Z501 Change of ECM and Integrins at different developmental stages of heart and phenylephrine-induced cardiac myocyte hypertrophy

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Cardiac hypertrophy is characterized by both remodeling of the extracellular matrix (ECM) and hypertrophic growth of the cardiocytes and leads to increasing myocardial stiffness, ventricular dysfunction and heart failure. A variety of agonists including phenylephrine (PE) induce hypertrophy in neonatal cardiomyocytes. To better understand the possible role of integrin in cardiac myocytes, we investigate the expression of the ECM and integrin isoforms at different developmental stages of heart as well as during PE-induced hypertrophy in cultured rat cardiac myocytes. Addition of PE to cultured neonatal cardiomyocytes stimulated sarcomeric organization, increased cell size, and induced atrial natriuretic factor as hypertrophic marker. In PE-induced cardiac hypertrophy, we investigated the change of cellular localization of ECM (fibronectin, laminin and collagen) and integrin (alpha1, alpha5, alpha7, alpha3A and beta1A) into sarcomeric banding pattern and a dramatic change of the expression level in integrin alpha1, alpha5, alpha7, alpha3A, beta1A, laminin, fibronectin and collagen. These data indicate that the signal transduction pathways leading to cardiomyocyte hypertrophy are strongly influenced by and/or dependent upon integrin-mediated singaling process.

Z502 The contribution of PKA pathway activated by Aplysia octopamine receptor becomes less effective on synaptic facilitation as the synapses becomes depressed

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To evaluate the contribution of cAMP/PKA signal pathway in short-term facilitation, we overexpressed Ap oa1 receptor in sensory neurons that do not normally express this receptor. We have previously showed that activation of this receptor in sensory cells, by a brief treatment with octopamine (OA), produced short-term facilitation such as membrane depolarization and increase in both membrane excitability and spike broadening and enhanced neurotransmitter release in non-depressed synapse. To assess contribution of PKA pathway on the different degrees of synaptic depression, we examined the ability of octopamine to facilitate depressed synapse in sensory cells expressing Ap oa1. When synaptic connections were moderately depressed to 30-40% of initial EPSP amplitude (ISI=1min) in the pleural-pedal ganglia, the application of OA to the sensory cells expressing Ap oa1 showed a moderate synaptic facilitation compared with that achieved by treatment with 10 uM 5-HT. In the case of highly depressed synapse (up to 10-30% of initial EPSP amplitude, ISI= 1min or ISI= 20sec), PKA pathway activated by Ap oa1 had less effect on synaptic facilitation than that of moderately depressed synapse in the pleural-pedal ganglia. These data are consistent with the previous observations that the role of PKA in synaptic facilitation becomes less effective as the sensori-motor synapse becomes depressed with repeated activity.