D127 Activation of p70S6K and ERK as well as Akt by Heat Shock and Hyperosmorality Stress is Phosphatidylinositol 3-kinase Dependent.

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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 present study, p70S6K and ERK as well as Akt were activated by cellular stress such as heat shock and hyperosmorality. Wortmanin, which is known as potent inhibtior of PI 3-kinase and normally inhibits growth factor induced activation of Akt, suppressed heat-shock and hyperosmorality induced activation of p70S6K and ERK as well as Akt. The results suggest that PI 3-kinase signal pathway involve in early events of the cellular response to stress.

D128 Effects of cytochalasin D on cell shape in cultured chick mesenchymal cells

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The altered morphology, disruption of actin filaments in cultured mesenchymal cells treated with cytochalasin D (CD) has been investigated. Treatment of CD (2 μM for 24h) to mesenchymal cells inhibited the chondrogenesis as determined by Alcian blue staining. Ultrastructurally, cells in control cultures showed the typical features of chondrocyte, but CD-treated cells became elongated and drastically altered cellular morphology like filament-rich fibroblast-like cells. Smooth muscle alpha-actin (α-SMA) is a functional marker for a myofibroblast. Both control and CD-treated cells expressed significant amount of α-SMA through the culture, whereas CD-treated cells overexpressed α-SMA on culture 1 day. The activation of some protein kinase C (PKC) isoforms regulates chondrogenesis of mesenchyme. The CD treatment resulted in the decreased PKCα protein expression. Increased β_1 integrin expression was observed in CD-treated cells but CD had no effect on the expression of α_5 integrin, talin, α-actinin, vimentin, paxillin, N-cadherin, P-cadherin throughout culture periods. An overexpression of α-SMA in CD-treated cells may be related to phenotypical changes and differential expression of β_1 integrin and PKCα may be related to inhibition of chondrogenic differentiation.