Z303 Activation of 70S6K and Akt by Heat Shock is Related with Growth Arrest and Blocking of Apoptosis

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Phosphatidylinositol 3-kinase (PI 3-kinase) is involved in a variety of cellular responses including cytoskeletal organization, cell survival, cellular stress and proliferation. Akt, a target molecule of PI 3-kinase, provides a survival signal that protects from apoptosis induced by various stresses. In the present study, treatment of heat shock (5-45 min at 45℃) induced activation of p70S6K and ERK as well as Akt and growth arrest. However, heat shock for 60 min did not changed activity of those molecules and induced apoptosis assessed by FACS analysis. When cells were pretreated with wortmanin, a potent inhibitor of PI3-kinase, activities of p70S6K and Akt stimulated by heat shock for 5-45 min were down-regulated, and apoptosis was induced. These results suggest that PI 3-kinase signal pathway involve in cell protection against apoptosis induced by heat shock for 1 hr.

Z304 Induction of Apoptosis by Vitamin E-succinate in HL-60 Cells Occurs through PKC Activation

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Vitamin E-succinate (VES) induced apoptosis of HL-60 human leukemia cells by 95% at 72 hr. VES increased degradation of apoptosis-related enzymes including caspase 3, poly(ADP-ribose) polymerase, and lamin B, and expression of p21 protein. To investigate the mechanism of induction of apoptosis by VES, the activation of PKC was examined. During the induction of apoptosis, translocation of PKC βII to the cell membrane was increased in VES-treated HL-60 cells, which was blocked by co-treatment of PKC inhibitor, GF-109203. The down-regulation of PKC by GF-109203 induced cell growth arrest at G2M phase of the cell cycle and inhibited VES-induced apoptosis. Taken together, these results suggest that VES-induced apoptosis is dependent on the activation of PKC βII in HL-60 cells.