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The herbal combination *allergina* has been used for the treatment of inflammatory diseases in South Korea. In this study, we investigated the immunosuppressive activities of *allergina* in more detail. Acute graft-versus-host disease (GVHD) is a major cause of morbidity and mortality in patients undergoing allogeneic bone marrow transplantation. Administration of *allergina* into Balb/c mice given total body irradiation followed by transplantation of bone marrow and spleen cells from C57BL/6 mice markedly reduced the mortality. In addition to systemic GVHD, *allergina* also showed therapeutic activity in local GVHD, in which *allergina* reduced lymph node weight of B6C3F1 mice transplanted of spleen cells from C3H mice. This herbal medicine also inhibited delayed-type hypersensitivity in which mice were intradermally injected with sheep red blood cells into the left footpad and were given *allergina* orally, suggesting that *allergina* inhibited cell-mediated immunity. *Allergina* also inhibited collagen-induced arthritis in DBA/1 mice, which were immunized with type II collagen, complete Freund adjuvant, and LPS. These results suggest that *allergina* could be a potent therapeutic agent for the treatment of immune-related diseases such as graft rejection, hypersensitivity, and autoimmune arthritis.

[PA1-48] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Comparisons of thermogenic activity among Ginseng Radix Alba (GRA), Ginseng Radix Rubra (GRR), and Panax Quinquefolii Radix (PQR)

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Uncoupling proteins (UCPs) are transmembrane proton transporters present in the mitochondria. In UCPs, UCP1 and UCP3 play an important role in adaptive thermogenesis by uncoupling mitochondrial oxidation of substrates from ATP synthesis.

PPAR γ coactivator 1 (PGC-1) regulates transcriptional activity of PPAR γ and other nuclear receptors and controls the expression of UCPs. We compared the adaptive thermogenic activity in ICR mouse treated with either GRA, GRR or PQR under the condition of either room (25 \rightarrow) or cold temperature (4 \rightarrow) by measuring rectal temperature, non-esterified fatty acid (NEFA), and glycerol. In addition, we investigated gene expressions of UCP1, UCP3 and PGC-1 in intrascapular brown adipose tissue, and gastrocnemius muscle using RT-PCR. Under of cold temperature, rectal temperature was increased to the level of room temperature by treatment. NEFA and glycerol decreased remarkably. In the mechanism study, expressions of UCPs and PGC-1 was upregulated by treatment. From these results, we may conclude that up-regulation of UCPs with treatment cause rectal temperature recovery under the cold temperature.

[PA1-49] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Anti-inflammatory, analgesic, and hypouricemic effect of *Actinidia polygama*(AP)

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The fruits of various *Actinidia* species are known to be used as a folk remedy for treatment of various inflammatory and analgesic ailments. Effects of the ethanol extracts and fractions from the fruits of *Actinidia polygama* (Sieb. et Zucc.) Maxim (Actinidiaceae) were studied using various in vivo and in vitro models of inflammation in mice and rats. In this study, we have evaluated the anti-inflammatory effects using acetic acid-induced vascular permeability and the carrageenin-induced rat paw edema and the analgesic activity using the formalin test, tail flick test and writhing assays. In addition, to clarify chronic effect, we performed the swelling and toxicity test in the Freund's complete adjuvant (FCA)-induced rheumatoid rat model. We also investigated the hypouricemic effect of AP on hyperuricemia induced by administration of the uricase inhibitor, potassium oxonate (250 mg/kg s.c., 1 h before the test drugs), and measured the plasma and urine urate in rats. 70 % ethanol extract of AP showed significant analgesic and anti-inflammatory activities in dose dependent fashion. In addition, AP ethanol extract also ameliorated the hyperuricemic state at a dose of 1000 mg/kg, though hypouricemic action of AP was inferior to that of either probenecid or allopurinol.

[PA1-50] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Effect of the Extract of Cultured Ginseng Roots on Hyperlipidemia in Rats

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We have investigated the effect of MeOH extract of cultured ginseng roots on hyperlipidemic rats induced by fat-rich diet. We also analyzed and compared ginsenosides of cultured ginsengs by HPLC. After oral administration of the extract to hyperlipidemic rats for four weeks, the variables including body weight, cholesterol, HDL, LDL, and triglyceride levels in serum were measured. One of the cultured ginseng roots (CBN3) decreased cholesterol and LDL-cholesterol and increased HDL-cholesterol levels in serum. HPLC analysis demonstrated that CBN3 contains more Rb1 and Rd ginsenosines than any other cultured ginsengs and cultivated ginseng. These results suggest that CBN3 with high contents of Rb1 and Rd may be useful in lowering blood cholesterol.

[PA1-51] [10/18/2002 (Fri) 09:30 - 12:30 / Hall C]

Antidiabetic effect and mechanisms of SPH-1 in db/db mice

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SPH-1 is a herbal medicine composing oriental prescription. We have studied the antidiabetic effect and mechanism of SPH-1 in insulin-resistant diabetic db/db mice. Mice were grouped and treated for 3 weeks as follows: control group was administrated with tap water orally; treated group was administrated with SPH-1 orally at dose of 500 mg/kg. SPH-1 lowered plasma glucose level by 67% as compared to the diabetic control. Total cholesterol, triglyceride and free fatty acid were all reduced in SPH-1 treated group. The control group showed hyperinsulinemia, whereas SPH-1 treatment decreased insulin level by 72% as compared to the diabetic control at the end of treatment. SPH-1 treated mice also exhibited low urinary glucose and albumin level as compared to the diabetic control, in parallel to the plasma glucose concentration. In the mechanism study, PPAR γ mRNA expression in epididymal fat were increased in SPH-1 treated group. GLUT4 mRNA expressions in skeletal muscle was also increased in SPH-1 treated group. We have also investigated glucose-6-phosphatase,