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Study identifies protein to repair damaged brain tissue in MS

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Vittorio Gallo, PhD, Director of the Center for Neuroscience Research at Children's National Health System, and other researchers have found a "potentially novel therapeutic target" to reduce the rate of deterioration and to promote growth of brain cells damaged by multiple sclerosis (MS). Current therapies can be effective in patients with relapsing MS, but have little impact in promoting tissue growth.

The brain produces new cells to repair the damage from MS years after symptoms appear. However, in most cases the cells are unable to complete the repair, as unknown factors limit this process. In MS patients, brain inflammation in random patches, or lesions, leads to destruction of myelin, the fatty covering that insulates nerve cell fibers called axons in the brain, and aids in transmission of signals to other neurons.

In yesterday's publication of *Neuron*, Gallo, who also is a professor of pediatrics at the George Washington University School of Medicine and Health Sciences (SMHS), reported identifying a small protein that can be targeted to promote repair of damaged tissue, with therapeutic potential. The molecule, Endothelin-1 (ET-1), is shown to inhibit repair of myelin. Myelin damage is a hallmark characteristic of MS. The study demonstrates that blocking ET-1 pharmacologically or using a genetic approach could promote myelin repair.

Repair of damaged MS plaques is carried out by endogenous oliogdendrocytle progenitor cells (OPCs) in a process called

remyelination. Current MS therapy can be effective in patients with relapsing and remitting MS, but "have little impact in promoting remyelination in tissue," Gallo said. Several studies have shown that OPCs fail to differentiate in chronic MS lesions.

Targeting ET-1 is a process that involves identifying signals in cells that could promote lesion repair. "We demonstrate that ET-1 drastically reduces the rate of remyelination," Gallo said. As such, ET-1 is "potentially a therapeutic target to promote lesion repair in deymyelinated tissue." It could play a "crucial role in preventing normal myelination in MS and in other demyelinating diseases," Gallo said.

More information:
www.cell.com/neuron/abstract/S0896-6273(13)01083-0

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