## Relation of Chitosan oligosaccharide-induced Melanin Production to The Activity and Expression of Tyrosinase in B16 Melanoma Cells

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To investigate the effect of chitosn oligosaccharide on skin care, we measured tyrosinase activity and melanin production in B16 melanoma cells, and elastase and hyaluronidase activity. Chitosan oligosaccharide itself did not have any anti-oxidant activity in DPPH radical scavenging, and did not affect the proliferation of B16 melanoma cells. Chitosan oligosaccharide dose-depednetly increased mealnin production in the absence or presence of MSH. However, chitosan oligosaccharide did not have any influence on the tyrosinase activity and tyrosinase expression in B16 melanoma cells. These results suggest that chitosan oligosaccharide-induced melanin production may be independent on tyrosinase in B16 melanoma cells. On the other hand, chitosan oligosaccharide increased neutrophil elastase activity but decreased hyaluronidase activity in a dose-dependent manner. From the above results. chitosan oligosaccharide dose-dependently appears to increase melanin production in B16 melanoma cells and inhibit hyaruronidase activity, suggesting that chitosan oligosaccharide may be used as sun-tanning agent and water conservative agent.

**[PB1-2] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Hypothetical Mechanisms of G protein-coupled neurodegeneration in glutamate excitotoxicity in human SH-SY5Y neuroblastoma cells

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The cellular mechanisms by which excess exposure to the excitatory neurotransmitter glutamate can produce neuronal injury are unknown. In this study, we found that glutamate induced cell death at IC (50) of 100 microM on the cultured human SH-SY5Y neuroblastoma cells. It has been hypothesized that glutamate excitotoxicity is related with the elevation of calcium (Ca) levels. To determine the dependence of glutamate neurotoxicity on Ca environment, extracellular (EDTA) and intracellular (BAPTA/AM) chelator were used. Pretreatment with EDTA (1 mM) did not suppress the glutamate induced cell death. However, pretreatment with BAPTA/AM (2 $\mu$M) prevented glutamate-induced cell death. For further investigation the role of intracellular Ca homeostasis in mechanisms of neuronal cell death, SH-SY5Y human neuroblastoma cells were treated with A23187 calcium ionophore. Interestingly, we found that the combination of glutamate and A23187 (0.5 $\mu$M) considerably attenuates neuronal cell viability, compared to glutamate alone, indicating the pivotal role of Ca in the process of glutamate neurotoxicity. In the parallel experiment dantrolene (40 $\mu$m), a ryanodine receptor antagonist, was also found to prevent the glutamate-induced cell death. Results of other test suggest that N-methyl-D-aspartate (NMDA) receptors may play a role in mediating glutamate-induced lethal Ca influx. The lethal effect of glutamate was abolished by the selective NMDA-receptor antagonist (+)-MK 801 (10 $\mu$M).On the other hand, Pertussis toxin (PTX) blocked glutamate-induced cell death, indicating possible involvement of G proteins in the process. Moreover it has been reported that glutamate exerts its effect by both activation of ionotropic and metabotropic receptors (mGluR). In future we hope to establish whether activation of mGluR is also involved in glutamate-induced cell death. The effect of mGluR agonist trans-ACPD and antagonist (+)-MCPG will be examined.

**[PB1-3] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Whitening Activity of Phenylpropanoid Compounds

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To investigate the relationship between structure and biological activity of phenylpropanoids, we measured effects of phenylpropanoids on anti-oxidant and whitening activity. In DPPH radical scavenging activity, caffeic