60 cells through activation of caspase in conjunction with cytochrome c release induced by a processed product of Bid. Now we are further investigating the relationship with the mitochondrial potential, ROS and Fas expression.

[PC1-13] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Intermedeol-Induced Apoptosis Involved Fas/Fas-L and cytochrome c dependent pathway in Human Leukemic cell HL-60

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We previously demonstrated that Intermedeol, a sesqueterpene isolated from Ligularia Fischery var., had a antitumor activity by induction of cell differentiation and apoptosis in HL-60. In this study, we examined signaling pathways implicated in Intermedeol up-regulation of Fas receptor expression and caspase 8 activation of Intermedeol. Bid is processed after Fas ligation and thus might activate the mitochondrial-dependent apoptotic cascade. Activated Bid preceded the release of cytochrome c without mitochondrial permeability transition. Cytochrome c release led to the activation of caspase 9 and downstream death effector, caspase 3. These finding suggest that Intermedeol induced cytochrome c-dependent apoptosis through Fas/Fas-L pathway.

[PC1-14] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Requirement for JNK activation in costunolide-induced apoptosis

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Costunolide is an active compound isolated from the root of Saussurea lappa Clarks, a Chinese medicinal herb, and considered as a therapeutic candidate for various types of cancers. In this study, we investigate the effects of costunolide on the induction of apoptosis in human leukemia cells and its putative pathways of action. Using diphenylamine and Hoechest apoptosis analysis, costunolide caused apoptosis of U-937 cells in a concentration- and time-dependent manner. Since costunolide-induced apoptosis was completely prevented in Bcl-2 overexpressed cells, these apoptosis was associated with Bcl-2, Furthermore, we demonstrated a requirement for c-Jun N-terminal Kinase, a member of the mitogen-activated protein kinase family in mediating cosunolide-induced apoptosis of human leukemia U-937 cells. JNK activation by costunolide contributed to apoptosis because transdominant-negative JNK significantly blocked costunolide-induced cell death. These findings cause the possibility that the JNK activation by costunolide can inhibit the Bcl-2 activity by phosphorylation.

[PC1-15] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Studies on the Growth-Inhibitory Effects of Pini Resina and Sodium Chloride against Oral Bacteria

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There has been considerable interest in the use of antimicrobial agents including a number of antiseptics, antibiotics and some natural products, as additives of some oral hygienic products for the purpose of treatment and/or prevention of periodontal disease.

In this report, water-, treated water- and ethanol-extracts of Pini resina, and water-extracts of branches of Mori albae were prepared and growth-inhibitory effects of these extracts, mixtures of extracts and sodium chloride against some representative oral bacteria were estimated by using agar diffusion methods and standard disk sus ceptibility testing procedures. In addition, dentifrice preparations containing these samples were also tested. the tested bacteria included ,Streptococcus mutams, stre [tpcpddus samguis, Actinomyces viscosus and Lactobacillus acidophilus for the agar diffusion methods and only S. mutans was used for the disk susceptibility tests.
MIC(Minimal Inhibitory Concentration) data of the tested samples were ambiguous to interpret due to the low solubility of these samples except the case of sodium chloride. Qualitative data from disk susceptibility test with ethanol-extracts of Pini resina suggested some potential applicability of this sample to the prevention of the periodontal diseases.
From this study, the following conclusions were made: In salt containing dentifrice, MIC is 5%(w/v%). 50% ethanol extracts are most inhibiting extracts on S. mutans, it was proved by performance standards for antimicrobial disk susceptibility tests. In Pini resina and treated Pini resina solution, its inhibition diameter is significantly equal to inhibition diameter of 1% chlorhexidine gluconate in 6.25, 12.5, 25㎍ inoculation.
Pini resina and treaed Pini resina extracts, Pini resina and treaed Pini resina extracts containing dentifrice might be useful for elimination of periodontal disease.

[PC1-16] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Mechanism of Manassantin A and B induced-differentiation in human leukemia HL-60 cell

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We already reported the differentiation inducing effect of Manassatin A and B, isolated from Saururus chinensi, in HL-60 human leukemia cells. These differeniation effect was further confirmed by esterase, phagocytosis and morphology change. The mechanism of differentiation was performed both western blot and RT-PCR techniques. Both Manassatin A and B exhibited a strong induction of mRNA and protein level of p21, CDK inhibitor at a concentration of 5 ㎍/㎖. The mRNA and protein level of c-myc was markedly suppressed in dose and time dependent manner. These results suggest that Manassatin A and B induced differentiation of HL-60 through up-regulation of p21 and down-regulation of c-myc mRNA and protein expression.

[PC1-17] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Suppression of RelA Transactivation Activity by Lignoids isolated from Saururus chinensis.

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In search for NF-kB inhibitors from natural resources, a novel dineolignan as well as four known dineolignans namned manasantin A (MNSA), manasantin B (MNSB), saucernetin, and sacerneol methylether were isolated from the MeOH extract of Saururus chinensis by activity-guided fractionation. The structure of a new compound was elucidated as saucerneol B on the basis of spectroscopic evidences. All of these compounds inhibited induced NF-kB activation by LPS or TNF-α in a dose-dependent manner. The relative potency of these compounds in NF-kB reporter assay was: MNSA = MNSB > saucerneol B > saucerneol methylether > saucernetin. However, these compounds did not