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A benzopyranyl derivative, KR32000, synthesized as a plausible KATP opener, has been shown to exert cardioprotective effect in vivo myocardial infarct model. In this study, we investigated whether KR32000 can produce cardioprotective effect against hypoxia- and reactive oxygen species(ROS)-induced injury in heart-derived H9c2 cells. Hypoxic injury was induced by incubating cells in anaerobic chamber (glucose-free, serum-free DMEM, 85% N2, 5% CO2, 10% H2) and oxidative stress was induced by buthionine sulfoximine(BSO). Cell viability was evaluated by MTT and LDH assay. KR32000 30 μM significantly decreased LDH release induced by hypoxia in H9c2 cells. This decrease in LDH release was inhibited by HMR1883, a blocker of sarcolemmal KATP channel, but not by 5HD, a blocker of mitochondrial KATP channel. KR32000 also decreased BSO-induced H9c2 cell death. ROS generation by BSO was decreased by 30 μM KR32000. These results suggest that KR32000 protects H9c2 cells from hypoxia- and oxidative stress-induced injury, at least in part, through sarcolemmal KATP channel and antioxidant effect. And also, we measured BSO-induced ROS generation to confirm whether KR32000 had protective effect from ROS-induced cardiac injury.
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[PB1-3] [10/17/2002 (Thr) 13:30 - 16:30 / Hall C]

Effect of Chitosan Oligosaccharide on Tyrosinase Activity

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Tyrosinase (monophenol, 3,4-β-dihydroxyphenylalanin oxygen oxidoreductase, EC 1.14.18.1), which plays a pivotal role in melanogenesis. It is single chain glycoprotein catalyzing the hydroxylation of tyrosine to β-3,4-dihydroxyphenylalanin (DOPA) and the oxidation of DOPA to DOPA quinone. To investigate whitening effect of chitosan oligosaccharide, we obtained chitosan oligosaccharide [(glucosamine)2-6] by NaNO2 oxidation and measured the effect of chitosan oligosaccharide on tyrosinase activity. Chitosan oligosaccharide dose-dependently inhibited tyrosinase (2 unit) activity and inhibited by 18.8% at dose of 100 μg/ml. Vitamin C, arbutin and kojic acid that are well known to be inhibitor of melanin production dose-dependently inhibited tyrosinase (2 unit) activity. These results suggest that chitosan oligosaccharide may be used as inhibitor of melanin production in melanocyte, which will be further studied.

[PB1-4] [10/17/2002 (Thr) 13:30 - 16:30 / Hall C]

Quercetin analogs extracted from Lidera erythrocarpa protects heart-derived H9c2 cells from oxidative stress-induced death

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Bioflavonoids are semi-essential food components that are ubiquitously present in nature. It has been reported that flavonoids act as anti-oxidant as well as anti-cancer agents. Quercetin is one of the most widely distributed bioflavonoids in the plant kingdom. The goal of this study was to investigate effects of quercetin analogs extracted from Lindera erythrocarpa, quercetin 3-O-α-arabinofuranoside and quercetin 3-O-α-L-rhamnoside, on oxidative stress-induced cell death. Cell death was induced by using BSO, buthionine sulfoximine, which inhibit GSH level and subsequently increase ROS level. Cell death was quantitatively determined by measuring lactate dehydrogenase(LDH) activity, by propidium iodide(PI)-uptake. The intracellular level of ROS was measured by using DCFH-DA.
BSO-induced LDH release and PI-uptake was significantly decreased by quercetin 3-O-α-arabinofuranoside and quercetin 3-O-α-L-rhamnoside. These components also reduced ROS production induced by treatment with BSO. In conclusion, our results suggest that quercetin 3-O-α-arabinofuranoside and quercetin 3-O-α-L-rhamnoside can protect heart-derived H9c2 cells from oxidative stress-induced death through antioxidant effect.
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