Commentary

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There are few Effective Therapies for those Living with Chronic Multiple Sclerosis Pain

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Description

More than half of people with multiple sclerosis experience severe quality of life impairment as a result of chronic pain. Experimental autoimmune encephalomyelitis is a model of multiple sclerosis that typically presents with paralysis of the hind limb, neuro inflammation, and neuro degeneration. Despite the absence of apparent hypersensitivity observed post-peak disease, this paralysis may prevent the use of pain behavior tests. We sought to optimize the pain phenotype of the traditional actively-induced EAE model. MOG35-55/CFA and 100–600ng pertussis toxin were used to induce EAE, and mechanical, cold, and thermal sensitivity was measured in the mice after 28 days. In order to evaluate demyelination and neuro inflammation, spinal cord tissue was collected 14 and 28 days after the injection. Mechanical hypersensitivity was only seen in mice that received 100ng of PTX. Demyelination, immune cell recruitment, cytokine expression, glial activation, and neuronal damage are hallmarks of disease pathology. EAE mice induced with moderate (200ng) doses of pertussis toxin outperformed EAE mice treated with low levels (100ng).In both EAE groups, immune staining revealed activated astrocytes and myeloid/microglial cells. Based on these findings, it may be possible to study pain behaviors in patients with EAE who still exhibit disease pathology.

Autoimmune Encephalomyelitis

The mechanisms underlying pain may be better studied with this altered model. Over 2.5 million people worldwide suffer from Multiple Sclerosis (MS); an autoimmune disease of the nervous system.MS is thought to be started by a CD4+T cell-mediated response that attacks the myelin sheaths that surround axons. This causes significant motor, sensory, and cognitive deficits, as well as autonomic dysfunction, fatigue, and pain. The exact cause of MS is unknown. A common murine model of multiple sclerosis that mimics many of these symptoms is experimental autoimmune encephalomyelitis, which can be induced by adoptive transfer of encephalitogenic T cells into naive animals or by administering myelin protein/peptides as an adjuvant. The onset of clinical symptoms one to two weeks after disease onset serves as a useful model for investigating potential mechanisms underlying disease outcomes. Neuro inflammation, demyelination of the brain and spinal cord, as well as changes in functional outcomes like motor function loss and altered sensory outcomes are hallmarks of EAE, which is similar to MS. Pain is a symptom that is very common in multiple sclerosis and has a positive correlation with disability and poor quality of life related to MS. MS pain can sometimes be lesional or secondary. However, the underlying causes of MS's chronic neuropathic pain are largely unknown. To investigate the pathophysiology of central neuropathic pain in MS, it is essential to employ a trustworthy animal model, such as those developed by other groups. Like individuals living with MS, enlistment of EAE in the mouse brings about the presence of cold and mechanical allodynia before the beginning of engine shortfalls; intriguingly, neither EAE animals nor MS sufferers typically exhibit thermal hyperalgesia, which is hypersensitivity to noxious stimuli like intense heat. However, there is a gap between MS and EAE pain: While most people with MS experience an increase in pain sensitivity as their disease and symptoms progress, EAE animals exhibit a diminished or absent response to sensory stimuli during the diseases peak and chronic phases. The hind limb loss of motion instigated by the EAE model prevents the utilization of usually utilized upgrade evoked torment conduct tests. The von Frey assay for mechanical sensitivity, the acetone test for cold sensitivity, and the Hargreaves radiant heat test for thermal sensitivity are examples of stimulus-evoked tests used to evaluate somatosensory responses in rodents. The animal's ability to respond to a stimulus applied to the hind paw is the foundation of these frequently used tests. As a result, the associated loss of motor function or ability, as observed in studies employing the EAE model, is a significant confounding factor in animal studies. As a result, we set out to improve a model that would allow us to study EAE's sensory phenotypes while keeping the characteristic changes in neuro inflammation and demyelination. We wanted to use the more common complete Freund's adjuvant and pertussis toxin model in our study, despite the fact that several modified models of EAE are used, such as those that use Quil-A or no pertussis toxin to cause disease. This was made possible by altering the amount of the pertussis toxin that was used to break through the blood-brain

barrier and allow immune cells to enter the central nervous system (CNS).

Clinical Signs of Disease and Other Hallmarks

The central demyelination and glial activation, were reduced when pertussis doses were decreased. According to our findings, it is possible to evaluate hypersensitivity in EAE animals when it is separated from motor function loss. Understanding the underlying cellular and molecular control of pain in autoimmune disease may now be easier thanks to this. All experiments utilized female C57BL/6J mice from Jackson Laboratories, Bar Harbor, ME, who were bred in-house and weighed between 16 and 21 grams. In accordance with a standard 12-hour light cycle, mice were housed in cages ranging in size from 2 to 5, and they had free access to food and water. The Canadian Council on Animal Care's guidelines was followed and all animal experiments and procedures were approved by the Queen's University Animal Care Committee. 50 mg of myelin oligodendrocyte glycoprotein was used to induce experimental autoimmune encephalomyelitis. Complete Freund's adjuvant Pertussis toxin and 100–600ng for behavioral work Mention Campbell Biological Laboratories. A second lot of PTX was also used for molecular biology research. xylazine: acepromazine, their lower backs were shaved to reveal the injection site, and 100 l of an emulsion of MOG35-55 and CFA was injected subcutaneously at two locations on the lower back. 100, 200, 400, or 600ng of PTX was dissolved in 200 l of phosphate buffered saline was injected intraperitoneally. 48 hours later, a second equal injection of PTX was given. An experimenter who was not aware of the treatment groups kept a 5-point clinical score on the mice every day up until 28 days after vaccination: zero; no motor symptoms;0.5, with a limp tail;1 tail paralyzed; 2, lack of coordination in one's movements; 2.5, a paralyzed hind limb; 3, the two rear appendages deadened;4, paralyzed forelimbs;5, defunct Once the mice achieved a score of 2 or higher, they were given moist food at the bottom of the cage and given daily injections of 1 ml saline. 100ng of PTX and CFA emulsified in phosphate buffered saline were injected into sham-injected controls. To prevent skin damage, green clay was applied to any lesions that appeared at the injection site during the study.