POSITIVE CORRELATION BETWEEN NGF, TRKA OR P75NTR LEVELS AND DISEASE STAGES IN EUTOPIC ENDOMETRIUM FROM ENDOMETRIOSIS WOMEN (FONDECYT 1120074)

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ABSTRACT.
Context: Endometriosis is an invasive disease highly associated with chronic pain.
Objective: To study the mRNA and protein of NGF and its receptors TRKA and p75 in eutopic endometria from women with endometriosis and their correlation with pain and endometriosis stages.
Design and Patients: Experimental study approved by Ethical Institutional Committees. The subjects, who donated eutopic endometrium after signing written informed consent, were women going to surgery for endometriosis diagnosis [Patients, n=70, Stages I-II:52% and III-IV:48% (ASRM), pain (VAS 2-10)] or for tubal sterilization or hysterectomy for no endometrial reasons (Control, n=46).
Methods: In endometria throughout menstrual cycle, NGF, TRKA and p75 mRNA (qRT-PCR real time) and protein immunohistochemistry (integrated-optical-density analysis) were studied. Statistical analysis: Anova/Tukey, t-test and Pearson tests.
Results: NGF, TRKA and p75 immunolocalized in endometrial gland and stromal cytoplasm in both groups of endometria. In eutopic endometria, the TrkA mRNA increased in control (3.5-folds) and patients (21-folds) and the NGF mRNA during mid secretory phase only in patients (60%) versus proliferative phase; the mRNA levels of each genes was 155% and 72% higher in patients than control endometria; p75NTR mRNA was similarly decreased from proliferative to secretory phase in both group of endometria. Positive correlation (p<0.05) was observed between NGF and p75 mRNA levels in endometriosis I-II and III-IV stages, and between NGF and TRKA only in the III-IV stages; no correlation was obtained between pain (VAS) and NGF, TrkA or p75NTR.
Conclusions: The high NGF and TRKA mRNA levels in eutopic endometria from endometriosis women and the positive correlation between NGF and p75 or TRKA depending on disease stages suggest that these molecules are involved in progression of the disease.

INSTITUTE.