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LUND UNIVERSITY

PO Box 117
221 00 Lund
+46 46-222 00 00

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Can PM2.5 be used as a proxy for deposited particle dose in the respiratory tract?

Kristensson A..[1], Rissler J.[2], Löndahl J.[2], Johansson C[3]., and Swietlicki E.[1]

[1]Department of Physics, Lund University, P. O. Box 118, SE-221 00, Lund, Sweden
[2]Department of Design Sciences, Lund University, P. O. Box 118, SE-221 00 Lund, Sweden
[3]Department of Applied Environmental Science, Stockholm University, SE-106 91 Stockholm, Sweden
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Presenting author email: adam.kristensson@nuclear.lu.se

Introduction

There are a few studies available that relate health effects to ambient concentrations of particle matter in residential areas where the exposure is high to wood combusted particles (Naeher et al., 2007). However, we do not know if particle measures such as PM2.5 or PM10 are good indicators of deposited dose in the respiratory tract. If they are, we cannot expect stronger associations with adverse health effects in short-term exposure epidemiological studies if we would use a different proxy for the deposited dose. On the other hand, if this is not the case, we would expect a stronger health association if we are able to predict the deposited dose in a more precise manner. In this study, we address this question and investigate whether PM2.5 is a satisfactory proxy for the deposited surface area and volume in the respiratory tract.

Methods

The respiratory tract deposition is obtained at a residential area in northern Sweden, in a town called Lycksele, where the contribution to air pollution from domestic wood combustion and road traffic is significant. The deposited surface area and volume have been calculated using hygroscopic particle data and data from the particle number size distribution. The particle measurement data is combined with the Multiple Part Particle Deposition model of respiratory tract deposition for hydrophobic particles (MPPD, version 1.0, Chemical Industry Institute of Toxicology, Research Triangle Park, NC, with inspiratory fraction of 0.5, particle density of $1 \cdot 10^3$ kg m^{-3}, and nasal breathing). This has resulted in data of the deposited concentration of particle surface area and volume as function of size and two hygroscopic growth modes, one less hygroscopic and one more hygroscopic mode. The principle methodology is described in more detail in Löndahl et al. (2009).

Results and Conclusions

The results show that when averaged over many hours or longer, there is a high correlation between PM2.5 and the deposited surface area and volume. For example, for an 11-hour running mean the Pearson product-moment correlation is $p \sim 0.91$ ($p^2 \sim 0.83$). This is proof that PM2.5 is indeed a satisfactory proxy for the deposited dose in short-term exposure epidemiological studies. Hence, we cannot expect more adverse health outcomes or stronger associations even if we would have data on the deposited surface area and volume.

Conversely, the correlation between one-hour averages of PM2.5 and the deposited surface area and volume is about $p \sim 0.75$, lower than for the 11-hour running mean. This might indicate that PM2.5 is not representative for the deposited dose during hour-long episodes of very high particle exposure. On the other hand, the elevated episodes in Lycksele linger more than a few hours, and hence the lower correlation on an hour basis might not have any practical implications for Lycksele or other towns with similar conditions.

Löndahl, J. et al. (2009). *Env. Sci. Technol.* **43**, 4659-4664.

Naeher, L. P., et al., (2007). *Inhal. Toxicol.*, **19**, 67-106.

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