

Abstract # 1557

Correlations Between Urinary Phthalate Metabolites and Phthalates, Estrogenic Compounds 4-Butyl phenol and o-Phenyl phenol, and Some Pesticides in Home Indoor Air and House Dust

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Background: Biomonitoring data provide critical information about chemical exposures, and combining biomonitoring with measurements in environmental media provides insight into sources and pathways of exposure.

Methods: In a study of 120 Cape Cod, MA, homes (CCHES), we collected 24-hour indoor air samples and house dust samples and analyzed them for 89 different chemicals thought to be hormonally active, including phthalates, alkylphenols, o-phenyl phenol, bisphenol A, parabens, pesticides, PBDEs, PCBs, and PAHs. First-morning void urine samples were collected from a female resident of each home and analyzed by US CDC for urinary phthalate metabolites. Creatinine-adjusted urinary monoethyl phthalate (MEP), monobutyl phthalate (MBuP), monobenzylphthalate (MBzP) and mono(2-ethylhexyl) phthalate (MEHP) concentrations in the Cape Study were generally comparable to a subset of the NHANES 1999-2000 study population similar in gender, age, and ethnicity; and to the overall NHANES 1999-2000 study population (years selected to match Cape Cod sample collection period). Kendall's tau rank correlation estimates (adjusted for censoring) were used to characterize the relationships within and between urine, air and dust values since the data are subject to multiple censoring levels. In general, Kendall's tau estimates of correlation tend to be lower in magnitude than the corresponding Pearson or Spearman correlation estimates.

Results: Within the Cape Study population, levels of the four phthalate metabolites were correlated (taus ranging from 0.22 - 0.39, $p < 0.001$ for all pairs); and significant correlations were also observed in the NHANES full sample and the subgroup comparable to the Cape Study population for both 1999-2000 and 2001-2002. Generally, strongest correlations were for MBuP with MBzP, MBuP with MEP and for MEHP with MBzP. Significant correlations were also observed between the parent phthalate in air and dust and the corresponding metabolite(s) in urine for MEP, and MBuP, but not MEHP and MBzP and air ($\tau = 0.13 - 0.27$, $p < 0.05$). In addition, several ubiquitous air and dust contaminants that have been shown to be weakly estrogenic were also significantly correlated with urinary phthalate metabolites, including o-phenyl phenol and 4-*t*-butylphenol; and some urinary phthalate metabolites were correlated with air or dust levels of phthalates other than their parent compound ($\tau = 0.12 - 0.17$, $p < 0.05$). The pesticides propoxur and permethrin and the synergist piperonyl butoxide in air or dust were also weakly correlated with urinary phthalate metabolites. This finding may reflect the use of phthalates as an inert ingredient in pesticide formulations.

Conclusions: Overall, these data show that concentrations of many EDCs in biological samples and indoor air and dust co-vary, suggesting that some EDC mixtures may originate from common exposure sources and highlighting potential confounding by other EDCs in health effect studies of phthalates. Future work will utilize factor analysis to identify source profiles of EDC mixtures that are associated with urinary phthalate levels.