**Supplemental Figure 1. Assessment of cardiac function with obesity or with changes in autophagy**

(**A**) M-mode echocardiographic images from WT and Ob/Ob mice by feeding either HFD or ND for 28 weeks. (**B**) M-mode echocardiographic images from *Myh6-cre* and *Myh6-cre/ulk1*fl/fl mice by feeding either HFD or ND for 28 weeks. (**C**) M-mode echocardiographic images from *Myh6-cre* and *Myh6-cre/Lpl*fl/fl mice by feeding either HFD or ND for 28 weeks. (**D**) M-mode echocardiographic images from *Myh6-cre* and *Myh6-cre/Lpl*fl/fl *ulk1*fl/fl mice by feeding either HFD or ND for 28 weeks. (**E**) M-mode echocardiographic images from WT and Ob/Ob mice by infusing with D-Tat-scramble or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet.

**Supplemental Figure 2. Cardiac lipids and ULK1 levels in obese hearts**WT and Ob/Ob mice were generated from Ob/+ heterozygous intercrosses. 6 week-old male mice were fed either a high fat diet (HFD; 60% cal from diet) or a normal chow diet (ND; 10% cal from fat) for 28 weeks. (A) DAG levels were measured using a DAG assay kit (n=6). (B-C) Ceramide and fatty acyl-CoA were performed by LC-MS/MS (n=6). (D-F) Cardiomyocytes (MLCV2), fibroblasts (Thy1), smooth muscle (α-SMA) and endothelial cells (CD31) were isolated from hearts. And 2x104 cells/ml were used to determine ULK1 expression in each cell line (n=6). Results were analyzed using one-way-ANOVA (graphs show means± SE of 6 experiments in each group). \*Significantly different from ND treated WT mice, *P* < 0.05. +Significantly different from other cell lines, *P* < 0.05 WB, Western blot; WT, Wild type; Ob/Ob, *Lep*ob/ob; TG, triglyceride; ND, normal chow diet; HFD, high fat diet.

**Supplemental Figure 3. Cardiac TG levels in obese or autophagy deficient hearts.**

Cardiac TG was stained with Oil-Red O and also measured using a PicoProbe triglyceride fluorometric assay kit. (**A**-**B**) WT and Ob/Ob mice were fed either HFD or ND for 28 weeks. The obese mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. (**C-D**) *Myh6-cre* and *Myh6-cre/ulk1*fl/fl were fed either HFD or ND for 28 weeks. The mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. Results were analyzed using two-way-ANOVA (graphs show means ± SE of 6 experiments in each group). \*Significantly different from ND treated WT mice, *P* < 0.05. #Significantly different from ND treated Ob/Ob mice, *P* < 0.05. @Significantly different from ND treated *Myh6-cre* mice, *P* < 0.05. †Significantly different from HFD treated *Myh6-cre/ulk1*fl/fl mice, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet. Scale bar = 100 μm.

**Supplemental Figure 4. Alterations in cardiac lipid-related mRNA and protein levels with obesity**

WT and Ob/Ob mice were generated from Ob/+ heterozygous intercrosses. (**A-D**) CD36, GPAT, DGAT, and ATGL protein levels were determined using Western blotting (n=6). Beta-actin was used to ensure an equal protein loading. (**E-H**) Total RNA was isolated from hearts, CD36, GPAT, DGAT, and ATGL mRNAs were measured using real time PCR (n=6). Results were analyzed using two-way-ANOVA (graphs show means± SE of 6 mice in each experiments). \*Significantly different from ND treated WT hearts, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob.

**Supplemental Figure 5. Autophagic flux in obese or autophagy deficient hearts.**

Mice were infused with saline, Bafilomycin A1 (1 μg/min per kg body weight) or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. (**A**) Cardiomyocytes were isolated and homogenized then subjected to Western blot for evaluating LC3. (B) autophagosome was measured using fluorescent assay and confocal microscope. Nucleus stained with DAPI shown as blue, autophagosome shown as green, and sarcomeric alpha actinin as red. Results were analyzed using two-way-ANOVA (graphs show means ± SE of 6 experiments in each group). (**C**) The isolated cardiomyocytes were subjected to Western blot for evaluating LPL. (D) Cardiomyocytes were isolated and homogenized then subjected to Western blot for evaluating p62. Results were analyzed using two-way-ANOVA (graphs show means ± SE of 6 experiments in each group). \*Significantly different from saline infused WT mice heart, *P* < 0.05. #Significantly different from saline infused *Myh6-cre* mice heart, *P* < 0.05. @Significantly different from saline infused *Myh6-cre/ulk1*fl/fl mice heart, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet.

**Supplemental Figure 6. Cardiac lipids and lipid-related protein levels in ULK1 deficient hearts.**Hearts were isolated and homogenized from WT, *Myh6-cre*, *Myh6-cre/Lpl*fl/fl, *Myh6-cre/ulk1*fl/fl, or *Myh6-cre/ulk1*fl/fl*Lpl*fl/fl mice. (**A-D**) CD36, GPAT, DGAT, and ATGL protein levels were determined using Western blotting (n=6). Beta-actin was used to ensure an equal protein loading. (**E**) DAG levels were measured using a DAG assay kit (n=6). (**F-G**) Ceramide and fatty acyl-CoA were performed by LC-MS/MS (n=6). Results were analyzed using two-way-ANOVA (graphs show means± SE of 6 mice in each experiments). \*Significantly different from *Myh6-cre* mice, *P* < 0.05, #Significantly different from HFD treated *Myh6-cre/ulk1*fl/fl mice, *P* < 0.05, WT, Wild type; Ob/Ob, *Lep*ob/ob; TG, triglyceride; ND, normal chow diet; HFD, high fat diet; WB, Western blot.

**Supplemental Figure 7. Role of D-Tat-beclin1 in cardiac lipids, lipid-related protein levels, and insulin sensitivity.**WT and Ob/Ob mice were generated from Ob/+ heterozygous intercrosses. 8 week-old male mice Ob/Ob mice were infused with D-Tat-scramble or D-Tat-beclin1 (4 μg/min per kg body weight for 3 weeks using osmotic pumps. After the infusion had finished, hearts were isolated and homogenized from the mice. (**A-D**) CD36, GPAT, DGAT, and ATGL protein levels were determined using Western blotting (n=6). Beta-actin was used to ensure an equal protein loading. (**E**) Mice were fasted for 14 h, blood glucose levels were measured during intraperitoneal glucose tolerance test (n=6). (**F**) Following the D-amino acids infusion to the animals, body weights were measured between 8 weeks and 12 weeks (n=6). (**G**) The isolated cardiomyocytes (2x104 cells/ml) were treated with 0.1μM insulin for 30 min and glucose uptake assay was done over a period of 30 min after the addition of 0.5 μCi/ml 2-deoxyglucose (n=6). (**H**) DAG levels were measured using a DAG assay kit (n=6). (**I-J**) Ceramide and fatty acyl-CoA were performed by LC-MS/MS (n=6). Results were analyzed using two-way-ANOVA (graphs show means± SE of 6 experiments in each group). \*Significantly different from D-Tat-scramble infused WT groups, *P* < 0.05. #Significantly different from D-Tat-scramble infused Ob/Ob groups, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob; TG, triglyceride.

**Supplemental Figure 8. Cardiomyocyte apoptosis in obese or autophagy-deficient hearts.**

Using TUNEL assay, cardiomyocyte apoptosis was determined in each group. TUNEL positive cells appear red (Arrows). Non-apoptotic nuclei remain Blue. (**A & D**) WT and Ob/Ob mice were fed either HFD or ND for 28 weeks. The obese mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. (**B**) WT hearts were isolated and perfused in the retrograde Langendorff mode with 35 min global ischemia and 40 min reperfusion. (**C & E**) *Myh6-cre* and *Myh6-cre/ulk1*fl/fl were fed either HFD or ND for 28 weeks. The autophagy deficient mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. Results were analyzed using two-way-ANOVA (graphs show means ± SE of 6 experiments in each group). †Significantly different from other group, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet. Scale bar = 100 μm.

**Supplemental Figure 9. Cardiac fibrosis in tissue sections from obese or autophagy deficient hearts.**

To determine cardiac fibrosis, cardiac sections were stained with Masson's trichrome in each group. (A & C) WT and Ob/Ob mice were fed either HFD or ND for 28 weeks. The obese mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. (B & D) *Myh6-cre* and *Myh6-cre/ulk1*fl/fl were fed either HFD or ND for 28 weeks. The mice were infused with saline or D-Tat-beclin1 (4 μg/min per kg body weight) for 3 weeks using osmotic pumps. Results were analyzed using two-way-ANOVA (graphs show means ± SE of 6 experiments in each group). \*Significantly different from ND treated WT mice, *P* < 0.05. #Significantly different from ND treated Ob/Ob mice, *P* < 0.05. @Significantly different from ND treated *Myh6-cre* mice, *P* < 0.05. †Significantly different from HFD treated *Myh6-cre/ulk1*fl/fl mice, *P* < 0.05. WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet. Scale bar = 100 μm.

**Supplemental Figure 10. Phosphorylation of Hsp25 or PKD in lean or obese hearts.**

WT and Ob/Ob mice were generated from Ob/+ heterozygous intercrosses. 6 week-old male mice were fed either HFD or ND for 28 weeks. (**A-B**) The mice hearts were isolated to evaluated phosphorylation of Hsp25 or PKD using western blotting and densitometry. Results were analyzed using one-way-ANOVA (graphs show means ± SE of 6 experiments in each group). WT, Wild type; Ob/Ob, *Lep*ob/ob; ND, normal chow diet; HFD, high fat diet.