REGULATION OF CELL PROLIFERATION BY PROTEIN KINASE C-ETA IN BREAST CANCER CELLS

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Cancer can develop due to an increase in cell proliferation or a decrease in cell death. Cellular pathways are deregulated in cancer, which allows for the proliferation of cells. The Protein Kinase C (PKC) family of proteins regulates cell proliferation, differentiation, and cell death. There are three subgroups of PKCs: conventional, novel, and atypical. PKCη is a member of the novel PKC family, but its regulation is distinct from other novel members of the PKC family. It is the only PKC isoform that is upregulated by tumor-promoting phorbol esters. The purpose of the present study was to determine how PKCη regulates cell proliferation in breast cancer cells. MCF-7 breast cancer cells were used in the study. The levels of PKCη and other proteins were determined by western blot analysis. Cell survival was monitored by clonogenic assay. PKCη knockdown decreased clonogenic cell survival of MCF-7 breast cancer cells.
The overexpression of PKCη caused an increase in clonogenic cell survival of MCF-7 breast cancer cells. The level of phosphorylated extracellular signal-regulated kinase (P-ERK), but not total ERK, was increased by PKCη overexpression. Thus, our study suggests that PKCη regulates cell proliferation through ERK.

Keywords: Protein kinase c-eta, breast cancer, cell survival, extracellular signal-regulated kinase