The administration of a mitochondrial antioxidant protects against the cardiac consequences associated with obesity in rats

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Obesity is associated with cardiac alterations in which oxidative stress can play an important role, with the mitochondria being the main source of reactive oxygen species. The aim of this study was to explore the impact of the mitochondrial antioxidant, Mito Q, in the cardiac changes observed in rats fed a high fat diet (35% fat; HFD) for 6 weeks. MitoQ (50 mg/Kg/day) attenuated the increase in body weight and cardiac hypertrophy observed in HFD rats. No significant differences were found in cardiac function among any group. However, HFD rats showed cardiac fibrosis and a reduced cardiac glucose uptake that were attenuated by MitoQ. Interestingly, MitoQ also prevented the increase in the levels of either fatty acid transporters (CD36 and CPT1A) or the enzyme involved in triglyceride synthesis (DGAT1). HFD rats also showed an increase in mitochondrial levels of mitofusin 1, and cyclophilin and a decrease in those of fumarase, which were reversed by MitoQ. In vitro, MitoQ prevent the increase in levels of CPT1A, mitofusin1 and cyclophilin and the reduction in fumarase expression induced by palmitic acid in cardiac myoblasts (H9c2). Our findings suggest that mitochondrial oxidative stress plays an important role in the cardiac alterations associated with obesity.