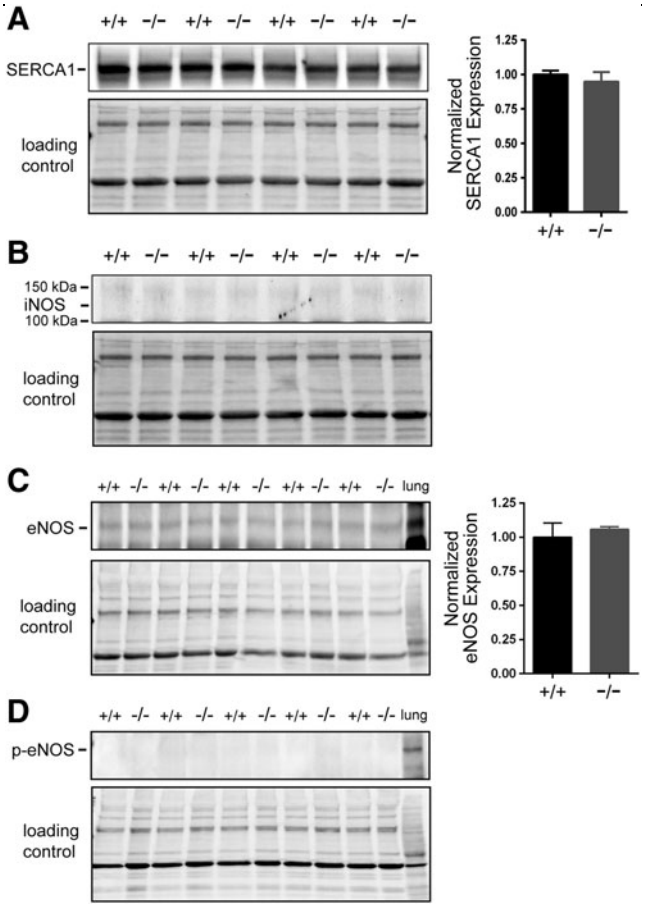

SUPPLEMENTARY FIG. S2. Mitochondrial density in gastrocnemius muscles is unaffected by loss of GSNOR. To test whether enhanced fatigue resistance could result from increased energy-producing capacity through induction of mitochondrial biogenesis, mitochondrial density was measured in gastrocnemius muscles. **(A)** Representative Western blot (*left panel*) showing mitochondrial marker VDAC1 protein expression in wild-type (WT, +/+) and GSNOR$^{-/-}$ muscle and total protein loading control (*right panel*). **(B)** Densitometric quantitation showed that VDAC1 protein expression was similar between wild-type and GSNOR$^{-/-}$ gastrocnemius. **(C)** Representative Western blots (*left panel*) showing expression of one subunit of each of the five multimeric mitochondrial respiratory chain complexes (CI-V) from wild-type and GSNOR$^{-/-}$ gastrocnemius, with protein loading control shown in the *right panel*. Complex subunit expression was similar between WT and GSNOR$^{-/-}$ muscle. **(D)** Densitometric quantitation confirmed that respiratory chain subunit expression did not differ between WT and GSNOR$^{-/-}$ gastrocnemius muscles. These data support findings in the TA (Fig. 5B, C) and argue against mitochondrial biogenesis as a contributing factor to the enhanced fatigue resistance of GSNOR-deficient muscle. $n = 6$ and 5 for WT and GSNOR$^{-/-}$ groups, respectively.