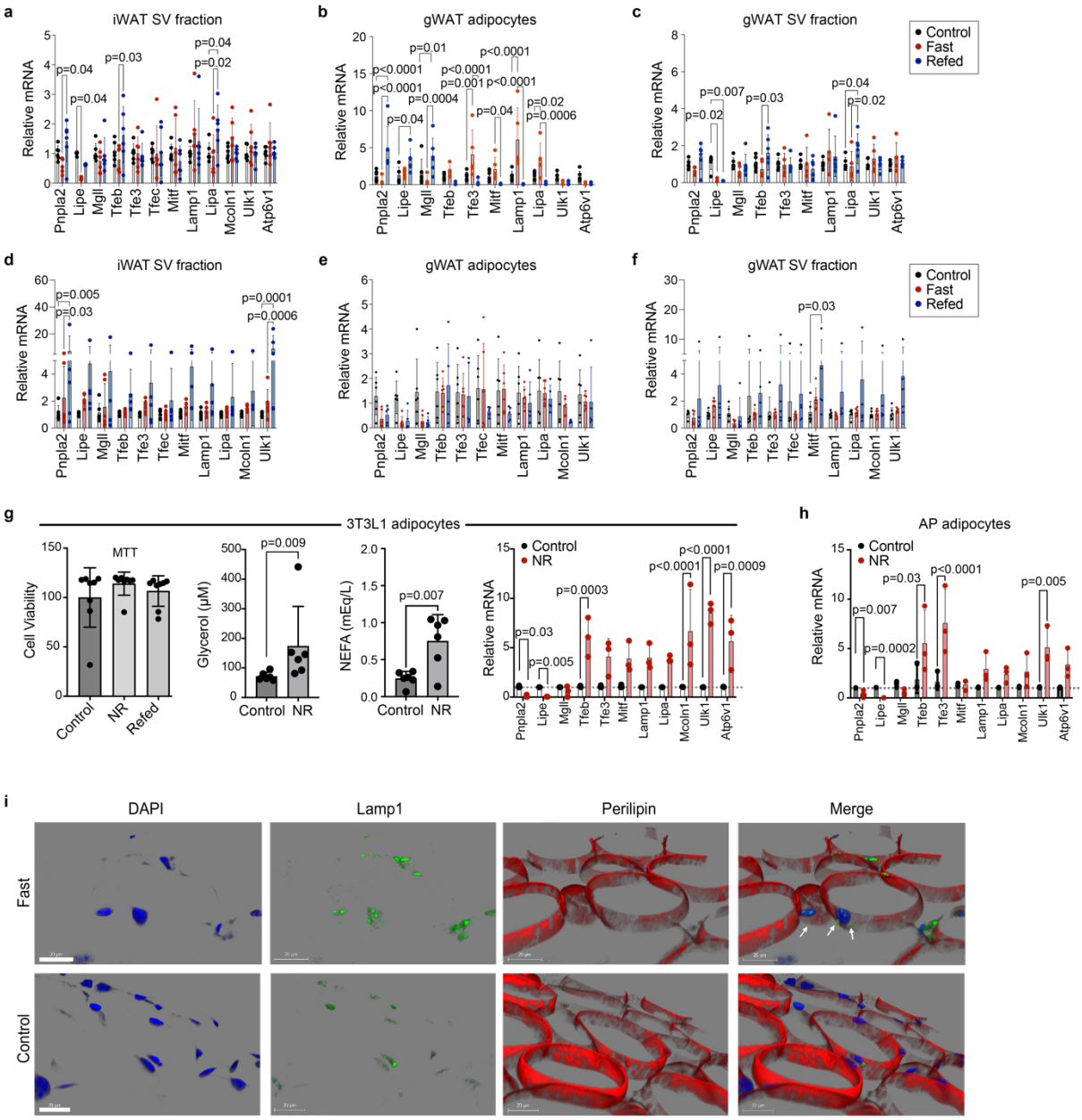


Figure S1. Fasting preferentially drives a lysosomal program in murine adipose tissue and adipocytes.

a. qPCR for canonical lipase genes and lysosomal genes in stromal-vascular fractions isolated from the inguinal adipose depots of fed and fasted male mice, the adipocyte fractions of which are shown in Figure 1c. Significance assessed by two-way ANOVA/Tukey's test.

b. qPCR for canonical lipase genes and lysosomal genes in adipocyte fractions isolated perigonadal adipose depots of fed and fasted male mice. Significance assessed by two-way ANOVA/Tukey's test.

c. qPCR for canonical lipase genes and lysosomal genes in stromal-vascular fractions isolated from the perigonadal adipose depots of fed and fasted male mice. Significance assessed by two-way ANOVA/Tukey's test.

d. qPCR for canonical lipase genes and lysosomal genes in stromal-vascular fractions isolated from the inguinal adipose depots of fed and fasted female mice. Significance assessed by two-way ANOVA/Tukey's test.

e. qPCR for canonical lipase genes and lysosomal genes in adipocyte fractions isolated perigonadal adipose depots of fed and fasted female mice. Significance assessed by two-way ANOVA/Tukey's test.
f. qPCR for canonical lipase genes and lysosomal genes in stromal-vascular fractions isolated from the perigonadal adipose depots of fed and fasted female mice. Significance assessed by two-way ANOVA/Tukey's test.
g. Adipocytes derived from 3T3L1 preadipocytes, exposed to nutrient restriction and removal of insulin from the media. The cells remained viable as assessed by MTT assay and released glycerol and non-esterified fatty acids (NEFA) into the media with 24 hours of nutrient restriction (NR). qPCR assessment of lipolytic genes (far right) demonstrated dynamic changes in canonical lipases and lysosomal genes. Significance assessed by two-way ANOVA/Sidak's test.
h. Adipocytes derived from primary adipocyte progenitor (AP), exposed to nutrient restriction (NR) and removal of insulin from the media. qPCR assessment of lipolytic genes demonstrated dynamic changes in canonical lipases and lysosomal genes. Significance assessed by two-way ANOVA/Sidak's test.
i. Confocal reconstructions of LAMP1+ puncta (arrows) in the perinuclear region and in close approximation to perilipin stained adipocyte lipid droplets. This corresponds to the images shown in Figure 1e. Scale bar=20 micron.
For a-h data presented as mean, S.D.M.

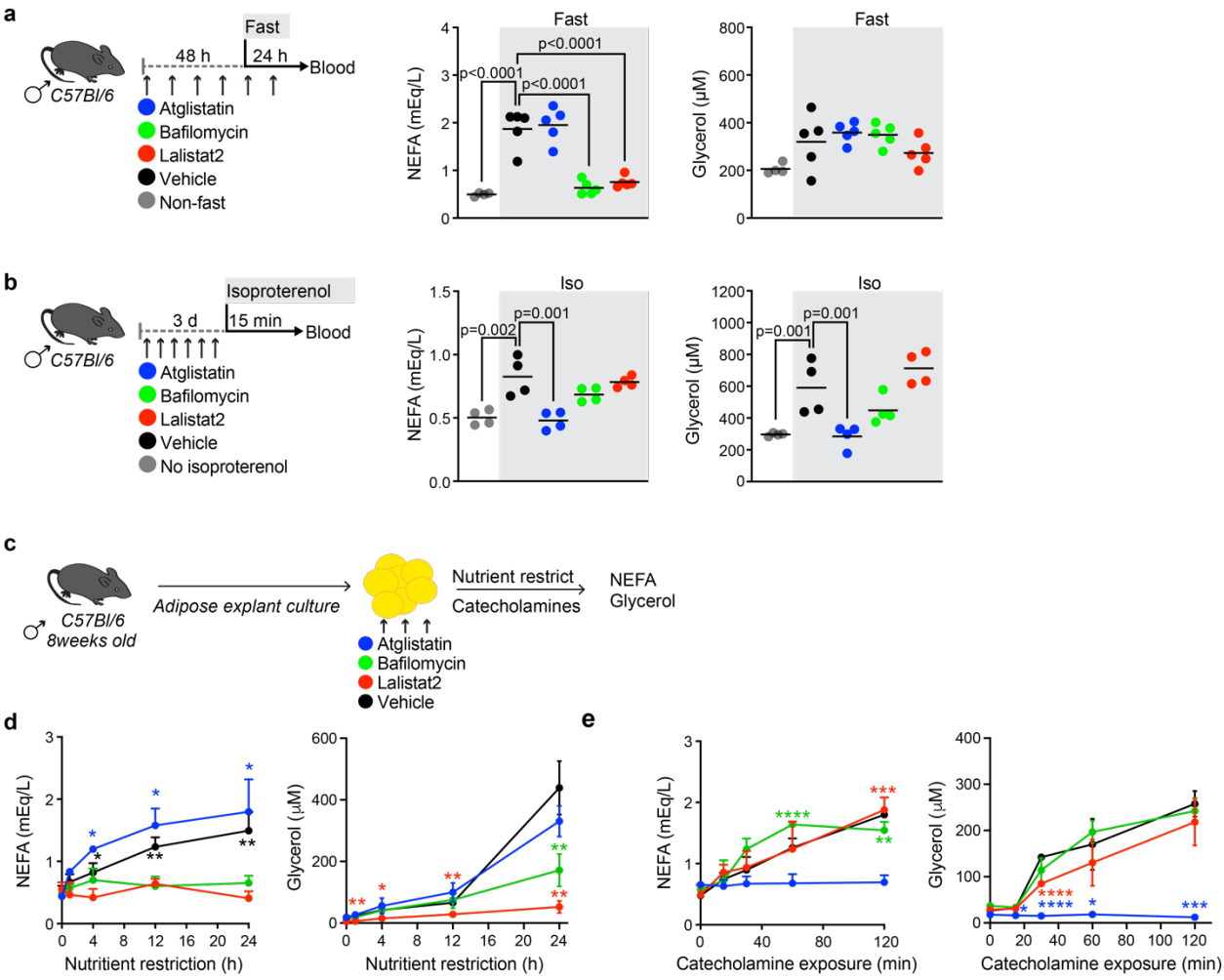


Figure S2. Lysosomal inhibitors neutralize lipolysis in adipose tissue explant culture with nutrient restriction.

a. Mice treated for 3 days with pharmacological inhibitors of lysosomes (Bafilomycin), lysosomal lipase (Lalistat2), the canonical lipase ATGL (Atglistatin), or vehicle with a 24 hour fast in the final day. Fed (non-fasted) controls are shown as gray dots. N=5 male mice, 8 weeks old. Significance assessed by one-way ANOVA/Dunnett's test.

b. Mice treated for 3 days with pharmacological inhibitors of lysosomes (Bafilomycin), lysosomal lipase (Lalistat2), the canonical lipolysis mediator ATGL (Atglistatin), or vehicle prior to administration of isoproterenol. N=4 male mice, 8 weeks old. Significance assessed by one-way ANOVA/Dunnett's test.

c. Schematic of adipose tissue explant culture.

d. Time course of adipose tissue explant culture with nutrient restriction and cultured with vehicle or inhibitors. Left: non-esterified fatty acids (NEFA). Right: glycerol.

e. Time course of adipose tissue explant culture with catecholamine stimulation and cultured with vehicle or inhibitors. Left: non-esterified fatty acids (NEFA). Right: glycerol. For D and E a single experiment was performed where each data point involved analyses of adipose tissue from separate mice, n=3 mice with data shown as mean, S.D.M.; significance assessed by one-way ANOVA/Dunnett's test. *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001

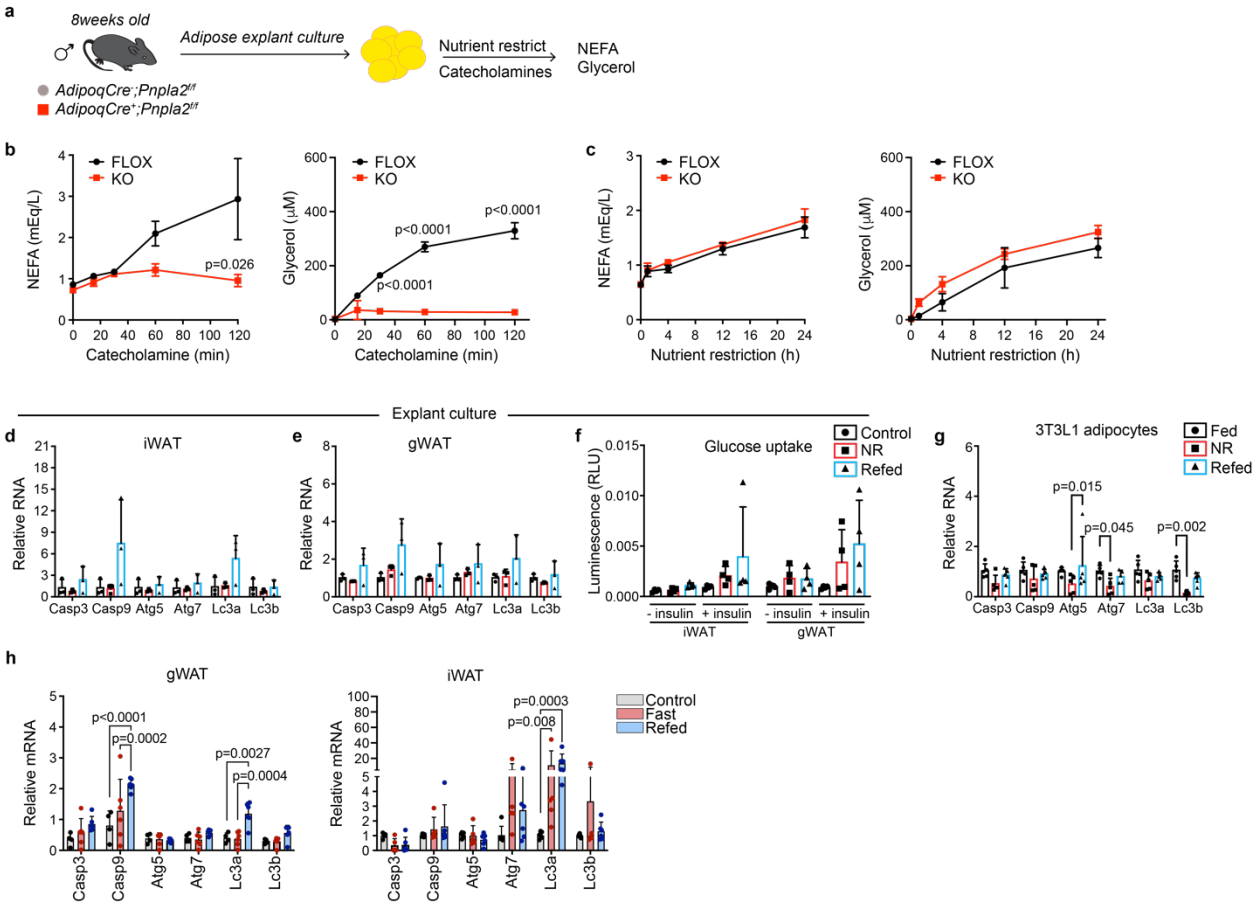


Figure S3. Response to nutrient restriction with adipocyte-specific targeting of *Pnpla2.*

a. Schematic of explant culture and stimulus with nutrient restriction or catecholamines.
b. Time course of catecholamine response for adipose tissue culture using explants from control (FLOX) versus adipocyte-specific ATGL knockout (KO). N=3 mouse donors per group. Two-way ANOVA/Dunnett's test. Data shown as mean, S.D.M.
c. Time course of nutrient restriction response for adipose tissue culture using explants from control (FLOX) versus adipocyte-specific ATGL knockout (KO). N=3 mouse donors per group. Two-way ANOVA/Dunnett's test. Data shown as mean, S.D.M.
d. Autophagy gene expression in inguinal white adipose (iWAT) explant culture with nutrient restriction (NR) or nutrient restriction followed by normalization of culture media ('Refed'). N=3 mouse donors per group.
e. Autophagy gene expression in perigonadal white adipose (gWAT) explant culture with nutrient restriction (NR) or nutrient restriction followed by normalization of culture media ('Refed'). N=3 mouse donors per group.
f. Nutrient restricted and 'refed' explant cultures exhibit insulin responsive glucose uptake. N=3 mouse donors per group.
g. Autophagy gene expression in 3T3L1 derived adipocytes with nutrient restriction (NR) with/without normalization of media ('Refed'). N=5 biological replicates. One-way ANOVA/Dunnett's test.
h. qPCR for autophagy genes in adipocyte fractions isolated perigonadal adipose depots of fed (n=4), fasted (n=6), and refed (n=6) female mice. Significance assessed by two-way ANOVA/Tukey's test.

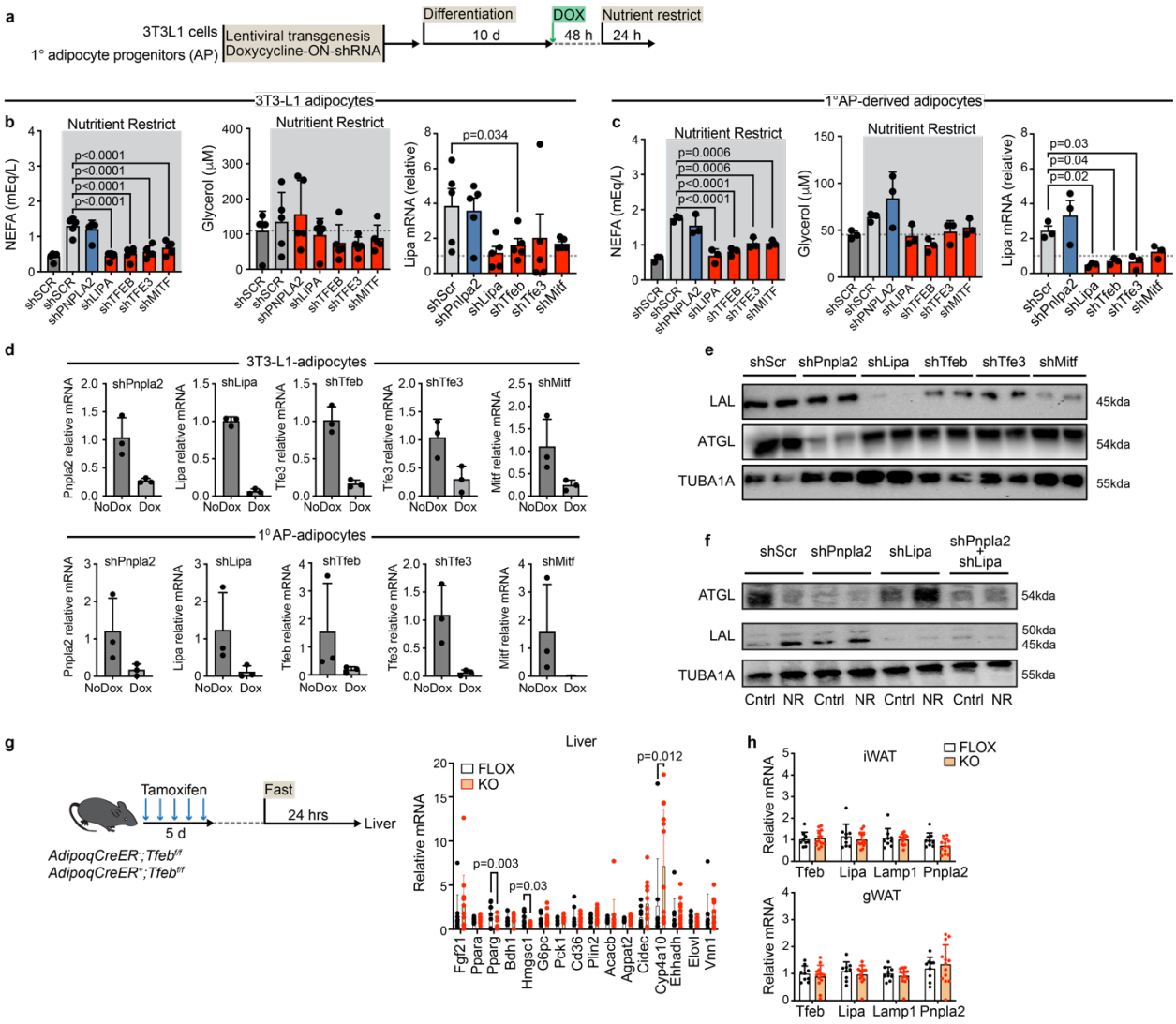


Figure S4. Targeting transcriptional regulators of lysosomal function attenuates lipolysis.

a. Schematic of doxycycline-inducible targeting of lipolysis genes in adipocytes derived from 3T3L1 preadipocytes or primary adipocyte progenitor (AP) cells. For data shown in b-g where each data point is a biological replicate experiment—i.e. mean of technical replicates.

b. Knockdown of *Pnlpa2* (blue) or lysosomal factors (red) in 3T3L1 adipocytes. Left: non-esterified fatty acids (NEFA); Middle: Glycerol; Right: *Lipa* mRNA with biological replicates (n=5) normalized to scramble control that was not exposed to nutrient restriction (dashed line). The gray shScr bar represents scramble control exposed to nutrient restriction. Friedman/Dunn's test.

c. Knockdown of *Pnlpa2* (blue) or lysosomal factors (red) in primary adipocyte progenitor (AP) derived adipocytes. Left: NEFA; Middle: Glycerol; Right: *Lipa* mRNA with biological replicates (n=3) normalized to scramble control that was not exposed to nutrient restriction (dashed line). The gray shScr bar represents scramble control exposed to nutrient restriction. One-way ANOVA/Dunnet's test.

d. Validation of gene knockdown in 3T3-L1 adipocytes (top) and primary AP derived adipocytes (bottom). Each dot is representative of a different experiment/biological replicate (n=3).

e. LIPA and ATGL western blots of 3T3L1 derived adipocytes with shRNA gene knockdowns. Note: tubulin controls run on separate gel. N=2 technical replicates from one biological replicate experiment.

f. LIPA and ATGL western blots of 3T3L1 derived adipocytes with/without nutrient restriction (NR). Note: tubulin controls run on separate gel. N=2 technical replicates from one biological replicate experiment.
g. Liver qPCR of lipid metabolic genes with inducible adipocyte specific TFEB targeting (KO, n=13) relative to control (FLOX, n=9) and 24 hours of fasting. Two-way ANOVA.
h. qPCR of stromal-vascular fraction from fasting Tfeb loss of function model as in ‘g’ showing no detectable effect on non-adipocyte cells from inguinal (iWAT) and gonadal (gWAT) adipose tissues.

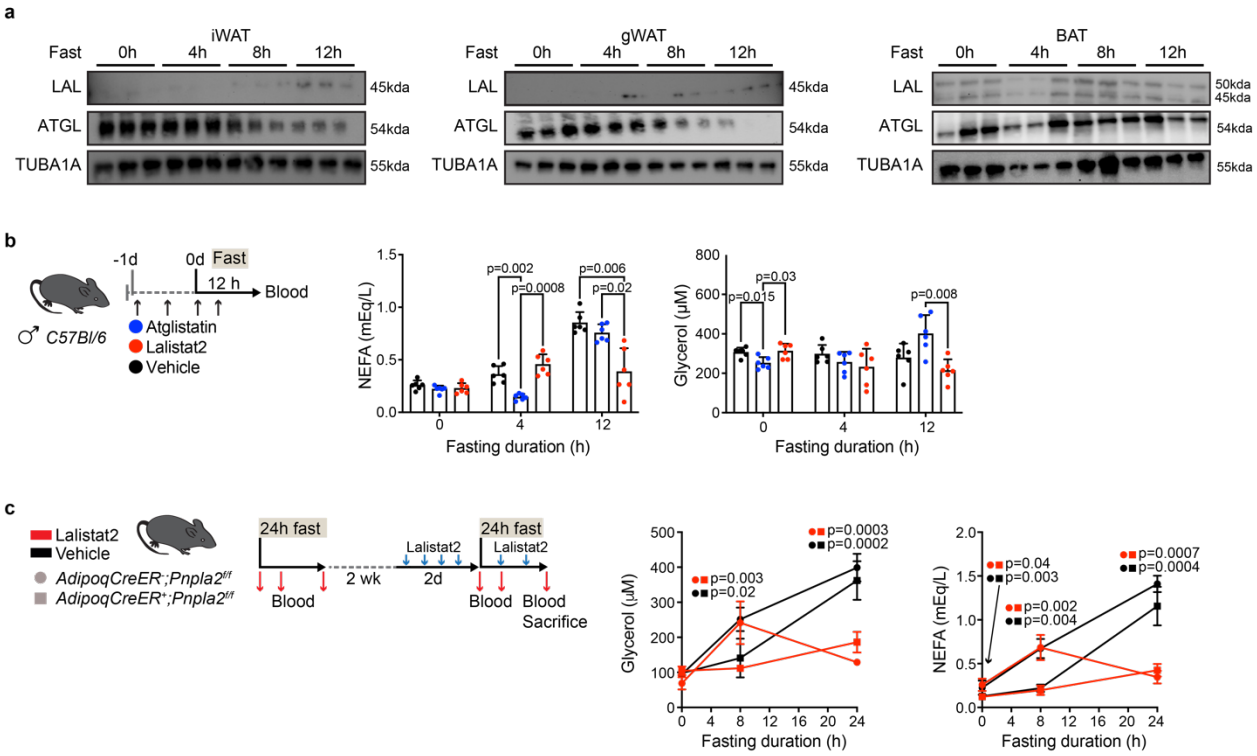


Figure S5. Temporal transition from ATGL-dependent to LIPA-dependent lipolysis with fasting

a. Immuno-blotting for LAL and ATGL in adipose tissues of fasting mice. ATGL protein signal diminishes when fasting progresses beyond 4h in inguinal white adipose tissue (iWAT, left) and in perigonadal adipose tissue (gWAT, middle). A consistent directional signal was not observed in brown adipose tissue (BAT, right). Note: tubulin controls run on separate gel.

b. Mice were treated for 2 days with pharmacological inhibitors of lysosomal lipase (Lalistat2) and ATGL (Atglistatin), or vehicle, with 4 or 12 hours of fasting in the second day of treatment. Left: plasma non-esterifed fatty acids (NEFA); Right: plasma glycerol. N=6 male mice, mean S.D.M.; two-way ANOVA/Tukey's test.

c. Adipocyte specific ATGL knockout (n=5) or control mice (FLOX n=3) males underwent a 24-hour fast followed by a two-week recovery and then another 24-hour fast with pharmacologic LIPA inhibition (Lalistat2). Two-sided t-tests were performed to assess two different hypotheses: (1) genotype effect = circle versus square and (2) lalistat2 effect = red versus black. N=3 floxed control mice; N=5 KO mice. Data shown as mean S.D.M.

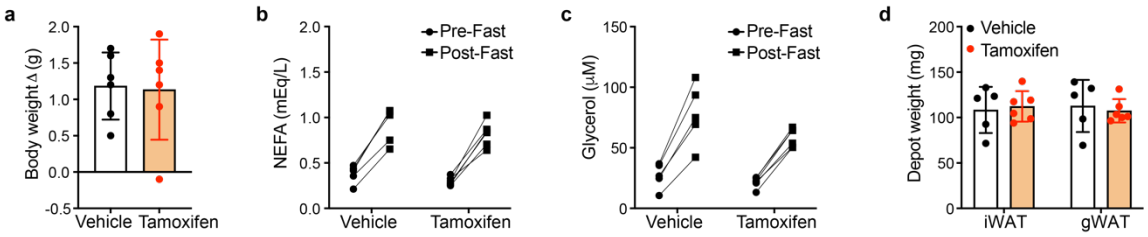


Figure S6. Fasting lipolysis metrics are preserved in mice after administration of tamoxifen recombination protocol.

a. Body weight change after 5-day tamoxifen protocol versus vehicle control in male C67Bl6 mice (n=5 control; n=6 tamoxifen). Mean, S.D.M.
b. One week later, mice underwent 24-hour fast with paired measurement of serum non-esterified fatty acids (NEFA). N=5 control; n=6 tamoxifen.
c. Paired measurement of serum glycerol before and after 24-hour fast.
d. Terminal adipose tissue weight after 24-hour fast. N=5 control; n=6 tamoxifen. Mean, S.D.M.

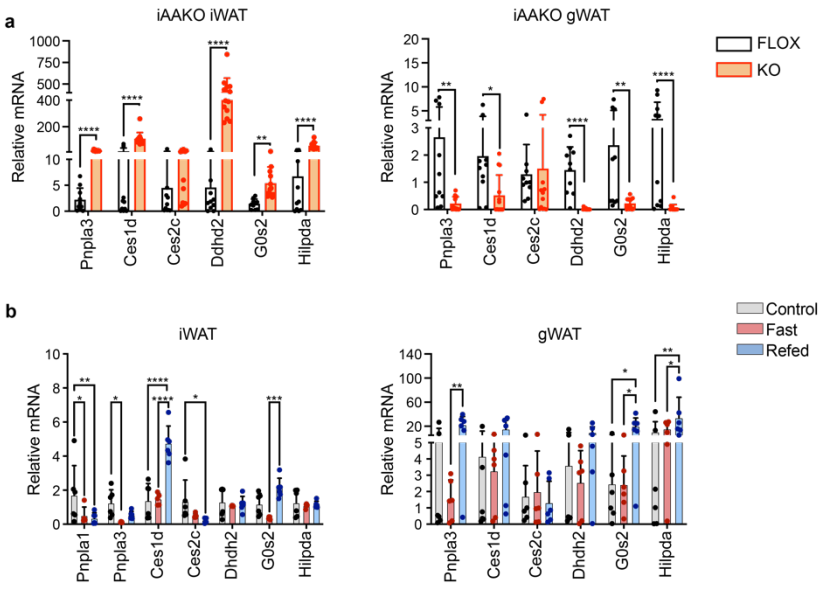
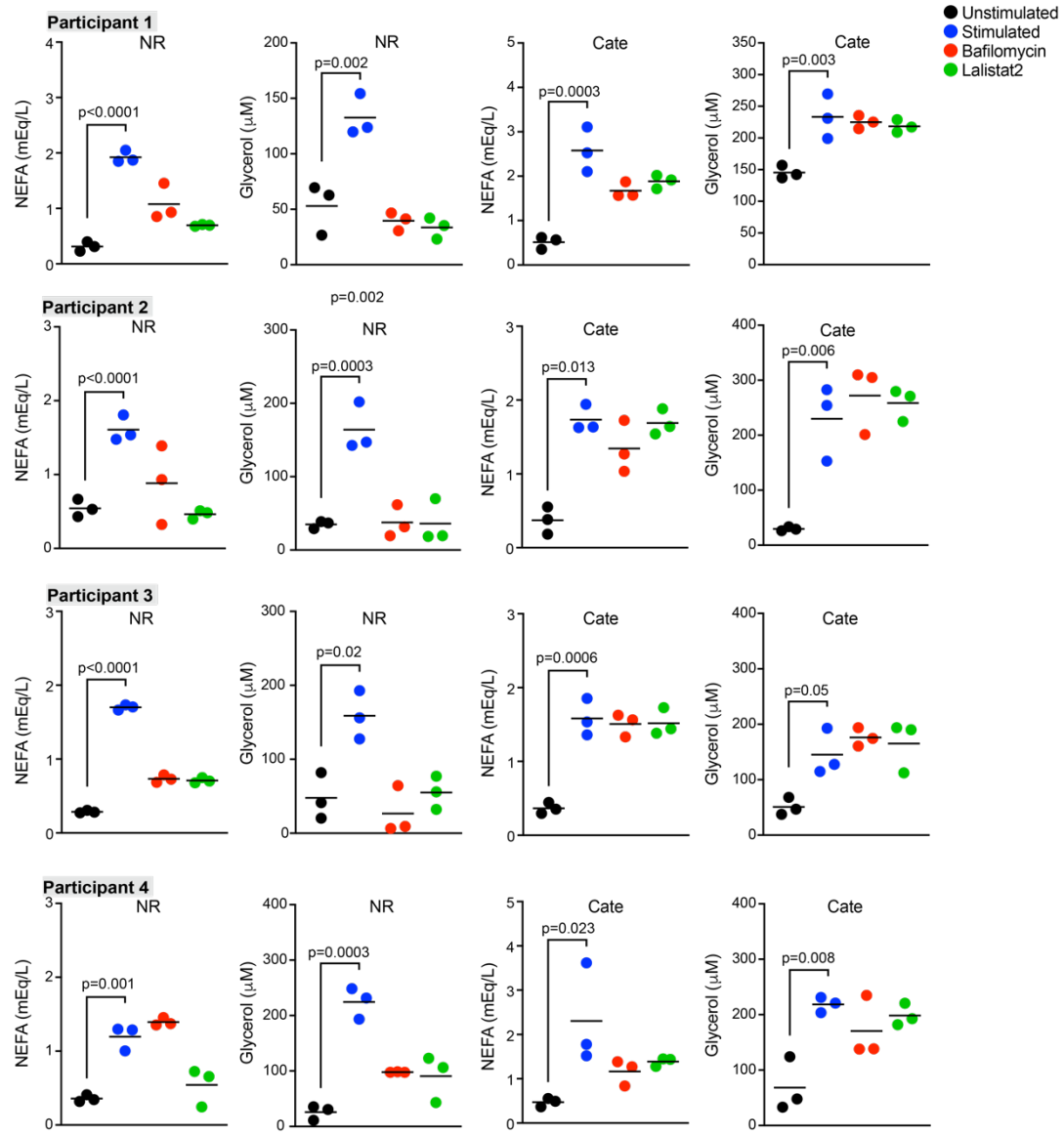


Figure S7. Candidate complementary lipolytic genes in fasting adipocytes.

a. Adipocyte qPCR from control (FLOX, n=10) and inducible adipocyte specific ATGL KO (iAAKO, n=13) adipocytes after 24 hours of fasting. Left, inguinal adipose (iWAT); Right, gonadal adipose (gWAT). Significance assessed by two-way ANOVA: *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001. Data expressed as mean, S.D.M.

b. Adipocyte qPCR from wild-type male C57Bl6 mice. iWAT: control (n=6), 24-hour fasting (FAST, n=5), and after 6 hours of refeeding (REFED, n=6). gWAT: control (n=6), 24-hour fasting (FAST, n=6), and after 6 hours of refeeding (REFED, n=6). Significance assessed by two-way ANOVA/Tukey's test *p<0.05; **p<0.01; ***p<0.001; ****p<0.0001. Data expressed as mean, S.D.M.

a



b

Participants	Sex	BMI	Diabetes
1	Female	53	No diabetes
2	Male	26	No diabetes
3	Female	38	No diabetes
4	Female	29	Diabetes

Figure S8. Nutrient restriction preferentially drives a lysosomal lipolysis in human adipose tissue explants.

a. Lipolysis assays in human explant culture. N=3 technical replicates for each donor specimen. NR=nutrient restriction; Cate=catecholamine stimulation. Significance assessed by one-way ANOVA/Dunnett's test.

b. Table of details for the 4 subjects for which discarded adipose tissue was studied (age mean=46.5 years-old; S.D.M.=5.7 years).

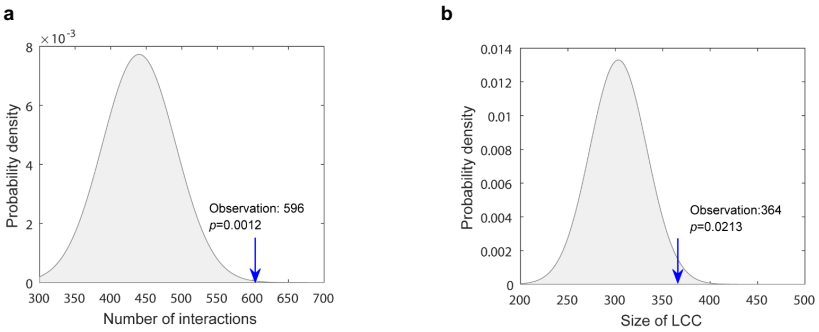
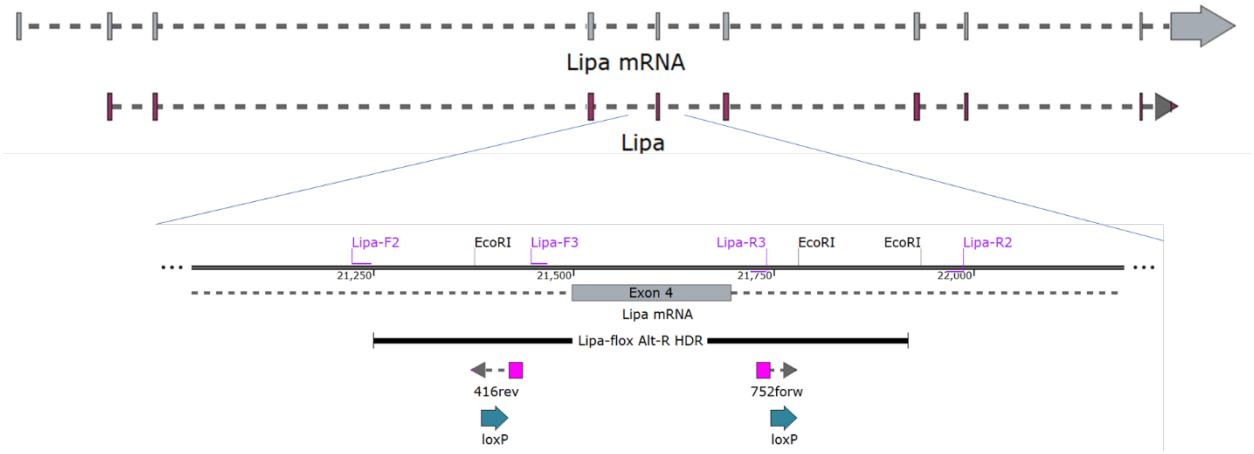


Figure S9. Fasting differentially expressed genes form a discrete subnetwork (module) in the human interactome.

a. The subnetwork has significantly more interactions than randomly expected.
b. The subnetwork has a significantly greater largest connected component (LCC) than randomly expected.



PCR	Forward	5' >3' Sequence	Reverse	5' >3' Sequence
5' LoxP site	Lipa-F2	AGGTATGCAAAACAGAAGAAAGAAA	Lipa-R3	CGAACGATACAGACAGACGC
3' LoxP site	Lipa-F3	GTTTGCTGAGAAGGTTGGAGC	Lipa-R2	GAGTTAACCTAGCAGCCCACA

Figure S10. Generation of the floxed *Lipa* mouse for conditional loss of function.
Schematic of the "*Lipa-flox*" allele. Lipa exons are in grey, and the coding sequence is in purple. The region surrounding exon 4 is detailed, with the relative position of the primers and EcoRI site. The SpyCas9 target sites are represented pink arrows and the LoxP site by blue arrows. Genotyping primers used for these mice are shown in the embedded table.

Table S1. The overlapping of fasting genes with aging-related endophenotypes.
The significance of network proximity was evaluated by creating 1000 random modules of the same size and comparing the observed proximity value with the null model (random control) through fitting normal distributions.

	Overlap with 732 fasting genes
Inflammation (909)	83 (p=2.54e-10)
Reactive oxygen species (359)	30 (p=7.05e-04)
DNA damage (406)	17 (p=0.461)
Cellular senescence (101)	3 (p=0.338)
Lysosomal function (133)	10 (p=0.073)
Genomic instability (189)	18 (p=0.002)

Table S2. Top 50 candidate mediators (out of 386) of the interaction between fasting and aging phenotypes based on normalized BC scores.

Gene names	Normalized BC scores	Gene names	Normalized BC scores
CDK1	1.000	TMEM206	0.067
AR	0.632	DAB2	0.066
PTPN11	0.439	FSCN1	0.065
RB1	0.407	CD14	0.062
IRS1	0.361	RUNX1	0.059
BAX	0.293	PLAU	0.057
H2AFX	0.284	CTSB	0.054
MAPK6	0.224	APOL2	0.053
PLK1	0.221	MYO5A	0.052
AURKA	0.196	ATF3	0.048
BTK	0.195	ACAT1	0.047
CAV1	0.182	ECHS1	0.047
PRDX1	0.181	ADAM17	0.047
RET	0.133	MKNK2	0.045
MITF	0.119	SREBF1	0.044
STMN1	0.111	KCNAB2	0.044
HMOX1	0.107	BIRC5	0.044
PTPN2	0.099	CCL7	0.043
EZH2	0.087	OXCT1	0.042
BUB1B	0.086	CTSD	0.041
TOP2A	0.083	ARPC5	0.041
ADIPOQ	0.083	BUB1	0.040
PRDX2	0.078	FOXM1	0.040
SCD	0.076	HNRNPC	0.040
CCL3	0.071	MDK	0.038

Table S3. Proximity of cardiometabolic disease modules to fasting module.
The significance of network proximity was evaluated by creating 1000 random modules of the same size and comparing the observed proximity value with the null model (random control) through fitting normal distributions.

	Overlap with fasting genes (732)	The proximity of fasting module
Type 2 diabetes (125)	12 (p= 0.010)	1.31 (p=0.010)
Non-alcoholic steatohepatitis (376)	29 (p=0.003)	1.26 (p=3.68e-07)
Coronary artery disease (1093)	89 (p=1.63e-08)	1.33 (p=1.23e-07)
Lipodystrophy (251)	22 (p=0.002)	1.21 (p=1.25e-09)

Table S4. Proximity of aging disease modules to fasting module.
The significance of network proximity was evaluated by creating 1000 random modules of the same size and comparing the observed proximity value with the null model (random control) through fitting normal distributions.

	Overlap with fasting genes (732)	The proximity of fasting module
HFPEF (78)	7 (p=0.058)	1.27 (p=0.008)
Alzheimer's disease (702)	58(p=3.87e-06)	1.32 (p=1.56e-08)
Parkinson's disease (219)	69 (p=5.0-06)	1.28 (p=4.83e-05)
COPD (698)	57 (p=6.68e-6)	1.29 (p=3.7e-09)
Breast Neoplasms (953)	66 (p=2.17e-4)	1.27 (p=2.43e-12)
Endometrial Neoplasms (332)	19 (p=0.156)	1.23(p=2.49e-12)
Prostate Cancer (152)	9 (p=0.235)	1.28 (p=1.56e-04)
Osteoporosis (924)	57 (p=0.007)	1.35 (p=1.4e-04)
Down Syndrome (140)	15 (p=0.001)	
Progeria (127)	8 (p=0.204)	1.23 (p=3.87e-05)
Turner Syndrome (155)	7 (p=0.463)	1.35 (p=0.0086)

Table S5. Network proximity of candidate anti-aging drugs to fasting genes and fasting module. The significance of network proximity was evaluated by creating 1000 random modules of the same size and comparing the observed proximity value with the null model (random control) through fitting normal distributions.

	Proximity to 732 fasting genes (p-value)	Proximity to fasting module (p-value)
Dasatinib, 23 targets	1.04 (p=0.00077)	1.26 (p=0.0013)
Quercetin, 27 targets	1.22 (p=0.0255)	1.04 (p=2.9E-6)
Rapamycin, 3 targets	1.0 (p=0.0988)	1.0 (p=0.031)
Metformin, 13 targets	1.31 (p=0.202)	1.54(p=0.322)

Table S6. qPCR mouse primer details

Gene	qPCR oligo sequence
Atp6v1e1_F	AAAAGATACGACCCGTTACCAAG
Atp6v1e1_R	GCACGATCATTCGAGGCTC
Lipa_F	CTGGTGAGGAACACTCGGTC
Lipa_R	AGCCGTGCTGAAGATACACAA
Lipe_F	CCAGCCTGAGGGCTTACTG
Lipe_R	CTCCATTGACTGTGACATCTCG
Pnpla2_F	ATGTTCCCGAGGGAGACCAA
Pnpla2_R	GAGGCTCCGTAGATGTGAGTG
Mcoln1_F	GCTGGGTTACTCTGATGGGTC
Mcoln1_R	CCACCACGGACATAGGCATAC
Mgll_F	CGGACTTCCAAGTTTTTGTCAGA
Mgll_R	GCAGCCACTAGGATGTGAGTG
Mitf_F	CAAATGGCAAATACGTTACCCG
Mitf_R	CAATGCTCTTGCTTCAGACTCT
Tfe3_F	ATCTCTGTGATTGGCGTGTCT
Tfe3_R	GAACCTTGAGTACCTCCCTGG
Tfeb_F	AAGGTTCGGGAGTATCTGTCTG
Tfeb_R	GGGTTGGAGCTGATATGTAGCA
Tfec_F	TAATGATCCTGATATGCGCTGGA
Tfec_R	AGCTGACGGTTAGCATGTTCTA
Ulk-1_F	AAGTTCGAGTTCTCTCGCAAG
Ulk-1_R	CGATGTTTTCGTGCTTTAGTTCC
Uvrag_F	ACATCGCTGCTCGGAACATT
Uvrag_R	CTCCACGTCGGATTCAAGGAA
Gapdh_F	TGGCCTTCCGTGTTCCTAC
Gapdh_R	GAGTTGCTGTTGAAGTCGCA
Casp3_F	ATGGAGAACAACAAAACCTCAGT
Casp3_R	TTGCTCCCATGTATGGTCTTTAC
Casp9_F	TCCTGGTACATCGAGACCTTG
Casp9_R	AAGTCCCTTTCGCAGAAACAG
Atg5_F	ATGCGGTTGAGGCTCACTTTA
Atg5_R	GGTTGATGGCCCAAAACTGG
Atg7_F	TCTGGGAAGCCATAAAGTCAGG
Atg7_R	GCGAAGGTCAGGAGCAGAA
Lc3a_F	GACCGCTGTAAGGAGGTGC
Lc3a_R	AGAAGCCGAAGGTTTCTTGGG
Lc3b_F	TTATAGAGCGATACAAGGGGGAG

Lc3b_R	CGCCGTCTGATTATCTTGATGAG
Fgf21_F	GTGTCAAAGCCTCTAGGTTTCTT
Fgf21_R	GGTACACATTGTAACCGTCCTC
Ppara_F	AACATCGAGTGTCGAATATGTGG
Ppara_R	CCGAATAGTTCGCCGAAAGAA
Pparg_F	TCGCTGATGCACTGCCTATG
Pparg_R	GAGAGGTCCACAGAGCTGATT
Bdh1_F	TTCCCCTTCTCCGAAGAGC
Bdh1_R	CCCAGAGGGTGCATCTCATAG
Hmgcs1_F	CGGATCGTGAAGACATCAACTC
Hmgcs1_R	CGCCCAATGCAATCATAGGAA
G6pc_F	CGACTCGCTATCTCCAAGTGA
G6pc_R	GGGCGTTGTCCAAACAGAAT
Pck1_F	CTGCATAACGGTCTGGACTTC
Pck1_R	GCCTTCCACGAACTTCCTCAC
Cd36_F	AGATGACGTGGCAAAGAACAG
Cd36_R	CCTTGGCTAGATAACGAACTCTG
Plin2_F	CTTGTGTCCTCCGCTTATGTC
Plin2_R	GCAGAGGTCACGGTCTTCAC
Acacb_F	AGGTTCCAGATGCTAATGGGT
Acacb_R	GCCCAGGATAAAGCTGGTCAT
Agpat2_F	CTTCAAGTACGTGTATGGCCTT
Agpat2_R	CTGTGAACATTAGCTCACGCT
Cidec_F	TGTCGTGTTAGCACCGCAG
Cidec_R	TTGCGCTGTTCTGATGGGG
Cyp4a10_F	TTCCCTGATGGACGCTCTTTA
Cyp4a10_R	GCAAACCTGGAAGGGTCAAAC
Ehhadh_F	ATGGCTGAGTATCTGAGGCTG
Ehhadh_R	ACCGTATGGTCCAAACTAGCTT
Elovl_F	GAAAAGCAGTTCAACGAGAACG
Elovl_R	AGATGCCGACCACCAAAGATA
Vnn1_F	TGTAACACCAGCGACTCTCAC
Vnn1_R	CCTGGGAATCAAACACCACAT

Table S7. qPCR human primer details

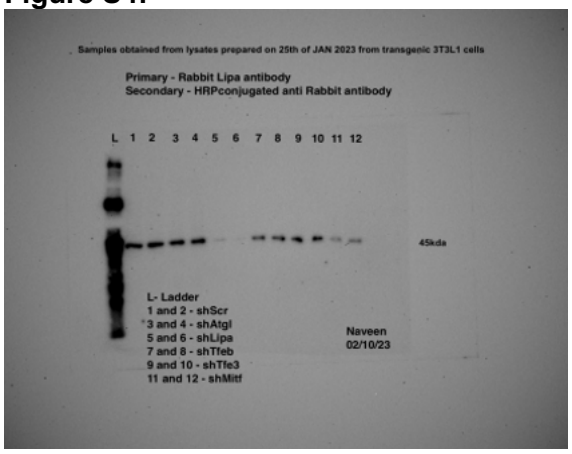
Gene name	qPCR oligo sequence
TFEB_F	ACCTGTCCGAGACCTATGGG
TFEB_R	CGTCCAGACGCATAATGTTGTC
TFEC_F	GGACAACCACAACCTCATTGA
TFEC_R	CACTTGATGTACTCCACTGATGC
MITF_F	CCTTCTCTTTGCCAGTCCATCTTC
MITF_R	GATCAATCAAGTTTCCCGAGACAG
TFE3_F	TGCCTGTGTCAGGGAATCTG
TFE3_R	CGACGCTCAATTAGGTTGTGAT
LIPA_F	CCCACGTTTGCACTCATGTC
LIPA_R	CCCAGTCAAAGGCTTGAAACTT
PNPLA2_F	GAGATGTGCAAGCAGGGATAC
PNPLA2_R	CTGCGAGTAATCCTCCGCT
MGLL_F	TCGTCAGGGATGTGTTGCAG
MGLL_R	AGGCGAAATGAGTACCATGCC
LIPE_F	AGGAGCCAGCATTGAGACAAA
LIPE_R	CGCAGGTGTTGATTCAGCTTC

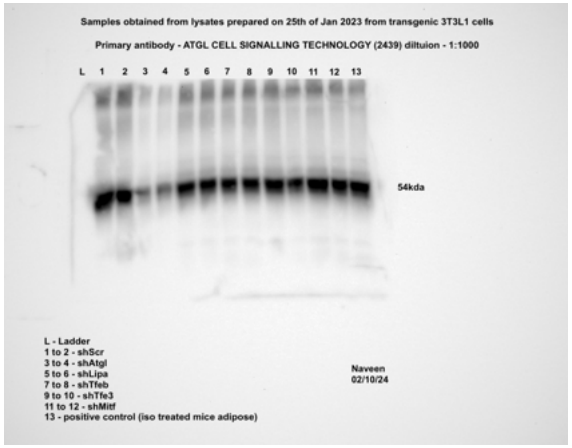
Table S8. Antibodies used in western blot and immunofluorescence

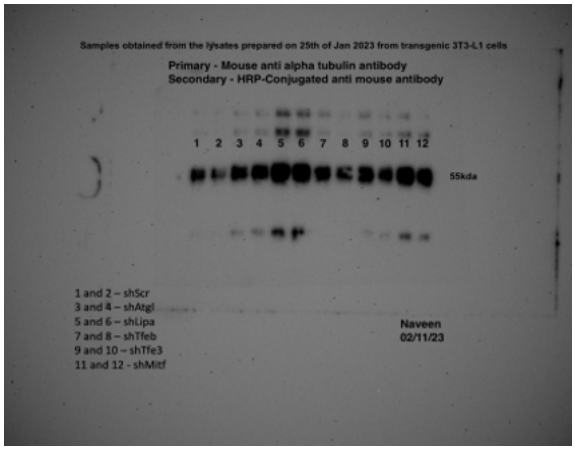
Antibody	Company	Catalog number
Perilipin	Cell signaling technology	D1D8
Lamp1	Invitrogen	14-1071-85
Atgl	Cell signaling technology	2439
Lipa (LAL)	Proteintech	12956-1-AP
Tfeb	Proteintech	13372-1-AP
Tfe3	Cell signaling technology	14779
Mitf	Cell signaling technology	12590
b-Actin	Cell signaling technology	D6A8
aTubulin	Proteintech	66031-1-Ig
anti-Rabbit HRP conjugated	Cell signaling technology	7074
anti-Rat HRP conjugated	Cell signaling technology	7077
anti-mouse HRP conjugated	Invitrogen	32430
Conjugated secondary antibody	**Company**	**Catalog number**
Anti-rabbit antibody	Invitrogen	A32734
Anti-rat antibody	Invitrogen	A21470

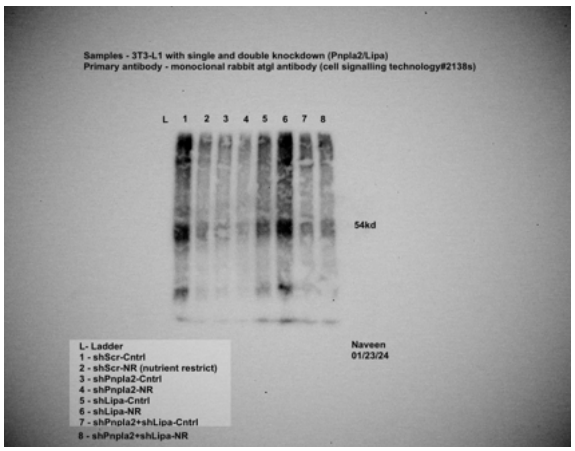
Uncropped blots, supplemental figures

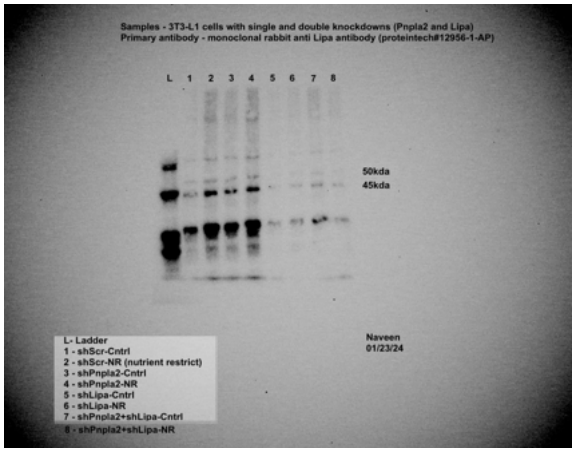
Figure S4:








Samples - 3T3-L1 with single and double knockdown (Pnpla2/Lipa)
Primary antibody - monoclonal rabbit atgl antibody (cell signalling technology#2138s)
L 1 2 3 4 5 6 7 8
54kd
L- Ladder
1 - shScr-Cntrl
2 - shScr-NR (nutrient restrict)
3 - shPnpla2-Cntrl
4 - shPnpla2-NR
5 - shLipa-Cntrl
6 - shLipa-NR
7 - shPnpla2+shLipa-Cntrl
8 - shPnpla2+shLipa-NR
Naveen
01/23/24


Samples - 3T3-L1 cells with single and double knockdowns (Pnpla2 and Lipa)
Primary antibody - monoclonal rabbit anti Lipa antibody (proteintech#12956-1-AP)
L 1 2 3 4 5 6 7 8
50kda
45kda
L- Ladder
1 - shScr-Cntrl
2 - shScr-NR (nutrient restrict)
3 - shPnpla2-Cntrl
4 - shPnpla2-NR
5 - shLipa-Cntrl
6 - shLipa-NR
7 - shPnpla2+shLipa-Cntrl
8 - shPnpla2+shLipa-NR
Naveen
01/23/24

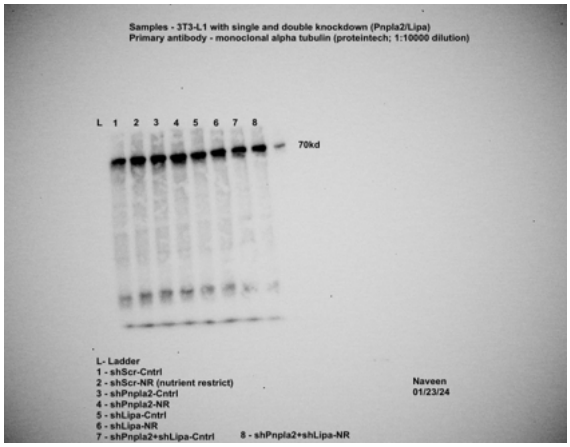
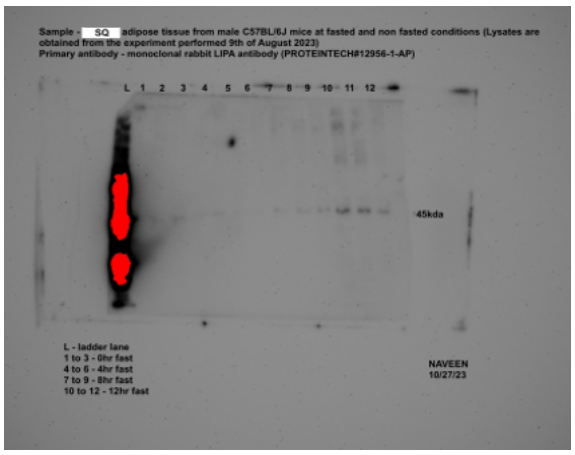
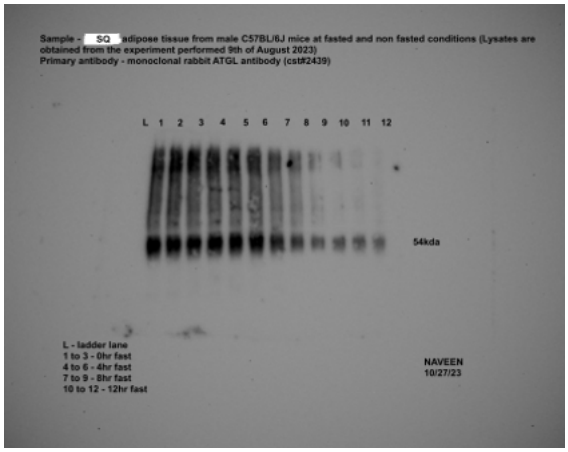
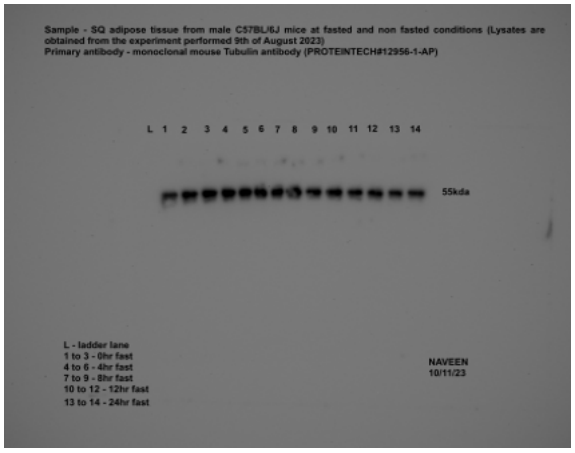
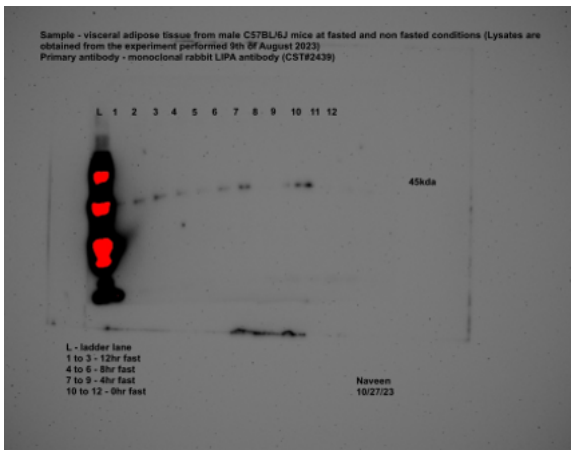

Samples - 3T3-L1 with single and double knockdown (Pnpla2/Lipa)
Primary antibody - monoclonal alpha tubulin (proteintech; 1:10000 dilution)
L 1 2 3 4 5 6 7 8
70kd
L- Ladder
1 - shScr-Cntrl
2 - shScr-NR (nutrient restrict)
3 - shPnpla2-Cntrl
4 - shPnpla2-NR
5 - shLipa-Cntrl
6 - shLipa-NR
7 - shPnpla2+shLipa-Cntrl
8 - shPnpla2+shLipa-NR
Naveen
01/23/24

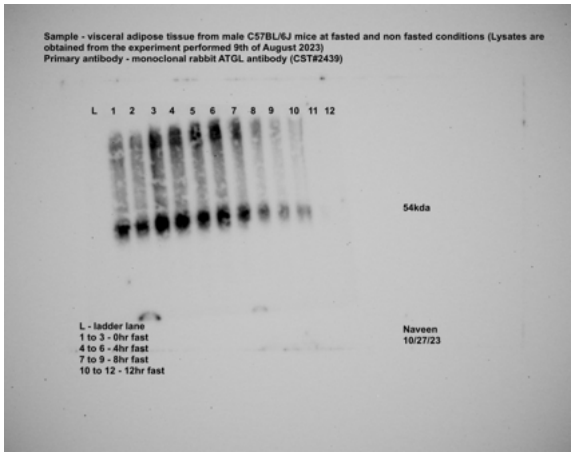
Figure S5

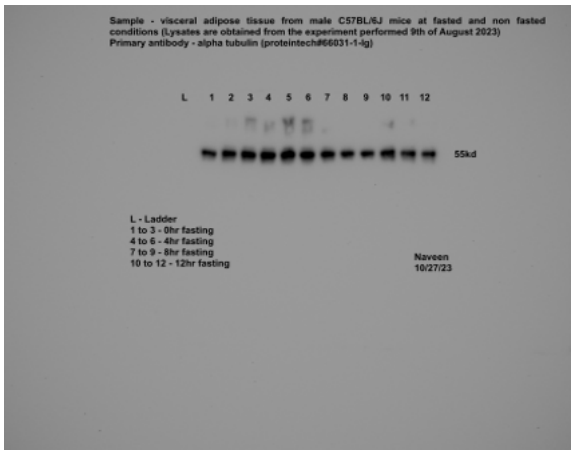

Sample - SQ adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit LIPA antibody (PROTEINTECH#12956-1-AP)
L 1 2 3 4 5 6 7 8 9 10 11 12
45kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
NAVEEN
10/27/23

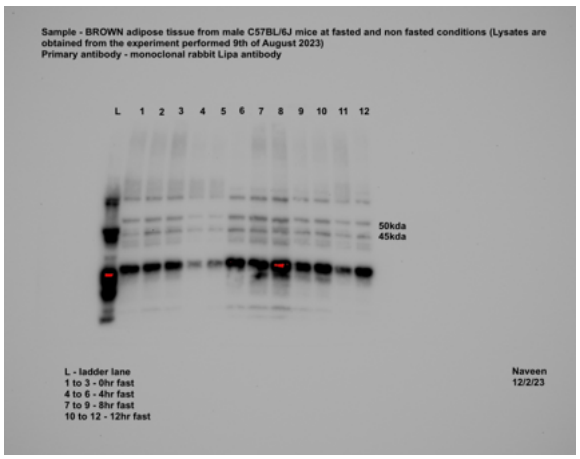

Sample - SQ adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit ATGL antibody (cst#2439)
L 1 2 3 4 5 6 7 8 9 10 11 12
54kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
NAVEEN
10/27/23

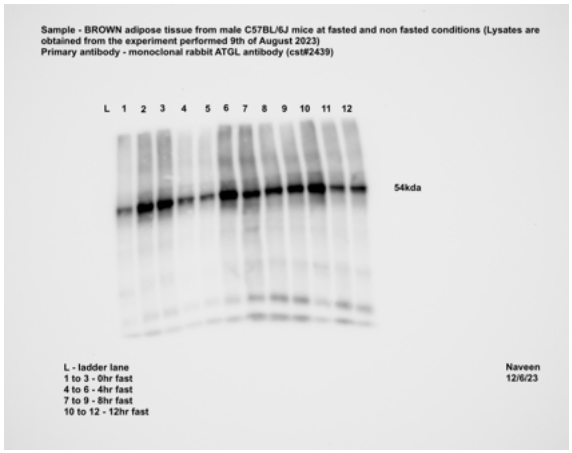

Sample - SQ adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal mouse Tubulin antibody (PROTEINTECH#12956-1-AP)
L 1 2 3 4 5 6 7 8 9 10 11 12 13 14
55kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
13 to 14 - 24hr fast
NAVEEN
10/11/23

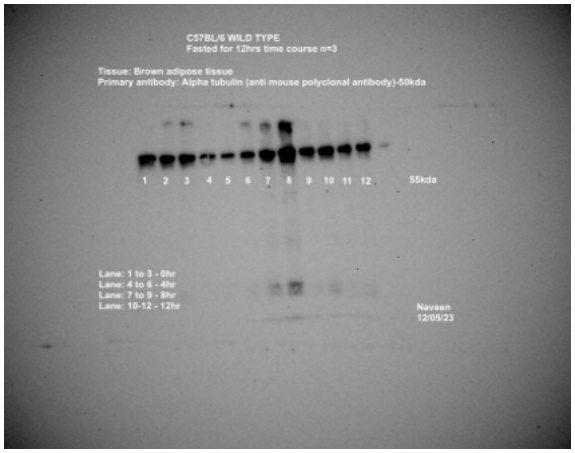

Sample - visceral adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit LIPA antibody (CST#2439)
L 1 2 3 4 5 6 7 8 9 10 11 12
45kda
L - ladder lane
1 to 3 - 12hr fast
4 to 6 - 8hr fast
7 to 9 - 4hr fast
10 to 12 - 0hr fast
Naveen
10/27/23


Sample - visceral adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit ATGL antibody (CST#2439)
L 1 2 3 4 5 6 7 8 9 10 11 12
54kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
Naveen
10/27/23


Sample - visceral adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - alpha tubulin (proteintech#66031-1-Ig)
L 1 2 3 4 5 6 7 8 9 10 11 12
55kd
L - Ladder
1 to 3 - 0hr fasting
4 to 6 - 4hr fasting
7 to 9 - 8hr fasting
10 to 12 - 12hr fasting
Naveen
10/27/23


Sample - BROWN adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit Lipa antibody
L 1 2 3 4 5 6 7 8 9 10 11 12
50kda
45kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
Naveen
12/2/23


Sample - BROWN adipose tissue from male C57BL/6J mice at fasted and non fasted conditions (Lysates are obtained from the experiment performed 9th of August 2023)
Primary antibody - monoclonal rabbit ATGL antibody (cst#2439)
L 1 2 3 4 5 6 7 8 9 10 11 12
54kda
L - ladder lane
1 to 3 - 0hr fast
4 to 6 - 4hr fast
7 to 9 - 8hr fast
10 to 12 - 12hr fast
Naveen
12/6/23


C57BL/6 WILD TYPE
Fasted for 12hrs time course n=3
Tissue: Brown adipose tissue
Primary antibody: Alpha tubulin (anti mouse polyclonal antibody)-50kda
1 2 3 4 5 6 7 8 9 10 11 12
55kda
Lane: 1 to 3 - 0hr
Lane: 4 to 6 - 4hr
Lane: 7 to 9 - 8hr
Lane: 10-12 - 12hr
Naveen
12/05/23