Altered dendritic development of cerebellar Purkinje cells in slice cultures from protein kinase Cγ-deficient mice

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Protein kinase C (PKC) is a key molecule for the expression of long-term depression at the parallel fiber-Purkinje cell synapse in the cerebellum, a well known model for synaptic plasticity. We have recently shown that activity of PKC also profoundly affects the dendritic morphology of Purkinje cells in rat cerebellar slice cultures suggesting that synaptic efficacy and dendritic development may be controlled by similar intracellular signalling pathways. Here we have analyzed the role of the gamma-isoform of protein kinase C (PKCgamma), which is strongly and specifically expressed in Purkinje cells, during dendritic development. After pharmacological treatment with PKC modulators, phosphorylation of PKCgamma at serine 660 was altered in cerebellar slices suggesting that a change of PKCgamma activity by these treatments was taking place within the Purkinje cells. In PKCgamma-deficient mice, Purkinje cell dendritic trees were enlarged and had an increased number of branching points compared to wild-type mice indicating a role for the PKCgamma isoform as a negative regulator of dendritic growth and branching. Furthermore, the branching-stimulating effects of the PKC inhibitors 2-[1-(3-dimethylaminopropyl)indol-3-yl]-3-(indol-3-yl)maleimide and Gö6976 found in wild-type cultures were abolished in the absence of PKCgamma. In contrast, the strong inhibitory effect on dendritic growth by the PKC activator phorbol-12-myristate-13-acetate (PMA) did not require the presence of the PKCgamma isoform since it was still present in the cultures of PKCgamma-deficient mice. Our results clearly demonstrate an involvement of PKCgamma in Purkinje cell dendritic differentiation in cerebellar slice cultures.

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