**PAH exposure and lung cancer risk assessment by internal dosimetry metrics: The case of biomass use for residential heating.**

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This study deals with the assessment of the cancer risk attributable to PAH exposure, attributable to the increased use of biomass for space heating in Greece in the winter of 2012-2013. Towards this aim, several methodological elements have to be integrated, including:

- PM measurements for the three main fractions (PM10. PM2.5 and PM1). This is essential, since smaller PM fractions have been associated to biomass burning. Measurements include two different sampling sites, so as to be able to differentiate the amount and the composition of PM attributed to contribution of different sources.
- PM chemical analysis, aiming at:
    - identifying their carcinogenic potency, through PAHs composition analysis.
    - identifying their origin, through levoglucosan and black carbon (BC) analysis. Levoglucosan is considered as the most specific biomass burning tracer, while BC is considered as product of internal combustion sources.
- Refining the exposure and associated risk assessment methodology. It is known that PM of different size interact differently with the Human Respiratory Tract (HRT). PM from biomass burning has specific characteristics, which have to be taken into account when estimating the associated risk. According to our methodology, this information is incorporated by employing an HRT deposition model. The latter allows us to estimate for a PM fraction of given size the actual amount of PAHs coming in contact with the lower respiratory tract. Using this refined exposure assessment approach, differences in physiology among different age groups/susceptible populations are taken into account in the corresponding cancer risk estimates.

Results showed that all PM fractions are higher in Greece during the cold months of the year, mainly due to biomass use for space heating. PAH and levoglucosan levels were highly correlated, indicating that particles emitted from biomass combustion are more toxic than PM emitted from other sources. The estimated lung cancer risk was non-negligible for residents close to the urban background monitoring site. Higher risk was estimated for infants and children, due to the higher bodyweight normalized dose and the human respiratory tract (HRT) physiology. HRT structure and physiology in youngsters favor deposition of particles that are smaller and more toxic per unit mass. In all cases, the estimated risk (5.7E-07 and 1.4E-06 for the urban background site and 1.4E-07 to 5.0E-07 for the traffic site) was lower to the one estimated by the conventional methodology (2.8E-06 and 9.7E-07 for the urban background and the traffic site respectively) that is based on Inhalation Unit Risk; the latter assumes that all PAHs adsorbed on particles are taken up by humans. With the methodology proposed herein, the estimated risk presents a 5 to 7 times difference between the two sampling sites (depending on the age group). Incorporating internal dose assessment metrics accounts for the increased children susceptibility to chronic exposure to airborne toxicants

such as particle-adsorbed PAHs. Besides the fact that body weight normalized dose of PM is higher in young children, the physiology of the human respiratory tract results in higher deposition fractions of smaller diameter PM in the lower respiratory tract, with multiple implications in the expected adverse health outcomes. These differences could not have been identified had we relied only on conventional risk assessment method. Consequently, the actual cancer risk attributable to PAHs on PM emitted from biomass burning would have been significantly underestimated.