

SUPPLEMENTAL MATERIAL

Renovascular hypertension induces myocardial mitochondrial damage, contributing to cardiac injury and dysfunction in pigs with metabolic syndrome

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Figure S1. MetS increases inter-mitochondrial junction (IMJ) number and electron density. A: Electron microscopy images of pig cardiomyocytes and quantification of IMJ density, electron density, and cristae density. *B:* Representative images showing the orientation of cristae at IMJs and quantification of IMJ cristae angles. * $p < 0.05$ vs. Lean+Sham; † $p < 0.05$ vs. MetS+Sham ‡ $p < 0.05$ vs. Lean+RVH.

Figure S2. MetS and RVH do not affect either mitochondrial biogenesis or dynamics. A. Myocardial expression of the mitochondrial biogenesis marker peroxisome proliferator-activated receptor gamma coactivator (PGC)-1 α , the fusion markers mitofusin (MFN)-1 and optic atrophy protein (OPA)-1, and the fission marker phosphorylated dynamin-related protein (pDRP)-1 did not differ among the groups.

Figure S3. Lean+RVH induces post-transcriptional regulation of mitophagy-related genes. A: Diagram of microRNAs targeting the mitophagy genes *BNIP3*, *PINK1*, and *MAP1LC3B* (miRWalk). *B:* Myocardial expression (quantitative-polymerase chain reaction) of ssc-miR-328, ssc-miR-22-3p, ssc-miR-29b, ssc-miR-370, ssc-miR-320, and ssc-miR-490-5p was lower in Lean+RVH compared to all other groups. * $p < 0.05$ vs. Lean+Sham; † $p < 0.05$ vs. MetS+Sham.