Numerous chemicals, both natural and manufactured, may act as disruptors of endocrine and immune system during critical periods of in utero development that could potentially manifests its effects by compromising adult reproductive health. It is estimated that more than 80,000 chemicals have been released into the environment within the United States alone over the past few decades and current regulations do not require prospective risk assessment of these multiple, potentially interactive chemicals on human health. Thus, this represents a significant challenge to the study the impact of environmental toxicants on the reproductive system. Endometriosis, the presence of endometrial glands and stroma outside the uterus, remains one of the most poorly understood conditions affecting not only women’s reproductive potential, but their overall quality of life. In 1993, Rier et al. reported the presence of laparoscopically documented endometriosis in a colony of rhesus monkeys 10 years after termination of a 4- year period of exposure to dioxin. In animal models, postnatal exposure to dioxin or dioxin-like compounds has been associated with reduced fertility and endometriosis in females although in spite of suggestive population based studies, solid evidence to support the hypothesis that dioxin exposure may lead to the development of endometriosis in women is lacking. Mechanisms through which EDC’s may modify endometrial physiology remain uncertain although studies have found that the endometrial phenotype of mice exposed to a dioxin derivative is markedly similar to that of women with endometriosis. In addition, modulation of the endometrial endocrine immune interface could mechanismistically link toxicant exposure to the development of the disease, and experimental evidence from rhesus monkeys shows that dioxin-exposed animals with endometriosis show long-term alterations in immunity associated with elevated levels of dioxin. Thus, dioxins and other EDC’s can mimic the endometriosis phenotype by inducing the development of an endometrium that exhibits both reduced progesterone responsiveness and hypersensitivity to proinflammatory stimuli. My presentation will focus of the utilization of a baboon model to study endometriosis and the mechanisms associated with the development of progesterone resistance and the impact of BPA exposure in-utero in a mouse model that impacts on uterine function and disruption of the decidualization response.