

In boiling aqueous solution, D-amygdalin usually begins to convert into neoamygdalin in 3 min and more than 30% of the initial D-amygdalin is found as neoamygdalin after 30 min. In this report, we establish methods for simple HPLC analysis and the inhibition of D-amygdalin conversion. D-Amygdalin and its conversion product, neoamygdalin, were clearly separated on reverse-phase column chromatography by an optimized eluent of 10 mM sodium phosphate buffer (pH 3.8) containing 6% acetonitrile. Linearity for analyzing D-amygdalin and neoamygdalin was observed in the range from 0.05 to 0.5 mM. The detection limits for D-amygdalin and neoamygdalin were ca. 5 μ M per injected amount. We found that D-amygdalin conversion was completely inhibited by adding 0.05% citric acid to the aqueous solution before boiling. To prevent the loss of pharmaceutical potency of Tonin, we applied this method to measure the conversion rate of D-amygdalin. We confirmed that D-amygdalin conversion in Tonin is effectively inhibited by acidic boiling solution with 0.1% citric acid.

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Effects of Aporphine Compounds on Dopamine Biosynthesis in PC12 Cells

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The effects of aporphine isoquinoline alkaloids such as liriodenine, anonaine and asimilobine on dopamine biosynthesis in PC12 cells were investigated. Treatment of PC12 cells with liriodenine (10 μ M), anonaine (0.05 μ M) and asimilobine (0.15 μ M) showed 33.6%, 37.7% and 35.1% inhibition of dopamine content for 12 h. The IC_{50} values of liriodenine, anonaine and asimilobine were 8.4 μ M, 0.05 μ M and 0.13 μ M, respectively. Dopamine content decreased at 3 h and reached minimal level at 12 h after the exposure to aporphine isoquinoline alkaloids described above. Under these conditions, tyrosine hydroxylase (TH) and aromatic amino acid decarboxylase (AADC) activities were also inhibited by aporphine alkaloids. These compounds did not show cytotoxic effects, which were examined by MTT test assay. These results suggest that aporphine isoquinoline alkaloids contribute partially to the decrease in dopamine content by the inhibition of TH and AADC activities in PC12 cells. Intracellular mechanisms of aporphine alkaloids need further studies.

*Supported by Korea Research Foundation

[PA1-31] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Inhibitory effect of Lonicera Japonica on trypsin-induced inflammatory mediator secretion from human leukemic mast cells

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Mast cells play an important role in inflammation by functioning as a source of histamine, tryptase, and proinflammatory cytokines. *Lonicera Japonica* (Caprifoliaceae, Lc) has been used to treat inflammation. We investigated whether the water extract of *Lonicera Japonica*(Lc) inhibit production of inflammatory mediators such as tryptase and tumor-necrosis factor (TNF)- α , and phosphorylation of extracellular signal-regulated kinase(ERK) in trypsin-stimulated HMC-1. Lc (0.01 mg/ml-1.0 mg/ml) significantly inhibited tryptase and TNF- α production in a dose-dependent manner. Moreover, Lc inhibited ERK phosphorylation in trypsin-stimulated HMC-1. Our results suggest that Lc may inhibit tryptase and TNF- α production via ERK pathway.