PH174 Atmospheric PAHs and dioxin-like toxicity size distributions and estimation of their depositions to human respiratory tract. Kameda, Y.¹, Shirai, J.¹, Masunaga, S.¹, Komai, T.² and Nakanishi, J.² Yokohama National University, Yokohama, Kanagawa, Japan. ²National Institute of Advanced Industrial Science and Technology, Tukuba, Ibaragi, Japan. In Japan, lung cancer has the highest mortality rate among all cancers. Polycyclic aromatic hydrocarbons (PAHs) and dioxin-like compounds, which are implicated in biological activity mediated through the aromatic hydrocarbon receptor (AhR), are considered to be the part of the cause. Therefore, it is necessary to characterize risk of atmospheric PAHs and find suitable indicator substances for estimating risk of total PAHs. Besides, their size distributions influence inhalation exposure that leads to lung cancer. Thus, the primary objectives of this research are to demonstrate total risk of 22 atmospheric PAHs and to find adequate indicator substances for estimating risk of total PAHs. The atmospheric particles and gases were collected and divided into 6 aerodynamic diameter groups in Yokohama in June and October 2002. The risk of PAHs was calculated as summation of the products of unit risk values of lung cancer and concentrations of individual PAHs. The risks in June and October were 6.15×10 -5 and 1.67×10 -4, respectively. They were in the same order of magnitude as that of benzene in atmosphere, which has already been regulated. Benzo[a]pyrene is considered to be an useful indicator, however, this study showed its risk contributed only 30.7% of summation. Consequently, this result revealed that a combination of dibenzo[a,h]anthracene, anthracene, benzo[b]fluoranthene and dibenzo[a,e]pyrene in addition to benzo[a]pyrene, which were >10% contributors, may be the indicator. The secondary objective is to estimate particle size contribution to three kinds of toxicity in human respiratory tract. They include dioxin-like toxicity, which was measured using H4IIE luciferase cell line, and toxic equivalent quantities of PAHs and PCDD/Fs. Based on these distributions, their TEQs in 5 regions in human respiratory tract were calculated using reference data for each regional deposition. These results showed that $<2\mu$ m size fraction contributed 82-97 % of total TEOs in alveoli region.