Introduction: Adiponectin is an adipose-derived cytokine which regulates body glucose and fatty acid metabolism. It also exerts cardio-protective effects in the heart, including a decrease in cardiac infarct size following LAD ligation and attenuation of TAC-induced cardiac hypertrophy. We previously reported that lack of Adiponectin Receptor 1 (AdipoR1) impairs cardiac function in non-diabetic animals, caused by ROS-induced mitochondrial uncoupling. A typical trait of diabetic cardiomyopathy observed in models of type 2 diabetes is impaired cardiac efficiency, related to increased myocardial FA utilization. Underlying mechanisms of impaired cardiac efficiency may include FA-induced ROS-mediated activation of mitochondrial uncoupling proteins, and/or oxygen waste for noncontractile processes. Since serum adiponectin levels are decreased in animal models of type 2 diabetes, we hypothesized that impaired cardiac energetics and reduced cardiac efficiency may be related to impaired cardiac adiponectin action.

AdipoRon treatment did not improve cardiac output and efficiency in db/db mice

Conclusions: AdipoRon treatment shifts myocardial substrate preference towards increased glucose utilization, likely by decreasing fatty acid delivery to the heart, but was not sufficient to improve cardiac output and efficiency in db/db mice.