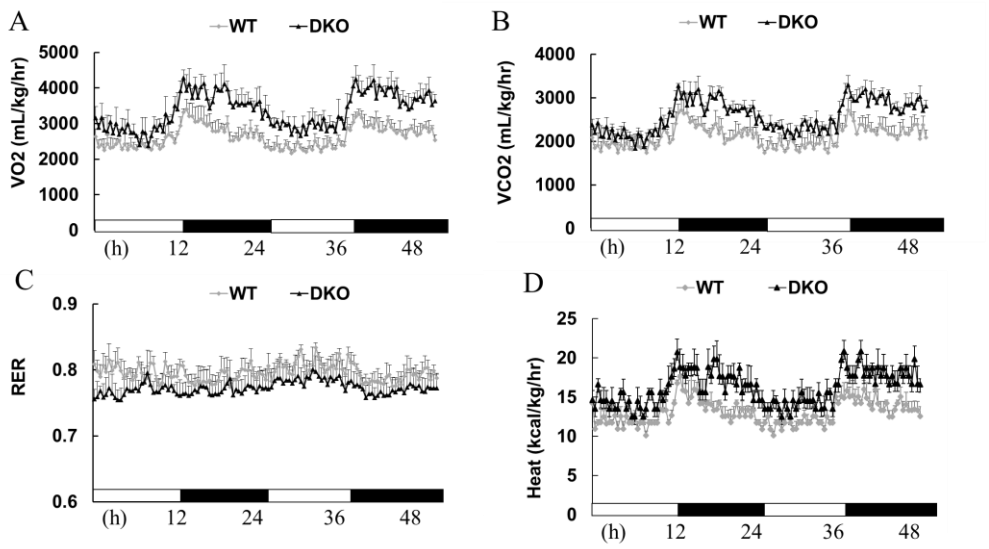
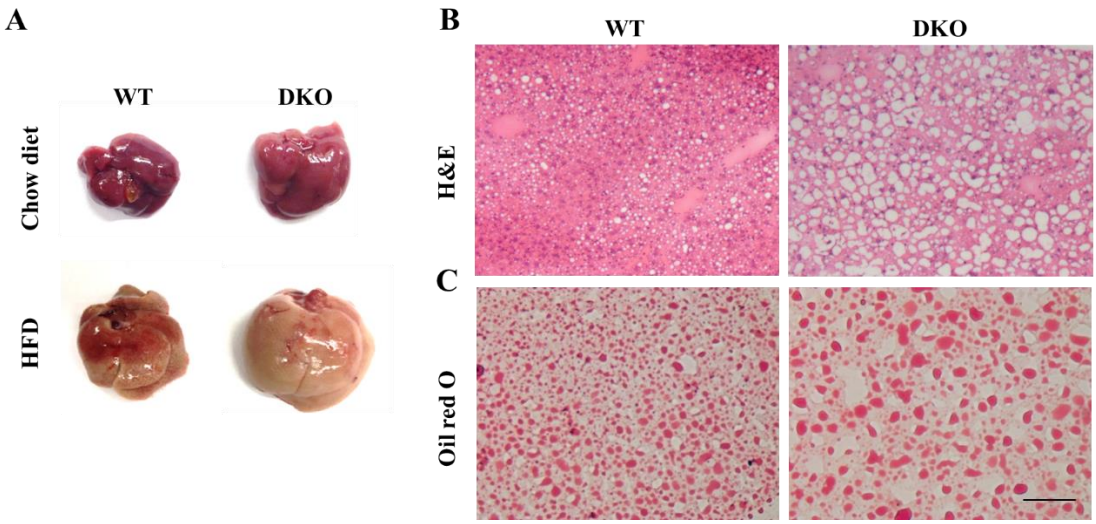
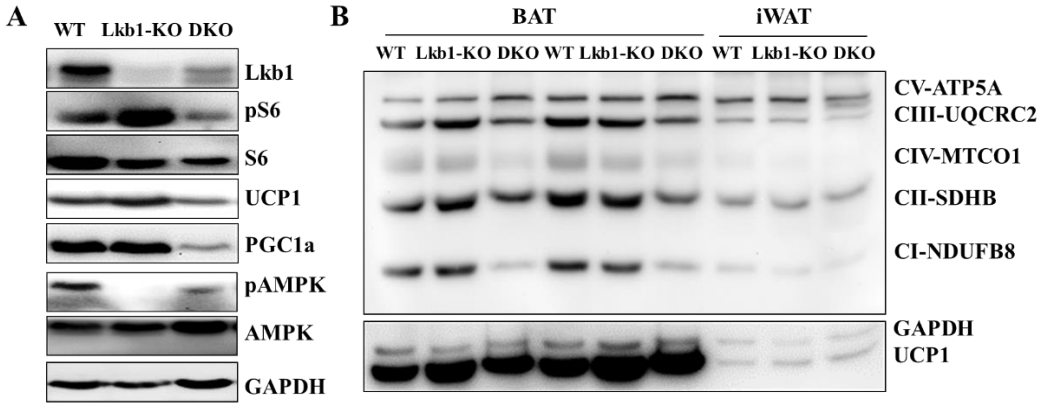


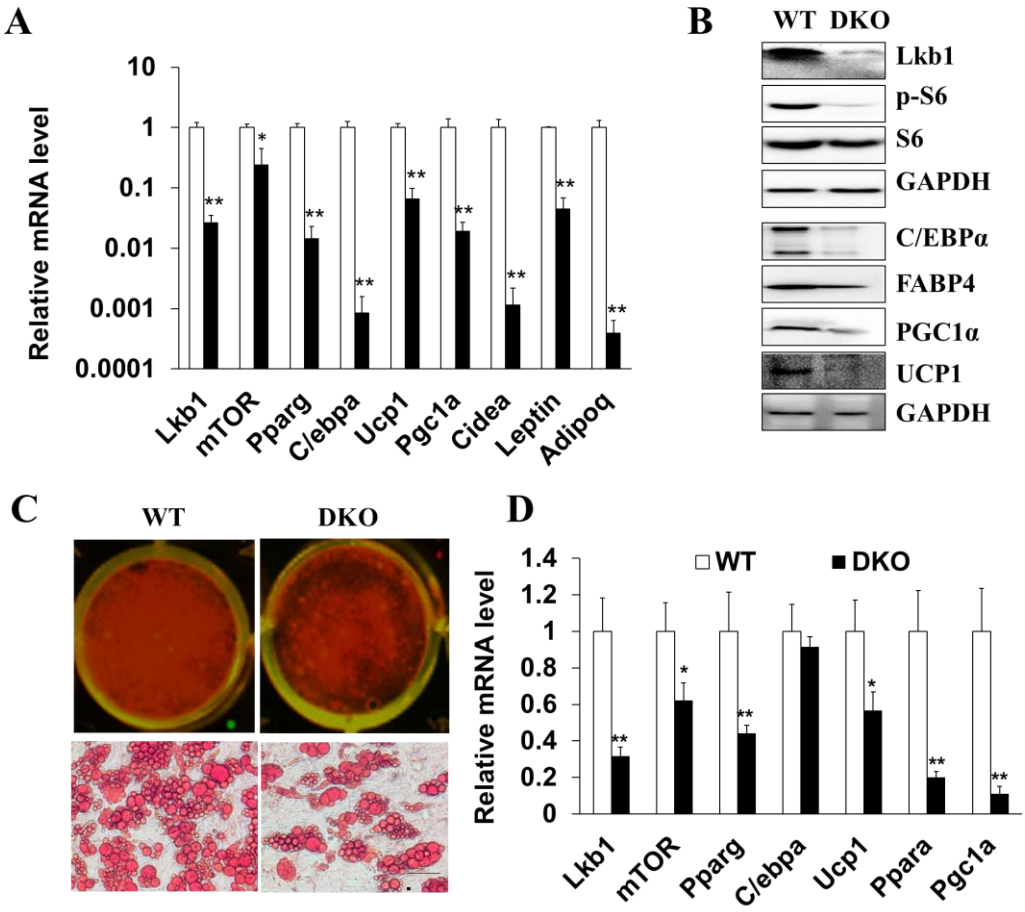
Supplemental Figure S1. Adipocyte-specific deletion of Lkb1 and mTOR does not affect systemic energy expenditure. Oxygen consumption (VO_2, A), carbon dioxide production (VCO_2, B), RER (C) and heat production (D) were measured by using indirect calorimetry system (n=5).



Supplemental Figure S2. Adipocyte-specific deletion of Lkb1 and mTOR causes fatty liver. (A) Representative images of liver after chow diet or high-fat diet (HFD) feeding. (B, C) HE (B) and Oil red O staining (C) of liver sections from WT and DKO mice after HFD-feeding. Scale bars: 100 μm.



Supplemental Figure S3. Double knockout of Lkb1 and mTOR decreased the protein levels of UCP1, PGC1α and mitochondrial related proteins in BAT. (A) The protein levels of Lkb1, pS6, S6, UCP1 and PGC1α, pAMPK, AMPK in BAT from WT, Lkb1-KO and DKO mice. (B) The levels of mitochondrial proteins in BAT and iWAT from different mice.



Supplemental Figure S4. Lkb1 and mTOR ablation decreases the expression of adipogenesis and mitochondrial function related genes in iWAT and cultured iWAT adipocytes. (A, B) mRNA (A) and protein (B) levels of adipogenesis and mitochondrial function related genes in iWAT from WT and DKO mice after 10-week of high-fat diet feeding (n=5). (C) Oil red O staining of cultured WT and DKO iWAT adipocytes. (D) mRNA levels of adipogenesis and mitochondrial function related genes in differentiated WT and KO iWAT adipocytes (n=3). Error bars represent SEM. * $p < 0.05$, ** $p < 0.01$.