

PublisherInfo		
PublisherName	:	BioMed Central
PublisherLocation	:	London
PublisherImprintName	:	BioMed Central

Display of targets in multiple sclerosis

ArticleInfo		
ArticleID	:	4469
ArticleDOI	:	10.1186/gb-spotlight-20020507-01
ArticleCitationID	:	spotlight-20020507-01
ArticleSequenceNumber	:	135
ArticleCategory	:	Research news
ArticleFirstPage	:	1
ArticleLastPage	:	2
ArticleHistory	:	RegistrationDate : 2002-5-7 OnlineDate : 2002-5-7
ArticleCopyright	:	BioMed Central Ltd2002
ArticleGrants	:	
ArticleContext	:	130593311

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Multiple sclerosis (MS) is a central nervous system autoimmune disease but molecular mechanisms underlying the disorder remain largely unknown. In May [Nature Medicine](#), Christopher Lock and colleagues from [Stanford University](#), California, show that microarray analysis of multiple sclerosis lesions has revealed potential new therapeutic targets (*Nat Med* 2002, **8**:500-508).

Lock *et al.* compared gene expression in acute and chronic MS lesions obtained at autopsy. They observed increased transcripts of genes encoding inflammatory cytokines, particularly interleukin-6 and -17, interferon- γ and associated downstream pathways.

In addition they chose two of these gene products as targets for therapy in experimental autoimmune encephalomyelitis (EAE), a murine model with similarities to MS. They observed that knocking out granulocyte colony-stimulating factor (upregulated in acute MS lesions) ameliorated EAE in the acute phase. In contrast, knocking out the immunoglobulin Fc receptor common γ chain (upregulated in chronic MS lesions) had the greatest effect on chronic disease.

"This is the first description supporting molecular differences in the two types of lesions, and provides data that could ultimately influence the choice of treatment for acute versus chronic stages of the disease" write Stephen Tompkins and Stephen Miller of [Northwestern University Medical School](#), Chicago, Illinois, in an accompanying [News & Views](#) article.

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