extracts tested showed more or less antioxidative activity. *Crassula cv. himaturi* showed the stongest antioxidant activity (EC_{50}, 10.45 ug/ml), followed by *Euphorbia mill var. splendens, Euphorbia ingens, Euphorbia submammillaris (Bgr.) Bgr* (EC_{50}, 45.31, 72.07, 96.29, respectively). The approximate flavonoid aglycone content ranged from 0.5 % up to 6.1 %. No significant relationship was observed between the antioxidant activities and the flavonoid contents.

[PA1-30] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

New Acetylenic Compound, Montiporyne A, from the Stony Coral Montipora sp. Induced Apoptosis in Human Skin Cancer Cells

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The antiproliferative effect of a new acetylenic compound, Montiporyne A, from the stony coral Montipora sp. on human skin cancer cells was investigated. As determined by MTT assay, Montiporyne A decreased cell viability in a concentration dependent manner. To test if the growth inhibitory effect of Montirporyne A was derived from apoptosis induction, general evaluation focusing on apoptosis was conducted. Characteristic manifestations of apoptosis, such as nuclear changes, the increased ratio of proapoptotic protein Bax to antiapoptotic protein Bcl-2, and cleavage of a specific subset of protein, poly(ADP-ribose) polymerase, via activation of caspase-3, were demonstrated. Human Fas ligand and its membrane receptor Fas, which trigger apoptosis, were induced. Hence, these results suggest that the newly isolated Montiporyne A are capable of inhibiting cell proliferation and inducing apoptosis in human skin cancer cells.

[PA1-31] [10/18/2001 (Thr) 14:00 - 17:00 / Hall D]

Protective effects of quinic acid derivatives on tetrahydropapaveroline- induced cell death in C6 glial cells: Possible role of a MAP kinase during cell death

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Tetrahydropapaveroline (THP) is biosynthesized in plant by Pictet-Spengler condensation of dopamine with dopaldehyde. THP was reported to inhibit mitochondrial respiration and thought to be a contributing factor in developing Parkinson's disease. THP was toxic to C6 cells in a dose-dependent manner. When cells were exposed to 10 □, M of THP, the activities of JNK and p38 kinase rapidly increased whereas the activity of ERK decreased significantly. Furthermore, pretreatment of C6 cells with 8-(4-chlorophenylthio)-cAMP or SB203580 prevented THP-induced cell death, while exposure to either PD98059 or LY294002 did not protect cells from THP-induced death, indicating the involvement of JNK and p38 kinase in THP-induced cell damage. The neuroprotective effects of quinic acids from Aster scaber on THP-induced cell toxicity were evaluated to see whether quinic acid derivatives are beneficial for neurodegenerative diseases. Quinic acid derivatives significantly diminished THP-induced cell toxicity. Among the quinic acid derivatives tested, (-) 4,5-dicaffeoyl quinic acid exhibited the highest protection effect against THP-induced cell toxicity. Preincubation of cells with quinic acid prior to THP exposure elevated the cell survival rate and the activities of glutathione peroxidase and catalase, but decreased the level of MDA and SOD activity. Types of cell death caused by THP were assessed by morphological observation of cells after staining with Propidium iodide and Hoechst 33342. Taken together, the results indicate that THP-induced cell death is likely to be mediated through the activation of JNK and p38 kinase and that quinic acid might be a potential agent for treating neurodegenerative diseases including Parkinsonism.