

# Traffic aerosol lobar doses deposited in the human respiratory system

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Abstract Aerosol pollution in urban environments has been recognized to be responsible for important pathologies of the cardiovascular and respiratory systems. In this perspective, great attention has been addressed to Ultra Fine Particles (UFPs<100 nm), because they efficiently penetrate into the respiratory system and are capable of translocating from the airways into the blood circulation. This paper describes the aerosol regional doses deposited in the human respiratory system in a high-traffic urban area. The aerosol measurements were carried out on a curbside in downtown Rome, on a street characterized by a high density of autovehicular traffic. Aerosol number-size distributions were measured by means of a Fast Mobility Particle Sizer in the range from 5.6 to 560 nm with a 1 s time resolution. Dosimetry estimates were performed with the Multiple-Path Particle Dosimetry model by means of the stochastic lung model. The exposure scenario close to traffic is represented by a sequence of short-term peak exposures: about  $6.6 \times 10^{10}$  particles are deposited hourly into the respiratory system. After 1 h of exposure in proximity of traffic,  $1.29 \times 10^{10}$ ,  $1.88 \times 10^{10}$ , and  $3.45 \times 10^{10}$  particles are deposited in the head, tracheobronchial, and alveolar regions. More than 95 % of such doses are represented by UFPs. Finally, according to the greater dose estimated, the right lung lobes are expected to be more susceptible to respiratory pathologies than the left lobes.

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# Introduction

Aerosol pollution in urban environments has been recognized to be responsible for important pathologies of the cardiovascular (Brook et al. 2002; Simkhovich et al. 2008) and respiratory systems (Delfino et al. 1998; Von Klot et al. 2002), it has also been associated with increased mortality and hospital admissions (Dominici et al. 2006). Directive 2008/50/EC states that for fine particulate matter (PM2.5), there is still no identifiable threshold below which PM2.5 would not pose a risk. Therefore, a general reduction of its concentrations in the urban background should be pursued to ensure that large sections of the population benefit from an improved air quality (Avino et al. 2002, 2004, 2015a; Avino and Manigrasso 2008).

More recently, the International Agency for Research on Cancer (IARC 2015) has considered outdoor pollution as a leading environmental cause of cancer deaths. Furthermore, particulate matter has been classified as carcinogenic to humans (Group 1). Within this context, the importance of aerosol size distribution measurements resides in that the doses deposited in the human respiratory system strictly depend on the particle sizes.

In this perspective, great attention has been addressed to Ultra Fine Particles (UFPs<100 nm) (Donaldson et al. 2001; Oberdörster et al. 2005; Fanizza et al. 2010), since they efficiently penetrate into the respiratory system and are capable of translocating from the airways into the blood circulation (Oberdörster et al. 2005. Moreover, the knowledge of their distribution in the respiratory tree is important because the airway

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pathologies caused by deposition of particulate matter have often been reported to occur at specific sites in the lung, particularly within specific lobes (Winkler-Heil and Hofmann 2009). Parkash (1977) observed that the right lung is more often the seat of carcinoma than the left lung, and the upper lobes more often than the lower lobes. The author speculated that since the right bronchus is wider, shorter, and runs almost as a continuation of the trachea, there is a greater chance of more particles being deposited in the right lung than in the left lung as a whole, which is likely to cause a higher frequency of malignancies. For this reason, it is important to assess particle deposition in the respiratory tree, considering the differences between and within the lung lobes. This study relies on time and size-resolved aerosol measurements and focuses on that task by estimating aerosol doses deposited in the lung lobes.

# Materials and methods

#### Aerosol measurements

Aerosol number-size distributions were measured by means of a TSI Fast Mobility Particle Sizer (model 3091, FMPS, Shoreview, MN, USA). The instrument counts and classifies particles, according to their electrical mobility, in 32 size channels, in the range from 5.6 to 560 nm, with a 1 s time resolution. FMPS operates at a high flow rate (10 L min<sup>-1</sup>) to minimize UFP diffusion losses. It operates at ambient pressure to prevent evaporation of volatile and semivolatile particles (TSI 2009; Manigrasso et al. 2009; Avino et al. 2013a). The performance of the FMPS was investigated by Jeong et al. (2009) by comparison with a Scanning Mobility Particle Sizer (SMPS). The authors evaluated that the SMPS number concentration, in the size range from 6 to 100 nm, is about 34 % lower than the FMPS measurements, due to the diffusion losses of particles in the SMPS. The diffusion loss-corrected SMPS number concentration is on average about 15 % higher than the FMPS data.

Aerosol measurements were carried out on a curbside in downtown Rome. The site  $(41^{\circ}53'46'' \text{ N}, 12^{\circ}29'46'' \text{ E})$  is located in an area characterized by a high density of autovehicular traffic, in a narrow double lane street (street width, *W*, of about 8 m), with high buildings on both sides (average height, *H*, of about 25 m). Such a street can be considered as a street canyon, as the aspect ratio *H*/*W* is about 3:1.

An hour of aerosol data collected during the morning traffic peak period has been selected to carry out dosimetry estimates. During such hour, the values of temperature and of relative humidity (average±standard deviation) were  $15.9\pm$  0.2 °C and  $73\pm1$  %, respectively.

#### Particle dose evaluation

Particle deposition in the human respiratory system was evaluated using the Multiple-Path Particle Dosimetry Model (MPPD v2.1, ARA 2009), which calculates the deposition and clearance of mono and polydisperse aerosols, from ultrafine to coarse particles in the respiratory system of humans and rats (Anjilvel and Asgharian 1995; Price et al. 2002; Manigrasso et al. 2015a, b). The model includes single and multiple-path methods to calculate airflow and aerosol deposition. Dosimetry estimates were made by means of the stochastic lung model, because it provides a more realistic lung geometry than the symmetric one considered in the ICRP model (ICRP 1994). Symmetric models do not consider the variability of the structural components of the lung. They assume that all airways in the same generation have the same values of diameter length and branching angle. The stochastic lung model takes into account (Koblinger and Hofmann 1985; 1990) the asymmetry of the lung structure and track each inhaled particle through a pathway randomly selected by means of the Monte Carlo method.

In the MPPD model, the ten stochastic lungs proposed by Asgharian et al. (2001) are ordered in a size (total number of airways) from the smallest to the largest and the approximate size percentile of each lung is provided. The 60th percentile human stochastic lung was considered in this study. The following settings were considered in the application of the MPPD model: (i) a uniformly expanding flow, (ii) an upright body orientation, and (iii) nasal breathing with a 0.5 inspiratory fraction and no pause fraction. Moreover, the following parameters were used for a Caucasian adult male under light work physical activity, based on the ICRP report (ICRP 1994): (i) a functional residual capacity (FRC) of 3300 mL, (ii) an upper respiratory tract (URT) volume equal to 50 mL, (iii) a 20 min<sup>-1</sup> breathing frequency, and (iv) an air volume inhaled during a single breath (tidal volume,  $V_T$ ) of 1.25 L.

Particle number regional deposition doses in the head (H), trachebronchial (TB), and alveolar (AI) left-upper (LU), left-lower (LL), right-upper (RU), right-middle (RM), and right-lower (RL) lung lobes were calculated as follows:

(i) instant regional doses:

$$D^{R}(t) = \sum_{i=1}^{32} F_{i}^{R} \times C_{i}(t) \times V_{T}$$
(1)

where t is the time,  $F_i^R$  is the deposition fraction at a given R region (H, TB, and AI entire lung) of particles classified in the *i*<sup>th</sup> size class (calculated by the MPPD model),  $C_i(t)$  is the concentration of particles in the *i*<sup>th</sup> size class averaged over a single respiratory act time interval. The summation in

Fig. 1 Temporal trends of total (5.6–560 nm) particle number concentration and of UFP (as %) contribution in a street canyon in downtown Rome



Eq. 1 is carried out over the 32 FMPS size classes. (ii) one hour regional cumulative doses:

$$D_h^R = \sum_{t=0}^{3600} D^R(t)$$
 (2)

where *R* stands for the head (H), tracheobronchial leftlower lobe (TBLL), tracheobronchial right-upper lobe (TBRU), tracheobronchial right-middle lobe (TBRM), tracheobronchial right-lower lobe (TBRL), alveolar left-upper lobe (ALU), alveolar left-lower lobe (ALL), alveolar right-upper lobe (ARU), alveolar right-middle lobe (ARM), and alveolar right-lower lobe (ARL).

The summation in Eq. 2 is carried out over a 1 h (3600 s) time interval divided in 3 s time segments, as the duration of respiratory act.

(iii) cumulative doses deposited at the end of an exposure period  $(D^{R}_{c})$  in head (H), tracheobronchial (TB) and alveolar (AI) regions as function of particle diameter and time. For the TB and AI regions the left-upper (LU), left-lower (LL), right-upper







Fig. 3 One hour regional cumulative doses  $(D^R_h)$  in the H, TBLU, TBLL, TBRU, TBRM, TBRL, ALU, ALL, ARU, ARM, and ARL regions, after 1 h exposure in traffic proximity. For the acronyms, see text

(RU), right-middle (RM), right-lower (RL) lung lobes were considered.

$$D_c^R = \sum_{t=0}^t D^R(t) \tag{3}$$

The summation in Eq. 3 is carried out over variable time intervals ranging from 0 to 3600 s increased by 3 s steps.

# **Results and discussion**

Figure 1 shows the temporal trend of total (5.6–560 nm) particle concentration throughout a 12 h time span together with the percent contribution of UFPs. We selected one-hour size distribution data (the *yellow box* in Fig. 1) to carry out dosimetry estimates. During such time interval, total particle concentration ranged from  $2.6 \times 10^4$  to  $5.8 \times 10^5$  particles×cm<sup>-3</sup>, with an average value of  $7.2 \times 10^4$  particles×cm<sup>-3</sup>. The upper limit of such an interval represents the spike concentration due to the instant exhaust emission of auto vehicles passing by the measurement site, whereas the lower limit represents the baseline value for the specific street canyon and time of day considered. In particular, during such 1 h time span, the UFP contribution is on average 95 %, whereas during nocturnal hours (1–4 a.m.) it drops down to 85 %.

Figure 2 describes the temporal evolution of aerosol number-size distribution (5 min averages of 1 s time resolution

data) as a function of time throughout the 12 h time period (Fig. 2a) and the average size number-distribution during the 1 h time interval considered for dosimetry estimates (Fig. 2b). In particular, during such period, a trimodal distribution with modes at 10, 19, and 34 nm was observed.

Based on such data, one-hour regional cumulative doses  $(D_h^R)$  deposited in the head, tracheobronchial and, alveolar regions are reported in Fig. 3. After 1 h exposure in proximity of traffic  $1.29 \times 10^{10}$ ,  $1.88 \times 10^{10}$ , and  $3.45 \times 10^{10}$  particles are deposited in the head, tracheobronchial, and alveolar regions. More than 95 % of such doses are represented by UFPs. Both in tracheobronchial and alveolar regions, doses in RU lobes are about twice as much as those in LU lobes, 24 and 31 % more, respectively, in TBRL than in TBLL and in PRL than in PLL lobes. Compared with the lung lobes, the highest dose was deposited in the H region, about 24 % of the total particle dose.

The relevance of these data resides in that they rely on aerosol measurements carried out in conditions that closely reproduce the real exposure of individuals in proximity of traffic, accounting for the rapid evolution of traffic aerosol. On the contrary, the regulatory approach currently adopted to estimate human exposure to airborne particles is based on the measurement of a 24-hour average mass concentration (PM10, PM2.5) at fixed outdoor monitoring stations (Directive 2008/50/EC; NEPC 1998, 2003; USEPA 2006). Such data are not representative of real outdoor exposure, because particle concentrations, high at the emission point,



Fig. 4 Instant regional doses  $(D^{R}(t))$  deposited in H, TB, and Al regions as a function of time. Figure **b** is a highlight of Fig. **a**. For the acronyms, see text

rapidly decay with the distance from the source (Marini et al. 2015). Air-quality standards are not defined in terms of number and surface area. Therefore, data from monitoring stations cannot be used to evaluate the doses deposited in the respiratory system. Moreover, short-term dose effects cannot be accounted for by means of average measurements. In fact, close to traffic, the exposure scenario is represented by a sequence of short-term peak exposures.

Such issue is addressed by Fig. 4a, where instant regional doses  $(D^{R}(t))$  deposited in the head, tracheobronchial, and alveolar regions are reported as a function of time throughout the same time interval considered to estimate the cumulative doses  $(D^{R}_{h}, \text{Fig. 3})$ . In this graphic, each data point represents the total particle number deposited after a tidal volume inhalation. In proximity to traffic, within a single respiratory act, the particle dose deposited in the H, TB, and AI regions may increase by about 500 % as shown in Fig. 4b.

In the contour plots of Figs. 5, 6, and 7, the cumulative doses of particle deposited  $(D^{R}_{c})$  are reported as a function of time and particle diameter for the head region and the five lung lobes both in the tracheobronchial and in the alveolar regions, respectively.

At the end of the 1 h exposure period, the size distributions of particle-deposited doses follow a trimodal distribution with modes at 10, 16, and 29 nm for the H and TB region and 10,



**Fig. 5** Cumulative dose  $(D^{R}_{c})$  for H region, as a function of time and particle diameter

19, and 29 nm for the alveolar region. Deposited in the H region are  $1.5 \times 10^9$ ,  $8.9 \times 10^8$ , and  $7.0 \times 10^8$  particles, respectively for 10, 19, and 29 nm particle diameters. At modal diameters, the particle-deposited doses are in the range of  $2.1 \times 10^8 - 5.0 \times 10^8$ ,  $1.4 \times 10^8 - 3.3 \times 10^8$ , and  $1.2 \times 10^8 - 2.7 \times 10^8$ , respectively for the TB region and  $2.8 \times 10^8 - 6.9 \times 10^8$ ,  $3.0 \times 10^8 - 7.0 \times 10^8$ , and  $3.2 \times 10^8 - 8.0 \times 10^8$ , respectively for the Al region.

It is worth observing that the mode at 10 nm represents the main contribution to the cumulative dose deposited in H and



Fig. 6 Cumulative dose  $(D^R_c)$  for lung lobes in TB region, as a function of time and particle diameter



**Fig.** 7 Cumulative dose  $(D^{R}_{c})$  for lung lobes in Al region, as a function of time and particle diameter

TB regions (Figs. 5 and 6), whereas for the AI region, the greater contribution is due to the mode at 29 nm (Fig. 7). In particular, for the daytime and traffic site considered, about 28 % of the particles below 10 nm are deposited in the head region. These particles are formed by the nucleation of semivolatile organic compounds in vehicle exhaust. Their persistence in the atmosphere is relatively low with respect to larger-sized particles. Then, they are predominant during the day when freshly formed and decrease significantly at night when traffic is less intense (Manigrasso and Avino 2012). They are responsible for the high frequency contribution observable in the temporal trend of total particle concentration (Fig. 1) (Avino et al. 2011, 2013b, 2015b; Manigrasso et al. 2013).

From the health point of view, UFPs may translocate from the blood circulation to other organs such as the liver, spleen, kidneys, heart, and brain (Peters et al. 2006). Indeed, the health relevance of the high UFP dose estimated for the head region (Fig. 3), in particular for the small-size fraction below 10 nm (Fig. 5) is linked to its ability to reach to brain. The central nervous system can be targeted by airborne UFPs. The most likely mechanism is from deposits on the olfactory mucosa of the nasopharyngeal region of the respiratory tract and subsequent translocation via the olfactory nerve (Oberdörster et al. 2004). Particles, on entering the brain, may possibly entail neurodegenerative consequences. Histological evidence of neurodegeneration has been reported in both canine and human brains exposed to high ambient PM levels, suggesting the potential for neurotoxic consequences (Terzano et al. 2010).

# Conclusions

Aerosol number-size distributions were measured with 1 s time resolution, on a curbside in a street canyon. Based on these, the aerosol doses deposited in the respiratory system were estimated for individuals exposed in proximity of traffic. During peak traffic hours, after 1 h exposure about  $6.6 \times 10^{10}$ particles were deposited into the respiratory system. Such dose was almost entirely made of UFPs and was asymmetrically deposited into the lung lobes: more in the right than in the left lung lobes. According to the greater dose estimated, the right lung lobes are expected to be more susceptible to respiratory pathologies than the left lobes. The cumulative doses deposited into the respiratory system followed a trimodal distribution with modes between 10 and 29 nm. For deposited particles below 10 nm, the highest dose was estimated for the head region, where from the olfactory mucosa they may possibly translocate to brain.

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