Abstract

Adaptation to chronic hypoxia confers long-term cardioprotective effects. We have shown, that protein kinase C (PKC) is involved in this cardioprotective phenotype. It has not been elucidated, which PKC isoform plays a role, but the most likely candidates are PKC ε a PKC δ. Therefore, the aim of this study was to analyze the expression and localization of PKC ε and PKC δ after the adaptation to chronic hypoxia. We have shown that adaptation to chronic hypoxia caused the activation and tranlocation of PKC δ to the mitochondrial and sarcolemmal membranes. Our study suggests that PKC δ plays an important role in the mechanism of cardioprotection induced by chronic hypoxia.