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## Oral Presentation

# Kininogen Deficiency Ameliorates Neuroinflammation by Reducing Immune Cell Trafficking

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***Abstract***

Enhanced immune cell trafficking into the central nervous system (CNS) and disruption of the blood brain barrier are pathophysiological hallmarks of neuroinflammatory disorders like multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE). However, recent studies suggest that the coagulation and the contact-kinin system might also be involved in MS development. For instance, it was shown that the coagulation factor XII modulates immune cell function and bradykinin influences the integrity of the blood-brain barrier (BBB). High molecular weight kininogen (HMWK) is a central constituent of the contact-kinin system. Here, we identify HMWK as a critical player in neuroinflammation. Deficiency of HMWK renders mice less susceptible to EAE and was accompanied by decreased numbers of infiltrated lymphocytes into the CNS, whereas the distribution of immune cells was unaltered as determined by flow cytometry analysis. Preliminary in vitro migration experiments showed that HMWK leads to an enhanced immune cell trafficking through an endothelial cell layer. Altogether, our study indicates that HMWK inhibition reduces cell invasion during autoimmune CNS disease and may offer a novel strategy to combat MS.

***Keywords:*** Central Nervous System, Mice, Animal Model

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