARP, Lopez JMR, 2024. Urban mobility and air pollution at the neighbourhood scale in the Megacity of São Paulo, Brazil. Discover Cities 1:13.

Guo X, Song Q, Wang H, Li N, Su W, Liang M, et al, 2022. Systematic review and meta-analysis of studies between short-term exposure to ambient carbon monoxide and non-accidental, cardiovascular, and respiratory mortality in China. Environ Sci Pollut Res 29:35707-22.

IBGE, 2025. Panorama. Available from: https://cidades.ibge.gov.br/brasil/sp/panorama.

Kang SY, Cramb SM, White NM, Ball SJ, Mengersen KL, 2016. Making the most of spatial information in health: a tutorial in Bayesian disease mapping for areal data. Geospat Health 11:428.

Lindgren F, Rue H, 2015. Bayesian spatial modelling with R-INLA. J Stat Softw 63:i19.

Ma Z, Cao X, Chang Y, Li W, Chen X, Tang N-jun, 2021. Association between gestational exposure and risk of congenital heart disease. Environ Res 197:111014.

Mamasoula C, Bigirumurame T, Chadwick T, Addor MC, Cavero-Carbonell C, Dias CM, Echevarría-González-de-Garibay LJ, Gatt M, Khoshnood B, Klungsoyr K, Randall K, Stoianova S, Haeusler M, Nelen V, Neville AJ, Perthus I, Pierini A, Bertaut-Nativel B, Rissmann A, Rouget F, Schaub B, Tucker D, Wellesley D, Zymak-Zakutnia N, Barisic I, de Walle HEK, Lanzoni M, Sayers G, Mullaney C, Pennington L, Rankin J, 2023. Maternal age and the prevalence of congenital heart defects in Europe, 1995–2015. Birth Defects Res 115:583-94.

Mathiarasan S, Hüls A, 2021. Impact of environmental injustice on children's health—interaction between air pollution and socioeconomic status. Int J Environ Res Public Health 18:795.

Miller A, Riehle-Colarusso T, Siffel C, Frías JL, Correa A, 2011. Maternal age and prevalence of isolated congenital heart defects in an urban area of the United States. Am J Med Genet A 155:2137-45.

Nogueira T, Kamigauti LY, Pereira GM, Gavidia-Calderón ME, Ibarra-Espinosa S, Oliveira GL, Miranda RM, Vasconcellos PC, Freitas ED, Andrade MF, 2021. Evolution of vehicle emission factors in a megacity affected by extensive biofuel use. Environ Sci Technol 55:6677-87.

Patel SS, Burns TL, 2013. Nongenetic risk factors and congenital heart defects. Pediatr Cardiol 34:1535-55.

Perez MT, Bucholz E, Asimacopoulos E, Ferraro AM, Salem SM, Schauer J, Holleman C, Sekhavat S, Tworetzky W, Powell AJ, Sleeper LA, Beroukhim RS, 2022. Impact of maternal social vulnerability and timing of prenatal care on outcome of prenatally detected congenital heart disease. Ultrasound Obstet Gynecol 60:346-58.

Pinto Júnior VC, Branco KM, Cavalcante RC, Carvalho Junior W, Lima JR, Freitas SM, Fraga MN, Souza NM, 2015. Epidemiology of congenital heart disease in Brazil. Rev Bras Cir Cardiovasc 30:219-24.

QGIS Development Team, 2025. QGIS Geographic Information System. Open Source Geospatial Foundation Project. Available from: https://qgis.org.

R Core Team, 2022. R: A language and environment for statistical computing. Available from: https://www.r-project.org/

Ribeiro AI, Krainski ET, Carvalho MS, de Pina MF, 2017. The influence of socioeconomic deprivation, access to healthcare and physical environment on old-age survival in Portugal. Geospat Health 12:581.

Riebler A, Sørbye SH, Simpson D, Rue H, 2016. An intuitive Bayesian spatial model for disease mapping that accounts for scaling. Stat Methods Med Res 25:1145–65.

Silva MD, Oliveira MCQD, Drumond A, Rizz LV, 2021. Air pollutants associated with surface meteorological conditions in São Paulo's ABC region. Rev Bras Cienc Ambient 56:459–69.

Sun L, Wu Q, Wang H, Liu J, Shao Y, Xu R, Gong T, Peng X, Zhang B, 2023. Maternal exposure to ambient air pollution and risk of congenital heart defects in Suzhou, China. Front Public Health 10:1017644.

van der Linde D, Konings EEM, Slager MA, Witsenburg M, Helbing WA, Takkenberg JJM, et al, 2011. Birth prevalence of congenital heart disease worldwide. J Am Coll Cardiol 58:2241-7.

Wang G, 2015. Effects of weather and landscape on the equine West Nile virus infection risk in Mississippi, USA. Geospat Health 10:357.

World Health Organization, 2025. Impact. Congenital disorders. Available from: https://www.who.int/health-topics/congenital-anomalies#tab=tab_3.

Zhan C, Xie M, Lu H, Liu B, Wu Z, Wang T, et al, 2023. Impacts of urbanization on air quality and the related health risks in a city with complex terrain. Atmos Chem Phys 23:771–88.