E107 Requirement of cAMP-mediated signal pathway at the early stage of chondrogenesis

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The cAMP-mediated signalling pathway is one of the major intracellular signal transduction pathways in cells. It has been suggested that cAMP signalling pathway is involved in chondrogensis of chick limb bud mesenchymal cells. Forskolin, an activator of adenylate cyclase, showed a stimulatory effect on chondrogenesis in a dose-dependent manner, as indicated by Alcian blue staining. Treatment of cells with specific inhibitors of protein kinase A(PKA), H89 or KT5720, blocked chondrogenesis. Thus, chondrogenic differentiation appears to be regulated by the increase in cellular cAMP level and activation of PKA. cAMP stimulates the expression of numerous genes through PKA-mediated phosphorylation of the nuclear factor, cAMP response element (CRE) binding protein (CREB). In chick chondroblast, CREB was expressed constituently throughout culture period, but it was highly phosphorylated immediately after seeding followed by progressive dephosphorylation. Electrophoretic mobility shift assays using consensus CRE sequence revealed one major complex bound with CREB as confirmed by anti-CREB supershift assay. Binding activity of CREB to CRE site was maximum within 4 hr after plating cells which is consistent with the level of phosphorylated CREB. Activating transcription factor(ATF), another CRE binding family, and CRE-binding protein (CBP) were also expressed at the early stage of chondrogenesis. Therefore, cAMP signal pathway to nuclear events for inducing genes apperard to be required at the early stage of chick limb bud chondrogenesis.

E108 Comparisons of Interleukin-6 and Lipopolysaccharide in Long-Term Potentiation in Rat Hippocampal Slices.

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A cascade of cytokines appears to be involved in the complex results of brain trauma, infection and inflammation. In this study, the effects of cytokine, IL6 and bacterial endotoxin, lipopolysaccharide(LPS) were studied on long-term potentiation(LTP) in the CA1 area of rat hippocampal slices, which is presumed to be the cellular mechanisms underlying learning and memory. Field excitatory postsynaptic potentials(fEPSP) were recorded extracellularly in the dendritic region of the CA1 area in response to electrical stimulation of Schaffer collaterals. Low frequency synaptic transmission was unaffected by IL6(1000 U/mL), but pretreatment with IL6 for 20 mins reduced the amplitude of LTP by 50 %. When perfused with IL6 after the induction of LTP, established LTP was not suppressed. Meanwhile, neural response to low frequency electrical stimulation (1 every 30 secs) was transiently reduced by the perfusion of 10 ug/mL LPS for 5 mins, which recovered after washout. To test the effects of LPS on LTP, slices were incubated in LPS-artificial cerebrospinal fluid for 1 hr before transferred to recording chamber. In these slices, LTP was not induced at all. Based on these results, it is our view that LPS exerts more extensive and direct influences on synaptic transmission and plasticity than IL6.