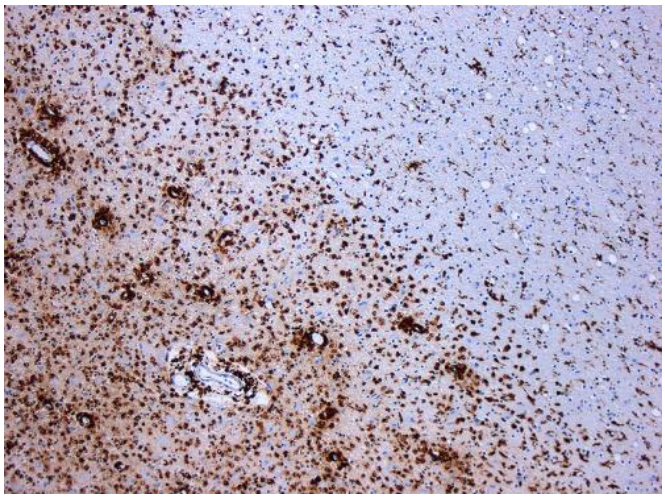
Experimental drug that may repair nerve damage in MS moves forward

14 April 2015



Demyelination by MS. The CD68 colored tissue shows several macrophages in the area of the lesion. Original scale 1:100. Credit: Marvin 101/Wikipedia

A new study suggests that an investigational drug for multiple sclerosis (MS) may repair myelin, the fatty material that protects nerves and is damaged in MS, according to a study released today that will be presented at the American Academy of Neurology's 67th Annual Meeting in Washington, DC, April 18 to 25, 2015.

"This study, for the first time, provides biological evidence of repair of damaged myelin in the human brain, and advances the field of neuro-reparative therapies," said study lead author Diego Cadavid, MD, with Biogen in Cambridge, Mass., and a fellow with the American Academy of Neurology.

The Phase 2 study involved 82 people who had their first incident of acute optic neuritis, a disease that typically affects one eye and is characterized by inflammation, damage to the nerve fibers and loss of myelin within the optic nerve. It is estimated that about half of people with optic neuritis will later develop multiple sclerosis.

All participants were treated with high dose steroids and then randomly selected with equal probability to receive either the experimental antibody, called anti-LINGO-1, or a placebo once every four weeks, for a total of six doses. Participants were then assessed every four weeks for six months and a final visit at eight months. The drug's effectiveness in repairing myelin was evaluated by comparing the recovery of the optic nerve latency in the damaged eye at six and eight months to the normal unaffected eye at the start of the study.

The main finding of the study focused on the latency of the visual evoked potential (VEP), a test that measures the visual system's ability to conduct electrical signals between the retina and the brain. The results showed that people treated with the experimental drug and who did not miss more than one dose (per protocol population) had significantly improved conduction as measured by latency recovery compared to people who received the placebo. At six months, those who received the drug improved on average by 7.55 milliseconds, or 34 percent, compared to placebo. The effect continued to eight months with an average improvement of 9.13 milliseconds or 41 percent over placebo.

In addition, the percentage of subjects whose VEP latency in the affected eye recovered to normal or nearly normal (within 10 percent of the normal eye) more than doubled, from 26 percent on placebo to 53 percent on the drug.

A substudy using an exploratory method of measuring latency called multifocal VEP revealed similar treatment effects.

"More studies are needed to evaluate whether these changes lead to clinical improvement," said Cadavid.

A second study of anti-LINGO-1 in people with multiple sclerosis is ongoing.

Provided by American Academy of Neurology