KV 7/M channels as targets for lipopolysaccharide-induced inflammatory neuronal hyperexcitability.

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Abstract:

Acute brain insults and many chronic brain diseases manifest an innate inflammatory response. The hallmark of this response is glia activation, which promotes repair of damaged tissue, but also induces structural and functional changes that may lead to an increase in neuronal excitability. We have investigated the mechanisms involved in the modulation of neuronal activity by acute inflammation. Initiating inflammatory responses in hippocampal tissue rapidly led to neuronal depolarization and repetitive firing even in absence of active synaptic transmission. This action was mediated by a complex metabotropic purinergic and glutamatergic glia-to-neuron signalling cascade, leading to the blockade of neuronal KV 7/M channels by Ca(2+) released from internal stores. These channels generate the low voltage-activating, noninactivating M-type K(+) current (M-current) that controls intrinsic neuronal excitability, and its inhibition was the predominant cause of the inflammation-induced hyperexcitability. Our discovery that the ubiquitous KV 7/M channels are the downstream target of the inflammation-induced cascade, has far reaching implications for the understanding and treatment of many acute and chronic brain disorders. This article is protected by copyright. All rights reserved.

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