

# Investigating the potential role of GnRH receptor type II in endometriosis pathogenesis



Endometriosis is a benign, gynaecological clinical entity characterized by ectopic growth of endometrial glandular and stroma cells, which can be mainly found in the ovaries, ligaments of the uterus, and the peritoneal cavity. The interpretation of endometriosis remains enigmatic, but it seems that more than one mechanisms make the ectopic endometrial cells capable of surviving in the peritoneal cavity. Modern studies are investigating the activity of immune and growth factors that are expressed locally and enhance the installation and maintenance of ectopic endometrial lesions. Hence, recent investigations have confirmed the mRNA expression of GnRH receptor type II in endometrial and ovarian cancer cells, which was found to exert a strong suppressive effect on proliferation of cancer cells. However, the expression of GnRH receptor type II on protein level has been studied in endometrial tissue, and in particular to eutopic and ectopic endometrium in bladder, or in the peritoneum, and the clinical significance of this is probably localized expression in these women than remains to be clarified. The purpose of this study is to investigate the expression pattern of GnRH-II receptor at mRNA and protein in pathological tissues, in normal

endometrium and peritoneum of those women who will undergo elective invasive treatment and the correlation of receptor's expression with clinical indicators of donor women. The apparent positive effect of GnRH-II receptor in controlling the growth of endometriosis cells is consistent with the objective of finding independent prognostic indicators and target molecules to achieve effective treatment of endometriosis. In the context of this thesis the presence and expression of GnRH- II receptor in the endometrium, peritoneum, endometriomas and biochemical condition was demonstrated. In further comparison of the presence between normal and abnormal peritoneum, a statistically significant presence was seen in the latter ( $p<0.05$ ). The data suggest that cell proliferation is increased in endometrial cells of women with endometriosis, suggesting that increased proliferative capacity may increase the likelihood of regressing endometrial cells implanting and surviving in the peritoneal cavity (Wingfield et al, 1995). The present study showed that GnRH II expression was increased in the ectopic endometrium of women with endometriosis. In light of the antiproliferative effect of GnRH II on endometrial cells, these findings may

be consistent with the view that endometrial cells in women with endometriosis have increased proliferative activity. This study is of particular scientific interest in the context of molecular medicine, aspiring that the findings of our research will lead to the proposal of a more effective treatment of endometriosis.



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