IGF-1 activates polyphosphoinositide hydrolysis, protein kinase C isoforms and ERK pathway in cultured neonatal rat cardiac myocytes

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Because IGF- lisa natural cardioprotective which migh; improve cardiac function and stimulates growth and proliferation of carfiac myocytes, there is considerable interest to elucidate the molecular f!iechanisms by which IGF-1 exerts these effects on cardiac myocytes We show here that IGF-1 stimulated polyphosphoinositide turnover (mrw at 30s, 65%) and a rapid translocation of PKC isoforms (a, E srd 8) from the soluble lo tiic paniculate fraction IGF-1 also increased both phospholipid- dependent and Ca2 phospholipid- dependent PKC activities (max. a 2-fold increase at 5 and 15 min for paniculate and soluble fractions, respectively). IGF-1 promoted translocation of ERK to the nucleus, associated with an activation and tyrosine phosphorylaticn of ERK (max at 5 min, 40% of ERK phosphorylated) Prolonged phorbol ester exposure of cells down-regulated subsequent activation of ERKs by IOF-1, suggesting a role of PKC isoforms in this ERK activation. IGF-1 stimulated protein synthesis rate and