Obesity is a strong risk factor for developing type 2 diabetes (T2DM), but the reasons for this are not fully understood. In particular, it is not known why some obese people develop T2DM while other obese individuals do not. This study tests whether differences in fat cells (adipocytes) are to blame. Adipose tissue is dynamic, expanding to accomodate increased energy intake and contracting to balance energy expediture with anatomic specificity. The Danforth hypothesis suggests that an impairment of the adipose tissue to create new adipocytes (adipogenesis) may underlie the development of insulin resistance and progression to T2DM. One consequence of impaired adipogenesis may be an increase in the average adipocyte cell size as the cells swell with lipid. In addition, large fat cells may secrete hormones and pro-inflammatory cytokines that can increase the risk for developing T2DM.

In order to understand whether adipocyte cell size is associated with insulin sensitivity, subcutaneous abdominal and gluteal adipose tissue biopsies from obese, insulin-resistant diabetic individuals (OIR) and obese, insulin-sensitive non-diabetic individuals (OIS) will be examined. We will determine whether adipocyte diameters differ between the subcutaneous and gluteal adipose depots of OIR and OIS subjects. These data will then be analyzed for correlations with cellular proliferation rates derived from stable isotope labeling, gene expression levels of adipokines and pro-inflammatory cytokines and clinical physiology data. We hypothesize that the OIR group will be characterized by having impaired adipogenesis and large (hypertrophic) adipocytes compared to the OIS group.