Summary of Research

Metabolic syndrome is thought to affect almost 1 in 3 people in the United States. Risk factors that define metabolic syndrome include hypertension, obesity, and insulin resistance. High dietary sodium intake increases the risk for hypertension and has recently been suggested to be a risk factor for adiposity and insulin resistance in adults, independent of caloric intake. While these correlations are striking, it is still unknown if high salt intake directly promotes fat deposition and insulin resistance, and if so, what mechanisms mediate this link.

Current research in our lab aims to determine if endothelin-1 (ET-1), a potent vasoconstrictor that is elevated by increasing salt intake, promotes facets of metabolic syndrome, such as obesity and insulin resistance. ET-1 exerts its physiological actions via two receptor subtypes, ET_A and ET_B. Both receptors are expressed in fat storing adipocytes, and both have profound effects on lipid metabolism and insulin signaling by adipocytes. ET_A receptor activation promotes the breakdown of lipids by adipocytes, while the ET_B receptor promotes insulin resistance by inhibiting the effects of insulin on adipocytes. We are currently using rodents to “knock-in” or “knock-out” ET-1 receptors in adipocytes to determine the physiological impact. The long-term goal of these studies is to develop a viable treatment strategy for use in patients with metabolic syndrome.