Summary of research

The prevalence of diabetes has increased dramatically in the US in the past several decades. Hypertension occurs twice as frequently in diabetic subjects as in the general population. The coexistence of diabetes and hypertension substantially increases the risk for onset and progression of chronic kidney disease. It is an ongoing target for scientists to identify the mechanisms and therapeutic methods responsible for diabetic-hypertensive nephropathy. TRPC6 is a member of the TRP superfamily, a group of non-selective cation channels, expressed widely in the kidney cells. In this project, we aim to understand 1) whether hyperglycemia in type 1 diabetes and moderately increased blood pressure synergistically promote kidney injury. Aorta constriction between the renal arteries will be used to induce hypertension in hyperglycemic and euglycemic mice. 2) Whether TRPC6 has a role in mediating the kidney injury induced by the combination of diabetes and hypertension. Global TRPC6 knockout mice and wild-type mice will be used to address this question. 3) The TRPC6 KO mice showed increased body weight when we initially set up the colony. To further demonstrate its role in obesity and metabolic function, we will measure their daily food intake, fasting/refeeding response, oral glucose tolerance, blood pressure, heart rate, energy expenditure, and motor activity, as well as the anorexic responses to leptin and melanocortin 4 receptor activation, and related gene expression.