Microtubules

Microtubules. By P. Dustin. Pp. 452. (Springer: Berlin, Heidelberg and New York, 1978.) DM148; $74.

IT is now fifteen years since Slaughterback first coined the term 'microtubule', and the literature has grown expodentially from a mere two papers in 1963 to several hundred per annum today. During this time, there have been intermittent review chapters and specialised monographs originating from symposia, but never a survey encompassing the whole field. Finally, Dustin has written a book which deals exclusively with microtubules, which should be in every library, and which will be obligatory reading for everyone interested in microtubules.

The first half of the book is primarily concerned with the structure and biochemistry of microtubules, the interactions between microtubules, and microtubule inhibitors, whereas the second half concentrates on aspects of cellular physiology in which microtubules have been implicated, such as movement, secretion, axonal transport, and of course, mitosis. The book is not without its faults: it is for example exclusively a summary of the extensive literature and is not a critical appraisal of the work. Furthermore, it has taken two years to produce since Dustin completed his search of the literature, and so inevitably many current ideas are absent. One example is that the book only briefly mentions microfilaments and lacks any discussion of a possible interaction between microfilaments and microtubules. As such it is interesting to see where we were at two years ago, in the same way that the author's earlier book (with Eigsti), *Colchicine*, is a fascinating account of the field a decade before the visualisation of microtubules. A more serious fault is the review of the literature concerning the biochemistry of microtubules and the kinetics of assembly/disassembly *in vitro*, which is superficial and which fails to relate the properties *in vitro* to the *in vivo* behaviour of microtubules, yet clearly our understanding of the role of microtubules in cellular events will develop from such comparative studies.

Finally, the book is written as a series of independent chapters, each with its own bibliography, with the consequence that it is difficult to search for specific references. Despite these reservations, this is a book that should be read by all cell biologists. **Roy Burns**

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Autoimmunity

Autoimmunity: Genetic, Immunological, Virologic and Clinical Aspects. Edited by N. Talal. Pp. 734. (Academic: New York, San Francisco and London, 1977.) $48.

AUTOIMMUNITY was considered thirty years ago as a contradiction in terms. If immunity, then regarded almost entirely to be due to antibodies, eliminated or neutralised microbes and other foreign substances which had penetrated the body it would be terribly dangerous to make antibodies against one's own tissues. Furthermore, no-one had convincingly shown that antibodies against self components were normally ever made. Antibodies could indeed be induced which reacted with an animal's own spermatozoa, lens protein or even brain tissues, but it was argued (sometimes correctly) that these were exceptions, as the antigens involved were in fact excluded from contact with cells of the immune system; and when brought into contact by infection or by injury they were essentially foreign. With improved techniques, however, undoubted autoantibodies were described in an increasing number of human diseases, such as haemolytic anaemia, thyroiditis, pernicious anaemia and rheumatoid arthritis. Autoantibodies became respectable, although it was still uncertain in most cases whether they caused the disease or were a secondary consequence of tissue damage, which had resulted in liberation of specialised tissue components that were normally hidden intracellularly but were recognised once they entered the blood stream.

The notion that autoimmunity did not occur—expressed by Ehrlich's term 'horror autotoxicus'—had been so widely accepted that few had bothered to question how the body could distinguish self from not self. In terms of current biochemical knowledge the phenomenon was inexplicable, but the clonal selection theory put forward by Burnet in 1959 made sense in terms of cell populations. It proposed that all lymphocytes potentially able to make antibodies against self antigens were eliminated if they met them during foetal life, and that auto-antibodies were the products of 'forbidden' clones, which had undergone somatic mutation after the period of obligatory development of self tolerance was over. This explanation had the added advantage of accounting for the increasing incidence of autoimmunity with age. However, in immunology few hypotheses remain intact for long. It was found that normal animals could quite easily be induced to develop autoantibodies against thyroglobulin by immunising with cross-reacting thyroglobulin, that inbred strains developed anti-thyroid antibodies spontaneously, and that normal animals contained lymphocytes with surface immunoglobulin receptors specific for thyroglobulin (and for other self components) even though they made no detectable antibodies. This implied that a considerable number of forbidden clones could not have been eliminated.

New light was shed when it was realised that specific T lymphocytes, recognising the 'carrier' part of antigens, are required to cooperate with B lymphocytes for the latter to secrete antibodies against a particular antigenic determinant. T lymphocytes are more easily inactivated by contact with antigen; the specific helper T lymphocytes having been eliminated, the B lymphocytes able to make autoantibody would only do so if an alternative set of helper cells were available. These could be provided by stimulating with cross-reacting antigens or self antigens altered in some way, such as by viral infection. Absence of autoimmunity could now be attributed to lack of helper T cells. Next came the recognition of the existence of suppressor T