Is the endometrium of women with endometriosis different?
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At present, the only way to conclusively diagnose endometriosis is laparoscopic inspection, preferably with histological confirmation. This contributes to the delay in the diagnosis of endometriosis which is 6-11 years. So far non-invasive diagnostic approaches such as ultrasound, MRI or blood tests do not have sufficient diagnostic power. In a clinical practice dealing with women with subfertility with or without pain, a non-invasive test of endometriosis with high sensitivity would allow the identification of those women with endometriosis who could benefit from laparoscopic surgery reported to improve these symptoms, ie increase fertility and decrease pain (D’Hooghe et al., 2003). As endometriosis can be progressive in up to 50% of women (D’Hooghe and Debrock, 2002), early noninvasive diagnosis has the potential to offer early treatment and prevent progression. Such a test would be useful especially in women with endometriosis which is not diagnosed by transvaginal ultrasound.

In this lecture, a review will be given on the difference in endometrial biology between women with and without endometriosis using a classical approach (hypothesis driven research investigating predefined biological compounds) and a "systems biology" approach using RNA microarray and proteomic approaches. There is considerable evidence that, when compared to eutopic endometrium from women with endometriosis, when compared with controls, is marked by increased expression of inflammatory cytokines (decreased expression of IL-1 R type II, increased expression of IL-8), of COX-2 and oxidative stress markers, of auto-antibodies like endometrial IgG, of aromatase, of tissue remodeling molecules like urokinase and metalloproteinase 2, 3 and 9, of angiogenic compounds like VEGF and angiopoetin, of glycoproteins like CA125 (only during menstruation) and of markers showing inhibition of apoptosis (TUNEL assay) and promotion of proliferation (Bcl-2, Ki67, telomerase activity, PCNA-1, Pak-1 and Phosphorylated ERK1/2) (May et al, xxxx).

The quality of these data will be discussed with respect to pathogenesis, to endometriosis-associated infertility, and to the semi-invasive diagnosis of endometriosis based on proteomic or mRNA endometrial analysis, or the presence of endometrial nerve fibers (Bokor et al, 2009; Kyama et al, 2010; Fassbender et al, 2012), taking into account recent recommendations for endometrial research (Altmäe et al, in press) and for biomarker development in endometriosis (Fassbender et al, 2013).

Furthermore, the question will be addressed if endometrial biological differences between women with and without endometriosis are caused by endometriosis, or do cause endometriosis. o make real progress, international agreement on biobank development is needed for standard operating procedures for the collection, treatment, storage, and analysis of tissue samples and for detailed clinical phenotyping of these samples. Furthermore, it is necessary to validate the diagnostic accuracy of any promising test prospectively in an independent symptomatic patient population with subfertility and/or pain without clear ultrasound evidence of endometriosis and with a clinical indication for surgery, divided into cases with laparoscopically and histologically confirmed endometriosis and controls with laparoscopically confirmed absence of endometriosis.

References:


