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Sommario/riassunto	Inflammation of the brain in the context of neurodegenerative disorders is an area of intense debate and discussion, not least in terms of its pathogenic significance and the extent to which it drives disease processes and pathology. This inflammation can take several forms including innate responses involving microglia, humoral responses involving antibody, complement mediated processes and cellular T-cell activation, of which the role and extent of each may differ between diseases. Whilst some diseases have been more intensely linked to inflammation and long-term degeneration (e.g. MS), more traditional chronic neurodegenerative disorders have been thought of in terms of intrinsic neuronal pathology with a secondary innate response. However, it has been described that microglia activation is an early event of many degenerative disorders and evidence is accumulating that it may play a critical role in actually causing pathology and driving disease processes. If true, this would have major therapeutic implications, but what is the evidence that this is the case?The initial observations by Patrick McGeer’s group of post-mortem tissue from patients with Parkinson’s disease revealed the presence of activated brain microglia long-term and has thus lead to the hypothesis that chronic inflammation could participate to neuronal degenerative processes. The significance of these original observations

has only been recently revisited, and the development of more powerful tools to study the brain immune response has certainly contributed to this field of research. Chronic inflammation in the brain can take many forms but of particular interest has been the resident microglia and the role they play in this process. In this context, microglia have often been thought to become activated only after the disease has begun and then to contribute minimally to the degenerative process. Emerging new concepts challenge this view by proposing that microglial senescence, for example, may release the disease process and/or accelerate it. In addition, microglia, once activated, can adopt different phenotypes which can be both pro-inflammatory and pro-repair and may impact not only on the healthy adult neuronal population but on those new neurons derived from neurogenic niches of the adult brain.In this Research Topic, we attempt to explore this by first considering the innate immune responses in the brain and the methods by which they can be studied experimentally and in patients with various neurodegenerative disorders. This sets the scene for then discussing a range of different disorders including Alzheimer’s, Parkinson’s, Huntington’s disease and amyotrophic lateral sclerosis. These papers seek to discuss the evidence for an innate immune response and whether this is beneficial or detrimental, as well as its therapeutic implications.