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Association of Long Term Exposure to Particulate Matter and Ozone with Health Status and Mortality in Patients after Myocardial Infarction.

Ali O. Malik, MD[a,b], **Philip G. Jones, MS**[a,b], **Paul S. Chan, MD MSc**[a,b], **Poghni A. Peri-Okonny, MD, MSc**[a,b], **Vittal Hejjaji, MD**[a,b], and **John A. Spertus, MD MPH**[a,b]

[a]University of Missouri - Kansas City

[b]Saint Luke's Mid America Heart Institute

Abstract

Background: Long term exposure to particulate matter <2.5μm in diameter ($PM_{2.5}$) and ozone has been associated with the development and progression of cardiovascular disease and, in the case of $PM_{2.5}$, higher cardiovascular mortality. Whether exposure to $PM_{2.5}$ and ozone is associated with patients' health status and quality of life, is unknown. We used data from two prospective myocardial infarction (MI) registries to assess the relationship between long-term $PM_{2.5}$ and ozone exposure with health status outcomes one year after a MI.

Methods and Results: TRIUMPH and PREMIER enrolled patients presenting with MI at 31 U.S. hospitals between 2003 and 2008. One year later, patients were assessed with the disease-specific Seattle Angina Questionnaire (SAQ) and 5-year mortality was assessed with the Centers for Disease Control's National Death Index. Individual patients' exposures to $PM_{2.5}$ and ozone over the year after their MI were estimated from the EPA's Fused Air Quality Surface Using Downscaling tool that integrates monitoring station data and atmospheric models to predict daily air pollution exposure at the census tract level. We assessed the association of exposure to ozone and $PM_{2.5}$ with 1-year health status and mortality over 5-years using regression models adjusting for age, sex, race, socioeconomic status, date of enrollment and comorbidities. In completely adjusted models, higher $PM_{2.5}$ and ozone exposure were independently associated with poorer SAQ summary scores at 1-year (β estimate per +1 SD increase = −0.8 (95% CI −1.4, −0.3 p=0.002) for $PM_{2.5}$ and −0.9 (95% CI-1.3, −0.4 p<0.001) for ozone). Moreover, higher $PM_{2.5}$ exposure, but not ozone, was independently associated with greater mortality risk (HR = 1.13 per +1 SD (95% CI = 1.07-1.20, p<0.001).

Conclusions—In our study, greater exposure to $PM_{2.5}$ and ozone was associated with poorer 1-year health status following an MI, and $PM_{2.5}$ was associated with increased risk of 5-year death.

Correspondence to: Ali O. Malik , University of Missouri-Kansas City/Saint Luke's Mid America Heart Institute, 4401 Wornall Road, 9th Floor Kansas City MO 64111 Phone: 702-831-8902; Fax: 816-932-5613, aomalik@saint-lukes.org.

Disclosures:

Dr. Spertus owns copyright for the Seattle Angina Questionnaire. He serves as a consultant to United Healthcare, Bayer and Novartis (modest). He has research grants from Abbott Vascular, Novarits and is the PI of an analytic center for the American College of Cardiology (significant). He has an equity interest in Health Outcomes Sciences (significant). The other authors report no disclosures.

Background

Exposure to air pollutants is an important risk factor for premature morbidity and mortality.[1] Fine particulate matter <2.5 μm in diameter ($PM_{2.5}$) and ozone are the most studied air pollutants and have been associated with a wide range of diseases.[1] Long-term exposure to $PM_{2.5}$ has been implicated in the development and progression of cardiovascular disease.[2-5] Complex and interlinked mechanisms have been proposed to explain this association with $PM_{2.5}$ exposure, including higher rates of atherosclerosis[6, 7] and the development of cardio-metabolic conditions, such as diabetes mellitus (DM), hypertension (HTN) and dyslipidemia.[8] In contrast to $PM_{2.5}$, there is some uncertainty between ozone exposure and cardiovascular outcomes. In a large study of over 400,000 participants, investigators did not find an association between ozone levels and cardiovascular mortality independent of $PM_{2.5}$.[9] Other studies have found an association between ozone exposure and mortality due to embolism[10], ischemic heart disease[11], heart failure[12] and stroke[13], but not all of these studies evaluated this association with ozone independent of $PM_{2.5.}$

Beyond mortality, patients are equally or more concerned about their health status: their symptoms, function and quality of life (QoL). To date, there have been no studies examining the association between exposure to $PM_{2.5}$ and ozone with patients' health status among patients with coronary artery disease (CAD). A deeper understanding of the potential impact of air pollution may further guide public policy regarding air quality standards, as the health benefits of stricter standards are better understood. Accordingly, we used data from the two prospective myocardial infarction (MI) registries to assess the relationship between long term exposure to these important air pollutants with health status one year after MI.

Methods

The investigators are willing to work with others who are interested in validating or extending the analyses. Air quality data for the US is publically available. The analytical codes can be made available but patient specific data will not be publically available.

Study Population

For this study, we included data on patients with an acute MI from two prospective registries with detailed information on patients' disease-specific health status. The TRIUMPH (Translational Research Investigating Underlying Disparities in Acute Myocardial Infarction) and PREMIER (Prospective Registry Evaluating Myocardial Infarction: events and Recovery) studies enrolled patients form 31 US hospitals between 2003 and 2008. Detailed design and methods for these registries have previously been described.[14, 15] Briefly, patients were required to have biomarker evidence of myocardial necrosis and additional clinical evidence supporting the diagnosis of MI, including prolonged ischemic signs/symptoms or electrocardiographic criteria of ST segment changes. Baseline data were obtained through chart abstraction and structured interviews by trained research coordinators. Health status data were obtained at baseline and during follow-up, including 1 year after the patients' MI, using standardized interview conducted by trained study personnel. Each participating site obtained Institutional Research Board approval and all patients provided written informed consent for the interview.

Assessment of Air Pollution Exposures

Each patient's exposure to ambient air $PM_{2.5}$ and ozone was estimated using publicly available data from Community Multi-Scale Air Quality Model (CMAQ) and point measurements provided by United States Environment Protection Agency (EPA).[16] For this study, exposure to $PM_{2.5}$ was based on average daily concentrations, expressed in microgram per cubic meter ($\mu g/m^3$). Exposure to ozone was based on the average of daily 8-hour maximum ozone levels, expressed in parts per million (ppm) moist air molecules in a fixed air volume. These metrics to assess long-term exposure are used commonly throughout the world and the EPA reports $PM_{2.5}$ and ozone exposure using these metrics.[16, 17]

Daily average $PM_{2.5}$ and 8-hour maximum ozone levels were derived from a Bayesian space-time downscaling fusion (DS) model by estimating concentrations at the census tract centroid of the patient's residence on the basis of National Air Monitoring Stations/State and Local Air Monitoring Stations and CMAQ model data in 12 × 12 km grids. Downscaler $PM_{2.5}$ and ozone estimates consider all monitors, as opposed to the most prevalent monitor, in areas where there are multiple monitors per site.[18] The DS model performance using the predictive mean absolute error showed that the model outperformed ordinary kriging or CMAQ models.[19]

The main focus of our paper was to examine an association of exposure to air pollutants with health status. However we also wanted to assess if previously described association of greater exposure to air pollutants with mortality was similar in our study cohort. Different approaches for the measurement of air pollutant exposure were used for the health status and mortality comparisons. Because the health status comparison was 1 year after the MI, we used average pollutant exposure over the year after a patient's MI. In contrast, as the mortality analyses examined survival after discharge, the air pollution exposure over the year prior to the MI was used.

Study Outcomes

Our main study outcome was health status at 1-year after MI. Disease-specific health status was assessed using the Seattle Angina Questionnaire (SAQ). The SAQ is a valid 19-item instrument with a 4-week recall period. It measures 5 domains of health in patients with coronary artery disease (CAD), which are angina frequency (SAQ AF), angina stability, QoL (SAQ QOL), physical limitation (SAQ PL) and treatment satisfaction.[20] Domain scores range from 0 to 100, with higher scores indicating fewer symptoms and better QoL. The primary outcome for this study was the overall health status, which is summarized using the SAQ summary score (SAQ SS) and reflects the average of the SAQ PL, AF and QOL domains.[21]

In addition, we assessed the effect of air pollutants on other health status measures. These included physical health status, as measured by the Physical Component Summary of the Medical Outcomes Study 12-item Short Form (SF-12 PCS).[22] The SF-12 is a reliable and valid measure of generic health status and provides summary component scales for overall physical and mental health.[23] As dyspnea is a common angina equivalent in patients with coronary artery disease (CAD)[24]and might also be associated with higher air pollution

levels, we also assessed the effect of air pollutants on dyspnea using a 4-level dyspnea item based on the Rose Dyspnea Scale (RDS). The RDS is a 4-item questionnaire with a 1-month recall period that assesses the patient's level of dyspnea with common activities.[25] Each activity associated with dyspnea is assigned 1 point, with scores of 0 indicating no dyspnea and increasing scores indicating greater limitation from dyspnea. The RDS has been used to assess symptoms in patients with coronary artery disease and has shown to be associated with QoL, rehospitalization, procedure success and long-term outcomes.[26] Lastly, mortality status over 5-years was determined through a query of the Centers for Disease Control's National Death Index.

Statistical Analysis

Baseline demographic and clinical characteristics of patients were compared across quartiles of average $PM_{2.5}$ exposure. Differences in patient characteristics across quartiles of exposure were compared using analysis of variance (ANOVA) for continuous variables and chi-square tests for categorical variables. Categorical variables are presented as mean ± standard deviation (SD) or median with interquartile range. Categorical variables are presented as number (n) with percentage.

We evaluate the association between exposures to average daily ambient air $PM_{2.5}$ and ozone with health status using generalized additive models with Gaussian errors for SAQ and SF-12 PCS scores and using a proportional odds logistic regression model for dyspnea scores. Smoothing or restricted cubic splines were used to allow for non-linear associations. Several models were defined *a priori*. In Model 1, we assessed unadjusted associations with $PM_{2.5}$ and ozone without adjusting for any covariates, except for ozone levels when assessing $PM_{2.5}$ and adjusting for $PM_{2.5}$ when assessing the impact of ozone. In Model 2, we additionally adjusted for covariates known to be associated with health status in patients with CAD.[27] These were demographics (age, sex and race), smoking, date of enrollment, and socioeconomic status (SES). SES has been shown to be associated with worse outcomes after MI[28] and was quantified using patients' education, insurance status, history of avoiding care due to costs, and end-of-the-month financial resources. We also adjusted for date of enrollment to account for temporal or seasonal effects. Since $PM_{2.5}$ has been previously shown to be associated with the development of HTN, DM, chronic kidney disease (CKD) (defined as eGFR <60) and heart failure[8], we constructed Model 3 to assess whether there remained an independent association between exposure to $PM_{2.5}$ and ozone and health status after accounting for these factors; which could potentially have either confounding or mediating effects, or both. Next, to evaluate whether air pollutants were also associated with change in health status following MI we constructed an additional model using change in health status from baseline to 1 year as the outcome with adjustment for the covariates included in the second model, as well as baseline health status. Finally as higher concentrations of ozone can amplify the adverse cardiovascular effects of $PM_{2.5}$[29] we augmented Model 2 to include an interaction between $PM_{2.5}$ and ozone levels, and estimated the effect of $PM_{2.5}$ at different ozone concentrations (10^{th} and 90^{th} percentiles and median of the distribution in our study).

While the main objective of our paper was to determine the association of air pollutant exposure with the health status of patients with stable ischemic heart disease who recently had a MI, we also wanted to see if the association of $PM_{2.5}$ with mortality was similar in our study cohort as has been described in previous studies. Hence we examined the association of 12-month average air quality parameters prior to patient's MI with all-cause mortality through 5 years after patients' index MI. We calculated crude survival rates by quartiles of $PM_{2.5}$ and ozone exposure using Kaplan-Meier methods, and we estimated hazard ratios using Cox regression models adjusted for patients' demographics (age, sex and race), SES, smoking status and comorbidities, including HTN, DM, CKD, heart failure, left ventricular function, prior to MI and Global Registry of Acute Coronary Events (GRACE) mortality risk score. We additionally adjusted for ozone levels when assessing the associations between $PM_{2.5}$ and outcomes and $PM_{2.5}$ levels when assessing association with ozone. The proportional hazards assumptions were assessed using Schoenfeld residuals.

All models included smoothing or restricted cubic splines for estimating effects of continuous variables to accommodate nonlinear relationships. In cases where no significant evidence of nonlinearity was found, associations were re-estimated using linear effects to simplify interpretation. All analyses were performed using SAS version 9.4 (SAS Institute Inc., Cary, NC) and R version 3.5.2.[30] P-values were evaluated at a 2-sided significance level of 0.05.

Results

Study Population

The PREMIER and TRIUMPH studies enrolled a total of 2,498 (between 1/1/2003-6/28/2004) and 4,340 patients (between 4/11/2005-12/31/2008), respectively. Some patients (n=1,198) could not be linked to EPA air quality data and the final study cohort for assessing 5-year mortality was 5,650. After excluding patients who did not have complete health status assessments due to death or loss to follow-up (n=1,727), our final study cohort for assessing 1-year health status was 3,913 patients (Supplemental Figure 1). Supplementary tables 1 and 2 compare the differences in patient characteristics between patients who were excluded and those who were not. There were significant differences in age, race, socioeconomic status and burden of comorbidities. Patients who were excluded were younger, had worse SES across all 4 SES variables that were assessed and a higher proportion had HTN, DM, CKD and heart failure. We adjusted for all these factors in our outcome analysis. Additionally patients who were excluded had on average lower peak troponin levels, lower GRACE mortality scores and a lower proportion presented with ST elevation myocardial infarction.

The mean age of the final analytical cohort was 60.6 ± 12.2, 33% were females and 25% were non-Caucasian. Comorbidities were common, with 64% of the patients having hypertension, 28% having diabetes and 40% having systolic dysfunction (EF < 50%). Fifteen percent of the patients were uninsured and 16% reported not having enough money at the end of the month to make ends meet. The mean 12-month average $PM_{2.5}$ and ozone exposure per patient were 11.96 ± 2.11 $\mu g/m^3$ (range = 4.3-20.5) and 0.0383 ± 0.0035 ppm

(range= 0.0267-0.0534). There was no significant correlation between average $PM_{2.5}$ exposure and average ozone exposure (Spearman r = −0.02, p=0.15).

Table 1 describes the baseline demographic and clinical characteristics, stratified according to quartiles of $PM_{2.5}$ concentration. Patients in higher quartiles of $PM_{2.5}$ concentration had lower Global Registry of Acute Coronary Events (GRACE) scores, lower peak troponin level, were more likely to avoid care due to costs, and were more likely to have HTN, prior MI, DM, heart failure, CKD, and chronic lung disease.

Supplementary table 3 describes the baseline demographics and clinical characteristics of patients stratified according to quartiles of ozone exposure. There were significant differences in the prevalence of diabetes, heart failure and chronic kidney disease in groups stratified by ozone exposure.

Association between $PM_{2.5}$ and Ozone with Health Status

The mean SAQ SS at 1 year was 89.2 ± 15.7. The mean dyspnea score was 0.80 ± 1.08 and the mean SF-12 PCS score was 44.1 ±11.8 respectively. Table 2 compares health status scores at one year in patients according to quartiles of $PM_{2.5}$ exposure. Patients with higher exposure to $PM_{2.5}$ had worse health status scores.

In unadjusted analysis (Model 1), there was a significant association of higher $PM_{2.5}$ concentration and ozone level with worse generic and disease-specific health status (Figure 1). Adjusting for demographics, smoking, SES and date of enrollment (Model 2) did not attenuate these associations. Even in Model 3, which additionally adjusted for comorbidities associated with air pollution, there remained a significant association with worse SAQ SS, dyspnea and SF-12 PCS scores. No significant nonlinearity was found for the effects of either $PM_{2.5}$ or ozone on any of the health status outcomes (p>0.1 for all), so all associations are summarized as linear effects. Furthermore, higher $PM_{2.5}$ and ozone concentration was significantly associated with worse recovery (i.e., change) in health status 1-year after an MI (Figure 2).

To assess whether ozone levels moderated the association of $PM_{2.5}$ and health status, we modified Model 2 to include an interaction between $PM_{2.5}$ and ozone. Although there was no significant interaction for disease specific health status (SAQ SS) and SF-12 PCS scores, higher ozone levels were associated with worse dyspnea for the same $PM_{2.5}$ concentration (Supplemental Figure 2).

Association between PM2.5 and Ozone with 5-year All-cause Mortality

There was no loss to follow-up in the ascertainment of all-cause mortality at 5 years outcome. Higher quartiles (Q) of $PM_{2.5}$ exposure in the year leading up to the MI were associated with lower crude 5-year survival (Q1: 85.2%, Q2: 82.1%, Q3: 76.8%, Q4: 73.1%; p<0.001). The association with ozone levels, while nominally significant in unadjusted analyses, was weaker (Q1: 79.7%, Q2: 80.7%, Q3: 79.6%, Q4: 77.1%; p=0.03) (Supplemental Figure 3). In Cox regression analysis adjusting for patient factors, higher $PM_{2.5}$ exposure was associated with greater mortality risk (HR = 1.13 per +1 SD, 95% CI = 1.07-1.20, p<0.001), while ozone level was not (HR = 1.01 per +1 SD, 95% CI = 0.96-1.06,

p=0.67). The proportional hazards assumption was satisfied for both parameters (p>0.3), and no evidence of nonlinearity was found (p=0.59).

Discussion

US policy on air pollution standards is in flux. Recent changes to the regulation of air pollution seeks to promote fossil fuels and abandon prior efforts to curb greenhouse gas emissions. This has raised concerns regarding the deleterious health effects of air pollutants such as $PM_{2.5}$ and ozone.[31] In this first analysis of the association of long-term exposure to $PM_{2.5}$ and ozone with health status and all-cause mortality after MI, we found small but statistically significant linear associations of higher long-term exposure to ambient air $PM_{2.5}$ and ozone with worse disease-specific and generic health status in patients with MI, as well as increased risks of 5-year mortality with higher exposures to $PM_{2.5}$ particles.

Previous studies have shown an association of increased long-term exposure to $PM_{2.5}$ with higher all-cause mortality[11] and increased cardiovascular events[32]. In patients with MI, long term exposure to $PM_{2.5}$ was associated with higher cardiovascular mortality.[33] Although some prior studies did not show an association of ozone with cardiovascular outcomes independent of $PM_{2.5}$ exposure[9], studies using more precise estimates of ozone exposure, independent of $PM_{2.5}$, show ozone to also be associated with cardiovascular mortality.[13] Our study confirms findings from previous studies regarding association of higher $PM_{2.5}$ exposure with mortality and adds to the evaluation of long term $PM_{2.5}$ and ozone exposure with cardiovascular outcomes by demonstrating that both $PM_{2.5}$ and ozone are independently associated with poorer 1-year health status after MI. Moreover, finding similar associations with $PM_{2.5}$ and post-MI mortality with previously reported studies (and the increased prevalence of comorbidities associated with air pollution in our study) provide external validity of our novel results regarding worse health status with higher air pollution levels.

Health status outcomes directly assess the impact of disease on patient's symptoms, function and QoL. In patients with cardiovascular disease, optimizing health status and disease-specific QoL have been recognized as important goals of treatment.[34] In addition to defining treatment factors that influence health status, it is important to consider other risk factors that may negatively affect patients' health status. Our analysis identifies $PM_{2.5}$ and ozone exposure as two such factors that negatively affect long-term health status of patients with cardiovascular disease. The mechanisms underlying the association of ozone and $PM_{2.5}$ with poorer health status are likely to be complex. There is abundant pathophysiologic evidence supporting the development and progression of coronary disease with air pollution. Long term exposure of $PM_{2.5}$ has been shown to be associated with oxidative stress[35] endothelial dysfunction[36] and increased propensity to coagulation.[37] These processes are thought to drive the association of exposure to $PM_{2.5}$ and development/progression of cardiovascular disease. Additionally, exposure to $PM_{2.5}$ is strongly associated with several risk factors for cardiovascular disease including HTN[38], DM[39] dyslipidemia[40] , CKD[41], obesity[42] and breathing disorders[43]. Ozone has also been shown to enhance the toxic effects of $PM_{2.5}$[29, 44] and could have independent effects contributing to adverse cardiovascular outcomes.[45, 46]

This complex interplay and biochemical relationships could underlie the observed associations between these air pollutants with health status and mortality after MI.

The observed effect sizes of greater air pollutant exposure to health status are small, although statistically significant. However, it is important to consider the results of our study in the context of the range of $PM_{2.5}$ and ozone exposure throughout the US and the world. While in our study the range for $PM_{2.5}$ was 4.3-20.5μg/m^3, in the US the range of average exposure to $PM_{2.5}$ is estimated to range from 5-50μg/m^3.[47] Worldwide, the range is even higher, with levels greater than 100μg/m^3 in some developing countries.[8] As different metrics for ozone concentration are used worldwide, a global comparison is difficult to make. However it is generally thought that ozone levels measured by daily average of 8-hour maximum vary considerably worldwide.[17] Given that we found a linear relationship with both health status and mortality (for $PM_{2.5}$), the true impact of both ozone and $PM_{2.5}$, could be even greater in patients exposed to higher concentrations of these air pollutants. The Clean Air Act[48] requires the EPA to set National Ambient Air Quality Standards, and the primary standard for average (per year) concentration of $PM_{2.5}$ has been set at 12μg/m^3 and 0.070ppm for ozone.[8] We observed a significant impact on mortality and health status at concentrations below these standards.

Our findings should be interpreted in the context of the following potential limitations. This study focused on the impact of ambient air $PM_{2.5}$ and ozone exposure on health status and mortality in patients after an MI and the primary exposure metric only focused on daily outdoor concentrations. Patients spend their time in various microenvironments, for example in their homes, offices, shopping malls, and could also be exposed to second-hand smoke. As there is likely to be a lot of variability in air pollutant concentrations in these different environments, it is possible that the total exposure of each patient to could have been misclassified, which would have biased our findings to the null. However, outdoor $PM_{2.5}$ and ozone levels have been shown to be strong proxies for total exposure for an individual patient.[49, 50] A second concern is that although we adjusted extensively for comorbid conditions, there is the possibility of residual confounding in this observational study. For example, we did not adjust for recruitment site, as there was a strong correlation between both $PM_{2.5}$ and ozone exposure and recruitment site (intraclass correlation coefficient (ICC) 79% for both), which is not unexpected. Nevertheless, there is the possibility that site-specific factors could have influenced the results, although the intra-class correlations between health status and site were much smaller (range 3-6%) than for air pollutant exposure (ICC = 79%). Importantly, even after adjusting for comorbidities that may be along the causal pathway between air pollution and cardiovascular outcomes, a significant association between $PM_{2.5}$ and ozone remained. Finally a number of patients were excluded as they had missing 1-year health status data or could not be linked to EPA air quality data, making it possible that our results could have been slightly different if we had complete data for all patients enrolled. However, it seems unlikely that patients with missing data would exhibit a different association between exposure and health status than those with complete data, particularly after adjustment for other factors.

Conclusions

We found a strong association of high long-term exposure to air pollutants with patients' 1-year health status and 5-year all-cause mortality after MI. These findings suggest that greater air pollution not only increases patients' risks for dying, but also worsens their symptoms, function and QoL. While further studies are needed to determine the mechanism of this increased risk, ongoing debates about the regulation of air pollution should consider the impact of higher exposure to $PM_{2.5}$ and ozone on patients' health status.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

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WHAT IS KNOWN?

- Long-term exposure to ambient air particulate matter <2.5μm ($PM_{2.5}$) and ozone has been associated with development and progression of coronary artery disease (CAD) and, in the case of $PM_{2.5}$, higher long-term mortality risk after an acute myocardial infarction (MI).

WHAT THE STUDY ADDS

- In patients with CAD 1 year after an MI, we found higher exposure to both $PM_{2.5}$ and ozone to be modestly but independently associated with poorer generic and disease-specific health status.
- These associations were not attenuated after adjusting for demographics, socioeconomic status and prevalence of comorbidities.
- The associations were also preserved after adjusting for baseline health status, indicating that poor air quality was also associated with recovery of health status following an MI.
- Similar to previous studies, higher exposure to $PM_{2.5}$ was associated with higher risk of mortality over 5 years after MI.

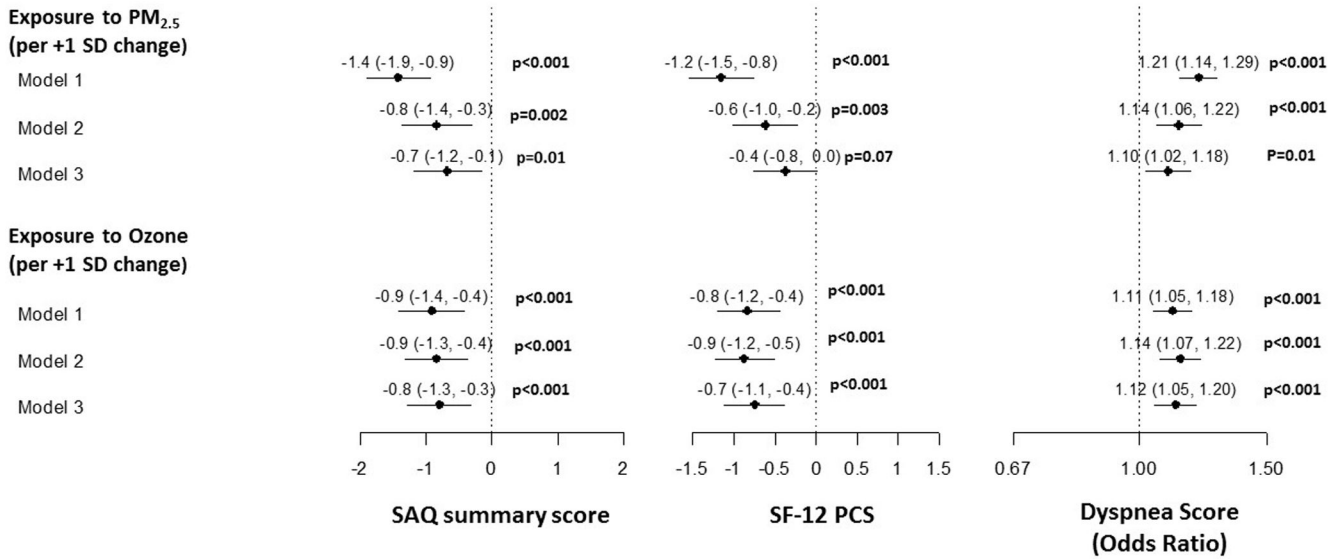

Figure 1.

Association of $PM_{2.5}$ and Ozone exposure with health status one year after MI.

MI= Myocardial Infarction, $PM_{2.5}$= Particulate Matter < 2.5μm

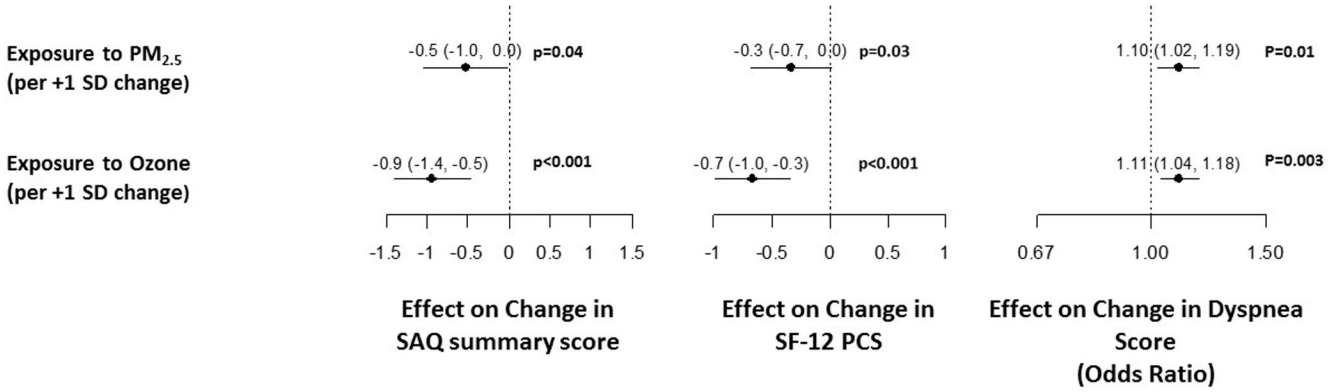

Figure 2.

Association of $PM_{2.5}$ and Ozone exposure with change in health status one year after MI

MI= Myocardial Infarction, $PM_{2.5}$= Particulate Matter < 2.5μm

Table 1.

Baseline demographic characteristics of patients compared according to exposure to $PM_{2.5}$ over 1 year after myocardial infarction.

	$PM_{2.5}$ Concentration (μg/m3)				
	Quartile 1 (4.3 to 10.6) n = 978	**Quartile 2 (10.7 to 12.0) n = 978**	**Quartile 3 (12.1 to 13.2) n = 978**	**Quartile 4 (13.3 to 20.5) n = 979**	**p-value**
Demographics					
Age (Mean ± SD)	60.4 ± 11.7	60.8 ± 12.1	60.4 ± 12.4	60.5 ± 12.7	0.860
Female	322 (32.9%)	271 (27.7%)	315 (32.2%)	372 (38.0%)	< 0.001
Caucasian Race	826 (84.7%)	841 (86.3%)	740 (75.9%)	518 (53.1%)	< 0.001
Ozone exposure (ppm) (Mean ± SD)	0.0384 ± 0.0036	0.0385 ± 0.0027	0.0382 ± 0.0036	0.0382 ± 0.0039	0.140
Socioeconomic Status					
High school education	843 (86.4%)	818 (84.0%)	798 (82.4%)	715 (74.4%)	< 0.001
Uninsured	98 (10.4%)	159 (16.5%)	135 (14.3%)	199 (21.2%)	< 0.001
Avoiding care due to cost	198 (20.4%)	198 (20.5%)	180 (18.8%)	205 (21.7%)	0.470
Not enough money left at end of the month	129 (13.4%)	34 (14.1%)	145 (15.3%)	188 (19.8%)	< 0.001
Comorbidities					
Current Smoker	168 (17.6%)	176 (18.3%)	192 (20.5%)	202 (21.8%)	0.08
Hypertension	594 (60.7%)	601 (61.5%)	613 (62.7%)	711 (72.6%)	< 0.001
Prior MI	173 (17.7%)	173 (17.7%)	197 (20.1%)	210 (21.5%)	0.085
Prior PCI	137 (14.0%)	182 (18.6%)	215 (22.0%)	173 (17.7%)	< 0.001
Prior CABG	93 (9.5%)	115 (11.8%)	126 (12.9%)	132 (13.5%)	0.035
Diabetes	238 (24.3%)	251 (25.7%)	291 (29.8%)	326 (33.3%)	< 0.001
Heart failure	51 (5.2%)	60 (6.1%)	55 (5.6%)	125 (12.8%)	< 0.001
Lung disease	63 (6.4%)	76 (7.8%)	91 (9.3%)	102 (10.4%)	0.009
CKD	46 (4.7%)	51 (5.2%)	59 (6.0%)	112 (11.4%)	< 0.001
MI presentation					
STEMI	462 (47.2%)	496 (50.7%)	470 (48.1%)	346 (35.3%)	< 0.001
Normal LV function	622 (63.6%)	556 (57.0%)	580 (59.4%)	585 (59.9%)	< 0.001
Peak troponin (Median (IQR))	7.6 (1.6, 31.6)	9.7 (2.3, 50.1)	8.7 (2.1, 37.7)	4.1 (0.9, 16.1)	< 0.001
GRACE score (Median (IQR))	7.6 (1.6, 31.6)	9.7 (2.3, 50.1)	8.7 (2.1, 37.7)	4.1 (0.9, 16.1)	< 0.001

CABG= Coronary Artery Bypass Graft surgery, CKD= Chronic Kidney Disease, MI= Myocardial Infarction, PCI= Percutaneous Coronary Intervention

Table 2.

Health status of patients at baseline and one year after MI compared according to exposure to $PM_{2.5}$ over 1 year after myocardial infarction.

	PM2.5 Concentration (μg/m^3)				
	Quartile 1 (4.3 to 10.6) n = 978	**Quartile 2 (10.7 to 12.0) n = 978**	**Quartile 3 (12.1 to 13.2) n = 978**	**Quartile 4 (13.3 to 20.5) n = 979**	**p-value**
Seattle Angina Questionnaire Summary Score					
Baseline	79.3 ± 17.2	78.6 ± 16.0	79.0 ± 17.3	75.4 ± 19.0	< 0.001
1-year	90.3 ± 14.5	90.2 ± 14.3	89.2 ± 15.4	87.1 ± 18.2	< 0.001
Dyspnea Score					
Baseline	1.02 ± 1.08	0.94 ± 1.07	0.93 ± 1.09	1.21 ± 1.23	< 0.001
1-year	0.71 ± 1.03	0.75 ± 1.07	0.79 ± 1.06	0.97 ± 1.15	< 0.001
SF-12 PCS					
Baseline	43.9 ± 12.1	44.0 ± 12.0	43.8 ± 12.0	41.9 ± 12.6	< 0.001
1-year	45.1 ± 11.7	44.6 ± 11.5	44.3 ± 11.8	42.2 ± 12.1	< 0.001

SF-12 PCS=Physical Component Summary of the Medical Outcomes Study 12-item Short Form