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

The use of illicit drugs by pregnant women may disrupt placental metabolism and exacerbate the effects of smoking.

chemicals and ionizing radiation. While an earlier study by other researchers did not find any correlation between increased placental UGT activity and maternal cigarette smoking, this study found a strong correlation for mothers who were both drug abusers and smokers. This suggests that illicit drugs compound the problems associated with smoking.

The study also reports the discovery of a previously unknown placental biochemical marker activity. Among maternal drug abusers, P1OH correlated consistently with ECOD activity but not with EROD activity. However, P1OH correlated with EROD or testosterone hydroxylase activity only among the cigarette smoking controls. The authors suggest that because ECOD and EROD activity always exhibit a positive correlation in normal cigarette smoking conditions, this may be evidence that in normal conditions a "silent" cytochrome P450 gene form may be responsible for P1OH activity. This change in the steroid metabolism profile *in vitro* suggests that maternal drug abuse may alter normal hormonal balance during pregnancy.

The authors suggest that these findings may mean that the combination of illicit drugs with certain prescribed medications could also complicate the expression of placental metabolizing enzymes. They urge that term placentas from mothers using any medication or illicit drug be evaluated after delivery in terms of total metabolizing activity *in vitro* and any possible link to adverse responses in later development of the infant. **–John S. Manuel**

Challenging the Assumptions

Risk of Effects from Radiation

For decades, scientists have agreed that cancer risks are best estimated by extrapolating from the high-dose effects observed in a cohort of atomic bomb survivors over the course of the subjects' lives. In this issue, Alice Stewart, a retired professor of epidemiology from the University of Birmingham in Great Britain, questions the traditional assumptions used to form cancer risk assessments: namely, that the only late effect of radiation is cancer, that there is no cancer risk at the radiation levels typically faced by nuclear workers, that the risk for leukemia far exceeds that for solid tumors, and that radiosensitivity is higher at the beginning of adult life than at the end [***EHP* 108:93–96**]. Stewart also presents the alternative view that, in addition to cancer, high-dose exposures to radiation may also result in irreversible damage to the immune system.

New research using a cohort of atomic bomb survivors questions how assessments of cancer risk from radiation exposure are made.

Stewart's views challenge the assumptions behind the radiation risk coefficients (or slope factors) espoused by the National Academy of Sciences' Committee on the Biological Effects of Ionizing Radiation and the International Commission on Radiological Protection. These coefficients are based on analyses of life span study (LSS) data gathered by the Radiation Effects Research Foundation in Hiroshima, Japan, which concluded repeatedly that the LSS cohort was homogeneous and representative of the general population.

Stewart and colleagues discovered that the noncancer death rate in the LSS cohort was lower in the middle of the dose range than at either extreme, prompting them to perform an independent analysis. This analysis, published in the March 1998 issue of the *European Journal of Oncology*, revealed significant differences between survivors with and without acute injuries such as burns. It also showed that only in a small group of survivors with multiple injuries was the leukemia death rate higher than normal, and only in a much larger group of survivors with no acute injuries was the cancer death rate exceptionally high among people exposed as young adults. Based on this assessment, Stewart concludes that thousands of early deaths from acute effects of immune system damage left the LSS cohort biased toward those who were exceptionally resistant to late effects of radiation. Furthermore, because many early deaths may have been incorrectly attributed to causes other than immune system damage, it was not a homogeneous population, nor was it representative of the general population.

Turning to the effects of low-dose exposures, Stewart reviewed the Oxford Survey of Childhood Cancers, which was the first survey to find evidence of a cancer risk at low-dose levels, and earlier studies by herself and colleagues that first found evidence of a cancer risk for nuclear workers at the Hanford Nuclear Site in Washington State. Based on her assessment of both studies, Stewart concludes that the risk of leukemia is not exceptionally high following low-dose exposures and that childhood cancers are typically the result of *in utero* mutations that have teratogenic as well as carcinogenic effects. She also concludes that the low cancer death rates found at Hanford resulted from the selective recruitment of exceptionally healthy individuals into the nuclear industry.

Stewart notes that a new methodology in which the annual dose of each worker contributes separately to the total risk has identified a cancer risk among Hanford nuclear workers. Furthermore, Stewart found that sensitivity to carcinogenic effects of radiation is increased in young embryos and in people over 50. **–Charles W. Schmidt**