

Preface

Up until approximately 20 years ago, the idea that the central nervous system (CNS) and components of the immune system were dynamically interactive was considered impossible (or at least highly unlikely) as the CNS was judged an immunosuppressive environment based upon experimental evidence highlighting the survival of tissue grafts within the brain. Additional evidence supporting this viewpoint included (i) the presence of the blood–brain barrier (BBB) which provides a physical and physiological obstruction that is difficult for cells and macromolecules to cross, (ii) the relative absence of MHC class I and II expression on CNS cells like astrocytes and neurons, and (iii) lack of abundant antigen presenting cells (APC) which are required for the generation of an adaptive immune response. However, in spite of these obstacles, it is now well-accepted that the CNS is routinely subject to immune surveillance under both normal as well as diseased conditions. Indeed, activated cells of the immune system such as T and B lymphocytes and monocyte/macrophages readily infiltrate and accumulate within the CNS following microbial infection, injury, or upon development of autoimmune responses directed toward resident antigens of the CNS.

The importance of studying events surrounding the initiation and maintenance of neuroinflammation is now recognized by scientists and clinicians alike as critical not only in characterizing the complex mechanisms associated with host defense following infection but also in contributing to neurologic disease. Much of our understanding of neuroinflammation has been derived from numerous animal studies of neurologic disease including autoimmune models of demyelination such as experimental autoimmune encephalomyelitis (EAE) and transgenic mice, microbial e.g. virus and bacteria infections, spinal cord injuries in mice and rats, and mouse models of Alzheimer's to highlight just a few. Moreover, the underlying molecular and cellular mechanisms governing inflammation are just now being understood and the importance of chemokines and chemokine receptors in recruiting targeted populations of leukocytes into the CNS is now appreciated. In addition, resident cells of the CNS e.g. microglia are recognized as important mediators in regulating innate defense mechanisms as well as disease.

This volume highlights important advances in our understanding of different aspects of neuroinflammation with a concentration on specific areas focusing on

glial activation, molecular signals regulating inflammation and neurotoxicity, immune responses concentrating within the CNS, and the emergence of transgenic models of neurologic disease. It was the goal of the editors to provide timely and insightful comments on these particular aspects of neuroinflammation and disease while recognizing that it was impossible to adequately address other equally important and relevant aspects of neuroinflammation not covered but certainly deserving of attention. In addition, the editors feel this text will be useful for researchers, clinicians, as well as a valuable resource for students interested in the fascinating arena of neuroinflammation.

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