

Novel α -L-Arabinopyranosidase have been purified from *Bifidobacterium berve* K-110, which was isolated from human intestinal microflora with ginsenoside Rb2-hydrolyzing enzyme. α -L-Arabinopyranosidase acted to the greatest extent on p-nitrophenyl- α -L-Arabinopyranoside and ginsenoside Rb2, but did not hydrolyze p-nitrophenyl- α -L-Arabinofuranoside and ginsenoside Rc. α -L-Arabinopyranosidase was purified to apparent homogeneity by a combination of DEAE-Cellulose, Butyl-toyopearl, Hydroxyapatite and Sephacryl S-300 column chromatography with the final specific activity of 8.8 μ mol/min/mg. Molecular weight of α -L-Arabinopyranosidase is 280 kDa by gel filtration, which is consisted of four identical subunits. (M.W. 72 kDa by SDS-PAGE) Its optimal pH was at pH 5.5-6.0. α -L-Arabinopyranosidase was potently inhibited by Cu²⁺ and PCMS.

Poster Presentations – Field C3. Cell Biology

[PC3-1] [04/18/2002 (Thr) 14:00 – 17:00 / Hall E]

Caspase-mediated cleavage of CDC6 and its functional implication in apoptotic process.

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Growing evidences suggest that proliferating cells are undergone to the arrest stage of the cell cycle prior to proceeding into apoptotic progression. CDC6, an DNA replication initiation factor has been shown to play an essential role in the regulation of re-initiation of DNA replication during the cell cycle. In the present study, we investigated whether CDC6 is involved in the stage of the cell cycle arrest in apoptotic progression. Here, we provide evidence that CDC6 undergoes caspase 3-mediated cleavage in the early phase of apoptotic progression. Proteolytic cleavage of CDC6 during apoptotic progression appears to occur in general since CDC6 similarly undergoes proteolytic cleavage in apoptotic HeLa cells induced by various apoptotic inducers such as Paclitaxel, Etoposide, and Ginsenoside Rh2. Immunoblot analysis demonstrated that the 62Kda-CDC6 is typically cleaved into a peptide fragment, a 44KDa. Interestingly, the proteolytic cleavage of CDC6 in the apoptotic cells was effectively blocked by treatment of the cells with Z-DEVD-fmk, the caspase 3 inhibitor in HeLa cells. In vitro experiments using [³⁵S]-Methionine labeled CDC6 and recombinant caspase 3 showed that CDC6 is a good substrate for caspase 3. To identify the cleavage site of CDC6 by caspase3, we constructed several point mutants and obtained the CDC6 mutant proteins using the in vitro translation system. The results from the cleavage mapping study showed that the cleavage site is located in the C-terminal that has been reported to contain the Nuclear Export Sequences (NES). From these results, we propose that proteolytic cleavage of CDC6 is functionally relevant event that can results in blocking the translocalization to subcellular location, which in turn, leads the cells into the cell cycle arrest and consequently into apoptosis.

[PC3-2] [04/18/2002 (Thr) 14:00 – 17:00 / Hall E]

Inhibition of PDGF-BB-Induced MAP Kinase(ERK1/2) Activation in Rat Aortic Vascular Smooth Muscle Cells by NQ12

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Several 1,4-naphthoquinone derivatives have been reported to possess many pharmacological effects such as anti-viral, anti-fungal, anti-cancer and anti-platelet activities. We have reported that 2-chloro-3-[4-(ethylcarboxy)-phenyl]-amino-1,4-naphthoquinone(NQ12) had potent inhibitory effect on the platelet aggregation in vitro and thrombosis in vivo. However, little has been known about functional role in vascular smooth muscle cells(VSMCs).