eTable 1. Selected organic chemicals measured in PM$_{0.25}$.

n-Alkanes	**Selected organic acids**	**Medium molecular weight PAH (4 ring)**
n-Dotriacontane	n-Decanoic acid	Benzo(a)pyrene
n-Hentriacontane	n-Dodecanoic acid	Benzo(b)fluoranthene
n-Heptacosane	n-Heptadecanoic acid	Benzo(e)pyrene
n-Heptatriacontane	n-Hexadecanoic acid	Benzo(j)fluoranthene
n-Hexacosane	n-Octadecanoic acid	Benzo(k)fluoranthene
n-Hexatriacontane	n-Octanoic acid	Perylene
n-Nonacosane	n-Pentadecanoic acid	
n-Nonatriacontane	n-Tetradecanoic acid	
n-Octacosane	Oleic acid	**High molecular weight PAH (5-6 ring)**
n-Octatriacontane	Palmitoleic acid	
n-Pentacosane	Phthalic acid	Benzo(ghi)perylene
n-Pentatriacontane		Coronene
n-Tetracontane	**Low molecular weight PAH (2-3 ring)**	Dibenz(ah)anthracene
n-Tetracosane		Dibenzo(ae)pyrene
n-Tetratriacontane	1-Methylchrysene	Indeno(1,2,3-cd)pyrene
n-Triacontane	9-Methylanthracene	Picene
n-Tritriacontane	Acephenanthrylene	
	Anthracene	
Hopanes	Benz(a)anthracene	
17α (H)-21β (H)-Hopane	Benzo(ghi)fluoranthene	
17α(H)-22,29,30-Trisnorhopane	Chrysene	
17β(H)-21A(H)-30-Norhopane	Cyclopenta(cd)pyrene	
22R-Bishomohopane	Fluoranthene	
22R-Homohopane	Phenanthrene	
22R-Trishomohopane	Pyrene	
22S-Bishomohopane	Retene	
22S-Homohopane		
22S-Trishomohopane		

Measurement of fractional NO in exhaled breath (FE$_{NO}$).

Exhaled breath samples were collected and FE$_{NO}$ measured using standard offline procedures,[1] with some modifications as suggested by Linn et al.[2] Exhaled breath samples were collected on Fridays of each monitored week at the same time in the afternoon (2:00-6:00 PM) to control for circadian variation. Subjects were asked to refrain from exercise or food or beverage intake one hour before sample collection. Subjects were instructed to inhale orally to total lung capacity and then immediately performed a slow vital capacity maneuver into an offline apparatus (Sievers Deadspace Discard Bag Collection & Sampling Kit, Ionics Inc., Boulder, CO) attached to a non-reactive 1.5 L Mylar reservoir bag. Contamination from the upper airways was reduced by venting approximately 200 ml of dead-space air prior to collecting the bag sample. Inspired ambient NO was controlled for using an NO/NO$_2$ chemisorbent filter placed at the air intake of the offline apparatus. Subjects breathed through the apparatus for 15 seconds ($\geq$ 2 tidal breaths) before sampling. Breath samples were collected in triplicate to assess reliability. An indoor air sample was collected to assess influence of indoor NO on FE$_{NO}$ (discussed below). The refrigerated (6 °C) sealed Mylar bags were analyzed within 20 hours for FE$_{NO}$ concentration with a chemiluminescence NO analyzer (NOA^{TM} 280i Sievers, GE Analytical Instruments, Boulder, CO).

Measurement of the potential of quasi-ultrafine particles (PM$_{0.25}$) to generate reactive oxygen species (ROS) in alveolar macrophage cells.

We assessed cellular production of ROS induced by PM$_{0.25}$ by examining the *in vitro* responses of rat alveolar macrophage cells (NR8383) to the aqueous extracts of 5-day composited PM$_{0.25}$ filters as preciously described.[3-4] The NR8383 cells were cultured from stocks obtained from the American Type Culture Collection. They have all the normal characteristics of primary macrophages including a functional mannose receptor present in human alveolar macrophages.[5] Details of the assay have been presented in the validation study by Landerman et al.[6] and are described briefly as follows. The composited PM$_{0.25}$ samples were extracted with purified water and then filtered with a 0.22 µm pore size filter to isolate the water-soluble components (dissolved, colloidal, and insoluble species that pass through the filter). Water soluble organic carbon was measured in an aliquot from the water extract as described in the text. Another aliquot from the water extract was buffered in a salt and glucose medium and a dilution series (each dilution in triplicate) prepared for cell exposures. The NR8383 cells were exposed to PM$_{0.25}$ and 2'7'-dicholorohidroflourescin diacetate in 96-well plates, and then incubated at 37°C for 2.5 hours. Fluorescence intensity after the incubation was measured using a Cytoflour II automated fluorescence plate reader. The fluorescence represents the oxidative generating capacity of PM. Positive and negative controls were analyzed along with each set of samples. A model of microbial particles, un-opsonized Zymosan (a β-1,3-polysachharide of D-glucose) served as a positive control because it binds to TLR-2 receptors on macrophage cells and then activates a strong respiratory burst and ROS production. Results are reported in units of Zymosan equivalents. We report results as µg Zymosan equivalents/m^3 air based the product of µg Zymosan/µg sample by 5-day average PM$_{0.25}$ in µg/m^3 air.

An assessment of cell viability is important in verifying that the ROS measurement is not compromised by cell injury. We routinely address this issue by (a) running the ROS assay with a detailed dilution series of each sample, and (b) measuring lactate dehydrogenase release. Cell damage/toxicity is revealed as non-linearity in dose-response curves and/or lactate dehydrogenase release above appropriate controls. The ROS data reported here was measured under experimental conditions where cell injury was negligible.

The rationale for our deliberate selection of murine cell line NR8383 is outlined in Landreman et al.[6] Briefly, our goal was to use a surrogate that exhibited all the functional characteristics of normal primary human alveolar macrophages. However, well-established and documented human alveolar macrophage cell lines are not available. We considered producing human alveolar macrophages by induction/differentiation from other human monocyte cell lines (e.g. U937 and THP-1, with PMA). However, these protocols were not considered sufficiently robust for our intended application. We therefore focused on murine alveolar cell lines, and after careful review determined that the rat alveolar cell line NR8383 best met our criteria. These cells exhibit the important functional characteristics of normal primary macrophage cells - being highly responsive to microbial, particulate, and soluble stimuli

with phagocytosis and killing. The cells display oxidative burst and secrete relevant cytokines (e.g. IL-1, IL-6, TNF-α, β). Importantly, the NR8383 cells express a functional mannose receptor, an attribute not present in other macrophage cell lines such as HL60, U937, and RAW264. The mannose receptor is an important phagocytic and endocytic receptor critical for immune response and host defense. Also, the attributes of this cell line have been exploited in numerous studies of the health impacts of environmental particles, and importantly the cell line has proven to be exceptionally robust and consistent in its response characteristics, which are traits critical for accurate comparison of studies and samples.

The relation of $F_{E_{NO}}$ to ROS adjusting for indoor NO
There was little difference in the associations for $F_{E_{NO}}$ reported in the manuscript when adjusting for indoor NO, and in some cases, associations increased (secondary OC, organic acids and O_3). Only the association of ROS with $F_{E_{NO}}$ was confounded somewhat by indoor NO (18% decrease). This apparent confounding effect may be a reflection of the effect on airway inflammation by air pollutants represented by ambient NO that infiltrates indoors rather than an error in the exhalant due to contamination by indoor NO. To assess this we tested confounding of the association between IL-6 and ROS by indoor NO, which is not a relevant confounder in this case. We found an even greater level of confounding than with $F_{E_{NO}}$ (43% decrease in the estimate of association with ROS). These findings suggest that adjusting for indoor NO may be inappropriate under the assumption that our use of an NO/NO_2 chemisorbent filter on the air intake of the Sievers offline apparatus is sufficient to reduce contamination.

Effect modification by medications, asthma and season.
In exploratory analyses, we found no clear evidence of effect modification of air pollutant associations with IL-6 or $F_{E_{NO}}$ by statins and few differences by angiotensin-converting enzyme inhibitors or angiotensin II receptor antagonists (not shown). There were no significant interactions between season and the 5-day average pollutants for IL-6 models (not shown). However, we previously reported stronger associations of IL-6 with longer-term averages of PN and EC (7- and 9-day) in the cooler phase than in the warmer phase.[7]

Below we present results of models evaluating effect modification of associations between $F_{E_{NO}}$ and air pollutants by season and by asthma diagnosis. Associations were more strongly positive in the cooler phase between $F_{E_{NO}}$ and macrophage ROS, $PM_{2.5}$, $PM_{0.25-2.5}$, $PM_{0.25-2.5}$, water soluble organic carbon, organic acids, and O_3 (eTable 2), even though concentrations of water soluble organic carbon and especially ROS and O_3 were lower then (Table 2 of manuscript). Adjustment for indoor NO did not confound associations with the exception of secondary OC and O_3, which were more strongly associated with increased $F_{E_{NO}}$ after adjustment (not shown).

Only organic acids and ROS showed significantly stronger associations with $F_{E_{NO}}$ among four subjects with asthma (eTable 3). Nevertheless, these pollutants were still positively associated with $F_{E_{NO}}$ among subjects without asthma. Adjustment for indoor NO did not confound the association of $F_{E_{NO}}$ with ROS (4.9% decrease in regression coefficient) or with organic acids (3.5%) in the asthma group (not shown).

Given the overall interaction of ROS and organic acids with season in relation to $F_{E_{NO}}$, we tested whether associations in the asthma and non-asthma groups differed by season. In both groups with and without asthma, we found significantly stronger associations between $F_{E_{NO}}$ and ROS during the cooler season (eFigure 1A). However, a contrasting pattern was seen for organic acids in that subjects with asthma showed stronger $F_{E_{NO}}$ associations during the warm season, whereas subjects without asthma showed stronger $F_{E_{NO}}$ associations during the cooler season. This suggests three-way interaction between asthma diagnosis, season, and organic acids (eFigure 1B).

It is possible that subjects with asthma are susceptible to photochemically-generated aerosols represented by organic acids in the warmer phase. This is consistent with our hypothesis about airway inflammation and secondary organic aerosols components (Figure 1 of manuscript). Organic acids likely serve as better tracers for secondary organic aerosols in the summer than in the winter when there may be other sources of these compounds. Because all of these exposures, except organic acids, were lower on average in the cooler season, we speculate that differences in association were due to variation in the mixture of particle components and/or particle size distribution, particularly nanoparticles, which we did not measure. We consider these findings of effect modification to be hypothesis-generating.

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eTable 2. Associations of FE$_{NO}$ (ppb) with 5-d average outdoor community air pollutants: effect modification by seasonal phase of study.

Air Pollutant	FE$_{NO}$ (ppb) Regression coefficient (95% CI)[a]	
	Warm Season	**Cool Season**
Macrophage reactive oxygen species	0.30 (-0.45, 1.05)*	1.52 (0.43 , 2.62)
Hourly PM mass and markers		
PM$_{2.5}$ Mass	-0.27 (-2.27, 1.74)**	2.13 (1.02 , 3.24)
Marker of primary and secondary organic aerosols:		
organic carbon	1.34 (-2.06, 4.73)	2.37 (-0.38, 5.12)
Markers of primary organic aerosols:		
elemental carbon	-0.52 (-2.14, 1.10)	-0.04 (-1.35, 1.27)
black carbon	-0.01 (-1.83, 1.80)	1.27 (-0.05, 2.59)
Primary organic carbon	-1.23 (-4.63, 2.17)	0.78 (-2.32, 3.89)
Marker of secondary organic aerosols:		
Secondary organic carbon	0.84 (-0.47, 2.15)	1.09 (0.04 , 2.14)
particle number	-3.03 (-5.35, -0.70)	-1.86 (-3.52, -0.19)
Size-fractionated PM mass		
PM$_{0.25}$ (enriched in primary organic aerosols)	-0.43 (-2.58, 1.71)	-0.04 (-2.06, 1.98)
PM$_{0.25-2.5}$ (enriched in secondary organic aerosols)	-0.63 (-2.51, 1.26)**	1.55 (0.77 , 2.34)
PM$_{2.5-10}$	-1.88 (-2.88, -0.87) [†]	1.57 (0.67 , 2.46)
Organic PM$_{0.25}$ Components		
Markers of primary organic aerosols:		
PAH total	1.16 (-0.72 , 3.05)	0.06 (-0.91 , 1.04)
PAH low molecular weight	0.92 (-0.53 , 2.36)	0.56 (-0.57 , 1.68)
PAH medium molecular weight	1.12 (-1.38 , 3.61)	-0.34 (-1.30 , 0.61)
PAH high molecular weight	1.61 (-0.78 , 4.01)	0.29 (-0.72 , 1.31)
Hopanes	0.17 (-0.54 , 0.88)	0.47 (-0.94 , 1.88)
Markers of secondary organic aerosols:		
water soluble organic carbon	0.19 (-0.87 , 1.26)*	1.67 (0.53 , 2.80)
Organic Acids	0.71 (-0.54 , 1.96)	1.91 (0.99 , 2.82)
n-Alkanes	0.09 (-1.56 , 1.74)	-0.01 (-0.18 , 0.16)
Hourly gases		
Markers of primary emissions:		
NO$_2$	0.65 (-1.06 , 2.36)	0.19 (-1.60 , 1.97)
NO$_x$	1.87 (-0.77 , 4.52)	-0.42 (-1.79 , 0.95)
CO	1.46 (-1.43 , 4.36)	0.51 (-0.96 , 1.98)
Marker of photochemistry:		
O$_3$	-0.08 (-1.86, 1.70)**	3.27 (1.01 , 5.53)

Fractional NO in exhaled breath (FE$_{NO}$); PM: particulate matter; PAH: polycyclic aromatic hydrocarbons.
P-value for interaction, warm vs. cool season: * $p < 0.1$, ** $p < 0.05$, [†] $p < 0.01$
[a]   Regression coefficients and 95% confidence intervals are for the expected change in the biomarker associated with an interquartile range change in the air pollutant (Table 2 of manuscript), mean centered by community and seasonal phase, and adjusted for temperature.

eTable 3. Associations of $F_{E_{NO}}$ (ppb) with 5-d average outdoor community air pollutants: effect modification by asthma diagnosis.

Air Pollutant	$F_{E_{NO}}$ (ppb) Regression coefficient (95% CI)[a]	
	No Asthma (N=56)	**Asthma (N=4)**
Macrophage reactive oxygen species	0.53 (-0.12, 1.18)	2.12 (0.27, 3.97)
Hourly PM mass and markers		
PM$_{2.5}$ Mass	1.66 (0.65, 2.67)	0.30 (-3.43, 4.04)
Marker of primary and secondary organic aerosols:		
organic carbon	2.34 (0.32, 4.36)	-0.26 (-7.69, 7.17)
Markers of primary organic aerosols:		
elemental carbon	-0.21 (-1.24, 0.82)	0.41 (-3.88, 4.70)
black carbon	0.74 (-0.36, 1.85)	3.36 (-1.04, 7.76)
Primary organic carbon	0.45 (-1.87, 2.77)	-1.38 (-10.3, 7.57)
Marker of secondary organic aerosols:		
Secondary organic carbon	1.08 (0.24, 1.92)	0.09 (-3.10, 3.29)
particle number	-1.90 (-3.30, -0.50)*	-6.88 (-11.7, -2.04)
Size-fractionated PM mass		
PM$_{0.25}$ (enriched in primary organic aerosols)	-0.37 (-1.88, 1.15)	4.02 (-1.03, 9.08)
PM$_{0.25-2.5}$ (enriched in secondary organic aerosols)	1.27 (0.52, 2.03)	-0.12 (-2.63, 2.39)
PM$_{2.5-10}$	0.10 (-0.62, 0.81)	-0.53 (-2.98, 1.92)
Organic PM$_{0.25}$ Components		
Markers of primary organic aerosols:		
PAH total	0.29 (-0.57, 1.15)	1.76 (-4.54, 8.05)
PAH low molecular weight	0.62 (-0.26, 1.50)	3.84 (-0.99, 8.67)
PAH medium molecular weight	-0.10 (-1.00, 0.79)	-2.49 (-7.82, 2.84)
PAH high molecular weight	0.44 (-0.50, 1.38)	3.01 (-3.62, 9.64)
Hopanes	0.18 (-0.47, 0.84)	0.91 (-1.52, 3.33)
Markers of secondary organic aerosols:		
water soluble organic carbon	0.87 (0.06, 1.68)	1.59 (-1.36, 4.54)
Organic Acids	1.37 (0.62, 2.13)	3.96 (0.55, 7.37)
n-Alkanes	-0.02 (-0.21, 0.16)	0.02 (-0.37, 0.42)
Hourly gases		
Markers of primary emissions:		
NO$_2$	0.20 (-1.13, 1.52)**	5.68 (1.52, 9.83)
NO$_x$	-0.04 (-1.29, 1.20)*	5.17 (-0.62, 10.95)
CO	0.68 (-0.66, 2.03)	3.00 (-5.76, 11.76)
Marker of photochemistry:		
O$_3$	1.33 (-0.10, 2.77)	2.96 (-2.68, 8.60)

Fractional NO in exhaled breath ($F_{E_{NO}}$); PM: particulate matter; PAH: polycyclic aromatic hydrocarbons.
P-value for interaction no asthma vs. asthma: * $p < 0.1$, ** $p < 0.05$, † $p < 0.01$
[a] Regression coefficients and 95% confidence intervals are for the expected change in the biomarker associated with an interquartile range change in the air pollutant (Table 2 of manuscript), mean centered by community and seasonal phase, and adjusted for temperature.

eFigure 1. Associations of fractional concentration of exhaled NO (FE$_{NO}$) with macrophage reactive oxygen species (ROS) production from PM$_{0.25}$ aqueous extracts and PM$_{0.25}$ organic acids: Effect modification by asthma diagnosis and season. A. FE$_{NO}$ with ROS; B. FE$_{NO}$ with organic acids. Expected change in the biomarker (adjusted coefficient and 95% CI) corresponds to an interquartile range increase in the air pollutant concentration (Table 2 of manuscript), adjusted for temperature, community and seasonal phase.

A.
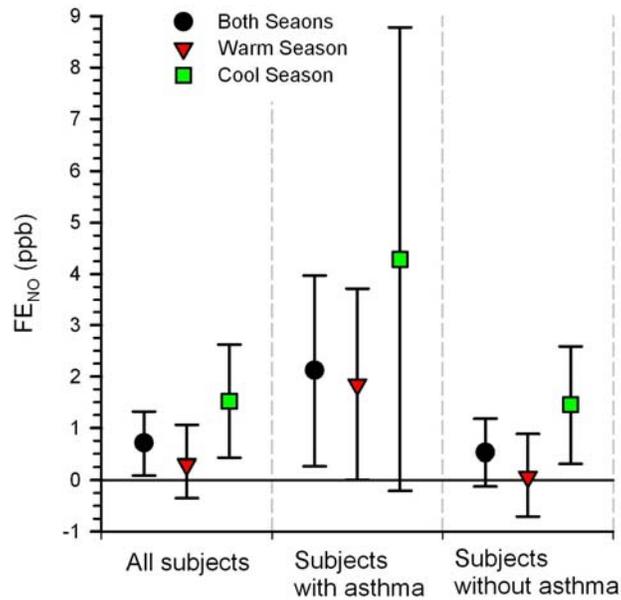
B.
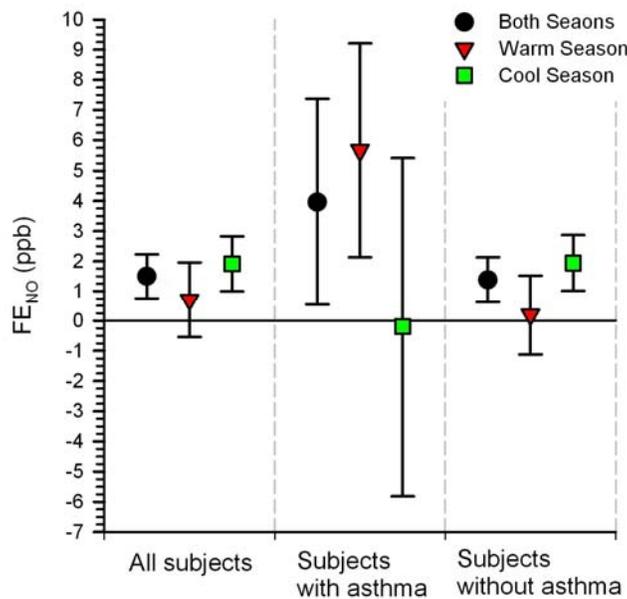
Effect modification by chronic obstructive pulmonary disease (COPD).

In exploratory analyses, we found no clear evidence of effect modification of air pollutant associations with FE$_{NO}$ by diagnosis of COPD (eTable 4) based on a lack of p-values < 0.1 for product terms. Nevertheless, although we had limited power, the positive associations of FE$_{NO}$ with air pollution seen for all subjects in Table 4 of the manuscript were somewhat stronger among the 55 subjects without COPD. This included two markers of secondary organic aerosols (secondary OC and water soluble organic carbon), total OC, accumulation mode PM (PM$_{0.25-2.5}$), PM$_{2.5}$, and O$_3$, but the opposite was observed for organic acids. Null findings for PM$_{0.25}$ and markers of primary combustion were also clearly

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seen in both groups with the exception of black carbon, and low molecular weight PAH which were nominally related to FE_{NO} in subjects without COPD.

eTable 4. **Associations of FE_{NO} with 5-d average outdoor community air pollutants: effect modification by COPD diagnosis.**

Air Pollutant	FE_{NO} (ppb) regression coefficient (95% CI)[a]	
	No COPD (N=55)	COPD (N=5)
Macrophage reactive oxygen species	0.78 (0.15, 1.41)	-0.43 (-2.73, 1.87)
Hourly PM mass and markers		
PM$_{2.5}$ Mass	1.67 (0.65, 2.68)	0.53 (-2.98, 4.04)
Marker of primary and secondary organic aerosols:		
organic carbon	2.31 (0.32, 4.30)	-1.55 (-10.11, 7.00)
Markers of primary organic aerosols:		
elemental carbon	0.02 (-1.02, 1.07)	-2.42 (-5.88, 1.03)
black carbon	1.07 (-0.04, 2.18)	-1.86 (-5.90, 2.19)
Primary organic carbon	0.77 (-1.54, 3.08)	-6.09 (-14.15, 1.97)
Marker of secondary organic aerosols:		
Secondary organic carbon	1.00 (0.17, 1.83)	1.25 (-1.97, 4.47)
particle number	-2.47 (-3.87, -1.08)	-0.13 (-5.01, 4.75)
Size-fractionated PM mass		
PM$_{0.25}$ (enriched in primary organic aerosols)	0.15 (-1.37, 1.67)	-2.05 (-6.76, 2.67)
PM$_{0.25-2.5}$ (enriched in secondary organic aerosols)	1.17 (0.41, 1.93)	1.03 (-1.40, 3.46)
PM$_{2.5-10}$	0.08 (-0.63, 0.80)	-0.42 (-2.76, 1.92)
Organic PM$_{0.25}$ Components		
Markers of primary organic aerosols:		
PAH total	0.37 (-0.51, 1.25)	-0.59 (-3.81, 2.62)
PAH low molecular weight	0.81 (-0.08, 1.70)	-1.06 (-4.55, 2.42)
PAH medium molecular weight	-0.09 (-1.01, 0.83)	-1.11 (-4.13, 1.92)
PAH high molecular weight	0.45 (-0.52, 1.41)	0.87 (-2.61, 4.34)
Hopanes	0.35 (-0.32, 1.02)	-0.71 (-2.57, 1.15)
Markers of secondary organic aerosols:		
water soluble organic carbon	0.97 (0.16, 1.78)	0.24 (-2.44, 2.93)
Organic Acids	1.38 (0.61, 2.14)	3.06 (0.34, 5.78)
n-Alkanes	-0.03 (-0.21, 0.15)	0.11 (-0.44, 0.66)
Hourly gases		
Markers of primary emissions:		
NO$_2$	0.78 (-0.53, 2.10)	-2.28 (-6.76, 2.19)
NO$_x$	0.30 (-0.97, 1.58)	-1.61 (-5.86, 2.64)
CO	0.84 (-0.55, 2.23)	-0.56 (-4.85, 3.72)
Marker of photochemistry:	1.56 (0.12, 3.00)	-0.32 (-5.50, 4.86)
O$_3$		

Fractional NO in exhaled breath (FE_{NO}); PM: particulate matter; PAH: polycyclic aromatic hydrocarbons.
[a] Regression coefficients and 95% confidence intervals are for the expected change in the biomarker associated with an interquartile range change in the air pollutant (Table 2), mean centered by community and seasonal phase, and adjusted for temperature. None of the product terms reached $p < 0.1$.

eTable 5. Associations of FE_{NO} and IL-6 with daily average outdoor community air pollutants.

Air Pollutant	Averaging time[a]	IL-6 (pg/mL) regression coefficient (95% CI)[b]	FE_{NO} (ppb) regression coefficient (95% CI)[b]
Particle number	1-day	0.35 (0.16, 0.53)	-1.16 (-2.16, -0.16)
	3-day	0.25 (0.03, 0.47)	-2.01 (-3.17, -0.85)
	5-day	0.22 (-0.04, 0.47)	-2.27 (-3.62, -0.92)
	7-day	0.34 (-0.03, 0.70)	-2.41 (-4.16, -0.65)
	9-day	0.40 (-0.01, 0.82)	-2.15 (-3.90, -0.40)
Elemental carbon	1-day	0.16 (0.01, 0.31)	-0.54 (-1.19, 0.10)
	3-day	0.26 (0.08, 0.44)	-0.08 (-0.88, 0.71)
	5-day	0.29 (0.07, 0.50)	-0.17 (-1.18, 0.83)
	7-day	0.49 (0.21, 0.78)	-0.16 (-1.44, 1.13)
	9-day	0.59 (0.25, 0.92)	-0.89 (-2.41, 0.64)
Black carbon	1-day	0.16 (0.02, 0.30)	0.19 (-0.42, 0.80)
	3-day	0.19 (0.02, 0.35)	0.76 (-0.02, 1.54)
	5-day	0.21 (-0.02, 0.45)	0.89 (-0.19, 1.96)
	7-day	0.19 (-0.10, 0.49)	1.25 (-0.08, 2.59)
	9-day	0.18 (-0.14, 0.50)	0.64 (-0.84, 2.11)
Organic Carbon (OC)	1-day	-0.02 (-0.38, 0.34)	1.92 (0.53, 3.30)
	3-day	-0.008 (-0.51, 0.49)	2.90 (0.99, 4.82)
	5-day	-0.02 (-0.53, 0.49)	2.11 (0.17, 4.06)
	7-day	-0.01 (-0.62, 0.59)	2.74 (0.37, 5.12)
	9-day	-0.10 (-0.73, 0.54)	0.27 (-2.24, 2.77)
Primary OC	1-day	0.30 (-0.05, 0.66)	-0.70 (-2.01, 0.61)
	3-day	0.50 (0.06, 0.94)	0.49 (-1.28, 2.25)
	5-day	0.48 (-0.06, 1.02)	0.28 (-1.96, 2.51)
	7-day	0.60 (-0.01, 1.22)	0.40 (-2.12, 2.92)
	9-day	0.66 (-0.07, 1.39)	-0.83 (-3.81, 2.16)
Secondary OC	1-day	-0.11 (-0.26, 0.04)	1.07 (0.50, 1.65)
	3-day	-0.17 (-0.37, 0.03)	1.24 (0.47, 2.02)
	5-day	-0.13 (-0.35, 0.09)	1.01 (0.20, 1.83)
	7-day	-0.19 (-0.48, 0.09)	1.62 (0.53, 2.70)
	9-day	-0.24 (-0.57, 0.09)	0.38 (-0.91, 1.67)

Air Pollutant	Averaging time	IL-6 (pg/mL) regression coefficient (95% CI)[a]	FE$_{NO}$ (ppb) regression coefficient (95% CI)[a]
CO	1-day	0.35 (0.17, 0.54)	0.26 (-0.66, 1.18)
	3-day	0.40 (0.20, 0.61)	0.54 (-0.49, 1.56)
	5-day	0.54 (0.27, 0.80)	0.73 (-0.60, 2.06)
	7-day	0.34 (-0.06, 0.74)	0.45 (-1.70, 2.59)
	9-day	0.31 (-0.07, 0.70)	-0.17 (-2.19, 1.85)
NO$_x$	1-day	0.32 (0.17, 0.48)	0.31 (-0.45, 1.06)
	3-day	0.32 (0.14, 0.49)	0.52 (-0.36, 1.40)
	5-day	0.42 (0.18, 0.66)	0.18 (-1.04, 1.40)
	7-day	0.56 (0.25, 0.87)	0.59 (-0.93, 2.11)
	9-day	0.54 (0.23, 0.85)	0.12 (-1.55, 1.78)
O$_3$	1-day	-0.16 (-0.39, 0.07)	0.83 (-0.33, 2.00)
	3-day	-0.16 (-0.45, 0.12)	0.05 (-1.35, 1.46)
	5-day	-0.14 (-0.44, 0.17)	1.41 (0.01, 2.81)
	7-day	-0.16 (-0.48, 0.16)	1.48 (0.03, 2.92)
	9-day	-0.24 (-0.60, 0.11)	1.71 (0.06, 3.35)
PM$_{2.5}$ [c]	1-day	-0.09 (-0.24, 0.05)	0.02 (-0.66, 0.71)
	3-day	-0.12 (-0.32, 0.08)	1.01 (0.06, 1.95)
	5-day	-0.25 (-0.48, -0.03)	1.57 (0.59, 2.54)
	7-day	-0.34 (-0.56, -0.12)	1.32 (0.34, 2.29)
	9-day	-0.39 (-0.64, -0.14)	1.15 (0.05, 2.24)
PM$_{0.25}$	1-day	0.30 (0.09, 0.51)	-0.08 (-1.04, 0.89)
	3-day	0.42 (0.15, 0.69)	-0.28 (-1.55, 1.00)
	5-day	0.26 (-0.06, 0.57)	-0.02 (-1.49, 1.44)
PM$_{0.25-2.5}$	1-day	-0.07 (-0.16, 0.02)	-0.06 (-0.47, 0.35)
	3-day	-0.20 (-0.38, -0.02)	0.09 (-0.75, 0.94)
	5-day	-0.19 (-0.36, -0.02)	1.16 (0.43, 1.88)
PM$_{2.5-10}$	1-day	0.11 (-0.03, 0.24)	-0.42 (-1.02, 0.18)
	3-day	0.15 (-0.06, 0.35)	-0.65 (-1.63, 0.33)
	5-day	0.01 (-0.15, 0.17)	0.04 (-0.65, 0.72)

Fractional NO in exhaled breath (FE$_{NO}$); PM: particulate matter.

[a]   We present averaging times that skip over averages by one day to simplify the presentation but still retain an evaluation of associations across the span of available exposure data (1-day, 3-day, 5-day, 7-day and 9-day averages).

[b]   Regression coefficients and 95% confidence intervals are for the expected change in the biomarker associated with an interquartile range change in the air pollutant (see Table 2 of manuscript), mean centered by community and seasonal phase, and adjusted for temperature.

[c]   PM$_{2.5}$ mass was measured with a Beta-Attenuation Mass Monitor for up to 9 days before biomarker measurements whereas the size-fractionated PM mass (PM$_{0.25}$, PM$_{0.25-2.5}$, PM$_{2.5-10}$) was measured with a Personal Cascade Impactor Sampler for up to 5 days before biomarker measurements.

eFigure 2. Associations of IL-6 with outdoor PM$_{0.25}$ mass coregressed with macrophage reactive oxygen species (ROS) production from aqueous extracts of the PM$_{0.25}$ samples.
Expected change in the IL-6 (adjusted coefficient and 95% CI) corresponds to an interquartile range increase in the air pollutant concentration (Table 2 of manuscript), adjusted for temperature, community and seasonal phase.
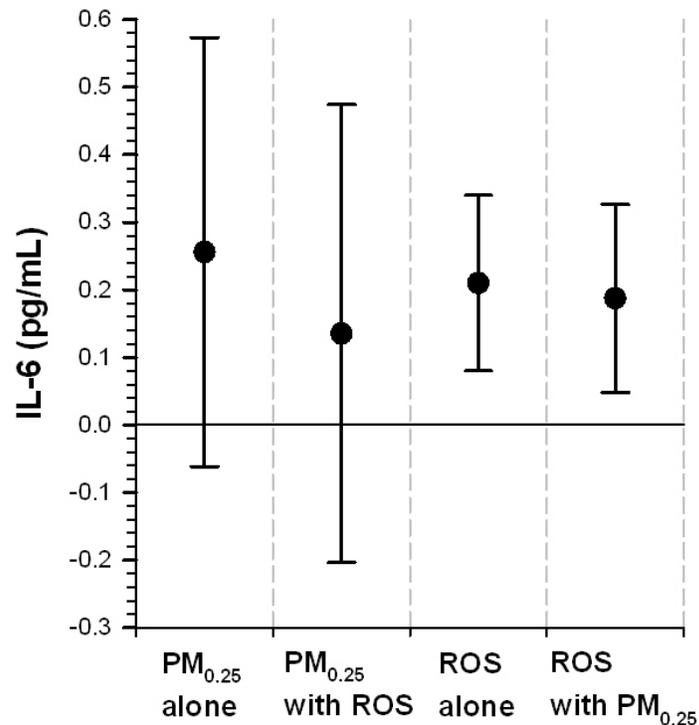
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