Inflammation is associated with both acute and chronic neurological disorders, such as stroke, multiple sclerosis, inflammatory neuropathies and Alzheimer’s disease (AD). The immune system interacts with the nervous system in regulation of physiological processes and is involved in pathophysiological mechanisms of various neurodegenerative disorders, including AD, frontotemporal lobar degeneration (FTLD) with loss of function progranulin mutations, and Lewy body dementia. Increased levels of pro-inflammatory factors such as cytokines and chemokines occurs in brains affected by neurodegeneration. Vascular dementia, as a result of different types of cerebrovascular disease, is also characterized by ischemia-associated brain inflammation, beside many other pathogenic factors. Systemic inflammation per se is a well accepted vascular risk factor and most of dementia cases are mixed dementia, with a vascular component. Studies in AD animal models showed that presenilin-1 is crucial for both age-dependent Abeta accumulation and inflammation and mutations in presenilin genes exacerbate inflammation in AD brains. Furthermore, according to recent reports, extracellular α-synuclein seems to trigger microglia activation, making these immune competent cells vulnerable to environmental toxic proinflammatory factors. Moreover, circulating IL-6 is increased in PGRN-mutated FTLD patients and PGRN-deficient mice brains show enhanced inflammation and progressive neurodegeneration. However, a regular anti-inflammatory treatment might not be beneficial in dementia patients, since in brain cytokins have dual roles, linked both to neurotoxicity and physiological intercellular signalling.