

increased proliferation and migration of Hs683 human glioma and C6 rat glioma cells in a dose-dependent manner. Since involvement of mitogen-activated protein kinases (MAPKs) in the cellular effect of GDNF has been suggested, we investigated the activation of JNK, ERK-1,2 and p38 by GDNF treatment in Hs683 cells. GDNF prominently increased phosphorylated form of p38 without affecting total p38 level. We also show that activation of other MAPKs, JNK and ERK-1,2, was also detected upon GDNF treatment, to a lesser degree compared to p38. Stimulatory effect of GDNF on Hs683 cells was suppressed by SB203580, p38 specific inhibitor. Moreover, PD98059, ERK inhibitor, inhibits effects of GDNF. Our data suggest that the stimulus effect of GDNF on glioma cell migration may possibly be mediated by activation of MAPKs.

Poster Presentations – Field C2. Microbiology

[PC2-1] [04/18/2003 (Fri) 09:30 – 12:30 / Hall P]

A Liquid Culture Bioassay System for the Detection of Quorum Sensing Signaling AHL Analogues

Kim YoungHee^o, Lee JaeGeun, Park SungHoon, Kim JungSun

Department of Biotechnology, Dongseo University & Department of Chemical Engineering, Pusan National University, Pusan, Korea

Recent studies have revealed that bacterial biofilm production by the gram-negative bacteria is regulated by the quorum sensing signaling molecules, AHLs (N-acyl homoserine lactones). This suggests that inhibiting the AHLs could enhance the effects of antibacterial agents. Halogenated furanones purified from the red algae *Delisea pulchra* have been known to decrease quorum sensing responses by competitive inhibition of the AHLs. In order to screen for these AHL inhibitors from marine natural products collected off Korean waters, an effective bioassay system has been developed using the AHL analogues-responsive recombinant *Agrobacterium tumefaciens* NTL4 (pCF218)(pCF372) strain. Compared to the previously developed plate bioassay, this novel liquid culture system was 100 times more sensitive and effective for quantitative analysis. Moreover, the lipophilicity of the AHL analogues seems to affect the response of the assay.

[PC2-2] [04/18/2003 (Fri) 09:30 – 12:30 / Hall P]

Purification and Characterization of β -Glucosidase and α -Arabinofuranosidase Metabolizing Ginsenoside Rc from *Bifidobacterium* K-103

Park SunYoung^o, Kim DongHyun

College of Pharmacy, Kyung Hee university

Ginsenoside, major components of ginseng have been reported to show various biological activities including an increase of cholesterol metabolism, stimulation of serum protein synthesis, immunomodulatory effects. To explain these pharmacological actions, it is thought that ginseng saponins should be metabolized by human intestinal bacteria after they are orally administered. Related to the biotransformation of ginsenosides, Bae *et. al.* isolated ginsenoside-metabolizing

bacteria. However, the purification of the glycosidases related to the metabolism of ginsenosides except β -glucosidase from K-110 have not been studied. We purified and characterized ginsenoside hydrolyzing β -glucosidase and α -arabinofuranosidase from *Bifidobacterium cholerium* K-103, a human intestinal bacterium. The specific activity of the homogeneously purified β -glucosidase was 0.65 $\mu\text{mole}/\text{min}/\text{mg}$. Molecular weight of the purified β -glucosidase was determined to be 360 kDa and the enzyme was composed of four identical subunits. Optimal pH range of β -glucosidase was 5.0~5.5 in phosphate buffer and the enzyme activity was inhibited by Cu^{++} . This enzyme transformed ginsenoside Rc to compound O. Specific activity of the homogeneously purified α -arabinofuranosidase was 0.76 $\mu\text{mole}/\text{min}/\text{mg}$. Molecular weight of the purified enzyme was determined to be 173 kDa and it has two identical subunits. Optimal pH range 5.5~6.0 in phosphate buffer and the enzyme activity was inhibited by addition of Cu^{++} . α -L-Arabinofuranosidase from *Bifidobacterium* K-103 transformed ginsenoside Rc to Rd but it didn't hydrolyze ginsenoside Rb2.

[PC2-3] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Antiallergic Activities of Daidzein, a Metabolite of Puerarin and Daidzin Produced by Human Intestinal Microflora

Park EunKyung^o, Choo MinKyung, Kim DongHyun

College of Pharmacy, Kyung Hee University

To evaluate the antiallergic activities of puerarin and daidzin from the rhizome of *Pueraria lobata*, *in vitro* and *in vivo* inhibitory activities of these compounds and their metabolite daidzein were measured. Daidzein exhibited potent inhibitory activity on the β -hexosaminidase release induced by DNP-HSA and potently inhibited the PCA reaction in mice. Daidzein administered intraperitoneally showed the strongest inhibitory activity and significantly inhibited the PCA reaction at doses of 25 and 50 mg/kg with inhibitory activity of 37% and 73%, respectively. The inhibitory activity of intraperitoneally administered daidzein was stronger than those of intraperitoneally and orally administered puerarin and daidzin. Therefore we believe that puerarin and daidzin in the rhizome of *Pueraria lobata* are prodrugs, which have antiallergic activities, produced by intestinal microflora.

[PC2-4] [04/18/2003 (Fri) 09:30 - 12:30 / Hall P]

Protective Effect of Kakkalide from *Puerariae Flos* on Ethanol-Induced Lethality and Hepatic Injury Is Expressed by Human Intestinal Microflora

Han YeoOk, Han MyungJoo, Park SungHwan^o, Kim DongHyun

Department of Food Science and College of Pharmacy, Kyung Hee University

The inhibitory effect of kakkalide isolated from *Puerariae flos* on ethanol-induced lethality and hepatic injury were investigated. Intraperitoneally treated Kakkalide was weakly reduced the mortality associated with administration of ethanol and did not reduce alcohol hepatotoxicity. However, orally administered kakkalide and intraperitoneally administered irisolidone significantly reduced the mortality and potently reduced serum ALT and AST activities on liver-injured mice by ethanol. When kakkalide (200mg/kg) was orally administered to rat, a main compound was irisolidone and kakkalide was not detected in blood and urine. Kakkalide was metabolized to irisolidone via kakkalidone when incubated with human intestinal microflora. Based on these