Z504 The aspects of protein kinase c isozymes during the induction of apoptosis by protein kinase c inhibitor in PC-3, human prostate cancer cell line.

Jeong Soon Park* and Won Chul Choi
부산대학교 자연과학대학 생물학과 세포생물학실

Apoptosis is one of the key mechanisms of physiological cell death as make controlling mechanisms in the embryo developments, growth, and differentiation. The activation of protein kinase c (PKC) is one of the earliest event in the cascade of signal transduction that induced a various cellular function as like growth of cell, differentiation, and gene expression. Apoptosis is induced by chemotherapy and ionizing radiation therapy of cancer cell, is characterized with membrane blebs, the condensation of chromatin, formation of apoptotic bodies, and production of DNA ladder. Therefore we have been studied about inductions by into PC-3 cell, a human prostate cancer cell line, the treatment of retinoic acid (RA), that is a anticancer agents, H-7 and HA1004 that are inhibitor of protein kinases, and PMA that is a tumor promoting factor. And we studied about the change of PKC isozymes during induction of apoptosis. The PC-3 cells were induced dose-dependently apoptosis by treatment with H-7, HA1004, and RA. The groups treated with H-7 or HA1004 have been inhibited apoptosis by PMA, that is announced inhibitor of induction of apoptosis, but the group treated with RA couldn't show the inhibition by PMA. PC-3 cells normally express multiple PKC isozymes including α, δ, ε, ζ, $\lambda(\iota)$, and μ. But PKC β, γ, η, θ isozymes did not appear to be expressed to significant levels. The expression of PKC α did not change in PC-3 cells, but PKC δ ε, ζ, $\lambda(\iota)$, and μ was shown the change of expression by treatment of H-7 and HA1004. Except of PKC ζ, PKC α, δ, ε, $\lambda(\iota)$, and μ isozymes was down-regulated by PMA. Especially, PKC δ, ε, and ζ were shown a more diverse variation than $\lambda(\iota)$ and μ isozymes. So that we suggested that PC-3 cell treated with H-7, HA1004, and RA was induced apoptosis by the pathway of calcium-independent.

Z505 Isolation and Characterization of Genes Responsibe for Ethanol Sensitivity in the Nematode *Caenorhabditis elegans*

Min Gi Hong and Junho Lee
Department of Biology, Yonsei University

The mechanisms and sites of action of volatile anesthetics and ethanol are not fully understood. In the hope of understanding the mechanisms of general anesthetics, we identified genes that control the sites of action for ethanol and anesthetics in the invertebrate system *Caenorhabditis elegans* . Because the response of *C. elegans* to ethanol and anesthetics is similar to that of mammals, elucidating the action mechanism of ethanol and anesthetics in the nematode will help understanding their mechanisms in higher organisms including humans. As a strat, we wanted to identify genes responsible for ethanol sensitivity by isolating mutations that confer ethanol resistance. Some ten thausand wild-type nematode individuals (N2) were subject to mutagenesis, and the offspring were screened in the second generation forresistance to ethanol exposure. We found 9 independent mutations so far. *ys9*, the first ethanol-resistant mutant isolated in our screen, was not different from N2 in shape and behavior, but showed resistance to 7 vol% ethanol treatment. *ys10* , a second mutation, was also wild type in shape and behavior. *ys11* animals displayed other phenotypes as well as ethanol resistance. The *ys11* animals are medium dumpy (Dpy), and their life cycle is longer than N2(5.5 days in 20C). Interestingly, none of the *ys9, ys10,* and *ys11* mutant animals survived after freezing and thawing, while wild-type animals did, indicating that there may be alteration in memebrane properties. Other mutations did survive freezing and thawing. We are currently performing genetic and molecular characterization of the genes for those mutations, and will present up-to-date results.