1. Record Nr. UNINA9910346753903321

Autore	Atsuyoshi Shimada
Titolo	Neuroimmune Interface in Health and Diseases
Pubbl/distr/stampa	Frontiers Media SA, 2017
Descrizione fisica	1 online resource (174 p.)
Collana	Frontiers Research Topics
Soggetti	Medicine
Lingua di pubblicazione	Inglese
Formato	Materiale a stampa
Livello bibliografico	Monografia

Sommario/riassunto

It is now well appreciated that the immune system, in addition to its traditional role in defending the organism against pathogens, communicate in a well-organized fashion with the brain to maintain homeostasis and regulate a set of neural functions. Perturbation in this brain-immune interactions due to inflammatory responses may lead to psychiatric and neurological disorders. Microglia are one of the essential cells involved in the brain-immune interactions. Microglial cells are now not simply regarded as resident tissue macrophages in the brain. These cells are derived from myeloid progenitor cells in the yolk sac in early gestation, travel to the brain parenchyma and interact actively with neurons during the critical period of neurogenesis. Microglia provide a trophic support to developing neurons and take part in the neural wiring through the activity-dependent synapse elimination via direct neuron-microglia interactions. Altered microglial functions including changes in the gene expression due to early life inflammatory events or psychological and environmental stressors can be causally related to neurodevelopmental diseases and mental health disorders. This type of alterations in the neural functions can occur in the absence of infiltration of inflammatory cells in the brain parenchyma or leptomeninges. In this sense, the pathogenetic state underlying a significant part of psychiatric and neurological diseases may be similar to "para-inflammation", an intermediate state between homeostatic and classical inflammatory states as defined by Ruslan

Medzhitov (Nature 454:428-35, 2008). Therefore, it is important to study how systemic inflammation affects brain health and how local peripheral inflammation induces changes in the brain microenvironment. Chronic pain is also induced by disturbance in otherwise well-organized multisystem interplay comprising of reciprocal neural, endocrine and immune interactions. Especially, early-life insults including exposure to immune challenges can alter the neuroanatomical components of nociception, which induces altered pain response later in life. Recently the discrete roles of microglia and blood monocyte-derived macrophages are being defined. The distinction may be further highlighted by disorders in which the brain parenchymal tissue is damaged. Therefore, studies investigating the dynamics of immune cells in traumatic brain injury and neurotropic viral infections including human immunodeficiency virus, etc. as well as neurodegenerative diseases such as amyotrophic lateral sclerosis are promising to clarify the interplay between the central nervous and immune systems. The understanding of the histological architecture providing the infrastructure of such neuro-immune interplay is also essential. This Frontiers research topic brings together fourteen articles and aims to create a platform for researchers in the field of psychoneuroimmunology to share the recent theories, hypotheses and future perspectives regarding open questions on the mechanisms of cell-cell interactions with chemical mediators among the nervous, immune and endocrine systems. We hope that this platform would reveal the relevance of the studies on multisystem interactions to enhance the understanding of the mechanisms underlying a wide variety of neurological and psychiatric disorders.