E125

Opposite Effect of Ca^{2+} and PKC on the Expression of Inwardly Rectifying K^+ Channel in Mouse Skeletal Muscle

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We report here that intracellular calcium and PKC exert opposite effect in muscle activity-dependent regulation of inwardly rectifying K^+ channel (IRK) expression. The amount of IRK mRNA was found to decrease upon denervation. Since nerve-induced muscle activity results in contraction, it was questioned whether the changes in IRK expression might be relevant to the increased intracellular calcium that functions as a cytoplasmic messenger in excitation-contraction coupling (ECC). Activation of either L-type calcium channels or ryanodine receptors, the major components of ECC, increased the expression of IRK. Ionomycin activated the IRK expression in time- and dose-dependent manners, which was abolished by treatment with EGTA. In contrast, 12-*O*-tetradecanoyl phorbol-13-acetate (TPA), an activator of PKC, markedly decreased the level of IRK mRNA, which requires protein synthesis. In addition, blockade of PKC further potentiated the expression of IRK. Therefore, the overall expression level of IRK is likely to be determined by the balance between a calcium-involved up-regulation mechanism and a PKC-involved down-regulation mechanism.

E126

Expression of Stress Proteins in *Drosophila* Kc Cells Exposed to Amino Acid Analogs

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Amino acid analogs, like other effectors of the stress response, induce the synthsis of stress proteins in mammalian cells. In this study, we examined the expression of stress proteins in Kc cells exposed to a proline analog, L-azetidine-2-carboxylic acid(AzC). Exposure to AzC resulted in the increased synthesis of the putative stress proteins, while the synthsis of normal proteins was inhibited by AzC in Kc cells. Especially, the mount of 70kD protein was corelated with the concentration of AzC and the exposure duration to AzC. The higher the concenturation of AzC was and the longer the exposure to AzC was, the larger mount of the 70kD protein was synthesized. The pretreatment and coincubation of cyclohexamide prior to or with AzC treatment inhibited the induction of the 70kD protein showing that incorporation of AzC into proteins was required for the induction of stress proteins. Induced 70kD protein was cross-reacted with antibody against HSP70 by western blot analysis. To investigate the regulation level of stress response by AzC, mRNAs was prepared from control and treated Kc cells, species and amount of mRNAs encoding stress proteins were identified by *in vitro* translation. The result indicates that induction of stress proteins by AzC is regulated mainly at the level of transcription.