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which sequester NF-kB complex in the cytoplasm. After a specific signal, NF-kB is released from IkB and translocates to the nucleus to control gene expression. The constitutive activation of NF-kB has been observed in many human cancers such as hepatocellular carcinoma. In some types of cancer, NF-kB activation is supposed to be the result of an inflammatory increase or as a consequence of an inflammatory microenvironment during the maturation process of the malignant tumor. The loss of NF-kB inducibility leads to a deregulation of the expression of genes involved in cell cycle regulation, apoptosis, migration and cell adhesion, then it is evident a hold relationship between NF-kB and carcinogenesis. In mouse and in human it has been described a new isoform of p65, called p65(-1). This new isoform contains an unknown exon (exon -1) located upstream to the first known exon of *Rel*A, codifing for p65 (exon 0). We identify the expression of p65(-1) by 2D-electrophoresis, of protein samples from liver tissue belonging to patients affected and not by liver diseases. The results obtained show a different pattern expression in liver samples with cirrhosis and hepatocellular carcinoma.

New proteomic evidence on decorin effects on breast cancer cells

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The establishment of a dynamic crosstalk between the malignant cells and several components of the extracellular matrix (ECM) is a crucial step of the tumor progression. The ECM is a highly intricate microenvironment in which many signals exert opposite effects on the tumor cells. Historically our research group focused the attention on the molecular mechanisms underlying the effects of ECM molecules on the behavior of breast cancer cells in vitro. In this context, the decorin, a small leucine-rich proteoglycan (SLRP) involved in the collagen fibrillogenesis, was found to play an "anti-oncogenic" reaction by affecting the growth and motility in vitro of cancer cells. The aim of the present study was to improve the knowledge about the effect of ectopic decorin on the breast cancer cells, starting from the results previously reported by Pucci-Minafra et al. 2008 (Connect Tissue Res. doi: 10.1080/03008200701820443), in collaboration with the universities of Pavia and Bologna. The experimental model used for this purpose was represented by the 8701-BC breast cancer cell line and by its clone, called DEC-C2, obtained by transfection of 8701-BC cells for the synthesis and secretion of ectopic decorin. The entire protein extract from confluent 8701-BC and DEC-C2 cells were processed for the 2D-IPG based proteomic followed by the MALDI-TOF mass spectrometry for protein identification. To date we identified about 400 proteins, so triplicating the number of identified proteins respect to our previous data. The new proteomic evidences strengthen the anti-oncogenic effects of decorin and highlight the attention on the decreased expression of the majority of the members of three protein classes closely related to the malignant phenotype: the metabolic enzymes, the S100 family and the cell motility proteins. In conclusion, our results confirm and extend the evidence for a anti-oncogenic role of decorin and support the possibility of its use for clinical applications.