

Neuroinflammation

MECHANISMS AND MANAGEMENT

Edited by Paul L. Wood

Humana Press, \$145.00, 375 pp

ISBN 0-89603-416-X, 1998

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Inflammation is the classic hallmark of infection and injury in most tissues. The nervous system, and particularly the brain, were thought to exhibit rather limited inflammatory responses, which were largely ignored by most biologists and clinicians. Recent research has changed this view fundamentally and has revealed that the central and peripheral nervous systems can and do exhibit many of the features of inflammation. Indeed such responses seem to contribute directly to numerous diseases of the nervous system. Neuroinflammation is now an area of intense research activity and considerable clinical and commercial interest. Therefore this book, the first major review of the topic, is most timely.

Neuroinflammation: Mechanisms and

Management, edited by Paul Wood, deals with all of the major issues relating to neuroinflammation, and discusses their relevance to diseases of the nervous system and potential therapeutic intervention.

The first chapter (by the editor) describing the role of CNS macrophages in neurodegeneration, provides an excellent, highly readable and very detailed account of the topic with useful tables and summaries and over 500 references. Indeed on the basis of this chapter alone, the book is an excellent guide to the subject. The rest of the book is organized into

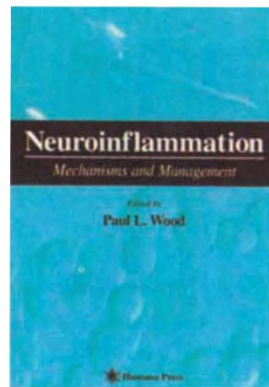
five sections: microglia, acute phase proteins, cytokines, free radicals and miscellaneous mediators, including 14 well referenced and illustrated chapters, each written by active researchers in the field.

As with many multi-author books there is some overlap and repetition between chapters. This overlap is probably compounded by the varied approaches of different contributors. Some address specific cell mediators or molecules (glia, complement, cytokines, free radicals, adhesion molecules, nitric oxide and cyclooxygenase 2), whereas other chapters deal with disease states (Alzheimer's, head injury, multiple sclerosis). As a re-

sult, specific mediators such as IL-1 and complement are discussed in depth, whereas others (eg TNF) are included in several separate chapters but are covered in less detail. Similarly certain clinical conditions (stroke, peripheral nerve injury) are discussed in less details than others (Alzheimer's, multiple sclerosis). Surprisingly the major feature of inflammation, invasion of inflammatory cells, is included only towards the end of the book in a chapter on adhesion molecules. The chapters on complement and IL-1, each combine eloquent descriptions of basic and research find-

ings with discussion of the applied implications for disease treatment.

This book will reach a wide audience including basic and clinical neuroscientists, and fills a significant gap in the literature. For those working within the field, it will be particularly useful as a reference material, and for "dipping into" chapters on specific topics. For neuroscientists wishing to attain a general understanding of neuroinflammation, the book provides an excellent starting point, and offers an insight into the rapid pace of discovery within a field which influences most aspects of nervous system function and disease.



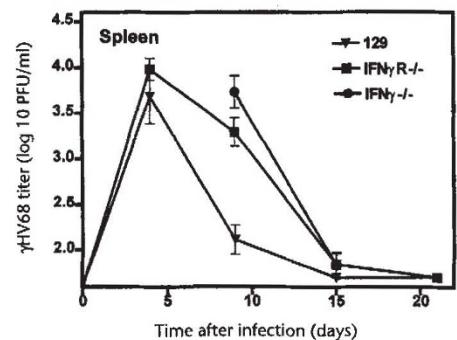
ERRATUM

Murine γ -herpesvirus 68 causes severe large-vessel arteritis in mice lacking interferon- γ responsiveness: A new model for virus-induced vascular disease

K.E. WECK, A.J. DAL CANTO, J.D. GOULD, A.K. O'GUIN, K.A. ROTH, J.E. SAFFITZ, S.H. SPECK & H.W. VIRGIN

Nature Med. 3, 1346–1353 (1997)

In the December 1997 issue, on page 1348, the x axis for Fig. 2 was incorrectly labeled. The correct figure is displayed to the right. We regret this error.



Correction

Adenovirus-mediated gene transfer into cold-preserved liver allografts: Survival pattern and unresponsiveness following transduction with CTLA4lg

K.M. OLTHOFF, T.A. JUDGE, A.E. GELMAN, X. DA SHEN, W.W. HANCOCK, L.A. TURKA & A. SHAKED

Nature Med. 4, 194–200 (1998)

The work described in the paper by Othoff *et al.*, was also supported in part by NIH grant AI37691 to L.A. Turka.