et al., was reinvestigated. Epididymal fat tissue from fasted Wistar rats was incubated in Krebs-Ringer bicarbonate buffer. Incubated tissues and media were then analyzed for free fatty acid (FFA) release. The three hypotheses examined, and the results of these investigations, were:

(1) *Calcium could reduce glucose entry into fat tissue and, by diminishing triglyceride synthesis, facilitate net lipolysis.* In the presence of 1 mg. per ml. ethylenediamine tetraacetate (EDTA) and 2 gm. per 100 ml. albumin, the lipolytic response to 1 ug. per ml. ACTH was found to be dependent on the presence of added calcium, irrespective of the presence or absence of glucose in the medium.

(2) *The lipolytic system activated by ACTH could require calcium to function.* EDTA suppressed ACTH-activated lipolysis only in the presence of albumin: in its absence, EDTA was wholly without liposuppressive effect.

(3) *Primary ACTH receptor sites might contain calcium whereas under EDTA-blockade less specific sites (for example, sulfhydryl groups), could permit ACTH attachment to fat tissue.* In the presence of EDTA, inhibition of ACTH-induced lipolysis was logarithmically related to albumin concentration in the incubation media. Sulfhydryl-group blockade proved ineffective in inhibiting ACTH-activated lipolysis in the absence of albumin. Several high molecular weight polypeptides and carbohydrate polymers were substituted for albumin in the presence and absence of EDTA, but had no liposuppressive effect.

These experiments suggest that the role of calcium in ACTH-activated lipolysis is to permit or facilitate ACTH attachment to adipose tissue.

THIRD PRIZE

Effects of Induced Hypokalemia on Carbohydrate Metabolism, FFA, Tissue Potassium, and Glycogen in the Rat

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The relationship between the potassium ion and carbohydrate metabolism has been the object of renewed interest. An animal study was undertaken to correlate changes in plasma glucose, free fatty acids, and tissue glycogen with alterations in plasma and tissue potassium.

Forty-eight male Wistar rats were divided into three groups and fed a low potassium diet for two weeks:

Group I—Controls, saline injected for seven days, diet, supplemented with potassium.

Group II—DOCA injected for seven days, no potassium supplementation.

Group III—Same as II but with potassium supplementation.

One week after the last injection, fasting animals were anesthetized and heart blood drawn for potassium, glucose, and FFA. An intracardiac injection of glucose (1 gm. per kg.) was given and tail blood sampled for glucose at five-minute intervals for thirty minutes. Two hours after glucose injection, heart blood was again collected for potassium, glucose, and FFA, the animal sacrificed, and tissue (muscle, liver, and epididymal fat pad) obtained for potassium and glycogen determinations.

1. Profound hypokalemia was established in Group II while normal serum potassium was maintained in Groups I and III.

2. Significantly, adipose tissue and liver showed no loss of potassium despite marked decreases in both plasma and muscle potassium in Group II.

3. Potassium depletion did not produce a significant change in glucose disappearance rate. Fasting and two-hour glucose values, however, were significantly elevated in potassium-depleted animals.

4. Liver and muscle glycogen was significantly increased in the potassium-depleted animals.

5. Plasma FFA (fasting and post-glucose) were unaffected by potassium depletion.

BOOK REVIEW

ANNUAL REVIEW OF BIOCHEMISTRY. *Edited by P. D. Boyer. $11.50. pp. 908, plus 91 pages of index. Annual Reviews, Inc., Palo Alto, California. Volume 35, Parts I and II, 1966.*

Significant recent developments in twenty-five different areas of biochemical research have been competently and critically analyzed in volume thirty-five of the *Annual Review of Biochemistry.* This comprehensive review affords an awesome glimpse of the enormous expansion that is taking place in biological knowledge; the deeper insight into biochemical processes that the newer observations provide is truly stimulating.

Important advances in our understanding of the composition, conformation and metabolism of macromolecules are de-

scribed in chapters devoted to proteins, nucleic acids and polysaccharides. Separate chapters are devoted to discussions of viruses, bacterial cell walls, contractile proteins, protein synthesis, messenger RNA and a description of the relationship of structure to function in immunoglobulins. Recent observations and basic concepts related to mechanisms of enzyme action and the regulation of enzyme activity are described in several well-written chapters.

Readers who are particularly interested in hormones and cellular metabolism will find several sections that deal with these subjects. Ira Pastan discusses the mechanism of action of various peptide hormones. W. A. Wood has reviewed newer aspects of carbohydrate metabolism with special emphasis on regulatory features. J. A. Olson has comprehensively reviewed observations concerning the metabolism of fatty acids and phospholipids. Phosphoglycerides and phospholipases are also discussed in detail by Van Deenen and De Haas. H. C. Pitot has contributed an analysis of biochemical aspects of malignancy.

Readers whose interests are highly specialized may like to know that reprints of individual chapters may be purchased separately.

ABSTRACTS

Abramson, Eugene; and Arky, Ronald (Thorndike Mem. Lab. and II and IV (Harvard) Med. Services, Boston City Hosp. and Dept. of Med., Harvard Med. Sch., Boston, Mass.): DIABETIC ACIDOSIS WITH INITIAL HYPOKALEMIA. THERAPEUTIC IMPLICATIONS. JAMA *196*:401-03, May 2, 1966.

The authors describe five patients with diabetic acidosis accompanied by initial hypokalemia, instead of the more usual normal or elevated serum potassium levels. The clinical background included long duration of poor regulation of diabetes (four patients) or recent prior acidosis (three weeks before, one patient). There was clinical aggravation of the hypokalemia by the usual intensive therapy and especially by sodium bicarbonate infusions. The use of the latter in the presence of hypokalemia should be either avoided or postponed until hypokalemia is corrected by adequate potassium therapy. Cardiac irregularities related to low serum potassium ensued in two patients and serum magnesium deficiency in one (characterized by circumoral paresthesia). S.B.B.

Albrink, Margaret J.; and Davidson, Paul C. (West Virginia Univ. Sch. of Med., Morgantown, W. Va.): IMPAIRED GLUCOSE TOLERANCE IN PATIENTS WITH HYPERTRIGLYCERIDEMIA. J. Lab. Clin. Med. 67:573-84, April 1966.

Elevation of plasma triglycerides is common in patients with frank diabetes. Abnormalities in carbohydrate tolerance are often found in patients with hypertriglyceridemia. In an effort to quantitate the relationships between carbohydrate tolerance and plasma lipids, the authors surveyed eighty-nine healthy or diseased subjects to obtain data permitting assessment of carbohydrate tolerance in persons having ranges of triglyceride values commonly found in health and disease. Standard oral glucose tolerance tests were done fifty-one times and intravenous glucose tolerance tests were done thirty-eight times. Nonesterified fatty acids, total fatty acids, cholesterol, lipid phosphorus and triglycerides were measured and statistical analyses were made. Subjects with hypertriglyceridemia displayed a slower disappearance of plasma glucose, a less marked diminution of plasma NEFA and a less marked reactive hypoglycemia after administration of glucose. Relationships between carbohydrate intolerance and lipid abnormalities were stronger with intravenous glucose tolerance testing than oral glucose tolerance testing. The results support the possibility that elevation of plasma triglycerides is a manifestation of insulin resistance and represents a stage in the development of maturity-onset diabetes or a diabetes-like state. T.G.S.

Anderson, J. M.; Milner, R. D. G.; and Strich, Sabina J. (Dept. of Neuropathy, Inst. of Psychiatry, The Maudsley Hosp., Denmark Hill, London, England; and Inst. of Child Health, Hammarsmith Hosp., London, England): PATHOLOGICAL CHANGES IN THE NERVOUS SYSTEM IN SEVERE NEONATAL HYPOGLYCAEMIA. Lancet *2*:372-75, Aug. 13, 1966.

Although sustained neonatal hypoglycemia may lead to death, microcephaly, mental retardation or cerebral palsy, there are no descriptions of the microscopic anatomy of the nervous system in infants dying with hypoglycemia. The authors studied the spinal cords and brains of two male infants who survived forty and fifty-five hours after birth in whom documentation of prolonged hypoglycemia was made. The chief changes consisted of acute degeneration of glial cells and neurones throughout the entire nervous system. The most striking abnormalities were fragmentation of the nuclei, loss of Nissl bodies and granularity of the cytoplasm of the nerve cells. There was no evidence of selective vulnerability of certain layers or regions as might be seen in anoxia. The only focal lesions were infarcts in the paraventricular white matter and these were seen in only one case. It is emphasized that premature babies and those having experienced intrauterine malnutrition are those with the greatest risk of neonatal hypoglycemia. T.G.S.

Antoniades, Harry N.; and Gershoff, Stanley N. (Protein Foundation Lab., Jamaica Plain, and Dept. of Nutrition, Harvard Sch. of Public Health, Boston, Mass.): "BOUND" INSULIN: BIOLOGIC EFFECTS IN INTACT, HYPOPHYSECTOMIZED OR ADRENALECTOMIZED RATS, FOLLOWING INTRAVENOUS ADMINISTRATION. Endocrinology *78*:1079-81, May 1966.

Preparations of "bound" insulin obtained from pooled normal human sera were injected intravenously into intact, hypophysectomized or adrenalectomized rats for comparison of the effects obtained from similar injections of crystalline insulin. Both "bound" and crystalline insulins produced a significant decline in blood glucose in hypophysectomized and adrenalectomized animals but not in the intact animals. Both insulins stimulated the incorporation of glucose-μ-C-14 into muscle