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I S—9 Receptor for 1, 25-dihydroxyvitamine D3 (VD3) in endometrial cancer and the effect of VD3 on endometrial carcinoma cell lines

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Objectives: In view of the potential of 1, 25-dihydroxyvitamine D3 (VD3) as a cell differentiation inducing agent in endometrial cancer, we examined the VD3 receptor in endometrial adenocarcinoma immunohistochemically, also investigating the effect of VD3 on cell growth as well as the phenotypic changes for cell maturation after treatment with VD3 in two endometrial carcinoma cell lines. **Materials & Methods**: The expression of VD3 receptors was analyzed by ABC method using anti-VD3 receptor monoclonal antibody in tissue obtained from twenty-one endometrial carcinoma patients. In two cell lines (AMEC-1, RL95-2) derived from endometrial carcinoma, the expression of VD3 receptor and the effect of VD3 on cell growth were examined, and the morphological changes when cultured in a collagen gel as well as the expression of cytokeratin analyzed by Western blot were examined after VD3 treatment. **Results**: The VD3 receptor was detected in 14 of 21 endometrial carcinoma specimens. The growth of RL95-2 cells expressing VD3 receptor was inhibited to 41% when cultured with 10^{-8}M VD3. However, the growth of AMEC-1 cells not expressing VD3 receptor was completely uninhibited even when cultured with 10^{-5}M VD3. The RL95-2 cells exposed to 10^{-8}M VD3 for 6 days had increasing expression for cytokeratin, and became columnar with pronounced polarity and formed gland structures when cultured in collagen gel. **Conclusions**: These results suggested that endometrial adenocarcinoma is a target for VD3 and that VD3 appears to function as a cell differentiation inducer that may prove to be an antineoplastic agents for the treatment of endometrial cancer.

I S—10 Hormonal regulations for c-erbB-2 and p53 oncogene expressions in human endometrial adenocarcinoma cells

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[Objectives]The mechamisms of autocrine regulations in endometrial adenocarcinoma cells mediated by estrogen inducing transforming grwoth factor-α (TGF-α) had been reported at previous meeting. In this study, we tested that the hormonal regulation for the growth related oncogenes as c-erbB-2(erb) express acting local receptors to TGF-α and tumor suppresser gene as p53. [Methods] The expressions erb and p53 were detected by immnocytochemical assay using monoclonal antibodies. The cells were treated with those antibodies and stained by PAP methods. The erb and p53 oncoproteins and gene were quantitatively detected by Western and Southern analysis. [Results]The expression of oncoproteins were correlated to the histopathological grading of primary tumors. Ten nanomolar of estradiol stimulated the expression of erb protein in hormone responsive cells but not unresponsive cells. In contrast, 10pM of TGF-α were stimulated the expressions of erb protein in both responsive and unresponsive cells. The amplification of erb gene were observed in two cells not relating to the grade and hormone responsiveness. The expressions of p53 in responsive cells were stimulated by R5020 as dose dependent manner. [Conclusions]In hormone responsive cells, the gene expression were regulated by hormones and TGF-α. But in unresponsive cells, those were only regulated by growth factors.

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