Mechanistic exposure assessment of traffic-originated ultrafine PM

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Abstract

Exposure to PM is linked to various acute and long-term health effects; however the lack of in depth understading of the mechanisms of toxicity obscures targeted risk management possibly resulting in overly conservative risk management. This study aims at the development of a methodology for refining PM exposure by incorporating a detailed particle number exposure model coupled to a lung deposition model, accounting for differences in PNC that are not always reflected by mass concentration measurments in terms of actual exposure.

Introduction

In urban areas combustion sources are the main contributors to submicrometric aerosol (Avino, et al. 2011) or else Ultra Fine Particles, defined as particles with an aerodynamic diameter less than 0.1 μm. In most of the existing studies UFPs vary about from 70% to 95% of total particle number concentration (PNC), with lower contributions observed during night-time hours (Buonanno, et al. 2011; Manigrasso and Avino 2012; Rodríguez, et al. 2007). UFPs penetrate efficiently into the respiratory system and are capable of translocating from the airways into the blood circulation (Oberdörster, et al. 2005). Although some epidemiological evidence suggests that exposure to ultrafine compounds (Delfino, et al. 2005) may be associated with higher cardiovascular risk (eg, an elevation of UFP count by 9748/cm^3 has been associated with an increase in cardiovascular mortality of approximately 3% within 4 days in Erfurt, Germany (Stolzel, et al. 2006) and adverse responses (Delfino, et al. 2009; Delfino, et al. 2008), there have been few such studies because they are challenging to conduct, for numerous reasons (Brook, et al. 2010). Only a few UFP monitors exist generally. The levels measured at regional sites may not accurately reflect the spatial heterogeneity of individual exposure. Furthermore concentrations may be dominated by local point sources of fresh combustion (eg, roadways), resulting in unclear associations to some extent (Peters, et al. 2005).

To better understand the etiology between PM air pollution and human disease, a deeper understanding of the actual exposure to different PM size fractions and the relevance of deposition and toxicity mechanisms to the outcomes is needed. The above elucidate the need for advancing PM exposure assessment to more informative exposure metrics, taking into account not only the overall mass concentration, but also the corresponding PNC. The latter is a descriptor of the actual size distribution of airborne particles. In addition, exposure to PM and more specific to UFPs is described comprehensively only if the actual size distribution is given. The latter is very important, since particle size distribution clearly affects deposition to the respiratory tract, affecting in a similar way possible health outcomes, always in relevance to the mediated biological mechanism.

Methodology

The study includes a set of UFP measurements and exposure modeling including deposition acroos respiratory tract. The UFP measurements were carried out in two urban sites in the city of Thessaloniki. The first one at the curbside of an intensivelly trafficked road (Egnatia Avenue, Venizelou square) and the second one at the suburbs of the city (Eptapyrgio), representing the urban background concentration. 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 sizechannels, in the range from 5.6 to 560 nm, with 1 s time resolution. FMPS operates at a high flow rate (10 L/min) to minimize diffusion losses of UFPs. In both sites, fixed monitoring stations of the regualtory monitoring network exist, providing average daily data for PM_{10} and $PM_{2.5}$. In addition, UFPs measuments were carried out inside a vehicle crossing Egnatia Avenue several times, so as to identify the differences in PM concentrations between the different traffic lanes and the curbside of the same road. These measurements were carried out by a hand-held Condensation Particle Counter Model 3800, covering a UFPs range of 0.015 to 1μm.

Internal exposure of UPF was carried out using the Multiple-Path Particle Dosimetry (MPPD) model. The algorithms in MPPD calculate the deposition and clearance of monodisperse and polydisperse aerosols in the respiratory tract of rats and human adults and children (deposition only) for particles ranging from ultrafine (0.01 microns) to coarse (20 microns) sizes. The models are based upon single-path and multiple-path methods for tracking air flow and calculating aerosol deposition in the lung. The single-path method calculates deposition in a typical path per airway generation, while the multiple-path method calculates particle deposition in all airways of the lung and provides lobar-specific and airway-specific information. Within each airway, deposition is calculated using theoretically derived efficiencies for deposition by diffusion, sedimentation and impaction within the airway or airway bifurcation. Filtration of aerosols by the head is determined using empirical efficiency functions. Results using our model show good agreement with experimental data for regional deposition in the rat and human lung (Heyder, et al. 1986).

Results and discussion

The results of the PNC distribution at the two monitoring stations and within the vehicle cabin are illustrated in Figure 1.

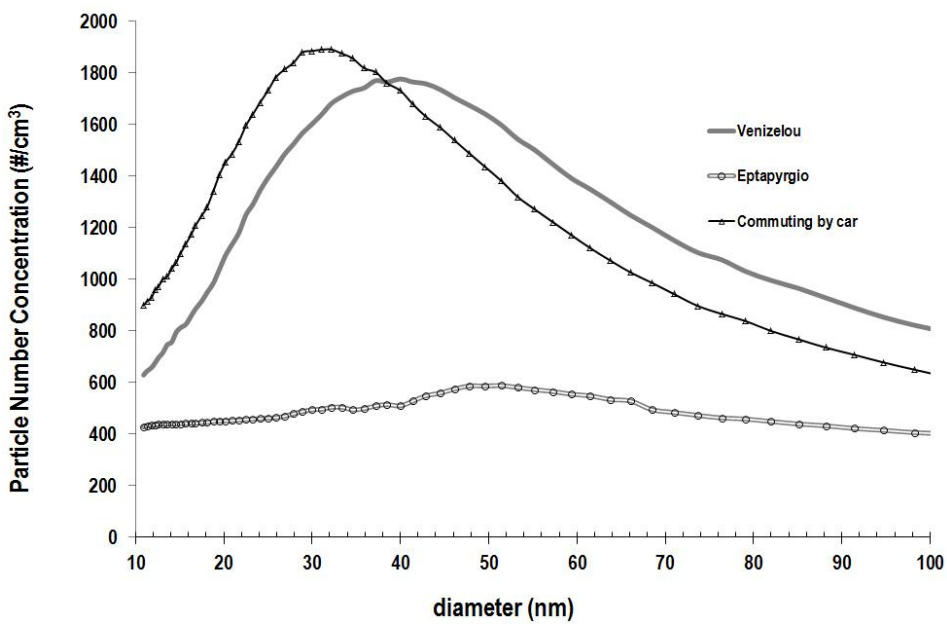

Figure 1. PNC in urban traffic, urban background and driving

For comparison, the annual $PM_{10}/PM_{2.5}$ concentrations for the two stations are 54/38 μg/m^3 (traffic station) and 33/23 μg/m^3 (background station) respectivelly. Although the $PM_{10}/PM_{2.5}$ ratio between the two sites does not differ substantially, the differences are much larger when it comes to PNC (77149 and 32459 particles/cm^3) – the corresponding UPFs mass concentration at the traffic and the backgound station is estimated equal to 5.9 and 2.4 μg/m^3 respectivelly.

Averaging the data from all measurements, the geometric mean diameter (GMD) for the traffic site is equal to 35.7 (sd 1.76) nm, while for the background site the corresponding value is equal to 44.7 (sd 1.99) nm. GMD intra-day variability is wider for the urban site, fluctuating between 32.4 to 50.6 nm. This variation is inverselly correlated to traffic intensity; the higher the intensity of traffic (and consequently, traffic emissions), the smaller are the UFPs. The lack of a dinstinct "peak" at the background monitoring site reflects the relative scarcity of direct tailpipe fumes there. The urban background monitoring station is located in such a way so as the sampled air is well mixed. Thus a number of particle fate processes such as nucleation form larger size particles. The results within the vehicle cab (windows open) indicate that during driving, commuters are exposed to an even higher fraction of smaller diameter UFPs, with an average GM of 31.9 nm (sd 1.76), PNC equal to 82061 and a mass concentration equal to 4.9 μg/m^3. The latter is explained by the fact that air from the traffic lane enters quickly in the vehicle cabin, probably faster than the time needed to travel to the curbside.

Overall, the results are in aggrement to the findings of recent similar studies (Manigrasso and Avino 2012) dealing with the rapid evolution of traffic emitted UFPs. From the centre of the street towards the curbside, there is very rapid decline on particle number, accompanied by a less significant decrease on the overall mass concentration, indicating the formation of larger PM towards the curbside. Aerosol arising from this variability in vehicular emissions very quickly gets mixed due to turbulent diffusion and what is usually measured is the result of these variable emissions and mixing. The time scale within which aerosol number size distributions evolve is determined by the changes that vehicular exhausts undergo upon exiting the tail pipes. These processes are dilution, nucleation of semivolatile organic compounds (SVOCs) to yield new particles. During traffic peak hours maxima are centered mainly between 10 and 20 nm, reflecting the vehicle exhaust emissions. In the early morning, between 2 and 3 a.m., when traffic levels are comparatively lower, maximum doses move towards higher diameters, between 30 and 40 nm, with frequent values also at about 60 nm. Relative humidity (RH) plays also a significant role due to hygroscopic growth of airborne particles (see Fig. 2) (Sarigiannis, et al. 2002; Sarigiannis, et al. 2004), since RH and GMD variation trends have been identified to be very similar (Manigrasso and Avino 2012).

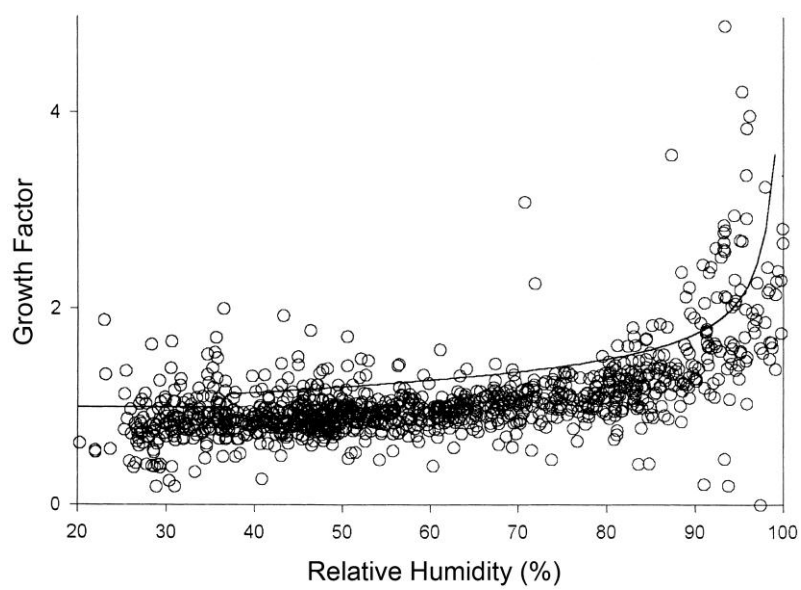

Figure 2. Particle growth as a function of relative humidity

To capture the implications of these dynamic processes for the overal UFP exposure, three exposure scenarios were built (exposure for 2 hours per day, 5 days per week), considering the differences in deposition across the respiratory tract. This is very important, since different size particles tend to deposite at different fractions along the respiratory tract. The results of these scenarios are presented in Figures 3 and 4, showing the UFP fraction and doses deposited in the three main regions of the respiratory system of an adult male breathing normally through the nose, doing light exercises. The

results indicate that exposure between the two measurment sites corresponds to different patterns of deposition within the lower repiratory tract. In fact, the overall deposition is almost four times higher at the traffic site vs. the urban background site. This variation is reflected neither in the overall UFP PNC, nor in the PM_{10} and $PM_{2.5}$ mass concentrations daily monitored in the respective sites. On the contrary, deposition patterns between the traffic site and car commuting were similar, with a higher fraction deposited in the second case, reflecting the lower GMD of the measured UFPs.

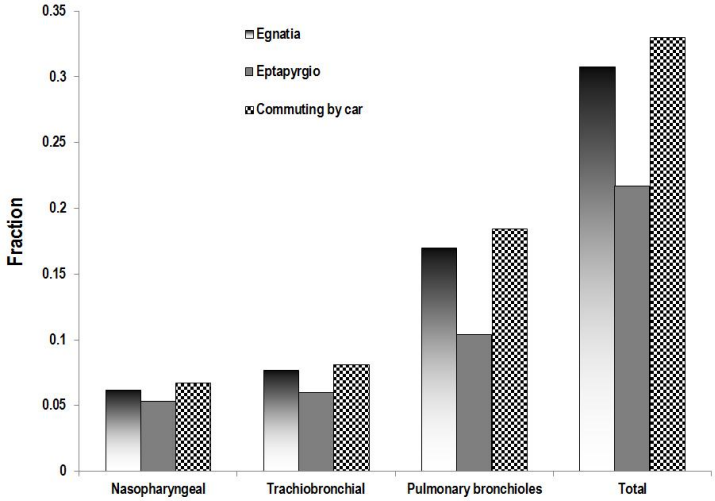

Figure 3. UFPs fraction deposition across the respiratory tract for the different exposure scenarios

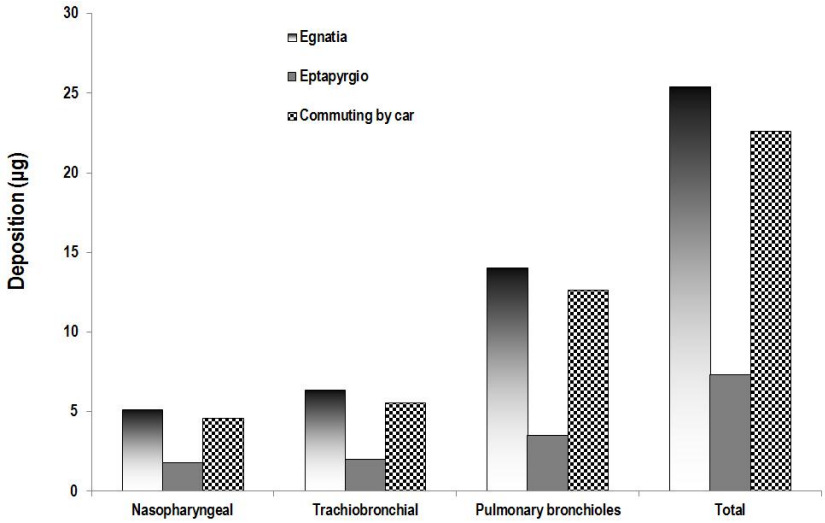

Figure 4. UFPs mass deposition across the respiratory tract for the different exposure scenarios

Concluding remarks

Recent epidemiological studies have started addressing the adverse effects on health of short-duration exposures to high particle concentrations (Brook, et al. 2002). However, a starting point for refining PM and more specifically UFPs exposure assessment, is to capture the fast-changing characteristics of traffic aerosol and to translate them into actual human exposure and deposition in the respiratory tract. Considering the fast evolution of aerosol from vehicle exhausts, estimation of short-term exposure in scenarios involving proximity to traffic requires higher UFP sampling time resolution. This is important

since inhalation of particles of different aerodynamic diameter results in completely different respiratory deposition patterns (Figure 5). Ultrafine particles are remarkably deposited to the lower respiratory tract (bronchioles); thus they are capable to enter systemic circulation and consequently induce or be associated to disease mechanisms beyond local (lung) inflammation.

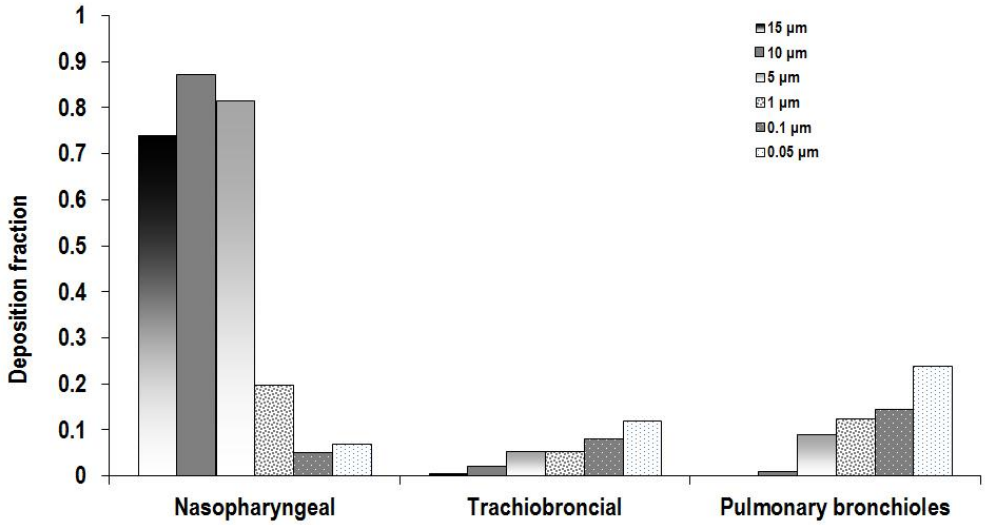

Figure 5. Fraction of deposition of different sized PM across the respiratory tract

The latter when combined to other exogenous (combined exposure to multiple chemicals) and endogenous (increased susceptibility due to health status or age vulnerability) risk modifiers may result in increased risk of disease. For example, in the latest study carried out by Brook et al. (2011), there are three general pathways that could link $PM_{2.5}$ exposure with changes in cardiovascular physiology: systemic inflammation (pathway 1), altered autonomic nervous system balance (pathway 2), and direct effects of particles or constituents reaching the circulation (pathway 3) (Brook 2008). Pathways 2 or 3 are considered as most probable. Taking this into account, the fraction of particles deposited in the lower respiratory tract and eventually translocated in the rest of the human body is of major importance in relevance to cardiovascular diseases, as well as to other health effects not attributed to lung or systemic inflammation. In addition, alterations in markers that indicate changes in thrombosis, fibrinolysis, and global coagulation have also been reported in the literature. An immediate elevation in soluble CD40-ligand concentration, possibly reflecting platelet activation, recently was found to be related to ambient UFPs and accumulation-mode particle ($PM_{0.1–1.0}$) levels in patients with coronary artery disease.

Our findings show that exposure to tailpipe traffic emissions (characterized by lower GMD) corresponds to increased internal exposure/deeper lung deposition compared to traffic exposure at the curbside and beyond. Thus the same magnitude of PM exposure in mass concentration will result in different response based on whether this corresponds to a low or high particles number, especially with regard to health effects relevant to UPFs translocation, such as cardiovascular effects. In conclusion, refined PM exposure -response functions could be developed if a holistic exposure-oriented framework as the one presented herein were used to perform environment and health association studies. Considering the practical difficulties to acquire such refined exposure data in the wider population, targeted measurement campaigns including also monitoring of pro-inflammatory markers of effect (C-reactive protein (CRP), fibrinogen, or white blood cell counts) in tandem with refined exposure measurements should provide a feasible strategy towards attaining that goal.

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