Factors controlling the lung dose of road traffic-generated sub-micrometre aerosols from outdoor to indoor environments

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Abstract
Estimates of lung dose of submicron particles in the human respiratory system play an essential role in assessing health outcomes of aerosol exposure. The objectives of this study are to calculate the regional lung dose of traffic-generated particles by different metrics from exposure in outdoor and indoor environments and to identify main factors determining the lung dose. Particle number size distributions were collected in both indoor and outdoor environments in two unoccupied apartments from 22nd February to 30th April 2012 in Bologna, Italy. The whole lung doses of outdoor aerosols by number, surface area and mass at a traffic site were $1.0 \times 10^{10}$ particles/h, 130 mm$^2$/h and 1.9 $\mu$g/h, respectively. A majority of particles by number and surface area were found to deposit in the alveolar region (65%). The physical properties of particles such as shape, hygroscopicity and density play an important role in the calculation of surface area and mass dose due to shifting of the lung deposition curve. Particle number can predict well the regional dose by number, while PM$_{2.5}$ and PM$_{10}$ are good metrics for the prediction of surface area and mass dose. Good correlations between NO$_x$ and the surface areas and mass dose ($r^2 \sim 0.8$) and number dose ($r^2 \sim 0.7$) of submicron aerosols suggest that NO$_x$ may be a good indicator for predicting the health outcomes of traffic-generated aerosols. The doses of indoor sub-micrometre aerosols are less than those of outdoor aerosols by factors of 4.1 (for number), 2.7 (for surface area) and 2.1 (for mass). Due to traffic emissions, the lung dose of outdoor aerosols in the traffic area was much higher than that in the residential area by 5 times for number and surface area and 2 times for mass. A different exercise level (standing, walking, running and cycling) has only a slight influence on the whole lung deposition fraction of submicron aerosols but has a large effect on the dose due to differences in ventilation rate.

Highlights
• Factors controlling the lung dose of road traffic aerosols are investigated.
• Indicators for monitoring of the lung dose rate of particles are evaluated.
• Exposure to traffic-generated particles from outdoor and indoor environments is studied.
• Increment of lung dose due to road traffic emissions is quantified.

Keywords Lung dose · Aerosols · Traffic emissions

Introduction
Road traffic is well-known as a major source of aerosol in cities. Recent source apportionment studies have found that road traffic accounts for 56.5 and 10.3% of particles by number and PM$_{2.5}$ mass in an urban background area in London, UK, respectively (Vu et al. 2016; Crilley et al. 2017). In addition, exposure to road traffic-generated aerosols is consistently associated with adverse health effects (Künzli et al. 2000; Weinmayr et al. 2015). Künzli et al. (2000) estimated the impact of outdoor traffic-related particulate matter on public health in three European countries and reported that motorised traffic was responsible for 20,000 mortality cases/year. Therefore, road traffic-generated aerosols and their health impacts have been of much concern in recent years.

In order to determine the health outcomes of aerosol exposure, the majority of epidemiological studies have used the particle mass (PM$_{2.5}$ or PM$_{10}$) only as an indicator (Harrison et al. 2010). However, growing evidence attributes a more
important role to sub-micrometre aerosols, which are most commonly measured in terms of their number concentrations, in the toxic effects of aerosols. This is due to their large surface area, oxidative capacity and potential for radical species formation that can lead to cellular DNA damage or induce inflammatory effects (Atkinson et al. 2010; Kreyling et al. 2004, 2006). In addition, when a particle enters into the lung, its deposition fraction is governed by many factors such as particle properties (size, shape, chemical properties), breathing pattern and lung structure, leading to a different deposition distribution in the respiratory system for different metrics (number, surface area and mass). Consequently, the estimation of the deposited dose of particles in the human respiratory system (regional lung dose) plays a vital role in determining the particle-induced biological response in toxicological studies and assessment of the health risk of aerosols in epidemiological studies. Harrison et al. (2010) indicate that assessing health outcomes based on particle mass metrics such as PM$_{2.5}$ likely underestimates the public health impacts, and suggest that the regional lung dose could be a better indicator for health impact assessment than their exposure concentration.

The lung dose of traffic-generated aerosols has been estimated in several previous studies (Londahn et al. 2009; Manigrasso and Avino 2012; Rissler et al. 2012; Vu et al. 2016). Londahn et al. (2009) measured the total lung deposition dose in nine healthy subjects breathing by mouth on a busy street in Copenhagen and found that the deposited dose of traffic exhaust particles was much higher (16 times by number and 3 times by surface area) than for the residential biofuel combustion particles. In a recent study, Manigrasso et al. (2017) measured traffic aerosol lobar doses and found that $3.45 \times 10^{10}$ particles could deposit in the human alveolar region after 1 h of exposure to aerosols in a high-traffic urban area. Buonanno et al. (2011) reported that transportation is one of the major contributions to alveolar and tracheo-bronchial particle number deposition in children (19 and 20%, respectively).

In our previous work, we found that traffic-related aerosol is not only a major contributor to aerosol number concentration in cities, but also contributes the main fraction of number dose to the lung due to their high concentration and deposition fraction (Vu et al. 2016). However, the factors controlling the regional dose of traffic-generated aerosols were not fully investigated in these previous studies. In addition, although people spend most of their time indoors, the dose of traffic-related aerosols in a house has yet to be well quantified, leading to a possible underestimation of the contribution of traffic to the total lung dose.

This study first estimates the regional lung dose of aerosols collected in a roadside area in Bologna, Italy. It then discusses the main factors governing the lung dose of particles and identifies the implications for lung dose. Finally, it compares the lung dose of particles released from vehicles due to exposure in outdoor and indoor environments and the dose increment of aerosols at the traffic site caused by road traffic emissions.

Methodology

Study design

Particle number size distributions (PNSD) were collected using a Fast Mobility Particle Sizer (FMPS) system (TSI, model 3091, 5.6–560 nm) in both the indoor and outdoor environments of two unoccupied apartments located in a busy traffic area (31,000 vehicles/day and close to a two-way street canyon) and a typical residential area in Bologna (Italy) in two sampling campaigns for the period from 22nd February to 30th April 2012. The FMPS system was deployed inside the first floor apartment. It was connected to a switching valve to measure both indoor and outdoor size distributions every 10 min, and a constant air exchange rate of 0.5 h$^{-1}$ was controlled by a novel mechanical system. The details of the sampling campaign were described in our previous work (Vu et al. 2017; Zauli Sajani et al. 2015). An hourly dataset of NO, NO$_2$ and NO$_x$ and daily PM$_{2.5}$ and PM$_{10}$ were collected at the Porta San Felice monitoring site (longitude 11.329, latitude 44.500) which is located in the urban traffic area and close to our traffic-influenced sampling site. Due to high uncertainties in measurements of particles in the smallest size bins, in this study, we only consider particles with the diameters ranging from 13 to 560 nm.

Lung dose calculation

Lung deposition model

The deposited fractions of aerosols in the human respiratory system were calculated based on the human respiratory tract model for radiological protection (ICRP model) from the International Commission on Radiological Protection Publication 66 (ICRP 1994). The deposited fractions of aerosol in the human respiratory tract are mainly controlled by the properties of aerosols (size, shape, density and hygroscopicity), breathing pattern and subject’s anatomy (lung structure). In this study, for the comparison of the dose of particles between outdoor and indoor environments or traffic and residential environments, we only calculate the lung deposition for a male adult. The input data information into the ICRP model includes particle properties (size, shape, density and hygroscopicity) and anatomical and physiological parameters (tidal volume, fraction of total ventilation airflow passing through the nose, volumetric flow rate of inspired air, functional residual capacity of the exposed subject, anatomical dead space).
for different exercise levels (standing, walking, running and cycling).

**Input properties of particles into the ICRP model**

Particle size: The ICRP model uses the thermodynamic particle diameter ($d_{th}$) which is determined by a measured diffusion coefficient. In the thermodynamic deposition of particles larger than 2 nm, $d_{th}$ is considered to be equal to the particle volume equivalent diameter, $d_{ve}$. The aerodynamic diameter ($d_{ae}$) is the diameter of a unit density sphere with the same terminal settling velocity in air as the particle of interest. The PNSD obtained by the FMPS system is represented by the electrical mobility diameter ($d_{in}$) which is the diameter of spherical particles with the same velocity in a constant electric field as the particle of interest. The traffic-generated aerosols contain not only spherical particles but also a very high proportion of agglomerates. Therefore, it is necessary to convert the particle mobility diameter to the volume equivalent or aerodynamic diameter before inputting these diameters into the ICRP model.

Particle shape ($\chi$) and effective density ($\rho_{eff}$): The shape and effective density of aerosols can be measured by tandem measurements which comprise multiple instruments such as a combination of a differential mobility analyser (DMA) and an aerodynamic particle sizer (APS)/aerosol particle mass analyser (APM) or transmission electron microscope (TEM) (Park et al. 2008). The tandem measurements by SMPS and APM are commonly used to measure the dynamic shapes and effective density of traffic soot (Geller et al. 2006; Park et al. 2003; Park et al. 2004; Rissler et al. 2012, 2014; Tavakoli and Olfert 2014). In this study, we assume the $\chi$ and $\rho_{eff}$ values for the soot based on the works of Park et al. (2004), Rissler et al. (2014) and Tavakoli and Olfert (2014). According to their work, the $\chi$ value is found to increase, while the $\rho_{eff}$ value decreases dramatically with the particle size (Avino et al. 2013; Manigrasso and Avino 2012): $\chi = 1.0$ and $\rho_{eff} = 1.0$ ($d_m = 14–50$ nm), $\chi = 1.5$ and $\rho_{eff} = 0.9$ ($d_m = 50–100$ nm), $\chi = 1.5–1.7$ and $\rho_{eff} = 0.9–0.6$ ($d_m = 100–200$ nm) and $\chi = 1.7–2.4$ and $\rho_{eff} = 0.6–0.2$ ($d_m = 200–520$ nm).

The equivalent and aerodynamic diameters are calculated from the following equations (Park et al. 2008):

$$d_{ve} = d_m \times \frac{C(d_{ae})}{C(d_m) \times \chi} \quad (1)$$

$$d_{ae} = d_m \times \sqrt{\frac{\rho_{eff} \times C(d_m)}{\rho_0 \times C(d_{ae})}} \quad (2)$$

where $d_m$, $d_{ae}$ and $d_{ve}$ are the mobility, aerodynamic and volume equivalent diameters (nm); $\rho_0$ is the unit density (1 g/cm$^3$); $\rho_{eff}$ is the effective density (g/cm$^3$); and $C(d)$ is the size-dependent Cunningham slip correction.

Tavakoli and Olfert (2014) reported that the relative standard errors (RSEs) in the measurements of effective density and shape factors are 9.3–11.1 and 3.8–3.9% for the soot particles in the mobility diameter range of 95–637 nm. Following Eqs. (1) and (2), the variation of shape factors and effective density causes the uncertainty in the calculation of equivalent diameters (RSE ~ 3.8–11.5%) and aerodynamic diameters (RSE ~ 1.8–3.5%) which are used as input variables in the ICRP model. We inputted these diameters with their uncertainties in the ICRP model and found that the RSE of deposition fraction of aggregates in the whole lung was in the range of 1.2–3.8%.

**Particle hygroscopic properties**: The hygroscopic properties of particles significantly influence the lung deposition fraction due to increase of the particle diameter when it penetrates into the very humid human respiratory system. Vu et al. (2015) reviewed hygroscopic growth factors of submicron aerosols from different environments and found that the traffic-generated aerosols are nearly hydrophobic, but during the atmospheric ageing process, their hygroscopic properties can change and become more hygroscopic. At roadside sites, the small particle (<50 nm) number fraction of the nearly hydrophobic group accounted for more than 80% of total particles, while for larger particles (>100 nm), the fraction of nearly hydrophobic particles decreases because of a higher contribution of hygroscopic urban background aerosol.

A comparison of lung deposited fractions of aerosols for aggregates, hydrophobic spherical particles and hygroscopic spherical particles is shown in Fig. 1 and will be discussed in the “Factors determining the dose of road traffic particle aerosols” section.

In this study, we assume that the particles from traffic emissions (fresh nucleation and soot mode) are hydrophobic agglomerates. The fraction of these particles was obtained from the positive matrix factorisation (PMF) results which will be discussed in the “Application of PMF to the particle number size distributions” section. The lung deposition of hygroscopic particles that originated from atmospheric nucleation and urban background aerosols is estimated following an approach which was described in our previous study (Vu et al. 2015).

**Lung dose calculation of aerosols by different metrics**

**Lung dose calculation**: In our study, we calculated the lung dose of aerosols in three main regions: extra-thoracic (ET), tracheo-bronchial (TB) and pulmonary/alveolar (AL).

The regional lung dose is defined as the proportion of inhaled particles deposited in the respiratory tract during an exposure time period ($\Delta t$) and is calculated from the following:

$$Dose_t = \sum_{j=1}^{26} V_E \times C_j \times DF_{ij} \times \Delta t \quad (3)$$
where $DF_{ij}$ is the total deposited fraction of particles in the $j$th size bin in the lung region ($i$). $f$ is the size bin in the FMPS measurements. $C_j$ is the total concentration of particles by number, surface area or mass in the $j$th size bin. $V_E$ is defined as the ventilation rate which is the volume of gas inhaled or exhaled from the lungs during a unit time period ($m^3/h$), and $\Delta t$ is the exposure time duration. In a given exposure scenario, the respiratory tract deposited particle dose rate, which is defined as the total amount of particles deposited in the respiratory tract during a given period of time, can be calculated as:

$$\text{Hourly Dose}_i = \sum_{j=1}^{26} V_E \times C_j \times DF_{ij} \quad (4)$$

The dose not only depends upon the measured particle concentrations in each environment but is also influenced by complex parameters including particle properties, breathing patterns, flow dynamics and lung structure.

**Calculation for the surface area and mass for spheres and agglomerates**

*For the spherical particles:* The surface area ($SA_{Sp}$) and mass ($M_{Sp}$) were calculated from the following equations: $SA_{Sp} = \pi (d_m)^2$; $M_{Sp} = \rho_{pp} \pi (d_m)^3 / 6$, where $d_m$ is equal to $d_{ve}$ and we assume the $\mu_t \sim 1.4$ g/cm$^3$ for dense aerosols based on the work by Geller et al. (2006) and Rissler et al. (2014).

*For the agglomerates:* The surface area ($SA_{Agg}$) and mass ($M_{Agg}$) were calculated as the following equations based on Rissler et al. (2012):

$$SA_{Agg} = \pi (d_m)^2 \left( d_m / d_{pp} \right) \left( \rho_{eff} / \rho_{pp} \right) \quad (5)$$

$$M_{Agg} = \rho_{eff} \pi (d_m)^3 / 6 \quad (6)$$

where $\rho_{pp}$ is the density of the primary particles (1.88 g/cm$^3$) and $d_{pp}$ is the primary particle diameter (27 nm).

**Application of PMF to the particle number size distributions**

The application of the PMF technique to the PNSD datasets has been widely used in recent years to apportion the sources of particles (Vu et al. 2016). This work utilises PMF on both outdoor and indoor PNSD datasets collected at houses in traffic and residential areas. The PMF results show that there are four sources of particles contributing at the traffic site, namely traffic nucleation mode, aged traffic nucleation mode, traffic soot mode and urban secondary aerosols. In a house in a residential area, traffic emissions, aged nucleation and secondary aerosols are the main sources of particles. The PMF application and PMF outcomes are described in detail in the Supplementary information.

The fractional contribution of road traffic to the sub-micrometre aerosols in both outdoor and indoor environments is shown in Fig. 2 as a function of particle size. Road traffic accounted for 91.3 and 84.2% of particle number collected in the outdoor and indoor environments in the traffic area. For aerosols collected in the residential area, its contribution was 43.5 and 23.7% for outdoor and indoor environments, respectively. For the lung dose calculation, we assumed aerosols generated from road traffic are aggregates and hydrophobic.

**Results**

**Lung dose calculation for road traffic-generated aerosols**

The average concentrations of traffic-related primary particles measured in front of an apartment located near to a busy street.
were $2.8 \times 10^4$ particle/cm$^3$, 620 mm$^2$/m$^3$ and 9.1 μg/m$^3$ by number, surface area and mass metrics, respectively. As shown in Fig. 3, more than 88.6% of particles by number were distributed in the ultrafine size range ($d_m < 100$ nm), whereas particles by surface area and mass were predominantly found in the accumulation mode ($d_m > 80$ nm). The median diameters of particle size distributions by number, surface area and mass were 51.7, 135.5 and 209.7 nm, leading to higher lung deposition fractions of particle number.

The deposition fractions of particles by number (for males in standing mode) were $0.07 \pm 0.01$, $0.12 \pm 0.01$, $0.36 \pm 0.03$ and $0.55 \pm 0.05$ in the ET, TB and AL regions and whole lung, respectively. Values expressed by surface area and mass were lower, and the deposited fractions (DFs) of submicron particles by surface area and mass in the whole lung were $0.32 \pm 0.02$ and $0.32 \pm 0.02$. To compare with previous studies, Vu et al. (2016) reported a value of DF for fresh traffic aerosol in London, UK, by number to be 0.57. Londahn et al. (2009) experimentally measured DFs of aerosols in nine healthy subjects by mouth at a busy street in Copenhagen, Denmark, and reported that DFs of the kerbside particles by surface area and mass in the whole lung were $0.60, 0.29$ and $0.23$ by number, surface area and mass, respectively. Rissler et al. (2012) experimentally determined the DFs of diesel aerosols in the human respiratory tract and reported the DFs by number in the whole lung were 0.47 (transient engine regime) and 0.65 (idling), while DFs were in the range of 0.27–0.32 for surface area and mass.

The hourly regional lung dose of aerosols was calculated and is shown in Fig. 4. The lung dose for particles by number, surface area and mass dose in the whole lung was $1.0 \times 10^{10}$ particles/h, 130 mm$^2$/h and 1.9 μg/h, respectively. A majority of particles by number were deposited in the AL region (65%), followed by the TB region (22%). A similar trend was found for particle surface area which mostly deposited in the AL region (65%) and TB region (20%). However, mass dose was found predominantly in the AL region (53%) and ET region (33%). Polar plots of the doses showed that the doses for all metrics were contributed by the local traffic sources. When wind speed increased above 4 m/s, the doses were decreased approximately twofold due to the dilution of particles.

Figure 3 shows the dependence of lung dose of particles by particle size. Lung dose by number was predominantly contributed by the small particles ($d_m < 100$ nm), while surface area and mass dose were mainly contributed by the accumulation mode particles ($d_m \sim 100$ nm). The increase of mass dose for particles larger than 250 nm was attributed largely to their hygroscopic properties rather than their size. In constructing Fig. 3, it was assumed that all non-traffic particles are hygroscopic, whereas traffic particles are hydrophobic, and data from Fig. 2 were used.

Factors determining the dose of road traffic particle aerosols

Particle properties

Particle properties including size, density, shape and hygroscopicity significantly influence the deposited fraction of particles in the human respiratory as shown in Fig. 1. Compared to a spherical particle with the same mobility diameter, aggregates which are mainly emitted from diesel combustion show higher deposition fractions. This result is in agreement with the results reported from previous studies (Broday and Rosenzweig 2011; Hofmann et al. 2009; Morawska et al. 2005). Because particles by number are mainly distributed in the ultrafine size range ($d_m < 100$ nm) and most of the particles collected at the traffic site are derived from road traffic-generated aerosol and are considered as hydrophobic particles, the properties of the particles have a minor effect on the deposited fraction and number dose calculation. However, as shown in Fig. 2, larger particles ($d_m > 100$ nm) are found to have a larger hygroscopic non-traffic fraction, and the
properties of the particles have a greater effect on the mass dose calculation.

**Physical patterns**

This section will compare lung dose for male adults in four physical modes, namely standing, walking, running and cycling. Table 1 shows the values of physiological parameters as the input variable in the ICRP model for a male adult at different exercise levels. Table 2 shows the results of calculations in which slight changes in deposition fractions of particles in the human respiratory system are seen. In terms of particle deposition fraction by number, there was a slight decrease from 0.55 for standing to 0.49 for running. The total surface area deposited fractions are also decreased slightly when the exercise levels increase, whereas the deposition fraction by mass increases from 0.32 for standing to 0.35 for running. As shown in Fig. 5, when exercise levels increase, it was found that the deposition of aerosols in the TB region halves. By increasing the exercise level, the deposition fractions in the

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**Fig. 3** Number/surface area/mass dose dependence upon particle size, using data from the outdoor traffic site

**Fig. 4** Hourly dose of outdoor aerosols in the human respiratory system from data from the traffic site, by number (left panel), surface area (central) and mass (right panel)
AL region increase for particles smaller than 34 nm, then decreases slightly for the larger particles. These results are consistent with earlier research by Löndahl et al. (2007). However, in another study conducted by Daigle et al. (2003), the DF by number at rest (0.63) was much lower than that (0.83) during exercise. The difference between this study and our results may be explained by the different size distribution. The count median diameter in the Daigle et al. (2003) study is 26 nm, which is much smaller than that (51.7 nm) in our study. As shown in Fig. 5, an increased exercise level causes a substantial increase in the deposition efficiency of small particles ($d_{50} < 30 \text{ nm}$).

From Eq. (4), the dose of aerosols into the lung not only depends on the DF but also the minute ventilation. The minute ventilation during running and walking is 3.5 and 1.45 m$^3$/h, respectively, which are 5.5 and 2.3 times higher than those during standing. Hence, the lung dose of submicron aerosols at different exercise levels is mainly controlled by the minute ventilation rate rather than the deposited fractions.

Table 1: Inputted physiological parameters in the ICRP model for different exercise levels based on previous studies by Adams (1993), ICRP (1994) and Zuurbier et al. (2009).

| Activity  | $F_R$ (min$^{-1}$) | $V_b$ (m$^3$/h) | $V_T$ (mL) | $V$ (mL) | $F_n$ | Other parameters |
|-----------|------------------|-----------------|------------|----------|-------|-----------------|
| Standing  | 13.8             | 0.64            | 774        | 355      | 1.0   | FRC = 3301 mL; $V_{dET} = 50$ mL; $V_{abn} = 49$ mL; $V_{dbb} = 47$ mL; height = 176 cm; $SF_1 = SF_b = SF_a = 1$ |
| Walking   | 19.6             | 1.45            | 1231       | 806      | 1.0   | $V_{dbb} = 49$ mL; $SF_1 = SF_b = SF_a = 1$ |
| (2 mph)   |                  |                 | 1231       | 806      |       |                 |
| Running   | 27.9             | 3.50            | 2094       | 1948     | 0.5   |                 |
| (5 mph)   |                  |                 | 2094       | 1948     |       |                 |
| Cycling   | 18.9             | 1.32            | 1162       | 734      | 1.0   |                 |
| (7.5 mph) |                  |                 | 1162       | 734      |       |                 |

$V_T$ and $V$ values were estimated based on their relationships with $V_b$ in different exercise levels for ICRP parameters for sitting, light exercise and heavy exercise.

Indicators for the monitoring of lung dose of outdoor particles (traffic site)

Table 3 shows the correlation between air pollutant (particles by number or mass, NO, NO$_2$ and NO$_x$) concentrations and the hourly lung dose deposition of sub-micrometre particles by different metrics for the male adult at rest. The hourly dose of particles by number in the human respiratory system is mainly controlled by their total number concentration ($r^2 \sim 1.00$). This can be explained by the lesser variation in the deposition fraction of particles ($0.55 \pm 0.05$) compared to the variation in their number concentration ($2.8 \pm 1.9 \times 10^4$ particles/cm$^3$). Particle number concentration has very good correlations with the lung dose of surface area in the AL and ET regions ($r^2 > 0.90$) and the TB region ($r^2 > 0.82$). However, for dose by mass, particle number is a poor predictor of the mass dose in the TB region ($r^2 = 0.42$), while the correlations between total particle number concentration with mass dose in the AL and ET regions are 0.70 and 0.88. In contrast, PM$_{2.5}$ and PM$_{10}$ are good predictors of the lung dose of mass or surface area (the latter only in the TB region), but they cannot predict the lung dose of particles by number ($r^2 < 0.5$). This conclusion is consistent with calculations reported by Harrison et al. (2010) from earlier size distribution measurements. Kristensson et al. (2013) reported that PM$_{2.5}$ can be used as proxy for the surface area and mass dose for long averaging times (> 10 h).

Good correlations between NO$_x$ and hourly lung dose of submicron aerosols by mass and surface area were found ($r^2 \sim 0.8$). At the traffic site, NO$_x$ is mainly emitted from diesel vehicles and the mass and surface area of aerosol are distributed mainly in the accumulation mode which is largely influenced by the diesel soot accumulation mode. NO$_x$ shows a moderate correlation with the number dose ($r^2 \sim 0.71$).

Table 2: Comparison of the deposition fractions of submicron aerosols from different exercise levels in the regional lung, using data from the outdoor traffic sites.

| Activity | Number | Surface area | Mass |
|----------|--------|--------------|------|
|          | AL TB ET | AL TB ET | AL TB ET |
| Standing | 0.36 0.12 0.07 | 0.21 0.06 0.05 | 0.18 0.05 0.09 |
| Walking  | 0.35 0.09 0.07 | 0.18 0.05 0.06 | 0.15 0.03 0.16 |
| Running  | 0.34 0.07 0.07 | 0.16 0.03 0.07 | 0.13 0.03 0.20 |
| Cycling  | 0.35 0.09 0.07 | 0.18 0.05 0.06 | 0.15 0.03 0.16 |
Lung dose of traffic-generated particles from outdoor to indoor environments

Deposition fractions of indoor aerosols (traffic site)

We estimated the deposition fractions of indoor traffic-derived aerosols by different metrics in the respiratory tract for male adults. DF values of aerosols by number in the AL, TB and ET regions and whole lung are 0.28 ± 0.02, 0.09 ± 0.01, 0.06 ± 0.01 and 0.43 ± 0.03, respectively. The lower DFs of indoor aerosols compared to those of outdoor aerosols are attributed to the change of their particle number size distributions which shifts from a small size range to a larger one. The count median diameter of outdoor PNSD was 51.7 nm, while that for the indoor PNSD was 67.2 nm. This size shift has a stronger influence on the DF by number in the AL region than other regions. Similarly, the DFs of aerosols by surface area and mass in the whole lung when they penetrate from outdoor into indoor environments show a slight decrease by 9.6 and 12.5%, respectively.

Comparison of diurnal lung dose of particles between outdoor and indoor environments

The hourly doses of indoor aerosols in the whole lung by number, surface area and mass were $2.5 \times 10^9$ particles/h, 48.2 mm$^2$/h and 0.91 $\mu$g/h which are less than those of outdoor aerosols by 4.1, 2.7 and 2.1 times. When aerosols enter into a house, their concentrations decline due to evaporation and deposition processes (Vu et al. 2017). Higher loss rates of

| Metrics | Dose by number | Dose by surface area | Dose by mass |
|---------|----------------|----------------------|-------------|
| Number | AL  | TB  | ET  | Total | AL  | TB  | ET  | Total | AL  | TB  | ET  | Total |
| PM$_{2.5}$ | 0.37 | 0.39 | 0.36 | 0.37 | 0.67 | 0.87 | 0.58 | 0.62 | 0.91 | 0.92 | 0.82 | 0.88 |
| PM$_{10}$ | 0.46 | 0.47 | 0.44 | 0.46 | 0.73 | 0.90 | 0.65 | 0.68 | 0.93 | 0.91 | 0.86 | 0.90 |
| NO  | 0.70 | 0.70 | 0.70 | 0.70 | 0.72 | 0.74 | 0.70 | 0.70 | 0.71 | 0.58 | 0.75 | 0.75 |
| NO$_2$ | 0.68 | 0.68 | 0.68 | 0.69 | 0.79 | 0.78 | 0.77 | 0.78 | 0.73 | 0.56 | 0.80 | 0.79 |
| NO$_x$ | 0.73 | 0.73 | 0.73 | 0.71 | 0.81 | 0.79 | 0.78 | 0.78 | 0.77 | 0.90 | 0.82 | 0.82 |

Particle number, NO, NO$_2$ and NO$_x$ were measured hourly. PM$_{2.5}$ and PM$_{10}$ were measured daily.
smaller particles result in a smaller indoor/outdoor (I/O) hourly dose ratio for particles by number (0.24) compared to those for the surface area (0.37) and mass (0.48).

Figure 6 shows the diurnal variation of hourly lung dose of both outdoor and indoor aerosols in the human respiratory tract. The lung dose of particles in terms of three metrics was highest around 7:00–9:00 a.m. and 17:00–19:00 p.m., corresponding to the morning and afternoon traffic rush hours. The outdoor total lung doses during rush hours were $16.8 \times 10^9$ particles/h, 192.5 mm$^2$/h and 2.4 μg/h for particle number, surface area and mass which were around two times higher than those during the non-rush hour periods. Daytime lung dose by number, surface area and mass was found to be higher by 2.3, 1.9 and 1.6 times than the nighttime lung dose.

**Increment of dose rate of particles in urban areas due to road traffic emissions**

Whole lung doses of outdoor traffic-derived submicron aerosols by number, surface area and mass in the residential area were $1.8 \times 10^9$ particles/h, 22.7 mm$^2$/h and 0.5 μg/h, while those of indoor submicron aerosols were $0.8 \times 10^9$ particles/h, 11.8 mm$^2$/h and 0.4 μg/h. The DF values of residential outdoor aerosols by number in the AL, TB and ET regions

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**Figure 6** A comparison of the diurnal patterns of the regional lung dose of aerosols between outdoor and indoor environments

**Figure 7** Hourly outdoor/indoor dose in the whole lung for a man standing in the traffic and residential areas
were 0.29, 0.10 and 0.06, while those of indoor aerosols were 0.27, 0.09 and 0.06. The DF values of those by surface and mass show an insignificant difference from those for aerosol collected at the traffic site. Lower DFs of aerosols by number in the residential area can be explained by the particle number size range. Count median diameters of outdoor and indoor residential particle number size distribution were 60.7 and 63.8 nm. In terms of regional lung dose, particles mainly deposited in the AL region (44–65%) for all metrics.

We compared the difference between the lung dose of aerosols collected in the traffic and residential areas at the same sampling times (16th–30th April 2012). Figure 7 shows a huge increment of lung dose at the traffic site due to road traffic emissions. The dose of outdoor aerosols in the traffic area was much higher than that in the residential area (5 times for number and surface area dose and 2 times for mass dose). Traffic emissions also caused an increased dose of indoor aerosols in terms of number and surface area (3 times) in the traffic area compared to the residential area, but there is only a slight increase in terms of the mass dose.

**Conclusion**

This study found that traffic emissions were the major source of submicron particles by number deposited into the human respiratory system. Due to their small size, the traffic-generated particles could penetrate deeply and deposit mainly in the AL region. The dose of sub-micrometre aerosols for both outdoor and indoor environments in the traffic area was much higher than that in the residential area, especially during the traffic rush hours. This is due not only to higher concentrations but also to a smaller count median diameter at the traffic site. The increment of lung dose of aerosols due to traffic emissions is different for different metrics, for example the dose of outdoor particle number in the traffic area was 5 times higher than that in the residential area, while mass dose in the traffic area was higher than that in the residential area by a factor of 2. Particle number can be used as an indicator for predicting the number dose, while PM$_{2.5}$ and PM$_{10}$ have good correlations with the surface area and mass dose. Due to the high correlation with the lung dose ($r^2 = 0.7$ for number, > 0.8 for surface area and mass), NO$_x$ could be a good proxy for predicting the lung dose of particles of sub-micrometre diameters.

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**Compliance with ethical standards**

**Conflict of interest** The authors declare that they have no conflict of interest.

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