

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 250µg/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 40µg/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-κB 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-κB. On addition of platycodin D into immortalized skin cells (HaCaT and SCC-13), NF-κB 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-κB activator. When N-acetyl-L-cysteine, a radical scavenger, was added into above reaction system, NF-κB production was significantly reduced. HaCaT and SCC-13 cell line have rather abnormal cell cycle, comparing to normal keratinocytes. It is likely that NF-κB 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-κB activation. Therefore, we suggest that the skin cell-death by Platycodin D might be the signal of apoptosis by NF-κB 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.