Abstract

The chemokine CXCL13 is expressed in monocyte/macrophage organs to induce immune cell recruitment and migration. Recent studies on arthritis and collagen-induced arthritis have shown that blocking CXCL13 with neutralizing antibodies reduced disease progression. We hypothesized that blocking CXCL13 in an adoptive transfer model of EAE would reduce clinical signs of disease.

Anti-CXCL13 Antibodies Inhibit Migration of Human pre-B and Tonsillar Cells and Murine Splenocytes

VX5378 Reduces Severity of Passive Th17-Induced EAE in SJL/J Mice

Generation of Anti-Human CXCL13 MAB

Antibody Specificity for CXCL13

VX5378 Mouse IgG1

Serum MPO

3D2 (Mouse IgG1)

VX5378 reduces inflammation and monocyte infiltration in vivo

Anti-CXCL13 Antibodies Inhibit Migration of Human pre-B and Tonsillar Cells and Murine Splenocytes

VX5378 Reduces Severity of Passive Th17-Induced EAE in SJL/J Mice

Confirmation of Anti-Inflammatory Effect: VX5378-IgG1 Reduces Severity of Collegen-Induced Arthritis

Summary

We have generated a mouse multiplex specific human vaccine and have demonstrated efficacy in Experimental Autoimmune Encephalomyelitis, a mouse model of Multiple Sclerosis.

VX53301, a humanized antibody derived from D2, shared epitopes and specificity and demonstrated significantly improved myelination and remyelination in chronic EAE in a mouse model.

VX53301 has been shown to be safe and well tolerated in preclinical studies, and has been advanced to clinical development for the treatment of Multiple Sclerosis.

VX53301 will undergo further development in preparation for an IND submission.