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370 Alterations of cytoskeleton in human endometrial carcinoma cells transferred by a chromosome #1. M.Nishida, S.Miyamoto, H.Kato, M.Sasaki\*, M.Oshimura\*, N.Wake, Dept.Obst.and Gynec., Med.Inst.Bioregulation, Kyushu Univ., Oita, \*Labo.Mol.and Cell Genetics, Tottori Univ., Tottori.

To identify the mechanism of morphological alterations induced in human endometrial carcinoma cells following the introduction of a normal human chromosome, we examined the amount and the cell distribution patterns of the three major cytoskeletal components in microcell hybrid cells. Microcell hybrids introduced by a single chromosome #1 showed the increased production of actin protein, compared with those of parental HHUA endometrial carcinoma cells and microcell hybrids containing a chromosome #9 or #19. The distribution of actin stress fibers in microcell hybrids containing a chromosome #1 appeared to be similar to that of normal endometrial cells though the distribution patterns were disrupted in the other cells. These findings were consistent with the results that the suppression of cell proliferation and tumorigenicity was most remarkable in hybrid cells transferred by a single chromosome #1, suggesting that the expression of the putative tumor suppressor gene locating on the chromosome #1 was responsible for the increased production of actin microfilaments and the reorganization of actin stress fibers.

371 Production of extracellular matrix components(ECMs) and expression of ECMs and cytoskeletons(CSs) in gynecologic tumor cell lines. T.Tsurunaga, Y.Seiki, M.Ueda, E.Iwai, T.Yamada, Y.Okamoto, O.Misaki, M.Ueki, O.Sugimoto, Dept. Obst. and Gynec., Osaka Medical College, Osaka.

The production of ECMs; laminin(La), collagenIV(Co), fibronectin(Fn) measured by RIA method and the expression of ECMs(La, Co, Fn), CSs; keratin (Ke), vimentin(Vi) and cell membrane components; ankyrin(An), fibronectin receptor(Fn-R) investigated by immunohistochemical technique were examined in 2 squamous carcinoma(SQ) cell lines; SKGIIIb, OMC-1, 3 adenocarcinoma(AD) cell lines; OMC-2, 3, 4 and 2 sarcoma(SR) cell lines; OMC-6, 7. The mainly produced ECMs in SQ cell lines was La, in AD was Co and in SR was both of them. These production was more increased in the early phase of logarithmical cells growth. There was no obvious difference in the immunohistochemical localization of La, Ke, Vi and An between SQ, AD and SR. On the other hand, Co, Fn and Fn-R were strongly expressed in AD and SR.

These findings suggest that abundantly produced Co in AD plays an important role in the re-construction of the basement membranes and strongly expressed Fn and Fn-R in AD and SR are utilized for the cell attachment and locomotion.

Human HHUA X normal fibroblast hybrids recover the tumor-forming ability by the loss of chromosome 4. <a href="M.Sasaki">M.Sasaki</a>, <a href="N.Wake">N.Wake</a>, <a href="Med.Inst.Bioregul.">Med.Inst.Bioregul.</a>, <a href="Kyushu Univ.">Kyushu Univ.</a>, <a href="Oita">Oita</a>.

We have examined 4 non-tumorigenic and 1 tumorigenic intraspecific human hybrid cells derived from fusion between human endometrial cancer cell line HHUA and normal human fibroblast. Tumorigenic segregants were obtained by inoculating these cells into nude mice subcutaneously. Karyotypes of non-tumorigenic clones and their tumorigenic segregants were compared to disclose the chromosomes associated with the recovery of tumorigenicity. Although three out of 4 non-tumorigenic clones contained 3 copies of #4 chromosome, only 2 copies were contained in tumorigenic segregants. One copy of chromosome #4 was also lost during the recovery of tumorigenicity in the remaining non-tumorigenic clone which contained 4 copies of the chromosome. The clone which had a tumor forming ability when the hybrids were formed, contained 2 copies of the chromosome. The chromosome composition was also identical in cells obtained from tumors. These results suggested that the chromosome 4 loss was compatible with the recovery of tumorigenicity in immortal endometrial cancer cells.