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Sommario/riassunto

Mechanisms of brain-immune interactions became a cutting-edge topic in systemic neurosciences over the past years. Acute lesions of the brain parenchyma, particularly, induce a profound and highly complex neuroinflammatory reaction with similar mechanistic properties between differing disease paradigms like ischemic stroke, intracerebral hemorrhage (ICH) and traumatic brain injury (TBI). Resident microglial cells sense tissue damage and initiate inflammation, activation of the endothelial brain-immune interface promotes recruitment of systemic immune cells to the brain and systemic humoral immune mediators (e. g. complements and cytokines) enter the brain through the damaged blood-brain barrier. These cellular and humoral constituents of the neuroinflammatory reaction to brain injury contribute substantially to secondary brain damage and neurodegeneration. Diverse inflammatory cascades such as pro-inflammatory cytokine secretion of invading leukocytes and direct cell-cell-contact cytotoxicity between lymphocytes and neurons have been demonstrated to mediate the inflammatory 'collateral damage' in models of acute brain injury. Besides mediating neuronal cell loss and degeneration, secondary inflammatory mechanisms also contribute to functional modulation of neurons and the impact of post-lesional neuroinflammation can even be detected on the behavioral level. The contribution of several specific immune cell subpopulations to the complex orchestration of secondary neuroinflammation has been revealed just recently. However, the

differential vulnerability of specific neuronal cell types and the molecular mechanisms of inflammatory neurodegeneration are still elusive. Furthermore, we are only on the verge of characterizing the control of long-term recovery and neuronal plasticity after brain damage by inflammatory pathways. Yet, a more detailed but also comprehensive understanding of the multifaceted interaction of these two supersystems is of direct translational relevance. Immunotherapeutic strategies currently shift to the center of translational research in acute CNS lesion since all clinical trials investigating direct neuroprotective therapies failed. To advance our knowledge on brain-immune communications after brain damage an interdisciplinary approach covered by cellular neuroscience as well as neuroimmunology, brain imaging and behavioral sciences is crucial to thoroughly depict the intricate mechanisms.