Background: In women, the androgen receptor (AR) is expressed in normal endometrium, with expression increased in epithelial cells in response to anti-progestins/progesterone withdrawal. We have demonstrated that androgen-dependent changes in gene expression can alter stromal cell proliferation, migration, and survival. Endometriosis is a hormone-dependent inflammatory disorder associated with chronic pelvic pain, characterised by growth of tissue derived from the endometrium in extra-uterine sites.

Hypothesis: Expression of AR in endometriotic lesions would render them a potential target for hormonal therapies aimed at reducing cell proliferation.

Materials and Methods: A mouse model of endometriosis was established; mice were treated with steroid hormones and given a single injection of BrdU prior to culling. Tissues (uterus, peritoneum, lesions) were recovered and either fixed or used for extraction of RNA/protein. Expression levels of AR and oestrogen receptor ? (ER?) as well as a marker of cellular proliferation (PCNA) were compared between normal uterus throughout the oestrous cycle and endometriotic lesions. Expression of androgen-regulated genes in uterus and lesions was also investigated.

Results and Conclusions: In the normal mouse uterus, AR was immunolocalised almost exclusively to stromal fibroblasts; ER? was most intense in the luminal and glandular epithelium and the same cells were actively proliferating. Expression of AR in lesions from the endometriosis mouse model was strikingly different to that in the normal uterus, with positive cell nuclei present in the epithelial compartment; results paralleled those in patient samples. Finally, there was no co-localization between AR and PCNA in endometriotic epithelial cells, highlighting the role of AR as an anti-proliferative mediator.