New MS target identified by Canadian researchers

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Multiple sclerosis (MS) is a disease caused by damage to myelin - the protective covering wrapped around the nerves of the central nervous system (CNS).

Previous studies have shown that certain white blood (immune) cells, called leukocytes, infiltrate the CNS and play a significant role in causing the damage that contributes to MS symptoms. It has also been shown that these leukocytes enter the CNS with help from a family of molecules called MMPs.

Using a mouse model, researchers have discovered that a molecular switch called EMMPRIN plays an important role in MS. The researchers explored how in MS, EMMPRIN affects MMPs and the entry of leukocytes into the CNS to result in disease activity.

"In our studies we inhibited EMMPRIN and noticed a reduced intensity of MS-like symptoms in mice," says Dr. V. Wee Yong, a professor of Clinical Neurosciences at the Hotchkiss Brain Institute at the University of Calgary's Faculty of Medicine and the study's principal investigator. "Our data suggests that if we target EMMPRIN in patients with MS, we may reduce the injury to the brain and spinal cord caused by immune cells."

In addition to working with animal models, the authors also found that EMMPRIN is significantly elevated in the brain lesions of MS patients, indicating its potential significance in the disease.

"This study has identified a new factor in MS, the blockade of which resolves disease activity in an animal model of MS. The results are exciting as they offer new insights into the MS disease process", says Dr. Smriti Agrawal, a postdoctoral fellow in Dr. Yong's lab and the study's lead author.

"The authors have extended our knowledge of the molecules that regulate the trafficking of immune cells into the nervous system as occurs in multiple sclerosis. The current study identifies a new factor that can serve as a potential target of MS therapeutics," says Dr. Jack Antel, Professor of Neurology at McGill University.

The research findings are published in the Jan 12th issue of the *Journal of Neuroscience*.

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