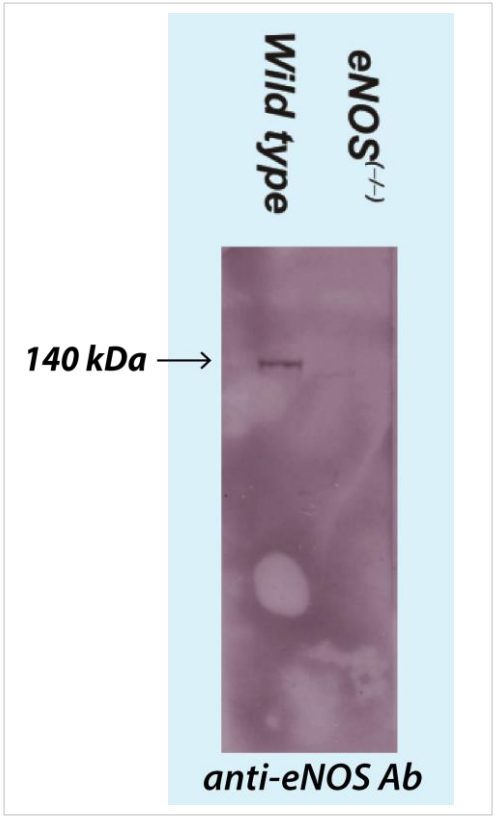
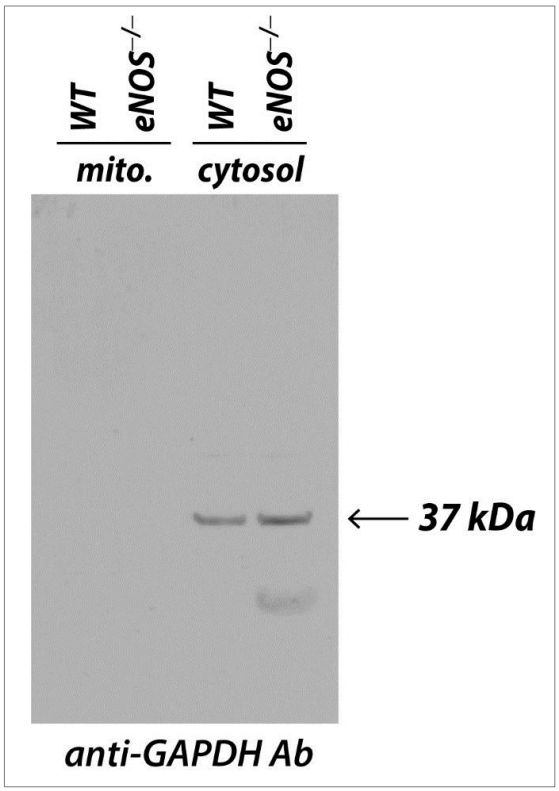
Supplementary Figure 1a. Immunoblotting of eNOS from the tissue homogenates (100 µg were used) of aorta from wild type and eNOS$^{-/-}$ mice using an anti-eNOS polyclonal antibody (1:250, Santa Cruz Biotechnology Inc., Dallas. TX)



Supplementary Figure 1b. Immunoblotting of glyceraldehyde-3-phosphate dehydrogenase (GAPDH) from the mitochondrial preparations (mito., 100 µg loaded) and cytosolic fraction (cytosol, 100 µg loaded) of murine hearts from wild type and eNOS$^{-/-}$ using an anti-GAPDH monoclonal antibody (1:500, Santa Cruz Biotechnology Inc., Dallas. TX)



Supplementary Figure 2. Effect of L-NAME treatment on the mitochondrial function and redox status of the myocardium from wild type mice. Mice were subjected to oral administration of L-NAME (1 mM in the drinking water) for 4 days. The mitochondrial fraction was isolated from the hearts as described in "Experimental Procedures". ***A-C,*** state 2, state 3, state 4, oxygen consumption rates, and respiratory control index of the isolated mitochondria were measured as described in "Material and Methods". ***D,*** the enzymatic activities of the ETC and the aconitase activity were measured as described in "Experimental Procedures". ***E,*** the redox activity of the myocardium was assessed by using EPR to measure the conversion of CM-H to the corresponding stable nitroxide as described in the legend of Fig. 3. ***F,*** the $^{\bullet}O_2^-$ generation by mitochondria at state 3 respiration was measured by EPR spin trapping with DMPO as described in "*Materials and Methods*".

Supplementary Figure 2