Iron deficiency in children with lead exposure

To the Editor:

There is an inherent uncertainty with observational studies of blood lead levels. A given blood lead level could be due to a specific amount of lead available for absorption or a lower amount of lead entering the body but a greater rate of absorption. Although there are clear animal data that iron deficiency increases lead absorption,[1] controlled studies of humans provided mixed results.[2,3] The hypothesis of the report by Serwint et al seems to be that the probability that a child with an elevated blood lead level will be iron deficient is the same as that of a child with a low blood lead level.[4] This was tested by analyses of differences between two groups defined by blood lead levels with parameters defined by serum ferritin alone or serum ferritin and hematologic criteria. They concluded that blood lead levels of 20 to 44 μg/dL should not be the sole criterion to initiate further testing or intervention.

In a review of iron deficiency cited by the authors, Oski counseled, "Because many of these tests (of iron deficiency) lack specificity, several tests are often used when rigorous definition is required."[5] A serum ferritin value of >10 μg/dL will fail to identify more than half of the children with a hemoglobin value of <11.5 gm/dL who are iron deficient as defined by a therapeutic response to iron supplementation.[6] The burden of iron deficiency is amplified for children who have lead toxicity, since there may be increased adverse effects from the same blood lead levels with iron deficiency,[7] as well as a possible effect on lead absorption. In situations of grave impact, such as the conclusion of Serwint et al, certainly a more rigorous definition of iron status should be used.

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Reply

To the Editor:

Dr Waldron expressed concern about the definitions we used for iron deficiency and iron deficiency anemia in our study.[1] To our knowledge, there is no unequivocal gold standard for diagnosing iron deficiency. We agree with Oski's statement that "Because many of these tests lack specificity, several tests are often used when a rigorous definition is required."[2] It is for this reason that we chose multiple definitions of iron deficiency and iron deficiency anemia, using up to 3 different hematologic measurements, which were suggested by Oski.[2] Since there are data to suggest that mild antecedent infections can cause transient anemia,[3] response to iron supplementation challenges in the face of recent infections may not be diagnostic of iron deficiency, but rather, may show a coincidental increase in hemoglobin after recovery. Our study compared children with moderate lead exposure with those with low exposure and, implementing commonly used measures of iron deficiency, demonstrated that there were no significant differences between the groups for any of the definitions used.

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On mice and men: An autosomal recessive syndrome of chronic recurrent multifocal osteomyelitis and congenital dyserythropoietic anemia

To the Editor:

In 1989, we reported on 3 related children with chronic recurrent multifocal osteomyelitis (CRMO) and congenital dyserythropoietic anemia (CDA) (Figure: IV-6, IV-10, IV-11). Two of them were brothers (IV-10 and IV-11), and their illness was also associated with Sweet syndrome.[1] Since then, a fourth child was born in this family with CDA and CRMO (IV-8). The parents of one sibship divorced, and the father remarried a non-related woman and had 3 healthy children (IV-1, IV-2, and IV-3). All patients were Palestinian Arabs.

Over a period of 9 to 16 years of follow-up, the course of CRMO in our patients was characterized by an early onset and dominated by frequent episodes, ranging between 1 and 3 per month, sometimes associated with mild fever. The 4 children failed to thrive; height and weight were below the 5th