Record Nr. UNINA9910136799503321

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Titolo Mechanisms of neuroinflammation and inflammatory

neurodegeneration in acute brain injury

Pubbl/distr/stampa Frontiers Media SA, 2015

Descrizione fisica 1 online resource (284 p.)

Collana Frontiers Research Topics

Soggetti Neurosciences

Lingua di pubblicazione Inglese

Formato Materiale a stampa

Livello bibliografico Monografia

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.

2. Record Nr. UNINA9910585941503321

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Titolo Occupational Health and Safety in the Healthcare Sector

Pubbl/distr/stampa Basel, : MDPI - Multidisciplinary Digital Publishing Institute, 2022

Descrizione fisica 1 online resource (282 p.)

Soggetti Humanities

Social interaction

Inglese

Lingua di pubblicazione

Formato Materiale a stampa

Livello bibliografico Monografia

Sommario/riassunto Healthcare workers are exposed to several different occupational risk

factors, and they pay an important tribute in terms of occupational diseases and work-related injuries. Currently, the COVID-19 pandemic has focused the attention on the problem of the infectious risk, which is certainly among the risks typically expected and specifically

recognized for the health personnel, but also other occupational risks

should not be overlooked, such as, e.g., the risks associated with workorganization factors and with the exposure to chemical and physical agents. The health consequences associated with the exposure to all these factors have relevant impacts in terms of induced diseases, DALYs, sickness absence from work and costs for the health systems. According to these premises, this reprint has collected manuscripts addressing topics related to the prevention of the occupational risks in the healthcare sector, including original articles and reviews on the prevention of work-related illnesses and injuries of the health personnel, as well as on the evaluation of the risks in the healthcare workplaces, and on the topics of risk perception and of the knowledge and attitudes of the workers towards the preventive procedures and the use of protections. The themes of the prevention of occupational infectious risk, biomechanical overload of the musculoskeletal system and work-related psychosocial factors are specifically discussed in the papers collected.