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# GROWTH

## Genetics & Hormones

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### Nutrition, Growth, and Growth Failure

Nutritional causes of short stature and/or poor growth often remain unrecognized by pediatricians and pediatric endocrinologists even though the need for adequate weight gain and body fat to sustain growth during puberty is well described. Although undernutrition resulting from the unavailability of food or psychosocial deprivation accounts for most cases of growth retardation throughout the world, it is a rare cause of short stature in the United States.

When a nutritional deficiency is suspected as a possible cause of short stature, the physician should first ascertain whether the deficit is due to decreased intake resulting from increased energy metabolism, or to increased caloric loss of protein or fat via the stool. A dietary history and/or a brief period of observation in a hospital usually reveals whether there is a decreased intake of calories and/or substrates, hyperactivity, or abnormalities of the gastrointestinal (GI) tract. Anorexia, which can occur as a nonspecific phenomenon secondary to disease or as a primary psychological disorder, is a classic example of poor intake. Nonspecific causes of anorexia—iron deficiency, for example—are seen during infancy and childhood.

**Iron deficiency** is the end result of an imbalance between the sum of the patient's iron endowment, intake, and absorption, and the sum of his iron needs for growth and replacement of losses. The peak incidence of iron deficiency in childhood is between 6 months and 1 year of age; another such peak is seen during early adolescence. The average American diet provides only 15 to 18 mg of iron per day, of

which only an average of 10% is absorbed. The normal daily requirement of elemental iron is 15 mg for an adolescent. It is therefore not surprising that as many as 10% of children have been found to have iron-deficiency anemia. Iron deficiency can also account for anorexia in some high school students.

In addition to looking for evidence of anemia, physicians should also determine serum iron levels, total iron binding capacity, and ferritin levels in infants with failure to thrive

and in older children with anorexia. If iron deficiency is found, one should try to determine whether it is the initiating cause of the anorexia or the result. Iron replacement over a period of two to three months may improve growth and appetite regardless of the etiology of the iron deficiency.

**Gluten sensitivity (celiac disease)**

is also associated with low caloric intake and may be associated with  
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### Problems in Assessing the Efficacy of Growth-Promoting Substances: The Role of Height Prediction

There are two objectives in treating children with growth-promoting substances such as growth hormone (GH) or anabolic steroids. The primary objective is to promote an increase in the child's ultimate height so that he will be taller than he otherwise would have been. The secondary objective is to accelerate the rate of growth, and thus permit the child to achieve an adult height sooner, even if the ultimate height

remains unchanged. To attain the second objective, the child must grow at an increased rate over a sustained period, but the increased rate must not diminish the ultimate adult height.

Growth hormone made by recombinant DNA techniques will soon be available, and the opportunity—or temptation—to use it on children who are short, but not GH deficient (GHD), will present itself. The problem is to know whether trials assessing GH in children have been successful and whether there is evidence, at the end of a single year, that GH administration is likely to achieve one or both of the aforementioned objectives.

The first difficulty with these questions relates to the compensatory deceleration of growth observed in GHD patients who are taken off GH therapy. In the year after the first year of therapy, the growth rate is

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anemia as well. Onset most often occurs during infancy. In Europe, much attention has been given to the association between celiac disease without significant GI symptoms and short stature. It is therefore reasonable to suspect celiac disease in children who are short without an adequate explanation. After the child has been challenged with a high-gluten diet for four to six weeks, an intestinal biopsy to confirm the diagnosis of celiac disease can be considered (see page 10 of this issue).

Adolescent girls with **anorexia nervosa** of the characteristic type are well recognized by most pediatricians and pediatric endocrinologists. The associated endocrine abnormalities found in these patients are related primarily to undernutrition and are similar to those seen in people with severe caloric deficiency in third world countries. Gonadotropins and somatomedin-C determinations are usually low. Growth hormone levels are usually normal. Since Crohn's disease can masquerade as anorexia nervosa, it should be considered in the differential diagnosis.

Similar to, but certainly not identical with, typical anorexia nervosa is "**fear of obesity**," so termed because it leads to self-imposed malnutrition (see page 9 of this issue). Numerous lay and scientific journals have published articles describing poor growth and delayed development in adolescents who have severely restricted their food intake or gone on fad diets. We recently recognized a group of 14 patients who failed to grow because of self-imposed malnutrition that was rooted in a fear of becoming obese. Their diets were deficient in calories, minerals, and vitamin D. After at least one year of inadequate weight gain, these patients showed signs of deteriorating linear growth and failed to attain puberty, the latter a characteristic also of anorexia nervosa. No organic causes were identified. Once the fear of obesity was recognized, the patients were given nutritional and psychological counseling. They resumed an adequate

caloric intake for their age and recovered, as demonstrated by improved linear growth and progression of adolescent sexual development. Only one patient had a permanent alteration of height potential, probably because of delayed diagnosis and treatment. Menarche occurred in this patient soon after adequate weight gain had been established, but her height increased only minimally.

These patients appear to differ from those with other bariphobic syndromes. They had not lost significant amounts of weight, but rather had ceased to gain weight as they progressed along previously defined height percentiles. They also did not have a distorted body image; they realized they were slim and wanted to stay that way. In contrast, patients with true anorexia nervosa lose weight rapidly over a short period and usually see themselves as heavy even though they are markedly undernourished.

Unlike classic anorectics, these patients fearing obesity had no self-induced vomiting, did not abuse laxatives or diuretics, did not exercise compulsively, and did not hoard food. We believe that fear of obesity as manifested in these patients represents an exaggeration of our social concerns with achieving and maintaining an "ideal" trim figure. The incidence of this syndrome is unknown, especially since patients with mild forms of the disorder may not even attract medical attention. Interestingly, we have recently identified infants with failure to thrive because of inadequate nutrition (calories were inappropriately withheld) stemming from parental concern about obesity in their children.

**Chronic inflammatory bowel disease** (CIBD) is another condition that retards linear growth. Growth failure and sexual infantilism (prevalence 30% to 85%) are major complaints in many adolescents with CIBD. Children, however, may be asymptomatic and present primarily for short stature and delayed development. Digital clubbing, aphthous stomatitis, arthritis, or pyoderma gangrenosum are clues to the un-

derlying GI pathology in short-statured patients with CIBD. Growth may slow down or cease without any other sign or symptom, sometimes for more than three years before GI complaints appear. Therefore, CIBD should be considered as a cause of inadequate growth even in the absence of GI complaints. Gut motility studies are helpful in confirming the diagnosis, as is an abnormal sedimentation rate, although not all patients with CIBD have abnormal rates.

Children with **Crohn's disease** or **ulcerative colitis** may not grow normally because of impaired nutrient absorption, decreased nutrient intake, specific nutrient deficiencies, or increased protein losses through the GI tract. Glucocorticoid excess during treatment with steroids is another cause. While some children with CIBD have intestinal malabsorption, the majority do not have significant steatorrhea and are able to absorb xylose normally. Thus, malabsorption of nutrients does not fully explain the poor growth in most of these patients. Anorexia, however, plays a significant role in patients who have abdominal pain following meals and who may also be losing protein through the GI tract.

Nutritional rehabilitation often promotes growth in growth-retarded children with CIBD. Short-term parenteral nutrition in a hospital, as well as long-term total parenteral nutrition at home, can produce marked increases in height and catch-up growth. Oral feedings may also promote catch-up growth if enough nutrients are ingested. On occasion, nutritional rehabilitation has induced remission of the disease, suggesting that adequate nutrition is needed to control it. Appropriate nutrition may also be necessary for medications such as sulfasalazine or steroids to exert their therapeutic effects, which in turn may permit the resumption of normal growth.

**Zinc metabolism and deficiency** are associated with a number of clinical syndromes. Moreover, many recent articles have implicated zinc deficiency as a cause of growth retardation.

Zinc is an essential nutrient. Adolescents and adults require 15

mg/d; infants and children require 3 to 5 mg/d in their first year and 10 mg/d until early adolescence. Zinc deficiency may result from malabsorption states, or it may develop during total parenteral nutrition or along with cirrhosis of the liver and renal disease. Symptoms of mild to moderate zinc deficiency include diminished taste sensitivity, anorexia, and growth retardation. Acrodermatitis enteropathica, diminished cellular immunity, and poor wound healing may also indicate zinc deficiency. High concentrations of dietary phytate (as seen in the typical Iranian or Egyptian diet) can diminish the availability of zinc and precipitate zinc deficiency syndromes. The clinical diagnosis of zinc deficiency can be confirmed by a low concentration of *plasma* zinc. Zinc levels in hair are unreliable indicators of deficiency.

In summary, physicians must recognize the important role that nutrition plays in normal and abnormal growth. Indeed, nutritional causes of growth disturbances may be as obscure and subtle as endocrine causes such as partial growth hormone or thyroid deficiency.

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References will be supplied upon request to Dr. Blizzard.

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