complete picture. So, despite this fine contribution, it could take another volume to answer the widely asked question: 'have antisense and gene therapy delivered their early promise?' With genetic therapies predicted by many (myself included) to form the mainstay of disease control in the next millennium, there will undoubtedly be another opportunity for Eric Wickstrom to provide the complete picture. This volume has certainly whetted my appetite!

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Neuroinflammation: Mechanisms and Management

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Multiple sclerosis is the archetypal disease of the brain and, for many years, meetings or reviews on neuroinflammatory disease were dominated by, if not wholly concerned with, discussion of multiple sclerosis and relevant animal models. In the past ten years this has changed. Neuroinflammation is of interest and concern to a diverse spectrum of basic and clinical neuroscientists, including those at the acute end of the disease spectrum studying neuroscience and head trauma, and those at the chronic end of the disease spectrum studying Alzheimer's and Parkinson's disease. It is interesting that the pharmaceutical industry has been as rapid to enter the arena as academic groups, and the significant contribution to this book by scientists from the industrial sector is testimony to this. There is a clear perception that this is an arena that will provide novel therapeutic targets in diseases for which effective treatments have yet to be found.

The inflammatory response in the brain is dominated in many circumstances by cells of the mononuclear phagocyte lineage and, in particular, the resident macrophages of the brain, the microglia. In no other organ of the body is there such intense interest in the contribution of the resident macrophages to inflammation, rather than the leukocytes recruited from the blood. It is apparent that macrophages can generate a plethora of inflammatory mediators, and so too can microglia *in vitro*. Wood provides extensive lists of receptors and secretory products expressed by microglia *in vitro*, and a few of these, especially components of the complement cascade (Walker, Rogers), and some cytokines (Martin) have been investigated *in vivo*. A clear message emerges that the microenvironment of the CNS imposes on the microglia a highly atypical macrophage phenotype. The normally quiescent microglia can be rapidly activated by a very diverse range of insults to the CNS, although what determines the mediators that the microglia synthesize under particular conditions is poorly understood.

A key question for those studying the atypical inflammatory response in the brain is whether the inflammation is a consequence of the disease, contributes to the disease, or is causal. It is clear that, in multiple sclerosis, inflammation is the cause of the disease, while in stroke (Kato) and Alzheimer's disease (Rogers and Griffin) there is evidence that the inflammatory response contributes to the outcome; the consensus opinion is that it contributes unfavourably. The mechanisms by which particular mediators contribute to the disease process are poorly understood. Although the actions of novel neurotoxins (Giulian and Li), nitric oxide (Boje) and eicosanoids (Rodger and Chan) are well studied *in vitro* and can be shown to be harmful to neurones, several authors highlight the need for data from good *in vivo* studies.

Few interested in neuroinflammation can fail to be aware of the importance of the trials with β-interferon in multiple sclerosis. These trials showed that inflammation in the brain in a chronic disabling disease was treatable and, at least in a proportion of patients, with significant benefit (Lechtenberg). The mechanism of action, however, remains to be defined. The relative lack of management of neuroinflammation is partly reflected in the fact that, while treatment of spinal cord injury with steroids (Hall) is discussed, as is β-interferon, these are the only two chapters given to the therapeutic issues. There is much to be done.

For those already working in the rapidly expanding area of neuroinflammation there is little that is new. The book might provide a useful reference source, but the rather limited index and the organization of references in each chapter, numbered in order of appearance in the text, are not user-friendly attributes. For those about to enter the field there are sufficient chapters in diverse areas that would make this a useful purchase but, at £110, not an essential one.

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