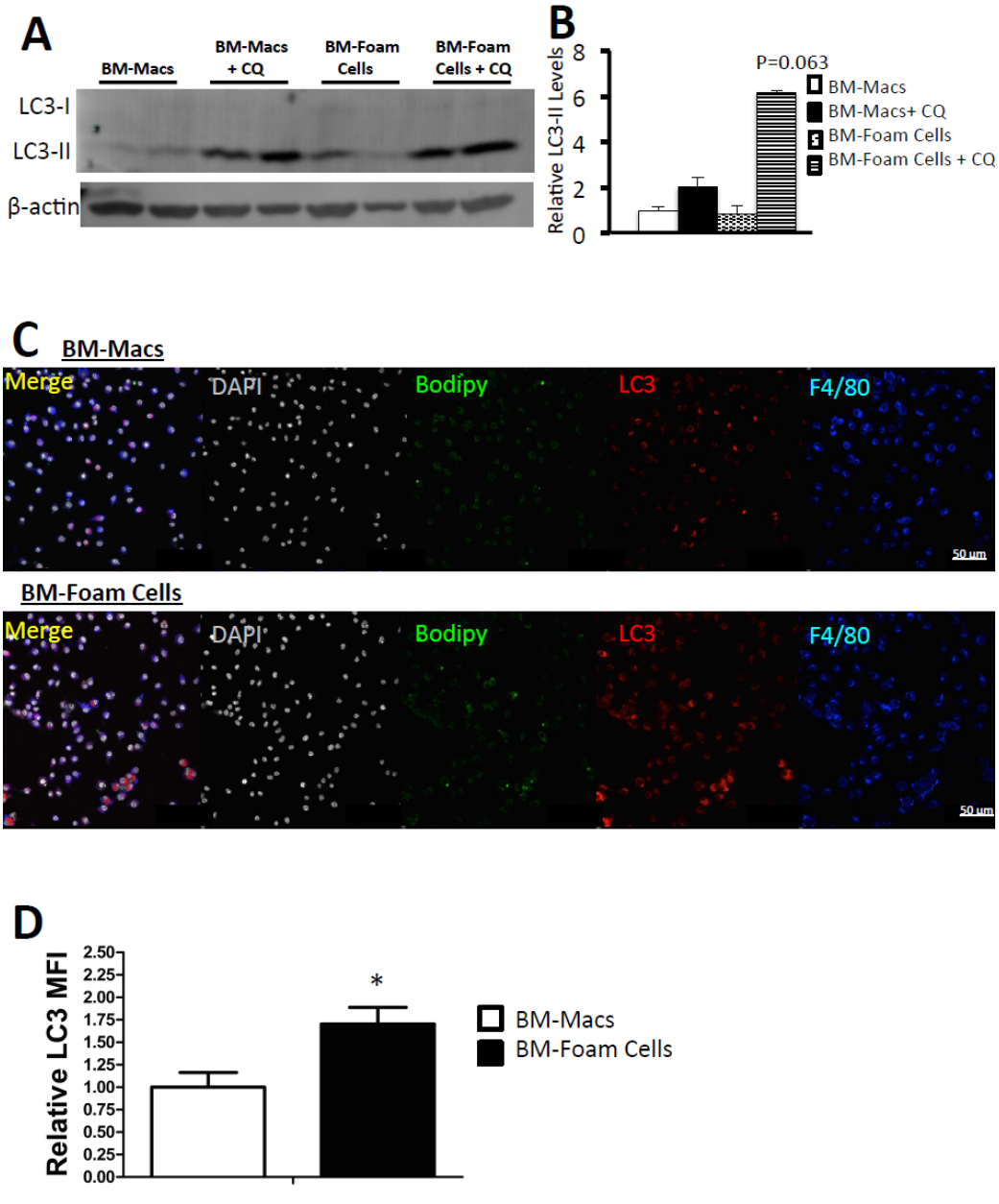
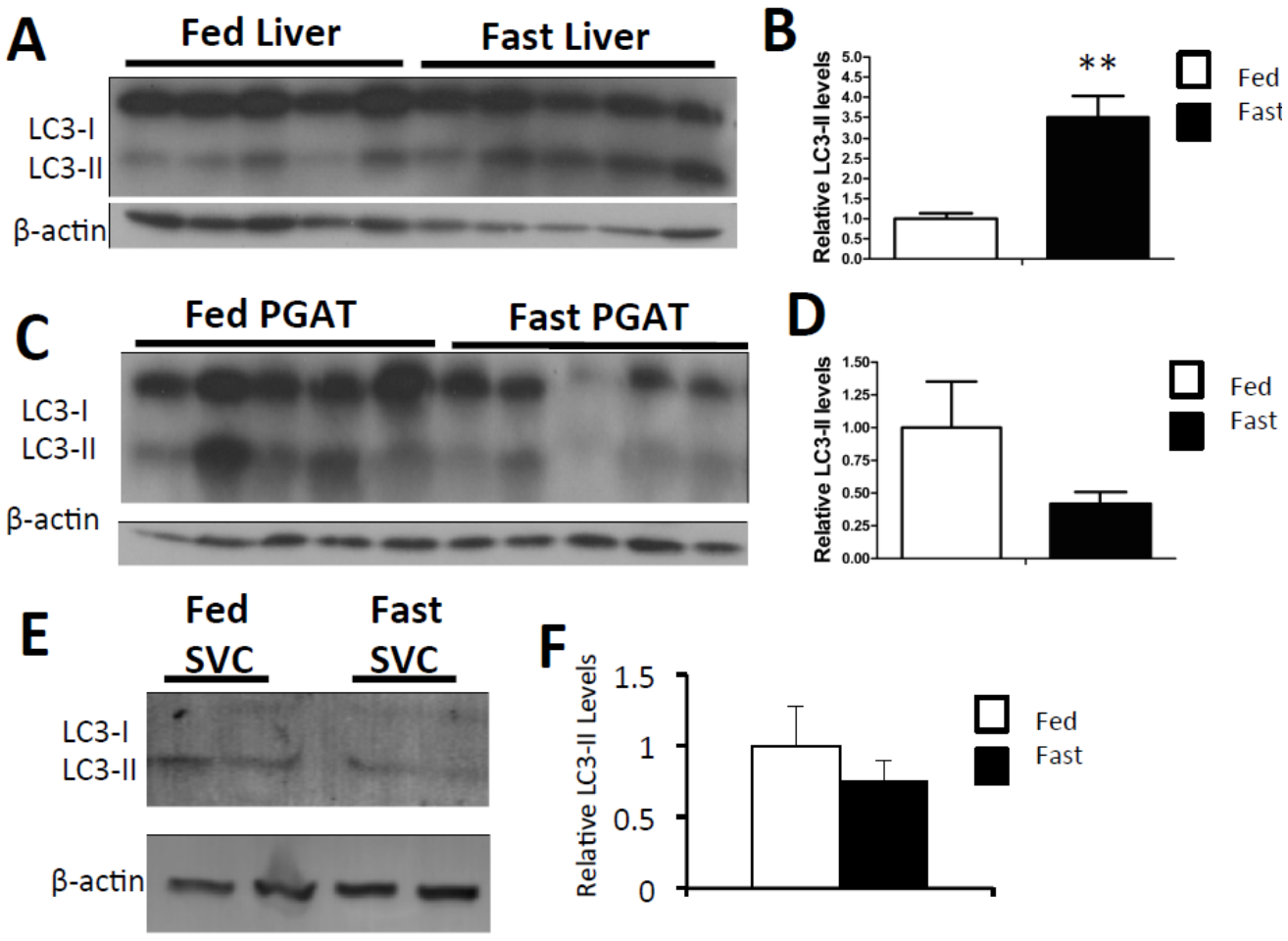
SUPPLEMENTARY DATA

Supplementary Figure 1. Autophagy is increased in Foam cells.
Bone marrow cells were isolated and differentiated with M-CSF to generate BM-Macs. BMMacs were treated with 150 ug/mL acetylated low-density lipoprotein to generate BM-Foam Cells. BM-Macs and BM-Foam Cells were treated with 20 uM chloroquine or diH_20 for 16 hours. *A:* Immunoblots were done against anti-LC3 and anti-β-actin antibodies in BM-Macs, BM-Macs + CQ, BM-Foam Cells, and BM-Foam Cells + CQ *B:* Quantification of relative LC3-II levels normalized to β-actin. n=2/ group mean ±SEM P-value BM-Macs + CQ vs BMFoam Cells + CQ *C:* Immunofluorescence staining of lipid (BODIPY), autophagy (anti-LC3 antibody), and macrophages (anti-F4/80 antibody) was done in BM-Macs and BM-Foam Cells Scale bar= 50µm. *D:* Quantification of relative LC3 mean fluorescence intensity in macrophage cells determined using Nikon NIS elements software White bar= BM-Macs Black bar= BM-Foam Cells n=11/group mean ±SEM * P<0.05 **P<0.01 *** P<0.005 Autophagy in adipose tissue macrophages



SUPPLEMENTARY DATA

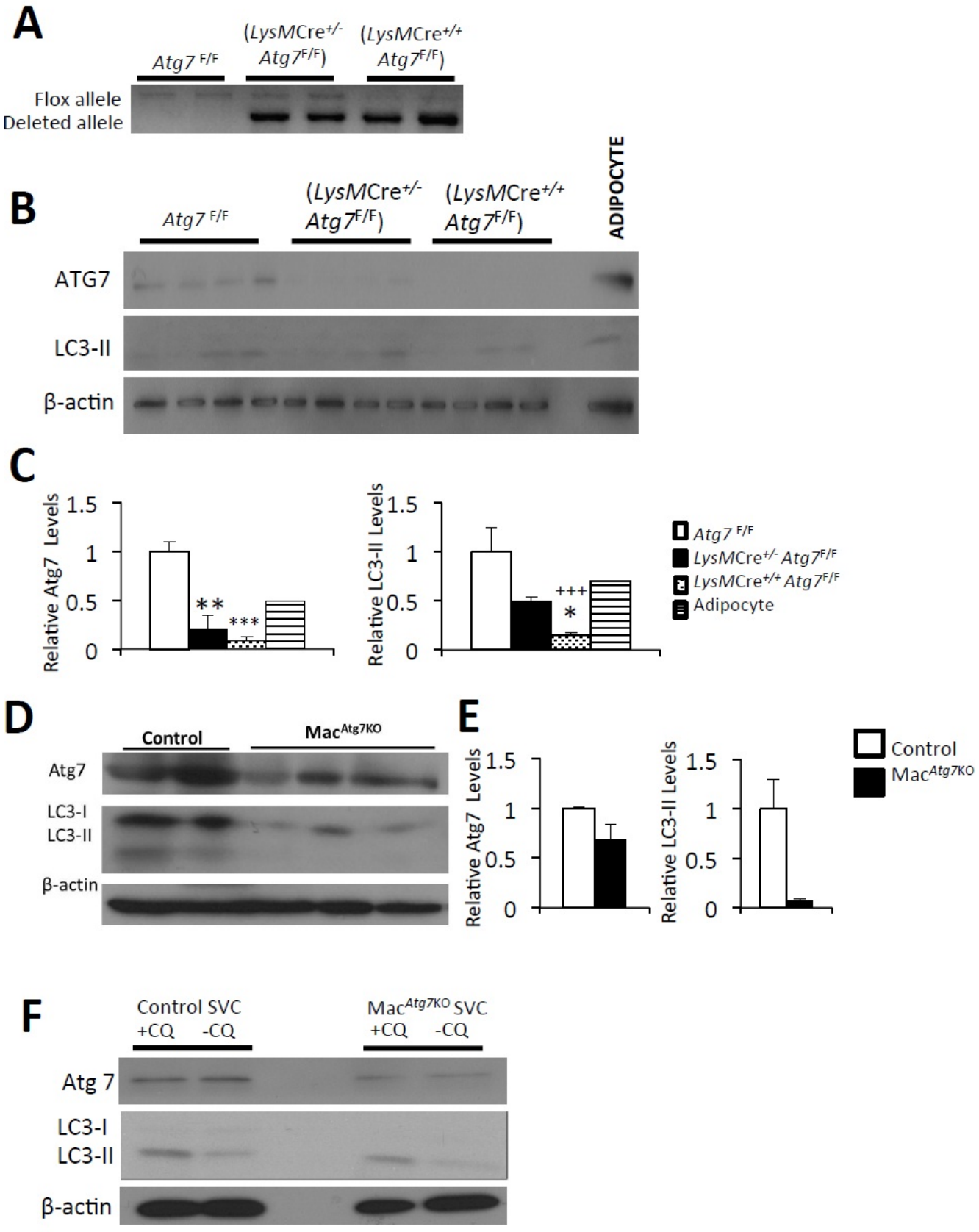
Supplementary Figure 2. Fasting does not induce autophagy in adipose tissue, stromal vascular cells, or BM-ATMs.
12-week-old lean male mice were fasted for 24 hours, half of which were refed ad libitum for 4 hours. Liver and perigonadal adipose tissue (PGAT) protein was extracted to assess autophagy using anti-LC3 antibody. Western blots of LC3 and β-actin protein levels in *A:* Liver and *C:* PGAT *B/D:* Quantification of relative LC3-II protein normalized to β-actin. n=5/group mean ±SEM * P<0.05 **P<0.01 *** P<0.005 *E:* Perigonadal adipose tissue stromal vascular cells were isolated from fasted and fed mice and protein was extracted. Western blot representing LC3 and β-actin protein levels in SVCs. *F:* Quantification of relative LC3-II protein normalized to β-actin. n=2 / group mean ±SEM



SUPPLEMENTARY DATA

Supplementary Figure 3. *Atg7* deletion in macrophages.
A: DNA was isolated from circulating white blood cells littermate controls (*Atg7*$^{F/F}$) and mice carry one (*LysM*Cre$^{+/-}$*Atg7*$^{F/F}$) or two copies (*LysM*Cre$^{+/+}$*Atg7*$^{F/F}$) of the Cre allele. PCR analysis revealed the presence of *floxed* allele and "*deleted"* alleles as expected *B:* Protein was isolated from BM-Macs from control mice (*Atg7*$^{F/F}$), Mac *Atg7* KO carrying one (*LysM*Cre$^{+/-}$ *Atg7*$^{F/F}$), or two (*LysM*Cre$^{+/+}$*Atg7*$^{F/F}$) *Cre* alleles. Atg7 and LC3 proteins were measured with β-actin as a control. *C:* Quantification of relative Atg7 and LC3-II protein levels normalized to β-actin. n=4 / group mean ±SEM * P<0.05 **P<0.01 *** P<0.005 Control vs *LysM*Cre$^{+/-}$ *Atg7*$^{F/F}$ or *LysM*Cre$^{+/+}$*Atg7*$^{F/F}$ + P<0.05 ++P<0.01 +++P<0.005 *LysM*Cre$^{+/-}$*Atg7*$^{F/F}$ vs *LysM*Cre$^{+/+}$*Atg7*$^{F/F}$ *D:* Protein was extracted from perigonadal adipose tissue from lean littermate controls (*Atg7* $^{F/F}$) and Mac $^{Atg7\ KO}$ (*LysM*Cre+*Atg7*$^{F/F}$). Western blots were done with antibodies against anti-Atg7, anti-LC3 and anti-β-actin. *E:* Quantification of relative Atg7 and LC3-II protein levels normalized to β-actin. n=2/ Control n=4 / Mac $^{Atg7\ KO}$ mean ±SEM *F:* Stromal Vascular Cells were isolated from lean littermate controls (*Atg7* $^{F/F}$) and Mac $^{Atg7\ KO}$ (*LysM*Cre+*Atg7*$^{F/F}$) mice, treated with chloroquine (+CQ) or diH_20 (-CQ) for 16 hours and Autophagy in adipose tissue macrophages protein was extracted. Immunoblot against anti-Atg7, anti-LC3 and anti-β-actin was performed.

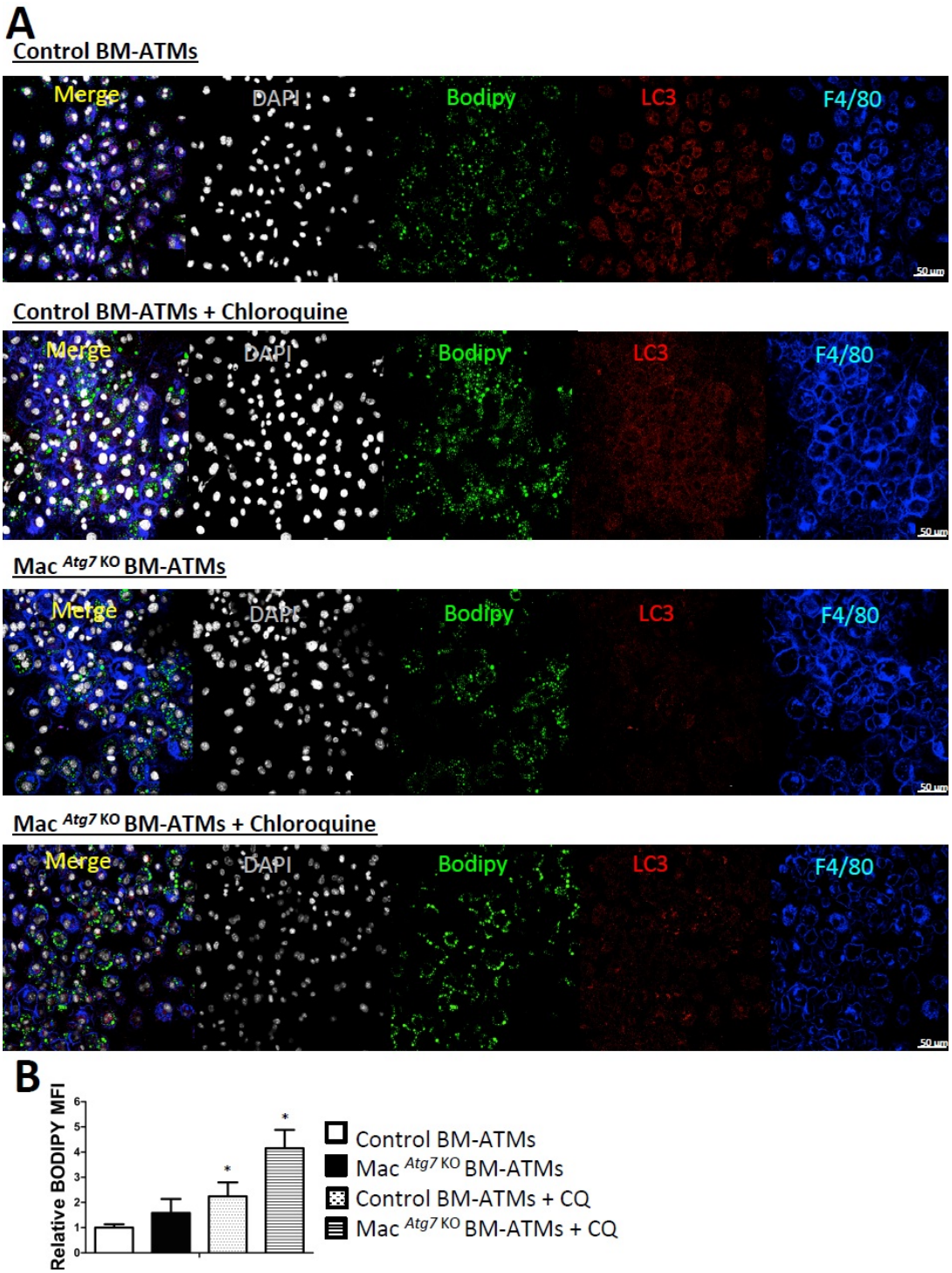
SUPPLEMENTARY DATA



SUPPLEMENTARY DATA

Supplementary Figure 4. Lysosomal inhibition results in lipid accumulation in control and Mac $^{Atg7\ KO}$ BM-ATMs.

BM-ATMs from control and Mac$^{Atg7\ KO}$ mice were treated with 20 uM chloroquine or diH_20 for 16 hours. *A:* Cells were stained using BODIPY, anti-LC3 (autophagy) antibody, and anti- F4/80 (macrophage) antibody. Scale = 50 µm. *B:* Quantification of relative BODIPY mean fluorescence intensity in macrophage cells was calculated using Nikon NIS elements software n= 4/group mean ±SEM * P<0.05 **P<0.01 *** P<0.005 comparison between nontreated and CQ treated



SUPPLEMENTARY DATA

Supplementary Figure 5. Neutral lipolysis is not increased as a compensatory lipid catabolism pathway in Mac $^{Atg7\ KO}$ mice.

PGAT was isolated from *A:* lean or *C:* DIO control and Mac$^{Atg7\ KO}$ mice. Protein was extracted and examined using western blots analysis using anti-Atgl and anti-β-actin antibodies. Quantification of relative ATGL levels in *B:* Lean *D:* DIO n=1-2/ Control n=4 / Mac $^{Atg7\ KO}$ mean ±SEM *E:* Bone marrow cells were isolated from controls (*Atg7*$^{F/F}$), and Mac $^{Atg7\ KO}$ (*LysM*Cre^{+}*Atg7*$^{F/F}$) mice and differentiated into BM-Macs with M-CSF. BM-Macs were further differentiated with M-CSF (BM-Macs) and with adipose tissue (AT) to generate BMATMs. Protein was extracted from BM-ATMs and adipocytes and western blot done using antibodies against anti-Atgl and anti-β-actin. *F:* Quantification of relative ATGL levels in Control, Mac$^{Atg7\ KO}$, and adipocyte. n=4 BM-ATMs and n=1 adipocyte mean ±SEM