

Animal Models of Multiple Sclerosis available in NDI Laboratories

Primate

Chronic, relapsing-remitting form of experimental allergic encephalomyelitis (EAE), which pathologically recapitulates the hallmark features of lesions of human MS

Rodent

EAE (Experimental Autoimmune Encephalomyelitis), both acute and relapsing/remitting versions

Primate model

The primate model is the marmoset, involving inoculation with whole human myelin. The principal endpoint is behavioral, with repeated measuring up to 3 or 4 months following disease induction, depending on the experimental design (prevention or treatment after onset of symptoms). This model is done for us by the person who developed it. There can also be imaging (MRI, for quantification of lesion volume), immunological (T cell reactivity, PBL's, splenocytes) and histopathological endpoints (inflammation and de-myelination quantification). This model provides much more information than the rodent model.

Rodent model

Our laboratory has used two rat models of MS, to reflect the two primary clinical forms of the disease, acute and relapsing/remitting. **With both models, we have pioneered a unique assay: *in vivo cell tracking of macrophage brain infiltration by Magnetic Resonance Imaging (MRI), using ultra-small superparamagnetic iron oxide (USPIO).***

What this assay allows: Early and accurate predictions of which specific animals will develop the disorder, and how severe it will be, following experimental induction of encephalomyelitis. These animals can then be used for testing the efficacy of various therapeutics. Our laboratory was the first to apply this novel approach pre-clinically in the EAE model, and now it is being conducted in the clinic.

The macrophage cell tracking approach is applied during the first clinical signs of MS. Potential therapies are then administered, and their efficacy monitored *in vivo* by MRI, which reveals the suppression of monocyte/macrophage brain infiltration. These *in vivo* observations are then followed by histopathology and immunohistochemistry, to confirm the extent of CNS inflammation (macrophages, T cells), demyelination, axonal damage and permanent axonal loss (see, for example: *Boullerne et al., Anti-S-nitrosocysteine antibodies are a predictive marker for demyelination in experimental autoimmune encephalomyelitis: implications for multiple sclerosis. J Neurosci. 2002 [22]: 123-132.*)

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