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BOOK REVIEWS

Post-translational Modification of Proteins by Lipids.

This book is essentially, as intended, a collection of methodologies and was derived from material presented at an EMBO course in 1987. Each chapter, however, is prefaced by a brief introduction to the subject matter which is adequately referenced for those who wish to explore the literature surrounding the topic in more detail. Although a fairly standard format is adopted for each chapter, the organisation of the methods is rather haphazard and some repetition is inevitable.

Concerning the reported methods, the detail presented is quite variable, but where lacking is again adequately referenced. Most of the methods are in quite common usage and are essentially variants on established themes. Most laboratories will be readily able to adopt these techniques with little modification of normal laboratory protocols and equipment. The main requirements are for radioactive measurements, SDS-PAGE and certain immunological techniques. The chapters can be grouped according to the following arbitrary headings: production and isolation of labelled lipid-modified proteins, enzymology of lipid-modified proteins, separation and identification of lipid and protein components and some miscellaneous headings. Among the latter is the longest chapter in the book (approx. 15% of the total) concerning the related topic of lipid-mediated protein glycosylation.

The examples chosen to illustrate the sources of lipid-modified proteins reflect their wide distribution in nature. These include the variant surface glycoprotein of *Trypanosoma brucei*, membrane anchored alkaline phosphatase of human choriocarcinoma cells, erythrocyte acetylcholine esterase, T-cell surface antigen Thy-1 and certain aquatic angiosperms amongst others.

The book is well produced and the text is laid out in such a way as to ensure a clear presentation of the experimental detail. This is not a book to inspire the newcomer into the field, but is rather to guide those who already have a definite interest to explore this new and expanding topic.

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FREE RADICALS IN DIGESTIVE DISEASES

Proceedings of the 1st International Symposium on Free Radicals in Digestive Diseases, Kyoto, 3 August 1987
Edited by M. Tsuchiya, K. Kawai, M. Kondo and T. Yoshikawa
Excerpta Medica, Amsterdam, 1988
244 Pages

The editors of this symposium remark in their preface that free radical disease is a very difficult concept in clinical medicine, firstly because free radicals cannot be separated and can hardly be measured and secondly because there is no definite procedure to protect tissues from free radical injury. At the present time the concept of free radical disease rests on ischaemia-reperfusion and neutrophil activation models of oxygen free-radical production. The problem is the significance of the models in particular clinical conditions. It is possible to entertain the idea of free radical pathology in a number of digestive diseases. This was the object of the Kyoto symposium. After a general section on free radicals in biology and medicine, the symposium was devoted to free radicals in the stomach; free radicals in the liver and pancreas; and free radicals in the pancreas and small intestine. By an editorial quirk the pancreas is addressed in two sections. It is difficult to detail the large amount of work presented on free radicals and the gastrointestinal tract, liver and pancreas, but some points can be made. Oxygen free-radicals produced after transient ischaemia of the gastric mucosa, such as that induced by stress, may trigger acute gastric ulceration but it is difficult to assign primacy to free radicals in the process. Of interest is protection given by derivatized copper-zinc superoxide dismutase with high affinity for plasma albumin, and therefore slow clearance from the circulation, and the property of accumulation in tissues with low pH, and therefore increased concentration of the perhydroxyl radical which is more toxic than the superoxide anion radical. Gastric mucin is able to mop up free radicals produced by luminal oxidants, as originally proposed by Halliwell. Gastric mucosa itself has relatively low superoxide dismutase and catalase activity and may be particularly susceptible to oxygen free-radical damage. Liver is more robust as shown by the safety of brief clamping of the hepatic artery and portal vein despite the episode of ischaemia-reperfusion. There is anatomical variation in the ability of liver cells to withstand free-radical intermediates and hydroperoxides. Perivenous hepatocytes are more active in xenobiotic detoxification than periportal hepatocytes but are less able to cope with depletion of reduced glutathione and accumulation of reactive intermediates. Oxygen free-radicals seem to be involved in models of acute pancreatitis with enhanced conversion of xanthine dehydrogenase to xanthine oxidase, as shown by the protective effect of superoxide dismutase, catalase and allopurinol, the standard tools of inference, but the clinical significance of these models is not clear. Superoxide dismutase, catalase and allopurinol are effective in the occluded common bile duct model of acute pancreatitis, which resembles the clinical condition of acute reflux pancreatitis, but injection of autologous liver bile into the common bile duct of the dog also liberates phospholipase A_2 (and other pancreatic enzymes) so that oxygen free-radicals cannot be considered sole pathogenic agents. Postischaemic tissue injury by oxygen free radicals was first proposed for small intestine. Shunting of blood across the base of intestinal villi probably aggravates ischaemic changes at the tip of the villi in partial hypoxia. This is but a fragmentary account of the provocative results and ideas presented at the Kyoto symposium. New

insights are welcome in clinical medicine. The work of the symposium will be of interest to gastroenterologists and hepatologists with enthusiasm for free radicals. The editors did not compile a subject index but it is not difficult to find one's way about the book.

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