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## Poster Presentation

### KCNK2 and Adhesion Molecules in an in-Vitro Blood Brain Barrier Model

Isabelle Plegge, Stefanie Bock, Tobias Ruck, Sven G. Meuth

Department for Neurology with Institute for Translational Neurology, University of Münster, 48149 Münster, Germany

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#### Abstract

Two-pore domain potassium channels, like KCNK2, are known to play an important role in inflammatory diseases such as multiple sclerosis (MS). Upregulation of cellular adhesion molecules in mouse brain microvascular endothelial cells (MBMECs) of *Kcnk2*<sup>-/-</sup> mice resulted in elevated leukocyte trafficking into the central nervous system under inflammatory conditions. The current project aims to gain deeper insights into the role of KCNK2 in the regulation of adhesion molecules and cell trafficking at the blood-brain-barrier (BBB). Therefore, we used a dynamic in vitro model of the BBB to investigate brain endothelial cell – T cell interactions under physiological and pathophysiological conditions. MBMECs from either wild type mice or *Kcnk2*<sup>-/-</sup> mice were seeded into flow chambers and T cell migration behavior was investigated under mild shear stress (0.25 dyn/cm<sup>2</sup>). Experiments showed so far increased T cell migration under inflammatory conditions and decreased migration while blocking cellular adhesion molecule ICAM1 on wild type MBMECs. In future experiments, we will use static transwell assays to assess how different subgroups of T cells are influenced by pharmacological KCNK2 modulation. Overall, our project might identify new therapeutic strategies to influence immune cell trafficking at the BBB.

**Keywords:** Blood Brain Barrier, Adhesion, Immune Cell

**\*Corresponding Author:**

**Email:** \_