

Growth Rate Reduction During Energy Restriction in Obese Adolescents

Amador et al studied the effects of energy restriction on growth and sexual development in a group of obese children who were participating in a multidisciplinary weight-loss program. Ninety-four children whose relative fat weight was determined to be above 25% but less than 40% in males and above 30% but less than 45% in females were studied. These children, aged 10.6 to 12.9 years, were all in Tanner stage II puberty and their body weight for stature was above the 97th percentile. The children were randomly classified into 2 groups: a control group in which energy intake was maintained (0.25 mJ/kg of expected body weight for height) and an experimental group in which energy intake was restricted to 30% of energy requirements (0.17 mJ/kg of expected body weight for height). All children participated in a program of physical activity, nutritional education, and behavioral modification. All subjects were measured and examined for height and stage of sexual development at the end of 6 months and again at 6 months

following the intervention. Seventy-eight children completed the 1-year study.

No differences between the 2 groups were found with respect to Tanner stage, body weight, lean body weight, or fat body weight at the initiation of the study. However, after 6 months of therapy, puberty progressed at a significantly slower rate in the group with the lower energy intake. In addition, there was a significantly greater reduction in body weight in this group, with significantly greater loss of fat body weight than in the control group. The height velocity was also significantly slower in the energy-restricted group. During the subsequent 6 months, catch-up growth was evident in the energy-restricted group, but pubertal development continued to lag behind the group with less restriction of energy intake.

The authors suggest that the restriction of energy intake in early adolescence should be avoided in the dietary management of overweight early adolescent children. They suggest that a nonrestrictive diet with the addition of physical activities, nutritional education,

and behavioral modification is a more appropriate method for achieving weight loss in this group.

Amador M, Ramos L, Morono M, et al. *Exp Clin Endocrinol* 1990;96:73-82.

Editor's comment: *This very interesting paper demonstrates once again the need for monitoring linear growth during weight-reduction therapy in children. Dietz et al (AJCD 1985;139:75) demonstrated previously that diets with even a mild restriction of energy may be associated with a reduction in height velocity. The present study confirms this finding and, in addition, demonstrates a reduction in the tempo of pubertal development in children whose energy intake is restricted. They have demonstrated that a multidisciplinary program of exercise, education, and behavioral modification is exceedingly important to weight reduction programs in children.*

William L. Clarke, MD

Identification of the 64K Autoantigen in IDDM as Glutamic Acid Decarboxylase (GAD)

An antigen previously found only in the beta cells of the islet cells here is reported also to be present in certain neurons that secrete gamma-aminobutyric acid (GABA) in the central nervous system (CNS). This antigen is identified as glutamic acid decarboxylase (GAD), the biosynthesizing enzyme of the inhibitory neurotransmitter GABA.

Individuals with stiff-man syndrome (SMS) frequently have associated insulin-dependent diabetes mellitus (IDDM). Individuals with SMS have autoantibodies to 64K antigen at much greater titers than do most patients with IDDM. The authors found that the GAD antibodies in SMS were 10 to 200 times higher than those found usually

in IDDM patients. A reference by Solimena et al in the *New England Journal of Medicine* in 1990 is quoted.

The authors used acceptable immunologic and microbiologic techniques to demonstrate that the 64K antigen in the beta cells and in the GABA-secreting neuron cells is the same. This finding is expected to motivate the creation of studies to elucidate the pathogenesis of IDDM and SMS, and to determine the mechanisms of generation of self-tolerance by the immune system and its failures.

The authors suggest there are components other than the GAD antibodies responsible for these diseases. For example, beta cells express major histocompatibility complex class I

molecules whereas CNS neurons normally do not.

Baekkeskov S, Aanstoot HJ, Cristgau S, et al. *Nature* 1990;347:151-156.

Editor's comment: *The pancreatic 64K beta-cell autoantigen is a major target of autoantibodies associated with IDDM. The finding that this antigen is identical to that found in GABA-secreting neurons is a significant contribution. The details as presented in the abstract above are convincing. Those readers who are the least bit interested in the possible role of autoimmunity as a cause of IDDM can benefit by reading the entire article.*

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