Studies of Marginal Zinc Deprivation in Rhesus Monkeys: (IV) Growth of Infants in the First Year and (V) Fetal and Infant Skeletal Effects

Growth retardation, delayed skeletal maturation, and defective bone mineralization are reported in rhesus monkeys subjected to marginal zinc deprivation during the prenatal period and throughout the first year of life. The subjects were offspring of mothers given either a zinc-deficient diet (4 mg/kg of zinc) or a control diet (100 mg/kg of zinc) throughout gestation and lactation. At weaning, the offspring were fed either a zinc-deficient or control diet corresponding to the maternal diet. A complete morphometric examination, a quinine acceptance test for taste sensitivity, blood samples for trace metals, and bone x-rays were performed at various intervals during the first year of life.

At birth, zinc-deficient males had significantly lower body weights, crown-rump lengths, and femur lengths than control males. These data suggest intrauterine growth retardation. Reduced rates of weight gain and crown-rump length growth were reported in the zinc-deficient group compared with controls at 9 to 12 months of age. The diminished rate of weight gain was positively correlated with reduced food intake, lower food-use efficiency, and decreased taste acuity at 1 year of age. Overall, zinc-deficient infants did not grow as well as the controls during the entire first year of life.

At birth, zinc-deficient infants demonstrated delayed skeletal maturation without defective mineralization. However, by 1 month of age, abnormal mineralization was reported in the zinc-deficient group. Specifically, there were changes suggesting rachitic syndromes with "frayed" metaphyses and "splayed" cortices.

As a result of these observations, the authors suggest that marginal zinc deficiency during gestation results in neonatal growth retardation that persists throughout the first year of life. Bone maturation delay and defective mineralization of the skeletal system also result from zinc deprivation.

Golub MS, Gershwin ME, Hurley LS, et al: *Am J Clin Nutr* 1984;40:1192; and Leek JC, Vogler JB, Gershwin ME, et al: *Am J Clin Nutr* 1984;40:1203.

Editor's comment*—These observations are important since they demonstrate that marginal zinc deficiency can lead to growth abnormalities in utero and to defective skeletal growth. These abnormalities resulted without inducing hypozincemia, but the mean values of the plasma zinc levels were lower in the zinc-deficient monkeys than in the control animals. Unfortunately, the maternal plasma zinc levels are not reported.*

These data also demonstrate the need for zinc in skeletal mineralization and the regulation of bone formation. Radiographic findings of rickets were associated with zinc-deficient diets. Unfortunately, vitamin D levels were not obtained. The above observations may be important clinically, since decreased intake of dietary zinc is often seen during pregnancy. In childhood, marginal zinc deficiency is often seen in conditions associated with poor growth.

Fear of Obesity: A Cause of Short Stature and Delayed Puberty

Fourteen of 201 children evaluated for short stature and/or delayed puberty over a 25-month period were found to fit a pattern of growth failure due to self-imposed restriction of caloric intake arising from a fear of becoming obese. Nine males and five females, ages 9 to 17 years, underwent a complete evaluation. They were all below the fifth percentile for weight and height. All showed deterioration of linear growth, which was preceded by one to two years of inadequate weight gain. The weight deficit for height was 5% to 23% of ideal body weight.

Seven of the older patients had delayed puberty. Physical examination and routine diagnostic laboratory examinations revealed no evidence of organic disease.

Review of the patients' 24-hour dietary intake by recall indicated that they ingested only 32% to 90% of the recommended caloric intake for age and sex. Nine skipped meals regularly. They tended to reduce the amount of animal proteins in the diet, but consumed increased amounts of cereals, fruits, and vegetables. The seven-day record in nine patients supported the data obtained by recall.

An open-ended interview of all patients revealed no evidence of psychiatric disease or anorexia nervosa. As a group, these patients were good students with compulsively shy personalities. Seven underwent the Diagnostic Interview for Children and Adolescents, which also revealed no psychiatric disease. Three patients did show evidence of an oppositional disorder (usually, argumentative or confrontational behavior).

After receiving nutritional counseling and nonstructured psychiatric counseling, the patients resumed an adequate caloric intake for age. Weight gain and a resumption of linear growth accompanied increased food intake, except in one female who underwent menarche and remained stunted.

Pugliese M, Lifshitz F, Grad G, et al: *N Eng J Med* 1983;309:513-518.

Editor's comment*—This paper describes a newly recognized cause of poor growth in adolescence. It remains to be seen whether fear of obesity, which may be prevalent in our population because of concern over being fat, is a distinct disease entity with its own natural history. This entity could also be a mild variant of anorexia nervosa. Whether a caloric deficiency or the inadequate intake of a specific nutrient caused the poor growth remains unclear.*