Abstract

Regional activities and levels of protein kinase C were measured after lateral fluid percussion brain injury in rats. At 5 min and 20 min after injury, neither cofactor-dependent nor -independent PKC activities in the cytosol and membrane fractions changed in the injured and contralateral cortices or in the ipsilateral hippocampus. Western blot analysis revealed decreases in the levels of cytosolic PKCα and PKCβ in the injured cortex after brain injury. In the same site, a significant increase in the levels of membrane PKCα and PKCβ was observed after injury. Although the level of PKCα did not change and that of PKCβ decreased in the cytosol of the ipsilateral hippocampus, these levels did not increase in the membrane fraction after injury. The levels of PKCγ were generally unchanged in the cytosol and the membrane, except for its decrease in the cytosol of the hippocampus. There were no changes in the levels of any PKC isoform in either the cytosol or the membrane of the contralateral cortex after injury. The present results suggest a translocation of PKCα and PKCβ from the cytosol to the membrane in the injured cortex after brain injury. The observation that such a translocation occurs only in the brain regions that undergo substantial neuronal loss suggests that membrane PKC may play a role in neuronal damage after brain injury.

Keywords: Brain injury; Fluid percussion; Protein kinase C; Diacylglycerol