ENVIRONMENTAL CARCINOGENS: EXPOSURE AND IMPACTS ON CHILDREN’S HEALTH

HRH Princess Chulabhorn Mahidol, Ph.D.

Chulabhorn Research Institute, Bangkok 10210, Thailand

Environmental factors play a major role in determining the health and well being of children who comprise over one third of the world’s population. Arsenic and carcinogenic compounds in air pollution are examples to illustrate how exposure to these compounds can potentially impact children’s health. Prenatal arsenic exposure in a human population resulted in alarming gene expression changes in the newborns. Class prediction algorithms identified gene expression signatures that predict arsenic exposure in a test population with about 80% accuracy. A highly predictive potential biomarker gene set composed of just 11 genes was identified. These genes are promising as genetic biomarkers for prenatal arsenic exposure. There is a robust prenatal response that correlates with arsenic-exposure levels that could modulate numerous biological pathways including apoptosis, cell signaling, inflammatory response, and other stress responses, and ultimately affect health status. The health impact of exposure to environmental carcinogens in air pollution during childhood was also examined. Personal monitoring of exposure and urinary metabolite excretion showed that city school children were exposed to benzene, 1,3-butadiene and PAHs at levels significantly higher than rural children, which was approximately 2-fold for benzene, 4-fold for 1,3-butadiene and 4-fold for PAHs. The early biological effects from exposure to carcinogens were assessed from DNA damage measured as 8-OHdG and DNA strand breaks and DNA repair capacity. 8-OHdG in leukocyte DNA which was 2.5 fold higher in the city school children compared with the rural school children was statistically significantly correlated with benzene exposure level. The levels of DNA strand breaks in peripheral blood samples from the city children were 1.5 fold higher than those in the rural children. Chromosome damage measured by the challenge assay were 1.7 fold higher in city children, indicating a reduction in DNA repair capacity in these children. Taken together, significantly higher levels of DNA damage believed to be the first step in development of cancer, coupled with a decreased DNA repair capacity, indicate that these children are at a higher risk for developing cancer later in life.