Effects of near-road and regional air pollution: the challenge of separation

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As people spend most of their lifetime at home, outdoor air pollution levels near the home remain an important determinant of personal exposure to pollution from outdoor sources. With every breath, we inhale a complex mixture of hundreds of such pollutants. A simplified categorisation of these pollutants distinguishes local, freshly emitted (primary) pollutants from regional, aged (secondary) pollutants. To understand people's exposure to the latter—more homogeneously distributed pollutants (eg, fine particles, PM_{2.5} or ozone)—many studies use data from fixed-site monitoring stations, considered to be representative of exposure in the entire community. For near-road local types—such as diesel soot, ultrafine nanoparticles, NO or CO—this approach is insufficient.¹ Freshly emitted pollutants from vehicle exhaust and resuspended particles show strong spatial gradients, with concentrations exponentially decreasing, reaching within 50–500 m of busy roads 10–30% of the levels observed on the street.² Thus, to understand exposure to these locally emitted pollutants, we need to use small-scale local measurements and models.

The main argument for making this distinction is that regional and near-road traffic-related pollutants may have different health effects. The literature on childhood asthma and air pollution supports this notion and reveals two consistent patterns: (1) comparison of the (multivariate-adjusted) asthma prevalence or incidence across communities with different air quality does not support the hypothesis that regional air pollutants play a relevant aetiological role³; (2) studies estimating exposure to near-road traffic-related pollutants indicate that children growing up close to busy roads have a higher risk of developing asthma than those living a few hundred metres away.^{4–5} The Southern Californian Children's Health Study (CHS) is one of several epidemiological studies that recognises the asthmogenic

role of near-road pollutants,^{6,7} confirming biologically plausible models.⁸

Landmark cohort studies initiated by the CHS team provide both cross-sectional and longitudinal evidence for regional ambient air pollution affecting lung growth during adolescence.^{9–10} The question of an independent role of near-road traffic-related air pollution was a later subject of analyses in two CHS cohorts, recruited in 1993 and 1996.¹¹ In those analyses—and in contrast with the asthma findings—lung function growth was associated with both regional pollutants and residential distance from highways as a marker of near-road pollution.

CHS researchers have explored the hypothesis that near-road and regional pollutants play a distinct role in the level of lung function among a cohort of 5–7-year-old children, recruited in 2002–2003.¹² In the new study, near-road pollutants were estimated for each child, linking residential location with land-use regression models and using NO, NO₂ and NO_x as markers of near-road pollution. Exposure to regional pollution relied on fixed-site monitor data, available in each community. Lung function levels were significantly associated with both the near-road and community mean levels of pollution. The coefficients observed for NO₂, listed as a marker for both near-road and regional pollution, indicate that the effects of near-road exposure were much stronger than those attributed to regional pollutants. With PM_{2.5} as a marker for regional pollution, regional effects were statistically significant.

Thus, for health outcomes such as lung function, where possibly both the near-road and regional secondary pollutants matter, the question then arises of whether we can investigate, separate and quantify these effects independently. First, this is of scientific interest, as these groups of pollutants have somewhat different characteristics that may act through distinct pathways. For example, the nano-sized ultrafine particle fractions—of particular relevance near roads—have some toxic properties and effects, such as possibly direct transitions into the brain, for example, that are unlikely to be relevant for larger particles. Second, the distinction is also relevant for policy makers

and risk assessors to more comprehensively target and quantify the burden of disease attributable to pollution and related clean air interventions.¹³ While both near-road and regional pollutants are very heavily driven by traffic in the Southern Californian region, this is not universally true. Moreover, whereas some near-road pollution problems may be solved by local action and tackled by local authorities (eg, retrofitting buses with particle filters), regional pollutants require regional and transnational action.

The CHS, with its multi-community design, provides a setting with sufficient contrasts in regional pollution across communities and in near-road pollutants within communities. For each subject, Urman *et al* determine the extent to which the home-outdoor NO_x concentration deviates from the community mean level to generate within-community exposure estimates that do not correlate with the between-community contrasts.¹² Although one interpretation of their findings is, indeed, that local pollutants result in independent effects, it must be emphasised that regional pollutant exposure relied on fixed-site monitor data alone. Thus, one may hypothesise that the finding of 'independent associations' is an artefact of the analytical approach. Imagine that, instead of testing the air pollution hypotheses, we are testing the association of pack-years of smoking with lung function in adults in a 10-community study. Let us pretend that the distribution of near-road pollutants shown in figure 1B of Urman *et al*¹² does not reflect residential NO₂ but the distribution of pack-years of cigarettes within each community. Let us further assume that we analysed the data in the same way, namely to show how the individual deviates from the community-mean pack-years. What would the association between pack-years and lung function look like for the within- and between-community estimates? In theory, one should expect the same pack-years coefficients for both the within-community and the between-community effects of smoking. The price we pay for such an analytical approach is some loss of statistical power, but the gain is the removal of unmeasured spatial confounders possibly related to 'communities'—a major concern in air pollution research. Moreover, the between-community estimate may be subject to measurement error, as it is not an individually assigned exposure. This point may be particularly relevant in an air pollution study such as that of Urman *et al*, where the regional fixed-site monitor value may be biased

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toward null, as the fixed-site monitor concentration was not equally representative of the community-specific, population-weighted, residential mean NO₂ exposures. This may, in part, explain why the coefficient for regional NO₂ was some four times smaller (and not significant) than the near-road NO₂ effect estimate. The between-community estimates were, however, significant for more homogeneously distributed particles. As these estimates did not correlate closely with the fixed-site monitor levels of NO₂, one may again endorse the interpretation of independent effects. However, these regional pollutants may also vary within communities, which was not taken into account with the fixed-site monitor approach, thus the true correlation between near-road and regional pollution remains unknown.

In light of such uncertainties, one question remains: what should one do to convincingly disentangle and quantify the long-term effects of both near-road and regional pollutants? I think we need studies where both the regional and near-road pollutants are estimated at the individual level, thereby negating the sole use of fixed-site monitor data to represent exposure to 'regional pollution'. Some studies have gone down this avenue,¹⁴ and, most recently, the European ESCAPE network adopted fully standardised measurements and land-use modelling approaches to individually assign home-outdoor (residential) estimates of seven markers of ambient air pollution to participants in more than 30 cohorts.¹⁵ Markers ranged from indicators of secondary regional pollutants (PM_{2.5})¹⁶ to local near-road markers (NO_x or reflectance of PM_{2.5}—indicating local diesel soot). As shown in ESCAPE, the approach may also face limits, as the contrasts between regional pollutants may be inherently low within small study areas. For example, for PM_{2.5}, the ratio between street site and urban background levels was as low as 1.02 and as high as 1.3 among ESCAPE regions, but was usually higher—up to 1.77—for the near-road marker of PM_{2.5} reflectance.¹⁶ In a recent analysis based on six childhood cohorts, NO₂, NO_x, PM_{2.5} absorbance and PM_{2.5} (but not PM₁₀, PM coarse or markers of neighbourhood traffic) were all associated with significantly lower lung function in 6–8-year-old children.¹⁷ Two-pollutant models for local (NO₂) and regional (PM_{2.5}) pollutants were challenged by multi-collinearities and thus did not conclusively support the notion of independent effects.

Mega-cohorts in very large regions—for example, in mega-cities in low-income economies with far more diverse and different sources of pollutants—may provide the setting for such individual approaches. For example, a land-use regression model for the mega-city of Tehran (Iran), developed for an area of 30×30 km, revealed very different patterns, with severalfold contrasts for both near-road traffic-related and (possibly) regional pollutants (PM₁₀, NO₂, SO₂) (publication in preparation—presented at the 2013 conference). The rather low spatial correlations among these pollutants provide a substantially different setting. Modelling both near-road and regional pollutants at the individual level will be needed to understand the specific long-term effects of near-road ultrafine particles.

In sum, while the landmark CHS research indicates independent effects of near-road and regional pollutants on lung function, this may not be generalisable. Further research is needed to better understand the public health role of different sources and characteristics of ambient air pollution. Whereas fixed-site monitor data remain very important for policy making and research using time-series analyses, trend assessment, calibration and modelling, epidemiological studies investigating the long-term effects of air pollution should not rely on fixed-site monitor data alone, but estimate exposure for both regional and local clusters of pollutants at the individual level. This strategy will also open doors to allow us to disentangle all health-relevant environmental stressors of traffic, namely primary and secondary exhaust-related and resuspended pollutants as well as noise, which remain a challenge to be separated and tackled.^{18 19}

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