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