Experimental Autoimmune Encephalomyelitis Model

Experimental Autoimmune Encephalomyelitis (EAE) is a commonly-used murine model for human Multiple Sclerosis (MS). EAE shares both immunological and pathological features with MS and the MOG_{35-55}-induced EAE model is a good tool for understanding the immune-mediated mechanism of neuroinflammation and demyelination associated with MS. Invivotek offers two well-characterized **MOG-induced EAE** models that are **pharmacologically-validated** and can be used in the development of a variety of MS therapeutics and as a mechanistic model for evaluating T cell function.

I. MOG-induced EAE model in C57BL/6 mice
II. MOG-induced EAE model in GFAP-luc® Tg/F1:FVBxC57BL/6 Albino

MOG-induced EAE model in C57BL/6 mice:

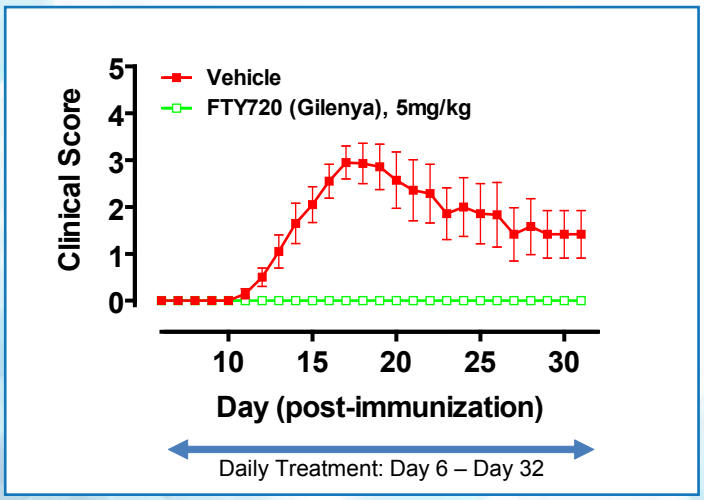
Characteristics of the Model:

- Perivascular infiltration of $CD4^+$ T- and mononuclear cells, followed by primary demyelination of axonal tracts in the central nervous system (CNS), leads to progressive hind-limb paralysis.
- Severity of the disease correlates with the levels of infiltrating $CD4^+$ T cells and macrophages and cytokines in the CNS.

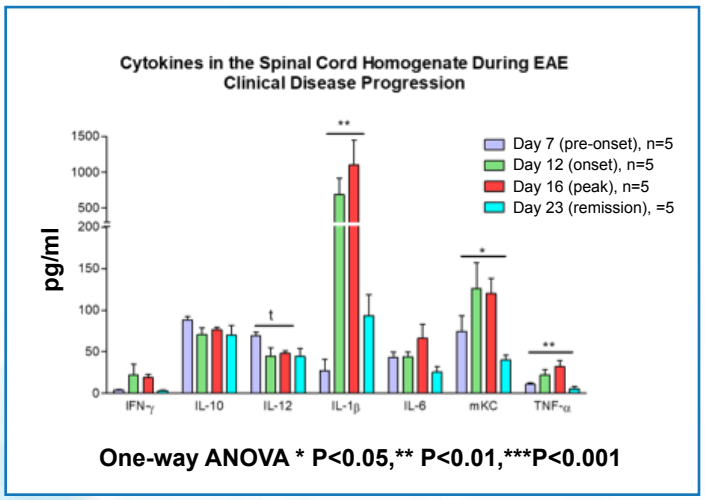
Model Assessment Modules:

- Clinical Score
- FACS analysis of cellular infiltration to the spinal cord
- Cytokine levels in the spinal cord, spleen and blood by the MesoScale Discovery platform®
- *In vitro* MOG-specific T cell response of primary splenocytes derived from mice with EAE

Pharmacological Validation:

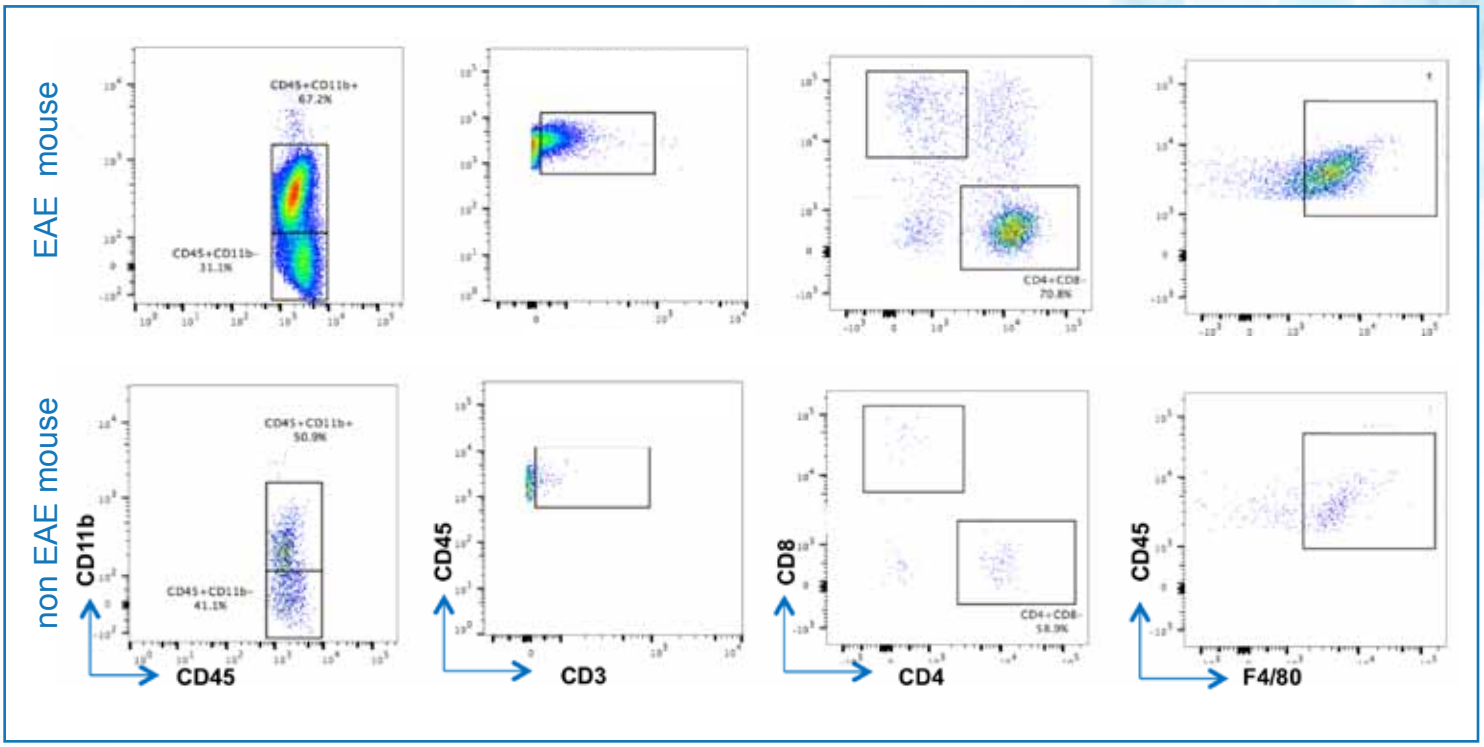


Cytokine Analysis in the Spinal Cord:



Cytokine analysis done on Day 7 (pre-onset), Day 12 (onset), Day 16 (peak) and Day 23 (remission)

FACS Analysis of Infiltrating Lymphocytes in the Spinal Cord:



MOG-induced EAE model in GFAP-luc® Tg/F1:FVBxC57BL/6 Albino mice

This model has the same characteristics as the classic MOG-induced EAE in C57BL/6 mice and allows application of the same model assessment modules. In addition, in the GFAP-luc® mice MOG-induced neuroinflammation can be monitored by biophotonic imaging. Neuroinflammation results in rapid activation of astrocytes and increased expression of luciferase driven by the glial fibrillary acidic protein (GFAP) promoter. The GFAP promoter-driven Luciferase expression activation precedes clinical manifestations of EAE by several days and bioluminescence can be used as an early read-out to evaluate effects of therapeutic interventions prior to the disease onset.

Visualizing neuroinflammtion in the brain and spinal cord in MOG_{35-55} induced EAE using the GFAP-Luc Tg/F1:FVBxC57BL/6 Albino Mice by IVIS Spectrum: