Summertime Blues

Childhood Lead Exposure Peaks in Warm Months

Previous studies have shown that children's blood lead concentrations tend to increase during hot summer months. In this issue, Lih-Ming Yiin and colleagues from the Environmental and Occupational Health Sciences Institute at Rutgers University and the Robert Wood Johnson Medical School in Piscataway, New Jersey, explore whether some of this elevation can be explained by seasonal increases in the amount of lead in residential house dust [*EHP* **108:177–182**]. Yiin and colleagues compared blood lead concentrations measured in children aged 6–32 months with three residential dust lead variables: concentrations of lead in dust obtained from floors, sills, and carpets; dust loading rates, which describe the amounts of dust typically found in each of these areas; and lead loading rates, described as the product of the dust loading rate and the lead concentration.

Blood and dust data were obtained from the Childhood Lead Exposure Assessment and Reduction Study (CLEARS), published in the September 1998 issue of the *Journal of Exposure Analysis and Environmental Epidemiology*, for which Yiin was an investigator. Samples for CLEARS were collected from families living in Jersey City, New Jersey, over a three-year period from 1992 to 1995.

Yiin found that both blood lead and residential dust lead concentrations were highest during warm months, peaking during the summer. Previous analyses performed during CLEARS indicated that nearly two-thirds of the lead in house dust was derived from outdoor sources. In the current study, Yiin attributes the high summertime amounts of leaded house dust to open windows and a greater overall frequency of indoor–outdoor movement. The best (strongest) correlations with elevated blood lead in the hot months were observed for floor lead loading, sill lead loading, and carpet lead concentrations. However, floor and sill loading data varied insignificantly from season to season, prompting Yiin to suggest that flaking paint chips contaminate floor and sill dust in a nonseasonal manner. In contrast, Yiin's study found that carpet dust and lead loadings were highest in the warm, cool, and cold months, and lowest during the hot months. This might reflect people's tendency to track mud and soil indoors during the winter. Carpet data were not strongly correlated with blood lead in the cold seasons, leading Yiin to suggest that summer lead sources may be more highly concentrated.

Summertime dust may raise the risk of childhood lead exposure.

Previously, researchers suggested that the increased blood lead concentrations observed in summer might result from biosynthesis of vitamin D by sunlight. Vitamin D promotes absorption of calcium and increases calcium concentrations in the blood; theoretically, it might have the same effect on lead, which has the same atomic properties as calcium. However, statistical studies performed in this study did not support this hypothesis. Rather, seasonal changes in blood lead were attributed almost exclusively to corresponding increases in residential house dust as well as to increased exposure to lead in soils during outdoor summertime play. **–Charles W. Schmidt**

This Is Your Placenta on Drugs

More Evidence against Maternal Drug Use

Maternal use of illicit drugs during pregnancy is a growing problem in modern society and one that appears to be causing increasing incidences of miscarriage and vaginal bleeding in mothers, as well as cognitive deficiencies in their offspring. During pregnancy, the placenta plays a vital role in producing and metabolizing a large number of steroids and hormones, which in turn regulate the health and development of the fetus. In this issue, Pauliina Paakki and colleagues demonstrate for the first time that abuse of illicit drugs alters the way the placenta functions as a steroid and hormone producer [*EHP* **108:141–145**].

The human placenta produces and metabolizes estrogenic steroids and metabolizes a host of foreign chemical agents, or xenobiotics. Cytochrome P450 enzymes play a particularly important role, metabolizing vitamins, fatty acids, and a wide range of medicinal drugs and chemical carcinogens. Thus far, research has primarily centered around the impact of cigarette smoking on placental xenobiotic metabolizing activities. Paakki's is the first study to demonstrate effects from maternal illicit drug abuse.

After collecting placental tissue from 13 drug-abusing mothers (women who through clinical history and/or urinalysis were determined to have used cannabis, methadone, opiates, cocaine, codeine, morphine, heroin, benzodiazepine, or barbiturates) at term and a control group of 24 nonabusing mothers, Paakki and colleagues conducted assays for microsomal protein concentrations including 7-ethoxycoumarin *O*-deethylase (ECOD), 7-ethoxyresorufin *O*-deethylase (EROD), pyrene 1-hydroxylase (P1OH), testosterone hydroxylase, UDP-glucuronosyltransferase (UGT), and glutathione *S*-transferase (GST). Comparisons were also made between the study group and cigarette smoking controls. According to classical analytical methods, no dramatic differences in metabolic or macroscopic characteristics between the study and control groups could be detected. However, using this extended panel of analysis containing P1OH and steroid-metabolizing (phase I) and UGT (phase II) activity determinations, some significant correlations were observed.

The researchers found that among maternal drug abusers, placental GST activity decreased in response to maternal drug load, a phenomenon identical to that resulting from exposure to industrial