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Sommario/riassunto	Microglial cells play a vital role in the innate immune response occurring in the Central Nervous System (CNS). Under physiologic conditions, microglia dynamically patrol the brain parenchyma and participate in the remodeling of active neuronal circuits. Accordingly, microglia can boost synaptic plasticity by removing apoptotic cells and by phagocytizing axon terminals and dendritic spines that form inappropriate neural connections. Upon brain and spinal cord injury or infection, microglia act as the first line of immune defense by promoting the clearance of damaged cells or infectious agents and by releasing neurotrophins and/ or proneurogenic factors that support neuronal survival and regeneration.Recently, two main pathways were suggested for microglia activation upon stimuli. Classical activation is induced by Toll-like receptor agonists and Th1 cytokines and polarizes cells to an M1 state, mainly leading to the release of TNF-alpha, IL-6 and nitric oxide and to grave neural damage. Alternative activation is mediated by Th2 cytokines and polarizes cells to an M2a state inducing the release of antiinflammatory factors. These findings have further fueled the discussion on whether microglia has a detrimental or

beneficial action (M1 or M2-associated phenotypes, respectively) in the diseased or injured CNS and, more importantly, on whether we can shift the balance to a positive outcome.Although microglia and macrophages share several common features, upon M1 and M2 polarizing conditions, they are believed to develop distinct phenotypic and functional properties which translate into different patterns of activity. Moreover, microglia/macrophages seem to have developed a tightly organized system of maintenance of CNS homeostasis, since cells found in different structures have different morphology and specific function (e.g. meningeal macrophages, perivascular macrophages, choroid plexus macrophages). Nevertheless, though substantial work has been devoted to microglia function, consensus around their exact origin, their role during development, as well as the exact nature of their interaction with other cells of the CNS has not been met.This issue discusses how microglial cells sustain neuronal activity and plasticity in the healthy CNS as well as the cellular and molecular mechanisms developed by microglia in response to injury and disease. Understanding the mechanisms involved in microglia actions will enforce the development of new strategies to promote an efficient CNS repair by committing microglia towards neuronal survival and regeneration.