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

Endometriosis is an estrogen-dependent disease that affects 5% to 10% of all women, causing pain and infertility. This disease occurs when uterine tissue grows outside of the uterus in the body cavity. It is unknown what causes this tissue to grow outside of the uterus in some women and not in others. Studies have shown that other hormone-dependent diseases may be related to prenatal exposure to environmental chemicals, 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 80% increased incidence of endometriosis. While pregnant women no longer take DES, there are compounds present in our environment that mimic estrogen action, and they can disturb the delicate balance of natural hormones in the body. These compounds are called endocrine disruptors, and they can be found in plastics, soy products, and some pesticides. The danger is that exposure to these environmental estrogens could increase the chance of developing estrogen-dependent diseases such as endometriosis. Thus, our hypothesis is that prenatal exposure to environmental estrogen increases the severity of endometriosis in adulthood. To test this hypothesis, pregnant mice were dosed with either DES or oil (control). The offspring were then grown to adulthood. Endometriosis was surgically induced, and the mice were collected four weeks later to measure the severity of the disease. Developmental DES exacerbated endometriosis in this model: lesions from mice that were exposed to DES were twice as large as lesions from the control. We also found that developmental DES exposure permanently decreased the expression of TIMP-1, a gene known to inhibit endometriotic growth. At the conclusion of this ongoing study, we expect to 1) demonstrate an estrogen-mediated fetal component to endometriosis, 2) identify potential markers of fetal environmental estrogen exposure, and 3) potentially open the door to new therapeutic approaches to treat and prevent endometriosis.