E101 Localization of PKC isoforms during chondrogenic differentiation in vitro

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HH-stage 23/24 chick limb bud mesenchymal cells were micromass cultured, and temporal and spatial distribution of PKC isoforms were analyzed by imnunohistochemistry. Expression of all the PKC isoforms including PKC α, γ, ε, ι, and λ was very weak in 1 and 2 day of culture and became obvious in 3 day of culture and increased as cultures proceed. PKC α and γ were localized in distinct areas of culture, especially on condensation area. On the other hand, distribution of PKC ι and λ was blurred all over the culture while distribution of PKC ε was very weak. Exposure of cells to PMA considerably decreased the expression of PKC α, γ, and ε and the distribution pattern of these proteins was quite different from that of control culture. PMA also decreased the expression of PKC ι and λ but less compared to that of PKC α, γ and ε. Lysophosphatidylcholine, a promoter of chondrogenesis and an activator of PKC, increased the expression of all the PKC isoforms. These results suggest that localization of PKC α and γ is required for cellular condensation which turns over chondrogenic differentiaiton.

E102 **Induction of apoptosis by H-7 in the prostate cancer cell lines**

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We investigated whether prostate cancer cell are able to undergo apoptosis by treatment with the protein kinase inhibitor H-7. Involvement of the PKC signaling pathway in prostate cancer cell was investigated by inhibitor(H-7 treatment) or activation(TPA; 12-o-tetradecanoyl-phorbol-13-acetate treatment) of PKC and characterization of PKC isozymes by western blot. We determined that cultured prostate cancer cell undergo apoptosis, in a dose-related manner, when treated with H-7. TPA confers partial and transient resistance to H-7 -induced apoptosis. The differential responses to individual inhibitor and activators of PKC may be related to the multiple PKC isozymes.