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**Title** Functionality of Cav1.2 and Cav2.1 calcium channels in astrocytes: implication in astrogliosis.

**Text** Voltage-gated calcium channels (VGCCs) are upregulated in reactive astrocytes in several CNS pathologies. In a previous work we showed that protein kinase C epsilon (PKCepsilon), which induces in astrocytes a stellated morphology according to a reactive pattern, also stimulates expression of specific VGCCs. Here we investigated whether the calcium channels formed in astrocytes as a consequence of PKCepsilon activation are functional. In addition, VGCC involvement in the morphological transformation induced by PKCepsilon was also explored. Patch-clamp experiments demonstrated that astrocytes infected with an adenovirus that expresses the active form of PKCepsilon display an increased number of VGCCs, as well as a prominent enhancement in total calcium current density. Moreover, PKCepsilon boosted the contribution of Cav2.1 calcium channels to the whole cell current. In another set of experiments, the implication of VGCCs in PKCepsilon-triggered morphological effects was tested by using nifedipine. This Cav1.2 inhibitor significantly reduced the astrogliosis induced by overexpression of PKCepsilon. In conclusion, we propose that astrocyte activation mediated by PKCepsilon involves increased expression of functional Cav1.2 and Cav2.1 calcium channel subunits, which contributes to the development of a stellated morphology. To clarify the link between these signalling events and neuroinflammation will require further work.

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**Theme** B - Neural excitability, synapses and glia: cellular mechanisms  
Ion channels - Calcium channels