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Modelling exposure of the Dutch population to air pollution

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Abstract

The AirPEx (air pollution exposure) model is a mathematical model to estimate the inhalatory exposure of humans to air pollution. The model quantifies individual and population exposures using data from air quality time series and activity pattern surveys. This paper presents the basic exposure concepts of the model, including contact, actual exposure concentration, intake rate, and standardisation. A case study including the exposure to ozone of the Dutch population in the 1991 summer demonstrates the application of the model. © 1998 Elsevier Science B.V. All rights reserved.

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1. Introduction

Toxicological and epidemiological research have indicated that inhalation of certain air pollutants may cause adverse health effects. Air pollution can well be regarded as a constant threat to the public health, because it is present throughout the biosphere. Evaluating the actual risks of air pollution for human health requires a chain of assessments to be performed [1]. This chain includes the following key elements: emission and dispersion of pollutants in the atmosphere, exposure of humans to pollutants, and adverse health effects of the pollutants on target tissues in the human body. Exposure assessment is an important area of investigation in this source–effect

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chain from both research and regulatory points of view [2]. Inhalatory exposure research focuses on the contact of humans with air pollutants and the entry of these pollutants into the human respiratory tract. The principal grounds for studying the inhalatory exposure of humans to air pollutants are formed by the need for realistic exposure/dose estimates to evaluate the health effects of these pollutants. Mathematical modelling has proven to be a useful and cost-effective tool in exposure assessments [2–4]. As an explicit form of quantitative knowledge modelling gives important support in analysing different abatement strategies and economic scenarios in environmental outlooks.

This paper gives an overview of the air pollution exposure (AirPEx) model, a mathematical model for calculating inhalatory exposure of individuals as well as populations from time series of air quality data and activity pattern surveys. The model is intended to support health effect assessments [5]. The aim of this paper is to present the basic concepts and input requirements of the AirPEx model and to illustrate the model's application. An example of application is presented focusing on the exposure of the Dutch population to ozone.

2. Materials and methods

2.1. Individual exposure

Exposure as a general term has been used in different ways in environmental monitoring and health effect studies to indicate contact between a target and a pollutant on a contact surface [3]. Methods to evaluate and assess inhalatory exposure quantitatively require mathematical definitions of exposure measures [2,3,6]. We contemplate four components in quantifying exposure: potential exposure, contact, actual exposure, and intake [6]. The potential exposure concentration, $E(x,t)$ is equal to the concentration in the air medium, which may vary in space, x, and in time, t. Each person (p) moves through space and time. If this path, $x_p(t)$, can be specified, the actual exposure concentration $E(x_p(t),t)$ that this person experiences can be determined. The average exposure during the exposure period, T, is evaluated from a time series of the actual exposure concentration [4]:

$$\overline{E_p} = \frac{1}{T}\int_T E\left(x_p(t),t\right)\mathrm{d}t \tag{1}$$

.

Another way of analysing a time series of actual exposure concentrations is to determine the time fraction, τ, that the actual exposure concentration is above a certain critical level, C_c

$$\tau = \frac{1}{T}\int_T P(t)\mathrm{d}t, \quad \text{with } P(t)=1 \text{ if } E\left(x_p(t),t\right) \geq C_c \text{ and } P(t)=0 \text{ if } E\left(x_p(t),t\right) < C_c \tag{2}$$

.

Entry of pollutants into the human body is depicted by the intake rate. The rate of intake can be approximated by multiplying the actual exposure concentration with the ventilation rate:

$$R(t) = I_{\mathrm{m}}(t)\,\eta E\left(x_{\mathrm{p}}(t), t\right) \tag{3}$$

.where $R(t)$ is the intake rate, t is time, $I_{\mathrm{m}}(t)$ is the rate at which the medium (air) is inhaled (ventilation rate), and η is the inhalability. Inhalability is the intake efficiency with which pollutants are inspired into the respiratory tract [7]. The inhalability is specific for each compound studied. The fourth component is the calculation of the total intake, which is given by the integral of Eq. (3) over an interval T:

$$U_T = \int_T R(t)\,\mathrm{d}t \tag{4}$$

.

Additional exposure measures can be derived from the above framework by standardising total intake to the exposure period and unit of receiving target entity [2,7]. As such, average intake rate is defined as intake divided by the period T:

$$\overline{R} = \frac{U_T}{T} = \frac{1}{T}\int_T R(t)\,\mathrm{d}t \tag{5}$$

.If the area of the air–tissue interface in the lungs, A, is known, then standardisation to area lung interface is possible [8]:

$$\overline{S} = \frac{U_T}{TA} = \frac{1}{TA}\int_T R(t)\,\mathrm{d}t \tag{6}$$

.

The exposure measures given by Eqs. (1) and (2) do not require any knowledge of the breathing physiology and anatomy of the target (ventilation rate, lung area), Therefore, these measures give a first impression of the exposure of humans to air pollution. However, breathing parameters are indispensable for quantifying the entry of pollutants into the respiratory tract, as given in Eqs. (3)–(6).

2.2. Population exposure

A population can be defined as a group of individuals with one or more common characteristics. One approach to estimate exposure of a population is to determine distributions of individual exposures in the population [2,3]. For an exposure measure x, we can estimate individual exposures for N persons taken from a certain population to compose a cumulative frequency (CF) distribution of these values. Dividing the cumulative frequencies by N gives the normalised cumulative frequency (NCF) distribution. If N is large and all persons are sampled randomly from the population, then the NCF distribution of the sample approximates the cumulative probability density function of the population. Analysis of the NCF distribution in terms of percentiles will yield information on the central tendency in the distribution of the population exposure, e.g. as indicated by the median. The variation and extremes in the exposure levels are quantified by the 10 and the 90% percentiles of the distribution compared to the median.

2.3. AirPEx model

The AirPEx model calculates individual and population exposures from time series of air quality data. The model employs a microenvironment approach, which divides space into a finite number of compartments. The potential exposure concentrations in the microenvironments are calculated as a function of time from air quality data obtained at monitoring stations. For this purpose, descriptive linear relations can be used, e.g. based on indoor/outdoor concentration ratios. The division of time into finite time steps is tuned to the time resolution of the air quality data.

The paths of individuals through the microenvironments, as well as their ventilation rate are derived from activity pattern surveys. These surveys have proven to be of critical importance in exposure assessments, as they give detailed data on the variation in the whereabouts and activities of humans in different microenvironments during the day [9]. Currently, AirPEx connects to a database containing the results of a large activity pattern survey among the Dutch population in 1994, including daily activity patterns (15 min time resolution) of 4985 individuals. For each individual, the location in different microenvironments as well as the level of activity were recorded for a period of 24 h. The periods were selected by a stratified sampling scheme. Weights were assigned to each activity pattern to compensate for deviations of the age distribution of the population in the survey from the general age distribution obtained in the Dutch mini-census of 1994. The database contains additional individual data including age, body mass, smoking habit, and the presence of pulmonary and cardiovascular diseases. The location of each individual in different microenvironments during the day is used to estimate the actual exposure concentration as a function of time. The ventilation rate as a function of time is derived from body mass and variation of the level of activity [10]. A correction factor based on data presented in clinical studies [11] accounts for relatively higher ventilation rates of smokers and subjects with compromised airways. Effective air–tissue interface areas (Eq. (6)) in the airways of the subjects are estimated from their age [12] and their health status, with potential reductions due to pulmonary disease [13]. It should be stressed that exposure models are always heavily depending on the quality of the input data [2]. Accordingly, the AirPEx model cannot operate well when activity pattern surveys and times series of air quality data with sufficient time resolution are missing.

The model has been implemented for use as a Windows 3.1 computer program. The program is versatile in displaying and analysing various exposure measures. This user interface allows interactive usage of the model to study different air pollution scenarios and to estimate exposure distributions in different populations. One important feature is the possibility to analyse the socio-demographic characteristics of the individuals that experience the highest exposures. This type of analysis enables the identification of groups at greater risk than the average.

2.4. Example: exposure of the Dutch population to ozone

We estimated the exposure of the Dutch population to ozone to demonstrate the application of the AirPEx model in inhalatory exposure analysis. The objective was to

evaluate distributions of various exposure measures as given by Eqs. (1)–(6) and to make a comparison between them. The basic boundary condition for the calculations was a time series of hourly averaged ozone concentrations recorded in the South East of the Netherlands during the 1991 summer (100 days starting from day 150). Eleven microenvironments were distinguished, including seven types of indoor environments and four types of outdoor environments. Concentrations of ozone in each environment were calculated from previously reported indoor/outdoor ratios and the level of urbanisation of the outdoor microenvironments. Concentrations of ozone in indoor environments are typically 0.1 to 0.5 times the outdoor concentration [5,14]. In the Netherlands, ozone concentrations in urban outdoor environments tend to be slightly lower than concentrations in the suburban and rural areas, while concentration in transit areas are much lower compared to the suburban and rural areas. This is probably caused by ozone scavenging by nitric oxide emitted from motor vehicles in areas with much traffic [15]. The above mentioned differences in ozone concentrations formed the basis of the ozone indoor/outdoor ratios used in AirPEx. We performed individual exposure calculations for 1480 individuals with different activity patterns recorded during the summer months at daily maximum temperatures below 25°C. As the number of activity patterns is large, the NCF distributions of the exposure measures can be considered as fairly good approximations of the distributions in the Dutch population. The distributions were analysed to identify the subjects with the highest exposures. The analyses included determination of the contribution of groups potentially at risk (children < 10 years, subjects with compromised airways, smokers and the elderly > 65 years) above the 90% percentile of the NCF distributions compared to the contribution of these groups in the total of 1480 individuals.

3. Results and discussion

The results of the exposure calculations are displayed in Fig. 1. The frequency distribution and the NCF distribution of the hourly averaged ozone concentrations monitored outdoors are given in Fig. 1a. The shape of the distribution results from repeated diurnal patterns with the highest concentrations in the late afternoon and lowest concentration in the early morning (pattern not shown). In many countries with moderate climates people spend most of their time indoors [16], where ozone concentrations are lower than outdoors [14]. Accordingly, the values in the frequency distribution of the average actual exposure concentration of the population (Fig. 1b) are lower than the median value of the ozone concentrations in the outdoor air (Fig. 1a). Nevertheless, during outdoor stays in the afternoon and early evening individuals may still be subjected to short periods of high ozone concentrations. This can be seen in Fig. 1c, which displays the time fraction that the individuals in the population are exposed to actual ozone concentrations above an arbitrary critical value of 100 μg m^{-3}. The actual exposure concentration patterns can be processed using Eq. (5) to yield a distribution of the average intake rate (Fig. 1d). This distribution is much more skewed than the distribution of the average actual exposure concentration (Fig. 1b). The long tail in Fig. 1d is mainly related to individuals in the population with a higher ventilation rate than

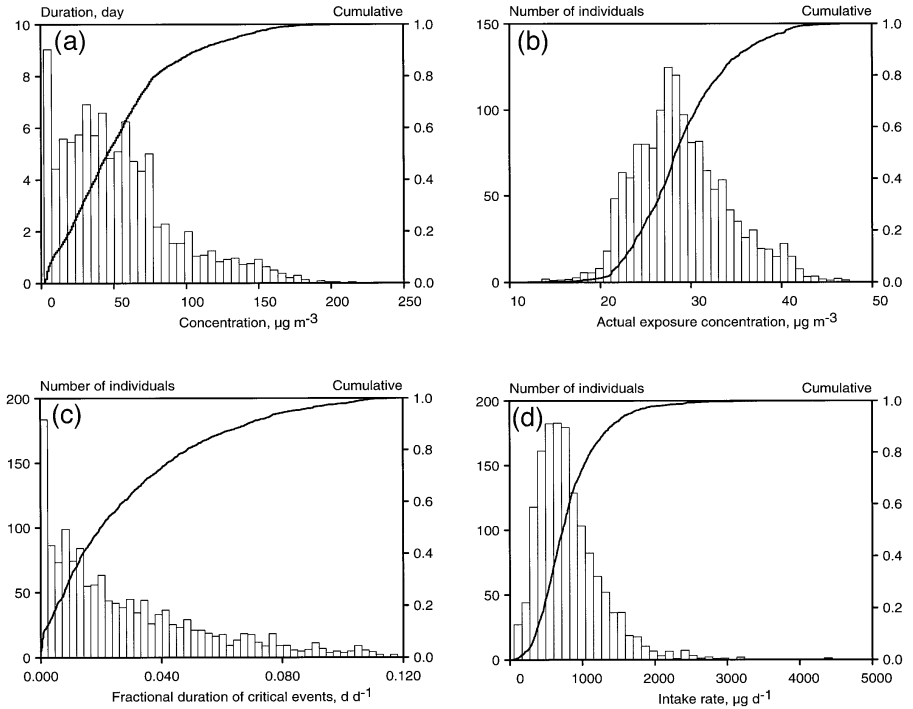

Fig. 1. Exposure of the Dutch population in SE Netherlands to ozone, summer 1991. Frequency distributions (bars, left axes) and NCF distributions (lines, right axes) of (a) total duration of hourly averaged concentrations at monitoring station, (b) average actual exposure concentrations for 1480 individuals, (c) fraction of time for 1480 individuals at hourly actual exposure concentrations above an arbitrary critical level of 100 μg m^{-3}, and (d) average intake rates of 1480 individuals.

average (smokers, subjects with compromised airways, individuals with a high level of physical exercise) that spend time outdoors during periods when ozone concentrations are at their maximum.

A summary of all exposure measures as defined in Eqs. (1)–(6) is given in Table 1, including the 50 and 90% percentiles. From the exposure levels at these percentiles it can be seen that the distributions of $\overline{R}$ and $\overline{S}$ are much more skewed than the distribution of $\overline{E}_p$. Table 1 also gives values of the abundance index (AI), which is defined as the ratio of the fraction of a certain group of subjects in the 10% of the highest exposures and the fraction of this group in the total population. A value of the AI higher than one indicates that persons in this group are more frequently present among the 10% of the highest exposures than among the total population analysed. The results show that when evaluating the average actual exposure concentration, $\overline{E}_p$, none of the potential risk groups in Table 1 predominantly belongs to the highest exposures. The average actual exposure concentrations only incorporates differences in path through space between subjects. If differences between breathing rate are also included, such as in the average intake rate $\overline{R}$, we can see that the relatively higher ventilation

Table 1
Exposure levels at 50 and 90% percentiles and abundance indices (AI) for different exposure measures calculated by the AirPEx model

Percentiles and indices	Exposure measure[a]		
	$\bar{E}_p$	$\bar{R}$	$\bar{S}$
50% percentile	28.3 μg m^{-3}	729.3 μg day^{-1}	10.1 μg m^{-2} day^{-1}
90% percentile	36.9 μg m^{-3}	1389 μg day^{-1}	23.3 μg m^{-2} day^{-1}
AI, children <10 years	0.901	0.000	2.084
AI, elderly >65 years	0.385	0.615	0.615
AI, compromised airways	0.592	2.053	9.408
AI, smokers	1.008	2.238	1.088

[a] $\bar{E}_p$ = average exposure concentration, $\bar{R}$ = average intake rate, $\bar{S}$ = average intake rate standardised to air–tissue interface.
The abundance index (AI) is defined as the ratio of the fraction of a certain group of subjects in the 10% of the highest exposures and the fraction of this group in the total number of exposures.

rates attributed to smokers and subjects with compromised airways [11] causes abundant presence in the 10% of the highest exposures (AI >2), while the opposite holds for children. Division of $\bar{R}$ by the air–tissue interface area in the lungs gives $\bar{S}$, which expresses the potential burden of the deep lung tissues. Children and subjects with compromised airways have relatively low effective air–tissue interface areas compared to the others [12,13], which results in high AI values for them. For the elderly it appears that none of the results indicate that they are subjected to high exposures.

From the above analysis of the subjects involved in the upper tails of the distribution of the three exposure measures it can be concluded that it is important to choose a relevant exposure measure. If contact between pollutant and exposed subject is aimed at, the average actual exposure concentration, $\bar{E}_p$ is fair. For a mass balance, where interest is focused on the total amount of pollutant that is inhaled the average intake rate, $\bar{R}$, may be of use. Effect relevant doses require the standardisation of the average intake rate to unit of target tissue. For example, doses can be standardised to area air–tissue interface in the lung, which gives $\bar{S}$ and expresses the potential burden of the deep lung tissues.

References

[1] J.V. Hall, Atmos. Environ. 30 (1996) 743–746.
[2] P.B. Ryan, J. Expos. Anal. Environ. Epidemiol. 1 (1991) 453–474.
[3] N. Duan, in: Total exposure assessment methodology. A new horizon, EPA/A and WMA, Pittsburg, PA, 1990, pp. 166–195.
[4] W.R. Ott, J. Toxicol. Clin. Toxicol. 21 (1984) 97–128.
[5] H.J. Van Scheindelen, M. Marra, P.J.A. Rombout, Exposure model AirPEx: Development and model description (In Dutch), Report No. 623710003, National Institute of Public Health and the Environment, Bilthoven, NL, 1995.
[6] M.P. van Veen, Risk Anal. 16 (1996) 323–330.
[7] ICRP, Human respiratory tract model for radiological protection, ICRP Publication 66, Annals of the ICRP 24(1-3), Elsevier, Oxford, UK, 1994, p. 42.

[8] A.M. Jarabek, M.G. Menache, J.H. Overton, M.L. Dourson, F.J. Miller, Health Phys. 57 (1989) 177–183.
[9] P.L. Jenkins, T.J. Phillips, E.J. Mulberg, S.P. Hui, Atmos. Environ. 26A (1992) 2141–2148.
[10] M.T. Kleinman, J. Expos. Anal. Environ. Epidemiol. 1 (1991) 309–325.
[11] M.J. Tobin, T.S. Chadha, G. Jenouri, S.J. Birch, H.B. Gazeroglu, M.A. Sackner, Chest 84 (1983) 286–294.
[12] C.P. Yu, G.B. Xu, J. Aerosol Sci. 18 (1987) 419–429.
[13] F.J. Miller, S. Anjilvel, M.G. Menache, B. Asgharian, T.R. Gerrity, Inhalation Toxicol. 7 (1995) 615–632.
[14] J.E. Yocom, J. Air Pollut. Control Assoc. 20 (1982) 500–520.
[15] T.R. Johnson, J. Expos. Anal. Environ. Epidemiol. 4 (1995) 551–571.
[16] D.W. Dockery, J.D. Sprengler, Atmos. Environ. 15 (1981) 335–344.