Comparison of insulin degrading activity of human kidneys and liver.

Since the liver and kidneys were obtained from the same human subject, it was of interest to compare total insulin-degrading activity of the two organs. Dialyzed extracts were prepared from the acetone powders and their insulin-degrading activity was determined using the same preparation of I-125-insulin. Kidneys were found to possess only one tenth of the insulin-degrading activity per unit protein compared with liver.

ACKNOWLEDGMENT

This work was supported by a Research Grant A-3854 from the National Institute for Arthritis and Metabolic Disease, U.S. Public Health Service.

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ABSTRACTS

Arky, Ronald A.; Veverbrants, Egils; and Abramson, Eugene A. (Thorndike Memorial Lab., the Second and Fourth Med. Serv., Boston City Hosp., and Dept. of Med., Harvard Med. Sch., Boston, Mass.): IRREVERSIBLE HYPOGLYCEMIA. A COMPLICATION OF ALCOHOL AND INSULIN. JAMA 206:575-78, Oct. 14, 1968.

Five adult insulin dependent diabetic patients were subject to severe hypoglycemia associated in each instance with acute alcoholic ingestion. Two died and three had severe irreversible neurological damage.

Six male volunteers aged thirty-five to fifty-eight years were studied by insulin tolerance tests (0.1 U. glucagon-free insulin per kg. body weight). The insulin was administered one hour after an infusion of 0.9 per cent saline at the rate of 2.0 ml. per minute alone or with 15 per cent ethyl alcohol (rate of administration, 236 mg. of ethyl alcohol per minute). The two curves were compared and the blood sugar decreased equally after the two types of infusion but there was a definite decrease in the rate of rebound of the blood sugar from its nadir (at approximately twenty-five minutes) in the patients who received ethanol.

It was concluded that excessive use of ethyl alcohol could predispose to dangerous hypoglycemia in insulin treated diabetic patients. S.B.B.

Bagdade, John D. (Div. of Metabolism, V.A. Hosp., Seattle, Wash.): BASAL INSULIN, AND OBESITY. Lancet 2:630-31, Sept. 14, 1968.

Human obesity is characterized by an increased number and size of fat cells. Furthermore, the fat cells are relatively resistant to the metabolic effects of insulin. The insulin resistance is compensated by increased levels of plasma immunoreactive insulin. In this report, basal levels of insulin were found to correlate with the degree of obesity as expressed in excess per cent of ideal body weight, and the author hypothesized that the glucose intolerance displayed by some obese subjects was the result of exhaustion of pancreatic insulinogenic reserve. A significant correlation between fasting triglyceride levels and insulin was also found in obese subjects. The cause of hypertriglyceridemia in obesity was thought to be increased synthesis of triglyceride-rich lipoproteins by a liver excessively stimulated by elevated insulin levels. The resistance to ketosis in obesity may be explained by an inhibition of fatty acid release from adipose tissue; and also related to high insulin concentration. T.G.S.

Barrett, Cynthia T.; and Oliver, Thomas K., Jr. (Dept. of Pediat., Univ. of Washington, Sch. of Med., Seattle, Wash.): HYPOGLYCEMIA AND HYPERINSULINISM IN INFANTS WITH