Traffic Related Air Pollution: Spatial Variation, Health Effects and Mitigation Measures

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Traffic Related Air Pollution: Spatial Variation, Health Effects and Mitigation Measures

Verkeersgerelateerde Luchtverontreiniging:
Ruimtelijke Variatie, Gezondheidseffecten
en Maatregelen
(met een samenvatting in het Nederlands)

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Chapter 1

General Introduction

Air pollution is probably the most intensely studied field in today's environmental health research. The extensive body of literature on health effects associated with air pollution exposure has led to the prioritization of air pollution as a public health risk factor,[1] and has resulted in air quality regulations worldwide.[e.g.2-4] However, even at concentrations below limit values, air pollution still has a significant health impact. Therefore, the debate on air quality policy is ongoing.

The policy debate focuses on fundamental questions; which government tier has the responsibility and which tier has the ability to make a difference? Moreover, the necessity to take action is often disputed. In that respect, reliable quantitative information on the health impact of air pollution is very important. The debate furthermore includes discussions of the relevance of specific components of air pollution to the observed health effects, the suitability of those specific components as targets for air quality regulations, the levels at which limit values should be set and the effectiveness of potential mitigation measures. Although in essence this is a debate in the political arena, science plays an important role in providing a solid evidence basis for the decision makers.

AIR POLLUTION AND ITS HEALTH EFFECTS

Air pollution

Air pollution is a complex mixture of many gaseous and particulate components originating from a large variety of natural and anthropogenic sources. Among anthropogenic sources, industry and traffic are most prominent.[1,5-7] From a health perspective, air pollution is most relevant when the population is exposed, like in residential areas. The main source of air pollution in residential areas in the Netherlands is traffic.[7,8] Traffic related air pollution originates from combustion and wear of tires, brakes and road surface and consists of many different components, such as soot, nitrogen oxides and particulate matter. Nitrogen dioxide (NO_2) is often considered an indicator of this mixture.[9]

The air pollution concentration at a specific location is determined by the presence of sources (such as traffic and industry), spatial characteristics (ranging from street and building configuration to the size and elevation of a city and its surroundings) and atmospheric processes (such as long-range transport of air pollution and meteorology).[10] Due to the variation in these characteristics, temporal and spatial differences in air pollution can be very large.[7-9,11,12] When looking at longer time periods (months or years), the spatial variation within a city is often larger than the temporal variation.[13-15]

Exposure assessment in epidemiological studies

To estimate exposure of participants in epidemiological studies, different methods are being used. In studies on the short-term (days to weeks) effects of air pollution, information on the temporal variation of air pollution is needed. Such data is often obtained from monitoring networks.[e.g.16] Exposure of participants in these health studies is estimated by the concentration measured at the monitoring site nearest to the participants' residential address.[e.g.6,17-23]

Exposure assessment in long-term (years) health effects studies started by assigning the annual mean concentration from monitoring data by the participants city of residence.[24,25] Later, approaches to estimate the variation of air pollution within cities were used. Since traffic is generally the dominant source of this small scale (meters) variation,[7,8,26-28] many studies used indicators of traffic near the residential address.[e.g.29,30] Examples of such indicators are proximity of different types of roads, traffic flow (number of cars per day) and/or its composition (cars, trucks) derived from questionnaires or Geographic Information Systems (GIS). These indicators, however, do not account for influential factors such as spatial situation, meteorology and urbanization. Modeled air pollution concentrations, accounting for such factors, may render a more valid estimation of exposure than indicators of nearby traffic.[31] Therefore, modeling techniques such as Land Use Regression (LUR)

and dispersion modeling became increasingly popular in epidemiological studies in the past few years.[e.g.14,32] Participants' long-term average exposure to air pollutants such as NO_2 (proxy of the traffic related air pollution mixture) is often estimated by applying these modeling techniques to the residential address.[e.g.9,14,32]

The estimated air pollution concentrations from dispersion or LUR modeling are quite close to measured concentrations at selected sites[14,28] and validity of this approach to estimate exposure has been shown.[e.g.33,34] Nevertheless, some misclassification may occur due to assumptions made. First, this approach assumes outdoor concentrations being representative for indoor exposure. Secondly, since exposure of an individual takes place at several locations of which residence is only one, exposure at a residential address is merely an indicator of long-term exposure. Furthermore, this approach does not account for personal activities such as occupation or time spent in traffic, which may influence exposure remarkably.

LUR models are increasingly popular in epidemiological studies as those models are a relatively simple method to extrapolate a limited number of measurements to a larger population. For the purpose of air quality management and regulation, however, dispersion modeling[10] is the method of choice in the Netherlands. Dispersion models are more complex models, for which a lot of input data is needed. Dispersion models furthermore have limitations in their applicability. The Dutch CAR model,[10] for instance, limits estimations to a maximum of 50 meters from a road for which input data is available. Only few comparisons have been made between these two modeling techniques.[26,35,36]

Air pollution health effects

Since the 1980s, the health effects of air pollution have been intensely investigated in episode and time-series studies (also called '*short-term studies*'), which showed that episodes of elevated air pollution levels were associated with increases in mortality, hospital admissions, and symptoms.[6,17-23] In the past decade, focus has shifted towards the health effects of long-term exposure to air pollution (also called '*long-term studies*'), and traffic related air pollution became a main priority.[37-40]

The first long-term studies showed that increased long-term average air pollution exposure was associated with increased mortality.[24,25] As air pollution variation may be larger within cities than between cities, later studies[e.g.37,41,42] used more sophisticated methods for the estimation of long-term exposure, such as LUR or dispersion modeling. Health effects shown to be associated with long-term exposure to air pollution are respiratory disease, such as asthma and chronic obstructive pulmonary disease (COPD), cardiovascular symptoms and disease, such as arteriosclerosis and ischemic heart disease (IHD), and mortality for these cardiopulmonary causes.[e.g.43-47] A hypothesis for

the biological mechanism underlying these health effects is that traffic related air pollution triggers systemic oxidative stress and inflammation in for instance endothelial cells and macrophages.[6,48] Such biological processes might also play a role in diseases such as arthritis and type 2 diabetes (also known as adult-onset diabetes), although data supporting an association with air pollution are limited.[49-53] Studies furthermore showed evidence for associations between air pollution and lung cancer,[e.g.47,54,55] lung development,[e.g.56,57] birth outcomes [e.g.42,58-61] such as preterm birth and low birth weight and cognition.[62]

Long-term studies showed larger effects of air pollution on cardiopulmonary mortality than short-term studies. This is explained by those cases of death in which air pollution is related to chronic disease leading to frailty but unrelated to timing of death, which are not detected in short-term studies.[63] Hospital admissions for cardiopulmonary causes only occasionally have been the subject of long-term studies.[41,64-69] Since the majority of these long-term studies on hospitalization have furthermore been done in specific sub-populations (e.g. children[64,69]), the health impact of long-term exposure to traffic related air pollution in the general population, remains largely unknown.

AIR POLLUTION POLICY IN THE NETHERLANDS

The European Union (EU) has applied air quality regulations ever since the 1970's, as "humans can be adversely affected by exposure to air pollutants in ambient air".[70] Under the current EU legislation (Directive 2008/50/EC), member states should empirically assess the ambient pollution levels. When concentrations above the EU limit values[3] are observed, air quality plans have to be developed to ensure compliance with the limit values.

A 2008 evaluation showed that air pollution levels exceeded the announced limit values for a large part of the country.[71] Therefore a national action plan (NSL: *Nationaal Samenwerkingsprogramma Luchtkwaliteit*) was prepared by the national government. The action plan comprises a number of general measures, such as traffic management at freeways, stimulation of cleaner vehicles, and a series of measures listed in the regional action plans (RSL: *Regionaal Samenwerkingsprogramma Luchtkwaliteit*, under provincial responsibility). Regional action plans consist of several municipal action plans listing local measures such as low emission zones, traffic management at specific crossways, limitation of driving speed and promotion of public transport and bicycle use. As part of the NSL, all aforementioned authority tiers are furthermore committed to provide data on local sources of air pollution and/or their emission (e.g. the number of cars at the main roads or the emission of a power plant) on a yearly basis. Using this information, the national government estimates past and future air pollution concentrations at all locations in The Netherlands, using a combination of modeling techniques (Monitoring tool: www.nsl-monitoring.nl). This monitoring also incorporates current and future spatial plans (such as neighborhood or road expansion and new business parcs). Based on the monitoring results, the action plans may be revised in order to meet EU limit values by the due date.

By applying this staged model over different authority tiers, responsibility for improving air quality has been assigned towards the local level. Local action plans are in part funded by the national government. As NSL has successfully been applied to get derogation from the EU (delay of the date at which the Netherlands will have to meet the EU limit values), all Dutch authorities involved are legally obliged to carry out their action plans.

In general, municipal action plans are prepared by a collaboration of municipal departments, such as the departments of environment and infrastructure, and the Public Health Service (GGD). Important factors when preparing such action plans are local air pollution levels, the contribution of local sources, the availability of tools to change the current situation and, last but not least, the political sense of urgency to take action.

EVIDENCE BASED PUBLIC HEALTH

The research presented in this thesis was conducted by the Public Health Service of Amsterdam in collaboration with the Institute for Risk Assessment Sciences of Utrecht University within the framework of the Academic Collaborative Center for Environmental Health. The Academic Collaborative Center for Environmental Health was funded by the Netherlands Organization for Health Research and Development (ZonMW) within the 'Academic Collaborative Centers' program. The aim of this program is to encourage academic research with high practical relevance in public health and to improve *evidence based public health* in Dutch Public Health Services.

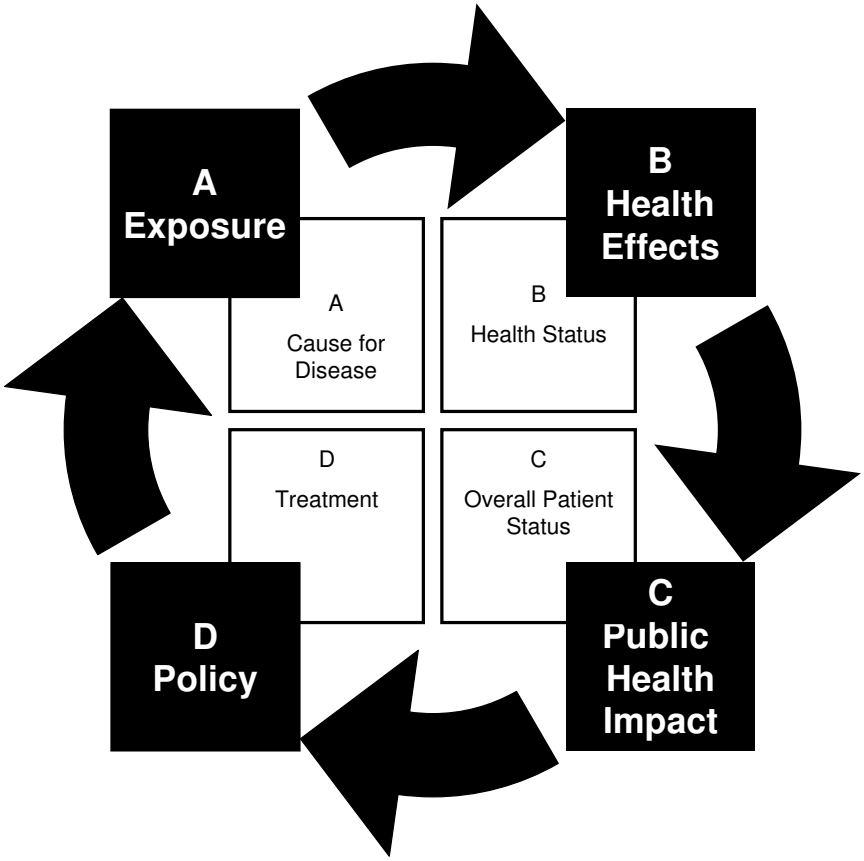

Figure 1. The cycle of clinical work (white) and public health (black) underlying 'evidence based medicine', and 'evidence based public health', respectively. In clinical work, cause(s) (inner Box A) of health problems (B) results in a doctors' diagnosis. The assessment of the overall situation of the patient (C) determines the treatment strategy (D) to positively affect the causes (A) and/or health (B). In public health, some exposure (A) may causes health problems in the population (B). The assessment of its relevance (C) may result in a policy (D) to abate the exposure (A) and improve public health (B). Ideally, all steps in both cycles are based on scientific evidence – evidence based medicine and public health, respectively. *Adapted from Künzli and Perez*[72]

Evidence based medicine is a well established paradigm.[73] In brief, evidence based medicine means that clinical expertise is integrated with the best available systematic research, and that decisions are made with the conscientious, explicit, and judicious use of the current best evidence. As stated by Künzli and Perez,[72] evidence based public health is the natural extension of evidence based medicine to the public health field. Their model of evidence based public health is shown in Figure 1.

The main complicating factor in the much less established 'evidence based public health' is that it deals with populations rather than individual patients. As a consequence there is a considerable difference in methods, actors, responsibilities and indicators of result. Especially the large variety of actors in the public health cycle, ranging from health professionals to technical engineers and governors at different authority tiers, poses a challenge for the Academic Collaboration Center of Environmental Health.

For air quality policy in the Netherlands, the different phases of the aforementioned cycle are carried out by different organizations. At the local level, for instance, the characterization of exposure (A) is done by engineers of the department of environment. The assessment of possible health effects (B) and their relevance (C) is done by Public Health Services. Policies to abate exposure (phase D) are carried out by different departments within a municipality. In Amsterdam, for example, traffic reduction measures are taken by the department of traffic and infrastructure, technical measures to reduce dust emission in coal handling are taken by the port of Amsterdam, mitigation measures to reduce exposure of vulnerable members of the population are taken by the department of youth and education, etcetera. For certain other policies, including those policies involving traffic management at freeways, national government bodies are in charge. Decision making processes may therefore become rather complicated.

Environmental health professionals from Public Health Services can be involved in all phases of the aforementioned cycle. By providing evidence based expertise they can contribute importantly to healthy air quality policies.

THIS THESIS

The primary objective of this thesis is to provide *evidence* for the association between health effects and traffic related air pollution, and potential mitigation measures relevant to Public Health Services in the Netherlands. The research in this thesis comprises three elements closely related to the work of Public Health Services: assessment of exposure (Chapter 2), its health effects (Chapters 3 and 4) and evaluation of mitigation measures (Chapter 5 and 6).

The aim of the first part of this thesis (Chapter 2) is to estimate the spatial variation in long-term average air pollution concentrations related to traffic in the West of the Netherlands. Chapter 2 describes three different approaches to model small scale variation of long-term exposure to traffic related air pollution. Two of these approaches were developed within the framework of this thesis, the third approach is the model required by national legislation. The approaches were evaluated regarding their ability to estimate concentrations at a number of independent measurement sites in Amsterdam.

The objective in the second part of this thesis (Chapters 3 and 4) is to explore the relationship between long-term exposure to traffic-related air pollution and morbidity. In Chapter 3, the relation between long-term exposure to traffic related outdoor air pollution and hospital admission for cardiovascular and respiratory disease in the total population of the West of the Netherlands is evaluated. Chapter 4 describes the associations between type 2 diabetes prevalence, as obtained through extensive screening of all 50-75 year old inhabitants of the region of Westfriesland, and different proxies of long-term exposure to traffic related air pollution.

The third aim is to assess the effectiveness of measures to reduce exposure to traffic related air pollution (Chapters 5 and 6). In Chapter 5 the effectiveness of a limitation of the maximum driving speed at the Amsterdam ring freeway in reducing the contribution of traffic emissions to the concentrations of several pollutants is evaluated. Chapter 6 describes to what extent different ventilation systems fitted with fine particle filters were able to reduce infiltration of outdoor air pollution into a school near a freeway.

In Chapter 7 the main findings of the studies presented in this thesis are discussed with respect to the framework of *evidence based public health*, together with the implications of the findings of this thesis. The experience and insights resulting from this work being done in the Academic Collaboration Centre for daily ‘air quality’-practice in Public Health Services are discussed.

Chapter 2

A Comparison of Different Approaches to Estimate Small Scale Spatial Variation in Outdoor NO_2 Concentrations

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ABSTRACT

In epidemiological studies, small scale spatial variation in air quality is estimated using land-use regression (LUR) and dispersion models. An important issue of exposure modeling is the predictive performance of the model at unmeasured locations.

In this study, we aimed to evaluate the performance of two LUR models (large area and city specific) and a dispersion model in estimating small-scale variations in nitrogen dioxide (NO_2) concentrations.

Two LUR models were developed based on independent NO_2 monitoring campaigns performed in Amsterdam and in a larger area including Amsterdam, the Netherlands, in 2006 and 2007, respectively. The measurement data of the other campaign were used to evaluate each model. Predictions from both LUR models and the CAR dispersion model were compared against NO_2 measurements obtained from Amsterdam.

The large-area and the city-specific LUR models provided good predictions of NO_2 concentrations [percentage of explained variation (R^2) = 87% and 72%, respectively]. The models explained less variability of the concentrations in the other sampling campaign, probably related to differences in site selection, and illustrating the need to select sampling sites representative of the locations to which the model will be applied. More complete traffic information contributed more to a better model fit than detailed land-use data. Dispersion-model estimates for NO_2-concentrations were within the range of both LUR estimates.

INTRODUCTION

Many epidemiological studies have shown that air pollution is associated with health effects such as cardiopulmonary morbidity and mortality.[6,17] Currently, the land use regression (LUR) method[74] is increasingly being used for estimating small scale variations in air pollution concentrations in European and North American epidemiological studies.[e.g.14,32] The quality of LUR-based exposure estimation of outdoor air pollution concentrations largely relies on coverage and quality of specific monitoring campaigns and the geographic data to support them. Information extractable from land use maps depends on resolution, which is often limited. Another common limitation is that digital geographic traffic information is usually not readily available, but needs to be collected from local and national authorities and linked to digital road maps.

Most LUR studies report good performance of prediction models, expressed as the percentage explained variation (R^2).[14] Validation is often performed by internal leave-one-out cross-validation from the database used for developing the model. An independent dataset for model validation is not often available. We had two independent datasets of NO_2 measurements in the city of Amsterdam available that allowed us to evaluate the performance of the LUR models in predicting concentrations from the dataset not used for model development.

Dispersion modeling is another method to estimate small scale variations in air pollution concentrations. In the Netherlands, the CAR dispersion model[10] is widely used for the purpose of air quality management and regulation. Few comparisons have been made between dispersion and LUR models.[26,35,75]

The aims of our study were 1) to evaluate the value of complete traffic data that is not standard available and high resolution land use data for improving LUR model performance, 2) to evaluate the performance of two LUR models with independent sets of NO_2 measurements, and 3) to compare the ability of the CAR dispersion model and two LUR models to estimate small scale variations in NO_2 concentrations.

METHODS

Study areas

The study area for the large area LUR is situated in the north-western part (6,000 km^2) of the Netherlands (Supplemental Material, Figure 1). It includes rural, suburban and urban areas among which major cities such as Amsterdam and Rotterdam. With 4.2 million inhabitants in almost 2 million households, this part of the Netherlands is densely populated and has a dense road network. The study area for the city specific LUR model consists of the greater city of Amsterdam (1 million inhabitants, 170 km^2, Supplemental Material, Figure 1).

Air quality

Two independent NO_2-monitoring campaigns were done. The campaign for the *large area model* took place in 2007 using Ogawa badges (Ogawa & co, Pompano beach, Florida). A total of 60 badges were distributed among traffic dominated urban sites (n=18), urban non-traffic sites (n=34) and rural sites (n=8). Eight additional badges were located at rural sites outside the study area to minimize border-effects when calculating background concentrations.[76] All badges were located at the façade of residential buildings and away from local sources other than traffic. One week monitoring (7 days +/- 3 hours, all starting at the same day) was performed in all four seasons (January, April, June and October). Sampling and analysis were done as described earlier.[33]

For the *city specific model*, data for the year 2006 from a routinely performed passive NO_2 monitoring program with Palmes tubes[77] in Amsterdam was used.[78] In contrast with the other campaign, Palmes tubes were not only located at the façade of residential buildings but also at (lamp)posts. As in the large area campaign, all sites were away from local sources other than traffic, measurements near hotspots such as traffic lights and bus stations were excluded. Tubes were put up at 62 locations in Amsterdam of which 25 were traffic dominated and 37 were not. Monitoring took place continuously. Tubes were replaced every 28 days and analyzed as described in Palmes et al.,[77] resulting in full-year data.

All monitoring locations were geo coded using a national GIS database (ACN) containing coordinates for all home addresses in the Netherlands. References for the geographical databases (including traffic and land use data) used in this study can be found in Supplemental Material, Annex A.

Traffic data

Geographical information on traffic flow was collected from all authorities responsible for traffic management in the study area. The National government is responsible for the freeways; Provinces for the highways, main connection routes and other country roads in rural areas; and municipalities

for all other roads and streets. In the large study area, there were 93 sources of traffic data: the national department of traffic, 3 provinces and 89 municipalities. All authorities provided data on traffic flow and traffic composition by road segment. For all freeways data were obtained from continuous automated counters, for most other roads traffic flow was estimated from yearly two to four week automated counts in combination with traffic models, most commonly OmniTRANS (www.omnitrans-international.com). Data were provided for 94.1% of the nationally, 58.2% of the provincially, and 48.1% of the municipally managed road length. Most authorities provided traffic data for the years 2004 (52% of the available road segments), 2005 (13%) or 2006 (31%). When no data for 2006 were available, data from the most recent previous year were used to estimate the expected 2006 traffic flow.[76] If no data were provided, quiet roads or small streets were assigned a minimal flow of 1225 vehicles per 24 hours[76] (applied to none of the nationally, 31.2% of the provincially and 44.6% of the municipally managed road length). Altogether, for 87.3% of the total road length in the study area traffic flow was available, for 86.9% also information on traffic composition was available. These data were linked to a geo-database of all roads in the Netherlands (NWB). For each measurement site we defined traffic flow in circular buffers (100m and 250m), distance to and traffic flow at the nearest road (distinguishing total and heavy duty traffic) for different road types (all roads, busy roads (traffic load of more than 5,000 vehicles per 24 hours), main roads (load of more than 10,000 vehicles per 24 hours), and freeways). All distances to roads were log transformed, a priori, to allow for the non-linear (exponential) decay of air pollution concentrations with distance to the road. All flow-variables were categorized by distance (25, 50, 100, 250 and 500m). All traffic variables used were derived using ArcGIS software (version 9, ESRI, Redlands CA, USA).

Land use data

Information on land use in the large study area was derived from the European land use database CORINE, available at a 100m*100m grid. For ten different categories (residential, industry, transport, port, airport, waste/construction, urban green, forest, agriculture, combined green space (urban green, forest and agriculture)) the percentage of land use in circular buffers with radii of 300 m, 1 km and 5 km around the monitoring sites were calculated (following,[76,79] adapted to the resolution of the available data when necessary, resulting in 30 land use variables).

For the city specific model, the percentage land use in 2006 from a 5m*5m grid map was calculated for circular buffers with radii of 25, 50, 100, 250 and 500m. Land use categories available in this detailed grid were railroad, road, freeway, building, business, industry, greenhouses, agriculture, urban green, forest, playground, sports ground, other tiled surfaces, water,

combined green space (agriculture, urban green, forest, play- and sports ground) and combined roads (road, highway and freeway).

For the large area and the city specific LUR-models, the number of inhabitants in circular buffers with radii of 100m, 300m, 1km and 5 km was calculated from the national population density database. The larger buffer sizes represent the potential impact of area level sources (e.g. all industrial or residential emissions) on measured concentrations, rather than the impact of a specific road or point source.

Imputation of missing concentration data

In the large area campaign, 10.6% of badges got lost, for the city specific campaign this was the case for 3.7% of the tubes. Based on the available data, missing values were imputed ten times using the MICE (Multivariate Imputation by Chained Equations) procedure in R (version 2.8.0, The R Foundation for Statistical Computing, Vienna, Austria), incorporating information on site type (rural, urban or traffic). The differences between the ten imputed datasets were small as only a small percentage of the observations was missing. From each imputed dataset the mean concentration was calculated for each location, which was calculated to estimate the annual mean values.

As a result of the multiple imputation applied to the measurement datasets, ten complete datasets for each of the two campaigns were available. Model parameters were calculated by imputation and then combined by the MIANALYZE procedure (SAS version 9.1, SAS Institute Inc., Carry NC, USA) to account for the uncertainty about the imputed values.

LUR model development and validation

The relationship between land use and traffic variables and NO_2 concentration at the measurement sites was studied by multiple linear regression analysis. Regression models were constructed using a supervised forward selection procedure.[79] Variables were added to the regression model in four steps: 1) traffic variables, 2) traffic related land use variables, 3) population density related land use variables, 4) other land use variables (such as industry and green space).

In each of these steps, the variable with the highest R^2 based on simple (or univariate) linear regression analysis was selected first. In selecting the best predictor, all categories (i.e. different buffer sizes) were tested separately and only the best predictor per group (i.e. each land use category) was selected for further testing, thus no overlapping categories were included in the model. Then, variables with the second, third (etc.) highest R^2 were added one by one and included in the multiple (or multivariate) regression model, if the adjusted R^2 improved by at least one percent and the sign of each of the regression coefficients remained as expected.

Because of the larger and more diverse area, the regional background concentration calculated as the inverse distance weighted mean concentration of rural background measurement sites within a radius of 50km (measurements done in the large area campaign) was a priori included in the large area model for all urban sites. For the rural background sites the locally measured concentration was used as the local background concentration.

After all of the available variables had been tested, the resulting model was re-examined. Variables with the highest p-values were excluded one at a time if the adjusted R^2 remained mostly unchanged (difference in adjusted R^2<1%). The reduced model was preferred.

The final model was evaluated using an internal leave-one-out cross-validation procedure.[14] We additionally evaluated the two models by comparing the concentrations predicted by one model for sites used to develop the other model. To study the additional value of the more complete traffic and land use data, the large area model was also developed using limited traffic data (without municipal road data) and the city specific model was also developed using less detailed land-use data (CORINE).

Dispersion model

In this study, the Dutch modeling tool CAR[10,80] was used, which is the model to be used in built up areas of the Netherlands according to Dutch air quality regulations to calculate traffic-related air pollution. An extensive description of the model is available in Supplemental Material, Annex B. CAR is an empirical dispersion model derived from a more comprehensive Gaussian dispersion model.[81] The model adds a local traffic contribution to a large scale concentration map, which is updated every year. This large scale concentration map is calculated from measurement data of the National Air Quality Monitoring Network (RIVM, Bilthoven, the Netherlands) and modeled contribution of sources in the Netherlands and other European countries. Traffic contribution is calculated by multiplying the traffic emissions with a dispersion factor. Traffic emissions are calculated from traffic intensity, -composition and default speed-dependent national emission factors. The dispersion factor depends on street configuration (buildings, trees), distance to the center of the road and on average annual wind speed (see Annex). The CAR model can be applied to a maximum distance of 60 meters from a road.

CAR version 6.1.1 was used to predict 2006 annual mean NO_2 concentrations in this study for both sets of monitoring locations, using meteorology for the year 2006. The information included in the model was: exact geo coded location, traffic flow (vehicles per 24 hours) and composition (percentage of cars, vans, trucks and busses), distance to the center of the road (m) and categorical information on driving speed, road type and the presence of trees.

Comparison of LUR and dispersion models

Since the CAR atmospheric dispersion model is used to predict air pollution concentrations for almost all roads for which traffic information is available in the Netherlands, concentrations observed at the measurement sites were compared with the CAR-predictions as well. Performance of the dispersion model was compared with the LUR models at the monitoring sites located in Amsterdam (13 monitoring sites of the large area campaign, and 62 monitoring sites of the city specific campaign). This was done by evaluation of scatter plots and correlations between observed and predicted concentrations, and between predictions by the different models.

RESULTS

Large area LUR model

Table 1 shows the distribution of the measured concentrations and the predictor variables for the large area model. Table 2 shows the change in NO_2 concentrations per inter quartile range increase in the predictors in this model and the explained variance of this model (R^2: 87%). Internal leave-one-out cross-validation resulted in a full model R^2 of 84%. Supplemental Material, Figure 2 shows a plot of the predicted and observed concentrations.

Table 1. Distribution of observed average NO_2-concentrations and predictor variables used in the large area (Northwest Netherlands) and city specific (Amsterdam) multivariate LUR models.

	Median	Range
Large area LUR model (N=60)		
Measured NO_2-concentration[a] (μg/m^3)	25.1	(10.5 to 53.1)
Regional background concentration (μg/m^3)	20.7	(10.8 to 25.4)
Traffic volume at nearest road (veh/24hrs)	1225	(195.4 to 37132.8)
Distance to nearest busy road[b] (m)	103.4	(0 to 1409.8)
Residential land use in a 5 km buffer (%)	28.5	(0.8 to 63.9)
City specific LUR model (N=62)		
Measured NO_2-concentration a (μg/m^3)	37.9	(24.8 to 75.1)
Traffic volume at nearest busy road[b] within 50m (veh/24hrs)	0	(0 to 29640.2)
Distance to nearest main road[c] (m)	113.5	(9.3 to 2845.1)
Green space in a 250 m buffer (%)	27.5	(0.5 to 76.3)
Water in a 100 m buffer (%)	4.9	(0 to 30.8)

[a] NO_2-concentrations: average of 10 imputed datasets
[b] busy road ≥5000 vehicles per 24 hours
[c] main road ≥10 000 vehicles per 24 hours

We also investigated the performance of the large-area model for the Amsterdam sub-region of the study area. The resulting R^2 of 79% (Supplemental Material, Figure 3) for these 13 sites was only slightly less than in the original model (internal cross-validated R^2: 84%). When we excluded all 13 Amsterdam sites from the model development (leaving 47 sites including the city of Rotterdam) the model performance expressed as R^2 was 87%.

In order to evaluate the added value of the more complete traffic data, the model was developed using traffic data for nationally and provincially managed roads only. This resulted in a model (Supplemental Material, Figure 4) including three predictor variables: background concentration (1) and percentage of land use categories residential (2) and port (3) in a 5 km circular buffer. The estimated coefficients for background concentration and residential land use were similar to those of the model with more complete traffic data (data not shown). The explained variance (R^2: 73%), however, was substantially lower than for the original model (R^2: 87%).

Table 2. Change in NO_2-concentrations per interquartile range increase in predictor variables used in the large area multivariate LUR model (R^2=87%, adjR^2=85%; cross-validation R^2=84% adjR^2=82%).

Large area LUR	Estimate[a]	SE[a]	p-value
Intercept	10.7	3.9	0.008
Background concentration (µg/m3)	3.4	0.8	<0.0001
Traffic volume at nearest road (veh/24hrs)	1.2	0.3	<0.0001
Distance to nearest busy road[b] (m)	- 4.0	1.2	0.002
Residential land use in a 5 km buffer (%)	6.1	1.1	<0.0001

[a] per interquartile range. Background concentration: 4.4µg/m^3, Traffic volume: 2668veh/24hrs, Distance: 110m, Residential land use: 26%
[b] busy road ≥ 5 000 motor vehicles per 24 hours

City specific LUR model

Table 1 shows the distribution of the measured annual mean concentrations for the city specific model. Concentrations ranged from 24.8-39.1 µg/m^3 at urban background sites and from 42.2-75.1 µg/m^3 at traffic sites. The change in NO_2 concentrations per inter quartile range increase in predictors for this model (R^2 72%, leave-one-out cross-validated R^2 65%) are shown in Table 3 (observed/predicted plot in Supplemental Material, Figure 2). As shown by this Figure, the model performs well for observed concentrations up to approximately 55 µg/m^3. At higher concentrations, the model underestimates the NO_2 concentration. A map of the predicted NO_2 contours for all of Amsterdam is shown in Figure 5 of the Supplemental Material.

Table 3. Change in NO_2-concentrations per interquartile range increase in predictor variables used in the city-specific multivariate LUR model (R^2=72%, adjR^2=69%; cross-validation R^2=65% adjR^2=63%).

Large area LUR	Estimate[a]	SE[a]	p-value
Intercept	56.2	5.5	<0.0001
Traffic volume at nearest busy road[b] within 50 m (veh/24hrs)	7.1	2.3	0.003
Distance to nearest main road[c] (m)	- 7.6	2.6	0.005
Green space in a 250 m buffer (%)	- 4.6	1.6	0.005
Water in a 100 m buffer (%)	2.7	1.5	0.076

[a] per interquartile range. Traffic volume: 14,052veh/24hrs, Distance: 249m, Green space: 26%, Water: 13%
[b] busy road ≥ 5 000 motor vehicles per 24 hours
[c] ≥ 10 000 vehicles per 24 hours

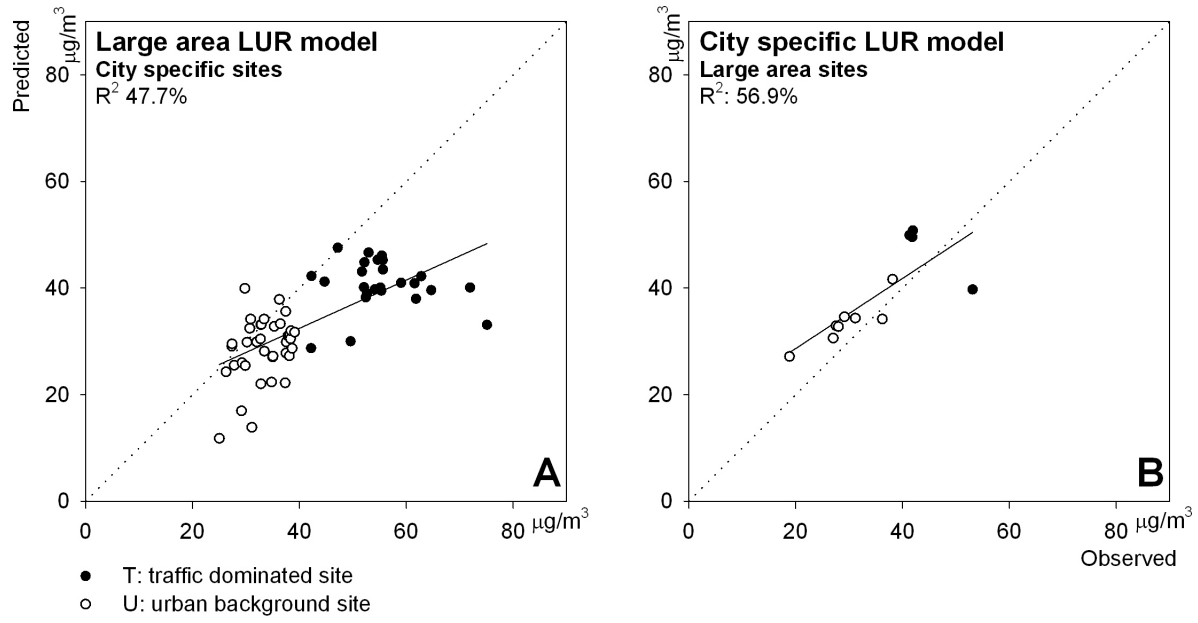
In order to evaluate the added value of high resolution land use data for this model the model was developed using CORINE land use data instead of high resolution land use data. In the final model (Supplemental Material, Figure 4) the same two traffic variables (distance to the nearest main road and traffic flow at the nearest busy road within 50 m) and the percentage of land use category 'port' in a 5km circular buffer were included. The explained variance (R^2) of the city specific model with lower resolution land use data was

69%, only slightly less than the explained variance of the original city specific model (72%).

LUR model evaluation by independent sets of measurements

In Figure 1, plots of the observed NO_2 concentrations at sites that were used to develop one LUR-model and predicted concentrations from the other LUR-model are shown. Both LUR models performed less well in predicting NO_2 concentrations at the sites that were used to develop the other model. Applying the large area model to sites of the city specific campaign (n=62, Figure 1A) resulted in an R^2 of 48%, which is much lower than the R^2 (72%, Table 3) of the city specific LUR for the sites of the city specific campaign that were used to develop the model and the internal cross-validation R^2. Applying the city specific model to the Amsterdam sites of the large area campaign resulted in an R^2 of 57% (n=13, Figure 1B), which is much lower than the R^2 of the large area model for the Amsterdam sites of the large area campaign (79%, Supplemental Material, Figure 3) and the internal cross-validation R^2.

Figure 1. Evaluation of large area and city specific LUR models for measurement sites in Amsterdam, the Netherlands: Predicted NO_2-concentrations from one LUR-model vs. observed concentrations at measurements sites that were used to develop the other LUR model. (A) Estimations by the city specific LUR model for the large area sites. (B) Estimations by the large area LUR model for the city specific sites. The dotted line indicates where observed equals predicted concentration.



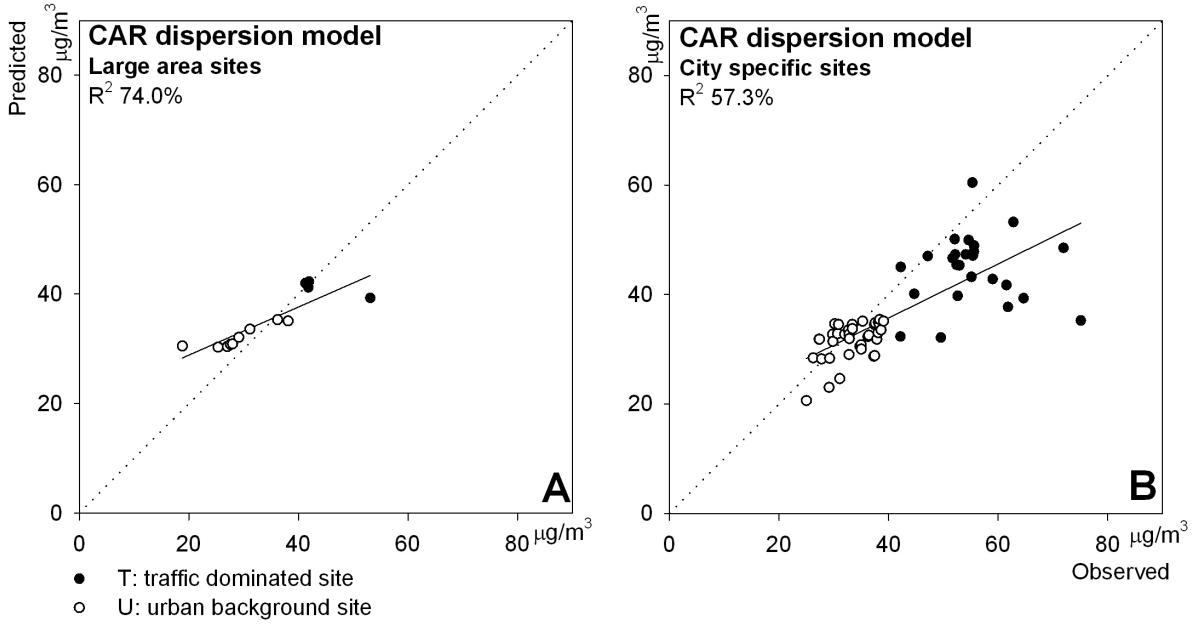
Dispersion model

Predictions from the CAR-model correlated highly with predictions from the two LUR models (Supplemental Material, Figure 6). The agreement between

CAR and both LUR models was higher for the 13 large-area campaign sites in Amsterdam (R^2 89%) than for the 62 city-specific campaign sites (R^2 75%).

Figure 2 shows the CAR dispersion model predictions and observed concentrations at the Amsterdam measurement sites of the large area campaign (Figure 2A) and the sites of the city specific campaign (Figure 2B). The CAR model predictions explain a large fraction of the variability in observed concentrations at the 13 Amsterdam sites of the large-area campaign (Figure 2A), but a systematic overestimation of background concentrations and underestimation of local traffic contributions to concentrations is evident. The CAR model explains a lower percentage of observed variability in concentrations at the city-specific sites (Figure 2B). As in the case of the city-specific LUR model, the dispersion model, systematically underestimates the highest exposed traffic dominated sites.

Figure 2. Observed and CAR dispersion model predicted NO_2-concentrations at measurement sites in Amsterdam, the Netherlands. (A) CAR estimations for the large area sites. (B) CAR estimations for the city-specific sites. The dotted line indicates where observed equals predicted concentration.



When we compare the percentage explained variability (R^2) of the LUR models at the independent monitoring sites, the CAR model performs slightly better than the two LUR models. The percentage explained variability at the city-specific sites was 57% for the CAR model (Figure 2B) and 48% for the large-are LUR model (Figure 1A). The percentage explained variability at the large-area sites was 74% for the CAR model (Figure 2A) and 57% for the large-area LUR model (Figure 1B). However, when we take into account the above mentioned under- and overestimation, we overall asses that the dispersion model does not perform better than the LUR models.

DISCUSSION

Two land use regression models were developed for two independent sets of NO_2 measurements. Both models explained a large percentage of the measured spatial variation (R^2 for the large area LUR 87%; for the city specific LUR 72%). Internal leave-one-out cross-validation R^2s were only slightly lower (84 and 65%, respectively). Both LUR models performed less well in predicting concentrations at an independent set of monitoring sites than was expected from internal cross-validation (R^2 large area: 48% vs. 84%, city specific: 57% vs. 65%). More complete traffic information improved the predictive power of the LUR models more than detailed land use data. The dispersion model CAR did not perform better in predicting concentrations at independent monitoring sites than the two LUR models.

Evaluation of LUR models

Two LUR models were developed that explained a high percentage of observed variability in measured NO_2 concentrations. In internal leave-one-out cross-validations, percentages of explained variability were high as well, suggesting good applicability of the models to unmeasured locations. However, the models explained less variability when applied to the monitoring sites from the other sampling campaign. The main reason for this is probably that the sampling sites have been selected in different ways (see discussion below). As LUR models are generally developed to estimate ambient pollution levels at unmeasured locations in the study area (e.g. homes of study participants), the implication is that the sampling locations need to be selected very carefully to reflect the type of locations to which the model will be applied. If residential exposure assessment is the goal of LUR model development, probably measurements at the façade are a better choice than measurements at curbside.

The two measurement campaigns used in this study differed in year of monitoring (2006 vs. 2007), sampler (Palmes tube vs. Ogawa badge), temporal resolution (continuous vs. four 1-week samples) and site selection criteria (the large area campaign was performed for the purpose of LUR modeling; the city specific campaign consisted of selected locations from a routine monitoring program), which may have influenced cross-validation results. In previous LUR studies, both strategies (purpose designed and routine monitoring) to collect measurement data have been used regularly.[e.g.35,76] However, the samplers in the city specific campaign were often placed slightly closer to the road than in the large area campaign. Although subtle, these systematic differences between measurement sites in both campaigns may explain in part the poorer predictions of the models for the sites used to develop the other model. Year of sampling may not have been important, as the correlation between concentrations measured in 2006

and 2007 at a subset of 35 sites from the city specific campaign was 0.98. Continuous measurements performed at an urban background site of the national network in Amsterdam further showed similar concentrations during both measurement campaigns (32.0 and 32.2 μg/m^3, respectively), indicating little (temporal) differences in NO_2 levels between campaigns. Since both samplers correlate highly with chemiluminescence monitors, differences between samplers are unlikely to be important. Several LUR studies have shown that spatial contrasts can be assessed with four 1- to 2-week sampling campaigns. However, absolute concentrations may deviate from annual mean concentrations.[14]

Few other studies have done out-of-sample validations of LUR models. In a study by Stedman et al.[82] the model R^2 was 97% (based on continuous NO_2 monitors), in validation (using passive measurements at other locations) this dropped to 36%. Henderson et al.[83], however, developed a LUR using passive measurements (model R^2 of 56%), which scored higher (69%) in validation using continuous monitors.

The scale of the large area model is somewhere in between the metropolitan[e.g. 84,85] or national[e.g. 76,82] scale of most other LUR models that have been developed before. The city specific model, however, is focusing on a metropolitan area. The availability of two LUR models for the same area provided the opportunity to compare the performance of LUR models originally developed for different geographical scales. The concentrations at traffic dominated sites of the city specific campaign, which were more often situated near complicated high traffic situations, were largely underestimated by the large area LUR model. Although still underestimating hot-spot concentrations, application of the city specific LUR model resulted in a better prediction with a much smaller mean residual of 2 μg/m^3. Predictions of both models for urban background locations in both campaigns and traffic dominated locations in the large area campaign, however, were within the range of the measured concentrations.

Value of detailed traffic and land use information

In this study we have put a large effort in gathering complete and detailed traffic information from all municipalities. Data from national and provincial authorities were readily available. As typically most of the streets that people live by are municipal roads, traffic on these roads are important for exposure assessment as used in epidemiological studies. Our effort resulted in all municipalities participating, providing traffic data for 31% of the municipal roads. Traffic load could thus be assigned to 87% of the total road length in the study area. In a previous Dutch study [76,76], 59% of the municipalities provided data resulting in data for 14% of the municipal roads. Recalculation of the large area model using limited traffic data (national and provincial only) resulted in a lower explained variance of that model (R^2 73 vs. 87% for the

recalculated and original large area LUR, respectively, Supplemental Material Figures 4 and 2). For other areas in which traffic is the main source of air pollution, the situation could be similar.

For Amsterdam high resolution land use data were available, which is reflected by the higher information density shown on the city map; smaller surfaces such as playgrounds or canals are not considered in a low resolution map, but can add up to an important part of the city surface area. Two of the high resolution land use variables (water and green space) were included in the city specific LUR model. Recalculation of the city specific LUR model using land use data at a lower resolution, however, showed that the added value of detailed land use data in the model fit was limited (R^2: 69 vs. 72% for the recalculated and original city specific LUR, respectively; Supplemental Material Figures 4 and 2). When forced to prioritize in future studies, obtaining complete traffic data would therefore be preferred over obtaining higher resolution land use data.

Comparison of LUR models and a dispersion model

Comparison of the three approaches to model NO_2-concentrations was done in Amsterdam. In the comparison, remarkable similarities between concentrations predicted by the large area LUR and the dispersion model were found: The model predictions were highly correlated and showed very similar levels. Possible explanations are that the same traffic data and similar traffic predictors (traffic flow at the nearest road and a distance-variable) were used in both models. Background concentration and residential land use together, as used in the large area LUR model, seem to be equivalent to the large-scale concentration included in the dispersion model. Measurements used to estimate background levels in the LUR model and to calibrate the large-scale concentrations in the dispersion model were done independently, thus not causing similarities. The restriction of the dispersion model to the estimation of concentrations at a distance of no more than 60m from a road [10] may explain the differences between the dispersion and the city specific LUR model.

The fit of the CAR dispersion model seems better for the 13 Amsterdam sites of the large area campaign than for the sites in the city specific campaign (Figure 2). Differences in the campaigns discussed above may have contributed to this finding. Differences in monitoring year and temporal resolution are unlikely explanations as these would have resulted in better agreement for the city-specific sites as CAR predictions were made for the year 2006 for both datasets. Possible explanations include the smaller fraction of traffic sites amongst the large area sampling sites (traffic sites are more difficult to model) and the range in concentrations. As in the case of the application of the large area LUR model to city specific sites and previous LUR studies,[35,74,85-87] the dispersion model was unable to predict the highest ('hot-spot') concentrations observed in the city-specific campaign well. Additional

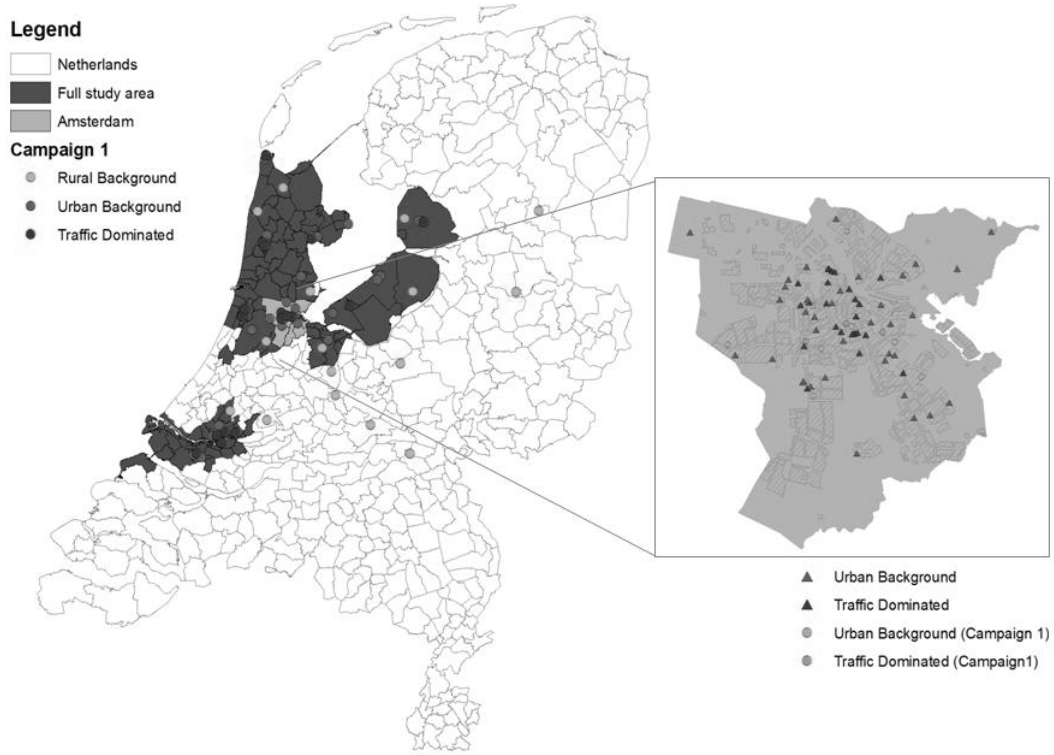
evaluations of the locations with the highest concentrations in the city specific campaign showed that most of these locations are situated near complicated high traffic situations such as congested busy roads. From this data it is hard to conclude which model is most appropriate for estimating concentrations in Amsterdam, as most of the measurement data available were used in developing the city specific model.

The few other studies comparing dispersion and LUR models have typically found that LUR models perform at least as well as the dispersion models considered.[81] The comparison however depends on the particular model and its ability to model small scale variations. The CAR model is a semi-empirical model derived from a more detailed Gaussian model and adapted to calculate air quality near roads.[81] The model is able to model small scale variations in urban areas, but not optimal for modeling dispersion along highways, so our results may not be generalizable to near highway applications.

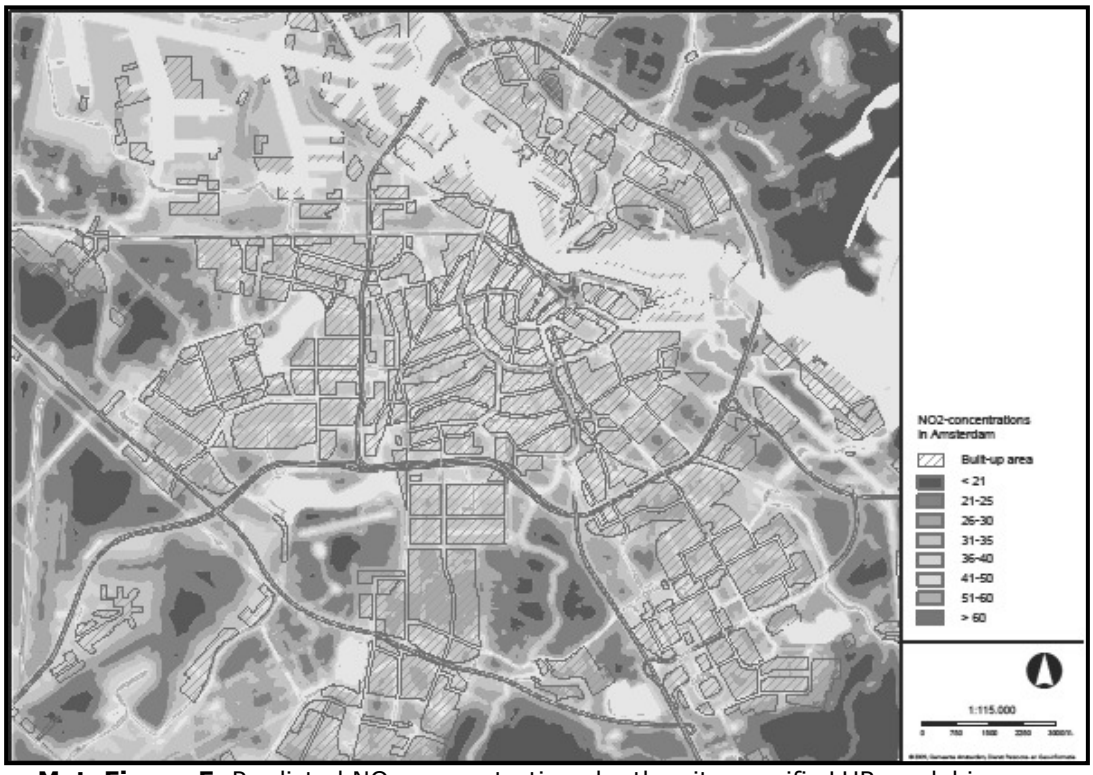
Conclusion

A large area LUR and city specific LUR model, developed for two independent sets of NO_2 measurements, explain a large percentage of the measured spatial variation. Both LUR models performed less well than results found from internal leave-one-out cross-validation, possibly related to differences in site selection. Evaluation of the value of using high resolution data showed that more complete traffic information adds much more to the model fit of LUR models than detailed land use data. The dispersion model CAR did not predict concentrations at independent monitoring sites better than the two LUR models.

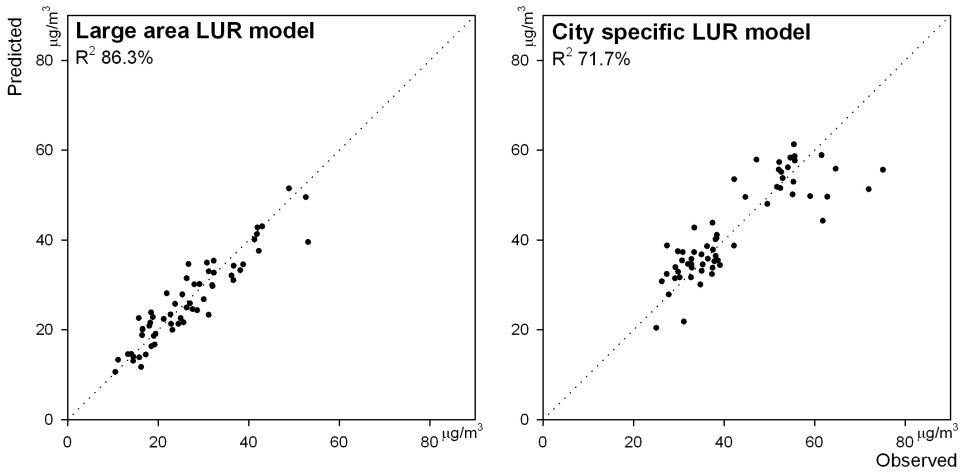
SUPPLEMENTAL MATERIAL



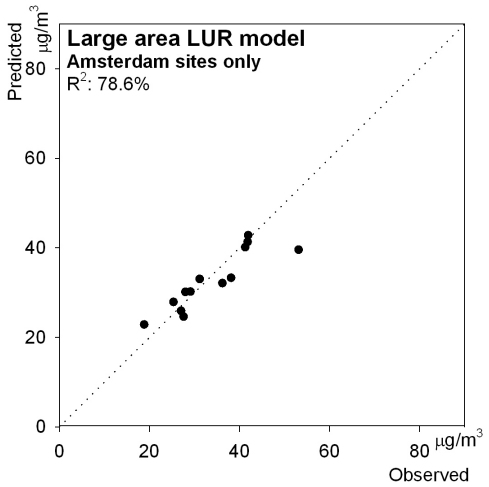
Supp.Mat. Figure 1. Maps and measurement locations for the large area LUR model (campaign 1, N=60) and the city specific LUR model (campaign 2, N=62)



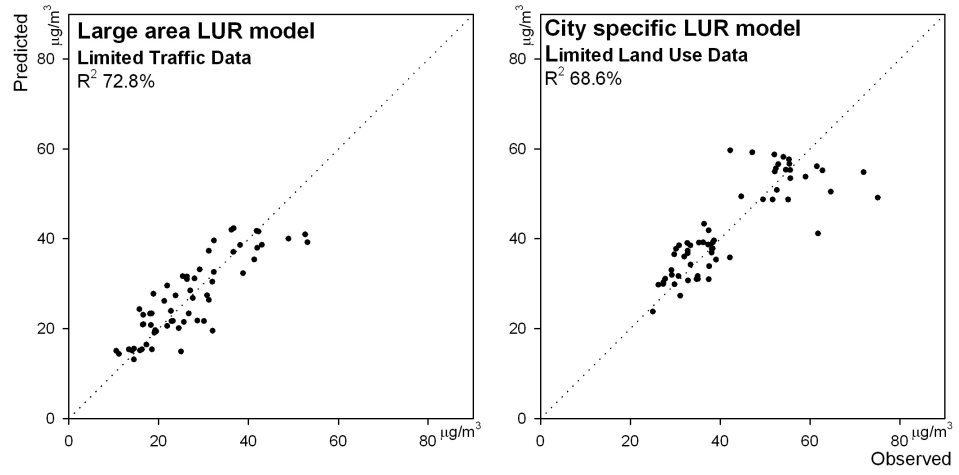
Supp.Mat. Figure 5. Predicted NO_2-concentrations by the city specific LUR model in Amsterdam



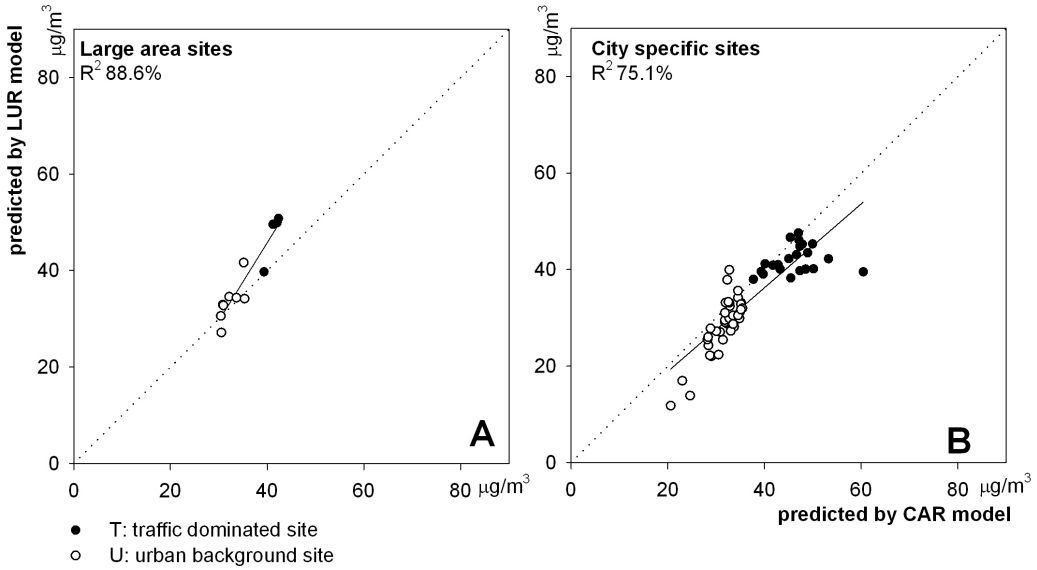
Supp.Mat. Figure 2. Observed and predicted NO2 concentrations for the large area (N=60) and city specific campaigns (N=62). The dotted line is where observed equals predicted concentration.



Supp.Mat. Figure 3. Observed and predicted NO2 concentrations for the Amsterdam sites of the large area campaign (n=13). The dotted line is where observed equals predicted concentration.



Supp.Mat. Figure 4. Observed and predicted NO_2-concentrations for the recalculated large area (limited traffic data, R^2=72.8%, adjR^2=71.3%) and city specific (limited land use data, R^2=68.6, adjR^2=67.0) LUR models.



A: CAR vs. City specific LUR at large area sites **B**: CAR vs. Large area LUR at city specific sites

Supp.Mat. Figure 6. Predicted concentration from CAR dispersion model vs. predicted concentration from both LUR models, for sites of the campaigns not used to develop the LUR models.

ANNEX A - Land Use Regression: Databases used

Air quality

ACN: Geocoding of measurement locations

Adres Coordinaten Nederland (translation: Address Coordinates Netherlands) 2005. Apeldoorn, the Netherlands, Kadaster, 2005.

Traffic data

NWB: Digital road map to which traffic infomation was linked

Nationaal Wegen Bestand (translation: National Road Database) 2006. Den Haag, the Netherlands, Ministerie van Verkeer en Waterstaat (Dutch ministry of Transport), 2006.

Land use data

CORINE: European land use (grid 100*100m)

Coordination of information on the environment (Corine) Land Cover 2000. Copenhagen, Denmark, European Environmental Agency, 2006.

KBKA: Amsterdam land use (grid 5*5m)

Kadastrale Basiskaart Amsterdam (translation: Cadastral Base Map Amsterdam) 2006. Amsterdam, the Netherlands, Dienst Geo- en Vastgoedinformatie, Gemeente Amsterdam (translation: Amsterdam Municipal Service for Geo- and Real Estate Information), 2007.

National population density database

CBS. National Population Database 2006. Heerlen, the Netherlands, Centraal Bureau voor de Statistiek (translation: Dutch Central Bureau of Statistics), 2006.

ANNEX B - CAR dispersion model

Model Description

In this study, the Dutch modeling tool CAR[10,80] was used, which is the model to be used in built up areas of the Netherlands according to Dutch air quality regulations to calculate traffic-related air pollution. CAR is an empirical dispersion model derived from the more comprehensive traffic model developed at TNO (Utrecht, the Netherlands), which is a Gaussian dispersion model, adapted to calculate air quality near roads based on an extensive program of wind tunnel experiments covering many different street configurations including street canyons.[81] The model adds a local traffic contribution on top of a large scale concentration map calculated with the Operational Priority Substances (OPS) dispersion model[88,89] and updated every year.[90] This large scale concentration map (at a 1*1km grid) is calculated from measurement data of the National Air Quality Monitoring Network (NAQMN, Bilthoven, the Netherlands) and modeling contributions of sources in the Netherlands and other European countries excluding local traffic. The OPS model calculates annual average concentrations based on emissions and their dispersion, transport, chemical conversion and deposition. The model uses a Gaussian plume for dispersion on a local scale and a Lagrangian trajectory for long-distance transport of compounds. The model calculates 5*5km concentrations, which have been interpolated to 1*1km grids.

The traffic contribution is calculated by multiplying the traffic emissions with a dispersion factor. The traffic emissions are calculated from traffic intensity, -composition and default speed-dependent national emission factors. The dispersion factor depends on street configuration (buildings, trees), distance to the center of the road and on average annual wind speed which is estimated on a 1x1 km basis (see 'Details from the manual'). The model calculates the NO_x concentration, which is transformed into NO_2 concentrations based on the fraction of directly emitted NO_2 and the transformation of NO to NO_2, using an empirical formula including the background ozone concentration (see 'Details from the manual'). The CAR model is updated yearly including updated traffic emission factors, meteorology and the updated map of large-scale concentrations. The CAR model has been calibrated using measurements from 14 NAQMN stations in busy streets for the period 2003-2006.[91] The CAR model can be applied to a maximum distance of 60 meters. A further discussion of the CAR model and its relation to other dispersion models is found in Vardoulakis et al.[81]

CAR version 6.1.1 was used to predict 2006 annual mean NO_2 concentrations in this study for both sets of monitoring locations, using meteorology for the year 2006. The information included in the model was: exact geo coded location, traffic flow (vehicles per 24 hours) and composition (percentage of cars, vans, trucks and busses), distance to the center of the road (m) and categorical information on driving speed, road type and the presence of trees.

Details from the manual

Details on the CAR dispersion model can be found in the model software user manual in Dutch. Here the main formulas are presented to calculate the street contribution.

1. Concentration

Concentration contribution from traffic in the street itself is calculated using formula 1. (1)

$$C_{jm-bijdrage} = E \cdot \theta \cdot F_b \cdot F_{regio}$$

Where
- $C_{jm\text{-}bijdrage}$ = Annual traffic contribution
- E = Emission (2)
- θ = Dilution factor (3)
- F_b = Tree factor (4)

F_{regio}	=	Regional factor concerning meteorology and windspeed (yearly updated and included in the model automatically, based on geographic coordinates)

The annual NO_x concentration contribution is calculated using function (1). The concentration of NO_2, is calculated using an empirical relationship including NOx, the background ozone concentrations and the fraction of directly emitted NO2 (formula 1a)

(1a) $$C_{NO2-jm} = F_{NO2} \cdot C_{NOx-jm} + \frac{B \cdot C_{achtergrond\_O3} \cdot C_{NOx-jm} \cdot (1 - F_{NO2})}{C_{NOx-jm} \cdot (1 - F_{NO2}) + K} + C_{achtergrond\_NO2}$$

where

C_{NO2-jm}	=	annual NO_2 concentration contribution
F_{NO2}	=	weight fraction of directly emitted NO_2
C_{NOx-jm}	=	annual NO_x concentration contribution (1)
$C_{achtergrond\_O3}$	=	background concentration of O_3[1]
$C_{achtergrond\_NO2}$	=	background concentration of NO_2[1]
B, K	=	empirical derived conversion factor, for NO to NO_2 (B=0.6, K=100)

[1] derived from the yearly updated large scale background map, based on geographical coordinates

2. Emission:

Emission is calculated from traffic intensity, traffic composition and default emission factors for the Dutch car fleet (formula 2). Emission factors are speed dependent.

(2) $$E = N \cdot ((1 - (F_m + F_v + F_b)) \cdot E_p + F_m \cdot E_m + F_v \cdot E_v + F_b \cdot E_b) \cdot \frac{1000}{24 \cdot 3600}$$

where

E	=	Emission (µg/m/s)
N	=	Number of vehicles per 24 hours ($24hrs^{-1}$)
Fm	=	fraction 'medium heavy' traffic (i.e. vans)
Fv	=	Fraction of heavy traffic (i.e. trucks)
Fb	=	Fraction of busses
Ep	=	Emission factor for cars
Em	=	Emission factor for 'medium heavy' traffic (i.e. vans)
Ev	=	Emission factor for heavy traffic (i.e. trucks)
Eb	=	Emission factor for busses

All emission factors are yearly updated based on roller bank measurements of vehicles.

3. Dilution Factor:

Dilution factors are differently derived for different road types. In CAR roads are categorized as:

1) Road through open terrain, incidental buildings or trees within a radius of 100m
2) Basic type, all roads not categorized in any of the other categories
3a) Buildings along both sides of the road, distance road-axis to building façade is smaller than three and larger than 1.5 times the building height.
3b) Street canyon: Buildings along both sides of the road, distance road-axis to building façade is smaller than 1.5 times the building height.
4) Buildings along one side of the road, at a distance smaller than three times the building height.

For road type 1 the dilution factor is derived from the following function:

(3a) $$\theta = a \cdot S^{b\frac{S+e}{S}} \cdot (c \cdot S + d)$$

For road type 2, 3a, 3b and 4 the factor function is:

(3b) $$\theta = a \cdot S^2 + b \cdot S + c$$

where θ = Dilution factor
S = Distance to road-axis
a, b, c, d, e = Road type specific parameters (from table below)

Parameter	wegtype				
	1	2	3a	3b	4
a	0,725	$3{,}1 \cdot 10^{-4}$	$3{,}25 \cdot 10^{-4}$	$4{,}88 \cdot 10^{-4}$	$5{,}00 \cdot 10^{-4}$
b	-0,77	$-1{,}82 \cdot 10^{-2}$	$-2{,}05 \cdot 10^{-2}$	$-3{,}08 \cdot 10^{-2}$	$-3{,}16 \cdot 10^{-2}$
c	-0,0011	0,33	0,39	0,59	0,57
d	1,20	n.v.t.	n.v.t.	n.v.t.	n.v.t.
e	2,70	n.v.t.	n.v.t.	n.v.t.	n.v.t.

4. Tree Factor:

Trees are included to take into account limited dispersion in case of high tree density in streets.

1 No trees at all, or an occasional tree
1.25 One or more rows of trees, less than 15 meters apart, openings between crowns
1.5 Crowns are touching and covering at least one third of the road with

Chapter 3

Long-term Exposure to Traffic Related Air Pollution and Cardiopulmonary Hospital Admission

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ABSTRACT

The objective was to explore the relation between long-term exposure to traffic related air pollution and cardiopulmonary hospital admissions.

This study was based on hospital registration (2001-2004) and general population data for the Western part of the Netherlands (population 4.04 million). At postcode area level (n=683) the association between the land use regression modeled annual average outdoor NO_2 concentrations and hospital admission for respiratory and cardiovascular causes was evaluated. Analyses were adjusted for differences in composition of the population of the postcode areas (age, sex, income).

Outdoor NO_2 concentration was positively associated with prevalence of hospital admissions for asthma, chronic obstructive pulmonary disease (COPD), all cardiovascular causes, ischemic heart disease (IHD) and stroke. For these causes of admission exposure-response relations were found (e.g. adjusted Prevalence Ratios (and 95%-confidence intervals) for the second to fourth quartile of exposure relative to the first quartile of exposure were 1.87 (1.46-2.40), 2.34 (1.83-3.01) and 2.81 (2.16-3.65) for asthma; 1.44 (1.19-1.74), 1.50 (1.24-1.82) and 1.60 (1.31-1.96) for COPD). Hospital admissions for all respiratory causes showed significantly elevated prevalence ratios with increased exposure, but no exposure-response relation.

Long-term exposure to traffic related air pollution was associated with increased hospital admissions for respiratory and cardiovascular causes.

INTRODUCTION

The cardiopulmonary health effects of air pollution have been widely investigated.[6,17] Several studies showed that episodes of elevated air pollution levels were related to increased hospital admission. In the APHEA study,[20,21] an increase in cardiac and respiratory hospital admission of about 1% per 10 $\mu g/m^3$ increase in daily PM_{10} was observed. In the USA, Zanobetti et. al[22] found admission to increase by 1.3% for cardiovascular causes and by 2.5% for chronic obstructive pulmonary disease (COPD) for the same PM_{10} increment. In a short-term exposure study looking at traffic related $PM_{2.5}$, Halonen et. al[23] reported increased emergency room visits for asthma among children and increased emergency room visits for asthma and/or COPD among elderly with increased air pollution concentrations.

In the past decade, the focus has shifted towards health effects of long-term exposure to air pollution, and traffic related air pollution became a main priority.[37-40] Most long-term exposure studies focus on mortality or prevalence of cardiopulmonary symptoms and disease. Furthermore, several cohort studies showed associations between air pollution and asthma incidence.[92-95] Hospital admissions for such causes have occasionally been studied in long-term exposure studies.[41,64-69] Most of these studies had a case-control design. One case-control study, reported an association of traffic with childhood asthma hospital admission, in absence of a dose-response relation [64]. A second case-control study on hospitalization for community acquired pneumonia among the elderly, using several methods to estimate exposure, found significant increases in hospitalization with increased exposures to NO_2 and $PM_{2.5}$.[67] A third case-control study found no association between hospital admissions for asthma among children and living close to busy roads in London.[69] Recently, a cohort study among adults in Denmark showed that the incidence of hospital admissions for COPD was associated with the residential 35-year mean NO_2 level (hazard ratio 1.08 per 5.8 $\mu g/m^3$).[41] In a case-control study based on the national stroke register in Sweden, no clear association between NO_x and ischemic stroke admission was observed.[68] An English ecological study[66] on admission for coronary heart disease among a population aged 45 and over found no clear association with NO_2 either. However, stroke admission was increased among subjects in the highest exposure category.[65]

In this population based study, we explore the relation between long-term exposure to traffic related air pollution and respiratory and cardiovascular hospital admission for a population of 4 million inhabitants of the Western part of the Netherlands.

MATERIALS AND METHODS

The study was conducted among inhabitants (4.04 million) in the West of the Netherlands (Supplemental Material, Figure I). The study area (approx. 7,300km^2) consists of rural and (sub)urban areas including the cities of Amsterdam and Rotterdam. This study was based on hospital admission data, obtained from the National Medical Registration (LMR),[96] population data provided by the Dutch national Central Bureau of Statistics (CBS, Heerlen/Den Haag, the Netherlands) and modeled NO_2 concentrations from a previously described land use regression model.[97] More information on the study area and population, hospital admission and covariates, and exposure is provided in the Supplemental Material.

The analysis focused on a priori selected admission causes: all respiratory (ICD-9: 460-519), asthma (ICD-9: 493), COPD (ICD-9: 490-492 and 494-496), all cardiovascular (ICD-9: 390-429), ischemic heart disease (IHD, ICD-9: 410-414) and stroke (ICD-9: 430-438). We analyzed the association between modeled NO_2 exposure and prevalence of hospital admissions (defined as the number of persons admitted at least once for respiratory and cardiovascular causes per total number of inhabitants in a postcode (PC4) area) by linear regression with the log of the PC4 specific prevalence as the dependent variable. PC4 areas with no admissions for a specific cause during the study period, were assigned a value of one-half of the smallest observed non-zero value. Smoothing splines (GAM procedure, mgcv-package, R version 2.8.0, R foundation for Statistical Computing, Vienna, Austria) were used to explore the functional relationship between admission prevalence and exposure. Since associations between hospital admission and NO_2 exposure were generally non-linear (Supplemental Material Figure II), NO_2 levels were categorized into four categories for all further analyses using quartiles as cut-off points. The lowest exposure quartile was used for reference. Results of regression analyses are presented as Prevalence Ratio (PR), calculated from the estimated regression coefficients beta as e^{β} with 95%-confidence intervals (95%-CI) with and without adjustment for confounders.

Table 1. Distribution of characteristics per postcode area (n=683) in the study area (West of the Netherlands), during 2001-2004.

Characteristic	minimum	25-percentile	median	75-percentile	maximum
Inhabitants per PC4 area (n)	25	1,605	5,178	9,059	22,934
Male (%)	36	49	50	51	99
Age <20 yrs (%)	0	21	25	27	43
Age >65 yrs (%)	0	9	12	17	80
Average income (€/month)	900	1,849	2,045	2,298	4,633
Urbanization (addresses/km^2)	7	341	1,342	2,304	11,439
NO_2 (µg/m^3)	7.7	17.4	24.0	31.2	82.5

We adjusted for a priori selected confounders age, sex and income. Age was included in the regression models using two variables: the percentage of young (<20 yrs) people per postcode area and the percentage of elderly (>65 yrs) people per postcode area. Sex was included as the percentage of men and income as mean income per month per postcode area. We performed all analyses for all hospital admissions (the total of emergency and planned) and emergency hospital admissions, separately. As there is evidence for hospitalization being associated with urbanization,[98,99] we performed sensitivity analyses with additional adjustment for addresses per km^2 as an indicator for urbanization. No information on smoking was available for the full population. Adjustment for diseases such as COPD and lung-cancer has been suggested in studies where information on smoking is not available.[100,101] We therefore performed sensitivity analyses with additional adjustment for COPD.

All analyses were done using SAS 9.2 (SAS Institute Inc., Cary, NC, USA).

Table 2. Hospital admissions in the study area (West of the Netherlands) during 2001-2004.

Cause of admission		Total number of admissions (N)	Inhabitants admitted per postcode area (%)					PC4 areas without admission (n)
			min.	25-perc.	median	75-perc.	max.	
All Respiratory	All	134,235	0	2.6	3.1	3.7	12.0	10
	Emergency	42,184	0	0.7	1.0	1.2	4.2	24
Asthma	All	5,252	0	<0.1	0.1	0.2	1.9	119
	Emergency	3,912	0	<0.1	0.1	0.1	1.9	142
COPD	All	10,769	0	0.1	0.2	0.3	2.4	77
	Emergency	6,741	0	0.1	0.1	0.2	1.9	101
All	All	113,116	0	2.1	2.7	3.3	12.8	6
Cardiovascular	Emergency	73,524	0	1.3	1.7	2.2	9.5	8
IHD	All	55,794	0	1.0	1.3	1.6	6.4	16
	Emergency	34,801	0	0.6	0.8	1.0	4.8	28
Stroke	All	28,600	0	0.4	0.6	0.8	2.9	28
	Emergency	20,161	0	0.3	0.5	0.6	2.5	41

RESULTS

The PC4 population distribution over the different age groups was quite homogenous (Table 1), as was the distribution of men and women. The inhabitants of one postcode area however, located in the Rotterdam industrial area, were almost exclusively male (99%). The distribution of estimated outdoor NO_2 concentrations over the PC4 areas is shown in Figure 1. Although the range in concentrations was large, for the majority of the postcode areas concentrations were between 17 and 32 $\mu g/m^3$; less than 10% of the concentrations were above 38 $\mu g/m^3$. The population living in the different quartiles of exposure was 0.5, 0.9, 1.2 and 1.4 million for the first, second, third and fourth quartile respectively. The highest concentrations occurred at urban locations near highways. Table 2 shows the distribution of the number of persons with hospital admissions per cause (247,351 in total) and the distribution of the prevalence of hospital admissions per PC4 area. The admission prevalence was not correlated with the total number of inhabitants per PC4 area (r=0.19 and 0.01 for all respiratory and all cardiovascular causes, respectively).

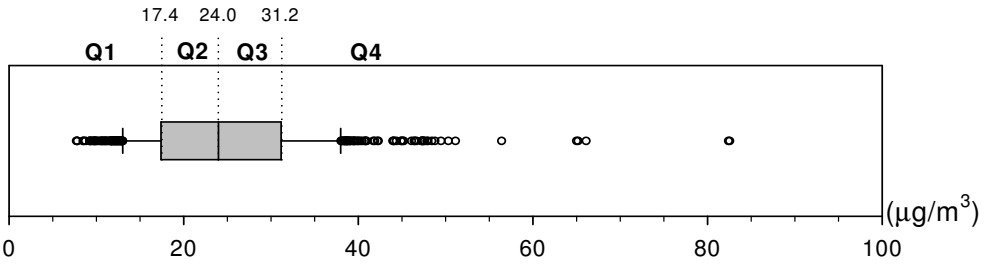


Figure 1. Distribution of NO_2-concentration over the postcode areas (mean: 25.19, SD 10.17).

For all respiratory causes for all and emergency admissions Prevalence Ratios (PR) were elevated relative to the reference quartile in crude analyses (Table 3). Adjustment for age, sex and income did not change these findings. There was no clear concentration-response relationship for 'All respiratory causes'. For asthma and COPD, however, increasing PR with increasing exposure was observed for all admissions. A concentration-response relation was also observed for emergency asthma admission.

For all cardiovascular hospital admissions, PR's were significantly increased comparing the three highest quartiles of exposure to the lowest quartile (Table 3). Adjustment for confounding reduced the effect estimates somewhat but most remained (borderline) significantly elevated. For emergency admissions for 'All cardiovascular causes' and IHD, an increased risk with increased NO_2 concentrations was observed but no concentration-response relation was seen.

Figure 2 shows the result of the sensitivity analysis exploring the effect of additional adjustment for urbanization. Adjustment for urbanization reduced effect estimates for 'All respiratory causes', asthma, COPD and stroke admission. However, prevalences remained significantly elevated relative to the reference quartile. The effect estimates for 'All cardiovascular causes' and IHD admission increased somewhat and the concentration-response relations for these outcomes were more apparent after adjustment for urbanization. The same adjustment among emergency hospital admissions (Supplemental Material Figure III) reduced effect estimates for all causes studied.

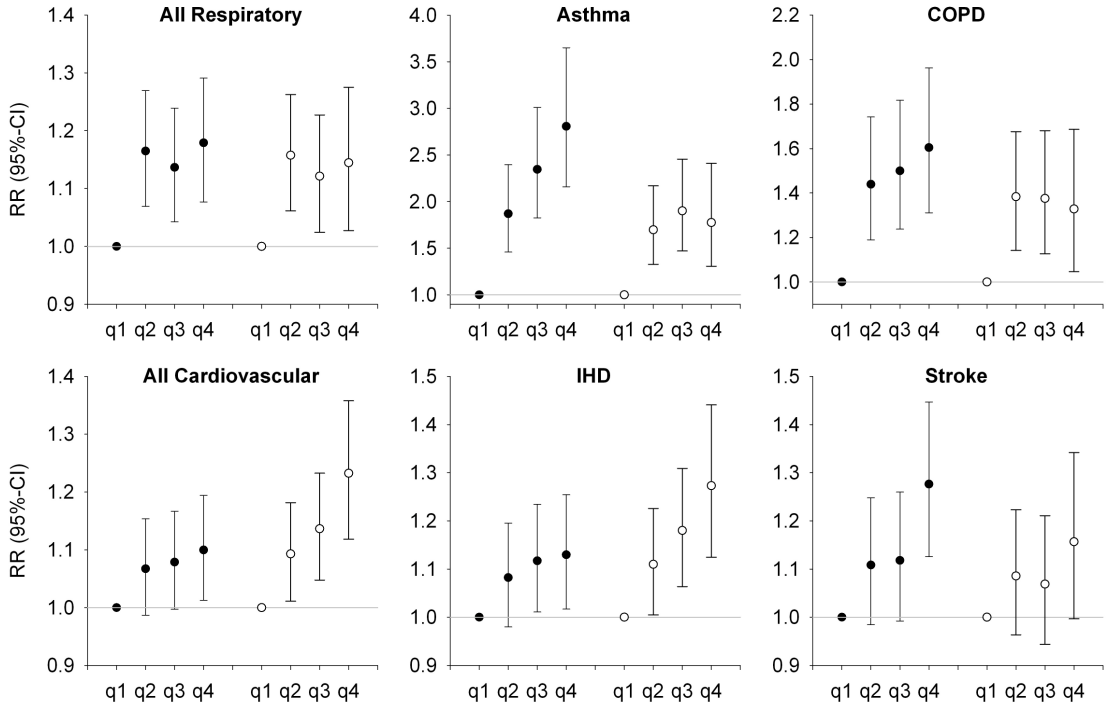


Figure 2. Associations between residential postcode area NO_2-concentration (in quartiles: q1-q4) and all hospital admissions per postcode area, adjusted for age, sex and income (filled dots) and adjusted for age, sex, income and urbanization (open dots).

No information on smoking habits was available for the full population. The Central Bureau of Statistics (CBS) had questionnaire derived smoking data available for a subset of people living in the study area (Permanent Quality of Life (POLS)-questionnaire) which were not directly available to us due to privacy regulations. Upon our request, CBS categorized the geographically well distributed 8,433 respondents to the questionnaire by exposure (aforementioned quartiles). Smoking was statistically significantly more prevalent among respondents with exposure in the highest quartile of exposure (35%) compared to the reference quartile (30%). However, no statistically significant difference was observed for the 2nd and 3rd quartile

comparing to the reference quartile (30 and 31%, respectively). Sensitivity analyses with additional adjustment for COPD as a proxy for smoking were performed. Results were very similar to those from analyses not adjusting for COPD. The COPD-adjusted PR's and 95%-CI's for all asthma admissions, for instance, were 1.82 (1.42-2.33), 2.27 (1.77-2.91) and 2.75 (2.12-3.57), for the second, third and fourth quartile respectively. For all admissions for 'All cardiovascular causes', these PR's and 95%-CI's were 1.06 (0.98-1.15), 1.07 (0.99-1.16) and 1.10 (1.01-1.19).

Table 3. Crude and Adjusted Associations (PR and 95%-CI) between Residential Postcode Area NO_2-concentration (in Quartiles) and Hospital Admissions per Postcode Area in the West of the Netherlands, During 2001-2004.

	Exposure category ($\mu g/m^3$ NO_2)	All Admissions				Emergency Admissions			
		Crude		Adjusted[a]		Crude		Adjusted[a]	
		PR	(95%-CI)	PR	(95%-CI)	PR	(95%-CI)	PR	(95%-CI)
All Respiratory	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.17*	(1.07 to 1.28)	1.16*	(1.07 to 1.27)	1.27*	(1.10 to 1.45)	1.24*	(1.09 to 1.41)
	Q3 : 24.0-31.2	1.15*	(1.05 to 1.26)	1.14*	(1.04 to 1.24)	1.32*	(1.15 to 1.52)	1.23*	(1.08 to 1.39)
	Q4 : >31.2	1.16*	(1.06 to 1.27)	1.18*	(1.08 to 1.29)	1.35*	(1.17 to 1.55)	1.27*	(1.11 to 1.46)
Asthma	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.83*	(1.41 to 2.36)	1.87*	(1.46 to 2.40)	1.65*	(1.28 to 2.12)	1.70*	(1.33 to 2.17)
	Q3 : 24.0-31.2	2.30*	(1.78 to 2.98)	2.34*	(1.83 to 3.01)	1.95*	(1.51 to 2.51)	2.00*	(1.57 to 2.57)
	Q4 : >31.2	2.83*	(2.19 to 3.66)	2.81*	(2.16 to 3.65)	2.46*	(1.90 to 3.18)	2.47*	(1.90 to 3.20)
COPD	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.50*	(1.20 to 1.86)	1.44*	(1.19 to 1.74)	1.39*	(1.11 to 1.75)	1.34*	(1.09 to 1.65)
	Q3 : 24.0-31.2	1.76*	(1.41 to 2.18)	1.50*	(1.24 to 1.82)	1.49*	(1.49 to 1.87)	1.29*	(1.05 to 1.59)
	Q4 : >31.2	1.84*	(1.48 to 2.29)	1.60*	(1.31 to 1.96)	1.52*	(1.52 to 1.90)	1.31*	(1.06 to 1.63)
All Cardiovascular	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.11*	(1.00 to 1.23)	1.07	(0.99 to 1.15)	1.15*	(1.03 to 1.28)	1.12*	(1.03 to 1.22)
	Q3 : 24.0-31.2	1.22*	(1.11 to 1.35)	1.08*	(1.00 to 1.17)	1.32*	(1.18 to 1.48)	1.17*	(1.28 to 1.08)
	Q4 : >31.2	1.18*	(1.07 to 1.30)	1.10*	(1.01 to 1.19)	1.14*	(1.02 to 1.27)	1.07	(0.98 to 1.18)
IHD	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.15*	(1.02 to 1.28)	1.08	(0.98 to 1.20)	1.21*	(1.21 to 1.38)	1.18*	(1.05 to 1.33)
	Q3 : 24.0-31.2	1.27*	(1.13 to 1.42)	1.12*	(1.01 to 1.23)	1.37*	(1.20 to 1.57)	1.24*	(1.10 to 1.40)
	Q4 : >31.2	1.24*	(1.10 to 1.39)	1.13*	(1.02 to 1.25)	1.18*	(1.03 to 1.36)	1.14*	(1.01 to 1.30)
Stroke	Q1: <17.4	Reference		Reference		Reference		Reference	
	Q2 : 17.4-24.0	1.13	(0.98 to 1.29)	1.11	(0.98 to 1.25)	1.15	(0.95 to 1.39)	1.12	(0.94 to 1.33)
	Q3 : 24.0-31.2	1.25*	(1.09 to 1.44)	1.12	(0.99 to 1.26)	1.28*	(1.06 to 1.55)	1.12	(0.94 to 1.33)
	Q4 : >31.2	1.32*	(1.15 to 1.51)	1.28*	(1.13 to 1.45)	1.18	(0.97 to 1.43)	1.14	(0.95 to 1.36)

*: $P < 0.05$

[a]: adjusted for age, sex and income

DISCUSSION

This study shows that long-term exposure to traffic related air pollution in the area of residence is associated with the community prevalence of hospital admissions for respiratory and cardiovascular causes.

Internal Validity

In this study, exposure was assessed by NO_2-concentrations modeled by land use regression. For less than ten percent of the postcodes estimated NO_2-concentrations were above the annual European Air Quality Limit Value which is 40 μg/m^3. Elevated risks for hospital admission were thus seen at levels well below the EU Limit Value. We recognize that NO_2 is a marker for a complex mixture of traffic-related air pollutants in studies such as ours.

We assigned the average concentration of all six-position coordinates to the four-position postcode (PC4) area. Estimation of residential NO_2-exposure at PC4 level instead of the individual street address may have caused some misclassification. Moreover, by estimating long-term exposure at residence, we assumed that residential exposure is representative for an individuals' total personal exposure and may have introduced further exposure misclassification,[102,103] especially for those subjects who live in rural areas and who commute to and work in urban areas. Residential mobility, on which no information was available, may have been another source of misclassification. Such misclassifications may have made it more difficult to see exposure-response relationships in our data. Another limitation of the exposure assessment may be that the land-use regression model was developed based on measurement data for 2007, which is after the period for which hospitalization data were available (2000-2004). Recent studies, however, showed reasonable long-term validity of LUR models for periods of up to almost 10 year.[104,105]

The degree of urbanization is associated with cardiovascular and respiratory hospital admission.[98,99] Air pollution may be one of the possible explanations for these associations. Urbanization, however, is also associated with differences in life-style and, for instance, access to hospitals. We therefore additionally adjusted our analyses for the number of addresses per km^2 as an indicator of urbanization. As one of the predictors in the land use regression model for NO_2 was the percentage of residential land use in a 5 km buffer, which is correlated with the number of addresses per km^2 (r=0.60), adjustment for urbanization possibly resulted in overcorrection. Adjustment for urbanization indeed reduced the effect estimates for NO_2 in respiratory admission (particularly asthma) and stroke. Nevertheless, these associations remained statistically significant. In contrast, effect estimates for IHD and 'All cardiovascular causes' increased slightly after adjustment for urbanization.

In this study, adjustment was only possible for a limited number of covariates. The distribution of the population per postcode area based on age, sex and income was associated with exposure as well as hospital admission. The analysis of questionnaire based smoking data in the study area showed that smoking prevalence was somewhat elevated in the highest air pollution exposure quartile only. This could indicate that the elevated risk in the highest exposure group may be partly attributable to smoking. For several causes for admission, however, we observed increased risks in the second and third quartile of NO_2 concentrations also, whereas the smoking prevalence was identical in the first three quartiles of the NO_2 distribution. Furthermore, adjustment for COPD as proxy for smoking prevalence did not change the effect estimates in our study. Due to the nature of our study, we cannot exclude that other unmeasured variables related to occupation, diet, lifestyle etc. may have confounded our results.

Respiratory hospital admission

The association between short-term exposure to air pollution and hospital admission due to respiratory illness has been studied widely.[e.g. 20,22,23,106] Long-term exposure, however, have been studied less. A recent study by Andersen et. al[41] showed that long-term exposure to NO_2 (35 year mean) was associated with hospital admission for COPD (hazard ratio: 1.08 (95%-CI: 1.02-1.14) per interquartile range of 5.8 $\mu g/m^3$) among a cohort of 50 to 64 year olds. We also found a clear exposure-response relationship, between exposure to traffic related air pollution and risk for COPD admission. Our study furthermore showed elevated risk in the absence of an exposure-response relationship for all and emergency admissions for 'All respiratory causes'.

A case-control study by Lin and colleagues[64] in Erie County, NY, in which exposure was estimated at the residential address, indicated that children aged 0 to14 years old who were hospitalized for asthma were more likely to live in close proximity to heavy traffic compared to children who were not hospitalized. However, no clear exposure-response relation was found.[64] A similar case-control study among children aged 5-14 living in London was done by Wilkinson et. al.[69] Proximity to traffic was assessed at the postcode centroid level, and showed no association with asthma admission. In our study, a clear exposure-response relation was observed for all as well as emergency asthma admission. The ecological design of our study allowed us to incorporate a higher number of asthma admissions than in the studies by Lin[64] and Wilkinson[69] (5,252 vs. 417 and 2,131 respectively). Both aforementioned studies furthermore used proximity to traffic as main exposure estimate, whereas modeled NO_2-concentrations were used in the present study, indicating that NO_2 could be a more valid measure of exposure.

Cardiovascular hospital admission

Cardiovascular hospital admission was shown to be associated with air pollution in short-term exposure studies.[e.g.21,22] Only few long-term exposure studies on cardiovascular hospital admission were done so far. One study found no association for stroke,[68] another study presented a Rate Ratio of 1.13 for the highest (>57.7 μg/m^3 NO_x) relative to the lowest (<49.6 μg/m^3 NO_x) exposed subjects aged 45 years and over,[65] while no association with coronary heart disease was seen in the same population.[66] We found an increased risk with increased exposure for admission for 'All cardiovascular causes', IHD and stroke. Like Maheswaran et. al[65,66] we did the analyses at an area rather than an individual level. The underlying total number of admissions in our study, however, was much larger (e.g. 28,600 vs. 5,122 stroke admissions).

The effects were clearer for all admissions than for emergency admissions. This could be due to an effect of long-term exposure to air pollution above that of short-term effects of episodes of air pollution. Such episodes, which elevate emergency admission, are primarily weather driven and may affect all study areas simultaneously. Consistently our effect estimates are substantially larger than typical effect estimates of short-term exposure studies over the same concentration range.

In conclusion; long-term exposure to traffic related air pollution was associated with increased hospital admissions for respiratory and cardiovascular causes.

SUPPLEMENTAL MATERIAL

ANNEX A - Methods

Study area and population

The study was performed among all inhabitants (4.04 million) of the provinces of Noord-Holland and Flevoland, and the Rijnmond area, all in the West of the Netherlands (Figure 1). The study area (approx. 7,300km^2) consists of rural, suburban and urban areas including the cities of Amsterdam and Rotterdam. The population was studied by four-position postcode (PC4) area. Residential location data of the patients was not available at a more detailed spatial scale since hospitals do not record the full address in the National Medical Registration due to privacy protection laws. In the Netherlands, PC4 areas typically represent small villages or neighbourhoods. The city of Amsterdam (740,000 inhabitants), for instance, consists of 78 PC4 areas. In this study area, the total number of PC4 areas is 683 with an average population size of 5,916 inhabitants.

Supp.Mat Figure I: The study area (provinces of Flevoland, Noord-Holland, including the city of Amsterdam and the (sub)urban area of Rijnmond, including the city of Rotterdam).

Hospital admission and covariates

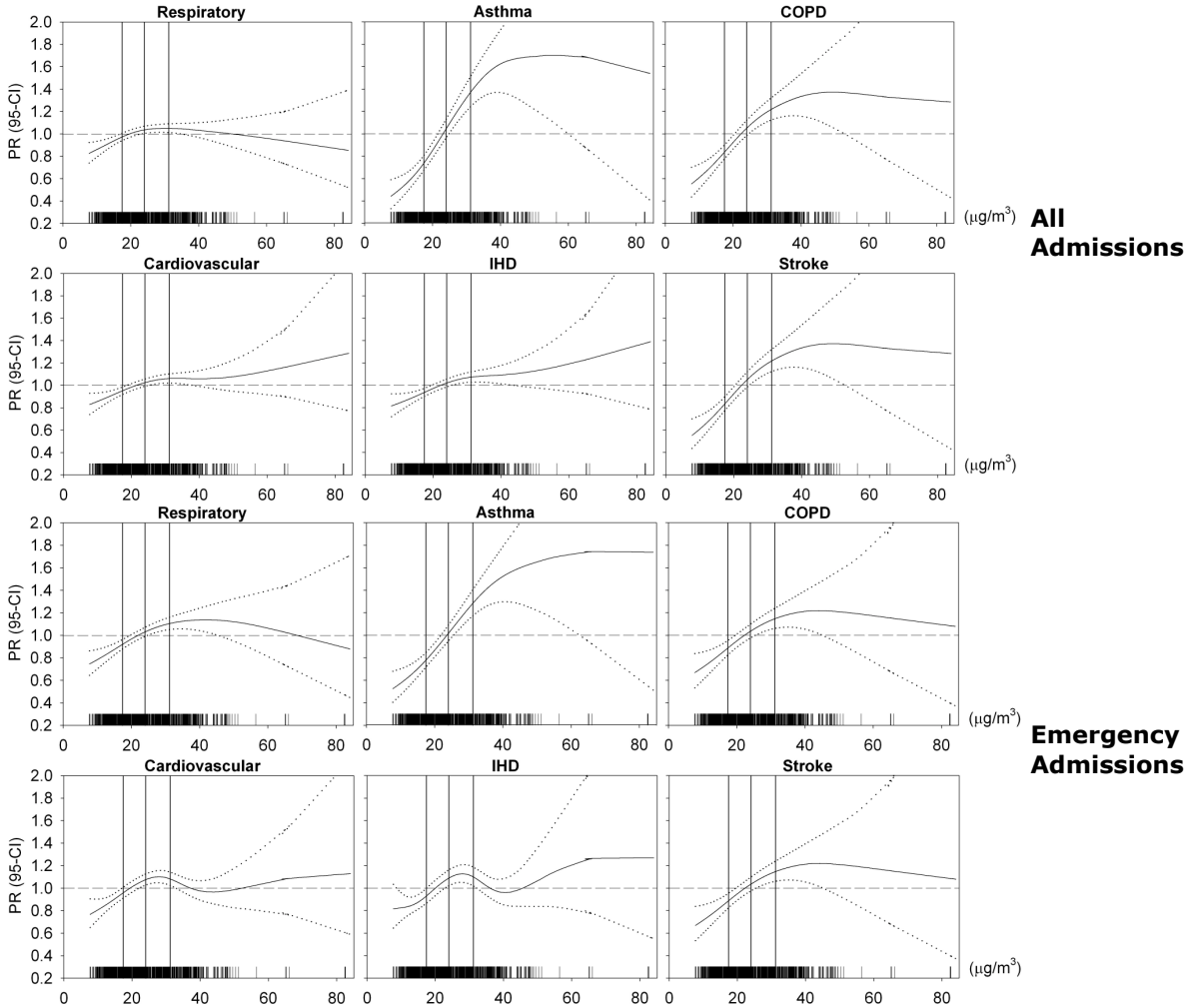
Hospital admission data were obtained from the National Medical Registration (LMR).[96] For the period January 1, 2001 to December 31, 2004, all hospital admission of inhabitants of the study area were registered. The registration consists of cause (ICD-9), date of admission and discharge, and whether the admission was planned or an emergency. Furthermore, registration of a limited number of personal characteristics (sex, date of birth, residential postcode) took place. In this study we focused on all (the total of emergency and planned) and emergency hospital admissions for respiratory (ICD-9: 460-519) and cardiovascular (ICD-9: 390-429) causes. Since no individual confounder or exposure data were available, analyses were done at postcode area level: we calculated the total number of persons admitted at least once for these ICD-9 codes per PC4 area. Multiple visits by the same person were counted once. The Dutch national Central Bureau of Statistics (CBS, Heerlen/Den Haag, the Netherlands) provided population data for the same period per PC4 area. For each PC4 area the total population and population by sex and age group (categorized as 0 to 20 years old, 20 to 65 years old and older than 65 years), mean income (Euro per month) and urbanization (in addresses per km^2) was available. Other population data generally provided by CBS, such as percentage of poor or rich households, had limited availability for PC4 areas included in this study due to privacy restrictions. For our analyses, the average values of the population data for the period 2001-2004 were used.

Exposure

Traffic related air pollution concentrations in this study were estimated using a land use regression model for NO_2. This model has been extensively described elsewhere.[97] In brief, NO_2 measurements were done using Ogawa badges at 60 sites located at rural, urban and traffic dominated locations in all four seasons of 2007. Potential predictor data were obtained from local authorities in the study area (traffic) and from geographic information system (GIS) databases (land use). Using ArcGIS software (version 9, ESRI, Redlands CA, USA), potential predictor variables such as land use in circular buffers, traffic volume at nearest road and distance to nearest freeway were created. A regression model was fitted by supervised forward selection to relate the mean measured concentration to the geographic land use and traffic data. The final

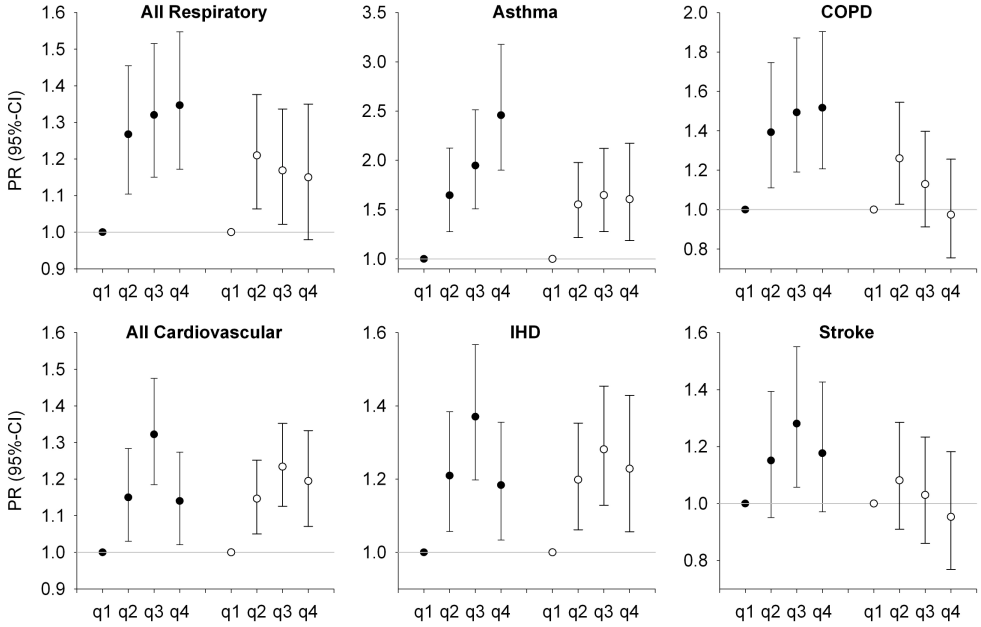
model included regional background concentration, traffic volume at the nearest road, distance to the nearest busy road and the percentage of residential land use in a circular buffer with a radius of 5 km. Leave one out cross-validation of the model resulted in a R^2 of 84%. Concentration estimations were done for the centroid of the six-position postcode (PC6) areas (approximately 20 addresses). For each PC4 area, the mean of the PC6 concentrations was calculated. On average, each PC4 area consisted of 141 PC6 areas (range 4-378).

Analysis



Supp.Mat Figure II: Smooth associations (PR and 95%-CI) between Residential Postcode Area NO_2 concentration and Hospital Admissions per Postcode Area

ANNEX B – RESULTS



Supp.Mat Figure III: Associations between residential postcode area NO_2 concentration (in quartiles: q1-q4) and emergency hospital admissions per postcode area, adjusted for age, sex and income (filled dots) and adjusted for age, sex, income and urbanization (open dots)

Chapter 4

Long-term Exposure to Traffic-related Air Pollution and Type 2 Diabetes Prevalence in a Cross-sectional Screening-study in the Netherlands

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ABSTRACT

Air pollution may promote type 2 diabetes by increasing adipose inflammation and insulin resistance. This study examined the relation between long-term exposure to traffic-related air pollution and type 2 diabetes prevalence among 50- to 75-year-old subjects living in Westfriesland, the Netherlands.

Participants were recruited in a cross-sectional diabetes screening-study conducted between 1998 and 2000. Exposure to traffic-related air pollution was characterized at the participants' home-address. Indicators of exposure were land use regression modeled nitrogen dioxide (NO_2) concentration, distance to the nearest main road, traffic flow at the nearest main road and traffic in a 250m circular buffer. Crude and age-, gender- and neighborhood income adjusted associations were examined by logistic regression.

8,018 participants were included, of whom 619 (8%) subjects had type 2 diabetes. Smoothed plots of exposure versus type 2 diabetes supported some association with traffic in a 250m buffer (the highest three quartiles compared to the lowest also showed increased prevalence, though non-significant and not increasing with increasing quartile), but not with the other exposure metrics. Modeled NO_2-concentration, distance to the nearest main road and traffic flow at the nearest main road were not associated with diabetes. Exposure-response relations seemed somewhat more pronounced for women than for men (non-significant).

We did not find consistent associations between type 2 diabetes prevalence and exposure to traffic-related air pollution, though there were some indications for a relation with traffic in a 250m buffer.

BACKGROUND

Many different factors are involved in the development of type 2 diabetes. Genetic predisposition, excess caloric intake and reduced physical activity are established and well-known determinants.[107] It has recently been hypothesized that long-term exposure to traffic-related air pollution might be an environmental risk factor for type 2 diabetes.[50-53]

Epidemiological studies have demonstrated that long-term exposure to traffic-related air pollution is associated with an increased risk for cardiopulmonary morbidity and mortality.[6,17] An hypothesis for the biological mechanism underlying these associations is that traffic-related air pollution triggers systemic oxidative stress and inflammation in for instance endothelial cells and macrophages.[6,48] These biological mechanisms are known to be involved in the development of insulin resistance seen in type 2 diabetes.[108,109] Consequently, it seems plausible that exposure to traffic-related air pollution could also be a risk factor for type 2 diabetes, like environmental tobacco smoke is.[110] At present, there is little data supporting this hypothesis. Recently, Sun et al.[52] demonstrated increased adiposity inflammation and whole-body insulin resistance in mice exposed to particulate matter air pollution. A study by Kramer et al.[51] further supported the plausibility of oxidative stress and inflammation as a biological mechanism for the relation between air pollution and type 2 diabetes, by showing that women with high C3c blood levels (a marker for subclinical inflammation) were more susceptible for particulate matter related excess risk of diabetes than were women with low C3c levels. That prospective study furthermore found a relation between traffic-related particulate matter and incident type 2 diabetes among elderly women in Germany.[51] Another epidemiological study, by Brook et al.,[50] found an association between modeled NO_2 exposure and type 2 diabetes prevalence among female patients, but not among male patients, of two respiratory health clinics in Canada. In addition, a recent American study found an association with distance to road among women, while no strong evidence of an association with particulate matter exposure was observed.[53]

The objective of the present study was to examine the relation between long-term exposure to traffic-related air pollution at the home-address and type 2 diabetes prevalence among subjects aged 50 to 75 years, living in a semi-rural region of the Netherlands.

METHODS

Study area and study population

The study was performed among residents of the semi-rural area of Westfriesland in the North-West of the Netherlands (Figure 1). The study area comprised three municipalities, consisting of seven towns and villages (Enkhuizen, Bovenkarspel, Grootebroek, Lutjebroek, Hoogkarspel, Westwoud and Oosterblokker). A large proportion of the estimated surface of 56 km^2 is used for agricultural activities, typically horticulture of tulips and cauliflower. Residents often commute to work in the area of Amsterdam, around 60 km away. No freeways are present in the study area. Two highways, known as provincial roads in the Netherlands, with a traffic flow of approximately 15,000 to 25,000 vehicles/24hrs, outline the North and South borders of the study area and are connected with the nearest freeway, located approximately 4 km to the west of the study area.

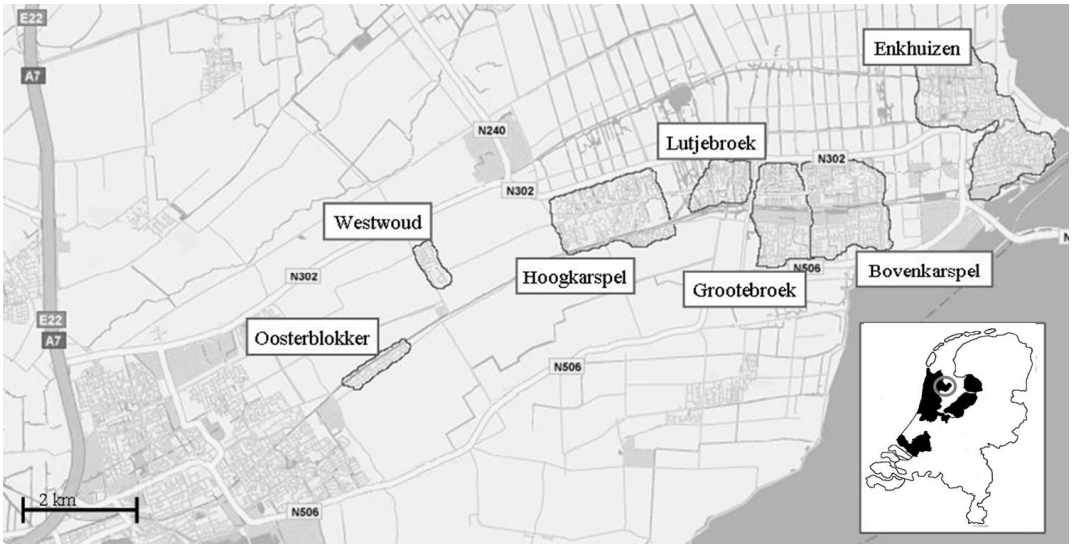


Figure 1. Study area and overview of specific location in the Netherlands. The study area consisted of three municipalities. Shown are the seven towns or villages within these municipalities, the highways (provincial roads) adjacent to the area and the nearest freeway, which is located to the west of the study area. The circle within the map of the Netherlands indicates were the study area is situated, the area marked in black is the area the NO2-model was developed for.

The study population has been described in more detail elsewhere.[111] In brief, between 1998 and 2000, all 50- to 75-year-old residents of the study area were invited to participate in the Hoorn Screening Study for type 2 diabetes. A total of 11,679 inhabitants received an invitation letter and the Symptom Risk Questionnaire, a screening instrument for undetected type 2 diabetes, which contained nine questions about age, gender, body length, body weight, family history of diabetes and health related problems like pain

when walking or frequent thirstiness.[112] BMI was derived of data on body length and -weight.

Of all responding participants (N=8,153), 417 (5%) reported previously doctor diagnosed diabetes. Participants with previously diagnosed diabetes were not required to complete the Symptom Risk Questionnaire and were not screened further. For the remaining 7,736 participants, risk-scores were calculated from the questionnaire. Participants with scores indicating a high risk profile for undetected type 2 diabetes (n=3,301) were asked to engage in further testing based on the 1999 World Health Organization guidelines for diagnosis of type 2 diabetes.[113] Further testing comprised fasting capillary glucose measurements. Depending on the outcomes of these capillary measurements, a venous fasting plasma glucose sample was taken, followed by either an oral glucose tolerance test or a second fasting plasma glucose measurement. The screening resulted in the diagnosis of 217 new cases of type 2 diabetes. Consequently, the Hoorn Screening Study population included 634 (8%) participants with type 2 diabetes.

The Dutch Central Bureau for Statistics provided additional population data on average monthly income of all residents in 2004 at a six-position postcode area level, which typically comprises about 20 dwellings.

Table 1. Characteristics of the total population and of participants with and without type 2 diabetes. Data are number (%) or mean (sd).

Characteristic	Total population	Type 2 Diabetes (Total)	Screening diagnosed Type 2 Diabetes	No Type 2 Diabetes
	(N=8018)	(N=619)	(n=213)	(N=7399)
Gender (male)	3,949 (49%)	330 (53%)	111 (52%)	3,619 (49%)
Age (years)				
50–55	2,753 (34%)	96 (16%)	28 (13%)	2,657 (36%)
55–60	1,795 (22%)	110 (18%)	38 (18%)	1,685 (23%)
60–65	1,446 (18%)	122 (20%)	45 (21%)	1,324 (18%)
≥ 65	2,024 (25%)	291 (47%)	102 (48%)	1,733 (24%)
BMI ($kg \cdot m^{-2}$)				
< 18.5	51 (1%)	3 (1%)	1 (1%)	48 (1%)
18.5–25.0	3,632 (45%)	130 (21%)	34 (16%)	3502 (47%)
25.0–30.0	3,344 (42%)	243 (39%)	108 (51%)	3101 (42%)
≥ 30.0	893 (11%)	145 (23%)	70 (33%)	748 (10%)
Missing	98 (1%)	98 (16%)	-	-
Average monthly income (€)	1,903 (417)	1,804 (407)	1,831 (464)	1,912 (417)
Total subjects with diabetes	619 (8%)	619 (100%)	213 (100%)	-
Subjects with pre-diagnosed diabetes	406 (5%)	406 (66%)	-	-

Exposure

Exposure to traffic-related air pollution was characterized at each participant's residential address at time of recruitment. All addresses were geocoded by means of the national GIS (Geographical Information System) database CAN,[114] which contains coordinates for all home addresses in the Netherlands. Exposure to traffic-related air pollution was defined by four different variables that have been demonstrated to be valid indicators of exposure:[37,115-117] modeled NO_2-concentration, distance to the nearest main road, traffic flow at the nearest main road and traffic within a 250 m circular buffer. NO_2 is considered an indicator of the complex mix of various gaseous and particulate components originating from both traffic combustion and wear of road and vehicles.

NO_2-concentrations at the home address were estimated by means of a land use regression model for the West of the Netherlands (Figure 1) that has been described elsewhere.[97] In brief, during one week in all four seasons of 2007, NO_2-measurements were performed using passive samplers at a total of 60 urban traffic dominated-, urban background- and rural background sites distributed over a large area (6,000km^2) in the West of the Netherlands, of which the current study area is part of. Traffic flow data were provided by all national, provincial and municipal authorities in the study area and were linked to a digital map of all roads in the Netherlands (NWB), using GIS. Other land use data were obtained from a European land use database (CORINE). Supervised forward selection was used to construct the land use regression model. The predictors in the final model were: background NO_2-concentration, traffic volume at the nearest road, distance to the nearest main road and residential land use in a 5km circular buffer. The cross-validation, adjusted, model R^2 was 82%.[97]

Furthermore, for each participants' residential address, other exposure indicators were derived from the traffic data described above using GIS: distance to the nearest main road (defined as a road with at least 5,000 vehicles/24hrs), traffic flow at the nearest main road (number of vehicles/24hrs), and total traffic per 24 hours on all roads within a 250 m circular buffer around the address. All GIS calculations were conducted using ArcInfo (ESRI, Redlands, CA).

Statistical analyses

Participants with missing values on exposure variables and the covariates age, gender and income were excluded from all analyses. We used penalized regression splines as implemented by Wood[118] in R (GAM procedure, mgcv-package of R version 2.8.0, R foundation for Statistical Computing, Vienna, Austria) to explore the functional relation between type 2 diabetes prevalence and the exposure variables. Since associations with type 2 diabetes seemed to be nonlinear, all exposure variables were analyzed in quartiles. As this

approach may have resulted in arbitrary intervals, which were sometimes quite narrow, smooth plots of the association between exposure and type 2 diabetes resulting from the GAM procedure were also presented for reference.

Logistic regression analysis was used to examine associations between type 2 diabetes prevalence and the different exposure variables. For each exposure variable, the quartile with the lowest level of exposure was chosen as the reference category. Analyses were performed with and without adjusting for a priori selected covariates age (continuous), gender, and average monthly income (continuous) as an indicator of neighborhood socio-economic status. Individually available covariates (gender, age and BMI) were also tested for effect modification. Stratified analyses were done by gender. Nationality was not adjusted for, as 99% of the population was Dutch. Since participants who reported previously diagnosed diabetes (n=417) were not required to complete the Symptom Risk Questionnaire, data on BMI was missing for 98 of these respondents. To be able to include all patients in the main analyses, we decided not to adjust for BMI in the main analyses, but to perform a sensitivity analysis to explore the potential confounding effect of BMI. In the sensitivity analysis we compared the results of covariate-adjusted (all previously mentioned covariates with and without additional adjustment for BMI) logistic regression analyses for the subgroup of participants with non-missing information on BMI. Additional sensitivity analysis was performed for type of diagnosis (self-reported previously doctor diagnosed and screening diagnosed), excluding participants with type 2 diabetes from the other diagnosis group. For all exposure variables, odds ratios (OR) and 95% confidence intervals (95%-CI) are presented. All analyses (besides the GAM analyses) were done with SAS 9.2 (SAS Institute Inc., Cary, NC, USA).

RESULTS

Participants living outside the study area (n=2), participants for whom geocoding of the home-address was not possible (due to a PO Box, boat or mail address, n=11) and participants with missing data on the covariates gender, age and income (average monthly income, n=118) were excluded from the study. This resulted in a study population of 8,018 participants, including 619 (8%) participants with type 2 diabetes, 406 previously diagnosed and 213 diagnosed in the Hoorn Screening Study. Forty-nine percent of the total population was male (Table 1) and median age of the total population was 58 years. The Box plots of the distribution of the exposure variables are presented in Figure 2. More detailed information about the distribution of the exposure variables and distributions for the participants with and without type 2 diabetes separately are presented in Table I of the Supplemental Material. Table I of the Supplemental Material also shows the distribution of the predictors of the NO_2 model. For one address the distance to the nearest busy road was outside the range of the distances for the monitoring sites based on which the model was developed (further away); all other predictors were within range of the original database.[97]

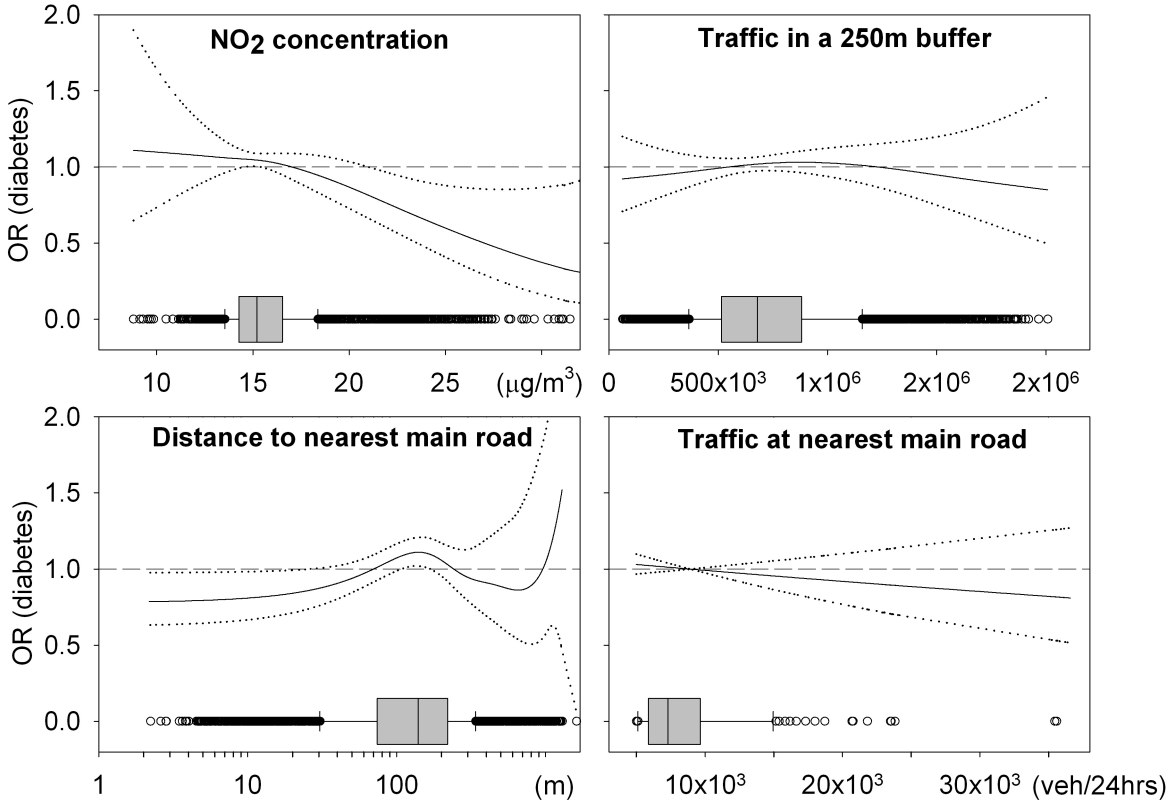


Figure 2. Smooth adjusted associations (OR and 95%-CI) between exposure variables and type 2 diabetes prevalence. Box plots on the x-axis present distribution of exposure variables.

Correlation between modeled NO_2-concentration and distance to the nearest main road was high (Spearman's r: -0.88). Distance to the nearest main road and traffic in a 250 m buffer were also correlated (0.63), as were modeled NO_2-concentration and traffic in a 250 m buffer (0.51). Traffic at the nearest main road was not correlated to the other exposure variables (r<0.2).

Table 2. Association between exposure variables and type 2 diabetes revalence: Odds Ratios with 95%-CI

Exposure Metric	Crude[a]	Adjusted[b]
NO_2-concentration ($\mu g \cdot m^{-3}$)		
Q1: 8.8-14.2	*reference*	*reference*
Q2: 14.2-15.2	0.98 (0.78 to 1.23)	1.03 (0.82-1.31)
Q3: 15.2-16.5	1.17 (0.94 to 1.45)	1.25 (0.99-1.56)
Q4: 16.5-36.0	0.80 (0.63 to 1.01)	0.80 (0.63-1.02)
Distance to nearest main road (m)		
Q1: 220-1610	*reference*	*reference*
Q2: 140-220	1.10 (0.87 to 1.39)	1.12 (0.88-1.42)
Q3: 74-140	1.22 (0.97 to 1.53)	1.17 (0.93-1.48)
Q4: 2-74	0.94 (0.74 to 1.19)	0.88 (0.70-1.13)
Traffic flow at nearest main road ($veh \cdot 24hrs^{-1}$)		
Q1: 5001-5871	*reference*	*reference*
Q2: 5871-7306	1.09 (0.87 to 1.39)	1.02 (0.81-1.29)
Q3: 7306-9670	0.98 (0.78 to 1.23)	1.03 (0.81-1.30)
Q4: 9670-	0.91 (0.72 to 1.16)	0.96 (0.75-1.22)
Traffic in 250 m buffer (10^3 $veh \cdot 24hrs^{-1}$)		
Q1: 63-516	*reference*	*reference*
Q2: 516-680	1.28 (1.01-1.61)	1.25 (0.99-1.59)
Q3: 680-882	1.15 (0.91-1.46)	1.13 (0.89-1.44)
Q4: 882-2007	1.13 (0.89-1.44)	1.09 (0.85-1.38)

[a]Crude model: not adjusted for any of the selected covariates
[b]Adjusted model: adjusted for average monthly income, age (continuous) and gender

Crude and adjusted associations between type 2 diabetes prevalence and the four indicators of exposure are shown in Supplemental Material-Figure I (crude smooth plots), Figure 2 (gender, age and neighborhood income adjusted smooth plots) and Table 2 (exposure quartiles, crude and adjusted). Both smoothing splines and analyses by exposure quartiles first show a slight increase in prevalence of diabetes with increasing modeled NO_2-concentration; then, when roughly modeled NO_2-concentrations exceeded the 75-percentile, the prevalence decreased and fell below the prevalence at the lowest modeled NO_2-concentrations. Overall, association between diabetes and modeled NO_2-

concentrations seems to be absent and is even slightly suggestive of an association counter to what was hypothesized.

The plots for distance to the nearest main road should be looked at reversely (highest distance means lowest exposure). To give a more true representation of the dispersion of air pollution from a road, the x-axis in the plots (distance) furthermore have a log scale. The plots, as well as the analyses per quartile, show an increasing prevalence with decreasing distance up until approximately the median. From there on, prevalence of diabetes drops and roughly at the 75-percentile, was below the prevalence at the largest distance (Table 2 and Figure 2). In some studies, distance to the nearest major road was dichotomized at cut-offs of 100m or 250m. In the present study, the age, gender and income adjusted OR for diabetes when living within 250m of a main road was 1.09 (95%CI: 0.87-1.36) relative to those living further away. For living within 100m this was 0.88 (0.74-1.05).

For traffic flow at the nearest main road, no association was seen with diabetes prevalence. Traffic in a 250m buffer, however, suggested some (statistically non-significant) increased diabetes prevalence for the higher exposures (roughly the upper three quartiles) although again prevalence decreases among the highest exposed.

Comparison of crude and adjusted models (Table 2, also Figure 2 vs. Supplemental Material-Figure I) demonstrates that inclusion of covariates in the adjusted models had little influence on the ORs and 95%-CIs. Additional adjustment for community did not change the results either (data not shown). Previous studies[50,51,53] suggest that gender could be an effect modifier, therefore analyses were stratified by gender (Figure 3). Patterns observed in the total population and described above seemed more pronounced among women than among men (also see Supplemental Material, Figure II). Statistically significantly increased odds were observed for modeled NO_2 and traffic in a 250m buffer (third quartile; 1.48 (1.07-2.04) and 1.44 (1.01-2.05), respectively). In regression analysis with exposure-gender interaction terms, however, the interaction was not statistically significant.

Sensitivity analyses were done to examine the potential confounding effect of BMI (Supplemental Material, Table II). In these analyses all participants with missing data on BMI (n=98), all of which had previously diagnosed diabetes, were excluded. Crude and adjusted analyses showed slightly higher ORs and wider 95%-CIs than in the total population (Table 2). Additional adjustment for BMI did not affect exposure-response patterns to a great extent. We therefore concluded that BMI was not an important confounder for the association between traffic related air pollution and diabetes prevalence in this population. We furthermore tested for effect modification, regression analysis with exposure-BMI interaction terms, did not show statistically significant interaction.

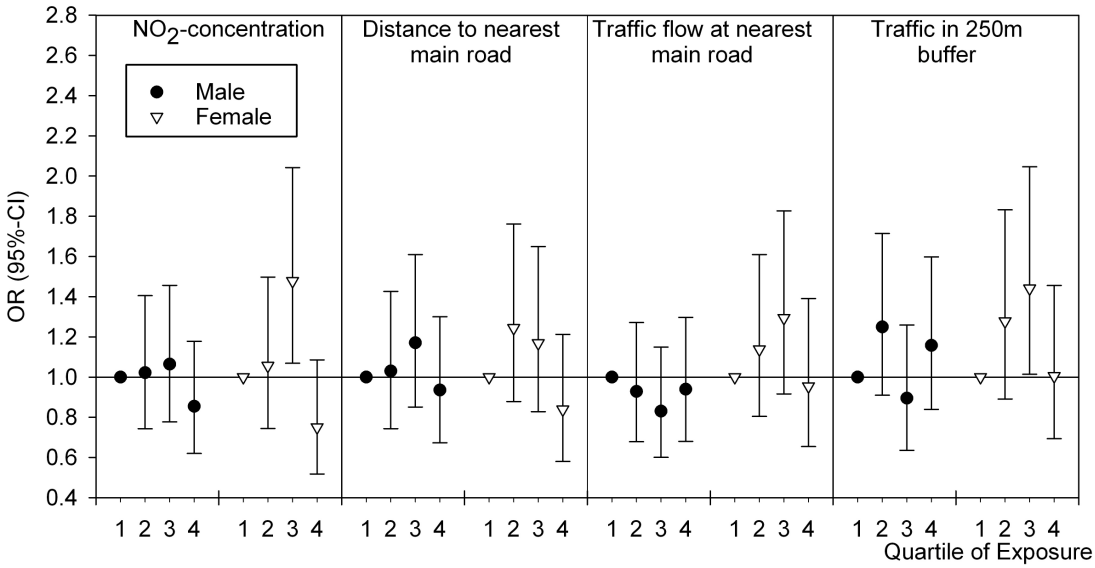


Figure 3. Analyses stratified by gender. Shown are ORs and 95%-CIs following from analyses adjusted for age and income.

We also performed sensitivity analyses for the different types of diagnosis (self-reported previously doctor diagnosed vs. diagnosed by the extensive screening in this study, Figure 4), showing that the participants with screening diagnosed diabetes contribute importantly to the findings of this study.

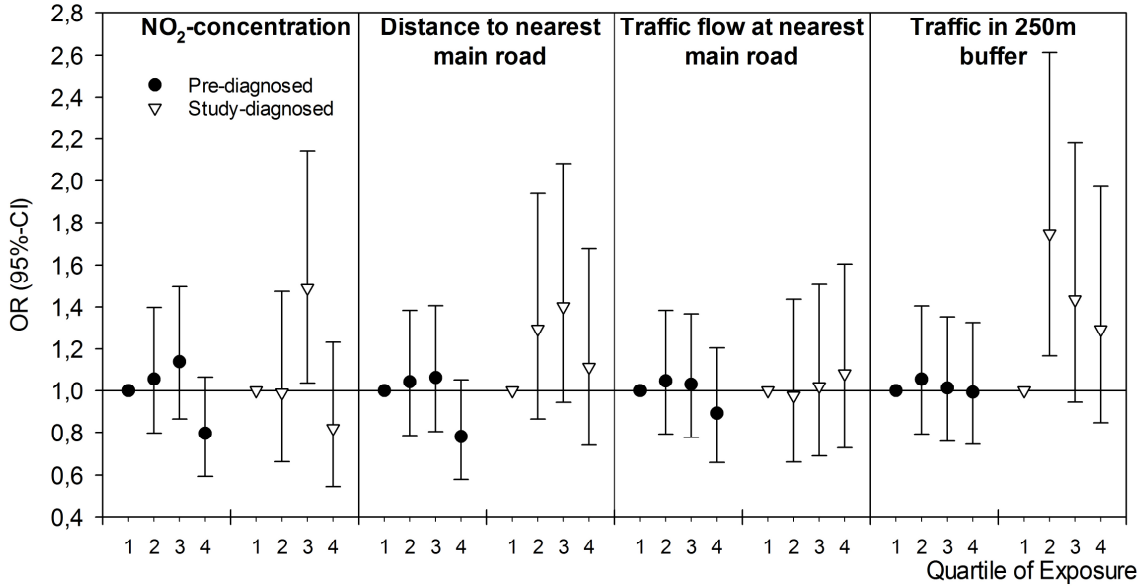


Figure 4. Analyses stratified by type of diagnosis. Shown are ORs and 95%-CIs following from analyses adjusted for age, gender and income. Dots are representing the ORs for self-reported previously doctor diagnosed diabetes (N=7,805), triangles represent screening diagnosed diabetes (N=7,612).

DISCUSSION

In this study, smooth plots of exposure versus type 2 diabetes risk supported some association with traffic in a 250m buffer. The prevalence of diabetes was (non-significantly) increased in the highest three quartiles compared to the lowest quartile, but did not increase with increasing quartile. Modeled NO_2-concentration, distance to the nearest main road and traffic flow at the nearest main road were not associated with diabetes. Associations seemed to be stronger for women compared to men.

Exposure in the study area

The area in which the Hoorn Screening Study was conducted has a relatively low level of air pollution, as documented with low NO_2-concentrations, and small exposure contrasts. Doing studies in areas with low exposures and small contrasts has advantages and disadvantages. One important aspect of such studies is that knowledge of possible health effects of air pollution at concentrations below current standards could be gained. A disadvantage is the potentially low study power. The latter may have limited our ability to detect a consistent association with traffic-related air pollution. Since other studies[e.g.119] observed effects in areas with low exposure and limited contrast, and several studies have shown largely linear associations between air pollution and e.g. cardiopulmonary mortality,[e.g.55] we considered exploration of a possible association in this study area to be worthwhile.

The limited ranges of exposure to traffic flow at the nearest main road and NO_2-concentration could have contributed to inconsistent findings. For instance, the interquartile range for NO_2-exposure in this study was only 2.3 $\mu g/m^3$, while in previous studies on air pollution and type 2 diabetes[50,51] this ranged from 5.8 to 15.0 $\mu g/m^3$. The relatively long tails at both ends of the exposure range, may furthermore have contributed to the absence of an exposure-response relation in this study: the range of exposure within the highest exposed quartile for NO_2 (16.5-36.0 $\mu g/m^3$) was much larger than the interquartile range. As shown in Figure 2, however, analysis exploiting the full contrast shows no increased odds with increased NO_2-concentration either.

Exposure-effect relation

In the present study, associations for different indicators of air pollution did not show consistent results. Whereas increased exposure as measured by traffic in a 250m circular buffer was associated with slightly increased odds for type 2 diabetes, this pattern was less clear for distance to the nearest main road and modeled NO_2-concentration and absent for traffic flow at the nearest main road. However, different associations for different exposure metrics were also observed in a cohort study on cardiovascular mortality in the Netherlands.[37] The exposure-response pattern for NO_2-concentration and

distance to the nearest main road in this study was similar, most likely due to the high correlation between the two variables. Distance to the nearest main road is a metric being increasingly used in policy practice, modeled NO_2-concentration, however, is probably a more precise metric of exposure to traffic related air pollution.

Potential misclassification of exposure

Exposure was characterized at the home-address. Despite high correlation between outdoor exposure at the home-address and overall exposure to traffic-related air pollution,[117] personal differences in exposure, caused by, for instance, occupational or commuting exposure could have resulted in exposure misclassification. In addition, it is unknown for what time period participants had resided in the study area at the time of enrollment. Residential mobility among elderly persons in the Netherlands, however, tends to be low[47,120] and therefore we believe that estimated exposures in the present study represent long-term exposures of the study participants. Exposure and participant data were furthermore obtained at different moments in time. As the study area is a stable environment where no major modifications in housing or the road network have occurred in the past twenty years, we do not think that spatial variation of exposure has changed much over time. Recent studies showed reasonable long-term validity of land use regression models.[104,105] Indicators such as distance to the nearest main road may be even more stable over time than air pollution concentrations.

As exposure was characterized at the geocoded home-address, spatial error in the database that was used for geocoding may have contributed to exposure misclassification. Geocoding was done with ACN, of which the accuracy is high (93.5% located at centroid of the correct building, 6.0% at the centroid of the correct parcel[121]). We therefore believe that misclassification of exposure due to spatial error in the geo coded home-address, if any, is small.

Study design

Ideally, epidemiological studies on the health effect of environmental exposures such as air pollution are conducted in a prospective cohort design. In order to study conditions such as type 2 diabetes in a cohort with sufficient power, a long follow-up time is needed and the size of the cohort has to be substantial. Since this is very time-consuming and costly, cross-sectional studies, such as the Hoorn Screening Study, can contribute to the understanding of such associations considerably in absence of cohort studies.

The Hoorn Screening Study is a cross-sectional study among a representative study population and the prevalence of diabetes is well-described. In questionnaire based studies, selection bias may be of importance. In the Hoorn Screening Study, selection bias was minimized by

inviting all 50- to 75-year-old inhabitants of the study area to participate and non-response was low (20%).[111] In general, type 2 diabetes remains undiagnosed in up to 30-55% of the cases. A strength of the present study is that many of these undiagnosed patients were detected.[111] About one third of the patients with type 2 diabetes in this study were diagnosed by the extensive screening procedure. Sensitivity analyses for type of diagnosis (self-reported vs. screen-detected, Figure 4) shows that the screening detected patients with type 2 diabetes contributed importantly to the findings of this study, a finding which may be of importance for setting up future studies. As subjects diagnosed in the screening were unaware of their disease, bias in especially this group seems unlikely. Although some misclassification might have occurred in the group of self-reported patients with type 2 diabetes, it is unlikely that this is related to exposure. This misclassification would therefore probably result in less pronounced effects, if any.

Confounding and effect modification

Comparison of crude and adjusted models indicated little confounding of the relation between type 2 diabetes and exposure variables. We cannot rule out residual confounding by other unmeasured factors such as lifestyle, personal socio-economic status, etc. For example, no data were available on smoking status or prior cardiovascular disease, which are important risk factors for type 2 diabetes. In the three published epidemiological studies exploring the relation between traffic-related air pollution and diabetes, Brook et al.[50] adjusted for the same factors as in our study, whereas Krämer et al.[51] and Puett et al.[53] had more detailed individual information available. Neither of these studies however indicated those characteristics to be important confounders in the association between diabetes and air pollution. In several studies on cardiopulmonary health[122-124], it also seemed that adjustment for important risk factors such as smoking, had little influence on the relation between cardiopulmonary health and traffic-related air pollution. This is consistent with our findings, in which adjustment for gender, age and an indicator of socio-economic status (neighborhood average income) indicated that these were not confounders for the relation with traffic-related air pollution. Sensitivity analyses on the potential confounding effect of BMI showed furthermore no indication of confounding by BMI in this population (Supplemental Material Table II, Model III vs. Model II) although residual confounding cannot completely be ruled out.

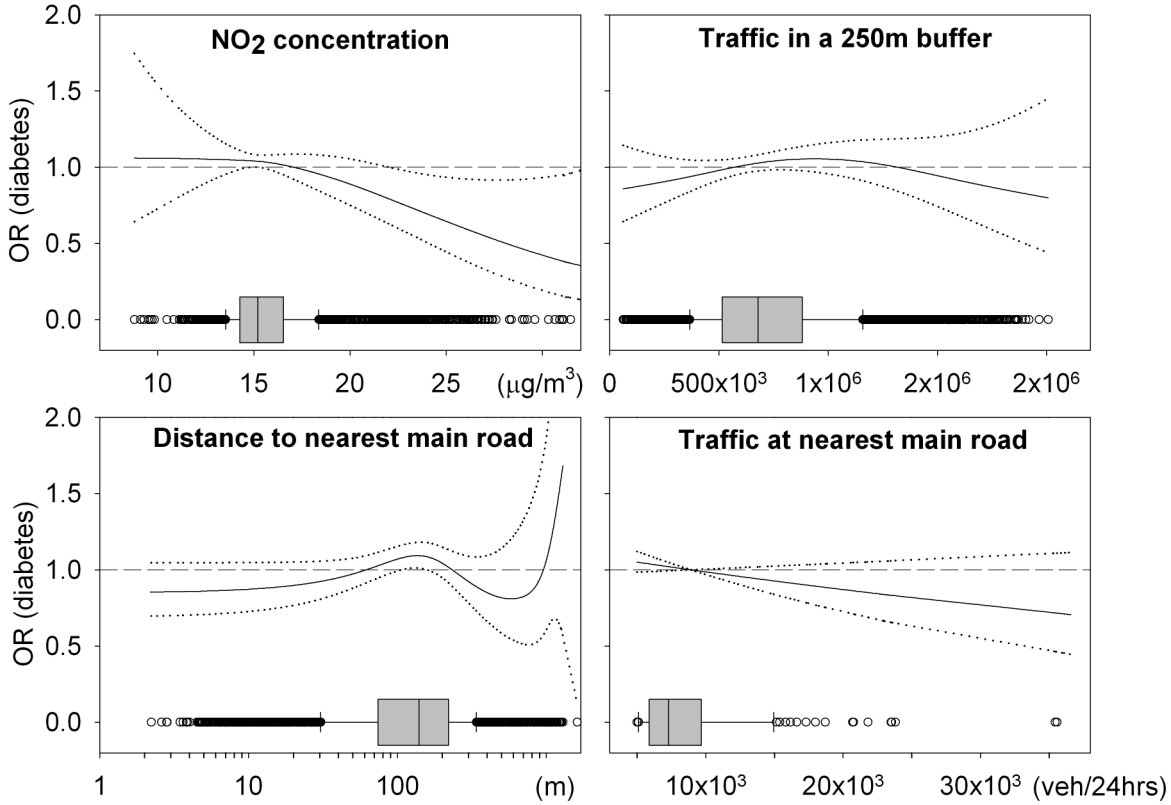
Krämer et al.[51] showed associations between traffic-related air pollution and incident type 2 diabetes among elderly women in a prospective study. For NO_2, the adjusted relative risk (RR) was 1.42 (95%-CI: 1.16-1.73) per 19 $\mu g/m^3$. Brook et al.[50] demonstrated a relation between modeled NO_2-concentration and type 2 diabetes prevalence among women (OR 1.04 (1.00-1.08) per ppb), but not among men. Puett et al.[53] observed an increased

hazard ratio of 1.14 (1.03-1.27) for living less than 50m versus ≥200m from a roadway among women. In our study, patterns observed in the full population seemed to be more pronounced among women, which is consistent with the studies by Brook, Puett and Krämer. In regression analysis, however, no statistically significant interaction by gender was shown. Among the potential explanations for a possible difference between men and women is accuracy of exposure estimation, which may be more accurate in women than in men. The women in this population are of a generation in which working outside of the home was rare. At the time of screening, women in this study therefore were more likely to have spent more time at home than men. Furthermore, susceptibility may differ between women and men.

Conclusion

This study did not find consistent associations between type 2 diabetes prevalence and exposure to traffic related air pollution, though there were some indications for a relation with traffic in a 250m buffer. Our study adds to the limited number of studies on air pollution as a risk factor for type 2 diabetes.[50-53] In contrast with previous epidemiological studies[50,51,53] we did not find consistent associations, though despite the limited level of exposure in the population studied, some indications for a relation were observed.

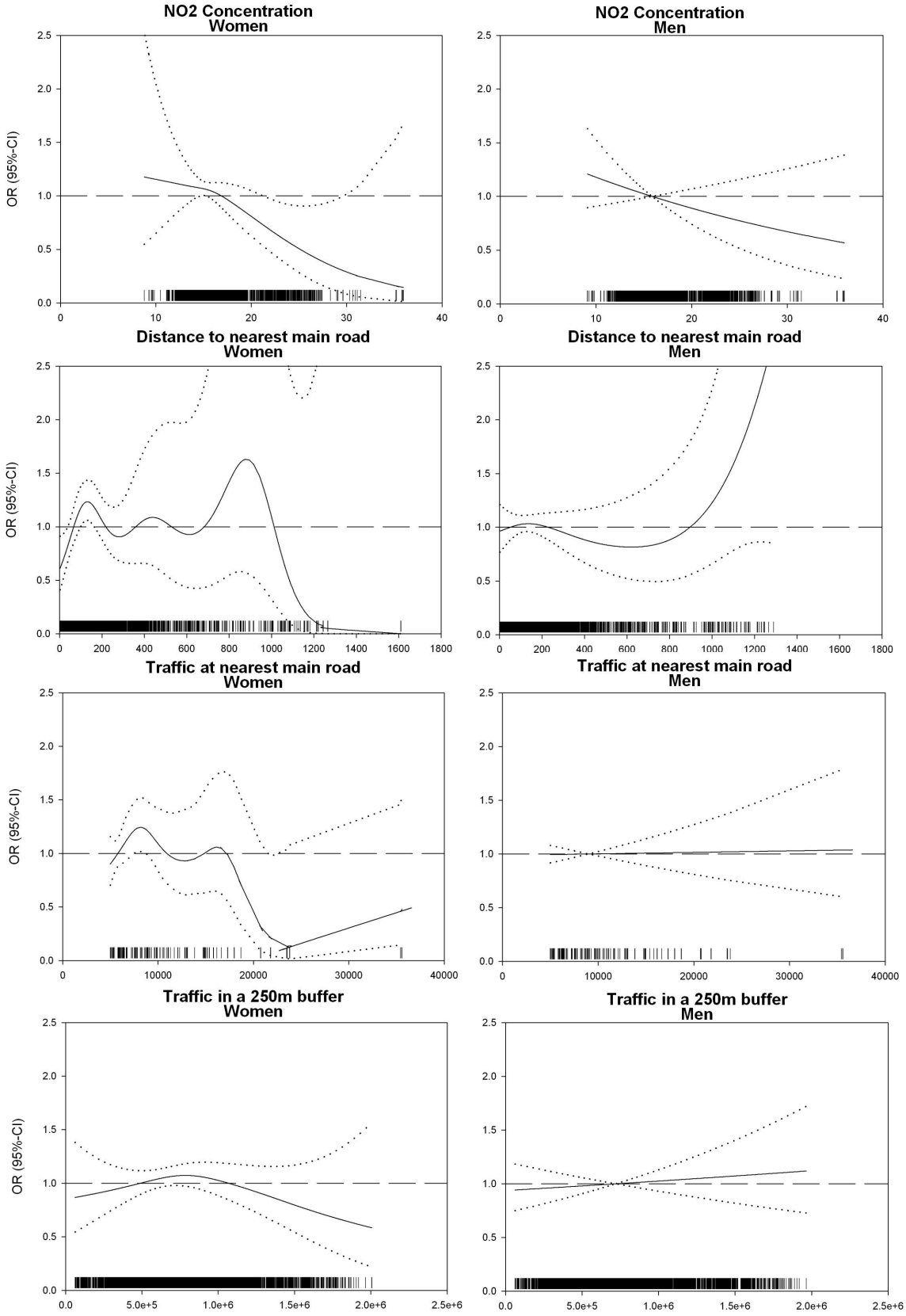
SUPPLEMENTAL MATERIAL



Supp.Mat. Figure I: Smooth crude associations (OR and 95%-CI) between exposure variables and type 2 diabetes prevalence. Box plots on the x-axis present distribution of exposure variables.

Supp.Mat. Table I: Distribution of exposure variables (and the predictors of the NO2 LUR model) for the total population and for the people with and without Type 2 Diabetes separately.

Exposure variable	minimum	10th percentile	25th percentile	median	75th percentile	90th percentile	maximum
TOTAL POPULATION							
Modeled NO_2-concentration ($\mu g \cdot m^{-3}$)	8.8	13.5	14.2	15.2	16.5	18.4	36.0
Predictors:							
Background concentration ($\mu g \cdot m^{-3}$)	*13.4*	*13.5*	*13.5*	*13.5*	*13.6*	*14.6*	*16.8*
Traffic flow at nearest road ($veh \cdot 24hrs^{-1}$)	*1225*	*1225*	*1225*	*1225*	*1225*	*4745*	*35567*
Distance to nearest main road (m)	*2*	*30*	*74*	*140*	*220*	*338*	*1610*
Residential land use in 5km buffer (%)	*4*	*9*	*10*	*13*	*14*	*15*	*17*
Distance nearest main road (m)	2	30	74	140	220	338	1610
Traffic flow nearest main road ($veh \cdot 24hrs^{-1}$)	5001	5115	5871	7306	9670	14970	35567
Traffic within 250 m buffer ($10^3 \cdot 24hrs^{-1}$)	63	367	516	680	882	1158	2007
TYPE 2 DIABETES							
Modeled NO_2-concentration ($\mu g \cdot m^{-3}$)	11.2	13.5	14.3	15.2	16.3	18.0	26.4
Predictors:							
Background concentration ($\mu g \cdot m^{-3}$)	*13.4*	*13.5*	*13.5*	*13.5*	*13.6*	*13.7*	*16.8*
Traffic flow at nearest road ($veh \cdot 24hrs^{-1}$)	*1225*	*1225*	*1225*	*1225*	*1225*	*1804*	*17304*
Distance to nearest main road (m)	*4*	*35*	*81*	*138*	*208*	*324*	*1266*
Residential land use in 5km buffer (%)	*8*	*9*	*10*	*13*	*14*	*15*	*16*
Distance nearest main road (m)	4	35	81	138	208	324	1266
Traffic flow nearest main road ($veh \cdot 24hrs^{-1}$)	5001	5115	5871	6790	9278	14736	35567
Traffic within 250 m buffer ($10^3 \cdot 24hrs^{-1}$)	65	392	531	683	878	1146	1860
NO TYPE 2 DIABETES							
Modeled NO_2-concentration ($\mu g \cdot m^{-3}$)	8.8	13.5	14.3	15.2	16.6	18.4	36.0
Predictors:							
Background concentration ($\mu g \cdot m^{-3}$)	*13.4*	*13.5*	*13.5*	*13.5*	*13.6*	*14.6*	*16.8*
Traffic flow at nearest road ($veh \cdot 24hrs^{-1}$)	*1225*	*1225*	*1225*	*1225*	*1225*	*4745*	*35567*
Distance to nearest main road (m)	*2*	*30*	*73*	*140*	*220*	*343*	*1610*
Residential land use in 5km buffer (%)	*4*	*9*	*10*	*13*	*14*	*15*	*17*
Distance nearest main road (m)	2	30	73	140	220	343	1610
Traffic flow nearest main road ($veh \cdot 24hrs^{-1}$)	5001	5115	5871	7306	9670	14970	35567
Traffic within 250 m buffer ($10^3 \cdot 24hrs^{-1}$)	63	367	516	680	882	1158	2006



Supp.Mat. Figure II: Smooth (age and income) adjusted associations (OR and 95%-CI) between exposure variables and type 2 diabetes prevalence, stratified by gender. Lines on the x-axis represent the distribution of exposure.

Supp.Mat. Table II: Association between exposure variables and type 2 diabetes prevalence: sensitivity analyses for BMI within a population of 7,920 participants, including 521 (7%) participants with type 2 diabetes. Excluded are 98 participants (1% of total population) with previously diagnosed type 2 diabetes, because of missing BMI data.

	Model I: Crude[a]	Model II: Adjusted[b]	Model III: Adjusted + BMI[c]
NO_2-concentration			
Q1	*reference*	*reference*	*reference*
Q2	1.08 (0.84 to1.39)	1.14 (0.88 to1.47)	1.11 (0.85 to1.44)
Q3	1.31 (1.03 to1.66)	1.39 (1.09 to1.78)	1.39 (1.08 to1.78)
Q4	0.84 (0.65 to1.09)	0.84 (0.64 to1.09)	0.84 (0.64 to1.10)
Distance to nearest main road			
Q1	*reference*	*reference*	*reference*
Q2	1.18 (0.92 to1.54)	1.21 (0.93 to1.57)	1.20 (0.92 to1.56)
Q3	1.36 (1.06 to1.74)	1.30 (1.00 to1.67)	1.30 (0.99 to1.68)
Q4	1.05 (0.80 to1.36)	0.98 (0.75 to1.28)	0.99 (0.75 to1.30)
Traffic flow at nearest main road			
Q1	*reference*	*reference*	*reference*
Q2	1.08 (0.84 to1.38)	1.01 (0.78 to1.29)	1.02 (0.79 to1.32)
Q3	0.96 (0.75 to1.23)	0.99 (0.78 to1.28)	1.01 (0.78 to1.31)
Q4	0.89 (0.69 to1.15)	0.93 (0.71 to1.21)	0.91 (0.70 to1.19)
Traffic in 250m buffer			
Q1	*reference*	*reference*	*reference*
Q2	1.43 (1.10 to1.84)	1.40 (1.08 to1.81)	1.34 (1.03 to1.75)
Q3	1.25 (0.96 to1.62)	1.22 (0.94 to1.59)	1.20 (0.92 to1.57)
Q4	1.24 (0.96 to1.62)	1.19 (0.91 to1.55)	1.19 (0.91 to1.56)

[a]Model I (crude model): crude relation between type 2 diabetes and exposure variables; not adjusted for any of the a priori selected covariates.
[b]Model II (adjusted model): adjusted for a priori selected covariates average monthly income, age (continuous) and gender.
[c]Model III (adjusted model +BMI): adjusted for a priori selected covariates and BMI.

Chapter 5

Air Quality Effects of an Urban Highway Speed Limit Reduction

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ABSTRACT

A speed limit intervention on part of the Amsterdam ring highway, adjoined with apartment buildings, was implemented.

The objective of this study was to assess whether, and to what extent, a lowering of the maximum speed limit from 100 to 80 kph had reduced traffic related air pollution in the direct vicinity of a highway. A monitoring station of the Amsterdam Air Quality Monitoring Network is situated adjacent to the intervened road section. Daily mean concentrations (PM_{10}, PM_1, Black Smoke and NO_x) in the first year since the intervention were compared with measured concentrations in the prior year. The intervention effect was adjusted for daily traffic flow, congestion and downwind exposure. The concentration changes were compared with those observed at a section of the same ring highway where the speed limit had not been reduced.

Since the intervention, the adjusted traffic contribution to PM_{10}-concentrations has decreased by 2.20 $\mu g/m^3$ (95%-CI: 1.43–2.98), PM_1 0.42 $\mu g/m^3$ (95%-CI: 0.01–0.82) and Black Smoke 3.57 $\mu g/m^3$ (95%-CI: 1.50–5.65). At the not intervened highway section the adjusted traffic contribution to PM10 and Black Smoke concentrations has also decreased by 0.97 and 2.43 $\mu g/m^3$ respectively. However, decreases were significantly greater for PM_{10} and PM_1 at the intervention site.

In conclusion, this study demonstrates a significant reduction of PM_{10} and PM_1 as a result of reducing the speed limit at an urban ring highway.

INTRODUCTION

Air pollution and associated health effects have been the subject of extensive research for many years.[6,17] More recently, health effects of traffic related air pollution have gained importance. Living near busy roads or attending school there has been shown to be associated with a reduction in lung-function growth[56] and an increase in chronic respiratory symptoms in children.[125-127] Other studies show that adults living near busy roads suffer more from respiratory symptoms[39] and heart disease,[120,128,129] compared to adults living further away from busy roads.

Because of the great public-health impact, Künzli et al.[130] concluded that traffic related air pollution should be a key target in public-health in Europe. To reduce general exposure to air pollution, the European Union has set air quality standards. In Amsterdam (730,000 inhabitants), air quality standards for PM_{10} and NO_2 are exceeded on many locations, especially along busy roads. All over Europe, many policy measures are taken to reduce traffic emission and thereby improve air quality. The effectiveness of such measures, however, has rarely been quantified. As stated by Int Panis et al.,[131] it is hardly ever feasible to directly measure the effect of a policy measure on vehicle emission and air pollution concentrations. To study these effects, emission- and dispersion modelling are often combined. In February 2003, a congestion charge was implemented in central London. Based on detailed traffic data and a local road traffic emissions model, an estimated emission reduction of 12% NO_x and 11.9% PM_{10} was achieved within the charging zone.[132] Using more extensive air pollution concentration modelling and exposure–response relationships from literature, Tonne et al.[133] modelled the resulting life expectancy impact of the London congestion charge. A modest benefit was found.

In the Netherlands, dispersion models[10] suggest that traffic related emissions at highways being substantially affected by the maximum driving speed. More strict speed limits on highways with many people living in close proximity are set to reduce exposure and related health effects. However, speed limitation measures taken elsewhere raised concern about air pollution concentrations which may increase due to delay and congestion.[134]

Starting November 2005 the Dutch National Department of Transport limited the maximum speed from 100 to 80 km per hour (62–50 miles per hour) on some specific stretches of urban highway. All over the country, the maximum speed for heavy duty vehicles already was 80 kph.

The Amsterdam ring highway (A10) is one of the busiest highways in the Netherlands. It typically consists of six lanes, three in both directions. During rush hours, congestion appears on every working day. Along the western section of the ring highway, apartment buildings are located at less than 20 m on either side of the road (<20 m), creating a situation resembling a street

canyon. This road section, which covers 6 km (3.7 miles), is where the 80-kph speed measure was implemented (Figure 1). Drivers are informed of this speed limit by many road signs, no additional devices causing traffic interruptions, such as speed control traffic signals, are used. This speed limit, however, is automatically adhered to through monitoring of vehicle specific trajectory driving speed and stringent fines.

Approximately 40,500 people live within close proximity that is within 500 m of the road section where the intervention was taken.

The Dutch National Transport Research Center conducted calculations prior to taking the policy measure. Estimated emission reduction on the Amsterdam highway was 14% for PM_{10} and 10–15% for NO_2. According to the Dutch National Department of Transport, this would lead to a concentration decrease of 0.5–1% for PM_{10} and 2–4% for NO_2 adjacent to the road.[135]

The objective of this study is to assess whether, and to what extent, the policy to lower the maximum speed limit from 100 to 80 kph on part of the Amsterdam ring highway has reduced measured traffic related air pollution in the direct vicinity of the highway.

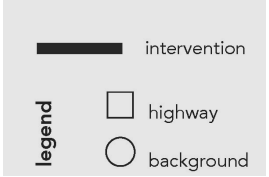
METHODS

Traffic

About 92,000 vehicles/day pass the western section of the Amsterdam ring highway (current speed limit: 80 kph), while about 140,000 vehicles pass the southern section (current speed limit: 100 kph, no intervention). Road management and continuous traffic monitoring are performed by the Dutch National Department of Public Works. Daily mean traffic flow, congestion parameters, as well as information on road closure and road works were obtained from this department.

Traffic flow measurements included all vehicles. Unfortunately, no data on heavy duty vehicles or other vehicle types were available. Daily mean intensities were available for analysis. As a parameter for traffic congestion, additional vehicle hours (AVH) were used. This parameter, commonly used by the Dutch National Department of Public Works, is the total of extra hours needed to complete the specified road stretch, for all vehicles passing, relative to the time it would take at a normalised driving speed. For this specific road section, the National Department of Public Works decided on a normalised driving speed of 70-kph. The provided AVH was calculated from minute to minute data on driving speed and traffic flow per lane. A daily total of AVH, referred to as traffic congestion, was used for analysis.

	Distance to nearest highway	**PM_{10}** $\mu g/m^3$ TEOM	**PM_1** $\mu g/m^3$ TEOM	**BS** $\mu g/m^3$ SX-200	**NO_x** ppb EcoPhysics
A10W	6.7 m	■	■	■	■
A10S	8.0 m	■	■	■	■
BN	1.4 km	-	-	■	■
BC	1.7 km	■	■	■	■
BW	1.7 km	■	-	-	-



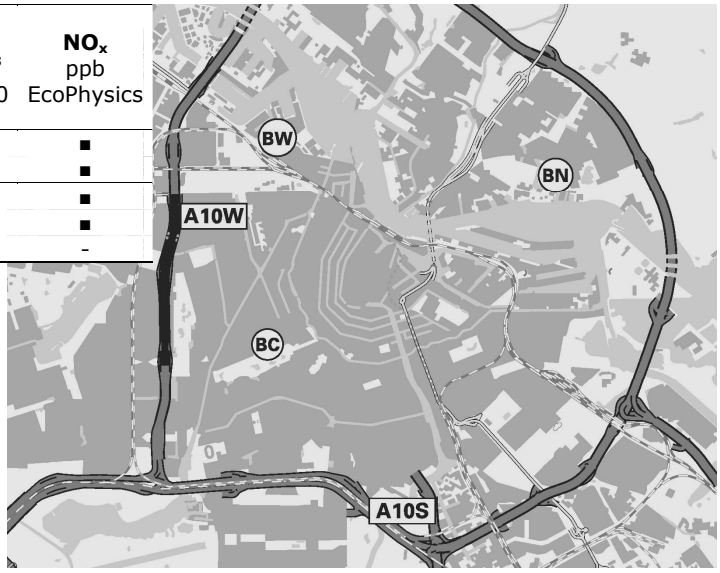


Figure 1. Location of the speed limit intervention road section, the monitoring stations in Amsterdam Air Quality Monitoring Network, and monitored components

Air pollution

Within the Amsterdam Air Quality Monitoring Network, particulate matter (PM_{10}, $PM_{2.5}$ or PM_1), nitrogen oxides (NO_x) and a proxy of soot (Black Smoke, BS) are continuously monitored at urban background and roadside locations in

the Amsterdam city area. One of the roadside stations is located along the western section of the ring highway (Figure 1). This is where the speed limit intervention measure was taken. The inlets of this monitoring station are located at 2.7 m elevation at 6.7 m East of the edge of the highway. The fixed site monitoring station was not specially configured to study the speed limit intervention.

Figure 2. Monitoring stations A10W (left) and A10S (right)

Another roadside station is located at the southern part of the ring highway. The southern station has a slightly different positioning; the inlets of this monitoring station are located at 2.7 m elevation at 8.0 m north of the edge of the highway. Figure 2 shows pictures of both monitoring stations. There are no buildings adjoining this road section. Furthermore, the monitoring network has three background stations (west, north and central), located at least 60 m from major roads. For most components, data on urban background concentrations are available from at least two urban background monitoring stations; PM_1 is available from one station only. Figure 1 shows locations of the air quality monitoring stations and monitored components.

Daily mean concentrations ($\mu g/m^3$) of PM_{10} and PM_1 were derived from continuous monitoring using tapered element oscillating microbalance (TEOM) as described in detail by Roemer and van Wijnen.[12] The reported concentrations are measured concentrations, so no additional correction for volatile components,[136] obligatory for legal purposes, was done. $PM_{2.5}$ was not available at the western roadside station. BS-concentrations in $\mu g/m^3$ were obtained using SX-200 continuous monitors.[12]

Nitrogen oxides are measured using chemiluminescence monitors (EcoPhysics, Switzerland, type CLD 700AL). For NO_x daily mean concentrations in ppb were available for analysis.

The Amsterdam Air Quality Monitoring Network complies with the accreditation criteria ISO/IEC 17025:2005 for test laboratories. Also, PM_{10} monitoring is in accordance with A 3580.9.8/NEN EN 1234 1, NO_x with NEN-ISO 7996. For PM_1 and BS no accreditation is available. PM_1, however, is

measured using a monitor identical to that for PM_{10}, only using a different inlet. BS monitoring and quality procedures are performed according to the manufacturer's recommendations (ETL, Hereford, England).

Meteorology and long range air pollution

Daily air pollution concentrations are also determined by factors other than local sources, such as meteorology and long range air pollution. In several studies, the contribution of traffic was studied by subtracting background concentrations.[136,137] In this study, Spearman's correlations between concentrations measured at three background sites in the same urban area (Figure 1) were high (PM_{10} 0.91, BS 0.75, NO_x 0.84), as were correlations between mean background and roadside concentrations (PM_{10} 0.86, PM_1 0.91, BS 0.64, NO_x 0.71). These coefficients reflect that meteorology (i.e. rainy days, warm and dry summer months or periods with specific predominant wind directions) and other long range atmospheric processes affect the concentrations over the whole city in a similar way. For both roadside monitoring stations, daily 'traffic contribution' concentrations were derived by subtracting same day mean background concentrations.

Table 1: Roadside and Background concentrations[a] of air pollutants in Amsterdam, one year prior to the intervention[b] (November 2004 to November 2005)

		Roadside			Traffic contribution[c]		
		N	Mean	Range (min-max)	N	Mean	Range (min-max)
PM_{10} ($\mu g/m^3$)	Highway West	331	29.72	(12.60 to 85.50)	331	8.18	(-2.40 to 23.95)
	Highway South	330	25.20	(6.60 to 80.40)	330	3.67	(-9.60 to 13.20)
	Background (mean)	334	21.52	(9.35 to 82.45)			
PM_1 ($\mu g/m^3$)	Highway West	332	14.78	(4.50 to 61.40)	322	3.72	(-11.80 to 12.10)
	Highway South	320	13.31	(4.60 to 58.40)	310	2.28	(-16.20 to 9.90)
	Background (mean)	324	11.03	(2.80 to 54.90)			
Black Smoke ($\mu g/m^3$)	Highway West	288	23.83	(0.43 to 104.06)	287	17.36	(-8.82 to 76.77)
	Highway South	330	20.12	(0.33 to 93.24)	329	13.66	(-1.82 to 54.75)
	Background (mean)	333	6.49	(0.43 to 41.49)			
NO_x (ppb)	Highway West	328	90.00	(8.80 to 334.40)	328	63.96	(2.80 to 157.00)
	Highway South	302	68.65	(8.00 to 322.40)	302	42.36	(-1.80 to 132.00)
	Background (mean)	334	26.10	(5.60 to 202.80)			

[a] August data were excluded
[b] Intervention: maximum driving speed reduced from 100 to 80 kph
[c] Traffic contribution: concentration at roadside minus daily mean background

As the air quality monitoring stations are located next to the ring highway, wind direction may affect the measured concentrations in addition to the meteorological conditions corrected for using 'traffic contribution'

concentrations. When the monitoring station is located downwind from the road, traffic emission is directed towards the monitoring station. When the wind is coming from the opposite direction, the opposite might occur. Wind direction data were not available for the exact monitoring locations.

Instead, daily wind direction data from measuring site Schiphol (Amsterdam Airport) were obtained from the Royal Netherlands Meteorological Institute. If the daily mean predominant wind direction was within 180° from parallel to the road (in both directions) to directly towards the monitoring station that day was considered downwind.

Analysis

In November 2005 the maximum speed for the western part of the Amsterdam ring highway was limited from 100 to 80 kph. In this study, daily mean concentrations in the year after the intervention were compared to daily mean concentrations in the year before. Due to holidays and maintenance works air pollution and traffic are generally untypical in August, and therefore August data were excluded from all analyses. All statistical analysis was done using SAS 9.1.3 (SAS Institute Inc., Cary, NC, USA).

The analysis consists of three phases:

- First, the effect of the intervention on the roadside concentrations was studied using linear regression.
- Secondly, the effect on 'traffic contribution' was studied using linear regression.
- Finally, the influence of traffic flow (T), traffic congestion (AVH) and wind direction (D) was taken into account.

Linear multivariate regression analysis was performed for the 'traffic contribution' concentrations of all components of air pollution. The multivariate regression equation was

$$\textit{'traffic contribution' conc.} = \alpha + \beta_1 * T + \beta_2 * AVH + \beta_3 * D + \beta_4 * \textit{intervention}$$

in which 'intervention' was included as a yes/no variable and β_4 is the intervention effect estimate. This final, fully adjusted analysis was replicated for data from the southern section of the highway, where no change in speed limit was implemented. This way, explanations for changes in air pollution other than caused by the speed limit intervention could be detected.

RESULTS

Table 1 summarizes the measured concentrations of the different components of air pollution in the year prior to the intervention. Concentrations at A10W (intervention highway section), A10S (non-intervention highway section) and mean background concentration are shown. Also the traffic contribution concentrations (daily roadside minus daily mean background) are shown for both highway locations. The data show that despite lower traffic density than at A10S, roadside concentrations of all components were highest at the intervened road section (A10W). Table 2 shows the measured concentrations for the year after the intervention took place.

Table 2. Roadside and Background concentrations[a] of air pollutants in Amsterdam, one year post-intervention[b] (November 2005 to November 2006)

		Roadside			Traffic contribution[c]		
		N	Mean	Range (min-max)	N	Mean	Range (min-max)
PM_{10} ($\mu g/m^3$)	Highway West	327	27.55	(11.60 to 59.20)	327	5.75	(-6.00 to 24.30)
	Highway South	316	24.21	(9.20 to 54.30)	316	2.63	(-25.55 to 13.60)
	Background[d]	334	21.73	(8.35 to 53.45)			
PM_1 ($\mu g/m^3$)	Highway West	320	14.23	(4.30 to 39.90)	313	3.14	(-1.70 to 14.40)
	Highway South	232	15.23	(4.70 to 57.00)	228	4.22	(-2.00 to 34.80)
	Background[d]	327	11.12	(3.70 to 38.70)			
Black Smoke ($\mu g/m^3$)	Highway West	312	19.41	(0.89 to 92.51)	311	13.46	(-13.04 to 75.10)
	Highway South	316	15.82	(0.63 to 53.93)	315	9.99	(-6.22 to 34.80)
	Background[d]	332	5.85	(0.32 to 30.50)			
NO_x (ppb)	Highway West	328	83.99	(8.80 to 218.40)	328	59.70	(-2.40 to 162.80)
	Highway South	314	61.60	(4.80 to 179.20)	314	37.09	(-8.00 to 103.60)
	Background[d]	334	24.13	(5.60 to 100.00)			

[a] August data were excluded
[b] Intervention: maximum driving speed reduced from 100 to 80 kph
[c] Traffic contribution: concentration at roadside minus daily mean background
[d] mean

The regression analysis of the effect of the intervention (Table 3) showed that roadside concentrations of PM_{10} and BS decreased statistically significantly at A10W. The traffic contribution to PM_{10}, PM_1 as well as BS was also found to be significantly reduced after the intervention.

Adjustment for daily traffic flow, congestion (AVH) and wind direction made no difference to this finding. PM_{10}-concentrations were estimated to decrease by 2.20 $\mu g/m^3$ since the speed limit reduction, PM_1-concentrations were reduced by 0.42 $\mu g/m^3$. A reduction of 3.57 $\mu g/m^3$ BS was achieved. The

Table 3. Speed limit intervention effects on concentration of PM_{10} ($\mu g/m^3$), PM_1 ($\mu g/m^3$), Black Smoke ($\mu g/m^3$) and NO_x (ppb) measured at roadside

	A10W: with intervention[a]					
	Roadside		Crude Traffic Contribution[b]		Adjusted[c] Traffic Contribution[b]	
	Change	(95%-CI)	Change	(95%-CI)	Change	(95%-CI)
PM_{10}	-2.30*	(-4.00 to -0.59)	-2.34*	(-3.13 to -1.55)	-2.20*	(-2.98 to -1.43)
PM_1	-0.47	(-1.56 to 0.62)	-0.54*	(-0.97 to -0.12)	-0.42*	(-0.82 to -0.01)
BS	-4.15*	(-6.78 to -1.52)	-3.72*	(-5.89 to -1.54)	-3.57*	(-5.65 to -1.50)
NO_x	-5.46	(-13.36 to 2.45)	-3.25	(-9.06 to 2.56)	-2.13	(-7.25 to 3.00)

	A10S: without intervention[a]					
	Roadside		Crude Traffic Contribution[b]		Adjusted[c] Traffic Contribution[b]	
	Change	(95%-CI)	Change	(95%-CI)	Change	(95%-CI)
PM_{10}	-1.36	(-2.88 to 0.17)	-0.63	(-1.41 to 0.16)	-0.97*	(-1.68 to -0.25)
PM_1	1.93*	(0.65 to 3.22)	2.19*	(1.56 to 2.82)	2.24*	(1.60 to 2.88)
BS	-1.99	(-4.12 to 0.14)	-2.18*	(-3.74 to -0.62)	-2.43*	(-3.80 to -1.05)
NO_x	-5.63	(-12.19 to 0.94)	-0.45	(-5.25 to 4.35)	-1.87	(-5.68 to 1.94)

* $p<0.05$
[a] Intervention: maximum driving speed reduced from 100 to 80 kph
[b] Traffic contribution: concentration at roadside minus daily mean background
[c] Adjusted for daily traffic flow, congestion (AVH) and wind direction

high variability of daily NO_x-concentrations is reflected in the wide 95%-confidence interval of the estimated intervention effect. No statistically significant effect was observed. The estimated reductions mount up to 27%, 11% and 21%, respectively, of the traffic contributions to PM_{10}, PM_1 and BS shown in Table 1. Table 4 shows the speed limitation effect relative to the roadside concentrations in the year before the intervention (Table 1).

Table 4. Speed limit intervention effects, relative to the ambient concentrations in the year before the intervention

	A10W			A10S		
	Intervention effect[a]	Relative to Roadside concentration[b]	(95%-CI)	Intervention effect[a]	Relative to Roadside concentration[b]	(95%-CI)
PM_{10}	-2.20* $\mu g/m^3$	-7.4%	(-10.0 to -4.8%)	-0.97* $\mu g/m^3$	-3.8%	(-6.7 to -1.0%)
PM_1	-0.42* $\mu g/m^3$	-2.8%	(-5.5 to -0.1%)	2.24* $\mu g/m^3$	16.8%	(12.0 to 21.7%)
BS	-3.57* $\mu g/m^3$	-15.0%	(-23.7 to -6.3%)	-2.43* $\mu g/m^3$	-10.2%	(-16.0 to -4.4%)
NO_x	-2.13 ppb	-2.4%	(-8.1 to 3.3%)	-1.87 ppb	-2.7%	(-8.3 to 2.8%)

* $p<0.05$
[a] Traffic flow, congestion and downwind exposure adjusted 'traffic contribution' speed limit intervention effect
[b] Roadside concentration in the year before the intervention

Table 5 shows the relation between air pollution concentrations at the two monitoring stations and traffic flow, congestion (AVH) and downwind exposure in the year before the intervention was implemented. The monitoring site being downwind from the freeway was significantly related to all air pollution components at A10S. At A10W this was only so for PM_1. Traffic flow was significantly related to almost all components at both stations, congestion was only related to some components.

Table 5. Effects of traffic flow, congestion and wind direction on traffic contribution[a] to air pollution concentrations measured at roadside

	A10W					
	Traffic Flow (per 1000 vehicles)		Traffic Congestion (AVH)		Downwind	
	Change	95%-CI	Change	95%-CI	Change	95%-CI
PM_{10}	1.09*	(0.61 to 1.56)	0.01	(-0.18 to 0.20)	0.07	(-1.13 to 1.26)
PM_1	0.78*	(0.53 to 1.03)	0.05	(-0.05 to 0.15)	-0.72*	(-1.34 to -0.09)
BS	2.78*	(1.96 to 3.60)	0.46	(-0.07 to 0.99)	1.11	(-2.18 to 4.40)
NO_x	10.78*	(7.58 to 13.98)	0.07	(-1.22 to 1.36)	-3.67	(-11.70 to 4.36)

	A10S					
	Traffic Flow (per 1000 vehicles)		Traffic Congestion (AVH)		Downwind	
	Change	95%-CI	Change	95%-CI	Change	95%-CI
PM_{10}	0.13	(-0.07 to 0.34)	0.08	(-0.03 to 0.19)	3.85*	(3.12 to 4.59)
PM_1	0.36*	(0.22 to 0.51)	0.07	(-0.01 to 0.15)	1.53*	(1.01 to 2.05)
BS	1.06*	(0.49 to 1.63)	0.32*	(0.01 to 0.63)	7.21*	(5.19 to 9.24)
NO_x	2.86*	(1.36 to 4.35)	0.85*	(0.04 to 1.66)	26.21*	(20.90 to 31.53)

* $p<0.05$

[b] Traffic contribution: concentration at roadside minus daily mean background

Figure 3 shows the estimated adjusted difference in traffic contribution between the year before and the year after the intervention for both monitoring sites. As the figure shows, there were reductions for PM_{10} and BS but not for PM_1 at the 'control' highway site as well. At both highway sites no statistically significant change in NO_x was seen. For PM_{10} and PM_1 the difference in estimated effect between the intervened and non-intervened road sections was statistically significant. The crude and adjusted effect estimates at the 'control' site (A10S) are shown in Table 3.

DISCUSSION

In this study, we have shown that particulate air pollution (PM_{10}, PM_1 and BS) at roadside has decreased since the speed limit reduction on a section of the Amsterdam ring highway. No significant effect on nitrogen oxides was observed. Although reductions were also observed at a section of the same ring highway without intervention, reductions in PM_{10} and PM_1 at the intervention site were significantly larger. The reductions on the non-intervened highway section might be explained by the governmental stimulation of reduced emission vehicles.

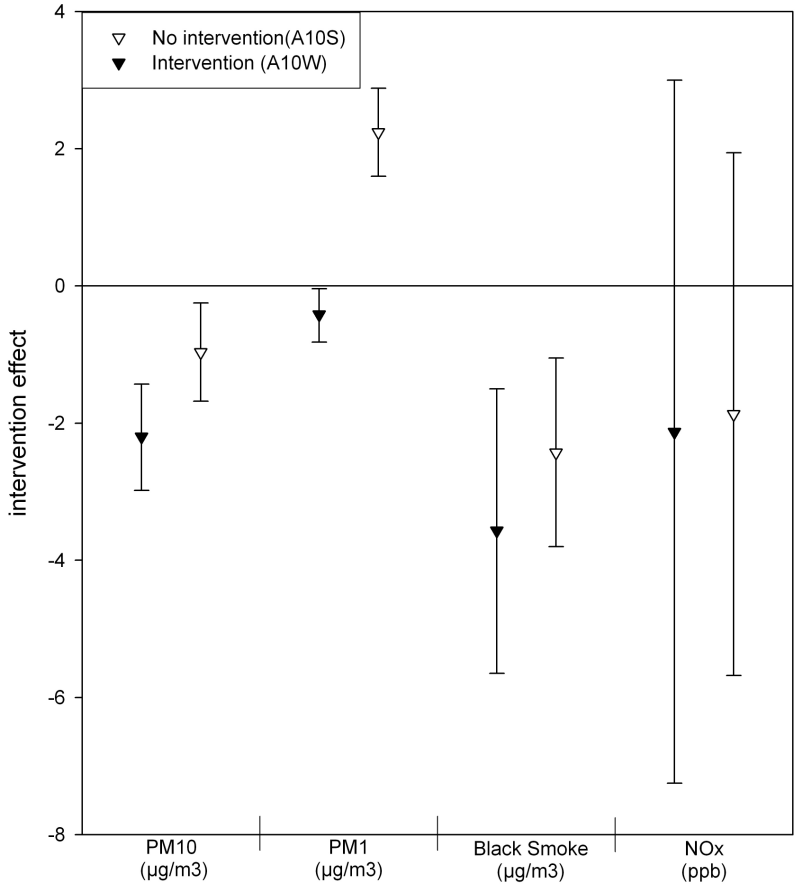


Figure 3. Speed limit intervention effect on traffic contribution measured at roadside at highway sections with and without intervention. Adjusted for traffic flow, congestion and wind direction.

Daily air pollution concentrations are not only determined by traffic, also other local sources and factors such as long range air pollution and meteorology are of influence. The correlations between the background monitoring stations reflect that these processes affect the concentrations over the whole city in a similar way. To correct for these factors, the traffic contribution concentrations were studied. Nevertheless, these processes may potentially influence the transport and dilution of pollution caused by local sources such as traffic as well, leading to both increasing and decreasing local

concentrations. In this study no further adjustment was possible as sufficiently detailed information on these processes was not available.

Apart from the difference in speed limit, the two highway sections are not exactly the same in some more features as well. While the western section has adjoining apartment buildings, the southern section is located in a relatively open area next to a river. Also, the embankment elevation of the two sections is different, 4.8 m at the western section, 7.6 m at the southern. These spatial differences are a probable explanation for the higher and more significant effect estimates for downwind exposure (Table 4) at the southern section. The negative association with PM_1 at A10W may be explained by the adjoining buildings.[137] Also, the wind direction data were obtained from Schiphol Amsterdam Airport, as no data from the monitoring sites were available. The monitoring sites are located 8 km northeast (A10S) to10 km north (A10W) of the airport. Possibly, local appearing wind directions might differ slightly. However, no change in intervention effect estimates was shown when the downwind exposure variable was excluded from the adjusted model. At the same time, the influence of traffic density on air pollution was much larger on the more enclosed A10W than at the A10S location, leading to higher pollution concentrations at the A10W site despite lower traffic densities.

Since the intervention, traffic flow on the intervened highway was somewhat decreased (intervention effect: -1823 vehicles per 24 h, 95%-CI: -4226 to 581), similar figures appear at the highway section without intervention (-1981 vehicles per 24 h). These changes are small, amounting to no more than 2% of total traffic flow. Congestion was higher at the intervened highway section than at the not intervened section. Since the intervention, daily traffic congestion at the western road section has not changed (intervention effect: 0.12 AVH, 95%-CI: -0.53 to 0.77), at the 'control' highway section, an increase was observed (1.13 AVH, 95%-CI: 0.47–1.80). The previously expressed concern of the speed limit intervention causing additional congestion,[134] showed not to be valid at this highway.

About two weeks before the intervention was implemented, a noise-barrier was installed along the western highway section. The screens were installed in the open spaces between the already present high-rise buildings (Figure 2). Noise screens are known to change the air flow at a road, thereby increasing the concentrations at the road itself, and lowering concentrations in the adjoining neighbourhoods.[138] In this study, the air quality monitoring station was situated between the road and the façade of the building (see Figure 2). Installation of the noise screen therefore could have caused an underestimation of the effect of the intervention on air quality.

Improving air quality by speed limit reduction has been predicted,[10,139] but has not been demonstrated by real life air quality measurements before. Based on dispersion models, the Dutch National Department of Transport predicted that PM_{10}-concentrations at roadside in this specific situation would

be reduced by 0.5–1%.[135] The observed reduction of 2.20 μg/m^3 is 7% of the mean concentration measured at roadside.

In addition, the monitoring network provided information on PM_1 and BS. Both fine particles (PM_1) and especially soot (BS)[9,117,140] are known to be directly related to traffic combustion and considered to be of health importance. In a country like the Netherlands, with a very dense population and road network, the observed reductions at roadside of 3 and 15% for PM_1 and BS respectively, are therefore of potential importance to health. The larger intervention effect on BS than on PM_{10} however, might be artificial. BS monitors are known to produce levels which are not real concentrations.[141] Relative to the traffic contribution concentration in the year prior to the intervention, the reduction in PM_{10} is larger than that in BS (26.8 and 20.6%, respectively).

Also, the relative PM_1 reduction (12.7%) is smaller than PM_{10}. A probable reason for the relatively large effect on the reduction of PM_{10} could lie in the fact that the traffic contribution concentration studied, is not only consisting of exhaust emission, but also of resuspended particulate matter. Along a busy street in London, about 20% of the traffic contribution concentration of PM10 is due to resuspension of particles.[136] In Berlin[142] this was about 50%. Traffic driving speed was one of the influential factors of resuspension, less resuspension occurs at lower driving speeds. Resuspension was dominated by the coarse fraction of PM_{10} (2.5 to 10 μm), the finer fractions of particulate matter are less influenced by the resuspension of road dust. In contrast to particulate matter, no clear effects on nitrogen oxides were observed but confidence intervals were wide, owing to the high day-to-day variation of the measured NO_x-concentrations.

In conclusion, this study demonstrated a significant reduction of PM_{10} and PM_1 as a result of reducing the speed limit at an urban ring highway.

Chapter 6

The Effectiveness of Different Ventilation and Filtration Systems in Reducing Air Pollution Infiltrating a Classroom near a Freeway

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ABSTRACT

Traffic related air pollution is associated with adverse respiratory health effects in children. We investigated whether fine particle filters in ventilation systems can improve indoor air quality at a school near a freeway in the Netherlands.

In an occupied classroom of a school located 130m from a freeway, three systems were tested: A) displacement ventilation with F7-filter, B) balanced ventilation and F7-filter, C) balanced displacement ventilation and F9-filter. Air quality ($PM_{2.5}$, soot, particle number concentration (PNC)) was measured indoors and outdoors simultaneously. The fraction of outdoor pollution infiltrating into the classroom (infiltration coefficient) was determined by linear regression.

During natural ventilation, the infiltration coefficients of $PM_{2.5}$, soot and PNC and were 0.44, 0.77 and 0.61, respectively. For PNC the infiltration coefficient was statistically significantly reduced by systems A (0.31) and C (0.30), and for $PM_{2.5}$ by system C (0.31), while no significant reduction was found for soot.

We conclude that mechanical ventilation systems equipped with fine particle filters may reduce infiltration of outdoor air pollution into classrooms. Overall, indoor air quality improved less than expected from filtration. Further work is needed to establish health benefits of retrofitting existing schools near busy roads with mechanical ventilation and filtration systems.

INTRODUCTION

Traffic related air pollution is associated with respiratory health effects in children residing or attending school near major roads.[56,117,126,143,144] Air pollution exposure at school, penetration of outdoor air pollution into the indoor environment, and related adverse health effects have recently received attention, scientifically as well as from a policy perspective.[145-148] Although, for instance in the Netherlands and California, USA, guidelines exist regarding school planning in urban high traffic areas,[149] civilians and local government seek additional protection measures, such as air filtration.[149-151] In north-western Europe, mechanical ventilation systems with forced air inlets are not very common in schools. However, to reduce energy consumption, ventilation systems equipped with heat recovery techniques are increasingly often retrofitted in school buildings.

Some form of air filtration is usually present in mechanical ventilation systems, to prevent clogging. Mechanical ventilation systems using filters with the ability to capture fine particulate matter are less common. Although the market in fine particle air filtration equipment has been expanding over the past few years, little is known about the practical effectiveness of these filters. Morawska et. al[152] reported on the redesign of a heat, ventilation and air conditioning system in a radio studio surrounded by busy roads. The particle number penetration rate was reduced from 42 to 14%. For $PM_{2.5}$, the penetration rate after the upgrade was approximately 18% and no baseline measurements were available. Parker et. al.[153] studied the particle size distribution and composition in a school which was applied with fine particle filters and found substantial protection against exposure to submicron aerosol (indoor-outdoor (I/O) ratio: 0.13).

In the present study, three mechanical ventilation systems with fine particle air filtration were tested in an occupied classroom of a primary school located at 130m from one of the busiest freeways in the Netherlands. The primary aim of this study was to get insight in the capability of filters to prevent traffic related air pollution from entering the classroom. The secondary aim was to study the effect of the ventilation systems on measured and perceived indoor climate.

MATERIALS AND METHODS

During the cold season of 2008/2009, three different mechanical ventilation systems equipped with fine particle air filters were tested successively. Each system was tested during four weeks, excluding school holidays. Four weeks before and two weeks after the tests, the natural ventilation situation was monitored. Each of the systems was installed during weekends or school holidays.

The study was done in one classroom at the ground floor of a primary school in Amsterdam, the Netherlands. The school was constructed in 1952 and was ventilated naturally. During the tests, the classroom was in use by a class of 22 pupils, on average nine years old. The distance between classroom and the Amsterdam ring freeway (about 92,000 vehicles per day) is 130m. A quiet residential street, which is also the access to the school, and an eight-storey apartment building are located between the school and the freeway.

Ventilation systems and fine particle filters tested

The three ventilation systems tested were selected to be representative from a larger inventory of what was commercially available at the time the study was done. All ventilation systems are indoor CO_2-concentration driven; when a sensor positioned at 1.5m above the floor, next to the blackboard in the front of the classroom, detected CO_2-concentrations of 800 ppm or higher, the mechanical ventilation switched on. The teacher was instructed not to open doors or windows for natural ventilation during the system tests.

The systems are schematically summarized in Figure 1, additional information and pictures are given in the Supplemental Material (Annex A). First, we tested a displacement ventilation system (system A), which has mechanical air inlet only (Airswitch, Bergschenhoek B.V., the Netherlands). The surplus of air is expected to leave the building naturally. Outdoor air was filtered by a F7 fine particle filter (classification according to EN 779, similar to ASHRAE class MERV-13). The air handling- and filter-unit were placed in the corridor next to the classroom. Fresh air is forced into the classroom through a textile duct hanging from the ceiling. The second system (system B) was a plug-and-play balanced ventilation system with heat recovery, in which a F7 (MERV-13) particle filter is applied (Monoline, Nedair B.V., the Netherlands). In this system, all mechanics such as air handling, filter-unit and heat recovery installation are built in a large free standing cabinet, which is to be placed in the classroom. Air in- and outlet ducts as well as electricity are then connected to the cabinet. Fresh air enters the classroom through valves at the upper side of the chest, used air leaves the classroom through the bottom of the cabinet, thereby generating airflow from the ceiling to the floor of the classroom. The third system (system C) was a balanced displacement ventilation system with heat recovery, which was fully placed outside of the

classroom (OCTO, Unifan, the Netherlands). In this system a F9 (MERV-16) particle filter is used. Air enters the classroom through porous ducts placed vertically at 0.2-1.5m from the floor. Used air leaves through vents placed at approximately 2.8m above the floor, thereby generating airflow from the floor to the ceiling.

Air quality

Indoor and outdoor air quality were measured continuously during the full testing period. All equipment was placed at the middle of a sidewall of the classroom, at 1.5m above the floor and 0.5-1.0m from the wall to measure air as breathed by the children. Outdoor measurements were done in the courtyard at the opposite side of the same sidewall.

	A	B	C
Inlet	Mechanical inlet	Mechanical inlet	Mechanical inlet
Fine Particle Filter	F7	F7	F9
In-class distribution	Textile distribution tube at ceiling of classroom	Nozzles at cabinet (>150cm)	Porous distribution tubes front classroom (<150cm)
Outlet	Overpressure - Natural outlet	Mechanical outlet	Overpressure - Mechanical outlet
		Porous frame at bottom of cabinet (<20cm)	Valves at front of classroom (>200cm)
		Balanced ventilation, Heat recovery	Balanced ventilation, Heat recovery

Figure 1. Schematic summary of the three ventilation systems tested.

Indoor and outdoor $PM_{2.5}$-concentrations were monitored according to the criteria of NEN-EN 14907, using low volume samplers (Klein Filter Gerät and PNS16, Derenda, Germany) and quartz filters (QMA, Whatman International, Maidstone, UK). The soot content of $PM_{2.5}$ was evaluated using both absorbance and elemental carbon content analysis. Absorbance was calculated from reflectance of the $PM_{2.5}$ filters as described elsewhere[154], using an EEL model 43 reflectometer (Diffusion Systems Ltd., London, UK). Elemental carbon (EC) content of the $PM_{2.5}$ filters was determined by thermal optical gas analysis, according to the NIOSH Manual.[155] Particle number concentrations (PNC) of particles in the ultrafine size-range of 0.01 to >1µm were measured

using handheld CPC 3007 equipment (TSI Ind, Shoreview, MN, USA). Measurements were done for approximately 5 hours (as the condensation liquid vaporizes fully in ca. 6 hours) every second school day during lessons. The equipment recorded PNC at a 1-minute interval. Previous quality assurance showed that readings of different CPC 3007 units differ slightly and may change over time.[156] A correction factor applied to the PNC readings was derived from parallel measurements at every fourth measurement day (running the units in parallel for the full day, indoors and outdoors alternately). Four measurement-days (1 day of system A and 3 days of system B) were deleted from further analyses due to technical failure of one of the units.

Table 1. Number of students, indoor and outdoor air pollution concentration, CO_2-concentration, temperature and perceived air quality in an occupied classroom in direct vicinity of a major freeway, during natural ventilation and the testing of three ventilation systems with fine particle filter. Number of observations (N), Mean and Standard Deviation (SD).

		Natural Ventilation		System A		System B		System C	
		N	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)
Number of students in the classroom		32	21.9 (0.3)	20	21.3 (1.0)	20	21.8 (0.5)	17	21.6 (0.7)
PM2.5[1]	Indoor	30	16.1 (7.2)	20	15.6 (6.2)	20	12.6 (4.2)	16	14.7 (4.8)
($\mu g/m^3$)	Outdoor		25.2 (14.8)		30.1 (13.4)		16.7 (7.2)		23.7 (14.3)
	I/O Ratio[2]		0.8 (0.3)		0.6* (0.2)		0.8 (0.3)		0.7 (0.3)
Absorbance[1]	Indoor	30	1.4 (0.3)	20	1.3 (0.4)	20	0.8 (0.3)	16	0.8 (0.3)
($10^5/m$)	Outdoor		1.7 (0.4)		1.6 (0.4)		1.1 (0.3)		1.2 (0.3)
	I/O Ratio[2]		0.9 (0.1)		0.8* (0.0)		0.7* (0.1)		0.7* (0.1)
PNC[3]	Indoor	33	24.1 (17.6)	25	10.2 (5.6)	15	17.3 (5.6)	28	13.0 (6.4)
($\#\cdot 10^3/cm^3$)	Outdoor		36.4 (26.9)		28.5 (13.3)		30.1 (8.2)		31.9 (15.5)
	I/O Ratio[2]		0.7 (0.2)		0.4* (0.2)		0.6 (0.2)		0.4* (0.1)
CO_2[4] (ppm)		31	929 (202)	20	811 (102)	18	737 (120)	17	770 (78)
Temperature[4] ($^\circ C$)		31	20.9 (1.0)	20	21.6 (2.6)	18	19.8 (0.8)	17	18.8 (2.0)
Perception students[5]		21	4.2 (0.5)	20	3.8 (0.7)	20	4.4 (0.3)	17	4.5 (0.1)
Perception teacher[6]		26	3.1 (0.5)	18	3.0 (0.0)	18	3.9 (0.3)	16	4.9 (0.3)

*: I/O ratio statistically significantly different from natural ventilation
1: 24-hr mean (N=number of days)
2: Indoor/Outdoor ratio
3: 1-hr mean (N=number of hours, measured at 8 (natural ventilation), 5 (system A), 3 (system B) and 6 (system C) days)
4: schoolday mean (N=number of days)
5: air quality; scored 1 (low quality) to 5 (high quality), 3 times per day (Mean score per school day. N= number of days)
6: air quality; scored 1 (low quality) to 5 (high quality), daily by all students (Mean score per school day, N= number of days)

For $PM_{2.5}$ and soot 24hr mean concentrations were available; for PNC hourly mean concentrations were calculated when data was available for at least 40 minutes of an hour. All school holidays and weekends were omitted from the analysis. Also, days with reported unusual in-classroom activities

which could affect air quality, such as a candle-lit Christmas celebration, were omitted.

Indoor climate

During the full study period indoor CO_2-concentration, temperature, perceived comfort and annoyance related to the ventilation system applied were monitored. Indoor CO_2-concentration, an indicator of ventilation rate for occupied rooms,[157] and temperature were measured every ten minutes (Q-track 8551, TSI Inc., Shoreview, MN, USA). The equipment was located at the same location as the air quality equipment. The teacher daily completed a questionnaire on draft, temperature, odour and ventilation related noise. In addition, a journal on number of pupils in the classroom, (additional) ventilation behaviour and in-classroom activities was kept by the teacher. The teacher was also interviewed about her general experience and opinion on each system tested. Teacher and students rated the perceived indoor air quality (scale of 1 to 5, in which 1 is low quality and 5 is high quality) three to four times per school day, depending on that days schedule. Instruction on the rating was given by the researchers.

Statistical analysis

The effectiveness of the fine particle filters was quantified by calculating infiltration coefficients for each component. Infiltration coefficients were defined as the slopes of regressions of indoor concentrations on outdoor concentrations derived from multiple linear regression models with system (none, A, B, C), outdoor concentration and system-outdoor concentration interaction terms. Mixed models with an autoregressive correlation structure (AR(1)) were used to account for the correlation between repeated hourly PNC measurements performed on the same measurement day. Regression analysis was used to compare all indoor climate and perception results during the system tests to the situation with natural ventilation. All statistical analyses were done using SAS version 9.2 (SAS Institute Inc., Carry, NC, USA).

RESULTS

Table 1 shows mean classroom occupation (number of students), indoor and outdoor concentrations and I/O ratios of $PM_{2.5}$, soot (absorbance) and PNC, and indoor CO_2-concentration, temperature and perceived indoor air quality as scored by teacher and students during natural ventilation and the three system tests. The outdoor concentrations observed during natural ventilation were capturing the range observed during the system tests (Table 1 and Figure 2).

Table 2. The infiltration coefficient and 95%-confidence interval of $PM_{2.5}$, absorbance and PNC during natural ventilation and the testing of the three ventilation systems with fine particle filter.

	Natural Ventilation	System A	System B	System C
$PM_{2.5}$	0.44 (0.37 to 0.52)	0.39 (0.29 to 0.50)	0.38 (0.19 to 0.57)	0.31* (0.20 to 0.42)
Absorbance	0.77 (0.69 to 0.86)	0.82 (0.72 to 0.92)	0.81 (0.67 to 0.96)	0.82 (0.67 to 0.98)
PNC	0.61 (0.55 to 0.68)	0.31* (0.15 to 0.47)	0.42 (0.13 to 0.70)	0.30* (0.19 to 0.42)

*: statistically significantly different from natural ventilation

The two soot indicators (absorbance and EC) showed very similar results (correlation >0.9, see Supplemental Material, Annex B) and therefore only results for absorbance are presented. The I/O ratios during the system tests were all lower than during natural ventilation (Table 1). Significantly decreased I/O ratios were observed for $PM_{2.5}$ (system A), soot (systems A, B and C) and PNC (systems A and C). The infiltration coefficients in Table 2 show that indoor air quality is importantly influenced by outdoor air quality, even after installation of ventilation systems with fine particle filters. The differences between I/O ratios and infiltration coefficients can be explained by the fact that the intercepts were different from zero (Figure 2). The infiltration of outdoor air pollution into the indoor environment was significantly decreased compared to natural ventilation for $PM_{2.5}$ with system C (infiltration coefficient 0.31 vs. 0.44) and for PNC with system A (0.31 vs. 0.61) and system C (0.30 vs. 0.61). In contrast to the I/O ratios, infiltration coefficients for soot were not statistically significantly different during the system tests in comparison with natural ventilation (see Table 2 for absorbance, Supplemental Material, Annex B for EC). The different findings for I/O ratios and infiltration coefficients for soot result from lower intercepts during all three system tests compared to natural ventilation (Figure 2).

In Table 3, the effect of the ventilation systems on indoor climate is shown. All systems tested reduced the mean CO_2-concentration, indicating increased ventilation, to a similar extent. With natural ventilation, 1200 ppm

(advised maximum CO_2-concentration for classrooms in the Netherlands [158]) was exceeded during 26% of time. With systems A, B and C 1200 ppm

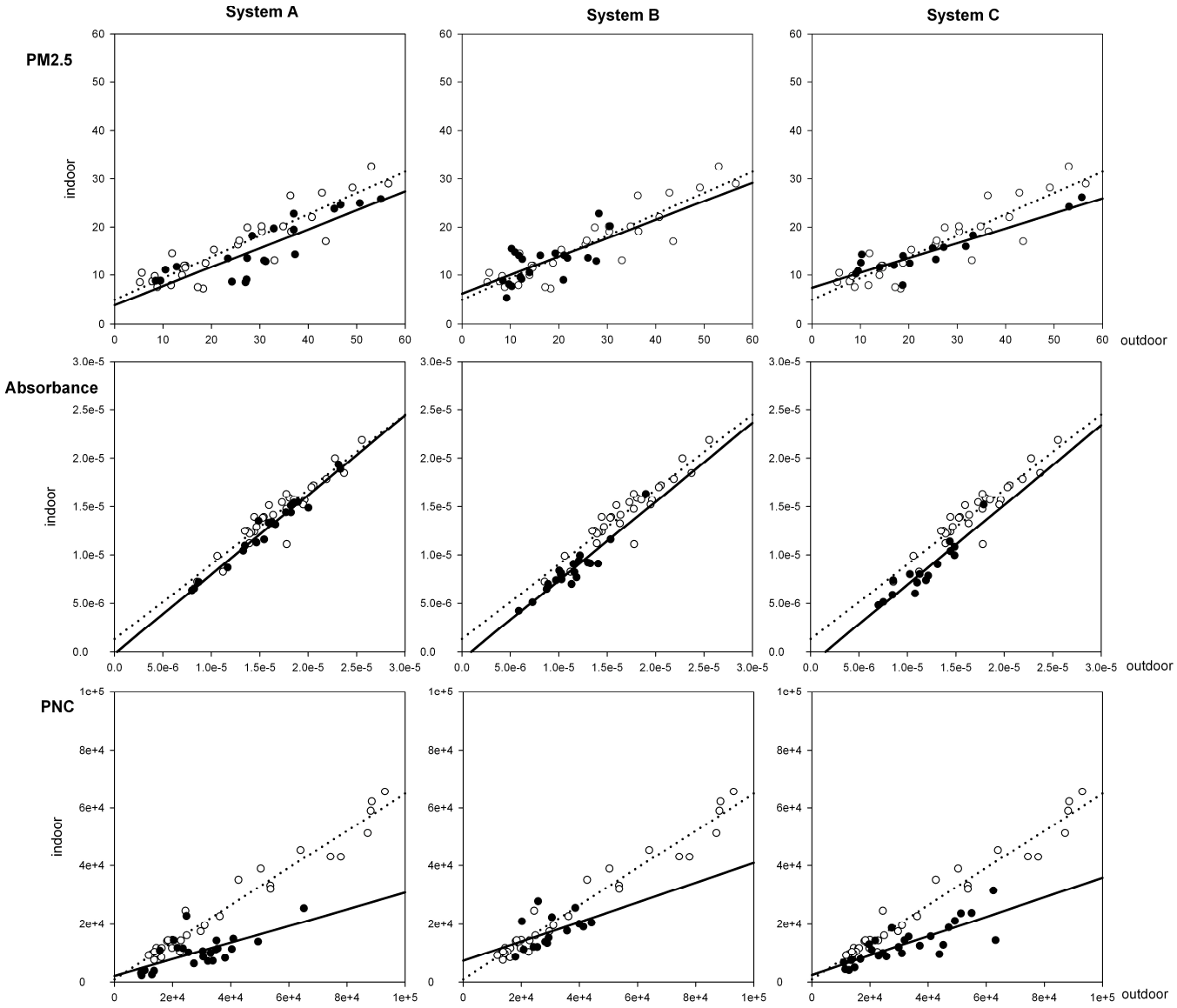


Figure 2. Indoor and outdoor concentrations of $PM_{2.5}$ (µg/m^3, 24-hr mean), absorbance (10^5/m, 24-hr mean) and PNC (number·10^3/cm^3, 1-hr mean) during natural ventilation (open dots, dotted regression line) and during the tests of the ventilation systems with fine particle filter (filled dots, solid regression line).

CO_2 was exceeded during 1, 6 and 2% of time, respectively. The mean indoor temperature decreased significantly during the tests of systems B and C (Table 3). The teacher indicated in the questionnaire that the indoor temperature was generally comfortable except for the period with system A (too cold 50% of the time and too hot 10% of the time). The teacher furthermore indicated that perceived indoor air quality increased during the testing of systems B and C (Table 3), students perceived a decrease in indoor air quality during the test of system A and an increase during the test of system C.

DISCUSSION

In this study we showed that installation of ventilation systems with fine particle filters in a classroom of a school located close to a main freeway, can result in improved air quality and indoor climate. Two systems statistically significantly reduced the infiltration of ultrafine particles (PNC) and one the infiltration of $PM_{2.5}$, while soot infiltration was not significantly reduced. Overall, indoor air quality improved less than expected from filtration. The balanced displacement system applied with F9 filter (system C) reduced $PM_{2.5}$ and PNC infiltration and was appreciated most by teacher and students.

Table 3. The average difference of the situation during the system tests and natural ventilation, and 95%-confidence interval, of the three ventilation systems on ventilation (CO_2-concentration), temperature and comfort relative to natural ventilation.

	System A	System B	System C
CO_2 (ppm)	-117* (-200 to -35)	-191* (-277 to -107)	-159* (-245 to -72)
Temperature (˚C)	0.7 (-0.3 to 1.6)	-1.0* (-2.0 to -0.1)	-2.1* (-3.1 to -1.1)
Perception teacher (score)[1]	-0.1 (-0.3 to 0.2)	0.8* (0.6 to 1.1)	1.9* (1.6 to 2.1)
Perception students (score)[1]	-0.4* (-0.7 to -0.1)	0.2 (-0.1 to 0.5)	0.3* (0.1 to 0.6)

*: statistically significantly different from natural ventilation
1: perceived air quality; scored 1 (low quality) to 5 (high quality)

Although there is a large body of literature on in-school climate and/or air quality,[153,159-166] we are not aware of any other study testing several mechanical ventilation systems in an occupied classroom. Moreover, our study was done in one classroom, and the only factors changing were ventilation system and weather conditions. In most other studies, circumstances differ, not allowing distinguishing effects of location and building from effects of method of ventilation.

As ventilation behaviour and perception may be influenced by season, we conducted the study in one (cold) season. To capture the natural within season variability of weather conditions, which can highly affect ambient air pollution concentrations,[167] each system was tested for four weeks. Measurement series in other studies were ranging between a few hours and two weeks.[159-165] Furthermore, the situation with natural ventilation was studied before as well as after the system tests to reduce seasonality effects. We were able to capture the typical range of outdoor concentrations for most components during most system tests (Figure 2): for PNC (system B) and soot (systems B and C), however, the majority of the measurements were at the lower end of the range of concentrations.

We presented infiltration coefficients in addition to I/O ratios as to account for the fact that indoor concentrations could be affected by particulate matter originating from other sources than traffic and/or resuspension e.g. by the children in the occupied classroom[166] and that intercepts could be positive rather than zero as assumed in I/O ratios. Intercepts were indeed positive for $PM_{2.5}$ and PNC for natural ventilation and during all system tests and for soot during natural ventilation (Figure 2). In contrast, we observed negative intercepts for soot for the three system tests, for which we do not have a conclusive explanation.

In this study we observed some unexpected results: system C was applied with a F9 filter, which is expected to have higher filtration efficiency for the smallest particles than F7 filters (systems A and B). For $PM_{2.5}$, we hence expected similar results for the different systems and a larger decrease of soot and PNC infiltration for system C. However, results indicated that system C was the only system decreasing infiltration of $PM_{2.5}$ and the decrease of PNC was similar for systems A and C. Furthermore, soot infiltration was not significantly affected by any of the systems and system B, which was applied with a F7 filter just like system A, did not significantly affect infiltration of any of the measured components. Taken together, these results suggest that during the test weeks, a major fraction of the air inside the classrooms had not passed through the filters but had entered the classroom through cracks or doors despite the positive pressure the systems A and C were supposed to have generated. This suggests that retrofitting existing schools and classrooms with mechanical ventilation and filtration systems should be accompanied with sealing efforts to ensure that outdoor air enters the classrooms primarily through the ventilation system.

Mechanical ventilation systems may introduce problems such as noise or draft and air pollution due to dusty or unhygienic ducts, filters and vents.[147,168,169] The data of our study show that during the four-week testing of three mechanical ventilation systems, such problems did not occur. In contrast with some previous studies,[161,170] teacher and students perceived a better indoor climate with the mechanical ventilation systems working. Perception, however, may have been biased as we were not able to conduct this study blindly (see pictures of the three systems in the Supplemental Material, Annex A). System technology and filter classification were nevertheless unknown by students and teacher. Moreover, perception and appreciation may possibly change over time when systems are used for longer periods. All three systems tested were designed to keep CO_2-concentrations in the classroom at approximately 800 ppm. Nevertheless, concentrations well above this value were observed regularly, showing that the systems on occasion were not able to supply sufficient outdoor air. During the testing of system B, the advised concentration of 1200ppm[158] was even exceeded for 6% of school hours.

From the three systems that were tested, system C improved indoor climate and air quality most. This study could be of value for the many urban areas in which spatial planning, especially for facilities aiming at vulnerable groups such as children, is complicated by air pollution problems.[149] However, further research is needed to confirm our findings in other classrooms, with other student populations and building characteristics, and at schools in other geographical settings. Furthermore, further research is necessary to find out if the observed improvement of indoor air quality is associated with reduction of the health risk experienced by children attending school at high traffic locations.

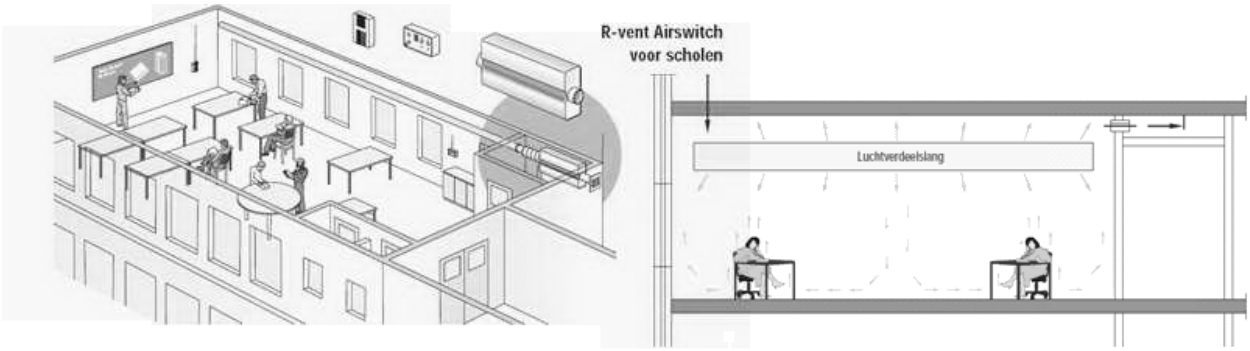
In conclusion, the ventilation systems tested provided limited improvements of indoor air pollution and climate. Further work is needed to establish health benefits of retrofitting existing schools with mechanical ventilation and filtration systems.

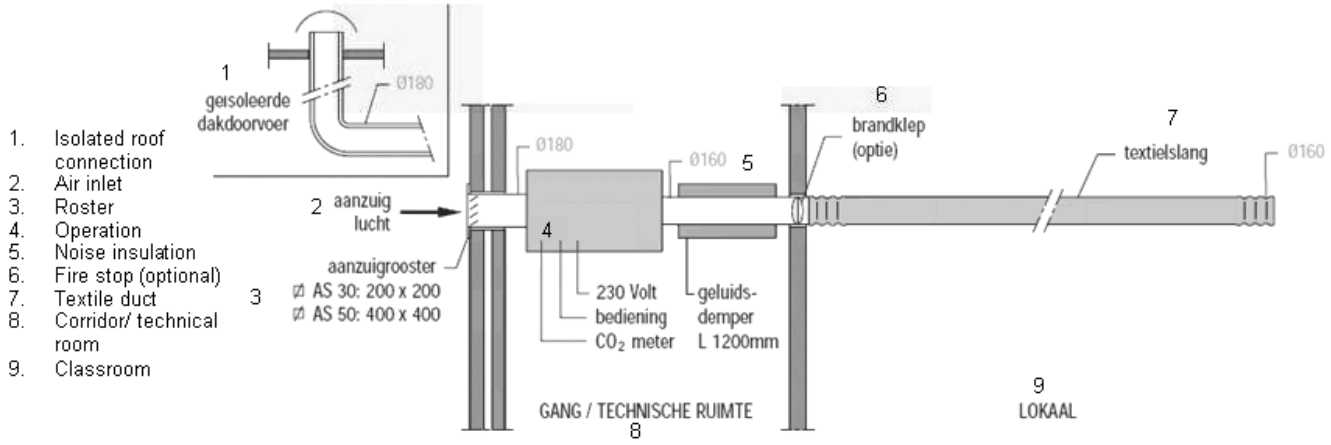
SUPPLEMENTAL MATERIAL

ANNEX A – Ventilation Systems Tested

System A

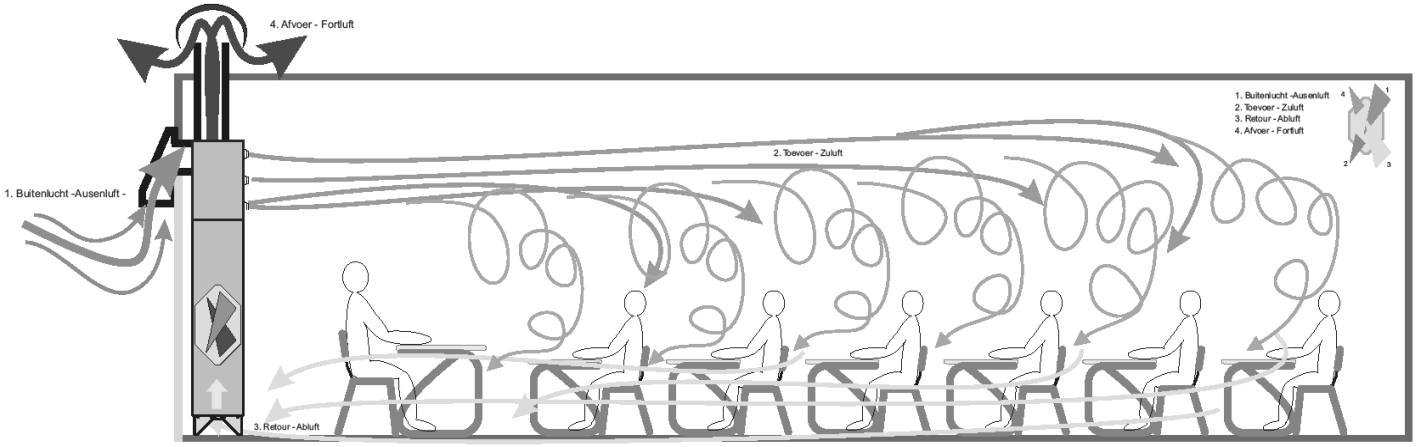
www.ihb.nl/site/downloads/Technische_documentatie_Rvent-Airswitch-062008.pdf (accessed; May 17th, 2011)





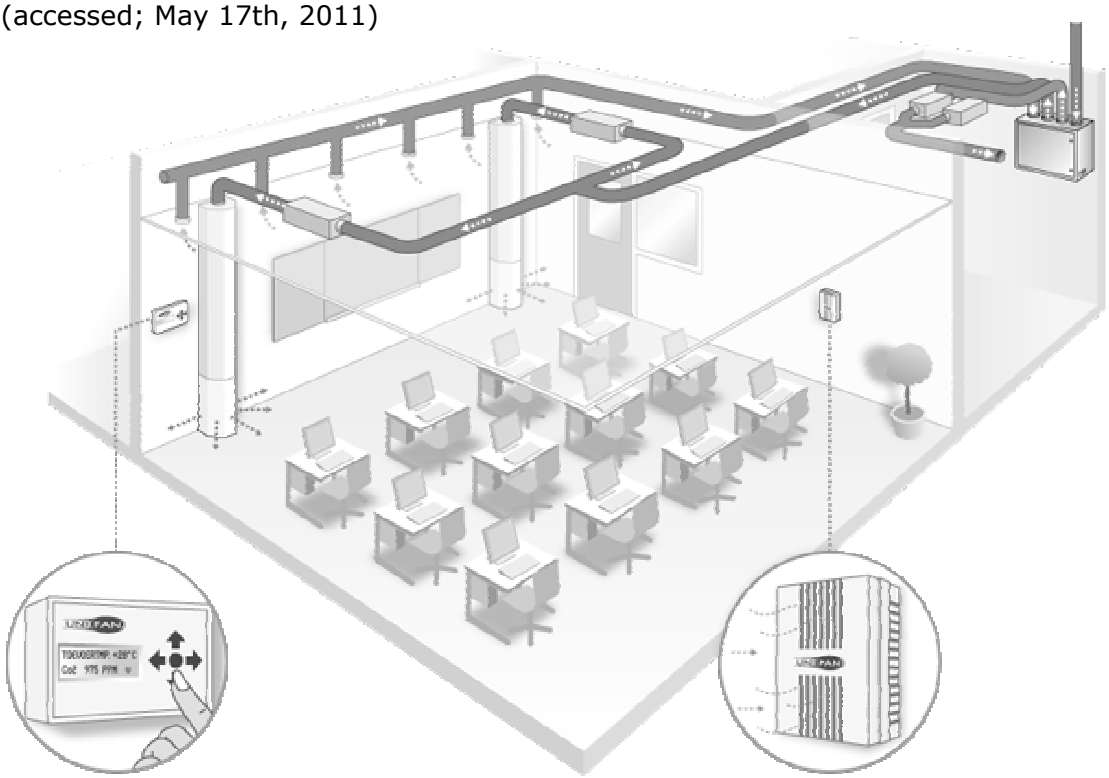
System B

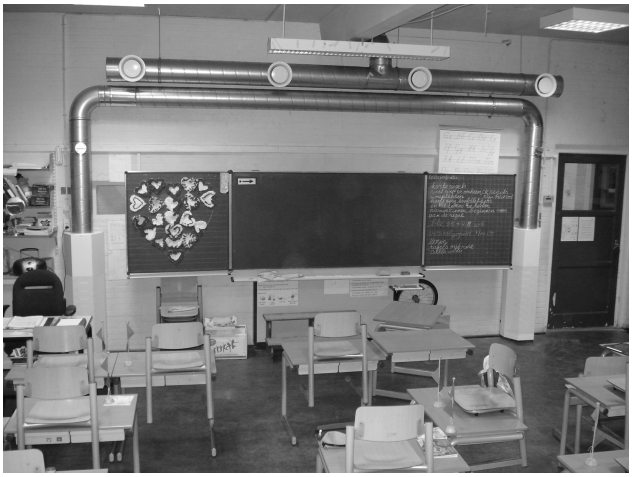
www.ned-air.nl/Documenten/Schoolventilatie/NL_MonoLine%20WTA%20HR%20600A_Versie%201.23.pdf (accessed; May 17th, 2011)



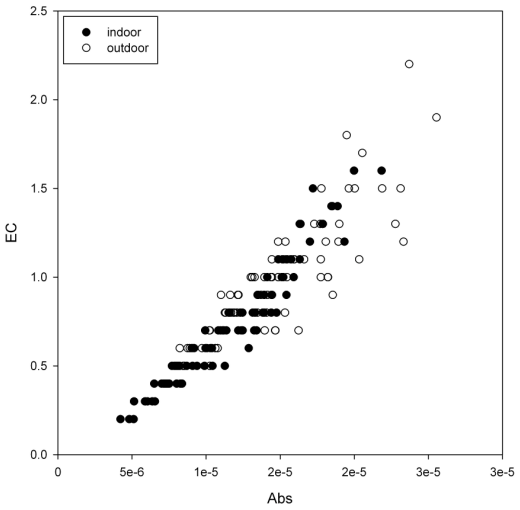
System C

http://www.unifan.eu/pdf/Technische%20specificaties%20OCTO%20producten.pdf (accessed; May 17th, 2011)





ANNEX B – Soot (Elemental Carbon)



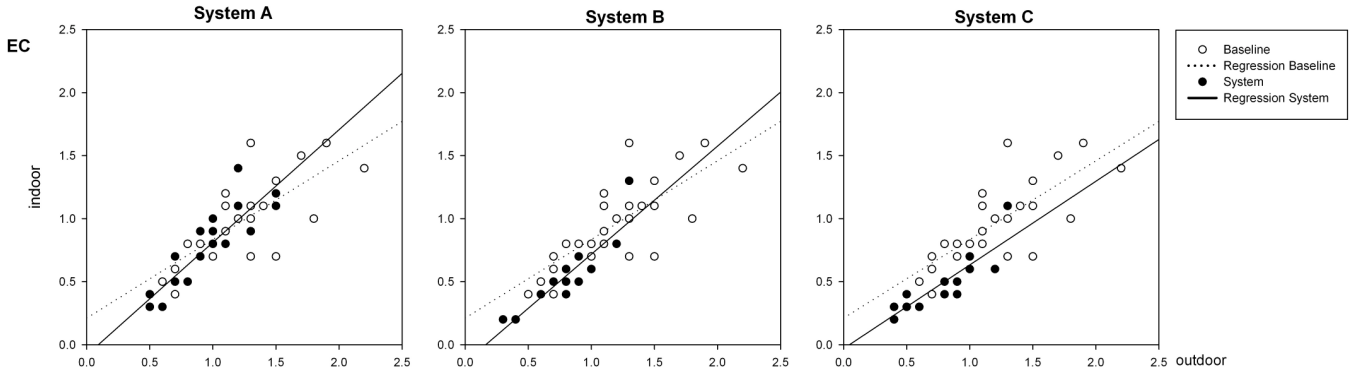
Supp.Mat Figure I: Scatter plot of indoor and outdoor measures soot indicators: Absorbance along the x-axis, Elemental Carbon along the y-axis. Correlation: indoor 0.96, outdoor 0.92.

Supp.Mat Table I: EC during natural ventilation and the testing of three ventilation systems with fine particle filter. Descriptives (N, mean, SD) and Infiltration Coefficient (with 95%-CI).

	Natural Ventilation		System A		System B		System C	
	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)	N	Mean (SD)
Indoor	30	16.1 (7.2)	20	15.6 (6.2)	20	21.3 (4.2)	16	14.7 (4.8)
Outdoor	30	25.2 (14.8)	20	30.1 (13.4)	20	16.7 (7.2)	16	23.7 (14.25)
I/O Ratio		0.8 (0.2)		0.8 (0.2)		0.7* (0.1)		0.6* (0.1)

*: I/O ratio statistically significantly different from natural ventilation

	Natural Ventilation	System A	System B	System C
Infiltration coefficient (95%-CI)	0.62 (0.48 to 0.77)	0.89 (0.64 to 1.14)	0.86 (0.56 to 1.16)	0.66 (0.38 to 0.95)



Supp.Mat Figure II: Daily mean indoor and outdoor concentration at baseline and during the tests of the ventilations systems with fine particle filter.

Chapter 7

General Discussion

The objective of the research presented in this thesis was to contribute scientific evidence on exposure to traffic related air pollution, its health effects and potential mitigation measures, relevant to Public Health Services in the Netherlands. The research presented in this thesis is the result of one of the projects of the Academic Collaborative Center for Environmental Health. The main aim of the Academic Collaborative Centers is to promote *evidence based public health*. The work presented in this thesis was largely performed at the department of Environmental Health of Public Health Service Amsterdam. The employees of the Public Health Service involved in the project were also involved in daily practice; e.g. assessing exposure and effects of traffic related air pollution, informing citizens and policy makers about this and promoting policies to reduce these effects. Experience and insights obtained in doing the research was therefore directly applied in daily practice, and vice versa.

In the Introduction (Chapter 1), a framework for evidence based public health, as proposed by Künzli and Perez[72] (Figure 1) was presented. In brief, exposure (phase A) may cause health problems in the population (phase B). The assessment of its relevance (phase C) may result in policy (phase D) to abate exposure (phase A) and improve public health (phases B and C). Ideally, practical expertise is integrated with the best available systematic research, and that decisions are made with the conscientious, explicit, and judicious use of the current best evidence: *evidence based public health*.

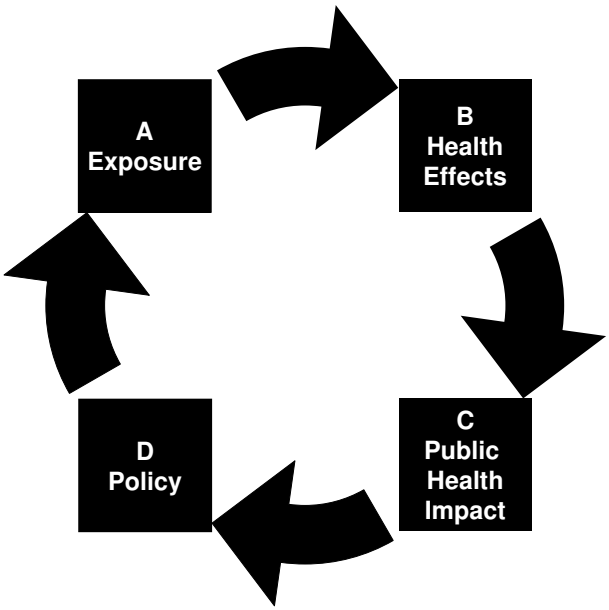


Figure 1. A framework for evidence based public health. Ideally, all steps are based on scientific evidence – evidence based public health. *Adapted from Künzli and Perez.*[72]

The framework of evidence based public health is used to discuss the work presented in this thesis. The research was done at Public Health Service Amsterdam, which resulted in crosspollination between science and practice in the field of air pollution. Several examples of this crosspollination are given, based on the experience obtained at the Public Health Service of Amsterdam.

EXPOSURE

The practice of environmental health starts with assessing exposure (Figure 1, phase A). At the Public Health Service of Amsterdam an air quality monitoring network is managed.[16] Comprehensive air quality measurements, however, can only be performed at a limited number of sites, providing exposure information for a limited part of the population. For population exposure assessment, models are therefore essential. Data from the monitoring sites do provide insight in source contribution and trends over time, which is impossible to get from models. Understanding of the contribution of specific sources is essential when seeking opportunities for possible abatement strategies. Knowledge on the trends over time are essential in evaluating the effectiveness of current policies.

In this thesis, two land use regression (LUR) models to estimate spatial variation of traffic related air pollution were developed and evaluated (Chapter 2). A LUR model can be used to estimate long-term air pollution concentration at a specific unmeasured location or address. In epidemiological studies, the estimated residential air pollution concentration is often used as a surrogate for long-term exposure to air pollution.[e.g.14,32] Two LUR models were developed, one for the West of the Netherlands (consisting of the Provinces of Noord-Holland and Flevoland and the Rijnmond area), and one for the city of Amsterdam (Chapter 2). The large area (West of the Netherlands) and city specific (Amsterdam) LUR models predicted NO_2-concentrations well (percentage of variability that was explained (R^2) of 87% and 72%, respectively).

As the LUR models were developed in order to estimate concentrations at unmeasured locations, insight in the predictive performance at independent measurement sites was desired. Concentrations estimated by the LUR models were therefore compared with independent NO_2-measurements obtained from measurement sites in Amsterdam. The percentage of variability that was explained was lower for the independent sites (large area model 48%, city specific model 57%) than for the measurement sites on which the LUR models were based. This out-of-sample-validation (applying a model to independent measurement sites) showed a much lower explained variance than internal leave-one-out cross-validation. Few other studies have done out-of-sample validations of LUR models, showing worse.[82] and somewhat better[83] performance than in leave-one-out cross-validation. In our study, the worse performance of the LUR models at independent measurement sites could be related to differences in site selection. The air pollution data on which our models were based, were obtained using passive samplers for NO_2, located at residential locations. The city specific campaign consisted of selected locations from a routine monitoring program, also using passive NO_2-samplers. This

program is carried out by the Public Health Service, commissioned by the city of Amsterdam. The large area campaign was performed for the purpose of LUR modeling. In previous LUR studies, both strategies (purpose designed and routine monitoring) to collect measurement data have been used regularly.[e.g.35,76] The two measurement campaigns described in this thesis differed slightly and the samplers in the city specific campaign were often placed slightly closer to the road than in the large area campaign. Our study thus illustrates the need to select sampling sites that are representative for the locations to which the model will be applied.

Several other studies have developed LUR models for metropolitan areas,[e.g.84,85] similar to the city specific LUR model developed here. The large area LUR model developed in our study is of a scale intermediate between metropolitan and national scale.[e.g.76,82] Traffic and air quality data used to develop both LUR models were at available at a high resolution, more similar to data used to develop metropolitan models rather than national models. Traffic data, for instance, was obtained from national, provincial and municipal authorities in the study area. Obtaining as much data on local traffic as possible is essential since most people live next to municipal roads, and knowledge about traffic on these roads is important for exposure assessment. In our study, authorities were approached through the networks of the Public Health Services, resulting in 100% participation. In a previous Dutch study[76] about 60% of the municipalities provided data, resulting in availability of traffic data for only 14% of municipal roads. In our study traffic data was available for 31% of the municipal roads and the roads for which no traffic data was available were almost exclusively small roads with little traffic. Using the Public Health Services networks thus most probably lead to a better exposure estimation.

In this study (Chapter 2), LUR models were developed for NO_2 only, as no measurement data on other components of the complex mixture of air pollution were collected. Based on other measurement campaigns, however, LUR models for particulate matter (including soot and ultrafine particles) in Amsterdam were developed recently.[15] The land-use and traffic information available for that study was identical to the data available for our study. Traffic was among the variables explaining the spatial variability of soot, ultrafine and coarse particulate matter ($PM_{2.5-10}$), in consistency with the NO_2 models from our study, but not for $PM_{2.5}$.

Since Dutch legislation orders air pollution concentrations to be estimated by a specific model (CAR dispersion model[10], incorporated in the Monitoring tool, Chapter 1), concentrations as estimated by this dispersion model were also compared with the same NO_2-measurements in Amsterdam (Chapter 2). In a dispersion model, the distribution of a component is modeled using understanding of principal physical and chemical processes. In the CAR

dispersion model, local air pollution concentrations are modeled using such processes and input on standardized emission per vehicle (yearly updated national emission factors), detailed traffic information, the local geographical situation (resulting in more or less dilution due to wind exposure) and background concentrations (as derived from the national monitoring network and emission inventory). In our study, the ability to predict concentrations at independent monitoring sites was similar for the CAR model and the two LUR models (Chapter 2). The few other studies comparing dispersion and LUR models have typically found that LUR models perform at least as well as the dispersion models considered.[81]

The dispersion model was unable to predict the highest ('hot-spot') concentrations observed in Amsterdam. This was also observed for the large area LUR model and in previous LUR studies.[35,74,85,86,171] In epidemiological studies this would result in underestimation of the exposure of subjects with the highest exposures. In general, errors in exposure estimation reduces the statistical power of a study[172] and is most likely to attenuate estimated effects towards zero.[173] This increases the likelihood that existing associations are not detected. In regulation this underestimation may result in air quality dropping on the political agenda. Underestimation of air pollution concentrations at 'hot-spot' locations will lead to less sites where air quality limit values are exceeded, which in turn means less urgency to act as most policy decisions are driven by the need to meet air quality limit values. Underestimation of 'hot-spot' locations by the legislations' monitoring tool may thus lead to trivialization of the air quality issue. Additional monitoring of air quality by measurements at the 'hot-spot' locations may therefore be very useful.

HEALTH EFFECTS

This thesis contributes to the body of evidence on air pollution health effects[e.g.6,17] (Chapters 3 and 4), fitting phase B in the evidence based public health cycle (Figure 1). Health professionals at Public Health Service Amsterdam apply knowledge from the many Dutch and international studies on health effects related to long-term exposure to air pollution in daily practice. These studies showed increased risk of cardiopulmonary mortality, symptoms and disease,[e.g.38,41,67,92,174-177] increased risk of lung cancer,[e.g.47,54,55] and despaired development of lung function.[e.g.56,57] Several international studies have furthermore suggested small adverse effects of maternal exposure to air pollution on pregnancy outcomes.[e.g.42,58,60,61,102,178] Pregnancy outcomes were also studied in the Public Health Service based Amsterdam Born Children and their Development (ABCD) prospective birth cohort study.[59] Exposure was estimated by a spatio-temporal NO_2-model, using the city specific LUR model (Chapter 2) and data from the Amsterdam air quality monitoring network.[16] No indications of a harmful effect on preterm birth, birth weight or the risk of 'small for gestational age' were observed among mothers with the highest exposures in Amsterdam.

In the West of the Netherlands (population of 4 million), long-term exposure to traffic related air pollution in the area of residence is associated with the community prevalence of hospital admissions for cardiopulmonary causes (Chapter 3). Exposure-response relation were shown for asthma, Chronic Pulmonary Obstructive Disease (COPD), Ischemic Heart Disease (IHD), stroke and all cardiovascular causes. The age, sex and income adjusted Prevalence Ratios for the comparisons of subjects with the highest exposure (>31.2 $\mu g/m^3$ NO_2) compared to subjects with the lowest exposure (<17.4 $\mu g/m^3$) were 2.8 (95%-CI: 2.2 to 3.7) for asthma and 1.6 (1.3 to 2.0) for COPD; 1.2 (1.0 to 1.3), 1.2 (1.1 to 1.4) and 1.3 (1.2 to 1.5), for all cardiovascular causes, IHD and stroke, respectively.

To our knowledge, no other studies have examined the long-term effects for air pollution on hospitalization for cardiopulmonary causes in the general population yet. Lin et al.[64] and Wilkinson et al.[69] studied the risk of hospitalization for asthma among children. Lin et al. found no clear exposure-response relation for hospitalization for asthma among children aged 0 to 14 years. However, children hospitalized for asthma were more likely to live in close proximity to heavy traffic compared to children who were not hospitalized.[64] In a similar case-control study among children aged 5-14 years, Wilkinson et al. found no association.[69] A Danish cohort study among 50 to 64 year-olds found a hazard ratio of 1.1 (95%-CI: 1.0 to 1.1, per 6 $\mu g/m^3$ increase in NO_2) for hospitalization for COPD.[41] For stroke hospitalization, one study found no association,[68] a second observed an increased risk among

those with the highest exposures,[65] while no association with coronary heart disease was found.[66] The ecological design of our study allowed us to incorporate a much larger number (double to ten-fold) of hospital admissions compared to previous studies.[41,64-66,68,69] In addition, many of the aforementioned studies used proximity to traffic as main exposure estimate, whereas modeled NO_2-concentrations were used in the present study. As in contrast with traffic indicators, modeling of air pollution concentrations accounts for multiple sources (urbanization) and influential factors such as geographic situation and meteorology, NO_2-concentrations are probably a more valid indicator of exposure than traffic indicators.[31]

A stronger association was found for the total of emergency and planned admissions than for emergency admissions alone (Chapter 3). When short-term exposure to air pollution (episodes) is assumed to be mainly associated with increases in emergency admission, this indicates that hospital admission is also strongly influenced by long-term exposure to air pollution.[20-23,106] Short-term variations may affect all study areas simultaneously as they are weather driven. Consistently, effect estimates found in our study are substantially larger than typical effect estimates of short-term exposure studies over the same concentration range.

Elevated risks for hospital admission were seen at levels well below the EU limit value. Only less than ten percent of the postcodes had NO_2-concentrations exceeding the limit value (40 $\mu g/m^3$). As in many other studies,[e.g.9,41] NO_2 was regarded as an indicator of traffic combustion in our study and the associated health effects are most probably not solely attributable to NO_2 itself. Nevertheless, NO_2 is the most critical legislated air pollution component in the European Union.[3] The EU adapted the WHO guideline for NO_2, even though WHO based its guideline value roughly on indoor studies and acknowledges that "a well established value based on the studies reviewed has not been possible".[1] The public and politicians, however, expect limit values to be health protective and legislative bodies tend to feed this assumption. The EU, for instance, states on its website that their legislation "establishes health based standards".[70]

It is hypothesized that traffic related air pollution triggers systemic oxidative stress and inflammation in for instance endothelial cells and macrophages.[e.g.6,48] This may be a biological mechanism underlying cardiopulmonary health effects, and might also play a role in the promotion of type 2 diabetes by increasing adipose inflammation and insuline resistance.[6,48] The limited number of studies on the relation between air pollution and type 2 diabetes,[50-53] suggest an association which is clearer among women than men.

In this thesis, the relation between long-term exposure to traffic-related air pollution and type 2 diabetes prevalence was studied among 8,018 subjects (aged 50 to 75) living in West-friesland, the Netherlands (Chapter 4).

In contrast with previous studies,[50,51,53] no consistent associations between type 2 diabetes prevalence and exposure to traffic-related air pollution were found, although there was some indication for a relation with traffic in a 250 meter circular buffer around the residential address. Due to the relatively small range in exposure in the study area, the statistical power of our study was limited. For instance, in our study the interquartile range for modeled NO_2-concentration was 2 $\mu g/m^3$, while in previous studies,[50,51] it ranged from 6 to 15 $\mu g/m^3$. An important strength of our study is that many previously undiagnosed diabetes-patients were detected, while in general up to 30-55% of the cases remain undetected.[111] About one third of the type 2 diabetes patients included in our study were diagnosed by the extensive screening procedure. Sensitivity analyses for type of diagnosis (self-reported vs. screening-detected) showed that the screening detected patients with type 2 diabetes contributed importantly to the findings of our study, which may be important for future studies.

PUBLIC HEALTH IMPACT

Technically, assessment of the relevance of the health effects described in the preceding paragraph (Figure 1, phase C: public health impact) was not part of the research presented in this thesis. The hospitalization study (Chapter 3) however, provides information on a large population (4 million) and due to its ecological design, results could be interpreted as public health impact results. The results of Chapter 3 are results applicable to the work of the Public Health Service Amsterdam. The almost threefold Prevalence Ratio of asthma hospitalization among those with the highest compared to those with the lowest exposures illustrates that current local variation of air pollution results in inequity of health risk within the population of a relatively small area (the West of the Netherlands). This type of information is of value for local policy makers when weighing policy priorities.

Although technically not part of this thesis, health impact assessment is among the key activities performed by Public Health Services because of its value for policy makers. Health professionals at the Public Health Services are supported in executing health impact assessments by national guidelines. These guidelines are composed by Working Groups consisting of staff of Public Health Services. Current scientific understanding, up-to-date knowledge of published scientific literature and experience on the type of information needed in daily practice is crucial when writing these guidelines. This knowledge and expertise can be obtained by staff involved in the projects of the Academic Collaborative Center for Environmental Health, thus resulting in a secondary benefit of the Collaborative Center. For example, staff of the Public Health Service Amsterdam who were involved in the work presented in this thesis, were among the co-authors of the guideline for Air Quality and Health,[179] published in 2008. In the guideline, several methods to assess air pollution health impact were introduced. The guideline advises to estimate life expectancy effects, based on a publication of Brunekreef,[5] or the number of Attributable Cases following the approach published by Künzli,[130] for local air pollution concentrations and accounting for the local population. The authors of the guideline are furthermore members of the national Public Health Working Group of Air Quality which provides health professionals at Public Health Services nationwide with updates from the academic, practical and policy field.

POLICY

According to Künzli and Perez,[72] a 'treatment' is applied to meet a certain target in the policy phase (Figure 1, phase D) of the public health practice cycle. In the case of air quality, limit values are that target.[72] Accordingly, evidence based practice should focus on proven effective 'treatments'. Recently, an international workshop on effective actions was held, titled 'From Good Intentions to Proven Interventions: Effectiveness of Actions to Reduce the Health Impacts of Air Pollution'. In the publication following this workshop, Giles et al. listed emerging strategies to address and mitigate air pollution-related health impacts (in no particular order): 1) reduce individual risk, 2) modify activity time, location and level to reduce dose, 3) abate traffic emissions, 4) separate sources and the public.[149]

Reduce individual risk

In order to reduce individual risks, interventions are usually directed at individuals, aiming at exercise, nutrition and medication.[149] Although the reduction of baseline risk of disease is among the main topics of public health, most of the topics proposed by Giles et al. are beyond the classic field of environmental health in the Netherlands. Mostly, these generic health issues are coordinated by the Public Health Service departments for Health Promotion. In the past few years the number of initiatives at Public Health Services in the Netherlands connecting the fields of environmental health and health promotion, however, is increasing. Examples of such initiatives are projects in which promoting physical exercise and abatement of traffic emission are combined, or projects incorporating different health issues in spatial planning.

Modify activity time, location and level to reduce dose

It was also proposed to modify activity time, location and level to reduce dose as air pollution concentrations may vary importantly over time and by location.[149] A high activity level may lead to a higher ventilation rate resulting in a higher dose (volume air taken in increases, dose of pollution increases). This is in line with a policy propagated by Public Health Services in the Netherlands.[179] For example, current advise by Public Health Services on outdoor sporting activities during ozone smog episodes (run in the morning, not late afternoon or night, as concentrations increase importantly at the end of the day) is very much in line with this proposal.[180]

The TRAVEL study (Transport Related Air pollution: Variance in commuting, Exposure and Lung function),[181] another project within the framework of the Academic Collaboration Centre for Environmental Health, resulted in improved insight in level and location of activity in relation to the inhaled dose. Exposure and dose of cyclists, car drivers and bus passengers

were compared. Cyclists were found to have lower exposure to traffic related air pollution, but due to their increased ventilation, a higher dose than car drivers or bus passengers. By adjusting the location of exposure, e.g. riding a low-traffic route instead of along the main connection route, exposure to and dose of air pollution of cyclists could be reduced importantly.[181]

Abate traffic emissions

Another strategy proposed by Giles et al. is to take policy measures abating traffic emissions.[149] In the wide range of possible mitigation measures, low emission zones and road pricing have shown to be effective.[133,182-184] In this thesis, the effect of a speed limit reduction measure taken on the Amsterdam ring freeway on air quality was studied (Chapter 5). Air quality improvement associated with speed limit reduction has been predicted in model studies.[131,134,135,139,185] The Dutch National Department of Transport predicted that PM_{10}-concentrations at roadside in this specific situation would be reduced by 0.5 to 1 percent.[135] This study was the first to evaluate such a measure using air quality measurements. In the year after the speed limit reduction, measured air pollution concentrations were significantly lower compared to the year before the speed limit reduction. Controlling for daily traffic, congestion and weather, the traffic contribution decreased by 2.2 $\mu g/m^3$ for PM_{10}, by 0.4 $\mu g/m^3$ for PM_1 and by 3.6 $\mu g/m^3$ for Black Smoke. The observed PM_{10} reduction of 2.2 $\mu g/m^3$ corresponds to 7 percent of the mean concentration measured at roadside, much larger than the 0.5 to 1 percent predicted by the Department of Transport.[135] Relative to the traffic contribution, which could be regarded a surrogate for traffic emission, a reduction of 38 percent for PM_{10} and a non significant reduction of 4 percent for NO_x was observed. The potential public health impact of this policy measure remains largely unknown from our study, as the effect of the speed limit reduction measure observed was at roadside and not in the neighborhoods.

Schram-Bijkerk et al. performed a health impact assessment (HIA) of several transport related measures, among which this same speed limit reduction.[186] Assuming a 2 to 4 percent NO_2 reduction as was estimated by the Department of Transport[135] and dispersion of traffic related air pollution to a maximum of 300 meters from the freeway, they estimated that exposure of relatively few people was affected. Consequently, the air pollution health impact of this measure was estimated to be limited.[186] The estimation of the health impact of the speed limit reductions in the evaluation by Schram-Bijkerk,[186] might be considered conservative. First, a much larger decrease in particulate matter than in NO_x was observed in our study, while the HIA was solely based on NO_2. Lefebvre et al.[187] furthermore expect a speed limit reduction on all Belgian freeways to be far more influential, and traffic and population density in Belgium and the Netherlands are similar. In their

modeling of soot dispersion, assuming distribution over an area much larger than 300 meters from the freeways, they optimistically estimated about 15 percent of the Belgian population would be affected by such a measure.[187]

Keuken et al. focused on changes in traffic dynamics and related changes in emission.[188] They used a combination of dispersion modeling and (independent) measurements along the same road section in Amsterdam, and another road section in Rotterdam. It was concluded that the emission reduction was in the range of 5 to 25 percent for PM_{10},[188] which is lower than the 38 percent we observed, and 5 to 30 percent for NO_x,[188] consistent with our study.

Separation of sources and the public

Giles et al. concluded with discussion on the potential effectiveness of separation of sources and the public as a policy measure to reduce the health impact of air pollution.[149] Local initiatives have been taken in Canada and California.[189,190] In the Netherlands, separation of dense traffic and the public has been promoted in the Public Health Services Guideline on Air Quality and Health since 2008.[179] In the guideline it is recommended to plan buildings facilitating vulnerable members of the population ('sensitive sites'), at a minimum distance of 300 meters from freeways and off the primary building edge of other busy (city) roads, regardless the air pollution concentrations. The guideline aims at the protection of the most vulnerable members of the population; children, elderly and those with pre-existing health conditions affected by air quality, such as respiratory or cardiovascular disease. Moreover, the guideline includes working definitions of the most sensitive sites (schools, daycare centers, nursing homes and residences) and of busy roads (a traffic flow of at least 10.000 motor vehicles per 24hrs). In addition, a user- and using-time based scaling system to score the sensitivity of other sites (such as sports fields and hospitals) was proposed. The guideline furthermore recommends aiming at new buildings, since large-scale restructuring is not anticipated by any authority.

In 2008, national legislation was implemented prohibiting new sensitive sites (excluding residences) when planned within 300m of a freeway or 50m of a provincial road and EU air quality limit values are not met. This legislation is expected to be temporal as the Ministry of Environment is anticipating to meet the EU air quality limit values nationwide by 2015. Although this legislation seems in line with the aforementioned guideline,[179] the inclusion of air quality limit values is affecting the impact dramatically as these limit values are not protecting health, but are merely a compromise between practical and economical feasibility and the objective to protect public health. Due to this unsatisfactory legislation, turmoil and discussions on this issue remain at a

local level. For instance, parents disagree with plans for relocation of a primary school near a freeway, resulting in long disputes including lawsuits.

Amsterdam was the first large city in the Netherlands in which policy regarding sensitive sites and air pollution was implemented by the City Council on advise by the Public Health Service.[191] Since spatial planning in such densely populated areas is a challenge, the policy was not fully adapted as recommended by the Public Health Service. First, residential sites were not listed as sensitive, as only a fraction of the users is part of the defined sensitive population (children, elderly, those with a frail health status). Secondly, the policy allows the relevant governor to deviate from the regulation after consulting the Public Health Service. When the governor decides to allow a specific sensitive site near traffic, the Public Health Service is asked to contribute to optimize the plans. Opportunities for optimization are often found in the location of the site within a spatial plan, location of the building within the proposed area or the function of rooms within the proposed building.

Reducing indoor exposure by filtration

Not mentioned by Giles et al.,[149] but often considered as an attractive opportunity by policy makers, especially when allowing a sensitive site near traffic, is limitation of infiltration of outdoor air pollution into the indoor environment. In Amsterdam, advise on filtration systems was requested by policy makers and governors in several situations. As very little evidence on the practical effectiveness of such systems was available, an experiment to obtain more knowledge was performed (Chapter 6).

In an occupied classroom, three different ventilation systems, each equipped with a fine particle filter were tested. Indoor and outdoor air pollution concentrations were measured and the infiltration of particulate matter during each of the system tests and during natural ventilation was studied. The infiltration of particulate matter was reduced by some systems; one system which worked by displacement ventilation (mechanical inlet and natural outlet) and had a F7 particle filter reduced infiltration of ultrafine particles (PNC) by one half; another system, with balanced displacement ventilation (mechanical in- and outlet, with overpressure) and a higher rated F9 filter, equally reduced PNC-infiltration and reduced $PM_{2.5}$-infiltration by 25 percent. No significant reduction of soot infiltration was observed. The demand-driven ventilation systems (by indoor CO_2-concentration) were not in operation during the full 24hr-interval at which $PM_{2.5}$ and soot were measured. In contrast, PNC was measured during school hours only. This possibly contributed to the somewhat inconsistent results for $PM_{2.5}$, soot and PNC and the finding that filtration improved indoor air quality less than expected. Further research in other classrooms, with other student populations and building characteristics, and at schools in other geographical settings is

needed. To find out if the observed improvement of indoor air quality is associated with reduction of the health risk experienced by children attending school at high traffic locations, more research is necessary.

Mechanical ventilation systems may introduce discomfort such as noise or draft and air pollution due to dusty or unhygienic ducts, filters and vents.[147,168,169] In our study, these types of problems were not reported. In contrast with some previous studies,[161,170] teacher and students perceived a better indoor climate with the mechanical ventilation systems working. Perception, however, may have been biased as it was not possible to conduct this study blindly. System technology and filter classification were nevertheless unknown by students and teacher. Moreover, perception and appreciation may possibly change over time when systems are used for periods longer than the four test weeks.

Although there is a large body of literature on in-school climate and/or air quality,[153,159-166] no other studies tested different mechanical ventilation systems in one classroom and kept all factors except weather conditions constant between all system tests. In most other studies, it was not possible to distinguish effects of location and building from effects of method of ventilation due to changing circumstances. In contrast with most other studies, the classroom studied was furthermore occupied resulting in a real-life test.

THE PROCESS OF POLICY MAKING

When aiming on evidence based public health, the process of policy making may be a challenge for health professionals as responsibilities, dynamics and culture are very different from the three other phases in the public health practice cycle (Figure 2). In current practice in Amsterdam, civil servants at different authorities (e.g. the city offices for traffic and infrastructure, environment or spatial planning), policy makers at the different government tiers and foremost local, provincial and national governors are important actors in the process of policy making. Although health professionals of the Public Health Service are involved in policy making, they carry limited formal responsibility. Policy making is furthermore a process in which many issues are considered, of which health is only one (Figure 2). In general, health is an issue valued highly by politicians and policy makers. Opinions on prioritization, costs and benefits, etcetera may differ importantly.

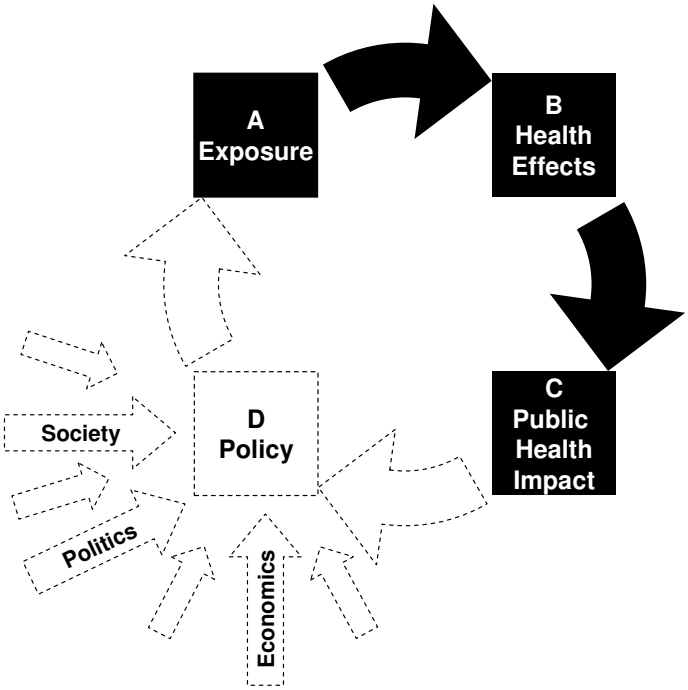


Figure 2. Policy making in the framework for evidence based public health; influenced by health, but also many may other issues.

The role of health professionals in the process of policy making is explained here using the policy cycle suggested by Lasswell[192] (Figure 3). According to Lasswell, policy making starts with *Problem Identification* (Phase I), which in the case of air pollution can be closely related to phase C (*Assessment of Public Health Impact*) of the evidence based public health cycle: Assessing the health impact of air pollution health effects and translating this into a comprehensible message, helps to identify and prioritize the topic and to place it on the policy agenda. In daily practice, the relevant actors are mainly civil servants with different backgrounds, including health professionals and environmental policy makers. Debates between these servants characterize this phase as for some servants legal obligation (EU legislation) may be the main motive for policy, whereas other servants are driven by motives such as public health protection.

In *Policy Formulation* (phase II), understanding of the mechanisms involved in the association between air pollution and health can be very useful.

From a legislation perspective, for instance, novel coatings which bind NO_2 from ambient air seem quite attractive as not attaining European NO_2-limit values might result in large penalty fees and possibly restrictions in development of infrastructure. From a health perspective, it is known that NO_2 should be considered as indicator component only. Removing NO_2 from the air pollution mixture while not addressing the other components will not result in 'healthier' air. In daily practice, it may be very challenging to get hold of this process, for instance due to the fact that actors are often largely unknown as they can be from many commercial organizations too. In this phase, health professionals may contribute to such discussions, though (environmental) policy makers are in charge.

In the *Adoption* of a potential policy (phase III), health arguments have to compete with many other interests, such as economical, political and legal issues (Figure 2). This phase is dominated by political argumentation rather than scientific evidence. The decisions are taken by the relevant governor, who often needs approval of the council or parliament. Debate between civil servants is for an important part formalized in notes and policy documents. Health professionals do not play any formal role, key actors in this phase are policy makers, politicians and foremost governors.

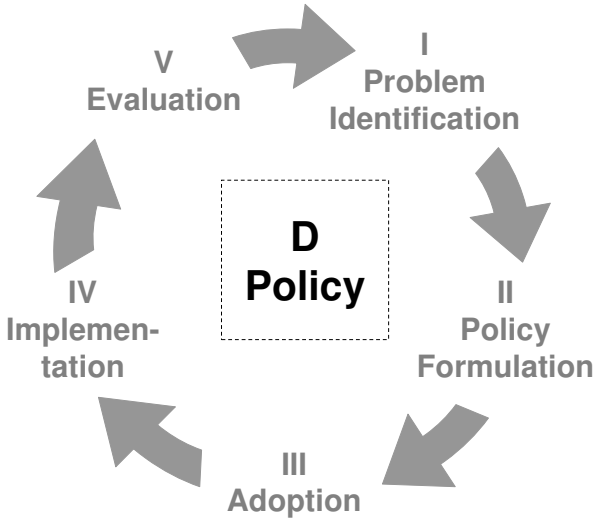


Figure 3. The Lasswell[192] policy cycle; relevant to Phase D (Policy) of the framework for evidence based public health.

In the phase of *Implementation* (IV), public support is of great importance for the governor responsible for the policy. In daily practice, policy makers tend to revert back to the original motive s to make a policy. As, in contrast with many other intentions, health is almost always considered a noble motive, health professionals are often requested to contribute to the build of public support by sharing arguments and evidence used in the previous two phases of the cycle. In Amsterdam, health professionals contribute importantly to activities aiming at building support, for instance by contributions to newspaper articles, TV interviews or at neighborhood gatherings. The practical or physical implementation of policies is controlled by other civil servants, depending on the actual policy. Traffic measures, for instance, are implemented by the municipal service for traffic and infrastructure.

The *Evaluation* phase (V) of the policy cycle may feed into, or even be part of, *Exposure Assessment* in phase A of the public health practice cycle (Figure 2). The studies on the effectiveness of a speed limit reduction measure (Chapter 5) and of fine particle filtration (Chapter 6) are examples of the scientific basis of such an evaluation. Thorough evaluation, however, may also include evaluation of *Health Effects* (Phase B) and *Health Impact* (Phase C). In the case of air quality policies in Amsterdam, no evaluation of health effects or –impact have been made yet. Policy evaluation may also include social, economical and administrational issues, which is currently coordinated from the municipality offices and the Public Health Service contributes to these evaluations limitedly.

As illustrated, health professionals are involved and embedded in the process of policy making to a limited extent. As a result, they can not guarantee full incorporation of evidence in this process. Consequentially, the phase of policy making is probably the Achilles' heel in the cycle of evidence based public health.

Though not in charge, health professionals can contribute importantly to effective (local) air quality policy, as for instance was shown in the Amsterdam policy for 'sensitive sites'. The limited involvement and responsibility of health professionals originates largely from the, good, democratic principle that voted politicians make the decisions and consider the many relevant issues of which health is one. The main challenge for health professionals may be to bring the health argument into the limelight as well as some of the other actors can do with their arguments or (commercial) solutions.

OUTPUT

The Academic Collaborative Center for Environmental Health aims to produce evidence to be applied in evidence based practice of environmental health. ZonMW (Netherlands Organization for Health Research and Development) initiated programs such as the Academic Collaborative Centers for Public Health to improve the societal use of science.[193] In recent years, key research organizations in the Netherlands, such as the Dutch Royal Academy of Sciences have initiated and participated in several projects[194,195] on the topic of societal use of research. Several contemplations on the processes involved were published,[193-199] summarized in Figure 3. In brief, society has its issues, in this case a health issue (health impact of air pollution). Science *responds* to this issue by distilling research questions and performing the subsequent research, resulting in *scientific output* (this thesis). The *relevance* of the scientific work determines the societal conclusion, resulting in *societal output*, which may have *impact*[200,201] on the health issue. In the case of the work presented in this thesis, different types of societal output were produced by the Public Health Service of Amsterdam.

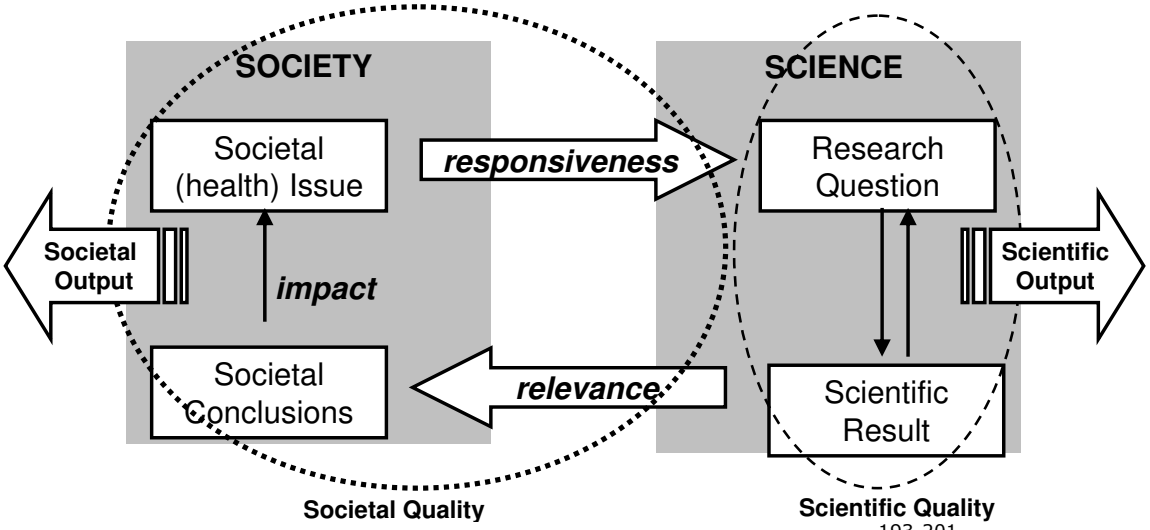


Figure 3. A framework for a societal research cycle.[193-201] The circles represent scientific and societal quality. Responsiveness, relevance and societal impact are the main indicators of societal quality.

There is a rich history of quantifying scientific output.[202] Journals are rated by impact factors. Throughout the academic world, scientists and departments are evaluated by numbers of publications and citations of scientists and departments, weighed by impact factor. For organizations such as Public Health Services, the scientific output is not measured as such.

Generally, societal impact is not evaluated in academia.[196] Although societal output is the main output of Public Health Services, output is not evaluated. According to ZonMW,[193] societal quality of research is determined by responsiveness (the significance of the research question for the societal

issue), relevance (the significance of the scientific result for societal conclusions) and societal impact (practical use of results and conclusions in practice or policy) as displayed in Figure 3. The responsiveness of the research presented in this thesis is considered high as the research questions were formulated by the Public Health Service. All questions had a policy or practice background. Specifically, the evaluation of measures was done at the request of a policy maker (Chapters 5 and 6). The research of Chapter 6 was even initiated by an Amsterdam alderman. That part of the research was also co-funded by municipal offices. The relevance of the scientific results for societal conclusions was high as well. All results have been, and will be, used in policy advice and public education activities of the Public Health Service. Following the results presented in Chapter 6, municipal tenders for school renovation were changed by the city of Amsterdam. The interest of societal organizations is furthermore illustrated by the large attendance at work shops and conferences.

Mostert et al.[203] recently proposed a method of quantifying societal impact of medical research. The concept proposed may be of use for illustrating the societal impact of Academic Collaborative Centers, although the field of public health differs considerably from the field of medicine as shown in the context of evidence based medicine vs. evidence based public health in Chapter 1. The societal output could be quantified by listing specific indicators by stakeholder-group. Figure 4 shows the Societal Quality Indicator Matrix proposed,[203] adapted to the case of air pollution at Public Health Services. In contrast to medicine, the stakeholder-group 'private sector', consisting of e.g. pharmaceutical, bio-technological and medical services, is of much less relevance for public health than for medicine and was removed from the original matrix. In the air pollution context, the group of 'health care professionals' may furthermore be extended by public servants in the fields of environment, traffic, spatial planning and health policy. In the matrix the stakeholder-group 'health care professionals' therefore was renamed 'professionals'.

Figure 4 furthermore shows the estimated output related to the current project per indicator. The different types of output in the public domain ranged from contributions to the public debate at discussion gatherings or in the media, opinionating articles in popular journals[e.g.204,205] and the Public Health Service Guideline on air quality health effects.[179] This output may have contributed to policies reducing air pollution levels and/or population exposure to air pollution. The aforementioned Amsterdam policy for sensitive sites is an example of such a policy.[191] It is impossible to show the exclusive output of the project, as it is closely related with, and connected to other activities within the collaborating Public Health Service and university. In rating the societal output of a project this may pose a challenge.

	Stakeholder-group	
	General public	**Professionals**
Knowledge production	• Contributions to: · television or radio programs *(5 to 10)* · newspapers or journals (non peer reviewed) *(15 to 20)* · public websites or news forums *(5 to 10)* · schoolbooks or study material *(none)*	• Publications in journals (non peer reviewed) *(3 to 5)* • Contributions to: · professional websites *(5 to 10)* · guidelines or protocols *(3 to 5)*
Knowledge exchange	• Memberships of public (funding) agencies or organizations *(3 to 5)* • Speeches for general public or contributions to public forums *(15 to 20)* • Information for scholars *(>20)*	• Memberships of advisory committees or professional associations *(10 to 15)* • Speeches at conferences *(5 to 10)*
Knowledge use	• Use of schoolbooks or study material in education programmes *(5 to 10)*	• Use of new charters or protocols in practice *(3 to 5)*
Earning capacity	• Charity funding *(none)*	• Indirect funding *(300k€)*

Estimated output is related to, yet not exclusively resulting from the work presented in this thesis.

Figure 4. Quantifying societal output. Indicator Matrix: indicators (estimated output) by stakeholder-group. *Adapted from Mostert et al.*[203]

An important limitation of the proposed matrix is that typical impacts of public health activities, such as improved life expectancy due to cleaner air, may take decades and are virtually impossible to attribute to a single project or department. Moreover, the matrix proposed by Mostert et al.[203] is lacking the stakeholder-group of policy makers and politicians. In the process of evidence based practice in the field of air pollution, this stakeholder-group is crucial as they are responsible for a number of key phases in the policy making (see *The process of policy making*). In order to be of use for quantitative evaluation in public health, the matrix in Figure 4 needs further development, accounting for these limitations.

In future projects aiming on improved evidence based public health, the difference in appreciation of scientific and societal output by universities and Public Health Services may be conflicting. For instance, it may be difficult to find the facilities and considerable amount of time needed to produce scientific output from a Public Health Service environment when this output is not appreciated by the relevant governor. Consistently, universities might find activities not resulting in scientific publications a waste of time. The Academic Collaborative Center for Environmental Health and its dedicated funding showed to be a successful method to guarantee the facilities and time needed to improve evidence based public health.

CONCLUSIONS

The objective of the research presented in this thesis was to contribute scientific evidence on exposure to traffic related air pollution, its health effects and potential mitigation measures, relevant to Public Health Services in the Netherlands. In evidence based public health, practical expertise is integrated with the best available systematic research, and decisions are made with the conscientious, explicit, and judicious use of the current best evidence. The research in this thesis is presented using a framework for evidence based public health.

Exposure Insight in the population exposure to traffic related air pollution was gained by development of several approaches to estimate traffic related air pollution (Chapter 2).

Health Effects Elevated prevalence of hospital admission is not only related to short-term episodes of increased air pollution levelsas is known from previous research by others, but also with long-term exposure to modest levels of air pollution (Chapter 3). In contrast with previous studies, no consistent association between type 2 diabetes prevalence and exposure to traffic related air pollution was found. (Chapter 4).

Public Health Impact Elevated risks for hospital admission were seen at levels well below the EU limit value (Chapter 3).

Policy Promising measures to mitigate air pollution showed to be effective, yet to a limited extent. Modest roadside concentration decreases were demonstrated by an emission reducing speed limit reduction (Chapter 5). Fine particle filtration of indoor air was able to limit the infiltration of pollution, though indoor air quality was still importantly dependent on outdoor concentrations (Chapter 6).

The process of policy making may be a challenge as responsibilities, dynamics and culture may be very different from the three other phases in the public health practice cycle. Health professionals are limitedly involved and cannot guarantee incorporation of evidence in the process of policy making, making this phase probably the Achilles' heel in the cycle of evidence based public health (Chapter 7).

It may be a challenge to find the facilities and time needed to improve evidence based public health in both universities and Public Health Services, as the different outputs (scientific vs. societal output) are traditionally appreciated differently. The Academic Collaborative Center for Environmental Health and its dedicated funding showed to be a successful method to guarantee time and facilities for evidence based public health (Chapter 7).

Chapter 8

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Chapter 9

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Chapter 10

Summary

Air pollution is probably the most intensely studied field in today's environmental health research. The extensive body of literature on health effects associated with air pollution exposure[e.g.6,17] has lead to prioritization of air pollution as public health risk factor by the WHO,[1] and has resulted in air quality regulations worldwide.[e.g.2-4] At current levels air pollution, however, still has a significant health impact.[e.g.92,175] The policy debate on the necessity to act, which tier is responsible and what are effective measures continues. Although in essence this is a debate in the political arena, science could play an important role by providing a solid evidence basis for the decision makers. The primary objective of this thesis is to provide *evidence* of the health effects of traffic related air pollution and potential mitigation measures relevant to Public Health Services in the Netherlands.

Air pollution, exposure assessment and related health effects were introduced and a brief overview of Dutch air pollution policy was given (Chapter 1). We furthermore introduced a framework for evidence based public health practice[72] consisting of four phases: health professionals starting the cycle by first looking at current exposure and then looking at possible health effects, subsequently performing a public health impact assessment followed by policy making, returning to assessment of exposure, health effects, etc. By bringing scientific evidence to each of the phases of the framework, environmental health professionals from Public Health Services can contribute importantly to healthy air quality policies.

Exposure

In the phase of Exposure Assessment, we developed and evaluated two land use regression (LUR) models to estimate long-term exposure to traffic related air pollution (Chapter 2). One model was developed for the West of the Netherlands (consisting of the Provinces of Noord-Holland and Flevoland and the Rijnmond area; large area model), the other model was developed for the city of Amsterdam (city specific model). Both models predicted NO_2 concentrations well (R^2s of 87% and 72%, respectively). As we developed these models to estimate concentrations at unmeasured locations, we wanted to obtain insight in the predictive performance at independent measurement sites. The modeled concentrations were therefore compared with independent measurements in Amsterdam, showing a lower percentage of variability that was explained (large area model 48%, city specific model 57%) than for the measurement sites on which the models were based. We also compared concentrations as estimated by the CAR dispersion model (the model authorities are obliged to use according to Dutch air quality legislation), with the same NO_2-measurements in Amsterdam. The ability to predict concentrations at independent monitoring sites was similar for the CAR model and the two LUR models.

Health Effects

We showed that in the West of the Netherlands (population of 4 million), long-term exposure to traffic related air pollution in the area of residence is associated with the community prevalence of hospital admissions for cardiopulmonary causes (Chapter 3). Age, sex and income adjusted Prevalence Ratios for the second to fourth quartile of exposure relative to the first quartile of exposure showed exposure-response relations for asthma, COPD (Chronic Pulmonary Obstructive Disease), IHD (Ischemic Heart Disease), stroke and all cardiovascular causes. The Prevalence Ratios for the comparisons of subjects with the highest to subjects with the lowest exposure were 2.8 (95%-CI: 2.2 to 3.7) for asthma and 1.6 (1.3 to 2.0) for COPD. Prevalence Ratios for all cardiovascular causes, IHD and stroke, were 1.2 (1.0 to 1.3), 1.2 (1.1 to 1.4) and 1.3 (1.2 to 1.5) respectively, for the same difference in exposure. Elevated risks for hospital admission were seen at levels well below the EU Limit Value, less than ten percent of the postcodes had NO_2-concentrations above the limit value.

It is hypothesized that air pollution may promote type 2 diabetes by increasing adipose inflammation and insulin resistance. In Chapter 4 we examined the relation between long-term exposure to traffic-related air pollution and type 2 diabetes prevalence among 8,018 subjects aged 50 to 75 years living in Westfriesland, the Netherlands. We did not find consistent associations between type 2 diabetes prevalence and exposure to traffic-related air pollution. However, there was some indication for a relation with traffic in a 250m buffer.

Mitigation Measures

We studied the air quality effects of a speed limit reduction from 100kph to 80 kph on a section of the Amsterdam ring highway (Chapter 5). At an air quality measurement site next to the freeway, air pollution concentrations were significantly lower in the year after the speed limit reduction compared to the year before. Controlling for daily traffic, congestion and weather, the traffic contribution decreased by 2.2 $\mu g/m^3$ for PM_{10}, by 0.4 $\mu g/m^3$ for PM_1 and by 3.6 $\mu g/m^3$ for Black Smoke. Decreases in air pollution concentrations during the same period were also observed at a freeway section without speed limit reduction. Decreases in PM_{10} and PM_1 concentrations, however, were significantly greater at the intervened freeway section.

Policy makers often consider limitation of infiltration of outdoor air pollution into the indoor environment by filtration an opportunity to reduce population exposure. As very little evidence on the practical effectiveness of such fine particle filtration systems was available, we performed an experiment to obtain more knowledge (Chapter 6). In a school, we tested three different ventilation systems, each equipped with a fine particle filters: A) worked by displacement ventilation (mechanical inlet and natural outlet)

and had a F7 particle filter, B) was a balanced ventilation (mechanical in- and outlet) which was also equipped with a F7 filter, C) was a balanced displacement system (mechanical in- and outlet, with overpressure) and had a higher rated F9 filter. We measured indoor and outdoor air pollution concentrations and studied the infiltration of particulate matter during each of the system tests and during natural ventilation. The infiltration of particulate matter was reduced by some systems; systems A and C reduced infiltration of ultrafine particles (PNC) by one half, system C also reduced $PM_{2.5}$-infiltration by 25 percent. No significant reduction of soot infiltration was observed. The demand-driven ventilation systems were not in operation during the full 24hr-interval at which $PM_{2.5}$ and soot were measured. This possibly contributed to filtration improving indoor air quality less than expected. To find out if the observed improvement of indoor air quality is associated with reduction of the health risk experienced by children attending school at high traffic locations, more research is necessary.

Evidence Based Public Health

In Chapter 7 we discussed the findings of our research within the framework of evidence based public health practice introduced in Chapter 1. The scientific work on exposure (Chapter 2) can be applied, together with the existing knowledge and data of the Amsterdam air quality monitoring network, in the Exposure phase of public health practice. The work presented in Chapters 3 and 4 contributes to the knowledge about Health Effects, which is the next phase of the framework. The ecological design of the hospitalization study (Chapter 3) also provides insight on the Public Health Impact (third phase of the framework). The fourth phase in the framework is Policy. Emerging strategies to address and mitigate air pollution-related health impacts[149] are: 1) to reduce individual risks, 2) to modify activity time, location and level to reduce dose, 3) to abate traffic emissions, 4) to separate sources and the public. The speed limit reduction studied in Chapter 5 is an example of a strategy to abate traffic emissions. Following a strategy to separate sources and a sensitive part of the public (children), policy makers wanted advise on possibilities to reduce infiltration of outdoor air pollution into the indoor (school)environment, which resulted in the research presented in Chapter 6 (fine particle filtration).

Responsibility, dynamics and culture may be very different in policy making than in the three other phases of the public health practice cycle. In current practice in Amsterdam, health professionals of the Public Health Service are involved in, but carry limited formal responsibility for policy making. In Chapter 7, we discuss this using the Lasswell[72] policy cycle: Although health professionals contribute importantly to the phase of problem identification, during the phases of policy formulation, adoption and implementation, they are hardly influential and key actors are policy makers

and foremost governors. Health professionals may play a role in evaluation, as shown in Chapters 5 and 6. Health professionals are limitedly involved and can not guarantee incorporation of evidence in the process of policy making, making this phase probably the Achilles' heel in the cycle of evidence based public health.

Another challenge of evidence based practice lies in a cultural difference in the evaluation of output between academia and practice. Whereas scientific output and impact (publication in scientific journals) is important at universities, the focus at Public Health Services is at societal output, such as policy advice, contributions to the public debate at discussion gatherings or in the media. Institutions funding scientific research acknowledge that societal impact of scientific work should receive more attention. Recently, a tool for evaluation of the societal impact of medicine was proposed, we discussed how this tool would be applicable to public health. The Academic Collaborative Center for Environmental Health and its dedicated funding showed to be a successful method to guarantee time and facilities for evidence based public health (Chapter 7).

Conclusions

The research presented in this thesis contributes to the understanding of today's impact of air pollution on public health in the West of the Netherlands. We gained insight in the population exposure to traffic related air pollution. Elevated prevalence of hospital admission is not only related to short-term episodes of air pollution as is known from previous research by others, but also with long-term exposure to modest levels of air pollution. Elevated risks for hospital admission were seen at levels well below the EU Limit Value.

Promising measures to mitigate air pollution showed to be effective, yet to a limited extent. Modest roadside concentration decreases were demonstrated by an emission reducing speed limit reduction. Fine particle filtration of indoor air was able to limit the infiltration of pollution, though indoor air quality was still importantly dependent on outdoor concentrations.

Chapter 11

Samenvatting

Luchtverontreiniging is waarschijnlijk het meest bestudeerde veld binnen het onderzoek naar Milieu en Gezondheid. De grote hoeveelheid gepubliceerde studies naar de gezondheidseffecten van luchtverontreiniging[o.a.6,17] hebben geleid tot prioritering van luchtvervuiling als gezondheidsrisico door de WHO.[1] Wereldwijd heet dit tot luchtkwaliteitsbeleid geleid.[o.a.2-4] Desalniettemin heeft luchtvervuiling, ook bij de huidige niveaus, een significante gezondheids-impact.[o.a.92,175] Het beleidsdebat over de noodzakelijkheid tot handelen, welke bestuurslaag verantwoordelijkheid draagt en wat effectieve maatregelen zijn woedt daardoor voort. Alhoewel dit debat hoofdzakelijk in de politieke arena speelt, zou de wetenschap een belangrijke rol kunnen spelen door beleidsmakers een solide wetenschappelijke basis te leveren. Het hoofddoel van dit proefschrift is wetenschappelijk bewijs (evidentie) over de gezondheidseffecten van verkeers-gerelateerde luchtverontreiniging en potentiële maatregelen te leveren die relevant is voor GGD'en in Nederland.

De inleiding (Hoofdstuk 1) bevat een korte introductie over luchtvervuiling, blootstellingkarakterisering, gerelateerde gezondheidseffecten en een beknopt overzicht van het Nederlandse luchtkwaliteitsbeleid. Ook werd er een kader voor *evidence based* milieu-gezondheidszorg[72] gegeven, welke uit vier fases bestaat: GGD medewerkers beginnen de cyclus door de Blootstelling in kaart te brengen, vervolgens worden de gerelateerde Gezondheideffecten bestudeerd, waarna de publieke Gezondheidsimpact wordt ingeschat, vervolgens leidt dit (mogelijk) tot Beleid, waarna wederom de blootstelling, gezondheid etc. worden bestudeerd. Door in iedere fase evidentie in te brengen, kunnen GGD medewerkers Milieu en Gezondheid in belangrijke mate bijdragen aan een gezond luchtkwaliteitsbeleid.

Blootstellingskarakterisering

In de Blootstellingskarakteringsfase hebben we twee land use regression (LUR) modellen ontwikkeld en geëvalueerd (Hoofdstuk 2). Met deze modellen kan langdurige blootstelling aan verkeersgerelateerde luchtverontreiniging worden ingeschat. Het ene model was ontwikkeld voor West Nederland (de provincies Noord-Holland en Flevoland en het Rijnmondgebied), het andere voor Amsterdam. Het LUR model voor het grotere gebied en die voor Amsterdam bleken NO_2-concentraties goed in te schatten (R^2 van respectievelijk 87 en 72%). Omdat we deze modellen ontwikkeld hebben om concentraties op locaties te schatten waar we geen metingen beschikbaar hadden, wilden we inzicht verkrijgen in de modelprestaties op onafhankelijke plekken. De gemodelleerde concentraties werden daarom vergeleken met onafhankelijke metingen in Amsterdam. Het percentage van de variabiliteit die door het model werd verklaard was lager voor de onafhankelijke meetpunten (LUR model voor grote gebied 48%, Amsterdams model 57%) dan voor de meetpunten op basis waarvan de modellen waren ontwikkeld. We hebben op dezelfde punten ook gemeten concentraties vergeleken met concentraties

gemodelleerd met het CAR dispersiemodel (het in de Nederlandse wetgeving verplicht gestelde rekenmodel). De kwaliteit van de inschattingen door middel van het CAR model was vergelijkbaar met de twee LUR modellen.

Gezondheidseffecten

Uit ons onderzoek bleek dat in het Westen van Nederland (populatie: 4 miljoen), langdurige blootstelling aan verkeersgerelateerde luchtverontreiniging in de buurt van de woning samenhangt met de prevalentie van ziekenhuisopnamen voor cardiopulmonaire aandoeningen (Hoofdstuk 3). De voor leeftijd, geslacht en inkomen gecorrigeerde Prevalentie Ratio's voor het tweede tot vierde blootstellingskwartiel, ten opzichte van het laagst blootgestelde kwartiel, lieten een blootstellings-respons-relatie zien voor astma, COPD (chronische obstructieve longziekten), IHD (ischemische hartziekten), beroerte en de verzamelde hart- en vaatziekten. De Prevalentie Ratio's voor astma en COPD waren 2.8 (95%-betrouwbaarheidsinterval: 2.2 tot 3.7) en 1.6 (1.3 tot 2.0) voor de gebieden met de hoogste blootstelling (>31.2 $\mu g/m^3$ NO_2) in vergelijking met de laagst blootgestelden (<17.4 $\mu g/m^3$). Prevalentie Ratio's voor hart- en vaatziekten, IHD en beroerte waren respectievelijk 1.2 (1.0 tot 1.3), 1.2 (1.1 tot 1.4) en 1.3 (1.2 tot 1.5) voor dezelfde blootstellingsverschillen. Een verhoogd risico op ziekenhuisopname werd bij concentraties ver onder de EU grenswaarden gezien, in minder dan 10 procent van de postcodegebieden was sprake van overschrijding.

Luchtvervuiling zou door toename van adipose ontsteking en insuline resistentie kunnen bijdragen aan het ontstaan van type 2 diabetes. In Hoofdstuk 4 hebben we de relatie tussen langdurige blootstelling aan verkeersgerelateerde luchtverontreiniging en type 2 diabetes prevalentie onderzocht onder 8018 personen (in de leeftijd van 50 tot 75), woonachtig in Westfriesland. We hebben geen consistente associaties tussen diabetes en luchtverontreiniging gevonden, wel was er enige aanwijzing voor een relatie met verkeer in een cirkel van 250m rond de woning.

Maatregelen

We bestudeerden de luchtkwaliteitseffecten van een beleidsmaatregel waarin de maximale snelheid op een deel van de Amsterdamse ring A10 werd teruggebracht van 100 naar 80 km per uur om de verkeersemissie van luchtvervuiling terug te brengen (Hoofdstuk 5). Op een meetpunt naast dit stuk snelweg waren de concentraties luchtvervuiling significant lager in het jaar na de maatregel dan in het jaar ervoor. De voor verkeer, file en weer gecorrigeerde verkeersbijdrage aan lokale PM_{10} concentraties daalde met 2.2 $\mu g/m^3$. Voor PM_1 was dit 0.4 $\mu g/m^3$ en voor Zwarte Rook 3.6 $\mu g/m^3$. Ook langs een deel van de A10 waar geen snelheidsbeperking gold werden in dezelfde periode lagere concentraties gemeten, maar de dalingen voor PM_{10} en PM_1 waren significant groter langs het stuk snelweg met snelheidsbeperking.

Beperking van de doordringing van buitenluchtvervuiling in het binnenmilieu door filtering wordt door beleidsmakers vaak gezien als een oplossing voor blootstelling, bijvoorbeeld op basisscholen. Omdat er erg weinig bewijs is over de praktische effectiviteit van fijn stof filters, hebben we een experiment uitgevoerd om meer kennis hierover te vergaren (Hoofdstuk 6). We hebben in een school drie ventilatiesystemen met fijn stof filter getest; A) een verdringingsventilatiesysteem (met mechanische invoer en natuurlijke afvoer) en een F7 fijn stof filter, B) een gebalanceerd systeem (in- en afvoer mechanisch) met een F7 filter, C) een gebalanceerd verdringings-ventilatiesysteem (in- en afvoer mechanisch, overdruk in de ruimte) met een fijner F9 filter. We hebben binnen en buiten luchtvervuiling gemeten en vergeleken de infiltratie van fijn stof gedurende de oorspronkelijke situatie met natuurlijke ventilatie met de infiltratie gedurende de tests. Toepassing van sommige systemen leidde tot beperking van de infiltratie van fijn stof; systemen A en C beperkten de infiltratie van ultrafijn stof (PNC) tot de helft, bij systeem C nam ook de infiltratie van $PM_{2.5}$ met een kwart af. De roetinfiltratie verminderde niet significant. De vraaggestuurde ventilatie-systemen waren niet in werking gedurende het volledige 24-uurs interval waarop $PM_{2.5}$ en roet werden gemeten. Dit heeft mogelijk bijgedragen aan minder dan verwachtte verbetering van de binnenluchtkwaliteit. Er is meer onderzoek nodig om na te gaan of de verbeterde binnenluchtkwaliteit leidt tot vermindering van de gezondheidsrisico's van kinderen die naar school gaan op locaties met een hoge verkeersbelasting.

Evidence Based Publieke Gezondheid

In Hoofdstuk 7 hebben we de resultaten van ons onderzoek in het kader voor evidence based milieu-gezondheidszorg (Hoofdstuk 1) gezet. De evidentie over blootstelling (Hoofdstuk 2) kan samen met bestaande kennis en data uit het Amsterdamse luchtmeetnet, worden toegepast in de Blootstellingsfase van het kader. In de fase Gezondheidseffecten kan het werk gepresenteerd in de Hoofdstukken 3 en 4 bijdragen aan de bestaande kennis. Het ecologische design van de studie uit Hoofdstuk 3 biedt daarnaast ook inzicht in de Gezondheidsimpact in het werkgebied (derde fase van het kader). De vierde fase is Beleid. Actuele strategieën in de aanpak van luchtvervuiling gerelateerde gezondheidseffecten[149] zijn: 1) het beperken van individueel risico, 2) het aanpassen van moment, locatie en intensiteit van activiteiten om de dosis te beperken, 3) beperken van verkeersemissies, 4) het scheiden van bronnen en publiek. De snelheidsbeperkingsmaatregel uit Hoofdstuk 5 is een voorbeeld van een maatregel die de verkeersemissie beperkt. Volgend op een beleidsdiscussie over het scheiden van een gevoelig deel van de bevolking (schoolkinderen) van bronnen (verkeer) wilden beleidsmakers advies over mogelijkheden om door middel van filtering de infiltratie van buitenlucht-

vervuiling naar binnen te beperken, wat resulteerde in het onderzoek in Hoofdstuk 6 (filtering).

Verantwoordelijkheid, dynamiek en cultuur zijn in de Beleidsfase heel anders dan in de drie andere fases van het kader. In de Amsterdamse praktijk zijn GGD medewerkers betrokken bij het maken van beleid, maar zij zijn daar slechts ten dele verantwoordelijk voor. In Hoofdstuk 7 wordt dit aan de hand van de beleidscyclus van Lasswell[192] besproken: Alhoewel GGD medewerkers een belangrijke bijdrage leveren aan de fase 'probleem identificatie', zijn zij nauwelijks van invloed in de fases van beleidsformulering, adoptie en implementatie, waarin beleidsmakers en vooral bestuurders de belangrijkste spelers zijn. GGD medewerkers kunnen een rol spelen in de evaluatie, zoals in Hoofdstukken 5 en 6 zichtbaar is gemaakt. Omdat GGD medewerkers slechts beperkt betrokken zijn in de beleidsfase, kunnen zij er niet voor instaan dat evidentie wordt betrokken in de besluitvorming, wat de beleidsfase waarschijnlijk tot de Achilleshiel van de evidence based milieu-gezondheidszorg maakt.

Een andere uitdaging van evidence based werken betreft de cultuursverschillen in de evaluatie van output tussen wetenschap en praktijk. Waar evaluatie op basis van wetenschappelijke output en impact (publicaties in wetenschappelijke tijdschriften) gebruikelijk is in de universitaire wereld, ligt de focus bij GGD'en op maatschappelijke output, zoals bijvoorbeeld beleidsadviezen, bijdragen aan het publieke debat op informatieavonden of in de media. De belangrijkste financiers in het wetenschappelijke onderzoek erkennen het belang van maatschappelijke impact van wetenschappelijk onderzoek en vragen hiervoor meer aandacht. Recentelijk is een tool ontwikkeld om de maatschappelijke impact van medisch onderzoek te evalueren. We bediscussiëren hoe een dergelijke tool bruikbaar zou kunnen zijn voor de milieu-gezondheidszorg. De Academische Werkplaats Milieu en Gezondheid en de toegewijde financiering blijken een succesvolle methode om tijd en faciliteiten voor evidence based milieu-gezondheidszorg te garanderen (Hoofdstuk 7).

Conclusies

Het onderzoek in dit proefschrift draagt bij aan het begrip van de huidige impact van luchtverontreiniging op de publieke gezondheid in het Westen van Nederland. Ons inzicht in de blootstelling van de bevolking aan verkeersgerelateerde luchtverontreiniging is vergroot. Hogere prevalentie van ziekenhuisopnamen blijkt niet alleen gerelateerd aan kortdurende episodes van luchtvervuiling – zoals bekend uit eerder onderzoek, maar ook aan langdurige blootstelling aan lagere luchtvervuilingsniveaus. Verhoogde risico's op ziekenhuisopname werden gezien bij niveaus ver onder de EU grenswaarde.

Veelbelovende maatregelen om luchtvervuiling aan te pakken bleken slechts beperkt effectief. Een emissiereducerende snelheidsverlaging leidde tot een geringe afname van de concentraties aan de rand van de snelweg. Filters bleken in staat de infiltratie van fijn stof te reduceren, maar de binnenluchtkwaliteit werd nog altijd in belangrijke mate door de buitenluchtkwaliteit beïnvloed.

Chapter 12

About the Author

ADDITIONAL SCIENTIFIC PAPERS PUBLISHED BY THE AUTHOR

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CURRICULUM VITAE

Marieke Bettine Alida Dijkema was born in Hoorn, the Netherlands, on June 20, 1980. After completion of her VWO at RSG Enkhuizen in 1998, she went to Wageningen University to study Environmental Sciences. As part of her MSc in Environmental Health, she did internships at the Public Health Service Arnhem, the Institute for Risk Assessment Sciences at Utrecht University, the National Institute for Public Health and the Environment (RIVM), and the University of the Sunshine Coast, Australia. In 2004 Marieke started her professional career at Public Health Service Amstelland-de Meerlanden as solitary environmental health professional, being in charge of all occurring activities. In 2006 Marieke changed to Public Health Service Amsterdam, where she spent half of her time on the project described in this thesis and half of her time on mostly air pollution related environmental health advice. Marieke will continue working at Public Health Service Amsterdam, where she will work as senior environmental health advisor and project leader for projects in the framework of the European Union Interreg IV-B program and the Academic Collaborative Centre for Environmental Health.

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