this volume, but a few other high points can be noted. It is evident from the work of O'Brien *et al.* that the mammalian "mini-ribosomes" of the mitochondrion contain normal numbers of ribosomal proteins even though they have smaller than normal ribosomal RNA's. Attardi *et al.* discuss the evidence for polyadenylate sequences in HeLa cell mitochondrial messenger RNA and review their experiments on molecular mapping of 4*S* and ribosomal RNA of mitochondria. Linnane, Slonimski, and Rabinowitz discuss gene purification in genetically marked petite mutants and the molecular markers (transfer RNA's and ribosomal RNA's) retained in specific petites, as well as the amplification and the structure of repeated sequences in petite mitochondrial DNA. There are also papers on various aspects of mitochondrial genetics in yeast, *Neurospora*, *Paramecium*, and tissue culture cells.

All in all, this is a very useful volume and indispensable for students of organelle biogenesis. Many of the papers are excellent, and the book gives a general picture of the state of the field at the moment and of the problems that lie ahead.

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Cellular Defense Mechanisms

Activation of Macrophages. Proceedings of a conference, Günzberg, Germany, Oct. 1973. W.-H. WAGNER, H. HAHN, and R. EVANS, Eds. Excerpta Medica, Amsterdam, and Elsevier, New York, 1974. xii, 346 pp., illus. $34.60. Workshop Conferences Hoechst, vol. 2.

In the course of infections such as tuberculosis macrophages may become metabolically activated and acquire an enhanced capacity to kill even unrelated microorganisms and, possibly, tumor cells. Thus activated macrophages could play an important role in defense of the host against malignancy as well as infection. This book deals with selected aspects of the physiology of macrophages, its modification by drugs and biological materials, and the interaction of macrophages with infectious agents and tumor cells.

This volume makes apparent some of the problems that have slowed progress in this field. One source of confusion is the lack of definition of what constitutes macrophage activation. Although it is now recognized that the widely dispersed macrophages of the body have a common hemopoietic origin, the cells of the mononuclear phagocyte system (MPS) can display considerable biosynthetic activity and express a wide variety of responses to stimuli in their environment. It is therefore essential to define macrophage activation in relation to a particular in vivo or in vitro model.

The classic models of *Listeria* and bacillus Calmette-Guérin (BCG) infection in the mouse were developed by Mackaness and North and their colleagues. These workers here summarize important new findings on the regulation of macrophage activation. Although activated macrophages represent the effector arm of cell-mediated immunity against *Listeria*, specifically sensitized T lymphocytes play a key role in mobilizing and activating monocytes, which migrate from the bone marrow, and in stimulating the proliferation of macrophages in infected foci. The control of T lymphocyte activation has also been studied in relation to the delayed type of hypersensitivity to sheep red blood cells. Circulating immune complexes inhibit activation of T lymphocytes in heavily immunized animals, but this effect can be counteracted by pretreatment with BCG, which increases removal of complexes by the MPS and also stimulates T lymphocyte proliferation, or by depressing antibody production with cyclophosphamide.

Although a variety of in vitro test systems has been devised to study the microbicidal activity of activated macrophages, their use has been restricted by technical difficulties. Mauel and his colleagues describe an attractive new model in which mouse macrophages can be activated by lymphocyte products to destroy intracellular amastigotes of the protozoan parasite *Leishmania enriettii*. Similar models for macrophage-mediated cytotoxicity against tumor target cells have been developed by Lohmann-Matthes and Evans and their collaborators.

Unfortunately, this volume also illustrates our ignorance of the cellular properties of the activated macrophage. For example, although Huber and Wiener present a useful review of current knowledge of macrophage membrane receptors, little is known about plasma membrane function in activated cells. The absence of suitable biochemical markers has hitherto been a particular handicap in the study of macrophage activation. The more recent discovery that induction and secretion of specific neutral proteinases, such as plasminogen activators, collagenase, and elastase, accompany macrophage activation not only should provide such markers, but also suggest a mechanism by which the activated macrophage could perform some of its effector functions.

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