CEA and HCG localization.

Conclusion: The tissue localization of carcino-fetal proteins (CEA, HCG) and loss of Isoantigen suspect the malignant change of the endometrium strongly.

178. Regulation of Growth of Human Endometrial Adenocarcinoma Cells in vitro by Prostaglandin E

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In recent progress, it becomes inceasingly a new knowledge that prostaglandins have actions to regulate the growth of adenocarcinoma cells in vitro & in vivo. In contrast to the mechanism of steroid hormone action, prostaglandins presumably combine with cell membrane receptor which may be regulated nuclear functions.

This paper demonstrate the effects of prostaglandin E on human endometrial adenocarcinoma cells in vitro in the influences of steroid hormones.

Cells in exponential growth were treated with various physiological concentration of prostaglandin E, for varying times, the result of administration was measured by studying doubling time, determining survival function in cell cycle kinetics by flow cytometer (FCM). We gauged numbers of cells in the G_1, S, and G_2 stage of the cell cycle, and compared cell numbers in S with these in control cell cycle group. 24-hour treatment with prostaglandin E yielded 59.0-91.5% decrease with 5×10^{-9}M/ml, and 48-hour treatment with prostaglandin E yielded 66.5-78.0% decrease with 5×10^{-8}M/ml. FCM demonstrated a decrease in the number of cells in the S stage of the cell cycle among the treatments. Collectively these results suggest a role for prostaglandin E in the regulation of cancer cell proliferation and indicate a possible locus for therapeutic intervention.

179. Regulation by Steroid Hormones to the Cell Cycle of Endometrial Adenocarcinoma Cells in vitro

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Steroid hormones are essential for the regulation of cellular growth and differentiation of adenocarcinoma cells of the endometrium. Based on the analysis, we demonstrate some results which was evaluated the influences of four kinds of steroid hormones to cultured SNG cells.

Steroid hormones consist of Estradiol-17B, Diethylsteilbestrol, Progesterone and Tamoxifen.

Hormone concentrations are physiological (5×10^{-8}M/ml) and therapeutic (5×10^{-6}M/ml) levels.

Results are as follows.

1) Anti-estrogenic Tamoxifen was remarkably decreased the cell growth after 24 hours in the administration of physiological and therapeutic concentration and Progesterone showed antagonestic effects after 60 hours in therapeutic concentration in contrast to that of estrogens.

2) Within 6 hours, Progesterone increased rapidly the effects of growth of cultured cells as well as estrogens.

Synthetic hormones such as Tamoxifen was delayed after 12 hours in their actions.

180. The Effect of Biotin on the Progesterone Induced Synchronous Culture of HeLa-S_3 Cells

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The effect of biotin on the progesterone induced synchronization of HeLa-S_3 cells was examined. The activity of thymidine kinase (T.K.) of the cells was measured as a function of time in the presence of progesterone (10 ng/ml, 1 μg/ml) with or without biotin (330 ng/ml). The peak level of T.K. was about 30% higher in the presence of progesterone and the addition of biotin suppressed the increase. The cytoplasm of HeLa-S_3 cells cultured in the presence of progesterone was autoradiographically labeled with ^{14}C-biotin, suggesting that a biotin-binding substance(s) was induced by progesterone. The molecular weights of the binding substance(s) in HeLa-S_3 cells and human endometrial cells were determined by Gel-chromatography and the amounts of the substance(s) were measured by sensitive assay methods using ^{14}C-biotin, bentonite and avidin. It was likely that the binding substance(s) was induced and increased in the HeLa-S_3 cells by progesterone added into the medium and molecular weights of that were over 20×10^4 in HeLa-S_3 cells, and 7×10^4 and over 20×10^4 in human endometrial cells. It is possible that the biotin-bind-