Type 2 diabetes (T2D) is a worldwide problem, and people with T2D have impaired postprandial glycemia (PPG). While current pharmaceutical therapies are targeted to reduce glycosylated hemoglobin and may not be effective in improving PPG, exercise is an effective treatment to enhance postprandial glycemic control in patients with T2D. As little as seven days of aerobic exercise has been shown to reduce PPG, but the mechanisms by which this occurs are not understood. Further, the assessment of PPG generally is done using a non-physiological glucose overload uncharacteristic of normal human feeding. Thus, the primary aims of this dissertation were: 1) to identify tissues contributing to changes in glycemic control after short-term exercise training and identify the systemic mechanisms by which exercise improves overall PPG in patients with T2D, and 2) to determine if a mixed meal tolerance test is a more valid tool for assessing improvements in glycemic control following exercise training than the standard oral glucose tolerance test in T2D. Our findings support that improved insulin sensitivity is an early adaptation of exercise training, but we did not see improvements in overall PPG in the sample studied. We also found that a mixed meal test is an effective alternative to the oral glucose tolerance test in assessing differences in PPG. Collectively, when viewed in the presence of the existing literature, these data suggest that while exercise training is ultimately known to enhance postprandial glycemic control, and testing this with a mixed meal test is a viable alternative to the current oral glucose tolerance test, subjects with T2D have variable responses and may not always see improvements in PPG following one week of aerobic exercise training.