

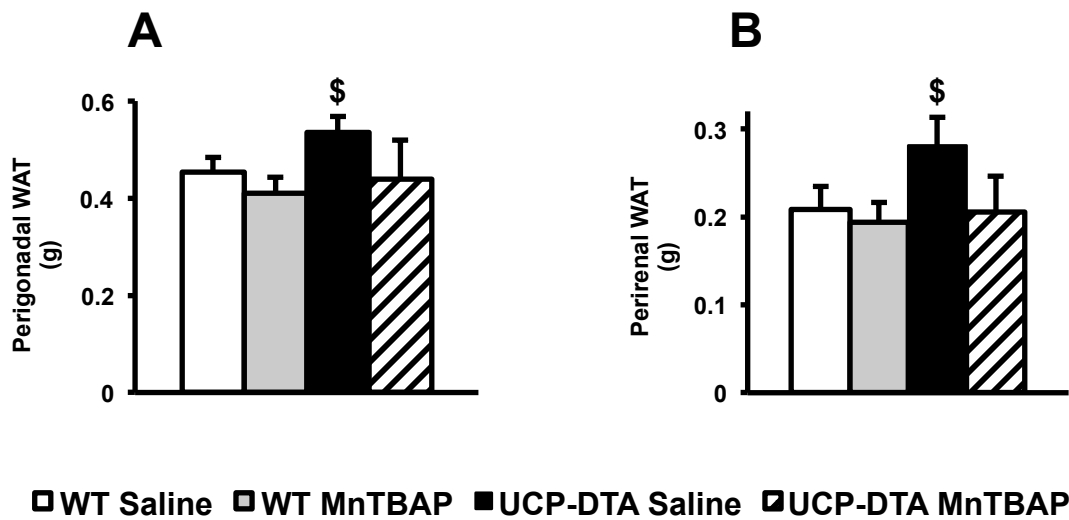
Supplemental Information for

Antioxidant treatment normalizes mitochondrial energetics and myocardial insulin sensitivity independently of changes in systemic metabolic homeostasis in a mouse model of the metabolic syndrome

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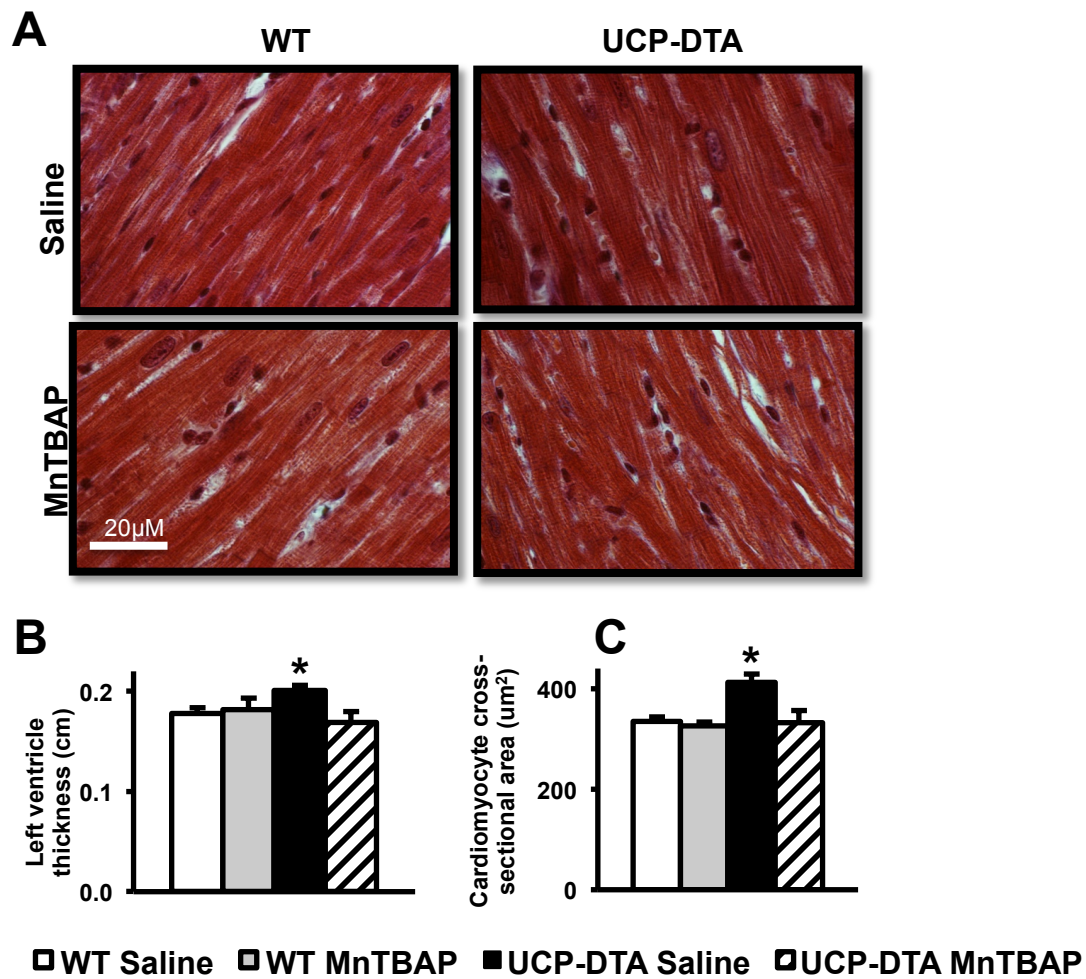
Supplemental figures

Supplemental Figure 1.



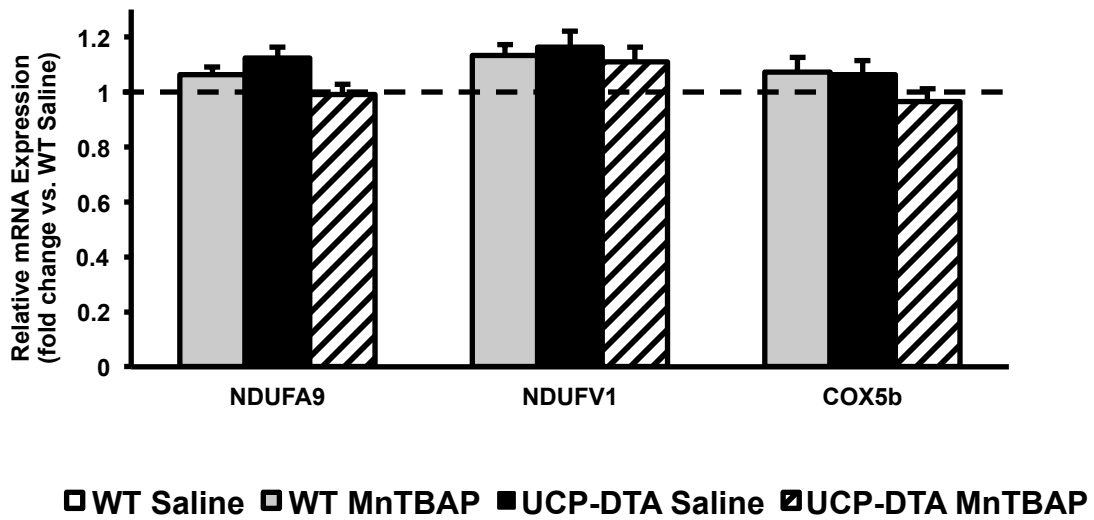
Supplemental Figure 1. Adipose tissue weights in 13 week-old UCP-DTA mice vs. controls. **A, B.** Perigonadal and perirenal fat mass, respectively (n=10-11). \$ p < 0.05 vs. WT. □ Saline-treated control wildtype (WT) mice, □ MnTBAP-treated WT mice, ■ UCP-DTA treated with saline, ▨UCP-DTA treated with MnTBAP.

Supplemental Figure 2.



Supplemental Figure 2. MnTBAP treatment decreases cardiomyocyte hypertrophy in UCP-DTA mice. **A.** Masson trichrome staining of paraffin-embedded left ventricular sections; Bar: 20µm. **B, C.** Stereological quantifications of cardiomyocyte cross-sectional area and left ventricular thickness (n=4-10). *p<0.05 vs. all other groups. □ Saline-treated control wildtype (WT) mice, □ MnTBAP-treated WT mice, ■ UCP-DTA treated with saline, ▨UCP-DTA treated with MnTBAP.

Supplemental Figure 3.



Supplemental Figure 3. Expression of genes encoding mitochondrial OxPhos subunits NDUFV1, NDUFA9, COX5b in the hearts of 24 week-old mice normalized to 16S ribosomal RNA (n=8). Saline-treated control wildtype (WT) mice are shown as dashed line. □ MnTBAP-treated WT mice, ■ UCP-DTA treated with saline, ▨ UCP-DTA treated with MnTBAP.