The cistrome of estrogen receptor beta in colorectal cancer cells

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- We defined the cistrome of ERβ in different CRC cell lines. From the preliminary data- we identified key binding motifs, including ERE and AP-1 sites.
- This work provides the basis for identifying ER,β genomic binding sites in CRC cell lines and key binding sites like PRR5, GREB1, TMEM246 were confirmed using QPCR.
- Ligand independent binding of ERβ to target genes was observed in colon cancer cell lines.
- Confirmed the binding of ERβ to PRR5 in intergenic region: Tumor suppressor gene implicated in breast, colon cancers and subunit of mTORC2.

Introduction
Colorectal cancer is the third most common cancer in the world and the most frequent cancer among men (3200 men and 2900 women diagnosed every year in Sweden). Studies in premenopausal women, long term hormone replacement therapy reduces the risk of colon cancer by 20%. Use of oral contraceptives have shown lower incidence of colorectal cancer. These findings suggest that estrogen may lower the risk of CRC (1,2).

ERβ is the predominantly expressed estrogen receptor in the colonic epithelium and several in vivo and epidemiological studies indicate a tumor suppression effect of estrogen receptor beta (ERβ) on colon cancer. We have shown before re-expression of ERβ in colon cancer cell lines has anti proliferative and anti-inflammatory properties (2,3). Here we explore the cistrome of ERβ in different colon cancer cell lines.

Objective
To identify ERβ DNA binding sites in colon cancer cells and to explore ERβ mediated colon cancer-preventive mechanisms.

Methods
Two different colon cancer cell lines with and without transduced ERβ expression were used.

Results

From SW480.ERβ ChIPseq data, We Identified 3200 binding sites for ERβ and confirmed several genes with QPCR.