At first sight the title of this book excited us enormously. There is an urgent need for a comprehensive source book summarizing current information on neuroinflammation and its management. However, in this respect the book fails to deliver. Paul Wood, an acknowledged expert on microglia and CNS macrophages, has assembled a group of like-minded authors, all except one from North America. Several focus almost solely on microglia, to the exclusion of other brain cells, notably astrocytes, which undoubtedly contribute to neuroinflammation. The book addresses both basic mechanisms and clinical applications, and many of the chapters are excellent reviews of their respective areas.
However, several important topics of clear relevance to neuroinflammation are not discussed. For example, there is no discussion of chemokines and chemokine receptors, little on the contributions of neurons and oligodendroglia, and almost nothing on apoptosis of brain cells in inflammation. Others will have their own lists of what is missing. Despite these shortcomings, perhaps inevitable in a brief work of this sort, there is much here which is worthy.

Part I (four chapters) is explicitly focused on microglia. A comprehensive (over 500 references) introduction by Wood takes us from properties of microglia and microglial cell lines in vitro through to descriptions of animal models in which microglia are implicated. Walker and Kato provide accounts of activation markers on microglia in neurodegeneration and stroke, respectively. Walker also introduces the concept of anti-inflammatory therapy for neurodegeneration, an important theme repeated through the volume. Guilian and Li discuss the evidence that destruction of neurons by activated microglia is responsible for much of the gradual cognitive decline typifying dementia.

Part II (two chapters) purports to describe the roles of acute phase proteins in neurodegeneration. Even for those, like us, biased in favour of the system, the focus here on complement is very surprising. Spiegel et al. provide a strong account of the complement system and its role in neurological disorders and add to this with a review of current strategies for inhibiting complement in vivo. It is unfortunate that some aspects are then repeated in the following chapter by Rogers and Griffin who give short shrift to the many other potential inflammatory mediators and spend almost half the chapter discussing the role of complement and its receptors.

Part III (three chapters) discusses several aspects of cytokines in CNS disease. Martin et al. provide an interesting account of the evidence that IL-1 drives inflammation in brain ischaemia and other forms of brain injury and neurodegeneration, and suggest that therapy with IL-1 receptor antagonist might dampen this inflammation. They make the point that other cytokine systems also play a role in neurological disorders and that a single cytokine cannot be considered in isolation. Indeed, Ott et al. hammer home this point by describing the pharmacopoeia of cytokines implicated in head injury and the evidence that this cytokine cocktail might drive brain and peripheral organ damage following head injury. Whether therapies aimed at inhibiting single cytokines in the cocktail will be effective remains to be seen. Lechtenberg focuses on multiple sclerosis and, after a long introduction, briefly discusses the use of interferons in therapy of this disease.

Free radicals are very much the flavour of the month in many fields and rate their own section. Hensley et al. provide a detailed introduction to reactive oxygen species and progress to summarise evidence of their involvement in diverse neurological disorders, notably neurodegeneration. The prospect of using simple antioxidant therapies in these conditions is indeed exciting. Hall describes the development of such agents, a series of powerful antioxidant steroids which have already been used to inhibit the generation of reactive oxygen species and subsequent lipid peroxidation in spinal cord injury. These agents have been clearly shown to improve neurological recovery in animal models and humans with minimal side-effects.

The final part contains a miscellany of chapters which didn’t quite fit anywhere else. Andrew provides a competent review of adhesion molecules involved in leukocyte trafficking into sites of inflammation. Leukocyte infiltration is important in acute and chronic inflammation in many organs, including the brain, and strategies aimed at interfering with the various stages of this process might well be of therapeutic value in CNS inflammation. Boje discusses the evidence that NO generation by glia might contribute to neuronal damage in neurodegeneration and following brain injury or infection, raising the prospect of using inhibitors of NO synthase in therapy. Finally, Rodger and Chan focus on the roles of proinflammatory prostaglandins such as PGE\textsubscript{2} in inflammation. Synthesis of these agents from arachidonic acid is catalysed by cyclo-oxygenases and the inducible isoform of cyclo-oxygenase, COX-2, is recognized as an excellent target for anti-inflammatory therapies.

In summary, this is a useful contribution to the literature which, while far from comprehensive, is current and does highlight some important areas of inflammation relevant to CNS disease. The plethora of anti-inflammatory approaches described in this brief volume gives some hope that effective therapies will follow, although the complexity of CNS inflammation and difficulties of delivery to the brain will no doubt continue to present major therapeutic challenges.

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