Figure S1. EPO decreases thapsigargin-induced lipid accumulation in mouse primary hepatocytes. (A) Hepatic lipid accumulation was visualized and quantified based on Oil Red O staining in Thp-treated cells with or without EPO. Scale bar, 100 $\mu$m. (B) The absorbance of Oil Red O staining was measured at 510 nm with a spectrophotometer. All data are shown as the mean ± standard error of the mean (n=3 independent experiments; $^{*}$P<0.05). EPO, erythropoietin; Thp, thapsigargin.

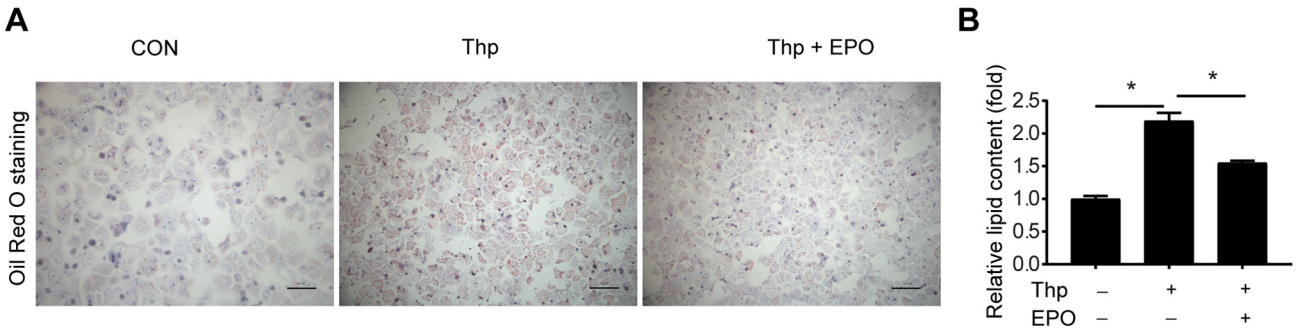

Figure S2. The expression level of SIRT1 was decreased by siRNA-mediated SIRT1 silencing. (A) The mRNA and (B) protein expression levels were determined in primary hepatocytes transfected with siSIRT1 or siCon. Data are presented as the mean ± standard error of the mean (n=3 independent experiments, $^{*}P<0.05$). SIRT1, sirtuin 1; siRNA, small interfering RNA.

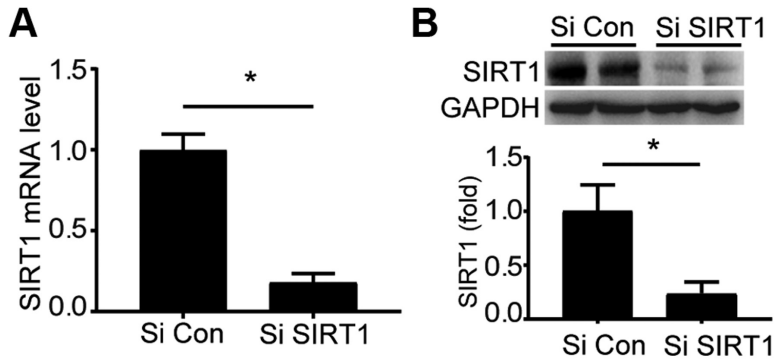

Figure S3. SIRT1 is required for EPO-stimulated hepatic FGF21 expression. (A) Quantification of FGF21 in the media of cultured primary hepatocytes treated with various concentrations of EPO. Data are shown as the mean ± standard error of the mean (n=3 independent experiments, *P<0.05, vs. 0 U/ml). (B) The serum levels of hepatic FGF21 were measured in mice treated with EPO or PBS for 7 or 14 days. (C) The serum levels of FGF21 and (D) the mRNA levels of hepatic PGC-1α were measured in SIRT1-LKO mice and their littermates with and without EPO treatment. Data are shown as the mean ± standard error of the mean (n=6/group, *P<0.05 vs. WT + PBS group). WT, wild-type mice. EPO, erythropoietin; SIRT1, sirtuin 1; FGF21, fibroblast growth factor 21; PGC-1α, peroxisome proliferator-activated receptor-γ coactivator α; SIRT1-LKO, hepatocyte-specific SIRT1-deleted.