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Effects of 17?-estradiol on endometrial cancer cell proliferation and HOTAIR expression

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Context: Endometrial cancer is the fourth most commonly diagnosed cancer among women, and the role of estrogen in the maintenance and development of endometrial cancer is well established.HOTAIR is the first-found antisense transcription long chain non coding RNA, which has been found to be highly expressed in many kinds of malignant cancers.
Objective: To investigate the role of 17?-estradiol in regulating HOTAIR gene expression and cell proliferation in Ishikawa cells?
Patients:None
Interventions:Endometrial cancer Ishikawa cells were divided into three groups: estradiol (E2) group, HOTAIR-siRNA+E2 group and control group.
Main Outcome Measures: We measured the expression of HOTAIR ,the expression of PRC2,and the cell proliferation ability.
Methods: Ishikawa cells were hormone-starved then treated or not with 17?-estradiol.HOTAIR expression was measured by qPCR,The role of HOTAIR in cell proliferation was measured following HOTAIR silencing using siRNA.The expression of PRC2(polycomb repressive complex 2), a histone H3 lysine27 (H3K27) specific methyl-transferase complex that interacts with HOTAIR ,was measured by immunoblot analyses.
Result:17?-estradio significantly induced cell proliferation in Ishikawa cells. Accordingly, 17?-estradiol significantly increased HOTAIR mRNA expression in Ishikawa cells compared to untreated cells.17?-estradiol increased PRC2 expression .The siRNA silencing of HOTAIR blocked 17?-estradiol-induced cell proliferation and PRC2 expression.
Conclusion: This study illustrates that estrogen induces HOTAIR expression in endometrial carcinoma cells. These results support HOTAIR as a therapeutic target in endometrial cancer.

[1] Obstetrics and Gynecology, [2] Obstetrics and Gynecology, [3] Obstetrics and Gynecology