Hypertension is the leading risk factor for morbidity and mortality globally. Reabsorption of sodium by the kidney plays a key role in determining blood pressure, and is the research focus of the McCormick lab. Sodium transporters including NKCC2 and NCC are activated following phosphorylation by the kinases SPAK/OSR1. SPAK/OSR1 are themselves activated by WNK kinases, which when mutated cause the disease Familial Hyperkalemic Hypertension (FHHt). FHHt is also caused by mutations in Cullin 3 and KLHL3, components of a Ubiquitin ligase complex that modulates WNK kinase abundance. Currently, our major goal is to understand the mechanisms by which mutant Cullin 3 causes FHHt, and the roles of Cullin 3 in normal renal physiology. Other interests include regulation of potassium homeostasis by the WNK-SPAK/OSR1-NCC pathway, and the role of Cullin 3 in Chronic Kidney Disease.