Antiplatelet and antithrombotic activities of Sunghyangjunggi-san

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As part of our continuing search for biological active anti-stroke agents from the herbal medicinal resources. We examined the possibility of Sunghyangjunggi-san and its ingradients as a novel antithrombotic agents in vitro and ex vivo, and its antithrombotic effect in vivo.
Among ingredients of Sunghyangjunggi-san, Arisaematis Rhizoma, Cinnamomi Cortex and Zingiberis Rhizoma potently inhibited ADP- and collagen-induced platelet aggregation in a dose-dependent manner in vitro. Sunghyangjunggi-san and most of its ingredients did not affect coagulation parameters as APTT, PT and TT in human plasma. Sunghyangjunggi-san, Arisaematis Rhizoma, Atractylodis Rhizoma Alba and Pinelliae Rhizoma significantly inhibited ex vivo rat platelet aggregation. Sunghyangjunggi-san, Alpiniae Fructus and Zingiberis Rhizoma showed significantly protection from death due to pulmonary thrombosis in mice.

[PC2-4] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

Metabolism of ginsenoside Rc by human intestinal bacteria

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Ginseng is frequently used in Asian countries for replenishment of viatal energy, tranquilization, mood elevation and prevention of aging. Its major components are ginsenosides, such as ginsenoside Rb1, Rb2 and Rc. These ginsenosides have been reported to show various biological activities including an anti-inflammatory and anti-tumor activities. To explain these pharmacological actions, it is thought that ginseng saponins must be metabolized by human intestinal microflora after orally taken them. Therefore, we investigate the metabolism of ginsenoside Rc by human intestinal bacteria. Ginsenoside Rc was metabolized to compound K and 20(S)-protopanaxadiol. Bifidobacterium K-506 transformed to compound K via compound Mb→compound F2 and/or compound Mc. However, Bifidobacterium K-103 transformed to compound K via compound Rd→ compound F2. Mb was a new compound, 3-O-(β-D-glucosyl)-20S-O-(α-L-arabinofuranosyl-1,6-β-D-glucopyranosyl)--protopanaxadiol (MW. 940[+Na]).

[PC2-5] [10/19/2001 (Fri) 09:00 - 12:00 / Hall D]

In vitro antagonistic acticity of Acharan sulfate against Helicobacter pylori infection to KATO III cell line

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Helicobacter pylori is recognized as a major etiological agent of acute and chronic gastritis. Infection with HP is strongly associated with pathogenesis of peptic ulceration and the development of adenocarcinoma of the distal stomach. HP adherences to sulfated carbohydrates, GM3 ganglioside, phosphatidylethanolamine and sialylactose of the mucous epithelial cell surface and the mucous layer lining the gastric epithelium.
Therefore, we investigate the antagonistic activity of acharan sulfate, which is an acidic glycosaminoglycan from Achatica fulica, on HP infection to a gastric cell line. This acharan sulfate was a