D121 The Effect of KCI-Induced Depolarization on Neurite Outgrowth and Neuronal Survival in the Cultured *Drosophila* CNS Neuron

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The effects of high K^+ -induced depolarization on the development and differentiation of neural cells were investigated in the CNS primary culture prepared from third instar larvae of *Drosophila*. High K^+ resulted in the reduction of neuronal survivability and inhibition of neurite outgrowth. Since depolarization induced by K^+ ion opens the voltage-sensitive Ca^{2+} channels, possibility of Ca^{2+} involvement in the process was also examined by using EGTA. The inhibitory effects of depolarization on neuronal survival and neurite outgrowth were at least, partially recovered by incubation with EGTA. Furthermore, depolarization inhibited neuronal cell adhesion and the formation of nerve growth cones. These inhibitory effects were also recovered by the blockage of Ca^{2+} influx. The results of our study strongly suggests that development and differentiation of the CNS neurons in *Drosophila* are influenced by depolarization and these effects are suggested to be mediated by the variation of intracellular Ca^{2+} concentration.

D122 Effect of PMA-Suppressed Membrane Ruffling in Chondrogenesis of Chick Fore Limb Bud Mesenchymal Cells.

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We studied the effect of membrane ruffling and inhibition of protein kinase C(PKC) in chick mesenchymal cells by flow cytometry and Western blot analysis. Chondrogenesis in chick fore limb bud mesenchymes requires the membrane ruffling and activation of $PKC\alpha$. $PKC\alpha$ down-regulated by long-term exposure to PMA produced complete inhibition of membrane ruffling, modification of cell cycle and reorganization of the actin cytoskeleton. Membrane ruffling is required for cell cycle arrest in the G1 and G2/M phase. However, PMA-suppressed membrane ruffling induced G1 arrest but failed to arrest cells at G2/M phase. Moreover, cyclin B may be involved the transition from G2 to M phase. In addition, suppressed membrane ruffling induced altered cell shape accompanying increased α -smooth muscle actin and P-cadherin. Taken together, we conclude that PMA-suppressed membrane ruffling is mediated by sustained $PKC\alpha$ and PMA-suppressed membrane ruffling is mediated by sustained $PKC\alpha$ and PMA-suppressed membrane ruffling in inhibition of chondrogenesis.