

# Promising therapy for relapsing multiple sclerosis

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An international team of researchers has found that adding a humanized monoclonal antibody called daclizumab to standard treatment reduces the number of new or enlarged brain lesions in patients with relapsing multiple sclerosis. This new study was published online Feb. 16, 2010, and in the March edition of the *Lancet Neurology*.

Multiple sclerosis (MS) is a debilitating disease in which the body's immune system attacks the fatty substance that surrounds and protects the [nerve fibers](#) in the brain and spinal cord. The resulting damage interferes with the transmission of [nerve signals](#) between the brain and spinal cord and other parts of the body, producing a variety of symptoms including problems with balance, coordination, vision, and even mental function. Approximately 85 percent of multiple sclerosis patients are initially diagnosed with relapsing MS, in which clearly-defined attacks of worsening neurologic function are followed by partial or complete recovery periods during which no disease progression occurs.

"Previous research has shown that treatment with daclizumab reduced multiple sclerosis disease activity," says John W. Rose, M.D., professor of neurology at the University of Utah School of Medicine, Neurovirology Research Laboratory, Veterans Affairs Salt Lake City Health Care System and the University of Utah, an author on the study. "Our work in the CHOICE trial shows that daclizumab significantly reduces MS lesion formation in people with active relapsing disease."

Monoclonal antibodies are immune system proteins that preferentially

bind to specific target cells, triggering the immune system to attack those cells. Daclizumab is a monoclonal antibody specific for CD25, a protein that is expressed on activated T cells, and binding of daclizumab to CD25 results in selective inhibition of these activated [T cells](#).

Daclizumab treatment has been studied in patients with human [autoimmune conditions](#), such as MS, that are characterized by abnormal T-cell responses.

Rose and his colleagues performed a randomized, double-blind, placebo-controlled study at 51 centers in the U.S., Canada, Germany, Italy, and Spain. They recruited 230 patients with relapsing MS who were taking interferon beta and randomly assigned them to receive add-on treatment with high-dose daclizumab, low-dose daclizumab, or placebo. The primary objective of the study was to assess whether daclizumab affected MS disease activity by measuring the total number of new or enlarged lesions in the brain during 24 weeks of treatment.

In addition to finding that add-on treatment with high-dose daclizumab resulted in a significantly lower number of new or enlarged MS lesions, the researchers found that patients treated with either high- or low-dose daclizumab had a seven to eight times higher number of immune cells called CD56bright natural killer cells (NK Cells). Previous research has shown that untreated MS patients have lower numbers of these NK cells than healthy individuals.

"Several lines of evidence point to a potential function for CD56bright natural killer cells in regulating the immune system," explains Rose.

"This study provides confirmatory data that daclizumab treatment causes an expansion of CD56bright natural killer cells and adds support to the theory that this expansion might mediate some of the effects of daclizumab on reducing multiple sclerosis lesion activity."

Further research is needed to clarify whether the risk-benefit of

daclizumab is better when the drug is used alone or in combination with interferon beta, as well as to determine the optimum dose and length of treatment needed to see the full therapeutic effects of the drug.

"The CHOICE trial showed that treatment with daclizumab was associated with both a significant reduction in MS lesion formation and a robust increase in important cells that help to regulate the immune system," concludes Rose. "Combined with previous research, these two findings strongly support further study of daclizumab as a clinical treatment for [multiple sclerosis](#)."

Provided by University of Utah Health Sciences

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