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Effects of prenatal exposure to xenobiotic estrogen and the development of endometriosis in adulthood

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Endometriosis is an estrogen-dependent disease that affects millions of women worldwide, causing pain and infertility. While it is known that retrograde menstruation places endometrial tissue in the peritoneal cavity, it is unclear why it invades and proliferates in women with endometriosis. Studies have shown that other hormone-dependent diseases have a fetal basis (e.g. breast cancer), suggesting that the presence of different hormones before birth may alter the incidence of endometriosis in adulthood. For example, women whose mothers took the synthetic estrogen diethylstilbestrol (DES) during pregnancy had an eighty percent increased incidence of endometriosis. Thus, our hypothesis is that prenatal exposure to xenobiotic estrogen will increase the severity of endometriosis in adulthood in a mouse model of surgically-induced endometriosis. To test this hypothesis, mice were time mated and dosed with vehicle control, 100 ng/kg DES or 10,000 ng/kg DES from days 11-17 of gestation. Surgical induction of endometriosis was performed in adulthood by autotransplantation of one uterine horn. The horn was removed, opened, divided into three pieces, and sutured to the arterial cascade of the intestinal mesentery. The implants became vascularized and formed endometriotic lesions. The mice were then collected at 2 or 4 weeks post-surgery, and the following endpoints were measured: 1) uterine weight; 2) implant size; and 3) implant weight. Additionally, implants were set aside for further analysis of 1) histology; 2) estrogen receptor indicator reporter gene activity; and 3) endometriosis-related gene expression. At the conclusion of this ongoing study, we expect to show whether there is an estrogen-mediated fetal component to endometriosis.