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41 Abnormal processing of arylsulfatase A in ovarian cancer. <u>T. Fujii</u>, <u>M. Ishikawa</u>, <u>T. Shimizu</u>, Dept. of Obst. and Gynec., Asahikawa Medical College, Hokkaido.

In the present study, we examined some modifications in the processing of AS-A in ovarian cancer. AS-A was purified from human placenta, ovary, and ovarian cancer tissues. The purified AS-A was composed from three non-identical peptides of molecular weight of 58kDa, 50kDa, and 10kDa on plolyacrylamide gel electrophoresis in the presence of SDS (SDS-PAGE). AS-A purified from normal ovary showed the similar pattern on SDS-PAGE. AS-A from ovarian cancer was composed from 58kDa band, and in addition 50kDa band decreased. In SDS-PAGE under non-reducing condition, the electrophoretic pattern of normal AS-A showed that 90kDa, 64kDa and 45kDa. Ovarian cancer 90kD increased and the 45kDa decreased. In polyacrylamide AS-A showed the gel isoelectric focusing, AS-A from normal ovary showed six-banded pattern with varying amounts of activity. Ovarian cancer AS-A showed the same banding pattern as normal ovary AS-A. AS-A from ovarian cancer had more oligosaccharidesn resistant to endoglycosidase H than AS-A from normal ovary. These findings suggest that the processing of both the ligosaccharide chains and the protein unit is modified in ovarian cancer.

42 An in vitro Experimental Model for Cancer-Epithelial Surface Interaction Using Human Amnion.<u>H.Sakamoto,K.Ohtani,K.Den,K.Satoh,</u> Dept.Obstet. Gynecol. Nihon Univ.Sch.Med.,Tokyo

In the present study, human ovarian cancer cell line was cultured on human amnion and the cell-epithelial/basement membrane interaction was studied. Human serous cystadenocarcinoma cell line (HRA, 105cells/plate) was plated on amnion (epithelial surface) fixed on a polystylene ring of 4.5x 4.5cm (total n=30). Cells were incubated at 37C under 5%CO2 in a medium containing 10% FCS (RPMI 1640 medium). Also cells were plated on a reversed surface (on compact layer lacking epithelium and basement membrane). Cells attached to the amnion were studied for their morphology under light and electron microscope and c-myc expression. Cells plated on the epithelial surface of the amnion showed growth and extension of the cytoplasm reaching the basement membrane through cracks in the epithelial layer. There seemed that the early stage of invasion is lead by one cell which has differentiated for the purpose. The front line of the invasion showed c-myc protein translation and marked microvilli development was observed. Cells, however, plated onto the reversed surface, did not show compatible growth. These observation suggest that recognition of the epithelial surface and basement membrane by cancer cells is a prerequisite for implantation.

43 A study on DNA leisons by cisplatin (CDDP) and its repair. <u>M.</u> <u>Iwata, S.Yoshida*, S.Maeda, T.Misawa, T.Kawai, K.Morikawa, F.Kikkawa, T.Kano, Y.</u> <u>Tomoda</u>. Dept.Obst.and Gynec., Nagoya Univ.Med.Sch., Aichi.*Lab.of Can.Cell Biol., Inst.for Disease Mech. and Cont., Nagoya Univ.Med.Sch., Aichi.

To investigate the lesions of DNA made by CDDP, single stranded DNA was incubated with cisplatin and the DNA was used as a template for DNA polymerase reaction. The reaction products were analysed on sequencing gel electrophoresis. T₇ DNA polymerase, mammalian DNA polymerase alpha and beta were used for this experiment.

To determine whether DNA repair activity is one of the mechanisms for ovarian cancer cells to become resistant against CDDP, cell growth inhibition was measured by MTT assay when cells were exposed to ultra violet (UV). Flow cytometric analysis of the CDDP resistant cells and CDDP sensitive cells exposed to CDDP were also examined.

DNA polymerase activity was inhibited strongly at GG and AG sequence. 7-fold CDDP resistant cell showed cross resistant to UV at 2-fold. After exposed to CDDP, cells at G_0 , G_1 phase decreased and G_2 , M phase increased. This pattern appeared earlier for CDDP resistant cell lines.