# Mechanistic exposure assessment of ultrafine PM

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Exposure to PM is linked to various acute and long-term health effects; however the lack of in depth understanding of the mechanisms of toxicity obfuscates targeted risk assessment possibly resulting in overly conservative risk management. To better understand the etiology of the association between PM air pollution and human disease, a better understanding of the actual exposure to different PM size fractions and the relevance of deposition and toxicity mechanisms to the observed health outcomes is needed. The above elucidate the need to use more informative PM exposure metrics taking into account not only the overall mass concentration, but also the corresponding Particle Number Count (PNC), as a descriptor of the size distribution of airborne particles.

The study includes a set of Ultra Fine Particles (UFP) measurements and exposure modeling including deposition across human respiratory tract (HRT). The UFP measurements were carried out in two urban sites in the city of Thessaloniki (traffic and an urban background one). In both sites, fixed monitoring stations of the regulatory monitoring network exist, providing average daily data for PM10 and PM2.5. PM concentration data comprise the input for a detailed exposure model which incorporates the dependence of inhalation rate on type of activity. The output of the exposure model is the input for the HRT Multiple-Path Particle Dosimetry (MPPD) model, which is used to estimate the deposition distribution of particles of different aerodynamic diameters.

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) respectively. 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 background station is estimated equal to 5.9 and 2.4 μg/m$^3$ respectively. 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 (Figure 1). This variation is inversely correlated to traffic intensity; the higher the intensity of traffic (and consequently, traffic emissions), the smaller are the UFPs. The urban background monitoring station is located in such a way so as the sampled air is well mixed, allowing the smaller particles to be subjected to processes such as nucleation and hydroscopic growth.

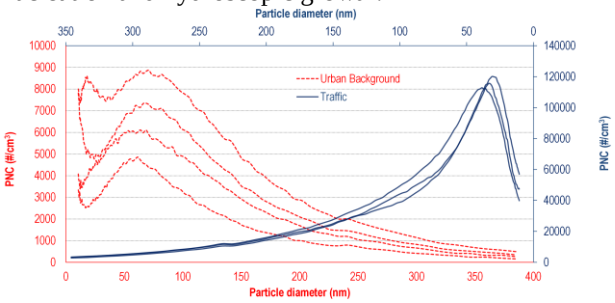


Figure 1. Typical particle count number distributions during peak hours.

To capture the implications of these dynamic processes for the overall UFP exposure, an exposure scenario was built (exposure for 2 hours per day, 5 days per week). This is very important, since different size particles tend to deposit at different fractions along the respiratory tract. The results indicated that exposure between the two measurements sites correspond to significantly different HRT deposition patterns, especially with respect to the lower respiratory tract, the overall deposition is almost four times higher at the traffic site vs. the urban background site (25.4 and 7 μg deposited respectively). This variation is reflected neither in the overall UFP PNC, nor in the PM10 and PM2.5 mass concentrations, which are monitored daily in the respective sites (Figure 2).

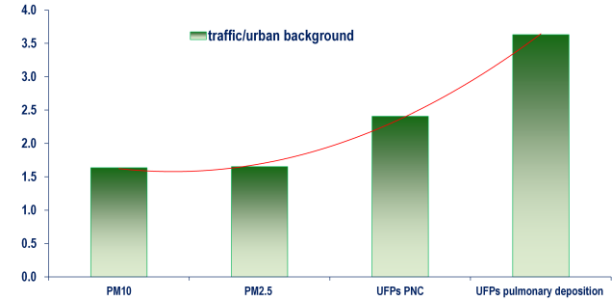


Figure 2. Non-linearity characterizing different PM exposure metrics.

The differences identified above, pose the question to what extent, the currently used concentration-response functions associating coarse and fine PM to mortality and morbidity reflect properly the causal association between actual exposure and health effects.