PROCEEDINGS OF THE BIOCHEMICAL SOCIETY

The 492nd Meeting of the Society was held jointly with the Nutrition Society at the University of Reading on Thursday and Friday, 27 and 28 March 1969, when the following papers were presented:

SYMPOSIUM ON 'BIOCHEMICAL ASSESSMENT OF PROTEIN NEEDS'*

The Absorption of Protein From the Intestine

By K. J. CARPENTER. (*School of Agriculture, University of Cambridge*)

The present state of knowledge about absorption of protein from the intestine will be reviewed and an attempt will be made to relate it to the other topics dealt with in this symposium.

Adaptation of Mammalian Protein Metabolism to Amino Acid Supply

By HAMISH N. MUNRO. (*Physiological Chemistry Laboratories, Department of Nutrition and Food Science, Massachusetts Institute of Technology, Cambridge, Mass., U.S.A.*)

Animals are equipped to make two major types of metabolic adaptation to the supply of nutrients. First, there are transient physiological adaptations to the intermittent intake of nutritionally adequate meals. These account for a large component of the diurnal variations that have been described in the protein metabolism of mammals (Wurtman, 1969). Thus liver polysome aggregation and the activities of some liver enzymes with short half-lives show rhythmic variations with a 24 hr. periodicity that appears to be related to the intermittent intake of protein in each meal. On the other hand, plasma amino acid concentrations undergo an independent diurnal cycle, which may be partly accounted for by amino acid deposition in muscle resulting from the influx of carbohydrate after each meal. Secondly, long-term adaptation of protein metabolism occurs when there is a decrease in the availability of essential amino acids in the diet. Under these circumstances, tissue constituents are lost to varying extents from different parts of the body. It is possible that changes in protein metabolism induced by prolonged amino acid deficiency may represent exaggeration of the normal diurnal metabolic adaptation. It is proposed to discuss mainly the subcellular mechanisms that allow short-term physiological adaptation to variations in amino acid supply.

* The full papers are published in the Proceedings of the Nutrition Society (1969) **28**, No 2.

During the absorptive period after consumption of protein the liver is subjected to an extensive increase in amino acid supply, much of which is diverted within the liver to hepatic protein formation, to plasma protein secretion and into catabolic pathways, so that only a relatively small part passes to the peripheral circulation as free amino acids (Elwyn, 1969). This increase in liver protein synthesis is accompanied by accumulation of polysome aggregates and diminution in dimer and monomer ribosomes and ribosome sub-units. There is evidence to suggest that the turnover of ribosomal RNA is retarded by this increase in the proportion of ribosomes present as polysomes; such an effect would account for the parallel changes in liver RNA and protein contents observed when amino acid supply is varied (Munro, 1968). The response of polysomes to amino acid influx is dependent on the mixture of amino acids undergoing absorption. Thus administration of various amino acid mixtures to rats after a brief period without food shows that polysome aggregation is dependent on the inclusion of tryptophan in the mixture (Proneczuk, Baliga, Triant & Munro, 1968). However, pre-treatment of the animals to alter the intracellular amino acid pools allows the administration of other amino acids to determine polysome aggregation in the liver (A. W. Proneczuk, Q. R. Rogers & H. N. Munro, unpublished work). The regulation of polysome aggregation in relation to amino acid supply appears to be effected by a cytoplasmic mechanism and can consequently be reproduced in cell-free systems (Baliga, Proneczuk & Munro, 1968).

In tissues that undergo cell multiplication, amino acid supply appears to influence cell division. Thus the rate of DNA synthesis in the intestinal mucosa is decreased by brief exposure to a protein-free diet (Munro & Goldberg, 1964), and multiplication of antibody-producing cells in the spleen in response to the challenge of an antigen is also inhibited (Kenney, Roderuck, Arnrich & Piedad, 1968; W. C. Cooper & H. N. Munro, unpublished work). Finally, protein deficiency at certain critical stages in the development of the rat affects the subsequent growth of cell numbers in vulnerable tissues (Winick