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.1 and Nakashita, J.2 1Yokohama National University, Yokohama, Kanagawa, Japan. 2National Institute of Advanced Industrial Science and Technology, Tsukuba, 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,h]pyrene in addition to benzo[a]pyrene, which were >10% contributors, may be the indicator. The secondary objective is to estimate particle size distribution 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 <2μm size fraction contributed 82.97% of total TEQs in alveoli region.

PH175 Comparative Effects of NNK and Resveratrol on COX-2 Activation in Lungs of Female A/J Mice. Nsaiif, R.H., Mehta, C.S. and Philip, O.M. Texas Southern University. NNK is a known lung tumorigen. Resveratrol (trans-3,4',5-trihydroxystilbene), is a polyphenolic phytoalexin found in red grapes. It is an anti-tumorigenic and anti-carcinogenic. The effect of NNK versus the effect of resveratrol on COX-2 activation in lungs of female A/J mice was compared.

PH176 Immunological Function is Altered after Lifetime Exposure to Trichloroethylene. Keil, D.E.1, Heesemann, L.2, EuDaly, A.2, Smythe, J.1, EuDaly, J.G.2, Gilkeson, G.G.2 and Peden-Adams, M.M.2,3 1Department of Health Professions, Charleston, SC. 2Department of Medicine/Rheumatology, Charleston, SC. 3Center For Marine Biomedicine and Environmental Science, Medical University of South Carolina, Charleston, SC. Trichloroethylene (TCE) is an industrial solvent used in the cleaning and degreasing of metal components in various machine industries. Not only is it commonly inhaled during occupational situations, but its widespread use has resulted in groundwater contamination leading to human exposure via drinking water. It has been reported, in murine studies, that TCE can both exacerbate autoimmune disease and suppress immune function. While these studies have addressed immunological effects in adult rodent models, none have explored immunological effects during developmental stages. Exposure to TCE in drinking water (1000 ppb or 10,000 ppb) began when pairs were mated [female C57 and male C3H mice] and continued through weaning [21-day old] or adulthood [56-days old]. The vehicle control group was administered Emulphor-treated water. At 21 days of age we have reported body weight and length were significantly decreased by the 10,000 ppb treatment. NK cell activity and T- and B-cell proliferation were not altered. IgM antibody responses to SRBC challenge were suppressed in both male and female pups by 10,000 ppb TCE and by 1000 ppb TCE in the male pups only. Few alterations were apparent in thymic T-cell populations, whereas there was a distinct decrease in splenic CD4+CD8– T-cells resulting in a concomitant decrease in the CD4+CD8+ ratio. At 56-days of age, current results have revealed and increased in delayed-type hypersensitivity responses. Although numbers of splenic B-cells were not altered at 56-days of age, primary IgM antibody responses were dose-responsively suppressed after exposure to TCE in both male and female mice. This study demonstrates that in a murine model lifetime exposure to TCE causes permanent deficits in humoral immunity and can alter cell-mediated responses.

PH177 Effects of brief exposure to hydrogen chloride on ventilation in awake rats. Gu, Z. and Januszkiewicz, A.J. Walter Reed Army Institute of Research, Silver Spring, Maryland, The United States. Military personnel, firefighters, even residents may inhale hydrogen chloride gas (HCl) released from burning products of polyvinyl chloride in fire environments. The effects of HCl inhalation on respiratory function are still very unclear. This work examined the changes in respiration resulting from exposures to different concentrations of pure HCl gas. Male Sprague-Dawley rats (250-380 grams) were randomly grouped and placed in nose-only position into restrainers connected to a polycarbonate exposure chamber. At first, animals breathed medical-grade air for five minutes. This time served as the baseline period. Subsequently, they were exposed to one of these concentrations for five minutes, afterwards, to medical-grade or ambient air. Respiratory activities during the periods of baseline and exposure, and at 1 hr as well as 24 hrs post-exposure were recorded for five minutes and analyzed using DATAQ-CODAS acquisition system. During the period of exposure to 500 ppm HCl, minute ventilation volume (MVV) was 82% of its baseline. In the 1000 ppm group, MVV dropped sharply to 40%, breath rate declined rapidly to 50% of their baselines. At 1 hr post-exposure, MVVs in 500, 1000 and 2000 ppm groups recovered to close to their baselines respectively. At 24 hrs post-exposure, in 500 and 1000 ppm groups, MVVs recovered to 96+/–11 and 77 +/- 21% (p > 0.05) of their baselines. While in 2000 ppm group MVV and tidal volume were 76 +/- 12% (P < 0.05), and 77 +/– 4 % (p < 0.05) of their baselines. These results suggest that exposure for as short as five minutes to HCl causes immediately ventilatory disturbance. In addition, exposure to HCl at concentration as high as 2000 ppm may cause delayed respiratory disorder.

PH178 Disposition of 5-Nitroacenaphthene in F-344 Rats. Austin, A.F., Nyanda, A.M. and Ramesh, A. Meharry Medical College, Nashville, TN, USA. Acenaphthene, a polycyclic aromatic hydrocarbon, occurs naturally in the environment by carbonization or pyrolysis of organic materials. With excess NO2 in