

## Special Report:

# Annual Scientific Meeting of the American Diabetes Association— June 6-9, 1987, Indianapolis, Indiana

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The plenary sessions of this annual meeting included symposia on lipid metabolism, insulin action, and the mechanisms and management of diabetic neuropathy. The Banting Memorial Lecture, given by Dr. Joseph Lerner (Charlottesville, Virginia), concerned insulin signaling mechanisms.

Several papers that were presented are of particular interest to those who are studying growth. MacGorman (Rochester, Minnesota) presented a paper on the importance of growth hormone in the maintenance of basal lipolysis in normal man. Using a study design in which somatostatin, insulin, glucagon, and glucose were infused in amounts necessary to maintain euglycemia, MacGorman et al infused labeled glucose and palmitate into seven normal human volunteers. Human growth hormone (hGH) was then administered either by constant infusion or by hourly boluses. When hGH was administered in a pulsatile fashion, palmitate levels were higher than when hGH deficiency was induced. However, no differences in glucose metabolism were observed. These results indicate that hGH is important in the maintenance of basal lipolysis during the night in normal volunteers and that free fatty acid metabolism may be more sensitive than glucose metabolism to hGH.

Jacob (New Haven, Connecticut) presented a paper entitled "Effect of IGF-I to Lower Blood Glucose and Its Effect on Hepatic Glucose Production." In this study, fasted rats were infused with either saline or recombinant human IGF-I (THR59) while simultaneously being infused with tritiated glucose; these infusions were done to determine hepatic glucose production. It was demonstrated that IGF-I produced hypoglycemia in rats by selectively enhancing peripheral glucose uptake. Liver glucose

metabolism was relatively unresponsive to IGF-I in comparison with insulin, which suggests that IGF-I and insulin affect hepatic glucose production by different cellular mechanisms.

In a paper entitled "Diabetes Mellitus Influences Growth by Regulating Hepatic Insulin-Like Growth Factors I and II Gene Expression," Yang (Madison, Wisconsin) compared the effect of streptozotocin-induced diabetes on the growth rate of young rats and on the transcription and translation of IGF-I and IGF-II. Although serum IGF-I levels correlated positively with hepatic mRNA and negatively with blood glucose concentrations, neither relationship held for IGF-II. Yang et al concluded that hepatic IGF-I mRNA, serum IGF-I levels, and growth rate are decreased by poorly controlled diabetes and are normalized by insulin therapy. In contrast, IGF-II synthesis and release are only slightly altered, suggesting that this somatomedin is less important in growth regulation.

Two papers concerning GH and diabetic retinopathy were presented. The first, by Shumak et al (Toronto, Ontario), was entitled "The Effect of Growth Hormone Suppression on Established Proliferative Diabetic Retinopathy." Four Type I diabetics with preproliferative retinopathy and varying degrees of macular edema received eight weeks of therapy with a long-acting somatomedin analog (SMS 201-995). Glycosylated hemoglobin  $bA_{1c}$  concentrations did not change during the study, while 24-hour integrated GH concentrations declined by about 42%. Visual acuity improved in all eight eyes but was not associated with detectable morphologic changes on stereofundus photography or fluorescein angiography. Within two months of discontinuing ana-

log treatment, visual acuity returned to pretreatment levels. The mechanism by which visual acuity improved is unclear, although subtle changes in the degree of macular edema may have occurred.

The second paper, presented by Sundkvist (Sweden), was entitled "Absent Elevations in Growth Hormone and Endothelial Factors During Exercise Predict a Resistance Against Retinopathy." Plasma levels of GH, endothelial factors, and GH factor VIII-related antigen, and plasminogen activator activity were recorded during exercise in 22 patients with insulin-dependent diabetes. The patients were reevaluated five to seven years later for the absence or presence of retinopathy in relation to previous exercise test results. Patients with retinopathy at follow-up showed significant elevations in GH factor VIII-related antigen and plasminogen activator activity during exercise. In contrast, patients without retinopathy at follow-up did not show significant elevations of these values during exercise. Sundkvist et al concluded that the absence of elevations in GH and endothelial factors during exercise may predict resistance against retinopathy in patients with insulin-dependent diabetes mellitus.

Further information regarding these and other papers concerned with growth can be found in *Diabetes* Vol. 36, Supplement I, 1987.

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