To determine the occurrence of vancomycin-resistant Enterococci in raw milk sample, we examined raw milk samples for 6 months. Enterococci were isolated directly from Enterococcal selective agar platessupplemented with 2mg of vancomycin per liter. 19strains were selected and identified by Vitek system. To determine resistance, 19 isolates were tested with vancomycin and teicoplanin.Vancomycin-resistant Enterococci were genotyped by PCR analysis and 5 of 19 isolates were VanC-1 type.

[PC2-12] [04/19/2001 (Thr) 15:30 - 16:30 / Hall 4]

Carrageenan-induced ulcerative colitis induces GAGs degrading enzymes of intestinal bacteria

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Ulcerative colitis (UC) is a non-infectious chronic intestinal inflammatory disease in humans. These animal models were mainly made by hydrolyzed carrageenan and 2,4-dinitrochlorobenzen. However, the mechanism underlying their pathogenesis are not well known. Therefore, we here studied the relationship between intestinal bacterial enzymes and carrageenan/DNCB-induced UC.
These UC model mice all showed signs of diarrhea, occult blood, prominent regenerations of the colonic mucosa and shortening of large intestine. In hydrolyzed carrageenan- and DCNB-induced UC model mice, GAGs degrading enzymes of intestinal bacteria, particulary chondroitinase and hyaluronidase, were potently induced. The hydrolyzed carrageenan exhibited the in vitro cytotoxicity against intestinal epithelial cell line (IEC18). The hydrolyzed carrageenan also induced bacterial GAGs-degrading enzymes in human intestinal bacterial culture system. These UCs were improved by antioxidant herbal drugs.

Poster Presentations - Field C3. Cell Biology

[PC3-1] [04/19/2001 (Thr) 15:30 - 16:30 / Hall 4]

A Role of NF-kB Activation on Melanogenesis in Transfectant Human HaCaT Keratinocytes

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NF-kB (nuclear factor- kappa B) plays a particularly central role in epidermal biology. It is well established that ultraviolet radiation (UVR) is one of the mechanisms to induce the activation of NF-kB in human skin. NF-kB activation by UVR is involved in immune or inflammation responses as well as growth control of cells. In order to demonstrate the role of NF-kB activation on meleanogenesis, we transfected pNF-kB-SEAP-NPT plasmid into human HaCaT keratinocytes. Transfectant cells released

the secretory alkaline phosphatase (SEAP) as a transcription reporter in response to the NF-kB activity and contain the neomycin phosphotransferase (NPT) gene for the dominant selection marker for geneticin resistance. Melanogenic inhibitors (niacinamide, kojic acid, hydroquinone, resorcinol. arbutin, and glycolic acid) were preincubated with transfectant HaCaT cells for 3 hrs and then UV was radiated. NF-kB activation was measured with the SEAP reporter gene assay using a fluorescence detection method. Of the melanogenic inhibitors, niacinamide, hydroquinone and kojic acid were the most potent inhibitors of NF-kB activation by UVR. Especially, the preincubation of niacinamide and kojic acid displayed that cell morphology have few damage in the dangerous UV-induced environment. These observations suggest that NF-kB plays an important role in the paracrine mediation of UV-induced melanogenesis and skin-whitening effect may be involved in NF-kB activation in the genetic molecular basis.

[PC3-2] [04/19/2001 (Thr) 15:30 - 16:30 / Hall 4]

P-glycoprotein is not functionally overexpressed, and bcr/abl and bax is down regulated in cisplatin resistant human chronic myelogenous leukemia K562 cells

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Human chronic myelogenous leukemia K562 cell lines were used in our laboratory to study the mechanism in the development of cisplatin resistance in cancer. Several reports have suggested that expression of bcr/abl tyrosine kinase renders chronic myelogenous leukemia cell lines such as K562 cell lines resistant to the induction of apoptosis by a variety of treatments. As assessed by WST-1 cytotoxicity assay, the K562/CDDP cell lines were 4.87-fold more resistant to cisplatin than the parent cell lines. Both cell lines were treated with cisplatin, for the purpose of studying drug accumulation and efflux. Drug accumulation and efflux were not showed significantly differentiation. Also, we have found that K562/CDDP has a no differentiation of expression pattern of p-glycoprotein with compared parental cell lines by immunoblotting. Further, DNA fragmentation analysis showed that K562/CDDP cell lines had significantly more resistant. This result suggests that the antiapoptotic functions may be responsible for cisplatin resistance. Additionally, expression pattern of apoptosis-regulating proteins analysis showed that K562/CDDP cell lines had reduction of bax expression. This result indicates that there may cause resistant to apoptosis through reduction of bax expression in CML cells having a drug resistance. On the other hand, the expression of bcr/abl had reduction in the K562/CDDP cell lines. Our studies did not establish whether the down-regulation of bcr/abl is transcriptionally or *posttranscriptionally regulated. If cisplatin resistance affects the transcription of the bcr/abl fusion* gene, this may also be mediated directly or indirectly by altered gene-transcription and expression brought about by p53.

[PC3-3] [04/19/2001 (Thr) 15:30 - 16:30 / Hall 4]

Absorption Enhancement of Heparin Disaccharide through Intracellular Regulation of Paracellular Permeability by Phytolaccosides from Phytolacca americana

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The effect of phytolaccosides on the intestinal absorption of heparin disaccharides was studied using Caco-2 cells. The absorption enhancing activity of these compounds (phytolaccoside B, D_2, E, F, G, I) was determined by changes in transepithelial electrical resistance (TEER) and the transport amount of heparin disaccharides across Caco-2 cell monolayers. With an exception of phytolaccoside G, all