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## **Choosing the Best Strategy to Prevent Childhood Iron Deficiency**

[Editorial]

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### **Outline**

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Twelve years ago, in the pages of THE JOURNAL, Stockman [\[1\]](#) celebrated a series of victorious battles against iron deficiency, but he warned that the war was far from over. He had good reason for both joy and caution. Following the introduction in 1972 of the Special Supplemental Food Program for Women, Infants and Children, iron deficiency anemia virtually disappeared in some populations of high-risk children. For example, the prevalence of moderate or severe anemia (hemoglobin levels <98 g/L) in young children in the inner city of New Haven decreased from 23% in 1971 to 1% in 1984. [\[2\]](#) From 1975 to 1985, the prevalence of anemia in young children enrolled in state public health programs monitored by the Centers for Disease Control Pediatric Nutrition Surveillance System declined from 7.8% to 2.9%. [\[3\]](#) Interventions other than the program for women, infants, and children, such as an increased emphasis on breast-feeding, delayed introduction of cow's milk, and improved iron fortification of infant foods, contributed to the better iron status of children in low-income families, and produced similar gains among middle-

class children. In a private pediatric practice in Minneapolis, the rate of anemia decreased from 6.2% in 1969-1973 to 2.7% in 1982-1986. [4] Government agencies, policymakers, physicians, and the food industry could point with pride to the success of their efforts.

However, Stockman also saw reason for concern. What about children who did not qualify for the women, infants, and children program and who might not be reached by other interventions, many of which depended on regular medical care and access to educational materials? Moreover, was the lower prevalence of iron deficiency anemia counterbalanced by emerging knowledge about the serious, long-term effects of this disorder? Regarding the first question, recent studies have found an alarming number of cases of severe iron deficiency anemia in young children. For example, Morad [5] reported 74 cases of children with hemoglobin levels less than 75 g/L occurring over 6 years. Kwiatkowski et al [6] documented 55 cases of children with hemoglobin levels less than 60 g/L occurring over 10 years. Many of these children belong to ethnic groups that may experience language, cultural, and other barriers to enrollment in public health programs or receipt and understanding of educational materials. [5,6] These findings suggest a reemergence of this disorder or possibly a redistribution among underserved populations.

Data addressing the question about systemic effects of iron deficiency are equally disturbing. Numerous studies, summarized by Oski [7] and by Booth and Aukett, [8] have demonstrated an association between iron deficiency anemia and impairment of mental and motor development in childhood. In addition, restoration of a normal hemoglobin level with iron replacement therapy may not fully reverse these changes. Children with moderately severe iron deficiency anemia at age 12 to 23 months still lagged behind their peers in mental and motor development at age 5 years, despite early correction of their anemia. [9] Thus, while the prevalence of iron deficiency anemia may be declining, the importance of early detection and treatment is increasing as its consequences are more fully understood.

In this issue of THE JOURNAL, Brugnara and colleagues [10] propose one solution to these remaining problems, namely a better screening test for iron deficiency. [10] These investigators have demonstrated the usefulness of the reticulocyte hemoglobin content (CHr) in identifying children with either iron deficiency or iron deficiency anemia. In their study, CHr proved more useful than such conventional tests as the ferritin level or red blood cell zinc protoporphyrin concentration, and it even outperformed the recently introduced transferrin receptor assay. When incorporated into an automated hematology analyzer, the measurement of CHr is an inexpensive and rapidly available alternative to biochemical iron studies for the detection of iron deficiency before the onset of anemia.

Some aspects of the study suggest the need for expanded trials of the clinical application of CHr. For example, 20% of the sample of 210 children had anemia with normal transferrin saturation, indicating the possible presence of confounding hematologic disorders. Perhaps the high rate of anemia in the control group helps explain the surprising diagnostic limitations of the time-

proven ferritin level or the highly touted transferrin receptor assay. Moreover, even at the chosen cutoff value of 26 pg, the sensitivity (70%) and specificity (78%) of CHr leave room for improvement to detect more affected children and to avoid unnecessary treatment and follow-up testing in healthy children. Nonetheless, in this pilot study, CHr appears to have the highly desirable ability to combine the ease of measurement of an automated hematologic parameter with the detection of an earlier stage of iron deficiency that usually requires more expensive biochemical assays.

The value of the CHr as an early screening test will increase exponentially if future studies demonstrate convincingly that adverse effects of iron deficiency, particularly those involving the brain, begin before anemia develops. At present, the link between nonanemic iron deficiency and delayed development remains uncertain, and the occurrence of irreversible mental or motor impairment at this early stage of iron deficiency is unproven. [8] Nonetheless, one could develop a rationale for early detection of iron deficiency with the CHr as a strategy for identifying children before their deficiency progresses to a stage with known hematologic and systemic effects. Field trials to determine the clinical utility of this approach are essential.

On the other hand, one might argue that further gains in the prevention of iron deficiency in infants and children will come from new public health initiatives rather than improved technology. After all, the extraordinary reduction in the prevalence of iron deficiency anemia in the past 3 decades was attributable to better nutritional advice and support, not to the development of new laboratory tests. For instance, a 2-year-old Vietnamese girl admitted recently with a hemoglobin level of 30 g/L and a mean corpuscular volume of 47 fL did not need better screening tests for iron deficiency. Instead, she needed earlier recognition and correction of a diet consisting mainly of cow's milk and rice, and she needed to receive the benefits of nutritional support programs despite the fact that her parents speak only Vietnamese. For children fortunate enough to receive regular pediatric care, careful dietary history taking, appropriate counseling, and targeted screening with a hemoglobin level may be sufficient to prevent the hematologic and other consequences of iron deficiency. Better tactics rather than more sophisticated weaponry ultimately may be the key to eradication of childhood iron deficiency.

Editorials represent the opinions of the authors and THE JOURNAL and not those of the American Medical Association.

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