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Funding Source: Life Sciences Undergraduate Research Opportunity Program

Neonatal exposure to xenobiotic estrogen alters the adult immune response and exacerbates endometriosis in mice

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Endometriosis is a common medical condition affecting 5-10% of women worldwide, and results in severe cramps, pelvic pain, and infertility. The cause of the disease is still unknown. Endometriosis occurs when endometrial tissue, which escapes into the peritoneal cavity via retrograde menstruation, adheres to other tissues in the cavity and causes irritated, inflamed lesions. Studies have suggested that the risk of developing endometriosis increases in women who have been exposed to xenobiotic (foreign to the body) estrogens during developmental stages of life. Thus, it is our hypothesis that programming of the immune system by xenoestrogens during development could potentially exacerbate endometriosis. This could occur by altering the peritoneal environment and/or the invading endometrial tissue. Therefore, it is our goal to study the effects of neonatal xenoestrogen exposure on the immune system; and ultimately, on the establishment of endometriosis in adulthood. In order to study this response, we dosed two strains of mice (CD1 and C57) with xenobiotic estrogens on postnatal days 2-14. In experiment A, CD1 mice were dosed with vehicle control (corn oil), 20 µg/kg/day, or 200 µg/kg/day bisphenol A. In experiment B, C57 mice were dosed with a vehicle control (corn oil) or 0.1 µg/kg/day diethylstilbestrol. At 8 weeks of age, endometriosis was induced in each strain via both a surgical induction and an injection technique. At 12 weeks, the endometriotic implants were counted and weighed to determine which mice had a greater susceptibility to the condition. Our next objective will be to analyze peritoneal fluid from the treated mice to identify key immune functions (for example, the release of certain cytokines) that may have been programmed by developmental xenoestrogen exposure.