Novel Approaches to Prevent Neuroinflammation by Targeting the Coagulation System

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Abstract

There is growing appreciation that other factors not traditionally considered components of the immune system foster inflammation in multiple sclerosis (MS) and its animal model, experimental autoimmune encephalomyelitis (EAE). The blood coagulation initiating factor XII was introduced as key mediator of central nervous system (CNS) autoimmunity by modulation of adaptive immune response. Moreover, these findings were indicated to be relevant in MS patients, as further demonstrated: levels of factor X (FX) and prothrombin are increased in treatment-naïve MS patients compared to healthy donors. This concept of a pivotal role of the coagulation system in neuroinflammation was further reinforced by our recent identification of the preventive effect of FX inhibition in EAE on neurological deficits and local inflammation compared to control animals. Platelets have been also recognized to contribute to EAE. Thus, the identification of platelet specific candidates relevant for disease activity is crucial for the understanding of platelet mediated contributions to CNS inflammation currently being addressed by ongoing studies. As the pathophysiology of MS remains poorly understood to date, the addition of novel mechanisms of neuroinflammation can broaden our knowledge about disease development and may even help in establishing novel therapeutic approaches targeting the coagulation system.

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