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ABSTRACTS

Albano, J. D. M.; Ekins, R. P.; and Turner, R. C. (Inst. of Nuclear Med. and Clin. Res., Middlesex Hosp. Med. Sch., London, England): A SENSITIVE, PRECISE RADIOIMMUNOASSAY OF SERUM INSULIN RELYING ON CHARCOAL SEPARATION OF BOUND AND FREE HORMONE MOIETIES. Acta Endocr. 70:487-509, July 1972.

For those who are interested in radioimmunoassay of peptide hormones, this article offers detailed information on a highly sensitive and precise method based upon adsorption of free hormone on to charcoal. S.P.

Bruno, O. D.; Metzger, Patricia; and Malaisse, W. J. (Labs. of Pathophysiology and Exp. Med., Brussels Univ., Brussels, Belgium): INHIBITORY EFFECT OF METYRAPONE ON GLUCOSE UTILIZATION BY BRAIN AND MUSCLE AND ON INSULIN RELEASE BY THE PANCREAS. Acta Endocr. 70:710-18, August 1972.

Verbatim summary. The effect of metyrapone on in vitro glucose metabolism in the rat muscle and brain and on insulin secretion by the rat pancreas was investigated. In the presence of increasing concentrations of metyrapone ditartrate, there was a significant progressive reduction in glucose uptake by incubated hemidiaphragms and in glucose uptake and oxidation by incubated brain pieces. Insulin output by incubated pieces of pancreas was also significantly inhibited by metyrapone ditartrate. A clear action-dose relationship is shown between the residual glucose uptake by the muscle and the uptake or oxidation of glucose by the brain on the one hand and the logarithm of the corresponding metyrapone ditartrate concentration on the other.

These data may help to explain some clinical and experimental findings, i.e., the simultaneous occurrence of hyperglycemia, signs of cerebral dysfunction and of growth hormone release, after the administration of metyrapone in vivo.

Cryer, Philip E.; Coran, Arnold G.; Keenan, Bruce S.; and Sode, Jonas (Naval Med. Res. Inst. and Naval Hosp. National Naval Med. Cent., Bethesda, Md. and the Bureau of Med. and Surg., Navy Dept., Res. Task No. M 4305.05-3056AGG 2): CESSATION OF GROWTH HORMONE SECRETION ASSOCIATED WITH ACUTE ELEVATION OF THE SERUM FREE FATTY ACID CONCENTRATION. Metabolism 21:867-73, September 1972.

Intravenous administration of fat with heparin to male baboons resulted in a marked rise in serum FFA and a rapid decline in serum growth hormone (GH). Neither fat nor heparin injections alone caused comparable changes. A late rise in GH did not occur, and the depression of GH was not explicable on the basis of hyperglycemia. The results suggest that the acute elevation of serum FFA concentrations caused a virtual cessation of GH secretion. Thus, FFA, in addition to glucose and amino acids, may be involved in the regulation of GH secretion. C.R.S.

Feldman, Jerome M.; and Lebovitz, Harold E. (Div. of Endocr. Dept. of Med., Duke Univ. Med. Cent., and Durham V.A. Hosp., Durham, N.C.): STRUCTURAL DETERMINANTS OF INDOLE AMINE ACTION ON IN VITRO INSULIN RELEASE. Endocrinology 91:809-16, September 1972.

Structural specificity for inhibition of glucose-stimulated insulin secretion by indole amines such as serotonin was investigated in an in vitro golden hamster pancreas system. Serotonin (5-hydroxytryptamine) is the most potent inhibitor and tryptamine is a weaker inhibitor, while 5-methyoxytryptamine, melatonin and 5-HIAA are without effect. The inhibitory effect of serotonin on insulin release is blocked by methysergide maleate but not by cinanserin, both being potent serotonin antagonists. These results indicate that in the indole amine and catecholamine series there is a decrease in the inhibitory potency with reduction in the number of hydroxyl groups on the aromatic ring; absence of the aliphatic amine group results in complete loss of inhibitory action. C.R.S.

Gagliardino, J. J.; Hernandez, R. E. with technical assistance of Gagliardino, Elma E. (Lab. of Endocr. Exp., Inst. of Fisiologia, Facultad de Ciencias Medicas, Univ. Nacional de La Plata, Argentina): RELATIONSHIP BETWEEN DIFFERENTIAL RESPONSIVENESS OF PANCREATIC B CELLS AND THE CIRCADIAN VARIATION OF SERUM IMMUNOREACTIVE INSULIN LEVELS. Endocrinology 91:822-25, September 1972.

Serum IRI levels in intact animals and insulin release from excised pancreas in vitro were studied in normal mice during a twenty-four-hour period. Both methods of study revealed a clear circadian rhythm with coincidence in the location of their highest and lowest values. These values rose from zero hour to the twenty hour time points. Based on these observations it is suggested that beta cell secretory activity could be the main factor responsible for the circadian rhythm in serum IRI levels. C.R.S.

Hait, Gershon; Gruskin, Alan B.; and Paulsen, Elsa P. (Dept. of Pediat., Albert Einstein Coll. of Med., New York, N.Y.): INSULIN SUPPRESSION IN CHILDREN WITH CONGESTIVE HEART FAILURE. Pediatrics 50:451-58, September 1972.

Oral glucose tolerance tests were performed in sixteen children with noncyanotic heart disease and various degrees of congestive heart failure, eight children with noncyanotic heart disease without congestive heart failure, and eleven normal children. Serum glucose levels were significantly higher in children with congestive heart failure than in the healthy subjects. Values in children with heart disease but without congestive failure were intermediate. Serum insulin concentrations during the glucose tolerance tests were significantly lower in children with heart failure than in the normal subjects. Children with heart disease without congestive failure were again intermediate. The impaired glucose tolerance observed in children with congestive heart failure would appear to be due, at least in part, to a decreased insulin response. It is suggested that the increased catecholamine levels previously reported in patients with congestive heart failure may be responsible. It is further suggested that these alterations in carbohydrate metabolism may contribute to the growth retardation frequently observed in children with congestive heart failure. The abnormalities in the children with heart disease but without congestive heart failure are unexplained. P.S.R.

Jackson, W. P. U.; Campbell, G. D.; Marine, N.; Major, V.; and Seedat, Y. K. (Endocrine Res. Group, Dept. of Med.; Groote Schuur Hosp. and Univ. of Cape Town, and Diabetes Clin. and Dept. of Med., Univ. of Natal, Durban, South Africa): TRIAMCINOLONE-AUGMENTED GLUCOSE TOLERANCE IN OFFSPRING OF DIABETIC COUPLES. Metabolism 21: 807-14, September 1972.

Two groups of offspring of Indian diabetic couples were investigated with standard oral and triamcinolone-augmented glucose tolerance tests (GTT). In a group studied in Cape Town, no appreciable difference was noted between offspring and controls. In a Durban group, fourteen of seventy-six offspring with normal responses to the standard GTT had diabetic reactions to the augmented GTT. Those with borderline results in the standard GTT usually had abnormal responses to the augmented test. A diabetic reaction to a standard GTT was generally unaltered by triamcinolone. Since the augmented test produced an abnormal curve in those with a high normal standard GTT, the triamcinolone-augmented test was no more discriminatory than a high normal or borderline standard GTT. Triamcinolone tests were abnormal particularly among older offspring whose standard tests were normal. C.R.S.

Kaneto, Akio; and Kosaka, Kinori (Dept. of Intern. Med., Tokyo Women's Med. Coll., Shinjuku-ku, Tokyo, Japan): EFFECTS OF LEUCINE AND ISOLEUCINE INFUSED INTRAPANCREATICALLY ON GLUCAGON AND INSULIN SECRETION. Endocrinology 91:691-95, September 1972.

Infusion of leucine into the pancreaticoduodenal artery of dogs resulted in a slight rise in glucagon (IRG) release from the pancreas and a more than twofold increase in plasma IRI in the pancreatic effluent blood. In the peripheral arterial blood, the IRG levels were unchanged while IRI showed significant enhancement. Isoleucine caused no rise in IRG in pancreatic effluent plasma but raised the IRI levels about threefold. There was a significant rise in peripheral arterial IRI following isoleucine administration. The results indicate a difference in the capacity of these amino acids to stimulate release of glucagon despite similarities in the magnitude of insulin-releasing potency. Insulin secretion by these amino acids is apparently stimulated by direct action rather than through the mediation of glucagon secretion. C.R.S.

Lees, Robert S.; Fiser, Robert H., Jr.; Beisel, William R.; and Bartelloni, Peter J. (Clin. Res. Cent. and Dept. of Nutrition and Food Science, Massachusetts Inst. of Tech., Cambridge, Mass., and U.S. Army Med. Res. Inst. of Infectious Diseases, Frederick, Md.): EFFECTS OF AN EXPERIMENTAL VIRAL INFECTION ON PLASMA LIPID AND LIPOPROTEIN METABOLISM. Metabolism 21:825-33, September 1972.

Sandfly fever, induced in healthy young males, was associated with a decline in plasma lipids and protein components of the low density lipoproteins, which occurred before or in conjunction with the onset of fever. Plasma triglycerides rose above the baseline in early convalescence. These observations suggest that triglycerides and other lipids may be used as metabolic fuel before and during the early febrile period. It is possible that the mechanisms mediating the metabolic response to even a mild viral infection may take precedence over the ordinary demands for energy balance. C.R.S.

Maruhama, Y.; and Macdonald, I. (Dept. of Physiol., Guy's Hosp. Med. Sch., London, England): SOME CHANGES IN THE TRIGLYCERIDE METABOLISM OF RATS ON HIGH FRUCTOSE OR GLUCOSE DIETS. Metabolism 21:835-42, September 1972.

Rats were fed diets high in glucose or fructose and were killed at weekly intervals after receiving a dose of labeled hexose intragastrically. Tissue and plasma analysis revealed that specific activity of the glycerol moiety of the liver and plasma triglyceride (TG) was higher than that in the fatty acid moiety. The specific activity in the adipose tissue was greater after glucose than after fructose feedings; the reverse was found in the plasma. These differences found between the responses to fructose and glucose became less apparent with longer periods of feeding. C.R.S.

Muhlbachova, Elfa; Chan, Peter S.; and Ellis, Sydney (Dept. of Pharmacol., Univ. of Texas Med. Branch, Galveston, Tex.): QUANTITATIVE STUDIES OF GLUCOSE RELEASE FROM RABBIT LIVER SLICES INDUCED BY CATECHOLAMINES AND THEIR ANTAGONISM BY PROPRANOLOL AND PHENTOLAMINE. J. Pharmacol. Exp. Ther. *182*:370-77, September 1972.

Verbatim summary. Log concentration-response (LCR) data on glucose release from rabbit liver slices by l-isoproterenol (ISO), l-epinephrine (EPI) and l-norepinephrine (NE) produced an order of potency of ISO >EPI >NE (42:8:1) and ED50 values of 5.9 x 10^{-8}M (ISO), 3.2 x 10^{-7}M (EPI), and 2.5 x 10^{-6}M (NE). Phentolamine, an alpha adrenergic blocking agent, at 1 x 10^{-4}M did not alter the EPI LCR curve. Propranolol (P) at 1 x 10^{-7} and 1 x 10^{-6}M caused parallel shifts to the right of the LCR curves of both ISO and NE, an indication of competitive antagonism. Since the shifts to the right of the LCR curves of ISO and NE with a tenfold increase in P were 0.5 to 0.6 log unit, rather than 1.0 log unit, the interactions of P and the catecholamines with the receptor sites do not appear to be simple competitive interactions of one molecule of P and one molecule of a catecholamine with one receptor site. Although data for the hyperglycemic potencies of catecholamines in intact rabbits suggest that the adrenergic receptors of the rabbit liver were quite unusual, this study of rabbit liver slices indicates that these receptors are indeed a type of beta adrenergic receptor, but the interaction of this receptor with a catecholamine and P appears complex.

Palumbo, Pasquale J.; Taylor, William F.; Molnar, George D.; and Tauxe, W. Newlon (Mayo Clin. and Mayo Foundation, Rochester, Minn.): DISAPPEARANCE OF BOVINE INSULIN FROM PLASMA IN DIABETIC AND NORMAL SUBJECTS. Metabolism *21*:787-98, September 1972.

Disappearance curves for radioiodinated and immunoreactive bovine insulins were measured in thirty subjects, twenty-five of whom were diabetic patients. Among the latter were eleven who had not received insulin and fourteen taking insulin, of whom eight were considered to have unstable diabetes. A significant negative association was noted between the disappearance of bovine insulin from plasma and the insulin-binding levels of circulating insulin antibody. The clinical characteristics of diabetes, whether stable or unstable, and the presence of chronic diabetic manifestations had no apparent influence upon the rate of disappearance of bovine insulin from plasma other than that attributable to the effect of circulating insulin-binding antibody. C.R.S.

Petersson, Birger; and Shopsin, Baron (Histology Dept., Univ. of Uppsala, Sweden, and Neuropsychopharmacology Res. Unit, Dept. of Psychiatry, New York Univ. Sch. of Med., New York, N.Y.): EFFECTS OF LITHIUM CHLORIDE ON THE ISLETS OF LANGERHANS IN GUINEA PIGS. Acta Endocr. 70:731-35, August 1972.

Lithium chloride was administered intraperitoneally for twenty-four days to guinea pigs. No significant change in blood glucose was observed. Upon histologic examination of the islet cells, a significant increase in the nuclear size of the A_1 cells was observed in the lithium-treated animals. No alterations were seen in the nuclear size of the glucagon-producing A_2 cells or the insulin-producing B cells. These observations suggest that the previously reported abnormalities in carbohydrate metabolism seen after administration of lithium are not associated with functional changes in the A_2 and B cells of the pancreatic islets. S.P.

Sherline, Peter; Lynch, Almorris; and Glinsmann, Walter H. (Sect. on Physiol. Controls, Lab. of Biomedical Sciences, National Inst. of Child Health and Human Development, NIH, Bethesda, Md.): CYCLIC AMP AND ADRENERGIC RECEPTOR CONTROL OF RAT LIVER GLYCOGEN METABOLISM. Endocrinology *91*:680-90, September 1972.

In the isolated, perfused rat liver the catecholamines, epinephrine, norepinephrine, phenylephedrine, and isoproterenol, all activate glycogen phosphorylase and increase glucose output. The alpha adrenergic blocker, phentolamine, prevents these actions while the beta blocker, propranolol, inhibits only the effect of norepinephrine. Cyclic AMP levels rise in liver and perfusate in response to epinephrine; this change is augmented by phentolamine and is blocked by propranolol. Isoproterenol causes a greater rise in cyclic AMP than does phenylephedrine, while the reverse is true with respect to phosphorylase. These data suggest two mechanisms by which catecholamines activate glycogenolysis: first, a beta receptor effect resulting in increased cyclic AMP levels which activates phosphorylase; and secondly, an alpha receptor effect which occurs by a cyclic AMP-independent mechanism, possibly involving vasoconstriction with resultant hyoxemia. In addition, there may exist a separate set of alpha receptors on hepatic cells which inhibit the beta receptor activity, thus preventing the rise in cyclic AMP. C.R.S.

Tzagournis, M.; Chiles, R.; Herrold, J.; and Skillman, T. (Dept. of Med., Div. of Endocr. and Metabolism, Ohio State Univ. Hosps., Columbus, Ohio): THE ROLE OF ENDOGENOUS INSULIN IN DIFFERENT HYPERLIPIDEMIC STATES. Diabetologia *8*:215-20, June 1972.

Verbatim summary. The relationship of insulin secretion to different types of lipid disorders was studied in fifty-four nonobese coronary patients with hyperlipidemia. Nineteen patients had hypercholesterolemia and normal triglycerides (type II), and thirty-five had endogenous hypertriglyceridemia with or without elevated cholesterol levels. Carbohydrate abnormalities and increased insulin concentrations were much more frequent in the hypertriglyceridemic group than in the type II group. Treatment designed to lower insulin levels resulted in no significant serum lipid changes in the type II subjects. In those with hypertriglyceridemia, a decrease in insulin accompanied appreciable falls in triglyceride and cholesterol levels. Mild carbohydrate abnormalities with insulin hypersecretion were closely associated with endogenous hypertriglyceridemia, but appeared to play no major role in type II hyperlipidemia.