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Neonatal exposure to xenobiotic estrogen may alter the adult immune response and exacerbate endometriosis in mice

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Endometriosis is a common medical condition affecting 5-10% of women worldwide and often results in severe cramps, pelvic pain, and infertility. The condition occurs when endometrial tissue, which escapes into the peritoneal cavity via retrograde menstruation, adheres to peritoneal cavity tissues 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 development. This could be due to developmental programming of the peritoneal environment, and specifically, an altered immune function within this environment. Therefore, it is our hypothesis that developmental programming by xenoestrogens alters the immune response to shed endometrial tissue and exacerbates endometriosis. To better understand the role of xenoestrogens in immune programming, we are conducting our studies using a mouse model of surgically induced endometriosis. In particular, we are concentrating on two major aspects of immunity: 1) the presence of immune cells and 2) the function of those cells. Our study of the former is being largely performed using methods of immunohistochemistry (IHC). IHC allows us to quantify the macrophages present in the peritoneal fluid of experimental mice (exposed to diethylstilbestrol) versus control mice (no xenoestrogen exposure). In order to study our second focus, immune cell function, we are using a cytokine antibody array to determine the relative cytokine concentrations in the peritoneal fluid samples. By identifying the degree to which certain cytokine concentrations differ, we hope to better understand the effect of xenoestrogen exposure on immune cell function.