Immune mechanisms of hypertension. Chronic inflammation and immune system dysfunction play an important role in the development of hypertension and cardiovascular disease. The kidneys are critical to the long term control of blood pressure and numerous studies show that inflammatory markers and renal immune cell infiltration are elevated in both humans with hypertension and in experimental animal models. The inflammatory mediators can alter kidney function and promote the development of hypertension. In order to understand how immunological changes contribute to the pathogenesis of hypertension, we utilize a mouse model with the chronic autoimmune inflammatory disorder, systemic lupus erythematosus (SLE). SLE is an ideal experiment model to examine the link between inflammation, the kidneys, and hypertension because its origins are rooted in immune system dysfunction and patients with SLE have prevalent hypertension and renal disease. Current studies in the laboratory are focused on the role of autoantibodies and various immune cell subsets in the pathogenesis of hypertension, and their impact on renal hemodynamic function.

Pregnancy and vascular function. Preeclampsia is a pregnancy specific syndrome that is defined as new onset hypertension after 20 weeks gestation with proteinuria, or any of several other characteristics including impaired liver function, renal insufficiency, thrombocytopenia, or impaired cerebral or visual symptoms. Patients with preeclampsia have increased risk for developing stroke, cerebral edema and seizure by mechanisms that remain unclear. We recently demonstrated that an experimental model of preeclampsia exhibits impaired cerebral vascular tone, blood flow autoregulation, and cerebral edema. Ongoing work in the laboratory is exploring how placental factors released during a preeclamptic pregnancy impact cerebral vascular function.