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## Poster Presentation

### The Role of Platelet Granules in Neuroinflammation

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#### Abstract

Platelets are known to contribute to vascular pathologies, however, their role in inflammatory disorders of the central nervous system (CNS), such as multiple sclerosis (MS) and its mouse model, experimental autoimmune encephalomyelitis (EAE), is thus far poorly defined. Although there is emerging evidence that platelets might accumulate in the CNS parenchyma along with an increased activation status and secrete proinflammatory factors thereby triggering immune response cascades during neuroinflammation, the role of platelet granules remains elusive so far. We investigate here the contribution of platelet granules to immune response during neuroinflammation. Therefore, we performed experiments using Munc13-4-deficient mice since mutation in Munc13-4 leads to abolished platelet dense granule secretion and compromised  $\alpha$ -granule release. We found that genetic deficiency of platelet granules renders mice less susceptible to EAE. This reduction in disease severity was accompanied by reduced numbers of interleukin (IL)-17A- and interferon (IFN)- $\gamma$ -producing proinflammatory effector T-helper cells as indicated by decreased cytokine levels of IL-17A and IFN- $\gamma$  compared to control mice. Taken together, our findings show that genetic inhibition of platelet granule release significantly reduces CNS inflammation in mice, potentially indicating a novel therapeutic strategy for the treatment of MS. To understand the underlying mechanisms, further investigations are required.

**Keyword:** Platelet Granules, Neuroinflammation, Central Nervous System

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