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RESEARCH/CLINICAL UPDATE

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Society-Funded Researchers Find Cells that May Promote Myelin Repair in MS

Researchers funded in part by the National MS Society's Promise:2010 campaign report on a potential source of cells in the brain to promote repair of nervous system damage that occurs in multiple sclerosis. Anne Baron-Van Evercooren PhD, and Brahim Nait-Oumesmar PhD (INSERM, Paris) and colleagues report their findings in the *Proceedings of the National Academy of Sciences* (2007 Mar 13;104[11]:4694-9). Drs. Van-Evercooren and Nait-Oumesmar are members of one of the four international, multidisciplinary teams funded through the Promise:2010 campaign's Repair and Protection Initiative to accelerate nerve tissue repair from basic research to human clinical trials.

Multiple sclerosis occurs when the immune system mistakenly attacks the myelin insulation of nerve fibers. Nerve fibers themselves also are damaged. Some spontaneous repair occurs via populations of immature stem cells resident in the brain, but this repair is not sufficient. Researchers are searching for ways to stimulate these natural repair resources in people with MS. Drs. Van-Evercooren and Nait-Oumesmar are members of the repair team headed by [Professor Charles ffrench-Constant](#) (University of Cambridge, UK), which is focusing on restoring myelin by identifying and amplifying natural repair factors in the brain and by attempting transplantation of replacement cells.

In the current study, the authors investigated an area deep in the brain that may harbor cells capable of promoting repair: the subventricular zone (SVZ). They studied brain tissue obtained from people with MS through autopsy and compared cells found in the SVZ with tissue obtained from people who did not have MS.

The researchers found evidence of two to three times as many stem cells showing molecular signs of being immature myelin-making cells in the SVZ of people with MS as in controls

without the disease. In areas (lesions) of active inflammation, the number of cells was eight times more than in areas where there was no inflammation.

These results show expanded capabilities for the SVZ in people with MS to produce cells that have potential to promote myelin repair. It remains to be seen why these cells fail to prevent disease progression, and how any obstacles might be overcome to take full advantage of the cells in therapeutic strategies for people with MS.

A full report on the progress of the Repair and Protection Initiative can be found in the Winter/Spring 2007 issue of "Research Highlights," at <http://www.nationalmssociety.org/ResearchProgress>.

-- Research and Clinical Programs Department