Inflammation is a frequent radiation-induced following therapeutic irradiation. Since the upregulation of adhesion molecules on endothelial cell surface has been known to be associated with inflammation, interfering with the expression of adhesion molecules is an important therapeutic target. We examined the effect if allicin, a major component of garlic, on the induction of intercellular adhesion molecule-1 (ICAM-1) by gamma-irradiation and the mechanisms of its effect in gamma-irradiated human umbilical vein endothelial cells (HUVECs). The inhibitory effect of allicin on ICAM-1 expression in gamma-irradiated HUVECs was assessed by ELISA and RT-PCR analysis, respectively. Also, the effects of allicin on transcription factors were determined by electrophoretic mobility shift assay (EMSA). Our data indicated that allicin significantly inhibited the surface expression of ICAM-1 and ICAM mRNA in a dose dependent manner. In EMSA analysis, AP-1 was activated in HUVECs by gamma-irradiation, whereas NF-kB was not. In addition, treatment with allicin resulted in the decrease of AP-1 activation. The data showed that treatment of JNK and p38 inhibitors were decreased radiation-induced expression of ICAM-1 by Western Blotting. We further investigated the effect of allicin on JNK and p38 MAP Kinase, and demonstrated that ICAM-1 expression induced by gamma irradiation was reduced by allicin in a dose dependant manner. And allicin decreases the level of p-p38 and p-pJNK in gamma-irradiated HUVCEs. These results suggest that allicin modulates expression of ICAM-1 via AP-1 dependent pathway in gamma-irradiated HUVECs and has therapeutic potential for the treatment of various inflammatory disorders associated with an increase of endothelial leukocyte adhesion molecules.

**[PB4-6] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Enhancing Effect and Action Mechanism of Interleukin-4 Production in Activated T Cells by Phytoestrogens

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Phytoestrogens are naturally occurring compounds derived from plants. Structurally, some phytoestrogens resemble endogenous estrogen of humans and animals. Phytoestrogens exhibit estrogen agonist/antagonist properties and have many biological effects such as prevention of hormone-dependent breast cancer, anti-oxidative activity, inhibition of tyrosine kinase activities and inhibition of angiogenesis. In this study we investigated whether biochanin A, a phytoestrogen, and its metabolites such genistein, p-ethylphenol and phenolic acid affect IL-4 production in EL-4 thymoma cell-line and primary lymph node cells. Biochanin A, genistein and p-ethylphenol significantly enhanced PMA-stimulated IL-4 production from EL-4 T cells in a dose-dependent manner while phenolic acid did not. This effect was not observed in primary lymph node cells. Biochanin A, genistein and p-ethylphenol induced IL-4 promoter activity in EL-4 T cells transiently transfected with IL-4 gene promoter constructs, but this effect was impaired in EL-4 T cells transfected with an IL-4 promoter construct deleted of P4 site carrying NF-AT and AP-1 binding sites. Furthermore, biochanin A, genistein and p-ethylphenol increased both NF-AT and AP-1 DNA binding activities, as demonstrated by electrophoretic mobility shift assay. The enhancing effects on IL-4 production and NF-AT/AP-1 DNA binding activities were, respectively, abrogated by specific inhibitors for PI3-K, PKC and p38 MAPK, indicating that biochanin A, genistein and p-ethylphenol might enhance IL-4 production by cross-talk between NFAT and AP-1 through PI3K/PKC or PKC/p38 MAPK signaling pathway. These results suggest that phytoestrogens and some their metabolites may increase allergic responses via enhancement of IL-4 production in T cells.

**[PB4-7] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## A small carbohydrate fraction from Artemisia Folium suppresses death of the mouse thymocytes in vitro by down-regulating the Fas death receptor gene

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Artemisia Folium is a preparation of dried leaves from Artemisia species and has been used traditionally to prevent or treat various kinds of woman's diseases. A similar preparation called Chinese Moxa has been used to

treat rheumatism by moxibustion in Chinese medicine. A small carbohydrate fraction of approximately 1,000 dlaton from the water-soluble extract of the Artemisia Folium promoted survival of the mouse thymocytes in culture. A mouse gene array study suggested that the fraction might modulate Fas/FasL dependent apoptotic cell death and thus had influence on the survival of the thymocytes in culture. RT-PCR analysis confirmed the down-regulation of the Fas gene by the treatment, supporting that the fraction modulated thymocyte death by suppressing the Fas gene expression.

**[PB4-8] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Effects of anti-inflammation and cell protection through biphenyl dimethyl dicarboxylate on Rat Microglia

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Biphenyl dimethyl dicarboxylate (DDB) is a by-product produced in process of synthesizing Schizandrin-C. Generally, DDB has known to protect hepatocytes and to decrease the index of liver enzyme (e.g. GOT and GPT) in chronic hepatitis. The present study was aimed to demonstrate whether DDB can protect the brain cell, especially the Alzheimer brain in vitro. As Alzheimers disease can be induced by activated microglia, a macrophage in the brain, through Abeta peptide (Aβ) produced from amyloid precursor protein (APP). Results showed that DDB attenuated the production of proinflammatory repertoire such as IL-1β, TNF-α, and Nitric oxide(NO) in 10μM to 25μM of DDB with the highest pick value at 24h. The attenuation was started from 6h and lasted up to 48h with clear evidences of cell protection (DAPI). The study suggested that DDB plays a important role in protecting the brain cells from the progressive Alzheimer's disease by inhibiting the chronic inflammation. In conclusion, we found that DDB can be used in neurodegenerative disease caused by inflammation and cell damages from stresses.

**[PB4-9] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Inductive Effects of Vibrio vulnificus Infections on Cytotoxic Activity and Expression of Inflammatory Cytokine Genes in Human Intestinal Epithelial Cells

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Vibrio vulnificus, a Gram-negative estuarine bacterium, is the causative agent of food-borne diseases, such as life-threatening septicemia. V. vulnificus penetrating into the intestinal epithelial barrier stimulates an inflammatory response in the adjacent intestinal mucosa. Therefore, interaction between V. vulnificus and intestinal cells is important for understanding of both the immunology of mucosal surfaces and V. vulnificus. In this study we investigated the effects of V. vulnificus infection on cytokine gene expression of human intestinal epithelial cells, Caco-2 and INT-407 cells. V. vulnificus infection significantly induced the expression of pro-inflammatory cytokines such as IL-1, IL-6, IL-8, IL-12, and IL-18 in both incubation time- and MOI-dependent manners, while did not affect TGF-beta, etc. expression. Especially, infection with V. vulnificus increased IL-8 mRNA level and also increased the binding activity of transcription factor NF-kB to the kB sites in both Caco-2 and INT-407 cells. Furthermore treatment with inhibitors for NF-kB activation and translocation abrogated the enhanced IL-8 gene expression by V. vulnificus infection, indicating that V. vulnificus infection induced IL-8 gene expression by increasing NF-kB binding activity in human epithelial cells.

**[PB4-10] [ 2003-10-10 09:00 - 13:00 / Grand Ballroom Pre-function ]**

## Allergenecity of soybean and soybean-based products

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