

alcohol dehydrogenase (ADH). In this study, ADH gene encoding a 104-kDa (p104) protein was identified and characterized. The deduced amino acid sequence of pneumococcal ADH shows homology with other members of the ADH family, and particularly with *Entamoeba histolytica* ADH2 and *E. coli* ADH. *S. pneumoniae* adh is composed of 883 amino acids and its estimated isoelectric point is 6.09. Although ADH is conserved between *S. pneumoniae* and *E. coli*, immunoblot analysis employing antisera raised against pneumococcus ADH demonstrated no cross-reactivity with ADH analog in *Escherichia coli*, *Staphylococcus aureus* and human HeLa cells. Also secretion of ADH was demonstrated by subcellular fractionation and immunoblot analysis of proteins. These results suggest that *S. pneumoniae* ADH could be a highly feasible candidate for both diagnostic marker and vaccine.

Poster Presentations – Field C3. Cell Biology

[PC3-1] [10/19/2001 (Fri) 09:00 – 12:00 / Hall D]

Caspase-dependent apoptosis by naphthoquinone analog in HL-60 cells

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Menadione has been known to exhibit a broad spectrum of antitumor activity in rodent and human cancer cells. Previous study showed that 2,3-dichloro-5,8-dihydroxy -1,4-naphthoquinone, one of the synthesized naphthoquinone analogs, have a anticancer effect on mouse leukemic L1210 and sarcoma-180 cells. Here we investigated the cellular effects and biochemical changes by naphthoquinone analog in human leukemic HL-60 cells. Naphthoquinone analog(NA) induced apoptotic cell death in HL-60 cells, which was shown by DNA ladder of fragments, a characteristic morphological change associated with apoptotic cells. NA induced the activation of caspases, release of mitochondrial cytochrome c into cytosol and upregulation of pro-apoptotic Bax protein but had no effect on anti-apoptotic proteins like Bcl-2 and Bcl-xL. The caspase inhibitor, z-VAD-FMK inhibited caspase activation and Bid cleavage by naphthoquinone analog but not cytochrome c release. These results show that naphthoquinone analog induces apoptosis through activating caspases and regulating Bcl-2 family proteins in HL-60 cells.

[PC3-2] [10/19/2001 (Fri) 09:00 – 12:00 / Hall D]

Studies on the Anticarcinogenic effects of Solanum tuberosum extracts

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Studies on the Anticarcinogenic effects of Solanum tuberosum extracts

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In this study, we performed to investigate the effects of cytotoxicity and quinone reductase(QR) induced activity of Potato(*Solanum tuberosum*) peel extracts on several human cancer cells, such as HepG2, HeLa and MCF-7. We extracted the peel of *Solanum tuberosum*(STP) with methanol and the methanol extract(STPM) was partitioned with n-hexane(STPMH), ethylether(STPMEE), ethylacetate(STPMEA), n-

butanol(STPMB) and aqueous(STPMA) solvent. Among five different partition layers, the ethylether (STPMEE) and the n-butanol(STPMB) partition layers showed the most efficient anti-proliferative effects at 250 μ g/mL which resulted ~90% on all human cancer cell lines which we used. We also measured quinone reductase(QR) activity extracts of STP on HepG2 cells. Among various partition layers of peel of Solanum tuberosum, QR activity induced by the ethylacetate partition layer(STPMEA) and the n-butanol partition layer(STPMB) dose of 40 μ g/mL on HepG2 cells showed 3.8 and 5.2 respectively compared to the non-induced control as 1.0. Moreover, the comparison on the cytotoxicity and quinone reductase induced effects of Solanum tuberosum are also under study.

[PC3-3] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Platycodin D Mediates ROIs-Induced NF- κ B Activation and Cytotoxic Effects in Immortalized Keratinocytes

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Platycodin D isolated from the root of platycodon grandiflorum A. DC. (Campanulaceae) is known to be an antiinflammatory agent, showing the mechanism as an inhibitor of inducible cyclooxygenase-2 (COX-2), thus, resulting in the decrease of prostaglandin E2 production. In contrast, we could get the opposite results using a cell-based assay system developed for the assay of NF- κ B. On addition of platycodin D into immortalized skin cells (HaCaT and SCC-13), NF- κ B was found to be activated. We postulate that auto-oxidation in the culture medium, caused the generation of reactive oxygen intermediates (ROIs), which are considered as NF- κ B activator. When N-acetyl-L-cysteine, a radical scavenger, was added into above reaction system, NF- κ B production was significantly reduced. HaCaT and SCC-13 cell line have rather abnormal cell cycle, comparing to normal keratinocytes. It is likely that NF- κ B activation is involved with apoptosis (programmed cell death) in cancer cells. Skin cell-death induced by Platycodin D is featured by DNA fragmentation, decrease of cell viability, and NF- κ B activation. Therefore, we suggest that the skin cell-death by Platycodin D might be the signal of apoptosis by NF- κ B activation.

[PC3-4] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Effects of *Aralia elata* Extract as an Absorption Enhancer on the Transport of Chondroitin Sulfate and Its Digestion Products in Caco-2 Cell Monolayers

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The absorption enhancing effect of *Aralia elata* extract was investigated in Caco-2 cell monolayers. The transport experiment on this extract was performed to evaluate the efficiency as an absorption enhancer to decrease the transepithelial electrical resistance (TEER) of the cell monolayers and to increase the intracellular permeability of the hydrophilic molecules. The addition of *Aralia elata* extract at the concentration of both 0.04% and 0.08% (w/v) to the cell monolayers decreased the TEER. The quantitative analysis of the transported intact chondroitin sulfate (CS) and its digestion products indicated that the extract increased the intracellular permeation of the anionic water-soluble molecules compared to the controls. Moreover we also evaluated the concentration range of the extract where they are relatively safe as an absorption enhancer. The results of MTT assay indicated that the cytotoxicity of the extract at the concentration below 0.1% (w/v) could be negligible. In conclusion, our results suggest that *Aralia elata* extract can be applied as an efficient absorption enhancer to make it easier for the hydrophilic molecules to permeate biological membranes.