

BOOK REVIEW

NUTRITION AND DIABETES MELLITUS, edited by E. R. Froesch and J. Yudkin, \$50.00, 687 pages. Milan, Casa Editrice Il Ponte, 1972.

The record of the Sixth Capri Conference, held in Italy in March, 1972, *Nutrition and Diabetes Mellitus* contains contributions from twenty-six participants and includes transcriptions of the discussions which followed presentation of the papers.

There is little that is new in this symposium, as the participants have generally reviewed data that have been presented previously, mostly in American and European journals. For those who have kept abreast of work proceeding rapidly on nutrition and diabetes, the symposium will have an element of déjà vu. Its value may derive from the integration of information and the generally generous bibliographies. The book is of more value to the investigator than to the general physician, who may prefer somewhat more predigested information.

The text is divided into six sections: nutrition and metabolism; interaction of nutrients and hormones; the relation between diabetes, obesity, arteriosclerosis and nutrition; the psychology of appetite; the dietetic treatment of diabetes; and the provocation and prevention of diabetes by nutrition. Each section will be discussed briefly here.

The section on nutrition and metabolism provides basic information. The first paper discusses the role of insulin and glucagon in the provision of substrate for energy utilization during pre- and postnatal life in the rat, with particular emphasis on glycogen levels and gluconeogenic enzymes. The second paper summarizes present knowledge regarding the genetics of diabetes in the Chinese hamster, discusses the influence of diet on the origin of diabetes and compares responses of the hamster and the mouse with published observations concerning man. A subsequent paper further emphasizes the utility of laboratory rodents as models for studying the influence of genetics and environment on diabetes; both elements may be strictly regulated in studies with rodents. The possibility of confusion arising from the use of different types of animal models is pointed out. Insulin response was measured in three types of obesity (obob, DBM and gold thioglucose) and also in controls and in spiny mice. In the three types of obese animals, three insulin secretion patterns were elicited. Generally, the obese mice tended to mimic the high basal insulin levels found in man, but differed in their first and second phases of insulin response. The spiny mice had normal basal insulin and impaired response to a variety of stimuli. This is a good summary of rodent models potentially useful for greater understanding of insulin secretion in obesity and hyperglycemia. The ensuing article, relating to the coexistence of obesity, hyperlipidemia and diabetes mellitus, is a simplistic review of existing literature. Finally, there are two articles on the role of ketones and amino acids in fuel utilization. The first presents data to support the concept that ketones are the preferential substrate for energy production; the second attempts to define the role of insulin and glucagon in regulating

amino acid metabolism and flux during the fed, fasted and exercising state. These latter two papers contain fine reviews of existing data and, in addition, the speakers' contributions to the field.

The second section of the symposium, on the interaction of nutrients and hormones, opens with a fair review of the effect of gastrointestinal hormones on pancreatic islet function, although too much emphasis is placed on the possible physiologic effects of hormones since the data available are on pharmacologic effects only. The second paper is a superficial review of the influence of carbohydrates and fat on the secretion of insulin and growth hormone. There follows an excellent study of the influence of proteins on insulin secretion by a member of the Michigan group that has done so much to enlighten us in this field. This is followed by a paper from another member of the same group discussing the effect of varying the protein in the diet on cortisol metabolism. The influence of some nutrients and metabolic substrates on glucagon secretion is then outlined, with special emphasis on the role of amino acids and of lactate. Finally, a member of the Seattle group reviews their data on improved glucose tolerance in mild diabetics after a high carbohydrate diet is prescribed without a change in total caloric intake.

The third section of the symposium, concerning the relation between diabetes, obesity, arteriosclerosis and nutrition, is composed of two good and two weak papers. The first reviews epidemiologic data for the relation of weight, sugar intake and fat intake to diabetes in various population groups. The epidemiologist, who has excellent command of the material, discusses the unequivocal role of fatness as a determinant of maturity-onset diabetes. He points out the diabetes prevalence among different population groups and notes that it is sometimes explainable by diet but often is not. He depreciates somewhat the sugar theory of diabetes induction by stating that, whereas rates of maturity-onset diabetes have correlated well with sugar consumption, a number of inconsistencies suggest that the relationship may be wholly or partially coincidental. In a discussion of the relationship between eating behavior and obesity, data are presented suggesting that frequency of eating is related to weight gain, those who eat less frequently being more at risk than those who take frequent meals. Though the data presented are far from foolproof, particularly as regards total caloric intake in the subjects investigated, the hypothesis is provocative.

The fourth section is a special lecture on the psychology of appetite. Given its importance in the book, it is unenlightening. The review of the mass of relevant literature is skimpy, and the data brought to bear on the regulation of body weight by regulation of meal size and meal frequency is flimsy. An opportunity for a thorough discussion of a fascinating topic has been missed.

In the fifth section, as the question of dietetic treatment of diabetes is debated, the confusion regarding the relative

usefulness of high or low carbohydrate intake in diabetes is evident. The first author considers carbohydrate limitation to be essential in diabetes. Though he recommends weight loss for obese diabetics, he does not for other obese persons. He then quotes studies showing that diabetics whose diet is not regulated do no worse than other diabetics. This is a confusing and poorly organized paper. A subsequent speaker deals with practical aspects of dietary treatment, but has nothing particularly new to say. The final paper is an interesting demonstration of the possible use and difficulties of teaching aids in the training of diabetics. Presented by someone actively engaged in teaching, it is well presented and will be of interest to those involved in teaching diabetics about the principles and practical aspects of diets.

The final section is entitled the "Provocation and Prevention of Diabetes by Nutrition." How the quantity and quality of the diet may affect laboratory rodents with spontaneously occurring hyperglycemic syndromes is discussed by the first speaker. Also, the effect of hyperphagia induced by destruction of hypothalamic structures on the appearance of diabetes in the animals is examined. Next is an extremely able discussion of the effect of diet restriction on the onset of diabetes in prediabetic Chinese hamsters. An interesting study of the dietary intake of diabetics and their nondiabetic siblings and other controls shows that diabetics eat significantly

more than nondiabetic controls. The symposium ends with an article propounding the thesis that the vascular diseases of retinopathy and atherosclerosis are dissociated, the first being caused by hyperglycemia and the second being related to diet. Again, the hypothesis is interesting but little data is brought to bear on the proposition.

This book is a mixed bag. Though great emphasis is placed on the role of carbohydrates, proteins and fats on the induction and control of diabetes, and also on caloric restriction in the obese diabetic, other nutritional aspects of diabetes are not mentioned. In exchange for some of the poorer presentations in this symposium, one might have substituted discussions pertaining to the influence of potassium and calcium on insulin secretion and of chromium on glucose homeostasis. Interest in the micronutrients is certainly growing, and diabetologists could profit from such discussions.

In summary, the proceedings of this symposium will be of interest primarily to the investigator, though the practicing clinician who desires to read in depth may find many of the chapters valuable. The Ponte publishing house, which sponsored the conference, may be congratulated for preparing the text with clear attractive type and with illustrations and tables that are easy to read. The text is printed in English and Italian, side by side, and the English translations are, on the whole, excellently done.

ABSTRACTS

Biener, J.; Jansen, F. K.; and Brandenburg, D. (Diabetes Forschungs-Inst., Düsseldorf, and Deutsches Wollforschungsinstitut, Aachen, F.R.G.): INSULIN LABELLED BY COUPLING WITH PEROXIDASE. *Diabetologia* 9:53-55, February 1973.

Verbatim summary. By coupling purified horseradish peroxidase with glutaraldehyde and subsequent reaction with insulin, conjugates were obtained. These were partially purified by ion exchange chromatography on DEAE-cellulose. A fraction with an average molar ratio of peroxidase to insulin 1:0.37 was analyzed by electrophoresis and gel filtration. The peroxidase activity of this fraction was found to be 19 per cent of normal and the immunological reactivity 0.6 per cent as compared with that of insulin.

Birnesser, H.; Reinauer, H.; and Hollmann, S. (Inst. of Physiol. Chem. and Diabetes Res. Inst., Univ. of Düsseldorf, F.R.G.): COMPARATIVE STUDY OF ENZYME ACTIVITIES DEGRADING SORBITOL, RIBITOL, XYLITOL AND GLUCONATE IN GUINEA PIG TISSUES. *Diabetologia* 9:30-33, February 1973.

Verbatim summary. In guinea pig tissues the activities of the enzymes D-gluconokinase, sorbitol dehydrogenase, D-ribulose and D-xylulokinase were measured. D-ribulose and D-xylulose were prepared by isomerization of D-ribose and D-xylose in pyridine and separated by preparative paper chromatography. The activity patterns of the pentulokinases were identical in all tested organs. The highest activities of these two enzymes were found in adipose tissue, when referred to soluble cell protein, and was higher than the activity in liver and kidney.

The high enzyme activities of the pentulokinases in adipose tissue may explain the antilipolytic effect of these pentitols and pentoses in diabetes. The activities of sorbitol dehydrogenase and gluconokinase showed a similar activity pattern in all tested organs of the guinea pig. The highest activities were found in liver and kidney and the lowest in the adipose tissue. The direct metabolism of gluconate in adipose tissue seems impossible. The activity of the pentulokinases is diminished in the tissues of the diabetic rat.

Bloom, S. R.; Daniel, P. M.; Johnston, D. I.; Ogawa, Olivia; and Pratt, O. E. (Inst. of Clin. Res., Middlesex Hosp., Dept. of Neuropathol. Inst. of Psychiat., and Dept. of Child Health, King's Coll. Hosp., London, England): RELEASE OF GLUCAGON, INDUCED BY STRESS. *Q. J. Exp. Physiol.* 58:99-108, January 1973.

Verbatim summary. When conscious, lightly restrained primates were startled by noise, the level of glucagon in the plasma rose rapidly and this rise was followed by an elevation of blood glucose but not of plasma insulin. In anesthetized animals similar effects were produced by unpleasant stimuli (rectal distension, drilling a burr hole in the skull or the passage of an electric current through the head). These experiments show that glucagon is rapidly released in response to various types of stress.

Bucher, W. H. (Medizinische Abteilung Interspital, Bern, Switzerland): MYOKARDINFARKT UND PLASMAFETTSAUREN. *Schweiz. Med. Wochenschr.* 103:199-203, February 1973.