Atopy is the genetic predisposition to develop IgE-mediated allergic responses. The abnormal reaction of the sensitized person's immune system against normally harmless environmental triggers or allergens results in allergic diseases such as allergic rhinitis, allergic asthma, atopic dermatitis, and food allergy.

Cytokines are immune regulatory molecules that can direct the nature of one's immune response. These are produced by CD4+ T Helper cells which are classified into Type 1 T Helper cells (TH1) or Type 2 T Helper cells (TH2) cells depending on their cytokine profile. TH1 cells produce interferon γ (IFN-γ), interleukin 2 (IL-2), and tumor necrosis factor (TNF) which promote cell-mediated immunity or delayed-type hypersensitivity and pro-inflammatory responses. TH1 dominated immune response is involved in organ-specific autoimmune disorder, acute allograft rejection, and unexplained recurrent abortion. In contrast, TH2 cells produce interleukins (IL-4, IL-5, IL-6, IL-9, IL-10, and IL13) which direct allergic or anti-inflammatory responses. The generation of TH2 cytokines has a crucial role in allograft tolerance and maintenance of normal pregnancy. This means that atopic or allergic diseases and normal pregnancy are both characterized by TH2 dominance. However, reports showed that TH2 dominance has been documented also in patients with recurrent pregnancy loss. Studies have shown that allergic women have more irregular menstruation, longer time to pregnancy, and reduced fertility. Hence, this lecture aims to present and understand the immunology of infertility, pregnancy losses, and atopy and to preclude pregnancy. In this talk, we will review the immunopathology in a non-human primate model from GBS and ZIKV perinatal infections. We will emphasize the correlation of key aspects of immunopathology with pregnancy outcome and fetal injury. Finally, we will present a few features of SARS-CoV-2 immunopathology in pregnancy.

Endometriosis is a common, estrogen-dependent, inflammatory disorder wherein endometrial-like tissue is found outside the uterus, mainly in the pelvis. While its pathogenesis is not well understood, genetics and environment contribute nearly equally to disease risk. Pelvic disease largely derives from retrograde transplantation of menstrual blood and endometrial cells that invade the mesothelium and elicit neo-neuroangiogenesis and an inflammatory response, resulting in pelvic pain, fibrosis, and infertility. While ~95% of women have some retrograde menstrual reflux, ~12% develop endometriosis, suggesting refluxed tissue avoids phagocytosis by peritoneal macrophages. Recent data demonstrate that peripheral and endometrial immune components differ in women with and without disease and the eutopic endometrium (lining the uterus) displays a pro-inflammatory phenotype. This, as well as low-grade systemic inflammation, likely contribute to endometrial-based infertility and poor pregnancy outcomes in affected women. Most cells of the innate and adaptive immune system are present in different amounts in endometrium and have altered cycle dependence and/or activation status in women with versus without disease, although data are sometimes conflicting. These cells, along with their secreted cytokines and chemokines contribute to the pro-inflammatory endometrial environment. Non-immune endometrial cells also display abnormalities in endometriosis women, including epithelial and mesenchymal lineages. Endometrial fibroblast differentiation to the decidual cell (“decidualization”), critical for pregnancy success, is regulated by progesterone, and in women with endometriosis the decidualization response is blunted and this cell type itself displays a pro-inflammatory phenotype. While progesterone “resistance” is