ABSTRACTS

Akerblom, Hans K.; Martin, Julio M.; and Cingolani, Horacio E. (Res. Inst. The Hosp. for Sick Children, Toronto 2, Ontario, Canada): CIRCULATING GLUCOSE, INSULIN, FREE FATTY ACIDS, AND ACETONE BODIES IN RATS GIVEN PROPRANOLOL. Amer. J. Physiol. *217*:1690-93, December 1969.

Verbatim summary. The beta-adrenergic blocking agent propranolol was injected into rats, either fasted or fed, at a dose of 1 mg./kg. (half iv, half ip), and circulating levels of glucose, IRI, FFA, and acetone bodies were measured at 0.15, and 45 min. In some experiments an oral glucose load (2.5 gm./kg.) was given simultaneously with propranolol. Control rats received saline instead of propranolol. The serum IRI response to glucose load was lower in propranolol-injected fed rats as compared to the controls, while in fasted animals, both treated and controls, the insulin response was equal. Beta-adrenergic blockade thus depressed the glucose-stimulated insulin release in fed rats. Serum FFA decreased insignificantly after propranolol. Ketogenesis decreased after glucose load in both fasted groups; however, the drop was greater in propranolol-treated animals than in controls, suggesting that propranolol depressed ketogenesis. Our results also suggest that beta-adrenergic receptors, in fed rats at least, have to be functionally intact for glucose to elicit full insulin response.

Azerad, E.; Lubetzki, J.; Duprey, J.; and Friedler, Denise (Serv. of Nutrition—Endocr., Hosp. Beaujon, F-92-Clichy, France): LACTIC ACIDOSIS IN DIABETES. A REPORT OF SEVEN CASES. Presse Med. 77:1705-08, Nov. 8, 1969.

In the last three years seven cases of fatal coma due to lactic acidosis were found among elderly diabetic patients whereas there were no cases among nondiabetics. The clinical features of this condition resemble ketoacidotic coma. The distinguishing features include elevation of lactic acid (mean 27 mEq./L.) an anion deficiency (mean 34 mEq./L.) and only moderate elevations of blood glucose and ketones. Intake of phenyl-ethyl-biguanide was common in all patients, whereas five patients had renal or hepatic disease as associated factors liable to affect the metabolism and/or the excretion of the biguanides and contribute to the excess lactate. M.C.B.

Biener, Jeana; and Vallance-Owen, J. (Dept. of Med., Queen's Univ. of Belfast, Belfast, Ireland): EFFECT OF SYNALBUMIN ON LIPOLYSIS. Lancet 2:1390-92, Dec. 27, 1969.

Human plasma albumin inhibits the effect of insulin on rat diaphragm in vitro and albumin from diabetic patients is more antagonistic than that of nondiabetics. In this study the effect of albumin on insulin induced antilipolytic activity of fat cells was evaluated. Albumin from nondiabetics antagonized insulin inhibition of glycerol release as its concentration rose from 0.5 to 2 per cent. Albumin from diabetics had a more marked antagonism. Since a primary defect in diabetes may be increased formation of free fatty acids from adipose tissue lipolysis, the role of synalbumin in inhibiting the insulin effect of retarding lipolysis could be important in the pathogenesis of the disease. T.G.S.

Calvert, Deane E.; and Lech, John J. (Dept. of Pharmacol., Marquette Sch. of Med., Milwaukee, Wisc.): INHIBITION OF LIPOLYSIS IN ISOLATED FAT CELLS BY SULFHYDRYL REAGENTS. J. Pharmacol. Exp. Ther. *171*:135-40, January 1970.

Verbatim summary. The sulfhydryl reagents iodoacetic acid, iodoacetamide, *p*-chloromercuribenzoate and N-ethylmaleimide were capable of inhibiting norepinephrine-stimulated lipolysis in isolated adipose cells. In addition, iodoacetamide inhibited lipolysis stimulated by theophylline and by dibutyryl cyclic adenosine monophosphate. The data indicate a probable intracellular site of action, possibly upon a triglyceride lipase. The dithiol dimercaprol was capable of preventing the inhibitory action of the compounds but could not reverse established inhibition. The monothiol mercaptoethanol could prevent the inhibition of N-ethylmaleimide. Elucidation of the ultimate mechanism of inhibition by these compounds awaits isolation of the entire system for lipase activation from adipose tissue.

Chazan, Bernard I.; Ferguson, B. Dan; Castelli, William P.; Touborg, Jens N. F.; Balodimos, Marios C.; and Rutstein, D. D. (Joslin Diabetes Foundation, New England Deaconess Hosp., and Depts. of Med. & Preventive Med., Harvard Med. Sch., Boston, Mass.): LIPEMIA RETINALIS: MICROCIRCULATORY CHANGES AND LIPID STUDIES IN A FAMILY. Metabolism *18*:978-85, December 1969.

Lipemia retinalis and massive hyperchylomicronemia in a patient with diabetic acidosis was corrected by insulin administration. Fat uptake by embryonic cells in tissue culture using the lipemic serum was marked and was enhanced by heparin. Red cell aggregation could be induced in retinal vessels at high lipid levels and persisted at lower levels of hyperlipemia. With clearing of hyperchylomicronemia, a pre-beta band appeared in the lipo-protein electrophoretic pattern which persisted when the blood lipids were normal. The patient's father, mother and brother had similar pre-beta bands despite normal blood lipid and insulin levels and normal glucose tolerance. The pre-beta band disappeared with heparin administration. This pattern may represent a prototype of carbohydrate-induced lipemia. C.R.S.

Chlouverakis, C.; and White, P. A. (Metabolic Reactions Res. Unit, Med. Res. Council, Dept. of Biochem., Imperial Coll. of Science, London, Eng.): OBESITY AND INSULIN RESISTANCE IN THE OBESE-HYPERGLYCEMIC MOUSE (obob). Metabolism *18*:998-1006, December 1969.

Insulin sensitivity was studied in obese-hyperglycemic mice with free access to food and after weight reduction on restricted diets and in lean littermates. The intact obese animals and their isolated tissues were resistant to the action of insulin in promoting glucose uptake compared to the other two groups. Following weight reduction the obese mice became sensitive to the action of the hormone. The in vitro results correlated with in vivo observations suggesting that insensitivity of the obese animals to insulin may reside in a tissue resistance. Restoration of the insulin effect after simple weight reduction indicates that insensitivity to the hormone is secondary to obesity or to a factor associated with it. C.R.S.

Coleman, D. L.; and Hummel, Katharine P. (Jackson Lab., Bar Harbor, Maine): EFFECTS OF PARABIOSIS OF NORMAL WITH GENETICALLY DIABETIC MICE. Amer. J. Physiol. *217*: 1298-1304, November 1969.

Verbatim summary. Adult diabetic mice (db/db) of the C57BL/Ks strain were joined in parabiosis with normal mice of the same sex. Little, if any, amelioration of the disease was observed in parabiont diabetics and no symptoms of diabetes were observed in the normal parabiont. Instead, the normal partners lost weight, became hypoglycemic, and died of apparent starvation within fifty days after surgery. In contrast, the diabetic partners gained weight rapidly and remained diabetic. The similarity of this response to that observed with normal rats in parabiosis with rats having lesions in the region of the ventromedial nuclei of the hypothalamus is discussed.

Conn, Harold O.; Schreiber, William; Elkington, Stephen G.; and Johnson, Thomas R. (Veterans Administration Hosp., West Haven, Conn.; Dept. of Intern. Med., Yale Univ. Sch. of Med., New Haven, Conn.): CIRRHOSIS AND DIABETES. I. INCREASED INCIDENCE OF DIABETES IN PATIENTS WITH LAENNEC'S CIRRHOSIS. Amer. J. Dig. Dis. *14*:837-52, December 1969.

Verbatim summary. The frequency of diabetes, defined as persistent fasting hyperglycemia, was evaluated in a consecutive group of 240 cirrhotic patients. A randomly selected group of 411 noncirrhotic patients of similar age, admitted to the hospital during the same period, served as a control group. Diabetes was significantly more common in the cirrhotic group (forty of 240, 16.7 per cent) than in the noncirrhotic group (twenty-nine of 411, 7.1 per cent). In the majority, cirrhosis was apparent before the diabetes was detected; in 18 per cent the diabetes was noted first.

The diabetes in the cirrhotic group was maturity-onset in type, characterized by fasting hyperglycemia, minimal glycosuria, relative freedom from vascular complications, and simple management with diet or oral agents. Although the features of cirrhosis were similar in the groups with and without diabetes, a history of diabetes in the immediate family (46 vs 16 per cent) and the presence of portacaval anastomoses (30 vs 13 per cent) were far more common in those with diabetes. There are many potential diabetogenic factors operative in cirrhosis which may induce diabetes. The appropriate disorders could result in diabetes of hepatogenous, pancreatic, hemosiderotic, kaliopenic, portal-systemic anastomotic, insulin-resistant, somatotropic, or genetic origin. The data suggest that these factors, acting individually or perhaps in various combinations, may precipitate diabetes in genetically susceptible patients or may even induce diabetes de novo in some cirrhotic patients.

Deschamps, I.; Heilbronner, J.; Canivet, J.; and Lestradet, H. (Hosp. Herold F-75-Paris (19e) France): VARIATIONS IN BLOOD INSULIN THROUGHOUT THE DAY AND NIGHT IN NORMAL SUBJECTS. Presse Med. 77:1815-17, Nov. 22, 1969.

Verbatim summary. Estimation of plasma insulin levels by the radioimmunological technic of Yalow and Berson with electrophoretic separation was carried out for twenty-four hours in ten normal subjects of different ages at rest and with normal carbohydrate diet and three meals in twenty-four hours.

This study shows that insulin levels reach a maximum at 12 a.m. and 2 p.m. and then fall progressively during the afternoon and night to reach their lowest level at 5 a.m.

Fager, Charles A. (Dept. of Neurosurg., Lahey Clin., Boston, Mass.): PITUITARY ABLATION—CURRENT SURGICAL TECHNICS. Lahey Clin. Bull. *18*:155-63, October-December 1969.

The author reviews the various procedures available for pituitary ablation. These include direct transsphenoidal microsurgical operation, stereotactic transsphenoidal thermal procedures (cryosurgery and heat coagulation), transsphenoidal introduction of yttrium-90, heavy particle radiation of the pituitary and finally intracranial operations (pituitary stalk section and total hypophysectomy). He further discusses in some detail the technic of intracranial hypophysectomy which he considers a safe and most practical operative procedure. M.C.B.

Giacometti, Luigi; and Barss, Margaret (Oregon Regional Primate Res. Center, Beaverton, Ore.): PAUL LANGERHANS: A TRIBUTE. Arch. Derm. *100*:770-72, December 1969.

In recognition of the Centennial anniversary of the discovery of the insulin producing islet cell masses in the pancreas, a succinct biography of Paul Langerhans' (1847-1888) is presented. Langerhans' life, from his birth in Berlin, to his death on the island of Madeira (off the coast of Portugal) is traced. Excerpts are quoted from Langerhans' doctoral thesis (The Microscopic Anatomy of the Abdominal Salivary Glands) which he completed in the laboratory of Virchow at the age of twenty-two. P.F.

Gordon, Edwin E.; and de Hartog, Maria (Dept. of Med., New York Univ. Med. Center, New York, N. Y.): THE RELATIONSHIP BETWEEN CELL MEMBRANE POTASSIUM ION TRANSPORT AND GLYCOLYSIS. THE EFFECT OF ETHACRYNIC ACID. J. Gen. Physiol. *54*:650-63, November 1969.

Verbatim summary. Cell membrane transport of K^+ stimulates the rate of glycolysis in Ehrlich ascites tumor cells. A study of the characteristics of this relationship indicates that the stimulation occurs under anaerobic as well as under aerobic conditions. The data suggest that glycolysis is stimulated by a K^+ transport mechanism that is coupled to Na^+ transport because the effect is blunted or abolished when the principal intracellular ion is lithium or choline. This stimulus to glycolysis is blocked by ouabain and ethacrynic acid, agents that have been shown to inhibit monovalent cation transport in erythrocytes. In contrast to the action of ouabain, glycolysis is inhibited by ethacrynic acid in Ehrlich ascites tumor cells in the absence of cell membrane K^+ transport. In studies with ghost-free hemolysates of human erythrocytes and with cytosol prepared from Ehrlich ascites tumor cells, ethacrynic acid significantly blocks lactate formation from fructose diphosphate demonstrating the direct inhibitory effect of this agent on one or more enzymes of the Embden-Meyerhof pathway. Since ethacrynic acid has no influence on lactate formation in intact erythrocytes utilizing an endogenous substrate, the presumptive site of inhibition is proximal to the 3-phosphoglycerate level.

Granitsas, A. N. (Lab. of Gen. Biology, Aristotelian Univ. of Thessaloniki Med. Sch., Thessaloniki, Greece): INCORPORATION OF ALANINE C-14 AND LEUCINE C-14 INTO PROTEIN OF ESCHERICHIA COLI UNDER THE INFLUENCE OF INSULIN. Arch. Biochem. *134*:275-78, November 1969.

It has been previously recognized that insulin exerts a net protein anabolic effect in the isolated rat diaphram. Reports from the author's laboratory had established previously that insulin also stimulates incorporation of radioactive amino acids into protein in isolated white cells. In the present report, evidence is presented indicating that insulin stimulates the uptake of amino acids by microorganisms. A disconcerting observation, however, was the wide variation in potency of various lots of insulin in causing amino acid uptake by E. coli. The data did not provide an explanation for these differences. T.J.M.

Gurson, Cihad T.; and Saner, Gunay (Dept. of Pediat., Univ. of Istanbul, Cerrahpasa, Istanbul, Turkey): LIPOPROTEIN LIPASE ACTIVITY IN MARASMIC TYPE OF PROTEIN CALORIE MALNUTRITION. Arch. Dis. Child. *44*:765-68, December 1969.

Verbatim summary. The effect of heparin on LPL activity, the kinetics of this enzyme in the plasma, and the FFA levels in fourteen cases of marasmus were investigated in the severe and recovery stages of the disease. The free fatty acid levels were found to be comparable to normal control values. This finding was in agreement with the absence of fatty liver enlargement in this type of protein-calorie malnutrition. LPL activity in the plasma after the administration of heparin did not rise as high as observed in the normal controls. This is probably the expression of a defect in the synthesis of this enzyme similar to defects in other enzyme systems observed in states of malnutrition. The defect in synthesis of the enzyme persisted for up to fourteen weeks.

Haynes, R. C., Jr., and Lu, Y. S. (Dept. of Pharmacol., Case Western Reserve Univ., Sch. of Med., Cleveland, Ohio): MEASUREMENT OF CORTISOL-STIMULATED GLUCONEOGENESIS IN THE RAT. Endocrinology *85*:811-14, November 1969.

An isotope dilution technic was employed to measure the rate of formation of glucose in fasted, adrenalectomized rats receiving cortisol. The rate of dilution of labeled blood glucose was not significantly increased in these animals. A significant rise in liver carbohydrate was demonstrated in response to cortisone representing the effect of gluconeogenesis. The data indicate a doubling of gluconeogenesis during cortisol administration despite no significant change in the rate of formation of free glucose. In these rats, the apparent space in which glucose was distributed was decreased during cortisol administration. C.R.S.

Kim, Ki-Han; and Blatt, Lois M. (Dept. of Biochem., Purdue Univ., Lafayette, Ind.): REGULATION OF HEPATIC GLYCOGEN SYNTHETASE OF RANA CATESBEIANA. 1. THE EFFECT OF INSULIN TREATMENT. Biochemistry *8*:3997-4004, October 1969.

Injection of insulin, 0.0025 U. per gram of body weight intraperitoneally, into tadpoles caused up to three-fold increases in activity of hepatic glycogen synthetase after three to six hours. Animals were maintained at 24° C. during the experiments. Hepatic glycogen synthetase was purified approximately 200-fold to 500-fold and its kinetic properties were examined. Insulin treatment caused a small increase in V_{max} but the main effect was a lowering of the apparent K_m for UDP-glucose. In contrast to the situation for mammalian glycogen synthetase, all forms of tadpole liver enzyme appeared to require glucose 6-phosphate for activity, and the apparent K_m for glucose 6-phosphate was not altered by insulin treatment. At suboptimal concentrations of glucose 6-phosphate, inorganic phosphate stimulated the activity of enzyme from control livers and from livers of insulin-treated animals. The injection of cycloheximide inhibited incorporation of radioactive leucine into total liver protein by 95 per cent, but did not prevent stimulation of glycogen synthetase by insulin. H.T.N.

Kuhns, L. R.; Spencer, M. L.; Bacon, G. E.; and Lowrey, G. H. (Univ. of Michigan, Ann Arbor, Mich.): LETTERS: CHLORPROPAMIDE-INDUCED HYPOGLYCEMIA IN A CHILD WITH DIABETES INSIPIDUS. JAMA *210*:907, Nov. 3, 1969.

Severe hypoglycemia with brain damage was induced in an eleven-year-old child weighing 29 kg. after administration of chlorpropamide for treatment of diabetes insipidus secondary to craniopharyngioma. The hypoglycemia occurred after adult sized dose of the drug (500 mg. per day) for three days. S.B.B.

Lee, John C.; Grodsky, Gerold M.; Caplan, Joan; and Craw, Linda (Dept. of Path., Univ. of California Sch. of Med., San Francisco, Calif.): EXPERIMENTAL IMMUNE DIABETES IN THE RABBIT. Amer. J. Path. *57*:597-616, December 1969.

Verbatim summary. Immunization of New Zealand white rabbits with bovine insulin in Freund's adjuvant produces circulating anti-insulin antibody that reacts with both endogenous and bovine insulins. Hyperglycemia or diabetes mellitus develops in some of these immunized animals. The pancreata of diabetic animals exhibit lymphocytic infiltration of the islets of Langerhans, while rabbits with anti-insulin antibody but without islet lymphocytes remain normoglycemic.

Immunofluorescence studies of the pancreata of immunized rabbits, both diabetic and normoglycemic, failed to demonstrate the presence of anti-insulin anitbody in islets. Electron microscope studies revealed minor alterations of beta cells in immunized nondiabetic rabbits and destruction of beta cells by lymphocytes in diabetic rabbits.

This experimental model of immune diabetes morphologically resembles acute-onset juvenile diabetes in man.

Letteri, Joseph M.; and Wesson, Laurence G. (Depts. of Med., Meadowbrook Hosp., East Meadow, N.Y.; & Jefferson Med. Coll. of Philadelphia, Phila., Pa.): CALCIUM AND MAGNESIUM EXCRETION DURING GLUCOSE AND MERCAPTOMERIN DIURESIS IN MAN. Metabolism *18*:1048-54, December 1969.

Calcium and magnesium as well as sodium, chloride and glucose excretions were studied using clearance methods during glucose and mercaptomerin diuresis in man. No specific depression of calcium or magnesium transport as related to sodium or chloride excretions was noted with administration of mercaptomerin. There was no correlation between the calcium and magnesium excretion and the extent of the glucose osmotic diuresis suggesting that glucose osmotic diuresis per se has little effect on calcium or magnesium excretion. C.R.S.

Long, W. Newton; and Freeman, Malcolm G. (Dept. of Gynec. and Obstet., Emory Univ. Sch. of Med., Atlanta, Georgia): DIABETES AND PREGNANCY. Johns Hopkins Med. J. *125*:258-61, November 1969.

Many previous studies starting from populations of pregnant women with abnormal glucose tolerance tests have repeatedly shown abnormally high fetal morbidity and mortality. The present investigation begins from a population of women with dead babies and assesses the abnormality of carbohydrate tolerance in this group.

Among 737 women whose babies were born dead or died in the first twenty-eight days of life at Grady Hospital between 1965 and 1968, the incidents of abnormally high glucose tolerance tests was 25.5 per cent, abnormally flat was 14.4 per cent, for a total abnormal of 39.9 per cent. The occurrence of a perinatal mortality is sufficient reason to suspect strongly that the mother will demonstrate abnormal carbohydrate metabolism. D.R.C.

Loridan, Liliane; and Senior, Boris (Dept. of Pediat., Tufts Univ. Sch. of Med., and Pediat. Endocrine-Metabolic Serv., New England Med. Center Hosps., Boston, Mass.): EFFECTS OF INFUSION OF KETONES IN CHILDREN WITH KETOTIC HYPOGLYCEMIA. J. Pediat. *76*:69-74, January 1970.

Verbatim summary. Four children with ketotic hypoglycemia and two control children were given infusions of B-hydroxybutyrate (15 gm. per square meter) after an overnight fast and following the induction of gluconeogenesis by a diet low in calories and carbohydrate. After the overnight fast the concentrations of glucose decreased markedly, to a greater degree than we had previously observed in adults, and lipolysis was inhibited. After the induction of gluconeogenesis the decrease in the concentration of glucose was no longer seen, although the inhibitory effect of lipolysis was still evident. In neither study did the levels of insulin change significantly nor were significant differences of response apparent between the patients and the control children.

Menendez, Celia J.; and Herskovits, Theodore T. (Dept. of Chem., Fordham Univ., New York, N.Y.): STUDIES OF THE LOCATION OF THE TYROSYL RESIDUES IN INSULIN. II. Biochemistry *8*:5052-59, December 1969.

Incubation of bovine insulin with trypsin produced desoctapeptide-insulin plus octapeptide and heptapeptide fragments derived from the carboxyl-terminal portion of the B chain. Solvent perturbation difference spectra were interpreted as showing that the desoctapeptide-insulin had one buried, one exposed, and one partly exposed tyrosyl residues. The tyrosyl residue at position B-26 appeared to be converted from a buried form to an exposed state when the peptide segment containing this residue was released by tryptic action. The two phenylalanyl residues in this segment of the B chain also appeared to be converted from a buried to an exposed state by tryptic hydrolysis. H.T.N.

Root, Allen W.; Bongiovanni, Alfred M.; and Eberlein, Walter R. (Div. of Endocr., Children's Hosp. of Philadelphia, and Dept. of Pediat., Univ. of Pennsylvania Sch. of Med., Philadelphia, Pa.): STUDIES OF THE SECRETION AND METABOLIC EFFECTS OF HUMAN GROWTH HORMONE IN CHILDREN WITH GLUCOCORTICOID-INDUCED GROWTH RETARDATION. J. Pediat. *75*:826-32, November 1969.

Verbatim summary. Plasma growth hormone responses to insulin-induced hypoglycemia, orally administered glucose, and the intravenous infusion of arginine were normal in four children with growth retardation secondary to prolonged treatment with glucocorticoids. The short-term administration of growth hormone to three of these subjects resulted in nitrogen retention comparable to that observed in a patient with hypopituitarism who was treated similarly. Transient acceleration of linear growth rate was observed when somatotropin was administered to each patient for six months. Two of the four patients developed serum gamma globulins capable of binding growth hormone. It has been concluded that glucocorticoids do not suppress growth by inhibition of growth hormone release, but rather by peripheral antagonism to the long-term growth-promoting activities of somatotropin.

Sachsse, R.; Sachsse, B.; Jahnke, K.; and Daweke, H. (Kinderabteilung der Waldklinik Hosel; Medizinische Klinik der Stadtischen Krankenanstalten Wuppertal-Elberfeld and II. Medizinische Universitatsklinik Dusseldorf, Germany): CALORIE REQUIREMENTS OF DIABETIC CHILDREN. Deutsch. Med. Wschr. *94*:2535-39, Dec. 5, 1969.

Opinions differ widely on the desirable level of caloric intake in healthy and diabetic children. The best way of determining daily food utilization is to weigh and calculate precisely the diet of every child. With this method, nutritional requirements were determined in 100 hospitalized diabetic children aged between three and fifteen years while they were under strict metabolic control. A control period of several weeks was followed by a test period of seven days during which time the food intake was measured. Physical activity was controlled during the week of testing. The proportion of total calories amounted to 17 per cent for protein, 38 to 39 per cent for fat, 44 to 45 per cent for carbohydrates. Total caloric requirement was higher in almost all children than that reported by earlier investigators on the basis of estimates or rough calculations alone. The high caloric requirements may be explained by the loss of energy which is caused in diabetic children by the inevitable and often considerable excretion of urine sugar. J.E.V.

Vaughan, Martha; and Murad, Ferid (Molecular Disease Branch, National Heart Inst., National Insts. of Health, Bethesda, Md.): ADENYL CYCLASE ACTIVITY IN PARTICLES FROM FAT CELLS. Biochemistry *8*:3092-99, July 1969.

A particulate subcellular fraction that contained smooth membrane fragments was prepared by differential centrifugation after disrupting rat adipose cells by freezing and thawing them in a hypotonic buffer solution. This preparation exhibited adenyl cyclase activity that was not appreciably diminished by storage at —80° for two weeks. Adenyl cyclase activity was enhanced several-fold, but usually to different maximal levels, by the addition in vitro of epinephrine, ACTH or glucagon. Higher concentrations of hormones were required for optimal stimulation of this particulate adenyl cyclase preparation than were needed for maximal stimulation of lipolysis in intact fat cells. The effects of maximally stimulatory concentrations of ACTH and glucagon on adenyl cyclase activity in the particulate system appeared to be additive. The enhancing effect of epinephrine was inhibited by dichloroisoproterenol and pronethalol. Insulin, prostaglandin E_1 and nicotinic acid did not diminish the stimulation of adenyl cyclase by maximally effective concentrations of epinephrine. H.T.N.