Gene Expression levels of Zfp407 and Zbtb9 in Relation to Glut4 During Fasting and Refeeding in C57BL/6J Male Mice

Type 2 diabetes affects over 300 million people worldwide. Initially, insulin resistance leads to decreased glucose uptake by peripheral metabolic organs. This decrease in uptake of glucose is in part due to the impaired function of Glucose transporter (Glut)-4, which is an insulin-dependent glucose transporter that primarily functions in muscle and fat cells. Previous studies demonstrated that Glut4 expression is regulated by food intake, but that this affect is blunted by obesity. We previously demonstrated that zinc finger protein 407 (Zfp407) controls Glut4 expression together with its interacting protein Zinc finger BTB domain containing 9 (Zbtb9). Therefore, we hypothesized that expression of Zfp407 and Zbtb9 during fasting and refeeding would correlate with that of Glut4. To test this, and better understand the physiological roles of these genes, we utilized two mouse models of obesity: 1) C57BL/6J male mice fed a control diet compared to a high-fat diet as well as 2) wild type (WT; C57BL/6J) mice compared to those with a genetic mutation in the leptin gene that induces obesity (ob/ob). Mice were subjected to fasting overnight for 16 hours and then a subset were refeed. Adipose tissue was collected at both time points for quantitative polymerase chain reaction analyses (qPCR) to determine gene expression levels. As expected, Glut4 expression patterns in both mouse models were as previously demonstrated, however, we saw no correlation between either Zfp407 or Zbtb9 gene expression and Glut4. This suggests that while these proteins interact and regulate Glut4 expression, regulation of Glut4 during refeeding is not controlled by changes in Zfp407 or Zbtb9 expression levels.