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Changes in CA125 release and surface expression caused by drugs in uterine cervix adenocarcinoma cells

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The effect of drugs on the release of CA125 antigen and the binding of anti-CA125 monoclonal antibody (MoAb) to malignant cells was evaluated *in vitro*. TMCC-1, uterine cervical adenocarcinoma cells, were exposed to dexamethasone (DEX), sodium *n*-butyrate (NaB), dibutyryl cyclic AMP (dbcAMP), retinoic acid (RA), calcitriol (VD3), and interferon-γ (IFN-γ). NaB, RA and VD3 increased CA125 release per cell and ^{125}I-labeled anti-CA125 MoAb binding to the cells. DEX also increased the ^{125}I-labeled anti-CA125 MoAb binding to the cells, and CA125 antigen release per cell was also slightly increased. IFN-γ suppressed both CA125 release and ^{125}I-labeled MoAb binding. A combination of DEX, VD3 and RA and increased the binding of MoAb to TMCC-1 cells, but the amount of bound MoAb was not significantly different from that obtained by single drug treatment. DbcAMP had no significant effect on enhancing MoAb binding. Drugs can increase the binding of anti-CA125 MoAb to malignant cells and they may be applied to increase the tumor uptake of radiolabeled MoAbs *in vivo*.

Key words: CA125, TMCC-1, monoclonal antibody, ovarian cancer, VD3