Supporting Information

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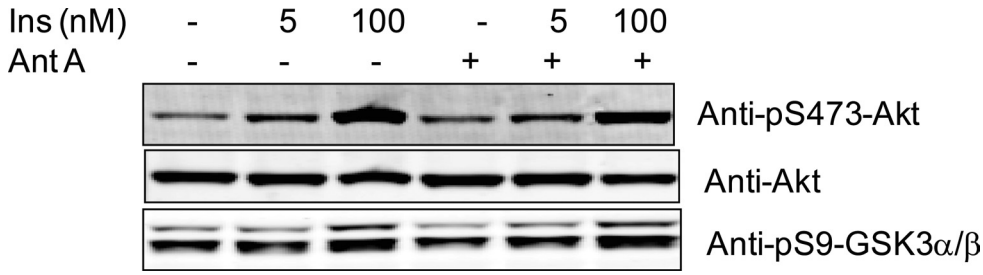

Fig. S1. Superoxide production does not alter maximal or submaximal insulin-stimulated Akt phosphorylation or Akt activity toward its downstream substrate GSK3. L6 myoblasts were treated with 50 nM antimycin for 10 min before insulin stimulation as described in Fig. 3*F*. Representative Western blots of three experiments are shown.

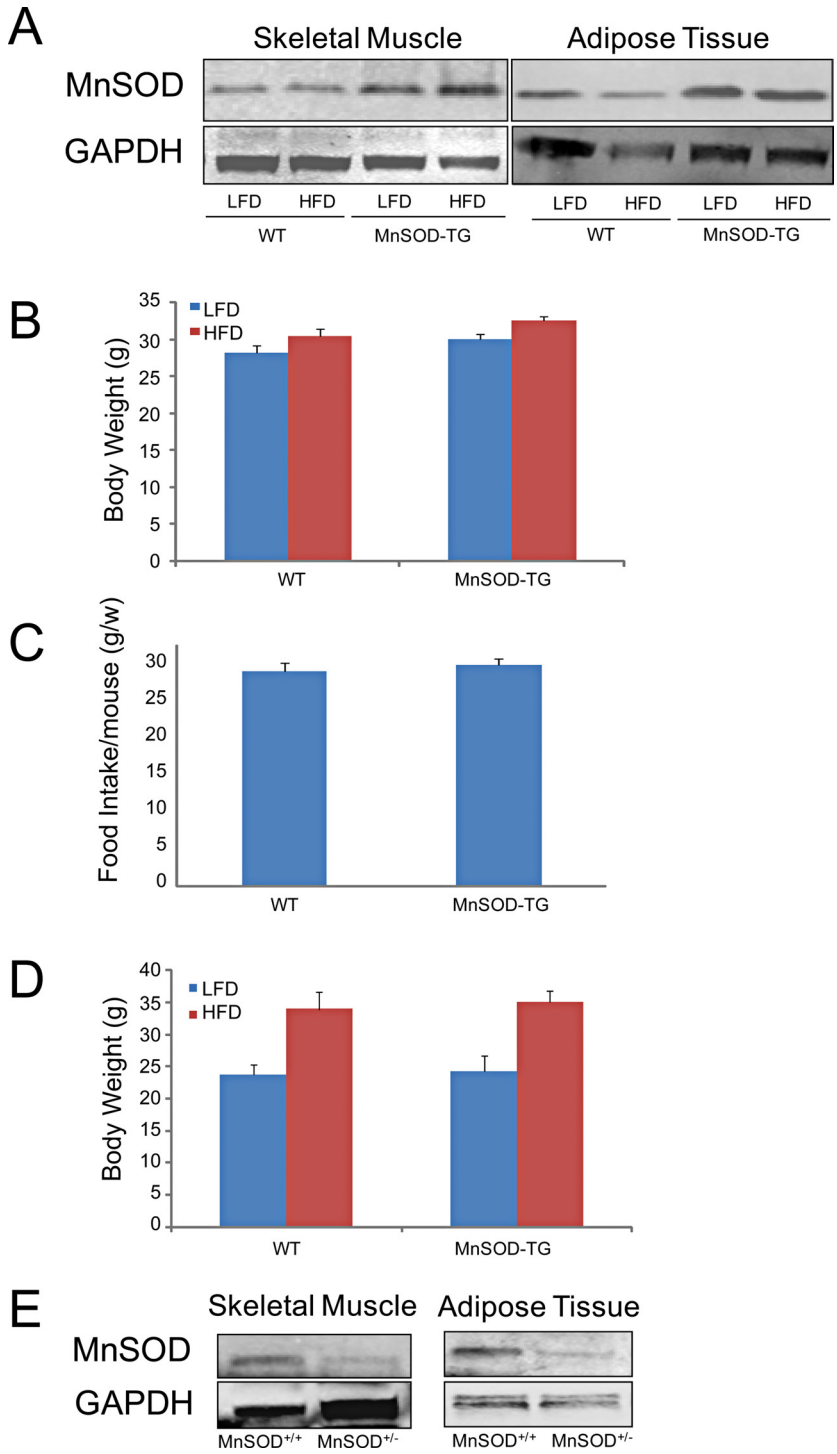

Fig. S2. Supplementary data comparisons between wild-type (WT) and transgenic mice used in this study. (*A*) Mitochondrial superoxide dismutase (MnSOD) expression in skeletal muscle and adipose tissue of WT and MnSOD transgenic mice on a low fat diet (LFD) or high fat diet (HFD). (*B*) Body weight of WT and MnSOD transgenic (MnSOD-TG) mice before and one week after HFD. (*C*) Food intake during the week of high fat feeding in (*B*). (*D*) Body weights of mice used in the long-term (12-week), high-fat feeding experiment. (*E*) MnSOD expression in skeletal muscle and adipose tissue of WT and MnSOD−/+ mice used in this study.

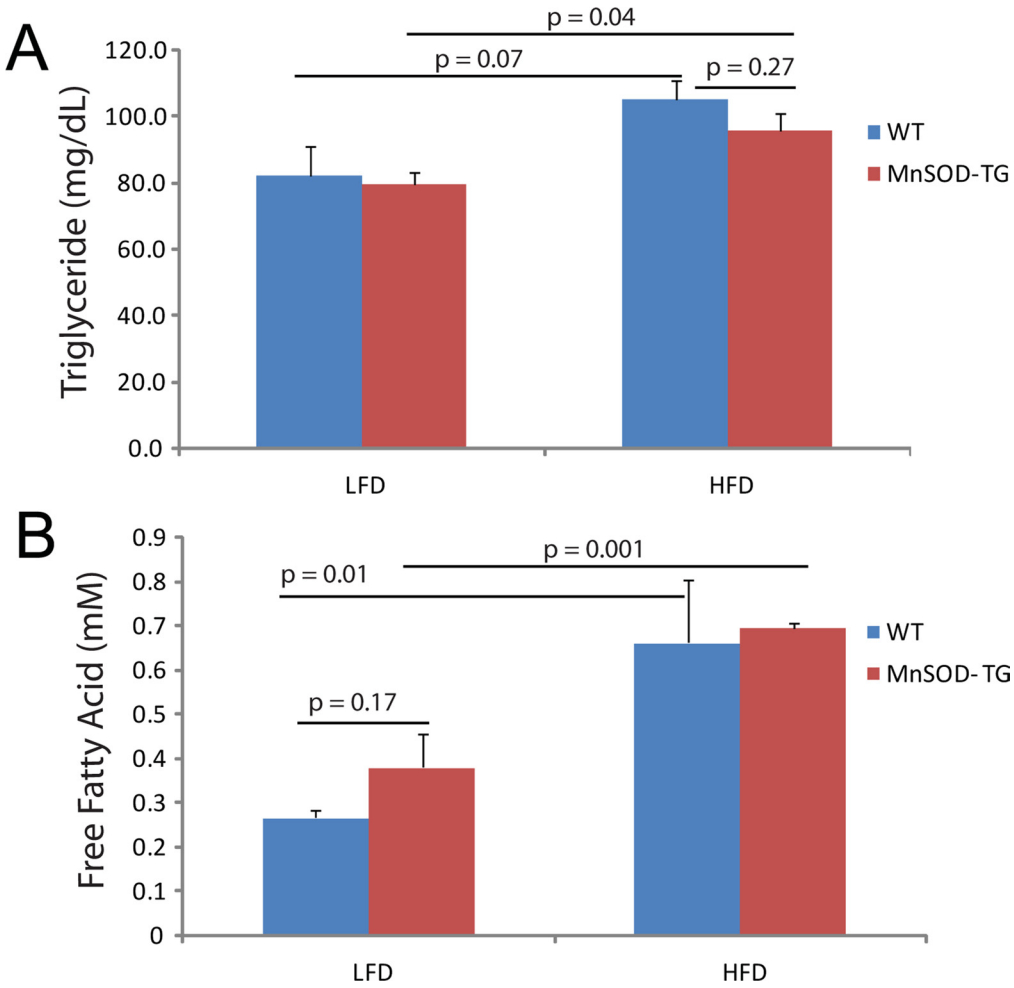

Fig. S3. Blood lipid comparisons between male WT and MnSOD-TG mice used in this study. (*A*) Plasma triglyceride levels were 82.3 ± 9.0 and 79.8 ± 3.3 mg/dL, respectively, for WT and MnSOD-TG fed chow diet and 105.3 ± 5.7 and 95.8 ± 5.3 mM, respectively, for WT and MnSOD-TG fed HFD for 16 weeks. (*B*) Plasma free fatty acid levels were 0.27 ± 0.02 and 0.38 ± 0.07 mM, respectively, for WT and MnSOD-TG fed chow diet and 0.66 ± 0.14 and 0.70 ± 0.01 mM, respectively, for WT and MnSOD-TG fed HFD for 16 weeks. $n = 4–5$ per group.