



ENVIRONMENTAL HEALTH PERSPECTIVES

<http://www.ehponline.org>

Air Pollution and Lung Function in Dutch Children: A Comparison of Exposure Estimates and Associations Based on Land Use Regression and Dispersion Exposure Modeling Approaches

**Meng Wang, Ulrike Gehring, Gerard Hoek, Menno Keuken,
Sander Jonkers, Rob Beelen, Marloes Eeftens, Dirkje S. Postma,
and Bert Brunekreef**

<http://dx.doi.org/10.1289/ehp.1408541>

Received: 10 April 2014

Accepted: 31 March 2015

Advance Publication: 3 April 2015

This article will be available in its final, 508-conformant form 2–4 months after Advance Publication. If you need assistance accessing this article before then, please contact ehp508@niehs.nih.gov. Our staff will work with you to assess and meet your accessibility needs within 3 working days.



Air Pollution and Lung Function in Dutch Children: A Comparison of Exposure Estimates and Associations Based on Land Use Regression and Dispersion Exposure Modeling Approaches

Meng Wang,¹ Ulrike Gehring,¹ Gerard Hoek,¹ Menno Keuken,² Sander Jonkers,² Rob Beelen,¹ Marloes Eeftens,^{1,3,4} Dirkje S. Postma,⁵ and Bert Brunekreef^{1,6}

¹Institute for Risk Assessment Sciences, Utrecht University, Utrecht, the Netherlands; ²TNO, Netherlands Organisation for Applied Scientific Research, Utrecht, the Netherlands; ³Swiss Tropical and Public Health Institute, Basel, Switzerland; ⁴University of Basel, Basel, Switzerland; ⁵University of Groningen, Department of Pulmonology, University Medical Center Groningen, Groningen, the Netherlands; ⁶Julius Center for Health Sciences and Primary Care, University Medical Center Utrecht, Utrecht, the Netherlands

Address correspondence to Meng Wang, Institute for Risk Assessment Sciences, Division Environmental Epidemiology, Utrecht University, PO Box 80178 3508 TD Utrecht, the Netherlands. Telephone: +31 (0)6 16697117. E-mail: M.Wang@uu.nl

Running title: Exposure modeling and lung function in children

Acknowledgment: The research leading to these results has received funding from the European Community's Seventh Framework Program (FP7/2007-2011): ESCAPE (grant agreement number: 211250). The PIAMA study is supported by The Netherlands Organization for Health

Research and Development; The Netherlands Organization for Scientific Research; The Netherlands Asthma Fund; The Netherlands Ministry of Spatial Planning, Housing, and the Environment; and The Netherlands Ministry of Health, Welfare, and Sport.

Competing financial interest: The authors declare they have no competing financial interest

Abstract

Background: There is limited knowledge about the extent to which estimates of air pollution effects on health are affected by the choice for a specific exposure model.

Objectives: We aimed to evaluate the correlation between long-term air pollution exposure estimates using two commonly used exposure modeling techniques [dispersion and land use regression (LUR) models], and in addition, to compare the estimates of the association between long-term exposure to air pollution and lung function in children using these exposure modeling techniques.

Methods: We used data of 1058 participants of a Dutch birth cohort study with measured forced expiratory volume in 1 second (FEV₁), forced vital capacity (FVC) and peak expiratory flow (PEF) at the age of 8 years. For each child, annual average outdoor air pollution exposure [nitrogen dioxide (NO₂), mass concentration of particulate matter with diameters <2.5 and <10 µm (PM_{2.5}, PM₁₀), and PM_{2.5} soot] were estimated for the current addresses of the participants by a dispersion and a LUR model, respectively. Associations between exposures to air pollution and lung function parameters were estimated using linear regression analysis with confounder adjustment.

Results: Correlations between LUR and dispersion modeled pollution concentrations were high for NO₂, PM_{2.5} and PM_{2.5} soot (R: 0.86-0.90) but low for PM₁₀ (R: 0.57). Associations with lung function were similar for air pollutant exposures estimated using LUR and dispersion modeling,

with the exception of associations of $PM_{2.5}$ with FEV_1 and FVC, which were stronger but less precise for exposures based on LUR compared with dispersion model.

Conclusions: Predictions from LUR and dispersion models correlated very well for $PM_{2.5}$, NO_2 and $PM_{2.5}$ soot but not for PM_{10} . Health effect estimates did not depend on the type of model used to estimate exposure in a population of Dutch children.

Introduction

Currently, there is an increased interest in estimating health effects with individual estimates of exposure taking into account intra-urban differences in air pollution levels (Brauer et al. 2008; Gehring et al. 2013; Molter et al. 2013) because of potential underestimation of health effects based on exposure assignment at community level (Jerrett et al. 2005a; Miller et al. 2007).

Land use regression (LUR) modeling and dispersion modeling have been extensively applied to characterize small-scale spatial variability of air pollution (Jerrett et al. 2005b). These approaches are based on distinctive methodological principles. LUR modeling combines data from air pollution measurements with data from geographic information systems (GIS) and stochastic modeling that exploits land use, geographic and traffic characteristics to explain spatial concentration variations at measured sites. Dispersion modeling relies on deterministic (e.g. Gaussian plume) equations and utilizes data on emission, meteorological conditions and topographical data to simulate the physico-chemical processes of transport and atmospheric chemistry when estimating outdoor air pollution concentrations (Jerrett et al. 2005b). Currently, comparisons of the prediction ability of LUR and dispersion models at cohort addresses are scarce (Beelen et al. 2010; Briggs et al. 2000; Cyrys et al. 2005; Dijkema et al. 2011; Gulliver et al. 2011; Marshall et al. 2008).

Recent studies have raised the importance of comparing alternative exposure metrics and relevant health effects in epidemiological studies (Baxter et al. 2013; Ozkaynak et al. 2013). The

impact of dispersion and LUR modeling on health effect estimates has only been investigated in a Californian study and a French study on the effects of air pollution on pregnancy outcomes.

These studies reported comparable results for the two modeling approaches (Sellier et al. 2014; Wu et al. 2011). However, only NO₂ and PM₁₀ models were compared in these studies.

The aims of this study were to 1) evaluate the agreement between long-term air pollution exposure estimates for NO₂, PM_{2.5}, PM_{2.5} soot, and PM₁₀ based on dispersion modeling and LUR modeling, and 2) to evaluate whether associations between long-term air pollution exposures and lung function in children differ depending on the exposure modeling approach used.

Methods

Study population

We included participants from the Dutch PIAMA (Prevention and Incidence of Asthma and Mite Allergy) birth cohort study. For the study, pregnant women were recruited in 1996-1997 during their second trimester of pregnancy from a series of areas in the north, West, and center of The Netherlands. Non-allergic pregnant women were invited to participate in a “natural history” study arm. Pregnant women identified as allergic through a validated screening questionnaire were primarily allocated to an intervention arm with a random subset allocated to the natural history arm. The study started with 3,963 newborns. Ethics approval to perform the study was obtained from the local authorized institutional review boards, and written informed consent was obtained from the parents or legal guardians of all participants. More information about the study design and population has been reported elsewhere (Brunekreef et al. 2002; Wijga et al. 2013).

The present analysis included participants from this cohort with successful lung function measurements at eight years of age; complete information on sex, age, height, and weight at the time of lung function measurement; and information on exposure to air pollution at the time of lung function measurement.

Lung function measurements

At age eight years, all children of allergic mothers and a random sample of children of non-allergic mothers (N=1552) were invited for a medical examination including pulmonary function testing, of which 1058 children responded with a visit to one of the study hospitals.

Children in the intervention and natural history groups were similar at age 8, and the intervention was shown not to have an effect on clinical outcomes (Gehring et al. 2012). In earlier work, we showed that combining these two groups did not affect associations between air pollution and lung function parameters (Gehring et al. 2013). A Jaeger pneumotachograph (Viasys Healthcare, USA) was used for pulmonary function testing. We investigated the following lung function parameters: force expiratory volume in 1 second (FEV₁), forced vital capacity (FVC), and peak expiratory flow (PEF). Body weight and height were measured during the medical examination by trained research staff using calibrated equipment (Gehring et al. 2013).

Air pollution exposure assessment

We used a local dispersion and an LUR model to estimate annual average air pollution concentrations of NO₂, PM_{2.5}, PM_{2.5} soot and PM₁₀ at the participants' home addresses at birth and at the time of the lung function tests.

- *LUR models* were developed using measurement data from the ESCAPE (European Study of Cohorts for Air Pollution Effects) study collected during 2008-2011. In brief, three two-week measurements within one year were conducted in at 40 (PM) and 80 (NO₂) locations respectively, throughout the Netherlands. The measurements were temporally adjusted using data from a continuous regional reference sites to generate annual average concentrations for LUR modeling. Model structures and performances have been shown in Table S1 (see Supplemental Materials). Details of the measurements and modeling efforts have been published elsewhere (Beelen et al. 2013; Cyrus et al. 2012; Eeftens et al. 2012a; Eeftens et al. 2012b). Detailed evaluations of model performances have been presented in a separate publication. (Wang et al. 2013).
- *The Dutch dispersion model* is a combination of a Gaussian plume model for the local scale and a Lagrangian trajectory model for long-distance transport (Van Jaarsveld 2004) which produces estimates of background concentrations of NO₂, PM_{2.5}, PM_{2.5} soot and PM₁₀ with a spatial resolution of 1×1 km. Annual average air pollution levels at the current address were based upon updated emission inventory data, actual meteorological parameters and dispersion modeling (Velders and Diederens 2009). Background concentrations of PM_{2.5} soot were derived from fractions of primary PM_{2.5} in combustion emissions depending on the type of fuel (biomass, coal, oil, diesel and petrol) as developed in the EUCAARI European research project (<http://www.atm.helsinki.fi/eucaari/>). Road traffic emissions were estimated by two standard Dutch models: “SRM1” a street canyon model for inner urban roads and

“SRM2” a line-source model for motorways. In SRM1, a source-receptor relationship has been specified as a function of the distance to the street axis for five different road types. SRM2 is based on a Gaussian plume model which takes into account vehicle-induced turbulence, the upwind roughness of the terrain, the presence of noise screens near the motorway and atmospheric stability. Emission factors for road traffic of regulatory components (NO_x/NO₂, PM_{2.5} and PM₁₀) are updated annually in the Netherlands, while for PM_{2.5} soot emission factors fractions of primary PM_{2.5} exhaust emissions have been used for diesel- and petrol-fuelled vehicles. More details about the applied dispersion models can be found elsewhere (Keuken et al. 2013; Wesseling 2003).

Statistical analysis

Pearson correlation coefficients were calculated to assess the agreement in estimated air pollution levels between different exposure modeling approaches and the agreement between the measured and dispersion modeled predicted concentrations at the ESCAPE sites. Paired t-test was applied to investigate the differences between the means of the distributions estimated by the two different models.

We used linear regression analyses with natural log (ln)-transformed lung function parameters as the dependent variables to estimate associations between continuous lung function parameters and air pollution levels at the birth address and at the home address at the time of the lung function measurement, as described elsewhere (Gehring et al. 2013). For each pollutant we specified models adjusted for sex, ln(age), ln(weight), and ln(height) only; and fully-adjusted

models that also included the following individual-level variables: ethnicity; parental allergies; parental education; breastfeeding; maternal smoking during pregnancy; smoking, mold/dampness, and furry pets in the child's home; recent respiratory infections. We used fully-adjusted models to compare associations with exposures estimated using the two different approaches. We also estimated associations using two-pollutant models that included both NO₂ and PM_{2.5} estimated using either the dispersion model or the LUR model, to determine whether mutually-adjusted effect estimates differed between the two exposure assessment methods. We estimated associations between air pollutants and lung function using fixed increments as used before in the ESCAPE study (Gehring et al. 2013). These increments were 10 µg/m³ for NO₂ and PM₁₀, 1 10⁻⁵/m for PM_{2.5} soot, 5 µg/m³ for PM_{2.5}. . Statistical significance was defined by a two-sided α -level $\leq 5\%$.

Results

Characteristics of the study population

The studied population included 1058 participants with average age of 8 years and with 50.4% female (Table 1). Mean (\pm SD) FEV₁, FVC and PEF were 1.80 \pm 0.25 L, 2.01 \pm 0.30 L and 3.79 \pm 0.63 L/sec, respectively.

Air pollution exposure

Table 2 presents the distributions of estimated annual average concentrations of air pollutants by different exposure models for the area of the PIAMA cohort. Although t-tests indicated significant differences between mean estimates based on dispersion and LUR models for all of

the pollutants ($p < 0.01$), mean values were similar. However, standard deviations (SD) were larger for dispersion model estimates than estimates from the LUR models. $PM_{2.5}$ soot concentrations were not directly comparable between LUR and dispersion models due to the use of different measurement techniques, with LUR estimates based on optical analysis reported as $10^{-5}/m$, and dispersion model estimates based on thermal analysis of elemental carbon reported as $\mu g/m^3$.

Performance evaluations of dispersion models with the measurements at the ESCAPE sites showed that the Pearson correlation coefficient was highest for NO_2 ($R=0.85$) and lowest for $PM_{2.5}$ ($R=0.54$) (Figure 1).

Figure 2 shows the scatter plots of the estimates between the dispersion and LUR models at the cohort addresses. Overall, the LUR model predictions correlated well with the estimates of the dispersion models for all the pollutants with an exception for PM_{10} ($R=0.57$).

Table 3 shows strongest correlations of concentrations between any pair of pollutants by the dispersion model ($R: 0.90-0.99$), followed by the measurements ($R: 0.75-0.93$), and the LUR model ($R: 0.63-0.91$). The values in the correlation matrix of air pollution predicted by the LUR model (LUR panel in table 3) were closest to the values in the correlation matrix between measured air pollutants (measured panel in table 3).

Associations between lung function and exposure estimated by different approaches

Overall, we found consistent negative associations between the lung function parameters FEV₁ and FVC and long-term exposure to air pollution estimated by both dispersion and LUR models at the current home addresses (Figure 3). The magnitudes of the effect estimates were similar for NO₂, PM_{2.5} soot and PM₁₀, but negative associations with PM_{2.5} were stronger for exposure estimates based on LUR compared with estimates based on dispersion modeling. 95% confidence intervals' (CI) ranges were similar for NO₂ and PM_{2.5} soot but larger for PM_{2.5} and PM₁₀ estimates by LUR models than for PM_{2.5} and PM₁₀ estimates by dispersion models. No significant associations were found between air pollution estimated by any of the exposure approaches and PEF. Effect estimates for concentrations estimated at the birth addresses were somewhat weaker than for the current addresses (results not shown). Associations with FVC were remained significant based on two-pollutant models for NO₂ and PM_{2.5} when exposures were estimated using the localized LUR models (–2.4% difference; 95% CI: –4.1, –0.8 and –9.5% difference; 95% CI: –18.2, –0.9 for NO₂ and PM_{2.5}, respectively) but were no longer significant when exposures were estimated using the dispersion models (–2.0 % difference; 95% CI: –4.2, 0.3 and –3.0 % difference; 95% CI: –7.8, 2.0, respectively).

Discussion

Model predictions of LUR and dispersion for PM_{2.5}, NO₂ and PM_{2.5} soot correlated very well. For PM₁₀ correlations between LUR and dispersion models were more moderate. For PM_{2.5} the variability in concentrations predicted by the LUR model was smaller than for the dispersion

model, whereas for NO₂ and PM_{2.5} soot variability was similar between the two models. LUR and dispersion predictions for PM_{2.5} soot are expressed in different units (PM_{2.5} absorbance in 10⁻⁵ m⁻¹ and µg/m³ for EC). If the average conversion factor in a recent review (Janssen et al. 2011) is applied (1 unit absorbance = 1.1 µg/m³ EC), the dispersion and LUR models predict slightly different absolute levels. The better agreement for NO₂ compared to PM mass agrees with a recent comparison between dispersion and LUR models (de Hoogh et al. 2014). The explanation offered by the authors was that both methods perform better for traffic-related pollutants than other pollutants, when appropriate input data are available. This interpretation is supported by our results for PM_{2.5} soot, which was not evaluated in the previous paper and is strongly affected by traffic emission in the Netherlands.

Previous studies have looked at correlations between LUR and dispersion modeled concentrations of NO₂ (Beelen et al. 2010; Cyrus et al. 2005; Marshall et al. 2008). Only the Cyrus et al. (2005) study documented a reasonably high Pearson correlation coefficient of 0.83 between the two models. The Pearson correlation coefficient of 0.90 we found compares favorably with the study as well as with recent multicenter study published by de Hoogh et al. (2014) which found a median Pearson correlation coefficient of 0.75 in 13 different European study areas. The Pearson correlation coefficient of 0.86 we found for PM_{2.5} was higher than the median Pearson correlation coefficient of 0.28 in de Hoogh et al. (2014) and our correlation (R) for PM₁₀ of 0.57 was also higher than the correlation (R) of 0.39 in that paper.

Comparison of the effect estimates of the association between long-term exposure to air pollution and lung function in children using LUR and dispersion models

This study shows that different exposure approaches revealed generally similar estimates of the association between long-term exposure to NO₂, and PM_{2.5} soot and lung function in a Dutch birth cohort. Effect estimates for PM_{2.5} and PM₁₀ were larger for the LUR estimates than for the dispersion estimates, but with wider confidence intervals. One explanation could be that the PM_{2.5} and PM₁₀ dispersion models did not predict the measured spatial variation of PM_{2.5} and PM₁₀ well (Figure 1). However, effect estimates were expressed over fixed concentration ranges. The dispersion models predicted wider concentration ranges for PM_{2.5} and PM₁₀ than the LUR models and as a consequence the 95% confidence intervals of the LUR modeled effect estimates were larger than those of the dispersion modeled effect estimates.

A strength of our study relates to the comparisons for PM_{2.5} and PM_{2.5} soot in addition to NO₂ and PM₁₀. Previous studies based on dispersion models were primarily focusing on NO₂ and PM₁₀ health effects (Downs et al. 2007; Jacquemin et al. 2013; Schultz et al. 2012; Sellier et al. 2014) with only one exception for PM_{2.5} in Oslo (Ofstedal et al. 2008). Moreover, our study employed well validated Dutch dispersion and LUR models with fine spatial resolution and reliable predictions of air pollution levels.

We estimated effects of a similar magnitude on lung function for all the strongly correlated air pollutants assessed by the dispersion models (Pearson correlation coefficients: 0.92-0.99, Table 3), probably because the sources are assumed largely the same: the dispersion model used

presumed fractions of PM emission factors derived from exhaust emissions and applied a scaling approach to estimate the PM metrics. In contrast, correlations between the air pollutants were weaker when estimated using the localized LUR models, and very similar to corresponding correlations between measured air pollutant concentrations. This was due to the fact that the LUR input data was from real measurements. Predictor variables in the LUR models frequently included population (or residence) density, a surrogate for sum of household activities (e.g. cooking and heating emissions) which were absent in the emission inventory for the dispersion modeling. Two-pollutant models with NO₂ and PM_{2.5} indicated more robust and independent effects of individual pollutants on FVC using the exposure estimates from the LUR models than from the dispersion models.

A limitation of this study is that we do not know how generalizable the findings of our analysis are to other cities and areas. We acknowledge that both dispersion and LUR models might produce exposure misclassifications and the degree of the impact depends on a variety of factors differentiating across geographical locations. For dispersion models, potential measurement errors may be affected by local emission inventory, the method of air pollution simulation, and the spatial resolution of grid cells. For LUR models, validity depends on the number of sampling sites, the quality of GIS variables, and the modeling procedures (Basagana et al. 2013).

In summary, LUR and dispersion model predictions for PM_{2.5}, NO₂ and PM_{2.5} soot were very well correlated (Pearson correlations 0.86–0.90). For PM₁₀ correlations between LUR and

dispersion models were more moderate. Health effect estimates did not depend on the type of model used to estimate exposure in the study population of Dutch children.

References

- Basagana X, Rivera M, Aguilera I, Agis D, Bouso L, Elosua R, et al. 2012. Effect of the number of measurement sites on land use regression models in estimating local air pollution. *Atmos Environ* 54:634-642.
- Baxter LK, Dionisio KL, Burke J, Ebel Sarnat S, Sarnat JA, Hodas N, et al. 2013. Exposure prediction approaches used in air pollution epidemiology studies: key findings and future recommendations. *J Expo Sci Environ Epidemiol* 23(6):654-9.
- Beelen R, Voogt M, Duyzer J, Zandveld P, Hoek G. 2010. Comparison of the performances of land use regression modelling and dispersion modelling in estimating small-scale variations in long-term air pollution concentrations in a Dutch urban area. *Atmos Environ* 44:4614-4621.
- Beelen R, Hoek G, Vienneau D, Eeftens M, Dimakopoulou K, Pedeli X, et al. 2013. Development of NO₂ and nox land use regression models for estimating air pollution exposure in 36 study areas in Europe - the ESCAPE project. *Atmos Environ* 72:10-23.
- Brauer M, Lencar C, Tamburic L, Koehoorn M, Demers P, Karr C. 2008. A cohort study of traffic-related air pollution impacts on birth outcomes. *Environ Health Perspect* 116:680-686.
- Briggs DJ, de Hoogh C, Guiliver J, Wills J, Elliott P, Kingham S, et al. 2000. A regression-based method for mapping traffic-related air pollution: Application and testing in four contrasting urban environments. *Sci Total Environ* 253:151-167.
- Brunekreef B, Smit J, de Jongste J, Neijens H, Gerritsen J, Postma D, et al. 2002. The prevention and incidence of asthma and mite allergy (piama) birth cohort study: Design and first results. *Pediatr Allergy Immunol* 13 Suppl 15:55-60.
- Cyrys J, Hochadel M, Gehring U, Hoek G, Diegmann V, Brunekreef B, et al. 2005. GIS-based estimation of exposure to particulate matter and NO₂ in an urban area: Stochastic versus dispersion modeling. *Environ Health Perspect* 113:987-992.

- Cyrus J, Eeftens M, Heinrich J, Ampe C, Armengaud A, Beelen R, et al. 2012. Variation of NO₂ and NO_x concentrations between and within 36 European study areas: Results from the ESCAPE study. *Atmos Environ* 62:374-390.
- de Hoogh K, Korek M, Vienneau D, Keuken M, Kukkonen J, Nieuwenhuijsen M, et al. 2014. Comparing land use regression and dispersion modeling to assess residential exposure to ambient air pollution for epidemiological studies. *Environ Intern*, in press.
- Dijkema MB, Gehring U, van Strien RT, van der Zee SC, Fischer P, Hoek G, et al. 2011. A comparison of different approaches to estimate small-scale spatial variation in outdoor NO₂ concentrations. *Environ Health Perspect* 119:670-675.
- Downs SH, Schindler C, Liu LJ, Keidel D, Bayer-Oglesby L, Brutsche MH, et al. 2007. Reduced exposure to PM₁₀ and attenuated age-related decline in lung function. *N Engl J Med* 357:2338-2347.
- Eeftens M, Beelen R, de Hoogh K, Bellander T, Cesaroni G, Cirach M, et al. 2012a. Development of land use regression models for PM_{2.5}, PM_{2.5} absorbance, PM₁₀ and PM_{coarse} in 20 European study areas; results of the ESCAPE project. *Environ Sci Technol* 46:11195-11205.
- Eeftens M, Tsai M, Ampe C, Anwander B, Beelen R, Cesaroni G, et al. 2012b. Spatial variation of PM_{2.5}, PM₁₀, PM_{2.5} absorbance and PM_{coarse} concentrations between and within 20 European study areas and the relationship with NO₂ - results of the ESCAPE project. *Atmos Environ* 62:303 - 317.
- Gehring U, de Jongste JC, Kerkhof M, Oldewening M, Postma D, van Strien RT, et al. 2012. The 8-year follow-up of the PIAMA intervention study assessing the effect of mite-impermeable mattress covers. *Allergy* 67(2):248-56.
- Gehring U, Gruzjeva O, Agius RM, Beelen R, Custovic A, Cyrus J, et al. 2013. Air pollution exposure and lung function in children: The ESCAPE project. *Environ Health Perspect* 121:1357-1364.

- Gulliver J, de Hoogh K, Fecht D, Vienneau D, Briggs D. 2011. Comparative assessment of GIS-based methods and metrics for estimating long-term exposures to air pollution. *Atmos Environ* 45:7072-7080.
- Jacquemin B, Lepeule J, Boudier A, Arnould C, Benmerad M, Chappaz C, et al. 2013. Impact of geocoding methods on associations between long-term exposure to urban air pollution and lung function. *Environ Health Perspect* 121:1054-1060.
- Janssen NAH, Hoek G, Simic-Lawson M, Fischer P, van Bree L, ten Brink H, et al. 2011. Black carbon as an additional indicator of the adverse health effects of airborne particles compared with PM₁₀ and PM_{2.5}. *Environ Health Persp* 119:1691-1699.
- Jerrett M, Burnett RT, Ma R, Pope CA, 3rd, Krewski D, Newbold KB, et al. 2005a. Spatial analysis of air pollution and mortality in los angeles. *Epidemiology* 16:727-736.
- Jerrett M, Arain A, Kanaroglou P, Beckerman B, Potoglou D, Sahuvaroglu T, et al. 2005b. A review and evaluation of intraurban air pollution exposure models. *J Expo Sci Environ Epidemiol* 15:185-204.
- Keuken MP, Zandveld P, Jonkers S, Moerman M, Jedynska AD, Verbeek R, et al. 2013. Modelling elemental carbon at regional, urban and traffic locations in the Netherlands. *Atmos Environ* 73:73-80.
- Marshall JD, Nethery E, Brauer M. 2008. Within-urban variability in ambient air pollution: Comparison of estimation methods. *Atmos Environ* 42:1359-1369.
- Miller KA, Siscovick DS, Sheppard L, Shepherd K, Sullivan JH, Anderson GL, et al. 2007. Long-term exposure to air pollution and incidence of cardiovascular events in women. *N Engl J Med* 356:447-458.
- Molter A, Agius RM, de Vocht F, Lindley S, Gerrard W, Lowe L, et al. 2013. Long-term exposure to PM₁₀ and NO₂ in association with lung volume and airway resistance in the maas birth cohort. *Environ Health Perspect* 121:1232-1238.
- Oftedal B, Brunekreef B, Nystad W, Madsen C, Walker SE, Nafstad P. 2008. Residential outdoor air pollution and lung function in schoolchildren. *Epidemiology* 19:129-137.

- Ozkaynak H, Baxter LK, Dionisio KL, Burke J. 2013. Air pollution exposure prediction approaches used in air pollution epidemiology studies. *J Expo Sci Environ Epidemiol* 23: 566-572.
- Schultz ES, Gruzieva O, Bellander T, Bottai M, Hallberg J, Kull I, et al. 2012. Traffic-related air pollution and lung function in children at 8 years of age: A birth cohort study. *Am J Respir Crit Care Med* 186:1286-1291.
- Sellier Y, Galineau J, Hulin A, Caini F, Marquis N, Navel V, et al. 2014. Health effects of ambient air pollution: Do different methods for estimating exposure lead to different results? *Environ Int* 66: 165-173.
- Van Jaarsveld JA. 2004. The operational priority substances model. Report 500045001/2004, national Institute for Public health and the Environment (RIVM), Bilthoven, The Netherlands:156 pp.
- Velders GJM, Diederik HSMA. 2009. Likelihood of meeting the eu limit values for NO₂ and PM₁₀ concentrations in the Netherlands. *Atmos Environ* 43:3060-3069.
- Wang M, Beelen R, Basagana X, Becker T, Cesaroni G, de Hoogh K, et al. 2013. Evaluation of land use regression models for NO₂ and particulate matter in 20 European study areas: The ESCAPE project. *Environ Sci Technol* 47:4357-4364.
- Wesseling JP. 2003. An intercomparison of the TNO traffic model, field data and wind tunnel measurements. TNO, Utrecht, The Netherlands:Report 2003/2207.
- Wijga AH, Kerkhof M, Gehring U, de Jongste JC, Postma DS, Aalberse RC, et al. 2013. Cohort profile: The prevention and incidence of asthma and mite allergy (piama) birth cohort. *Int J Epidemiol*.
- Wu J, Wilhelm M, Chung J, Ritz B. 2011. Comparing exposure assessment methods for traffic-related air pollution in an adverse pregnancy outcome study. *Environ Res* 111:685-692.

Table 1. Description of the study population and lung function measurements.

Variable	N	Percent or Mean±SD
Female sex	1058	50.4
Respiratory infections	1054	24.2
Allergic mother	1058	66.1
Allergic father	1055	33.3
Dutch ethnicity ^a	1044	95.7
High maternal SES	1055	38.6
High paternal SES	1043	42.9
Breastfeeding	1058	52.6
Mother smoked during pregnancy	1044	15.4
Smoking at child's home ^b	990	15.7
Mold/dampness in child's home ^b	985	28.8
Furry pets in home ^b	970	49.9
Height (cm)	1058	132.90±5.60
Weight (kg)	1058	28.90±4.80
Age (years)	1058	8.10±0.30
FEV1 (L)	1058	1.80±0.25
FVC (L)	1058	2.01±0.30
PEF (L/sec)	1058	3.79±0.63

^aEthnicity: Dutch; ^bAt the age of the lung function measurement.

Table 2. Descriptive of estimated annual average air pollution levels (N=1058).

Models	Mean±SD	Min	P25	Median	P75	Max
NO₂ (µg/m³)						
Dispersion	23.0±8.2	9.8	14.9	23.7	28.1	44.8
LUR	22.1±6.3	9.4	17.5	22.4	26.2	52.1
PM_{2.5} (µg/m³)						
Dispersion	15.9±1.9	12.6	13.6	16.8	17.3	20.0
LUR	16.3±0.6	14.9	15.6	16.5	16.7	19.3
PM_{2.5} soot^a						
Dispersion	0.7±0.2	0.3	0.4	0.7	0.8	1.6
LUR	1.2±0.2	0.9	1.0	1.2	1.3	2.1
PM₁₀ (µg/m³)						
Dispersion	23.8±2.3	19.7	21.1	24.9	25.5	28.6
LUR	24.8±1.0	23.7	24.0	24.5	25.1	29.8

^aPM_{2.5} soot estimated by dispersion model using thermal detection method (µg/m³) and by LUR models using optical method (10⁻⁵/m).

Table 3. Pearson Correlation Coefficients (R) between measured air pollution concentrations at the ESCAPE monitoring sites (NO₂: N=40 and PM: N=80) or modeled pollutants at PIAMA addresses (n=1058), respectively.

Models	Pollutants	NO₂	PM_{2.5}	PM_{2.5}soot	PM₁₀
Measured ^a	NO ₂	1			
	PM _{2.5}	0.75	1		
	PM _{2.5} soot ^b	0.93	0.84	1	
	PM ₁₀	0.86	0.85	0.86	1
Dispersion	NO ₂	1			
	PM _{2.5}	0.92	1		
	PM _{2.5} soot ^b	0.95	0.93	1	
	PM ₁₀	0.90	0.99	0.92	1
LUR	NO ₂	1			
	PM _{2.5}	0.75	1		
	PM _{2.5} soot ^b	0.91	0.86	1	
	PM ₁₀	0.78	0.63	0.88	1

^aMeasured: Measured concentrations at the ESCAPE sites for LUR model development in The Netherlands. ^bPM_{2.5} soot estimated by dispersion model using thermal detection method (µg/m³) and by LUR models using optical method (10⁻⁵/m).

Figure Legends

Figure 1 Pearson correlation coefficients of dispersion modeled NO₂ (N=80), PM_{2.5}, PM_{2.5} soot, PM₁₀ (N=40) with the same pollutants measured at the ESCAPE sites.

Figure 2 Pearson correlation coefficients of air pollution estimates between localized dispersion and LUR models at the PIAMA addresses (N=1058).

Figure 3 Adjusted associations (model b) of annual levels of air pollutants estimated by dispersion and LUR modeling approaches with FEV₁, FVC, and PEF level (N=1058) at the PIAMA current addresses. The increment of each pollutant is calculated by 10 µg/m³ for NO₂ and PM₁₀, 1 10⁻⁵/m for PM_{2.5} soot, 5 µg/m³ for PM_{2.5}.

Figure 1.

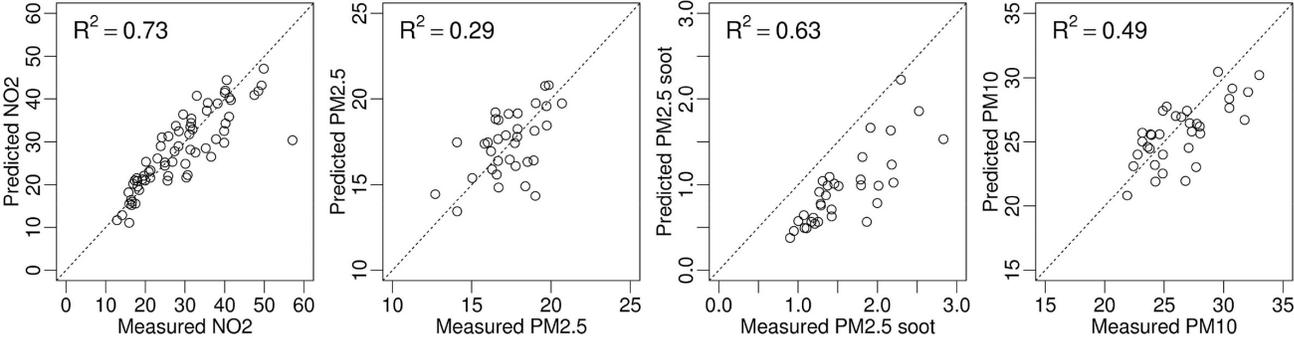


Figure 2.

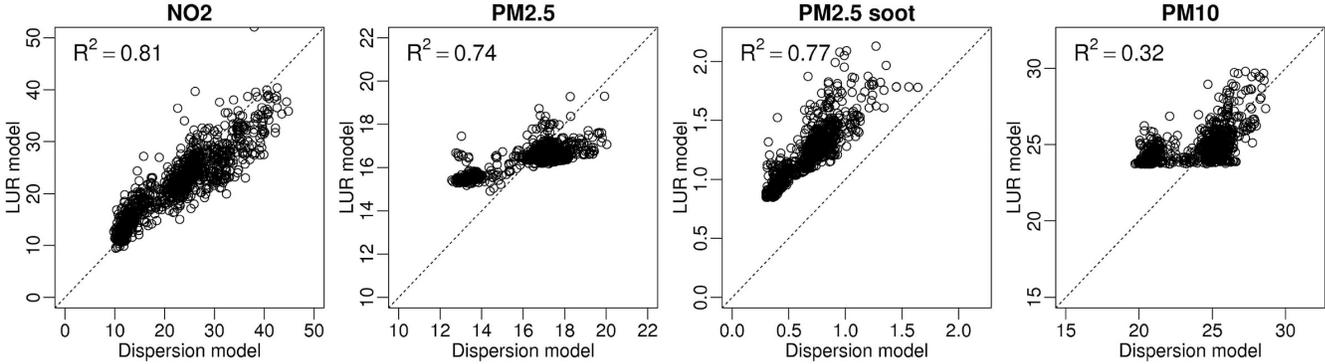


Figure 3.

