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Editorial

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Immune Cells Silence Neurons

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Damage to the brain gray matter plays an important role in the progression of multiple sclerosis. This study now shows that such damage can be caused by inflammatory reactions that lead to loss of synapses, which impairs neural activity. Gray matter damage associated with multiple sclerosis progression may be caused by inflammatory reactions that lead to synaptic loss. This study now shows that such damage can be caused by inflammatory reactions that lead to loss of synapses, which impairs neural activity. These immune cells preferentially eliminate spines, which contain high levels of calcium. We assume that the inflammation reaction itself triggers an influx of calcium, which destabilizes the spines," says Kerschensteiner.

These results contrast with findings in patients with progressive MS, in whom the cerebral cortex is permanently damaged. The interplay between the immune and nervous systems has been acknowledged in the past, but only more recent studies have started to unravel the cellular and molecular players of such interactions. Mounting evidence indicates that environmental signals are sensed by discrete neuro–immune cell units (NICUs), which represent defined anatomical locations in which immune and neuronal cells colocalize and functionally interact to steer tissue physiology and protection. The latter comprises neurons and glial cells organized in nerve bundles, coming from the CNS. Immune responses have been shown to be modulated by the peripheral nervous system. We evaluate the potential benefits to the immune system that arise by taking advantage of some of the brain's unique features, such as its capacity to integrate and synchronize physiological functions, its predictive capacity and its speed of response. The sensory nervous and immune systems, historically considered autonomous, actually work in concert to promote host defense and tissue homeostasis. These systems interact with each other through a common language of cell surface G protein-coupled receptors and receptor tyrosine kinases as well as cytokines, growth factors, and neuropeptides. Lung nociceptors initiate cough and bronchoconstriction. To elucidate if these fibers also contribute to allergic airway inflammation, we stimulated lung nociceptors with capsaicin and observed increased neuropeptide release and immune cell infiltration.

Lung nociceptors initiate cough and bronchoconstriction. To elucidate if these fibers also contribute to allergic airway inflammation we stimulated lung nociceptors with capsaicin and observed increased neuropeptide release and immune cell infiltration. Immune responses in the CNS are common, despite its perception as a site of immune privilege. These responses can be mediated by resident microglia and astrocytes, which are innate immune cells without direct counterparts in the periphery. The nervous system regulates immunity and inflammation. The molecular detection of pathogen fragments, cytokines, and other immune molecules by sensory neurons generates immunoregulatory responses through efferent autonomic neuron signalling. Loss of synapses - the structures that serve as functional contacts between nerve cells - is an early indicator of damage to the cerebral cortex in cases of progressive MS.

The researchers therefore suspected that the synapses are the key to the neuronal damage that ensues in this stage of the disease. Our results indicate that nociceptors amplify pathological adaptive immune responses and that silencing these neurons with QX-314 interrupts this neuro-immune interplay, revealing a potential new therapeutic strategy for asthma.

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