## F. 생리. 생화학 및 생물리

F101

Neurotoxin 6-Aminonicotinamide(6-AN) Induces the Casein Kinase Activities in L6 Skeletal Muscle Cell Minyoung Jang<sup>P</sup>, In Kook Park<sup>C</sup>

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Effects of nerutotoxin 6-aminonicotinamide(6-AN) on activities of casein kinase and other enzymes in L6 skeletal musclel cell were investtigated. 6-AN increased activities of casein kinase, catalase and glutathione peroxidase but decreased activities of ATPase and glyceraldehyde-3-phosphate dehydrogenase. The induction of casein kinase was further confirmed by the immunoblotting assay using casein kinase antibody. The analysis of SDS-PAGE revealed the molecular mass of casein kinase to be 43 kDa. In addition, RT-RCR analysis demonstrated that casein kinase mRNA appeared to the induced by 6-AN treatment compared to the control group and its molecular size was 650 bp. N-terminal sequence of casein kinase wasd determined to be N-Pro-Phe-Ser-Asn-Thr-His-Asn-His-Lys-Leu-Lys-Ser-Pro-Glu-Glu-Glu-Glu-Phe-Pro-C. 6-AN also caused profound morphological changes which were akin to the apoptosis. It is concluded that neurotoxin 6-aminonicotinamide may specifically trigger the overexpression of casein kinase gene which may be associated with the maintenance of cell shape.

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Hepatoprotective Effects of Onion Extract on Acetaminophen-induced Liver Damage in Mice Deok Song Kim<sup>P</sup>, Eun Sun Seo<sup>1</sup>, Jin Heo<sup>1</sup>, Kyung-Jin Lee<sup>1</sup>, Myung Suk Na<sup>2</sup>, Jong Bin Lee<sup>C</sup>

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The protective effects of onion extract (OE), on acetaminophen-(APAP)-induced hepatotoxicities and the possible mechanisms involved in this protection were investigated in mice. Pretreatment with OE prior to the administration of APAP significantly prevented the increase in serum alanine and aspartate aminotransferase activities and hepatic lipid peroxidation in a dose-dependent manner. In addition, pretreatment with OE significantly prevented the depletion of reduced glutathione content in the liver of APAP-intoxicated mice. APAP-induced hepatotoxicity was also prevented, as indicated by a liver histopathologic study. The effects of OE on the cytochrome P450 (P450) 2E1, the major isozyme involved in APAP bioactivation were investigated. Treatment of mice with OE resulted in a significant decrease of P450 2E1-dependent p-nitrophenol and aniline hydroxylation in a dose-dependent manner. Consistent with these observations, the P450 2E1 expressions were also decreased, as determined by immunoblot analysis. These results show that the protective effects of OE against the APAP-induced hepatotoxicity may be due to its ability to block bioactivation of APAP mainly by the inhibition of expression and activities of P450 2E1.

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8-Cl-cAMP and its Metabolite, 8-Cl-adenosine Induce Growth Inhibition through the Same Pathways; Protein Kinase C Activation and Cyclin B Down-regulation Young-Ho Ahn<sup>P</sup>, Joong Mok Jung<sup>1</sup>, Seung Hwan Hong<sup>C</sup>

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Among the cAMP analogues, 8-Cl-cAMP is known to be most effective in growth inhibition and differentiation of a number of cancer cells. And its cellular metabolite, 8-Cl-adenosine also induces growth inhibition in lots of cell lines. In this study, it was found that an adenosine kinase inhibitor (A-134974) and adenosine deaminase could reverse growth inhibition induced by 8-Cl-cAMP and 8-Cl-adenosine. 8-Cl-cAMP could not inhibit cell growth in the presence of IBMX, a PDE inhibitor, but 8-Cl-adenosine could. These results suggest that it is not 8-Cl-AMP but 8-Cl-adenosine which induces growth inhibition, and 8-Cl-cAMP must be metabolized to inhibit cell growth. Also, we found that a PKC inhibitor, GF-109203x could restore this growth inhibition, and both 8-Cl-cAMP and 8-Cl-adenosine could activate enzymatic activity of PKC, which was recovered by A-134974. Furthermore, after 8-Cl-cAMP and 8-Cl-adenosine treatment, cyclin B was down-regulated and this down-regulation did not occur when co-treated with A-134974 and GF-109203x. These results suggest that there might exist signaling cascade such as PKC activation and cyclin B down-regulation after 8-Cl-cAMP and 8-Cl-adenosine treatment.

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Cloning and Functional Expression of Acyl-CoA Desaturase-encoding cDNAs Isolated from Pheromone Gland of *Helicoverpa assulta*, the Oriental Tobacco Budworm (Noctuidae, Lepidoptera)

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Seven desaturase cDNAs were isolated from pheromone glands of Helicoverpa assulta, a moth producing a sex pheromone blend with high Z9-16:Ald and low Z11-16:Ald, opposite to what is found in other heliothine moths such as Helicoverpa zea. Six of the seven sequences map onto recently defined lepidopteran desaturase sequence lineage and the other is orthologous to a desaturase sequence previously reported in H. zea. The levels of desaturase-encoding transcripts in pheromone glands were determined and the three most abundant ones were functionally expressed in a desaturase-deficient mutant strain of Saccharomyces cerevisae. The HassNPVE transvript, shown to encode a 9 desaturase producing more Z9-18:Acid than Z9-16:Acid, was the most abundant, followed by the HassKPSE transcript, shown to encode a 9 desaturase producing more Z9-16:Acid than Z9-18:Acid, and by the *HassLPAQ* transcript, shown to encode a 11 desaturase producing only Z11-16:Acid. Thus, the relative amounts of transcripts encoding two 9 desaturases and a single 11 desaturase in H. assultapheromone were consistent with the relative amounts of unsaturated fatty acid precusors required to produce the major and minor sex pheromone components of this species.