Letters to the Editor

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A Documented Case of Perinatal Lead Poisoning

We present an unusual case of lead poisoning from a ceramic pitcher, with complete prenatal and postnatal lead history and follow-up. The case illustrates important points for obstetricians, pediatricians, and public health workers who work with populations at risk and for professionals who examine lead-damaged children.

The mother was a patient in a long-term prospective lead study. After an uneventful pregnancy she delivered an apparently healthy girl with no complications. The only unusual finding was an extremely high cord plasma beta-endorphin level (1064 pg/ml). The child was constipated in the hospital, and the first and subsequent bowel movements were manually stimulated.

During the pregnancy the mother's blood lead level oscillated around the mean for the study sample (10 µg/dl) until week 36 (Figure 1). Laboratory processing delays brought the abrupt rise in maternal lead level after week 36 to our attention 6 weeks after delivery. The mother was at that time being discharged from a readmittance with complaints of lumbar pain radiating to the abdomen, paresthesia in the lower extremities, nausea, occasional vomiting, and hypertension. Initial diagnoses of pyelonephritis, alteration of the dorsal lumbar spinal column, or lithiasis were not confirmed in clinical and laboratory work-up, and she was released when symptoms diminished.

Upon interview, she disclosed she had purchased at week 36 a glazed ceramic pitcher for mixing and storing lemonade. The glazed interior finish of the pitcher had deteriorated and reflected a rainbow of colors. Using FDA procedures for testing releasable lead in hollow ware we found 933 ppm of lead in the elution by argon plasma emission spectrometry.

Although the family stopped using the pitcher and was counseled about breast-feeding, nursing continued, as a method of behavioral control, until the baby was 27 months old. After being refused admittance at the only available clinic for lead detoxification because there were no symptoms, the child was examined by the family pediatrician. He told the family the child was perfectly normal and the mother should not be told anything more about lead because it was upsetting her. We secured family permission to continue seeing the child only on condition that we would not discuss lead.

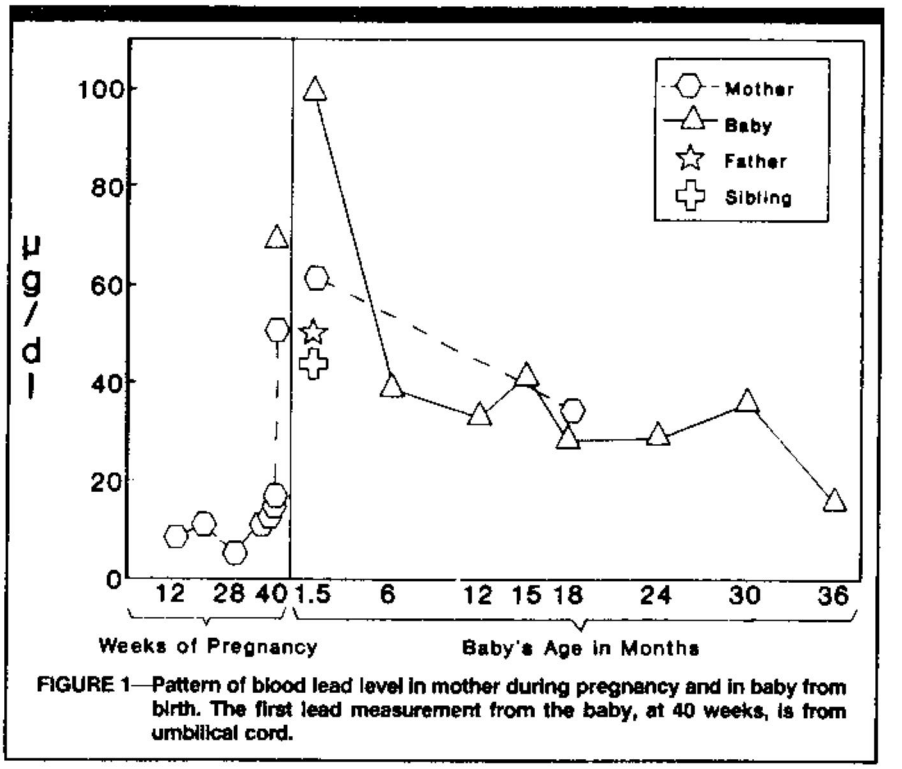

FIGURE 1—Pattern of blood lead level in mother during pregnancy and in baby from birth. The first lead measurement from the baby, at 40 weeks, is from umbilical cord.

Brazelton Neonatal Behavioral Assessment Scales and the Graham/Rosenblith neurological soft signs scale showed hypertonia, irritability, abnormal cry, and other neurological soft signs at 2, 15, and 30 days. Brainstem auditory evoked responses and clinical EEG were essentially normal at 20 days, 3, 6, and 12 months. EEG sleep pattern was fragmented at 20 days and 3 months, and abnormal respiratory patterns were noted to 6 months. Psychometric (Bayley Scales, Terman Merril and McCarthy Scales) and diagnostic testing (Fagan Test of Infant Intelligence) yielded scores within normal limits out to 3 years. At every examination, however, testing protocols noted some combination of restlessness, agitation, distractibility, high energy level, lack of persistence, short attention span, and poor fine motor control. The mother describes the child as very difficult, with low frustration tolerance.

Lead-glazed ceramic ware, common in Mexico, is widely used by Hispanics in the United States, who often bring it from Mexico.[1,2] Tourists import such items as gifts, and the number of recalls of commercially imported ceramic ware likely underestimates the quantity of leaded items available.[3]

Uninformed physicians can adversely affect treatment of lead poisoning. Toxic levels of lead produce symptoms that can be confused with other disorders. The pattern of hospital admission, unconfirmed diagnoses, reduction of symptoms when the patient is removed from the lead source during hospital stay, discharge, and re-exposure has been noted before.[4] Mothers with high lead levels expose their infants through maternal milk. Standardized psychometric tests are frequently without value in detecting damage from lead in children up to 3 years, even though behavioral disturbances are clear. □

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Could Sunscreens Increase Melanoma Risk?

Topically applied chemical sunscreens prevent sunburn.[1] One of the most common sunscreens, para-aminobenzoic acid (PABA), was invented in 1922, and commercial products containing sunscreens became available in 1928.[2] High sun protection factor (SPF) sunscreens, largely based on PABA and its esters, became widely available by the late 1960s and early 1970s.[3,4] High SPF sunscreens have been widely recommended for the prevention of skin cancer, including melanoma.[5–7] It has been assumed that the action spectrum for initiation and promotion of melanoma and basal cell carcinoma is identical to that of sunburn.[8] Sunscreens have been strongly recommended for persons with fair coloring and those with a history of skin cancer,[9,10] and use of sunscreens has become widespread. A large proportion of adults in the United States report using sunscreens during recreation,[11] and the American Medical Association has recommended that frequent use of sunscreens should become a standard procedure for children.[6]

Although sunscreens, including PABA and its esters prevent sunburn,[1,2,12] there has never been any epidemiological or laboratory evidence that they prevent either melanoma or basal cell carcinoma in humans.

Worldwide, the countries where chemical sunscreens have been recommended and adopted have experienced the greatest rise in cutaneous malignant melanoma, with a contemporaneous rise in death rates. In the United States, Canada, Australia, and the Scandinavian countries, melanoma rates have risen steeply in recent decades, with the greatest increase occurring after the introduction of sunscreens.[13–17] Death rates in the United States from melanoma doubled in women and tripled in men between the 1950s and the 1990s.[18] The rise in melanoma has been unusually steep in Queensland, Australia, where sunscreens were earliest and most strongly promoted by the medical community.[19] Queensland now has the highest incidence rate of melanoma in the world.[20] In contrast, the rise in melanoma rates was notably delayed elsewhere in Australia,[20] where sunscreens were not promoted until more recently.

The SPF of sunscreens concerns solely their ability to absorb ultraviolet B (UV-B) light.[21] Even sunscreens with high SPF factors can be completely transparent to ultraviolet A (UV-A),[21] which includes 90% to 95% of ultraviolet light.[22] UV-A blocking ingredients, which have commonly been added to most sunscreens since 1989, block only half the UV-A spectrum and provide a protection factor against delayed UV-A induced erythema of only 1.7 at usual concentrations.[23]

Both UV-A and UV-B have been shown to mutate DNA and promote skin cancers in animals.[24,25] UV-A also penetrates deeper into the skin than UV-B.[26] Because of the energy distribution of sunlight[22] and filtering by the outermost layers of the skin,[26] melanocytes receive up to 70 photons of UV-A for every photon of UV-B.

While largely transparent to most of the UV-A spectrum, sunscreens effectively block UV-B. UV-B is the normal stimulus for accommodation of the skin to sun, such as thickening and increased pigmentation.[27] Sunscreens also inhibit the skin's production of vitamin D, which is similarly dependent on UV-B.[28] Laboratory findings indicate that vitamin D metabolites suppress growth of melanoma cells,[29] suggesting the possibility that vitamin D deficiency in the skin may have a role in the etiology of melanoma.

While few epidemiologic studies have examined the relationship of sunscreen use and skin cancer, two studies suggest that sunscreens may not be effective in preventing skin cancer. A large