Non-Tailpipe Traffic Emissions and Airway Dysfunction

Evidence from a Real-World Crossover Study (IONA)

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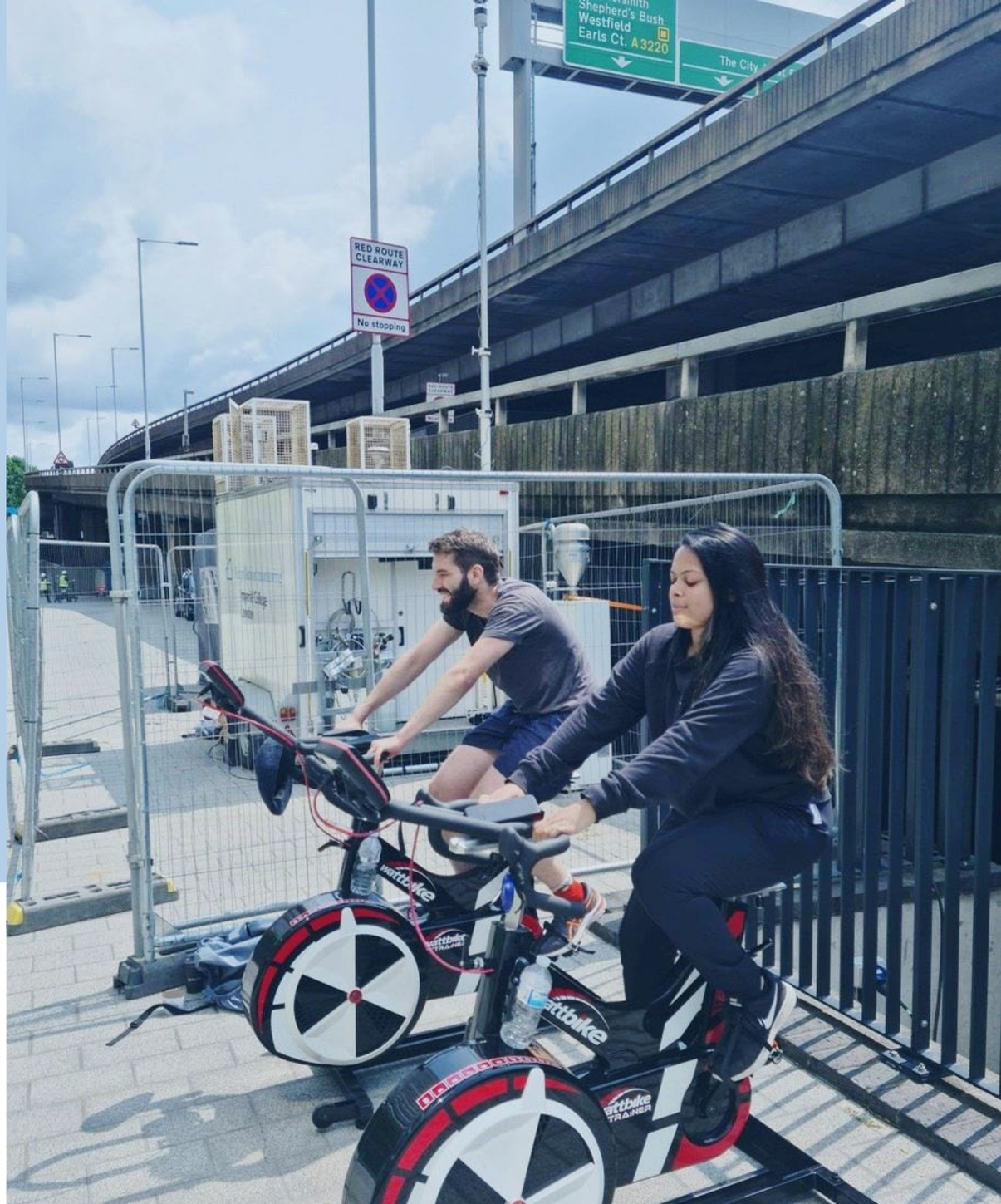


Why focus on non-tailpipe emissions?

The challenge

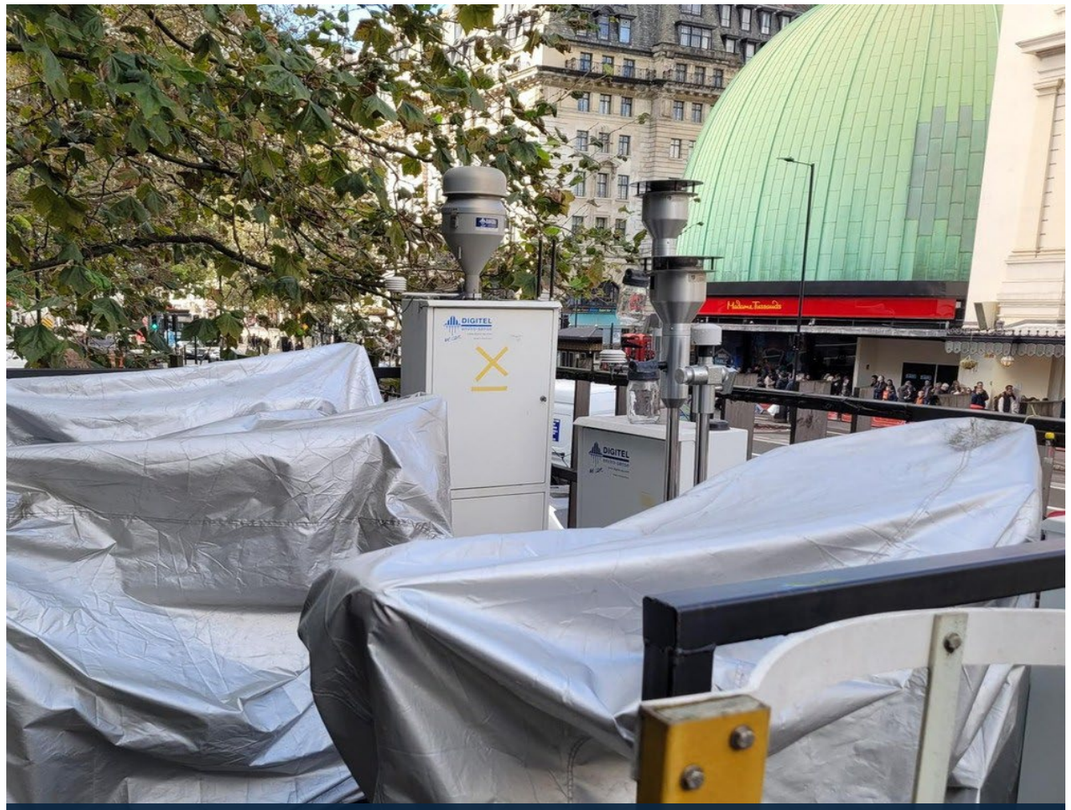
Non-tailpipe PM is a mixture of mechanically generated particles and metals, often co-varying with exhaust traffic markers.

- ❑ **Brake wear**: transition metal-rich particles, linked to stop-go driving
- ❑ **Tyre and road wear**: coarser and finer fractions generated by road friction

Asthma provides a sensitive model for acute airway responses

Aim

To create sufficiently distinct exposure gradients to determine whether source-related airway dysfunction could be detected under real-world conditions.



Ambient, real-world exposure: not a chamber, not a single pollutant.

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Randomised Crossover Exposure Study

44 recruited • **34** completed all three sites • standardised intermittent cycling for 2.5 hours

Each participant acted as their own control across three London microenvironments.

Marylebone Road

stop-go traffic
expected brake-wear contrast

White City (A40)

faster continuous traffic
expected tyre/road-wear contrast

Honor Oak Park

urban background
control reference site

Exposure Visit Timeline:

Pre-Exposure
- Baseline spirometry
- Questionnaires

Mid Exposure (1.25 h)
- Mid-exposure spirometry
- Check symptoms
- Heart rate monitoring

Post- Exposure (2.5 h)
- Post-exposure spirometry
- Biological samples

Next Day Exposure (29 h)
- Next-day spirometry
- Biological samples

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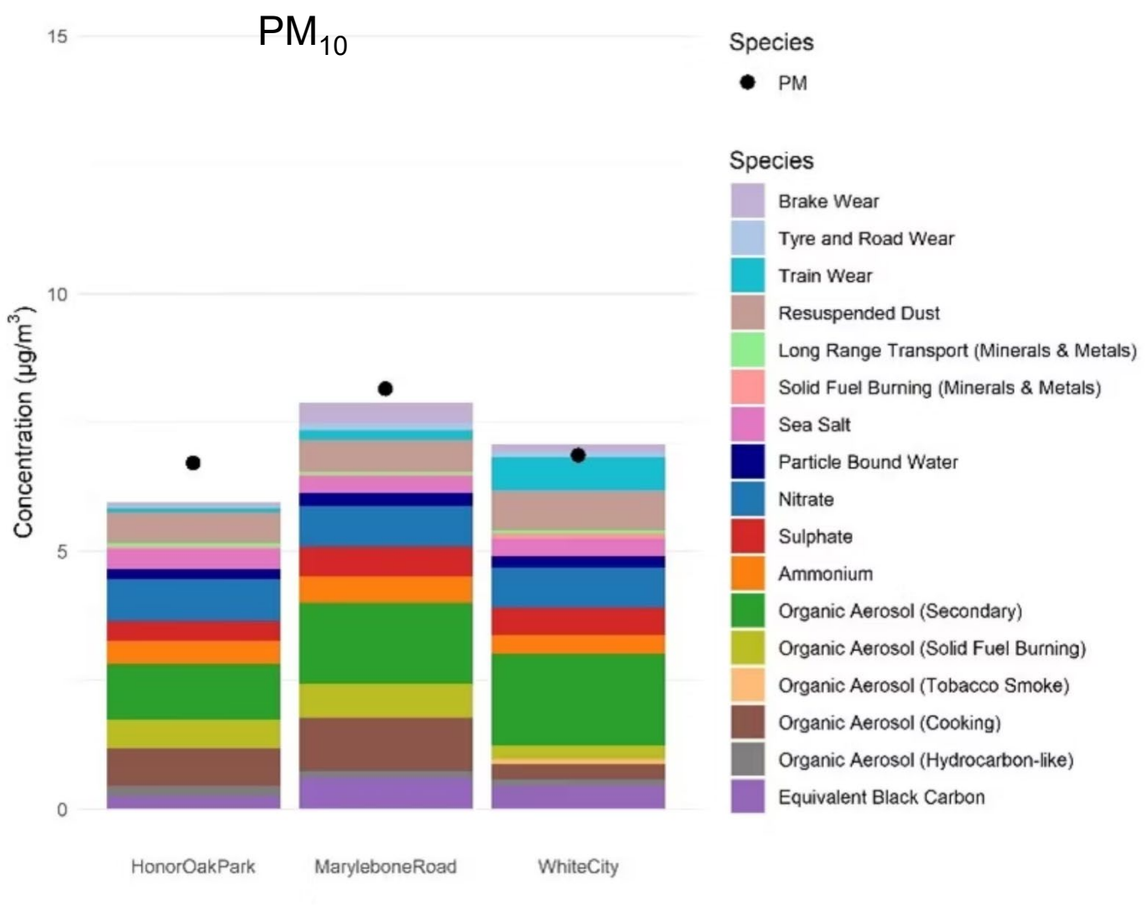
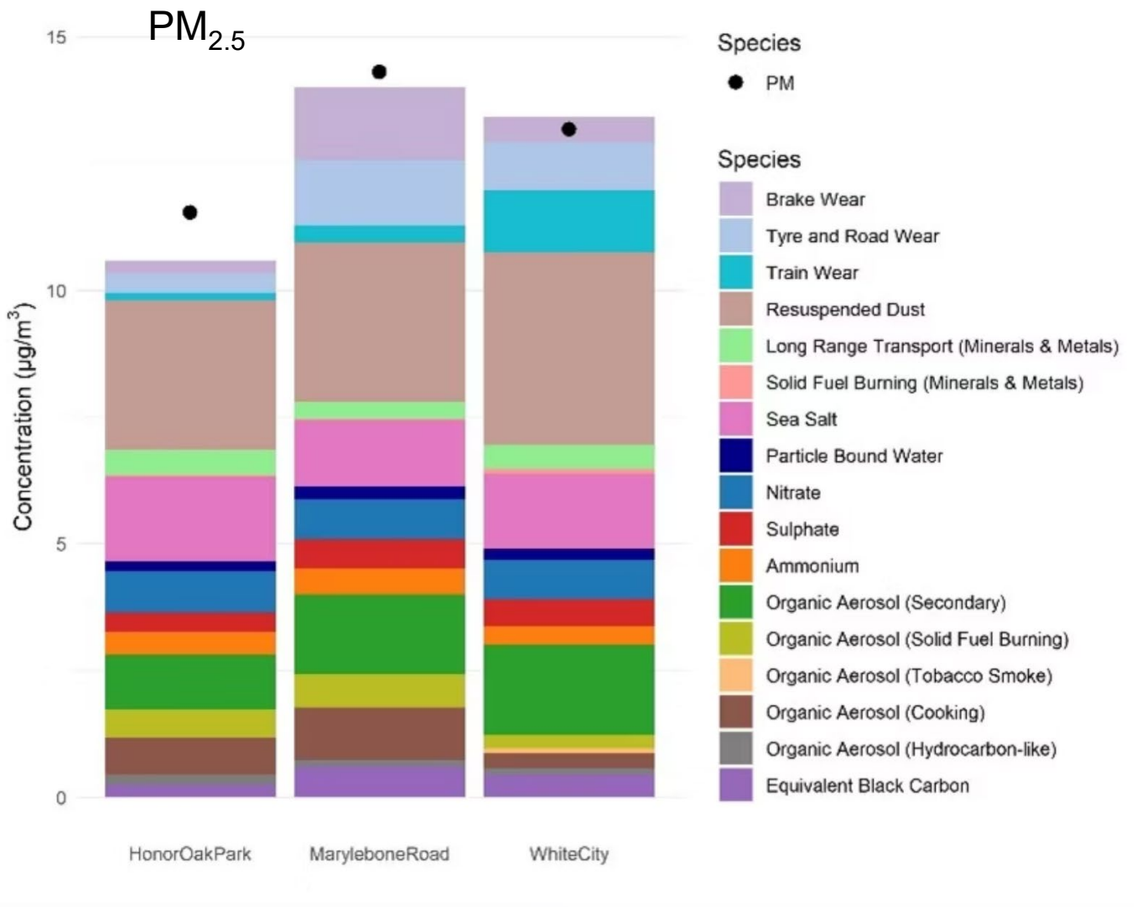
High-resolution source apportionment

Moving from “roadside PM” to brake wear and tyre/road wear fractions.





Stacked bar plots of data from reference equivalent measurements of $PM_{2.5}$ and PM_{10}

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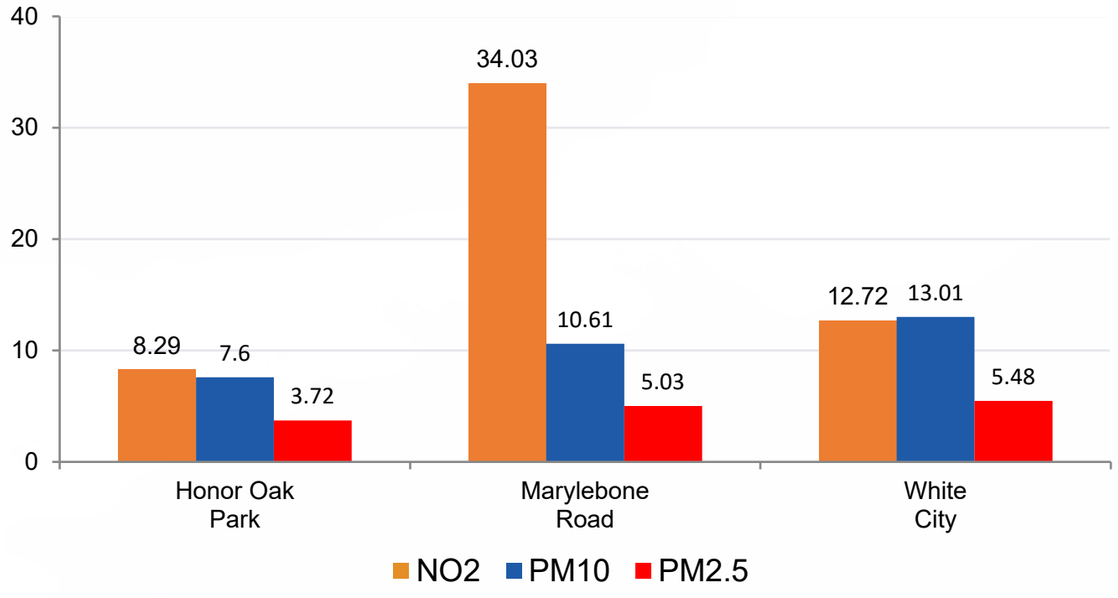
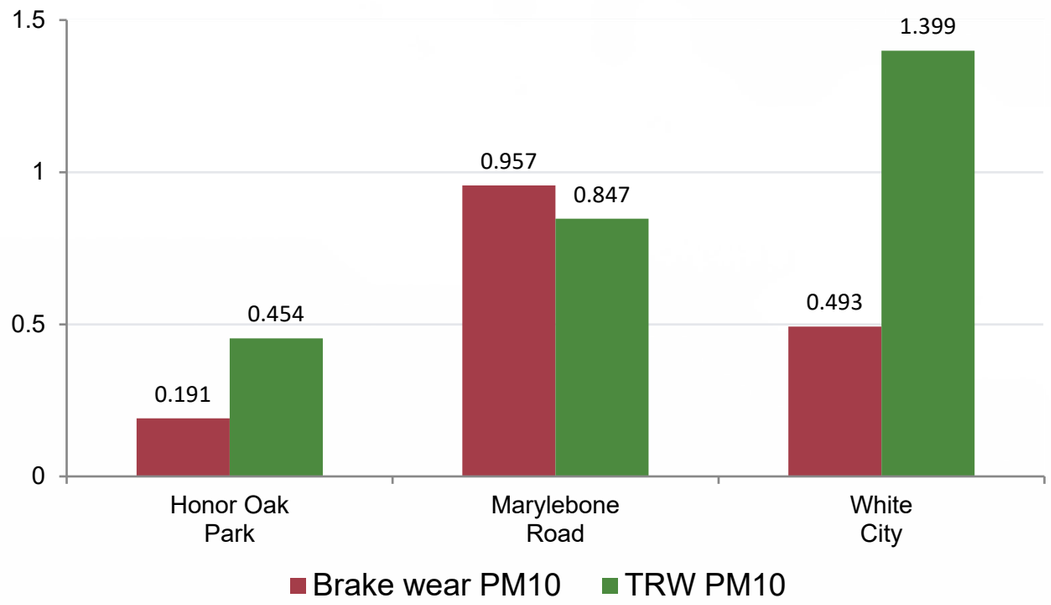
The exposure contrasts worked

The three sites separated traffic mixtures during participant exposure periods.

Criterion pollutants (µg/m³)



Source-resolved PM_{10} fractions (µg/m³)



NO_2 highest at Marylebone Road

$PM1_0$ and $PM_{2.5}$ highest at White City

Brake wear highest at Marylebone; TRW highest at White City

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By site difference: Lung function (FEV1, Forced expiratory volume - L)

Mixed models comparing each site with the urban background site (Honor Oak Park)

Site	Time Point											
	Mid-Exposure				Post-Exposure				Following Day			
	Coefficient	95% CI		P-value	Coefficient	95% CI		P-value	Coefficient	95% CI		P-value
Honor Oak Park	Ref				Ref				Ref			
Marylebone Road	-0.09	-0.20	0.01	0.073	-0.07	-0.30	0.16	0.531	0.01	-0.07	0.10	0.747
White City	-0.15	-0.25	-0.04	0.005*	-0.24	-0.47	-0.01	0.042*	-0.09	-0.18	0.00	0.04*

Results from mixed models with FEV_1 (L) as dependent variable, contrasting sites (categorical covariate) with only participants who completed all three exposure sites (completers) and no other adjustment except FEV1 0min

- Across numerous sensitivity analysis **140-240mL (6 to 8%) reductions** were seen between sites and these remained almost 30 hours after exposures.
- This reduction in breathing capacity was significant enough to be considered **important for health**.

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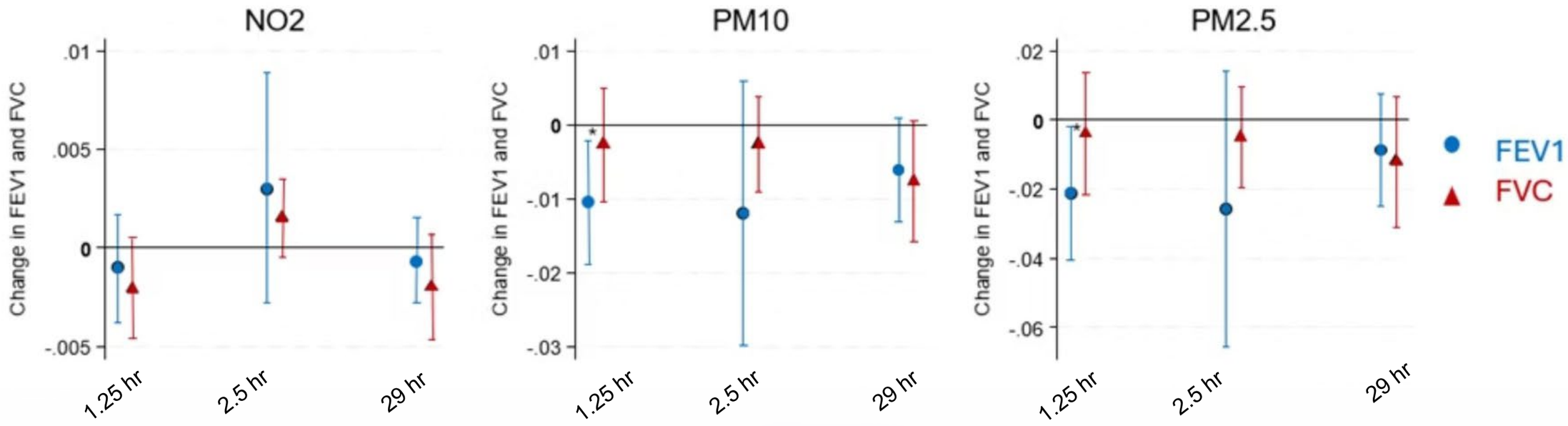
Pollutant-level Models

Criterion pollutants show consistent, partly statistically significant adverse patterns.



Coefficients of changes in FEV_1(L) and FVC(L) per interquartile range increase in pollutant at the times indicated FEV_1: Forced expiratory volume in one second, FVC: Forced vital capacity. *Represents P<0.05. n=38

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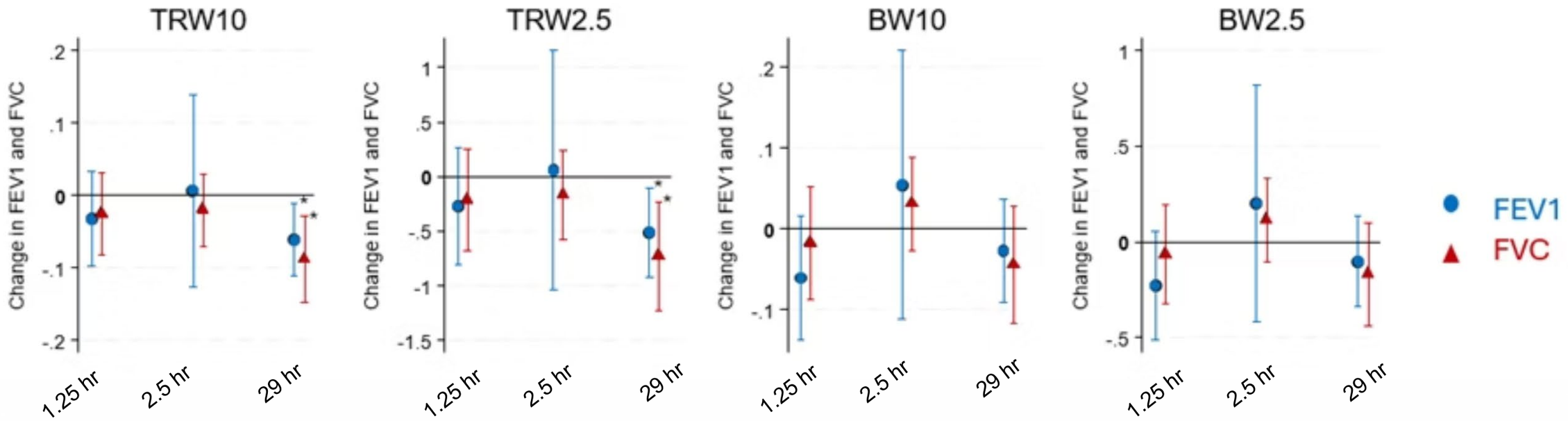
Source-apportionment Models



Coefficients of changes from baseline in FEV1(L) and FVC(L) per pollutant source at the times indicated FEV1: Forced expiratory value in one second, FVC: Forced vital capacity. TRW: Tyre and road wear, BW: Brake wear. * Represents P<0.05

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Key finding: Differential effects by pollutant source

- ❑ Non-tailpipe emissions are not benign
- ❑ Pollution composition matters — not only PM mass
- ❑ Different sources may trigger different biological pathways
- ❑ Responses persisted almost 30 hours after exposure
- ❑ Findings remained robust after multiple model adjustments

Take-home message

Distinct non-tailpipe emission sources produced measurable and clinically relevant respiratory responses.

What next?

Epidemiology
test at population scale

Chambers
mechanisms without ambient confounding

Policy
support regulation of evolving PM sources

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Thank you

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