Sodium glucose cotransporter-2 (SGLT-2) inhibitors are a recent new category of drug prescribed for the treatment of hyperglycemia in patients living with type 2 diabetes. These drugs are currently undergoing clinical trials examining drug efficacy and safety for the treatment of type 1 diabetes (T1D). Recently, studies have begun to investigate the effects of SGLT-2 inhibitors on their potential for augmenting glucagon secretion from the alpha cells of the pancreas in healthy and diabetic animal models and humans. Increases in glucagon secretion may have protective effects when hypoglycemia develops during exercise, or with insulin overtreatment, but may also increase the potential for ketone production. This project aims to evaluate the interaction of empagliflozin, a commonly prescribed SGLT-2 inhibitor for patients with type 2 diabetes, with insulin-induced hypoglycemia and with exercise in a rat model of type 1 diabetes. It is hypothesized that the SGLT-2 inhibitor empagliflozin will increase the secretion of glucagon, thereby reducing the fall in blood sugars seen during exercise and insulin treatment. Insulin treated Wistar rats with streptozotocin-induced diabetes were divided into 4 groups: i) inactive with placebo; ii) inactive with drug; iii) active with placebo; and iv) active with drug. Rats were given 8 days of placebo or SGLT-2 inhibitor treatment twice daily, with the active groups receiving 4 hours/day of voluntary running wheel access. At the end of this treatment period, all rats underwent an insulin-induced hypoglycemia challenge. Our preliminary findings to date suggest that SGLT-2 inhibition may reduce a fall in blood glucose levels during exercise by improving the glucagon response. SGLT-2 inhibition does not appear to reduce the likelihood of hypoglycemia during insulin overtreatment. Future studies will aim to confirm our preliminary findings that SGLT-2 inhibition improves glucagon counterregulation during exercise in rats with type 1 diabetes and will examine potential sex differences in blood glucose response to exercise and SGLT-2 inhibition.