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# Short-term Effects of Particulate Matter Gamma Radiation on:

## Oxidative Stress Biomarkers in COPD Patients

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## Abstract

Inhalation of particulate matter (PM) radioactivity is an important pathway of ionizing radiation exposure. We investigated the associations between short-term exposures to PM gamma radioactivity with oxidative stress in COPD patients. Urinary concentrations of 8-hydroxy-2'-deoxyguanosine (8-OHdG) and malondialdehyde (MDA) of 81 COPD patients from Eastern Massachusetts were measured 1-4 times during 2012-2014. Daily ambient and indoor PM gamma activities (gamma-3 through gamma-9) were calculated based on EPA RadNet data and indoor-outdoor infiltration ratios. Linear mixed effects models were used to examine the associations between biomarkers with PM gamma activities for moving averages from urine collection day to 7 days before. Our results indicate that ambient and indoor PM gamma activities were positively associated with 8-OHdG, with stronger effects for exposure windows closer to urine collection day. For per IQR increase in indoor PM gamma activities averaged over urine collection day and 1 day before, 8-OHdG increased from 3.41% (95%CI: −0.88, 7.88) to 8.87% (95%CI: 2.98, 15.1), adjusted for indoor black carbon. For MDA, the timing of greatest effects across the exposure week varied but was nearly all positive. These findings provide insight into the toxigenic properties associated with PM radioactivity and suggest that these exposures promote systemic oxidative stress.

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The authors declare no conflict of interests, including relevant financial interests, activities, relationships, and affiliations.

**Keywords**

exposure; radiation; particle gamma radioactivity; oxidative stress; chronic obstructive pulmonary disease (COPD)

## Introduction

Chronic obstructive pulmonary disease (COPD) is an irreversible chronic inflammatory lung disease and is one of the leading causes of morbidity and mortality worldwide.[1] In 2015, 174 million people had COPD[2] and 3.2 million people died from COPD[3], while in the U.S., COPD was the 4th most common cause of death in 2016[4]. Oxidative stress may promote more severe disease when free radicals react with biomolecules (e.g., DNA and lipids).[5,6] Oxidative stress biomarkers increase during COPD exacerbations, which also contribute to COPD-related morbidity and mortality.[7-9]

Exposure to particulate matter (PM) and its components has been linked to increases in oxidative stress biomarkers in the general population, including COPD patients, who have a higher vulnerability to air pollution exposure.[10-16] As COPD patients spend most of their time indoors, exposures to indoor pollutants may be especially important for their health. Indoor pollution exposure has been shown to be responsible for over one-third of premature COPD deaths in adults in low and middle-income countries.[17] We have previously demonstrated that greater exposure to indoor black carbon (BC) in PM was associated with an increase of oxidative stress biomarkers in COPD patients.[16]

One hypothesized mechanism that contributes to the adverse effects of PM involves natural radionuclides that attach to airborne particles. Environmental radioactive nuclei from background sources, i.e., natural terrestrial and extra-terrestrial, can attach to respirable particulate matter (PM).[18] PM radioactivity can be carried into the human body by inhalation.[18,19] Previous studies have found associations between PM radioactivity with biomarkers of increased systemic inflammation in a general population cohort[20] and increased blood pressure and reduced pulmonary function in a cohort of elderly men.[21-22] As COPD patients are more vulnerable to air pollution exposure,[15,23] it is possible that PM associated radioactivity, even with short-term ambient and indoor exposures, is associated with increases in oxidative stress in COPD patients. However, no study has linked these exposures to oxidative stress in COPD patients.

The aim of this study is to investigate the association between exposures to PM radioactivity and biomarkers of oxidative stress in COPD patients. We investigated the associations between short-term (up to one week) exposures to PM gamma activities (surrogates of PM radioactivity), including ambient and indoor exposures, with urinary oxidative stress biomarkers, 8-hydroxy-2'-deoxyguanosine (8-OHdG) and malondialdehyde (MDA), in 81 COPD patients from Eastern Massachusetts, USA. Previously in this cohort we reported associations between exposures to gamma PM radioactivity with increases in systemic inflammation and decreases in pulmonary function assessed up to one week after exposures.[18,24]

# Methods

## Study population

Participants were enrolled in the COPD and Air Pollution Study.[16,25] We recruited participants at the VA Boston Healthcare System between November 2012 and December 2014. We identified potential participants by reviewing medical records of VA Boston pulmonary, primary care, and pulmonary function clinic encounters, with ICD-9 codes 490–493 and 496. We included participants if they were at least 40 years old, former smokers with a smoking history of at least 10 pack years, had physician-diagnosed COPD, and had a ratio of forced expiratory volume in one second (FEV1) over forced vital capacity (FVC) on post-bronchodilator spirometry less than 0.70 or emphysema on a clinical CT scan report. We excluded individuals if they had a history of any malignancies other than stable skin cancer or prostate cancer; were smokers or lived with smokers; had important indoor sources such as wood stove, fireplace or candles at the time of study entry. Each participant attended clinic visits 1-4 times per year (roughly three months apart) for urine collection and completed questionnaires about demographics, lifestyle factors, health history and home characteristics.

Our study protocol was approved by Institutional Review Boards at VA Boston and Harvard Medical School. We obtained informed consent from all participants prior to study procedures.

## Urinary oxidative stress biomarkers

Urine samples were analysed for 8-OHdG, which represents the oxidative damage of DNA; and MDA, which represents the oxidative degradation of lipids. There are two forms of MDA: total and free MDA. Free MDA is unbound to proteins, while total MDA includes both the unbound and bound forms. As free MDA is likely explained by the binding of MDA to protein molecules in the urine, total urinary MDA may be a better indicator of lipid peroxidation.[16,26] Therefore, we used total MDA in the urine to represent lipid peroxidation in the current study.

Urinary concentration of 8-OHdG was measured using a HPLC-ESI-MS system.[27] MDA was measured using a HPLC system with fluorescent detection, with the exception of an added alkaline hydrolysis step prior to sample extraction procedures.[28] Details regarding analyses have been published elsewhere.[16]

## Assessment of PM gamma activities

Hourly ambient PM gamma activities of different energy ranges (101-2200 keV) were obtained in counts per minute (CPM) from the US Environmental Protection Agency (EPA) RadNet website.[29,30] The energy ranges are represented as separate gamma channels as shown in Table S1 in the Supplementary Information. Each EPA air radiation monitoring station is equipped with a Total Suspended Particle (TSP) high volume air sampler. The gamma radiation detector is a NaI(Tl) scintillation crystal, sampling at $60 m^3$/hr and positioned above the TSP filter and connected to a 1,024-channel multi-channel analyzer and a local processing unit. The hourly PM gamma activity data (from 0:00 to 23:00) were

averaged into daily measurements for each channel. We did not include gamma2 in our analysis as over 25% of the data were missing.

The gamma spectrometer from RadNet stationary air monitors are not well-shielded, therefore, measurement of gamma activities may be influenced by background terrestrial and cosmic radiation, which exhibit a small seasonal variability.[31] In order to estimate the PM associated gamma activities (PM gamma activities), we subtracted from the RadNet daily gamma activities an average baseline signal that represents background radiation. We assessed different averaging periods that included 7, 21, 28 and 90 days before measurement day for the background levels. Finally, we selected the average of the previous 21 days to represent the background level, as corrected ambient PM gamma activities using this averaging period had the best correlation between ambient $PM_{2.5}$ mass exposures ($p<0.05$).[24] Fig. 1 shows the variability of the RadNet and corrected PM gamma-9 activity radiation. Similar plots for the other PM gamma activities (gamma-3 to gamma-8) are shown in Fig. S1 in Supplementary Information.

## Assessment of ambient and indoor exposures to other pollutants

Prior to each clinic visit, we collected weekly indoor PM samples in the main activity room of each home, using the Harvard T.H. Chan School of Public Health (HSPH) Micro-environmental Automated Particle Sampler (APS).[32] Ambient $PM_{2.5}$ samples were collected daily at the Harvard Supersite located on the roof of the Harvard Medical School Library (Boston, MA). Filters were analysed using an electronic microbalance (Micro-Gravimetric M5, Mettler Instruments Corp, Hightstown, NJ) for indoor and ambient $PM_{2.5}$ mass concentrations. Subsequently, a smoke stain reflectometer (model EEL m43d, Diffusion System Ltd., United Kingdom) for indoor BC concentrations and an aethalometer (Magee Scientific Company, model AE-16, Berkeley, CA) for ambient BC concentrations.[33,34] Indoor nitrogen dioxide ($NO_2$) was collected using Ogawa passive sampling badges clipped to the HSPH Micro-environmental APS, and then extracted from the filters and analyzed using ion chromatography.[35] Ambient $NO_2$ and $O_3$ concentrations were obtained from US EPA regulatory Boston-area monitoring stations and the adjusted mean values calculated.[36] We calculated the weekly ambient exposures concurrent with the indoor weekly exposures by averaging daily ambient concentrations. We used the indoor-to-outdoor sulfur (S) ratio measured in indoor and outdoor $PM_{2.5}$ filter samples[37] as proxy for the $PM_{2.5}$ infiltration ratio. Previous studies have shown that the greatest activity fraction of ambient radioactive aerosols is in the accumulation mode, which includes particle sizes between 0.1 and 2.5 μm.[38,39] Therefore, in the absence of major indoor combustion sources, indoor PM gamma activities can be estimated by multiplying the ambient PM gamma measurements by indoor-outdoor sulfur ratio for each home. For the indoor analysis we excluded observations with sulfur ratios greater than 1.1, as this was an indicator of indoor sources.[32] To examine patterns of effects over the week before the clinic visit, we constructed different exposure windows averaged from the day of urine collection (day 0) up to seven days before urine collection (day7) for ambient and indoor PM-gamma activities

## Covariates

We *a* priori included variables known to be associated with oxidative stress or PM gamma activities in our model as potential confounders or precision variables. These variables included demographics, such as age and race; health information, such as heart disease, diabetes, and body mass index (BMI); and meteorological parameters, such as temperature, humidity, season and time of the day for urine collection. We also adjusted for urine dilution using urinary creatinine concentration.

Information on demographics and health conditions were obtained from questionnaires. Age was calculated by subtracting date of birth from date of visit, and BMI was calculated using height and weight measurements taken at the visit. Daily ambient temperature at each home was estimated using an exposure model based on satellite remote sensing, land use, and ground level temperature data.[40] Daily ambient relative humidity was obtained from measurements at the Boston Logan International Airport weather station.[34] Season was categorized into winter (December, January, February), spring (March, April, May), summer (June, July, August), or fall (September, October, November) based on the month of the clinic visit. Urinary creatinine was measured by a colorimetric method.[28] As we previously found positive associations between indoor BC and oxidative stress biomarkers in the same cohort,[16] we adjusted for weekly ambient BC and indoor BC exposures for the ambient and indoor gamma models, respectively. In order to consider the possible confounding effects of other pollutants, we also performed sensitivity analyses by adjusting the indoor and ambient PM gamma radioactivity models for indoor weekly and corresponding ambient exposures to other pollutants. Specifically, we adjusted for indoor $PM_{2.5}$ and $NO_2$ in the regression models that included indoor PM gamma radioactivity and adjusted for ambient $PM_{2.5,}$ $NO_2$, and $O_3$ in the regression models that included ambient PM gamma radioactivity. We also assessed models unadjusted for any other pollutants for comparison.

## Statistical methods

We fit linear mixed-effects models (using the “lme4” package in R) with random intercepts for each participant to account for the correlation among repeated observation within individuals[41] to examine the associations between exposures to each of the ambient and indoor PM gamma activities (gamma-3 through gamma-9) in different exposure windows with oxidative stress biomarkers. Each biomarker was natural log-transformed to meet the linear model assumption, i.e., an approximate normal distribution for residuals. The results are presented as percent changes of each biomarker per interquartile range (IQR) increase in exposure to each of the PM gamma activities.

We examined that we had enough sample size for our linear mixed-effects models based on power-calculation for standard design (using package “sjstats” in R). The validity of model assumptions was confirmed by examination of model residuals. We consider results with $p < 0.05$ as statistically significant. All statistical analyses were performed using R software.

## Results and Discussion

### Characteristics of participants and exposures

A flow chart of participants in our cohort is shown in Fig. S2 in Supplementary Information. We included data from 81 participants with 231 clinic visits, including 28 participants followed for 4 clinic visits, 22 with 3 clinic visits, 22 with 2 clinic visits, and 9 with 1 clinic visit. Clinic visits were conducted evenly throughout the seasons. Characteristics of the 81 study participants as well as levels of biomarkers of oxidative stress are shown in Table 1. All participants were male, and 89% were white, with an average age of 72.7 years old. Furthermore, 45.2% of the subjects were obese (BMI ≥ 30 $kg/m^2$), 24.4% had healthcare provider-diagnosed diabetes, and 50% had heart disease requiring treatment in the past 10 years.

Table 2 presents the distributions of the ambient and indoor daily PM-gamma activities, ambient temperature and relative humidity on the urine collection day, and ambient and indoor weekly BC concentrations before urine collection. The estimated PM gamma activities have both negative and positive values, as they are relative estimates of daily fluctuations from average background exposures. The distributions of ambient and indoor PM gamma activities for each of the different moving averages are shown in Tables S2 and S3.

The number of observations included in each analysis varied depending on the availability of the exposures, with a maximum number of 231 for ambient models and up to 222 for indoor models (Supplemental Tables S1 and S2).

### Associations between PM gamma activities exposure and biomarkers of oxidative stress

We assessed the associations between ambient and indoor PM gamma activities starting with the urine collection day through 7 days before. Figs. 2 and 3 show the associations between ambient and indoor PM gamma activities with 8-OHdG and MDA, expressed in percent increases in each biomarker per IQR of exposure, adjusted for black carbon exposure. The values of percent increase with per IQR PM gamma activities across all channels, and the *p*-values are summarized in Table S4 and S5 in Supplementary Information.

We observed positive associations between 8-OHdG with ambient and indoor PM gamma activities (Fig. 2a and 2b) with stronger associations for shorter exposure windows. The increases in 8-OHdG with per IQR indoor PM gamma activities were slightly higher than those with ambient exposure, with the strongest associations and greatest effects for the moving average starting 1 or 2 days before urine collection across all gamma channels. For example, for the moving average starting from 1 day before urine collection: percent increases with per IQR increase of PM gamma activities ranged from 2.09% (95%CI: −0.79, 5.05) to 5.65% (95%CI: 2.08, 9.34) for the ambient, and from 3.41% (95CI: −0.88, 7.88) to 8.87% (95%CI: 2.98, 15.1) for the indoor.

The associations between MDA with ambient and indoor PM gamma activities were not as consistent as 8-OHdG. However, the associations across each gamma channel were all generally positive (Fig. 3a and 3b). For moving averages starting from 1 day before urine

collection, the percent changes in MDA ranged from −0.37% (95%CI: −5.47, 5.00) to 1.68% (95%CI: −3.88, 7.57) per IQR increases of ambient PM activities. For moving averages of indoor PM gamma activities starting from 1 day before urine collection percent increases in MDA ranged from 0.23% (95%CI: −8.97, 10.4) to 5.89% (95%CI: −4.64, 17.6) with per IQR across gamma channels 3 through 9.

We also explored the effects of other pollutants, including ambient $PM_{2.5}$, $NO_2$ and $O_3$, and indoor $PM_{2.5}$ and $NO_2$ as potential confounders in the models for ambient and indoor gamma activities, respectively. The results of these models (Fig. S3-S8 in the Supplementary Information) were similar to the results of models adjusted for ambient and indoor BC (Fig. 2 and 3). The results of models unadjusted for other pollutants (Fig. S9-S10 in the Supplementary Information) were similar to the adjusted models.

### Effects of BC, $PM_{2.5}$, $NO_2$ and $O_3$

For models including ambient PM gamma activities, the effect of ambient BC on 8-OHdG ranged from 5.86% (95CI%: −1.20, 13.4) to 8.16% (95CI%: 1.02, 15.8) increase per IQR (Fig. S11(a), Supplementary Information). For models including indoor PM gamma activity, the effects of indoor BC on 8-OHdG ranged from 1.38% (95CI%: −3.78, 6.81) to 3.18% (95CI%: −2.01, 8.65) increase per IQR, depending on the specific gamma channel (Fig. S11(b), Supplementary Information). For MDA, the effect of ambient BC ranged from 5.27% (95%CI: −6.05, 17.9) to 6.64% (95%CI: −4.70, 19.3) increase per IQR and 3.10% (95%CI: −3,64 10.3) to 3.90% (95%CI: −2.82, 11.1) for indoor BC exposures (Fig. S12, Supplementary Information). These results are similar to our previous findings regarding indoor BC.[16]

The associations between ambient $PM_{2.5}$, $NO_2$ and $O_3$, and indoor $PM_{2.5}$ and $NO_2$ with 8-OHdG and MDA are shown in Fig. S13-S18. Although not statistically significant, we found positive associations between ambient $PM_{2.5}$ (Fig. S13(a)) and ambient $NO_2$ (Fig. S15(a)) with 8-OHdG, but not with indoor $PM_{2.5}$ (Fig. S13(b)), indoor $NO_2$ (Fig. S15(b)) or ambient $O_3$ (Fig. S17). There were also positive associations between indoor $PM_{2.5}$ (Fig. S14(b)) and ambient $NO_2$ (Fig. S16(a)) with MDA, but not with ambient $PM_{2.5}$ (Fig. S14(a)) indoor $NO_2$ (Fig. S16(b)), nor ambient $O_3$ (Fig. S18). The reasons for the differences in $PM_{2.5}$, $NO_2$ or $O_3$ on MDA and 8-OHdG are uncertain.

## Discussion

In this study, we observed positive associations between PM gamma activities and biomarkers of oxidative stress. Specifically, we found positive associations between both ambient and indoor PM gamma activities with 8-OHdG (a biomarker of DNA oxidation), with slightly higher effects for indoor exposures; and more consistent positive associations between MDA (biomarker of lipid oxidation) with indoor PM gamma activities than the associations with ambient PM gamma activities. The more consistent associations with 8-OHdG than those with MDA suggest that exposure to PM radioactivity maybe more likely to cause oxidative damage to DNA than to lipids. The increases in biomarkers with the increase indoor PM gamma activities suggest that there are effects of ambient exposures infiltrating the homes of COPD patients. As these COPD patients spend most of their time indoors, our

results suggest that staying indoors are not protective of the effects of ambient PM gamma activities. We adjusted for ambient and indoor weekly BC, and found positive effects of BC, consistent with the effects of BC in our previous study. We also adjusted for $PM_{2.5}$, $NO_2$ and $O_3$ respectively, and found the results of PM gamma activities were similar to models adjusted for BC and unadjusted models.

To the best of our knowledge, this is the first study investigating the short-term effects of exposures to PM radioactivity on oxidative stress biomarkers in COPD patients. Exposure to ionizing radiation has been found to be associated with an increase in oxidative stress.[42,43] In the only other study to date of the impacts of PM radioactivity on oxidative stress, there was no association between PM gross beta radioactivity with plasma 8-epi-prostaglandin F2α or myeloperoxidase (MPO).[20] This may be due to differences in the populations of these two studies; our study was composed of males with COPD who were receiving treatment at VA Boston, while the other analysis was conducted among male and female volunteers from the Framingham Heart Study who may have been less susceptible to adverse effects. In addition, other studies also suggested systemic effects of exposures to PM radioactivity. These studies include report of a positive relationship between PM radiation and diastolic and systolic blood pressure in elderly men,[21] as well as the association between exposure to PM radiation and decrease in lung function[22]. The above studies[20-22] used PM gross beta activity as the surrogate of PM radioactivity. However, as gamma radiation is mostly emitted after the emission of alpha or beta radiation, it is possible that PM gamma activities are better surrogates for the sum of environmental PM alpha and beta activities.[44,24] Huang et al.[18] and Vieira et al.[24] used PM gamma activities as surrogates of PM radioactivity and found that ambient and indoor PM gamma activities were associated with increases in plasma inflammation biomarkers and decreases in pulmonary function ($FEV_1$ and FVC) in the same COPD cohort as in this report. All these previous studies indicate potential adverse health effects of PM associated radioactivity and support our findings.

There are some limitations in this study. One limitation is that we did not measure PM gamma activities directly in the indoor environment. As we estimated indoor PM gamma activities using infiltration ratio (sulfur ratio) without considering the contribution of indoor radiation sources (such as radon) we may underestimate the full burden of indoor exposures. Second, our study participants were all white males living in the Northeastern US, which may restrict the generalizability of the study if the mechanisms or exposure levels are different in individuals of other ages, races, or in females. Third, our study only included participants who were able to travel to VA Boston for a study visit and were able to receive and ship samplers back to the field team. Thus, or results may not be generalizable to individuals with very severe COPD who may have greater systemic response to PM gamma activities.

Our study also has several strengths. To the best of our knowledge, we ae the first to describe the effects of previously unrecognized exposures to PM radioactivity on oxidative stress biomarkers in COPD patients. Additionally, our study considers exposures to PM gamma activities in indoor environment where COPD patients spend most of their time. Finally, all participants in our study have well documented COPD.

## Conclusion

Our results indicated positive associations between ambient and indoor PM gamma activities (surrogates of PM radioactivity) with urinary oxidative stress biomarkers. Our findings suggest that PM with attached radionuclides may promote the oxidative degradation of DNA and lipids in COPD patients and contributes to understanding effects of low-level ambient radiation.

## Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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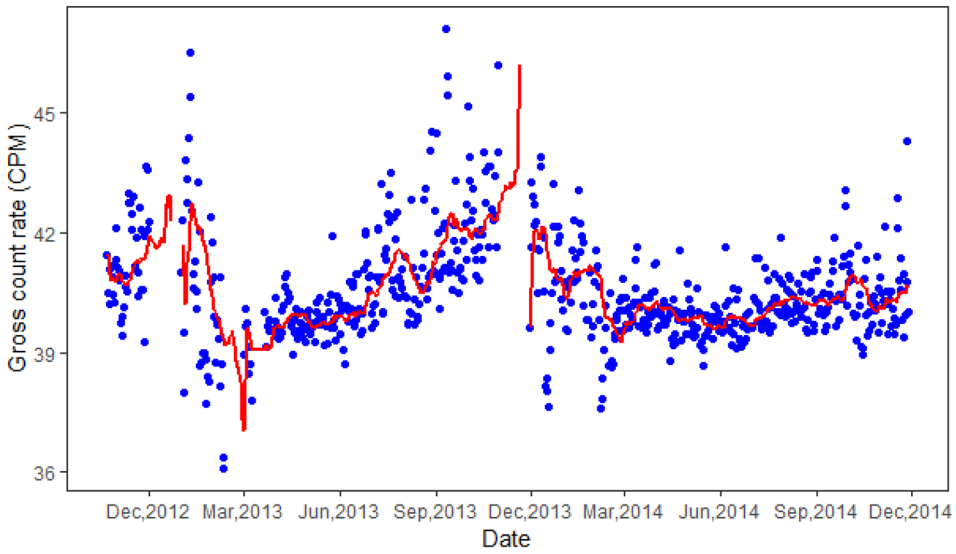
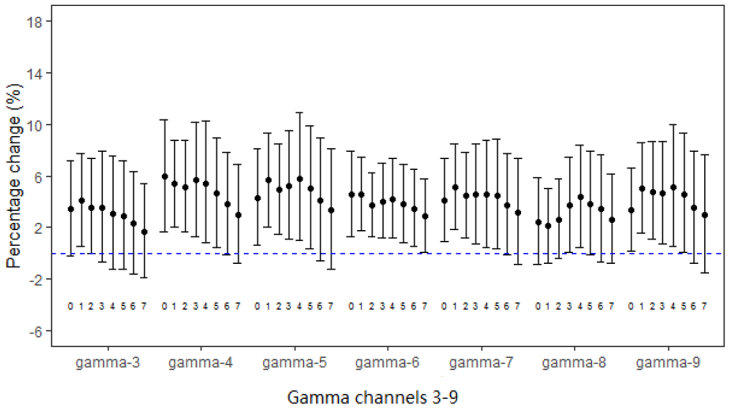


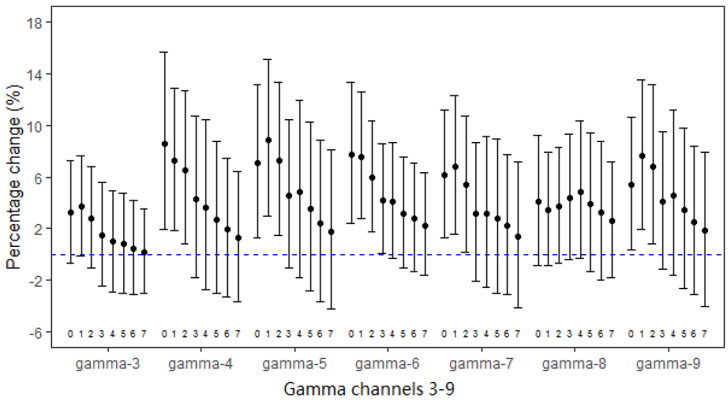

**Fig. 1.**

Daily measured gamma-9 activity (blue dots) from RadNet and 21-day moving average (red line) representing background radiation during study period.

Footnote: The red line is not continuous because of the missing of gamma activities for some dates from RadNet.



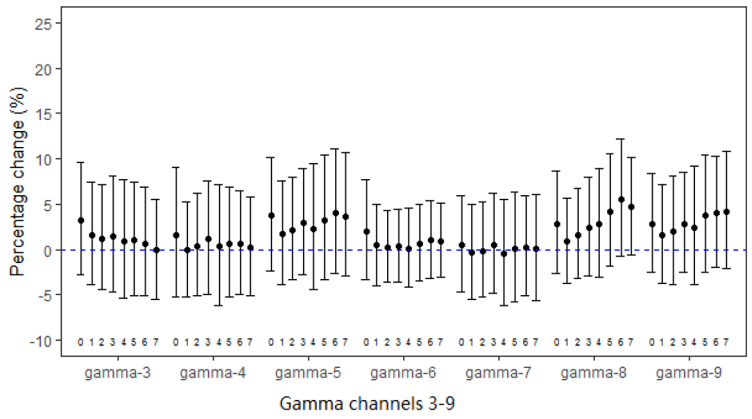

(a) Effect of ambient PM gamma activities



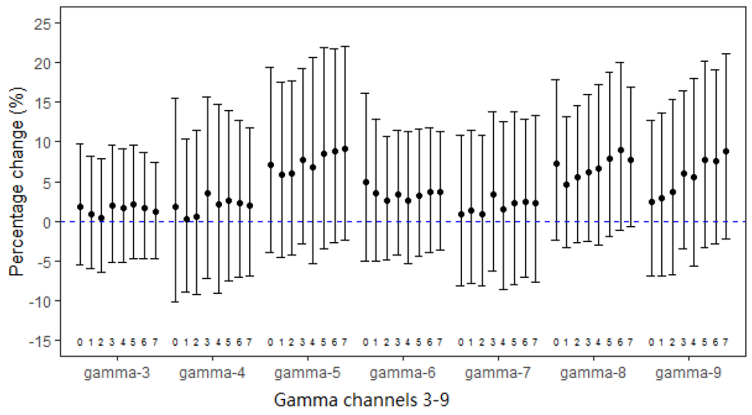

(b) Effect of indoor PM gamma activities

**Fig. 2.**

Percent changes in 8-OHdG per IQR increases in PM-gamma activities (gamma-3 through gamma-9) for daily moving averages (2a for ambient and 2b for indoor) starting with the day of urine collection (day 0) through 7 days before urine collection (day 7), adjusted for weekly black carbon.




(a) Effect of ambient PM gamma activities




(b) Effect of indoor PM gamma activities

**Fig. 3.**

Percent changes in MDA per IQR increases in PM-gamma activities (gamma-3 through gamma-9) for daily moving averages (3a for ambient and 3b for indoor) starting with the day of urine collection (day 0) through 7 days before urine collection (day 7), adjusted for weekly black carbon.

**Table 1.**

Descriptive information for the 81 participants, and urinary oxidative stress biomarkers collected during up to 4 clinic visits (n=231)

| (a) Descriptive information of 81 participants | | | |
|---|---|---|---|
| **Variables** | | **Mean (SD)** | **Range** |
| **Age (yrs)** | | 72.7 (8.4) | 46.7 – 90.7 |
| **BMI (kg/m$^2$)** | | 29.8 (5.8) | 18.8 – 50.8 |
| | | **N (%)** | |
| **Race** | **White** | 72 (88.9%) | |
| | **Non-White** | 9 (11.1%) | |
| **Diabetes** | **Yes** | 20 (24.7%) | |
| | **No** | 61 (75.3%) | |
| **Heart disease** | **Yes** | 41 (50.6%) | |
| | **No** | 40 (59.4%) | |
| **(b) Seasons for clinic visits** | | | |
| | | **N (%)** | |
| **Season** | | **Winter** | 41 (17.7%) |
| | | **Spring** | 52 (22.5%) |
| | | **Summer** | 72 (31.2%) |
| | | **Fall** | (28.6%) |
| **(c) Biomarkers for 231 observations** | | | |
| **Biomarker** | | **Median (25 – 75 percentile)** | **Range** |
| **8-OHdG (mg/mL)** | | 4.4 (2.6 – 7.5) | 0.5 – 28.1 |
| **MDA (μM)** | | 12.6 (8.9 – 19.7) | 1.2 – 275.6 |
| **Creatinine (mg/dL)** | | 114.0 (71.9 – 158.1) | 10.1 – 626.8 |

**Table 2.**

Ambient and indoor PM gamma activities, temperature and relative humidity on the day of urine collection (day 0), weekly BC and season of assessment in 81 COPD patients (n=231 for ambient and n=222 for indoor).

| Variables | Median (25-75$^{th}$ percentile) | Range |
|---|---|---|
| **Daily temperature (°C)** | 13.1 (4.4 – 20.0) | −12.2 – 28.0 |
| **Daily relative humidity (%)** | 63.8 (52.3 – 77.6) | 30.7 – 97.0 |
| **Ambient weekly BC concentration (μg/m$^3$)** | 0.54 (0.41 – 0.78) | 0.27 – 1.20 |
| **Ambient weekly PM concentration (μg/m$^3$)** | 6.03 (5.02 – 7.97) | 3.64 –13.4 |
| **Ambient weekly $NO_2$ concentration (ppb)** | 24.8 (21.2 – 29.7) | 14.5 – 41.4 |
| **Ambient weekly O concentration (ppb)** | 53.1 (44.2 – 62.6) | 26.2 – 75.6 |
| **Ambient daily PM-gamma3 activity (CPM)** | 4.3 (−25.6-51.1) | −195.6 - 265.5 |
| **Ambient daily PM-gamma4 activity (CPM)** | −0.01 (−6.1-10.0) | −53.9 - 43.4 |
| **Ambient daily PM-gamma5 activity (CPM)** | 1.0 (−4.5-6.6) | −37.2 - 31.4 |
| **Ambient daily PM-gamma6 activity (CPM)** | 0.23 (−1.6-2.7) | −25.3 - 14.3 |
| **Ambient daily PM-gamma7 activity (CPM)** | −0.04 (−2.4-3.7) | −26.3 - 18.6 |
| **Ambient daily PM-gamma8 activity (CPM)** | 0.60 (−2.9-4.3) | −26.7 - 43.4 |
| **Ambient daily PM-gamma9 activity (CPM)** | 0.08 (−0.43-0.44) | −3.2 - 3.2 |
| **Indoor weekly BC concentration (μg/m$^3$)** | 0.18 (0.09 – 0.31) | −0.42 – 0.95 |
| **Indoor weekly $PM_{2.5}$ concentration (μg/m$^3$)** | 6.90 (4.78 – 8.93) | 0.26 – 42.8 |
| **Indoor weekly $NO_2$ concentration (ppb)** | 12.3 (8.45 – 19.5) | 1.09 – 112 |
| **Indoor weekly O concentration (ppb)** | 19.6 (26.4 – 47.6) | 0.02 – 463 |
| **Indoor daily PM-gamma3 activity (CPM)** | −0.03 (−22.2-32.5) | −151.1 - 205.2 |
| **Indoor daily PM-gamma4 activity (CPM)** | −0.26 (−4.3-5.5) | −38.4 - 38.8 |
| **Indoor daily PM-gamma5 activity (CPM)** | 0.00 (−3.3-3.9) | −24.4 - 29.1 |
| **Indoor daily PM-gamma6 activity (CPM)** | 0.01 (−1.1-1.6) | −15.1 - 15.7 |
| **Indoor daily PM-gamma7 activity (CPM)** | −0.19 (−2.1-2.2) | −16.1 - 17.3 |
| **Indoor daily PM-gamma8 activity (CPM)** | 0.37 (−1.9-3.4) | −22.0 - 29.8 |
| **Indoor daily PM-gamma9 activity (CPM)** | 0.00 (−0.31-0.30) | −2.0 - 3.0 |