Diabetes is a disease characterized by reduced insulin action and secretion, leading to elevated blood glucose. In the 1990s, studies showed that intravenous injection of fatty acids led to a sharp negative response in insulin action that subsided hours after the injection. This dissertation is focused on the formulation of a model built around the known mechanisms of glucose and fatty acid storage and metabolism within myocytes. Data from euglycemic-hyperinsulinemic clamp with fatty acid infusion studies are used to validate the qualitative behavior of the model and estimate parameters. Analysis and numerical simulation of the model demonstrates critical bifurcations implicating nutrient excess as a driver of muscular insulin resistance. Finally, clinical applications of this class of models are discussed. (Received September 26, 2017)