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ORIGINAL ARTICLE

Investigating the association between ambient particulate matter (PM_{10}) exposure and blood pressure values: Results from the link between the Portuguese Health Examination Survey and air quality data

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KEYWORDS
Particulate matter;
Blood pressure;
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Air quality data

Abstract
Introduction and objectives: High blood pressure (BP) remains a major modifiable cardiovascular (CV) risk factor. Several epidemiologic studies have been performed to assess the association between air pollution exposure and this CV risk factor but results remain inconsistent. This study aims to estimate the effect of short-term PM_{10} exposure (average previous three-day concentration) on diastolic (DBP) and systolic (SBP) blood pressure values of the resident mainland Portuguese population.
Methods: Our study was based on available DBP and SBP data from 2272 participants from the first Portuguese Health Examination Survey (INSEF, 2015) living within a 30 km radius of at least one air quality monitoring station, with available measurements of particulate matter with an aerodynamic equivalent diameter $\leq$10 μm (PM_{10}). We used data from the air quality monitoring network of the Portuguese Environment Agency to obtain the individual allocated PM_{10} concentrations. Generalized linear models were used to assess the effect of PM_{10} exposure on DBP and SBP values.
Results: No statistically significant association was found between PM_{10} exposure and both DBP and SBP values (0.42% DBP change per 10 $\mu g/m^3$ of PM_{10} increment (95% confidence interval (CI): −0.85; 1.70) and 0.47% SBP change per 10 $\mu g/m^3$ of PM_{10} increment (95% CI: −0.86; 1.79)). Results remain unchanged after restricting the analysis to hypertensive or obese participants or changing the PM_{10} assessment methodology.

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Conclusions: In view of the PM_{10} levels observed in 2015, our results suggests that exposure to PM_{10} concentrations have a small or no effect on the blood pressure values. Other air pollutants and mixtures of pollutants that were not included in our study should considered in future studies.

PALAVRAS-CHAVE
Partículas;
Pressão arterial;
INSEF 2015;
Qualidade do ar

Investigando a associação entre a exposição a partículas ambientais (PM_{10}) e os valores da pressão arterial: resultados da ligação de dados do Inquérito Nacional de Saúde com Exame Físico e da qualidade do ar

Resumo
Introdução e objetivo: A pressão arterial elevada (PA) continua a ser um importante fator de risco cardiovascular (CV) modificável. Vários estudos epidemiológicos têm sido realizados para avaliar a associação entre a exposição à poluição do ar e este fator de risco CV, mas os resultados permanecem inconsistentes. Este estudo tem como objetivo estimar o efeito da exposição de curta duração às PM_{10} (concentração média dos últimos três dias) nos valores da pressão arterial diastólica (PAD) e sistólica (PAS) da população residente em Portugal Continental.
Métodos: O nosso estudo baseou-se nos dados disponíveis de PAD e PAS de 2272 participantes do primeiro Inquérito Nacional de Saúde com Exame Físico (INSEF, 2015) que viviam num raio de 30 km de pelo menos uma estação de monitoração da qualidade do ar com medições disponíveis de material particulado com diâmetro aerodinâmico $\leq$10 μm (PM_{10}). Foram utilizados os dados da rede de monitoração da qualidade do ar da Agência Portuguesa do Ambiente para atribuir as exposições individuais de PM_{10}. Os modelos lineares generalizados foram utilizados para estimar o efeito da exposição às PM_{10} nos valores de PAD e PAS.
Resultados: Nenhuma associação estatisticamente significativa foi encontrada entre a exposição a PM_{10} e os valores de PAD e PAS (0,42% de incremento de PAD por cada aumento de 10 $\mu g/m^3$ de PM_{10} [IC de 95%: −0,85; 1,70] e de 0,47% de incremento de PAS por cada aumento de 10 $\mu g/m^3$ [IC 95%: −0,86; 1,79]). Os resultados permaneceram inalterados após restringir a análise aos participantes hipertensos ou aos participantes obesos ou após alteração da metodologia para atribuir as exposições individuais de PM_{10}.
Conclusões: Tendo em consideração os níveis de PM_{10} observados em 2015, os nossos resultados sugerem que a exposição às concentrações de PM_{10} terá um efeito pequeno ou nenhum efeito sobre os valores da pressão arterial. Outros poluentes atmosféricos e misturas de poluentes que não foram incluídos no nosso estudo devem ser analisados em estudos futuros.

Introduction

High blood pressure (BP) or hypertension remains a major modifiable cardiovascular (CV) risk factor, being responsible for at least 45% of all deaths due to heart disease and 51% of deaths due to stroke.[1,2] Worldwide, it is estimated that around 31% of adults aged 25 years and over had hypertension, defined as systolic BP (SBP) $\geq$140 mmHg and/or diastolic BP (DBP) $\geq$90 mmHg.[3] In Portugal, according to the Portuguese Health Examination Survey (INSEF), the overall prevalence of hypertension, in the population aged 25–74 years of age, was 36.0%, in 2015, defined as having a SBP $\geq$140 mmHg and, or, a DBP $\geq$90 mmHg and, or, taking antihypertensive medication.[4]

The causes of high blood pressure are complex and are related to genetic, behavioral and environmental factors, including air pollution exposure.[5] Over the last decades, several epidemiological studies assessed the association between air pollution exposure, including particulate matter (PM), and BP values. Evidence from a recent meta-analysis which pooled studies published before May 2017, supports the association between PM_{10} and a DBP elevation. The authors of this meta-analysis found a DBP elevation of 0.86 mmHg, 95% CI (0.37; 1.35) and 0.15 mmHg, 95% CI (0.01–0.29) per each 10 $\mu g/m^3$ increase of PM_{10} in long ($\geq$30 days) and short-term (<30 days) periods of exposure, respectively.[6] The evidence compiled in this systematic review failed to support the association between PM_{10} and SBP values.

Direct PM translocation into the blood, oxidative stress, inflammatory mediator production and neuroendocrine activation are the potential biological mechanisms responsible for the pathophysiological mechanisms through which PM exposure can raise blood pressure, including vascular dysfunction and aortic stiffness,[7] sodium retention at the kidney level[8] and chronic kidney diseas.[9] According to Newman and his colleagues, the first three pathophysiological responses associated with BP elevation will occur in a few hour to days after exposure and are reversible whereas the last physiological response will occur mainly in a long-term exposure context and will be irreversible.[10]

To the best of our knowledge, in Portugal, the association between PM exposure and blood pressure values has never been published before. Consequently, our study aims to assess/investigate the association between individually allocated environmental PM_{10} concentrations and individual level SBP and DBP, considering a short period of time (previous three days before blood pressure measurements) as the window of exposure, in the mainland resident Portuguese population aged between 25 and 74 years old, in 2015.

Methods

Study population

This study used data from the first Portuguese National Health Examination Survey (INSEF), collected between February and December 2015. This survey was described in more detail by Nunes et al.[11] The present analysis was restricted to the subsample of INSEF participants from mainland Portugal (n=3467) who consented to have their data linked, provided a residence zip code number, were living within a 30-km radius of an air quality monitoring station with available PM_{10} concentration values and had blood pressure values measured in INSEF (n=2272). The INSEF survey received approval from the Ethics Committee of the Portuguese National Health Institute Doutor Ricardo Jorge, the National Data Protection Authority (Authorization no. 9348/2010) and from the regional Ethics Committees.

Health related data

Health data collection in INSEF was performed by trained health professionals, according to the European Health Examination Survey (EHES) procedures.[12] Blood pressure values were measured according to the previous published study of Rodrigues and colleagues.[4]

Sociodemographic (age, sex, educational level and occupation), lifestyle (smoking, excessive alcohol consumption, sedentary and unhealthy diet) and health status variables (diagnosed-dyslipidemia, diagnosed diabetes, lipid-lowering medication usage and diabetes medication usage) were obtained by self-report through the interview. Smoker's definition includes current daily and occasional smokers and excessive alcohol consumption was defined as reporting three or more days/week of consumption of at least one of the following alcoholic beverages: wine, beer, brandy/spirits, Port wine/martini/liqueur, whiskey/gin/vodka. Unhealthy diet was defined as no consumption of fruit and vegetables at least once a day and sedentary was defined as reporting reading, watching television or other sedentary activities as the best description of leisure time activities during the last 12 months.

Environmental exposure assessment

Hourly validated PM_{10} values were obtained from the QualAr database, available online at the Portuguese Environment Agency (APA) website (https://qualar.apambiente.pt/) and the individual allocated PM_{10} concentrations were obtained as previously reported by Gaio et al.[13] Briefly, for each individual the allocated three-day average PM_{10} concentration was the weighted average of three-day averaged PM_{10} concentrations of all stations within a 30 km radius from each participant's residence. This average was weighted by the inverse of the squared distance between the residence and the air quality monitoring stations.

The individually allocated three-day average temperatures were also obtained using data from the National Oceanic and Atmospheric Administration database (www.ncdc.noaa.gov) and we assumed the three-day average value of the closest temperature monitoring station as being representative of the individual exposure.

Statistical analysis

The statistical analysis was performed using the R statistical package version 3.6.3.[14] All estimates were weighted to account for different selection probabilities resulting from the complex sample design of INSEF 2015 and to match the geographic region, age group and sex population distribution of 2015. T-test and the Wilcoxon test were used to access differences of quantitative variables according to their adherence to the normal distribution or not. Proportions were compared using Pearson's chi-squared test. The significance level for all analysis was set at 5%.

Regression coefficients of effect (β) of PM_{10} on DBP and SBP with the corresponding 95% confidence intervals (CIs) were obtained by generalized linear regression models analyses for each 1 $\mu g/m^3$ increment of PM_{10}. We used the svyglm function from the ''survey'' R package to run each Gaussian family model with a link log function (family=gaussian (link=''log'')). Then, percent change per 10 $\mu g/m^3$ increment of PM_{10} with corresponding 95% CIs were calculated by using the formula $100\times[\exp(\beta)-1]*10$. Additionally, to obtain estimates comparable with previous studies, we also run the models using the (family=gaussian (link=''identity'')) to obtain results in mmHg increase per each 10 $\mu g/m^3$ increment of PM_{10} and these results are presented as supplementary data.

First, an unadjusted exposure-outcome model was fitted for each outcome. Then, a second model confounder-adjusted for sex (Male/Female), age group (25–49 years; 50–74 years), educational level (Low education/Medium education/High education), occupation (White-collar occupation/Blue-collar occupation), smoking (smoker/no smoker), excessive alcohol consumption (Yes/No), sedentary (Yes/No), unhealthy diet (Yes/No) and individual allocated three-day average temperature (continuous) was performed for each outcome. Additionally, as the objective was to assess only the short-term effects of PM_{10} exposure,

Table 1 Description of the general characteristics of the study participants.

Characteristics	Total (n=2272)	Males (n=1037)	Females (n=1235)
Age (n=2272) – %			
25–49 years old	53.0	53.6	52.4
50–74 years old	47.0	46.4	47.6
Level of education (n=2271) – %[a]			
Low education	**57.8**	**62.1**	**54.0**
Medium education	**21.9**	**21.9**	**21.9**
High education	**20.3**	**16.0**	**24.1**
Occupation (n=2102) – %[b]			
White-collar occupation	**63.6**	**56.1**	**70.7**
Blue-collar occupation	**36.4**	**43.9**	**29.3**
Lifestyles variables – %			
Smokers (n=2272)[c]	**21.5**	**27.0**	**16.6**
Excessive alcohol consumers (n=2271)[d]	**35.8**	**53.5**	**20.0**
Unhealthy diet (n=2271)[e]	**36.4**	**46.0**	**27.8**
Sedentary (n=2258)[f]	45.0	42.0	48.0
Hypertension (n=2272) – %[g]	**36.4**	**40.8**	**32.4**
Hypertensive medication (n=2272)[h]	25.1	24.4	25.7
Diastolic blood pressure (n=2272) – mmHg (mean, SD)	**74.0 (10.2)**	**76.1 (9.9)**	**72.0 (9.9)**
Systolic blood pressure (n=2272) mmHg (mean, SD)	**125.1 (16.0)**	**130.0 (14.3)**	**120.8 (16.3)**
Individual allocated			
3-Day average temperature (n=1887) – °C (mean, SD)	16.1 (4.0)	16.1 (4.0)	16.1 (4.0)
3-Day average PM_{10} (n=2272) – °C (mean, SD)	18.7 (8.3)	18.6 (8.2)	18.7 (8.4)

[a] Low education: levels 0–2 of the ISCED 2011; medium education: levels 3–4 of the ISCED 2011; high education: levels 5–8 of the ISCED 2011.
[b] White-collar occupation: Managers, Professionals, Technicians and Associate Professional, Clerical Support Workers and Services and Sales Workers; Blue-collar occupation: Skilled Agricultural Workers, Craft and Related Trades Workers, Plant and Machine Operators and Elementary Occupations.
[c] Smokers include current daily and occasional smokers.
[d] 3 or more days/week of consumption of at least one of the following alcoholic beverages (wine, beer, brandy/spirits, port wine/martini/liqueur, whisky/gin/vodka).
[e] No consumption of fruit and vegetables at least once a day.
[f] Reading, watching TV or other sedentary activities declared as the best description of the leisure time activities during the last 12 months.
[g] Defined as measured SBP ≥140 mmHg or measured DBP ≥90 mmHg or self-report of taking antihypertensive medication.
[h] Self-report of taking antihypertensive medication. Results in bold are those with statistically significant difference between females versus males, according to the Pearson's chi-square test (p<0.05).

an adjustment for the long-term effect of PM_{10} was also included by adding the variable one-previous year average of PM_{10} concentrations.

To assess the sensitivity of our analysis to the 30 km radius criteria, we also fit the models considering only participants living within a 20 km radius of an air quality monitoring station with available PM_{10} measurements. To evaluate the sensitivity of our analysis regarding the exposure assessment method, we also fit the models considering the modeled PM_{10} concentrations obtained by the application of a numerical air quality model – the WRF-CAMx modeling system that has been extensively applied over Portugal region with satisfactory skills, as previously described.[13]

Finally, we also repeat the analysis after excluding the participants taking anti-hypertensive medication and also when considering only more vulnerable groups, namely restricting the analysis to the hypertensive participants (defined as measured SBP ≥140 mmHg or measured DBP ≥90 mmHg or self-report of taking antihypertensive medication) or participants with abdominal obesity (assessed by using the formula: waist to height ratio >0.5).

Results

Participants' characteristics and PM_{10} values description

The general characteristics ((Table S1) analyzed were similar among included and excluded participants. Among the 2272 participants in our study, 52.8% were females, 53.0% aged between 25 and 49 years old, 57.8% had low education level and 63.6% had a white-collar occupation. Most participants reported to be non-smokers (78.5%), non-excessive alcohol consumers (64.2%), to have a healthy diet (73.6%) and to be not sedentary (55.0%). The prevalence of hypertension was 36.4% and 25.1% of the participants reported to take antihypertensive medication. The mean values of DBP

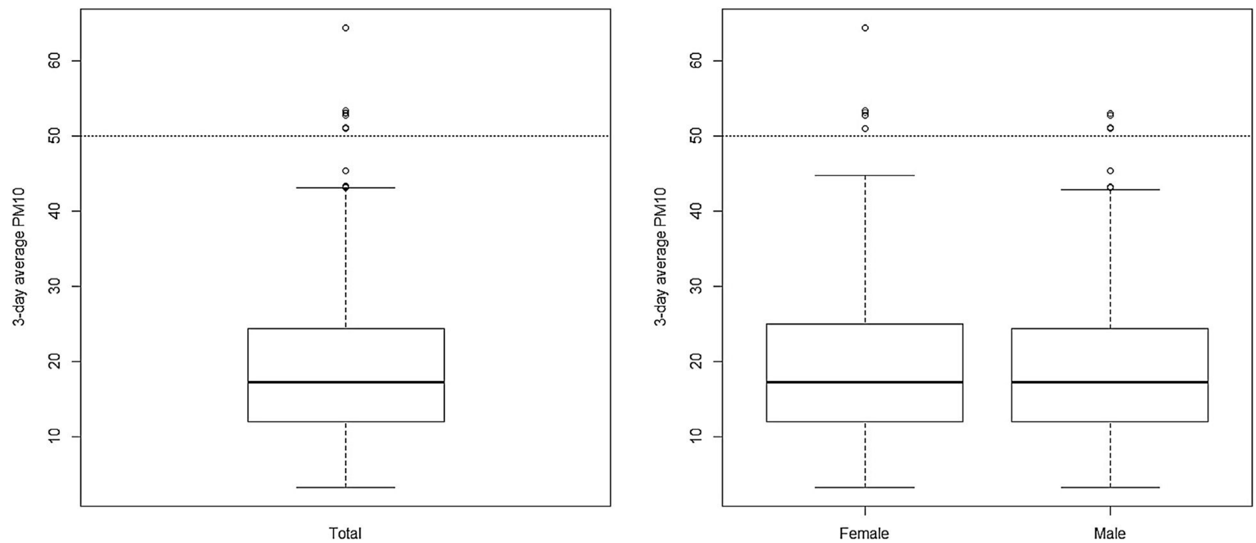

Figure 1 Boxplot of the PM_{10} distribution according to sex. The dashed lines represent the daily limit value given by the EU Ambient Air Quality Directives (not to be exceed on more than 35 days/year) and also by the World Health Organization air quality guidelines (not exceeded more than 3 days/year).

and SBP were 74.4 mmHg and 125.1 mmHg, respectively and the Individual allocated three-day average temperature was 16.1 °C. Differences between males and females were found regarding level of education, occupation, smoking, alcohol consumption, unhealthy diet, hypertension prevalence and blood pressure values (Table 1).

The individual allocated three-day average PM_{10} concentration values ranged between 3.31 $\mu g/m^3$ and 64.35 $\mu g/m^3$ (median=17.19 $\mu g/m^3$, IQR=12.40–24.27 $\mu g/m^3$) and differences between males and females were not found (Figure 1). The distribution of the three-day average PM_{10} concentration values is above the daily limit value given by the EU Ambient Air Quality Directives (not to be exceed on more than 35/year) and also by the World Health Organization air quality guidelines (not to be exceed more than three days/year).

Association between PM_{10} and diastolic blood pressure and systolic blood pressure values

There was no association between individual allocated three-day average PM_{10} concentration and DBP or SBP values (0.42% DBP increase per each 10 $\mu g/m^3$ PM_{10} increment, 95% CI: −0.85%; 1.70% and 0.47% SBP increase per each 10 $\mu g/m^3$ PM_{10} increment, 95% CI: −0.86%; 1.79%). Correspondent estimates in mmHg increase per each 10 $\mu g/m^3$ increment of PM_{10} are presented as supplementary data, in Table S9 (0.33 mmHg DBP increase per each 10 $\mu g/m^3$ PM_{10} increment, 95% CI: −0.62%; 1.27% and 0.57 mmHg SBP increase per each 10 $\mu g/m^3$ PM_{10} increment, 95% CI: −1.10%; 2.54%). Similar results were obtained in the stratified analysis, when considering only males or females (Table 2).

Sensitivity analysis

Regarding the sensitivity analysis, when we restricted our sample to the participants living within a 20 km radius of an air quality monitoring station with available PM_{10} values, similar results were found when considering the total sample and also when considering both males or females. Per each 10 $\mu g/m^3$ PM_{10} increment, there was a 0.51% (95% CI: −1.35; 2.37) increase in the DBP values and a 0.75% (95% CI: −1.02; 2.53) increase in the SBP values, being these results not statistically significant (Table S2). When we considered different exposure periods of time (2 previous days or 5 previous days instead of three previous days) similar results were found (Tables S3 and S4). Additionally, when we considered the individual allocated PM_{10} concentrations obtained by the air quality modeling system (WRF-CAMx), no associations were neither found (Table S5). Finally, when we restricted our analysis to participants not taking hypertensive medication, or when we considered only more susceptible individuals like those obese or diagnosed with hypertension, results remain similar (Tables S6–S8).

Discussion

Main finding of this study

In this study, no statistically significant association was found between PM_{10} exposure and both DBP and SBP values. However, the point estimates obtained were similar or even higher than those from the last published meta-analysis about the global association between ambient air pollution and blood pressure[6] (DBP: 0.33 versus 0.15 mmHg increase per each 10 $\mu g/m^3$ PM_{10} increment; SBP: 0.57 *versus* 0.21 mmHg increase per each 10 $\mu g/m^3$ PM_{10} increment). These results were consistently maintained by the sensitivity analysis performed, especially when considering a more restrictive criteria to the exposure assessment (participants living within a 20 km radius of an air quality monitoring station with available PM_{10} values), when considering the two or five previous days instead of three previous days as the exposure period of time, when considering an individual

Table 2 Percent changes in DBP and SBP per 10 μg/m^3 increment of PM_{10} according to sex.

	% Change per 10 μg/m^3 of PM_{10} increment	
	DBP	SBP
Total (n=2272)		
Unadjusted model	−0.73 (−1.92; 0.47)	−0.60 (−1.88; 0.69)
Adjusted model*	0.42 (−0.85; 1.70)	0.47 (−0.86; 1.79)
Males (n=1037)		
Unadjusted model	−0.61 (−1.95; 0.73)	−0.50 (−1.87; 0.88)
Adjusted model*	−0.04 (−1.09; 1.02)	0.68 (−0.81; 2.18)
Females (n=1235)		
Unadjusted model	−0.80 (−2.84; 1.25)	−0.64 (−2.75; 1.46)
Adjusted model*	0.76 (−1.42; 2.94)	0.16 (−1.98; 2.31)

DBP: diastolic blood pressure; SBP: systolic blood pressure.
* Adjusted for age, sex, educational level, occupation, smoking status, excessive alcohol consumption, unhealthy diet, sedentary, individual allocated ambient temperature, individual allocated 1-year average of PM_{10} concentrations.

allocated PM_{10} concentrations obtained from an air quality model instead of the direct measurements from the air monitoring stations or when considering only hypertensive, participants with abdominal obesity or participants not taking anti-hypertensive medication.

What is already known on this topic

According to a recent systematic review and meta-analysis performed on the association between ambient air pollution and blood pressure, that pools studies published before May 2017,[6] evidence already supported the association between short-term PM_{10} exposure and DBP values (0.15 mmHg, 95% CI: 0.01; 0.29, per each 10 μg/m^3 increase of PM_{10}), which our study was unable to confirm. However, the point estimate obtained in our study was higher than the one reported by the cited meta-analysis (0.33 versus 0.15 mmHg increase per each 10 μg/m^3 PM_{10} increment). Additionally, the same meta-analysis did not support the association between PM_{10} exposure and SBP values (0.21 mmHg, 95% CI: −0.01; 0.43, per each 10 μg/m^3 increase of PM_{10}), which is in concordance with our results.

It is important to mention that the polled estimates of the meta-analysis previously mentioned[6] were obtained using data from studies mostly from America and Asia regions where the levels of PM_{10} are higher in comparison to the majority of the European countries. When we consider only the pooling of the estimates from studies performed in European countries,[15–18] we found a −0.01 mmHg DBP decrease (95% CI: −0.14; 0.13) per each 10 μg/m^3 increase of PM_{10}. Accordingly, when considering only European countries, the published meta-analysis also did not support the association between short-term exposure to PM_{10} and diastolic (DBP) or systolic (SBP) blood pressure values, which is corroborated by our study.

After the publication of the previously cited meta-analysis,[6] additional studies were published on this issue but the association between short-term PM_{10} exposure and blood pressure values remains controversial.[19,20] The majority of the studies, mostly performed in China, provide evidence of a positive association between PM_{10} and blood pressure values[19,21] but there are some other studies that did not support this association.[20] For instance, in a panel study performed in Italy and Sweden, with a range of PM_{10} values comparable to those from Portugal, no associations were found between PM_{10} levels and either systolic (SBP) or diastolic (DBP) blood pressure but short-term exposure to PM_{10} results in reductions in carotid elasticity among elderly population which means that PM exposure could be associated with other important CV outcomes.[20]

What this study adds

This study suggests that exposure to PM_{10} concentrations in the levels observed in Portugal, similar to other European countries, has little or no effect on the blood pressure values, however it could have impact in other important cardiovascular outcomes. In fact, in a recent published study performed in the Portuguese population,[13] a significant association between short-term PM_{10} exposure (3-day average PM_{10} concentration) and white blood cells (WBC) among females was found (a 2.76% WBC increase, 95% CI: 0.65–4.87, per each 10 μg/m^3 PM_{10} increment) suggesting that PM_{10} exposure could impact on other outcomes with potential to mediate a cardiovascular event. Additionally, it is also important to mention that studies reporting significant results are more likely to be published and the possibility of a publication bias on this issue cannot be rejected.[22,23]

The point estimates obtained in this study were not statistically significant, which will be probably due to the lack of statistical power to estimate effects of this magnitude with a sample size of just over two thousand participants. However, even if the point estimates had statistical significance, we considered that obtained estimates would represent a small effect of the exposure to PM_{10} concentrations on blood pressure, taking into account the daily mean values of PM_{10} concentrations observed in the Portuguese context, in 2015 (three-day average PM_{10}: 18.7 μg/m^3).

Limitations of this study

Our study has some limitations. A first one is related to the exposure assessment method, due to the high distance considered between participants' residence and the air quality monitoring stations (30 km) in the inclusion criteria. The number and the spatial distribution of air quality monitoring stations in the Portuguese mainland does not allow us to apply a more restrictive distance due to a substantial sample reduction that could compromise the power of the estimates and result in selection bias. However, our results remain similar when we apply more restrictive criteria (including only participants living within a 20 km radius of an air quality monitoring station with available PM_{10} measurements) or when we used data from a numerical model.

A second limitation of our study is related to the possibility of an erroneous assumption on the temporal relationship between exposure to PM_{10} and the BP changes. In fact, the considered time window of exposure, that was the three previous days in our study, varied a lot in the previously published studies, ranging from days to months and even years. Despite the time window of PM exposure that is causally linked to BP levels is not well established, according to the perspective of Newman and his collaborators,[10] it will be on the order of a few days given the rapid occurrence of the physiological responses whereby PM influences on BP. Consequently, we assumed that the average three previous days' PM_{10} concentrations were adequate to represent the individual exposure of each participant. Additionally, we also tested the associations when considering two and five previous days instead of the three previous days and results remain the same.

Finally, even considering the adjustment for several potential confounders, there is still a possibility that the observed estimates could have been affected by residual confounding due to other unmeasured confounders. In addition, effect estimates presented in this study were based on a single-pollutant model and considering only PM_{10}, due the scarce available air quality monitoring data for the remaining air pollutants, but there are important interactions between the atmospheric pollutants, namely the potential additive effects of multiple pollutants, that should be considered in future studies.[24,25]

Conclusions

To the best of our knowledge, this is the first study assessing the association between an air pollutant exposure and the blood pressure values in Portugal. Our results suggest that exposure to the PM_{10} levels observed in 2015, have a small or no effect on the blood pressure values. However, we only analyzed one of a large amount of air pollutants, and future studies should investigate whether the exposure to other air pollutants, including particulate matter of a smaller size, can explain some of the causal link between short-term exposure to ambient air pollution and diastolic or systolic blood pressure values.

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Conflicts of interest

The authors declare that they have no conflicts of interest.

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Appendix A. Supplementary data

Supplementary material associated with this article can be found in the online version at doi:10.1016/j.repc.2022.02.011.

References

1. Lawes CM, Hoorn SV, Rodgers A. Global burden of blood-pressure-related disease, 2001. The Lancet. 2008;371:1513–8.
2. Stanaway JD, Afshin A, Gakidou E, et al. Global, regional, and national comparative risk assessment of 84 behavioural, environmental and occupational, and metabolic risks or clusters of risks for 195 countries and territories, 1990–2017: a systematic analysis for the Global Burden of Disease Study 2017. The Lancet. 2018;392:1923–94.
3. Mills KT, Bundy JD, Kelly TN, et al. Global disparities of hypertension prevalence and control: a systematic analysis of population-based studies from 90 countries. Circulation. 2016;134:441–50.
4. Rodrigues AP, Gaio V, Kislaya I, et al. Sociodemographic disparities in hypertension prevalence: results from the first Portuguese National Health Examination Survey. Rev Port Cardiol. 2019;38:547–55.
5. Mills KT, Stefanescu A, He J. The global epidemiology of hypertension. Nat Rev Nephrol. 2020;16:223–37.
6. Yang BY, Qian Z, Howard SW, et al. Global association between ambient air pollution and blood pressure: a systematic review and meta-analysis. Environ Pollut. 2018;235:576–88.

7. Rajagopalan S, Al-Kindi SG, Brook RD. Air pollution and cardiovascular disease: JACC state-of-the-art review. J Am Coll Cardiol. 2018;72:2054–70.
8. Tsai DH, Riediker M, Wuerzner G, et al. Short-term increase in particulate matter blunts nocturnal blood pressure dipping and daytime urinary sodium excretion. Hypertension. 2012;60:1061–9.
9. Wu MY, Lo WC, Chao C, et al. Association between air pollutants and development of chronic kidney disease: a systematic review and meta-analysis. Sci Total Environ. 2020;706:135522.
10. Newman JD, Rajagopalan S, Levy P, et al. Clearing the air to treat hypertension. J Human Hypertens. 2020;34:759–63.
11. Nunes B, Barreto M, Gil AP, et al. The first Portuguese National Health Examination Survey (2015): design, planning and implementation. J Public Health (Oxford, England). 2019;41:511–7.
12. Tolonen H. EHES manual. Part B. Fieldwork procedures. Helsinki; 2013.
13. Gaio V, Roquette R, Monteiro A, et al. Exposure to ambient particulate matter increases blood count parameters with potential to mediate a cardiovascular event: results from a population-based study in Portugal. Air Qual Atmos Health. 2021;14:1189–202.
14. Core Team. R: a language and environment for statistical computing. Vienna, Austria: R Foundation for Statistical Computing; 2020.
15. Bilenko N, Van Rossem L, Brunekreef B, et al. Traffic-related air pollution and noise and children's blood pressure: results from the PIAMA birth cohort study. Eur J Prevent Cardiol. 2015;16:4–12.
16. Pieters N, Koppen G, van Poppel M, et al. Blood pressure and same-day exposure to air pollution at school: associations with nano-sized to coarse PM in children. Environ Health Perspect. 2015;123:737–42.
17. Tsai DH, Guessous I, Riediker M, et al. Short-term effects of particulate matters on pulse pressure in two general population studies. J Hypertens. 2015;33:1144–52.
18. Harrabi I, Rondeau V, Dartigues JF, et al. Effects of particulate air pollution on systolic blood pressure: a population-based approach. Environ Res. 2006;101:89–93.
19. Guo P, He Z, Jalaludin B, et al. Short-term effects of particle size and constituents on blood pressure in healthy young adults in Guangzhou, China. J Am Heart Assoc. 2021;10:e019063.
20. Scheers H, Nawrot TS, Nemery B, et al. Changing places to study short-term effects of air pollution on cardiovascular health: a panel study. Environ Health. 2018;17:1–8.
21. Xu N, Lv X, Yu C, et al. The association between short-term exposure to extremely high level of ambient fine particulate matter and blood pressure: a panel study in Beijing, China. Environ Sci Pollut Res. 2020;27:28113–22.
22. Mlinarić A, Horvat M, Smolčić VŠ. Dealing with the positive publication bias: why you should really publish your negative results. Biochemia Medica. 2017;27:447–52.
23. Koletsi D, Karagianni A, Pandis N, et al. Are studies reporting significant results more likely to be published? Am J Orthodont Dent Orthop. 2009;136:e1632–5.
24. Oakes M, Baxter L, Long TC. Evaluating the application of multipollutant exposure metrics in air pollution health studies. Environ Int. 2014;69:90–9.
25. Davalos AD, Luben TJ, Herring AH, et al. Current approaches used in epidemiologic studies to examine short-term multipollutant air pollution exposures. Ann Epidemiol. 2017;27:145–53.