Supplemental Data

Table 1S: Primers for qRT-PCR.

	Accession Number	Forward	Reverse	Species
ATGL	NM_025802.3	TGTGGCCTCATTCCTCCTAC	TCGTGGATGTTGGTGGAGCT	Mouse
ADPN	NM_054088.3	ATTCCCCTCTTCTCTGGCCTA	ATGTCATGCTCACCGTAGAAAGG	Mouse
TGH	NM_053200.2	CTCAGAGACCCACAGAGCCCTTGTC	AAGCTGTGCACGCAGCAAGAGA	Mouse
AADAC	NM_023383.1	TCCCACCCACGTCTGATGAGC	AAGCCCCGCCTGAGAGCCAT	Mouse
CGI58	NM_026179.2	CTTGCTTGGACACAACCTG	GAGGTGACTAACCCTTGATGG	Mouse
Cyclophilin	NM_008907.1	CTTCTTGCTGGTCTTGCCATTCCT	GGATGGCAAGCATGTGGTCTTTG	Mouse
CPT1a	NM_013495.2	TTAACAGCAACTACTACGCC	CCAGAAGACGAATAGGTTTGAG	Mouse
CPT2	NM_009949.1	CAGTTCAGGAAGACAGAAGTG	CGAAGTGTCTTCAGAAACCG	Mouse
LCAD	NM_007381.3	AAACGTCTGGACTCCGGTTC	GTACCACCGTAGATCGGCTG	Mouse
MCAD	NM_007382.4	TCAAGATCGCAATGGGTGCT	GCTCCACTAGCAGCTTTCCA	Mouse
SCAD	NM_007383.2	GTGACCTGCAACCGAGAAGA	AGGGCGGCTCAAATGAAGAA	Mouse
SIRT3	NM_022433.2	CGCTAAACTTCTCCCGGGTT	ACACAGAGGGATATGGGCCT	Mouse
PDK4	NM_013743.2	GCCAACCCTACGGATCCTAAC	TGTTCACTAAGCGGTCAGGC	Mouse
ATGL	NM_020376.3	AAGCGGAGGATTACTCGCAG	AAGCGGATGGTGAAGGACAG	Human
Cyclophilin	NM_021130	AAGACTGAGTGGTTGGATGG	CGAGAGCACAAAGATTCTAGG	Human
CPT1a	NM_001031847.2	AGTTCTCTTGCCCTGAGACG	GTGATGTCCATGGTCTCCTCC	Human
CPT2	NM_000098.2	GTAGCACTGCCGCATTCAAG	GCCATGGTACTTGGAGCACT	Human
LCAD	NM_001608.3	ATACGGTTGCCAGCTAGTGC	TGCACTGTCTGTAGGTGAGC	Human
MCAD	NM_000016.4	GCTGCAGGGTCCTGAGAAGTA	TATTCTGCAGCCACTGGGATG	Human
SCAD	NM_000017.2	GGAACATCTCTTCCCAGCGG	GAGGGCAAAGCAGCCAATTT	Human
SIRT3	NM_001017524.2	TGGCGGCAGGGACGATTATT	ATCGTACTGCTGGAGGTTGC	Human
PDK4	NM_002612.3	TGGTTTTGGTTACGGCTTGC	AGTGTCCCTCTTCACATGGC	Human
MTTP	NM_008642.1	CACAATTATGACCGTTTCTCCA	CAATCACCACCTGACTACCA	Mouse
FGF21	NM_020013.4	TACACAGATGACGACCAAGAC	AAAGTGAGGCGATCCATAGAG	Mouse

LXRα	NM_013839.3	GTTTCTCCTGATTCTGCAACG	ACCCTATCCCTAAAGCAACC	Mouse
PGC1β	NM_133249.2	GTGTTCGGTGAGATTGTAGAG	CAGATGTGGGATCATAGTCAG	Mouse
CYP7α	NM_007824.2	TGGAATAAGGAGAAGGAAAGTAGG	AGGGAGTTTGTGATGAAGTG	Mouse
PPARγ2	NM_011146.3	GAGCACTTCACAAGAAATTACC	TCTACTTTGATCGCACTTTGG	Mouse

Supplementary Figures

FIGURE 1S. Effect of diet and Elov/5 activity on glycerol-3-phosphate 2-O-acyl transferase 1 (GPAT1) expression in livers of lean and obese mice. Mouse liver RNA from fasted and refed lean and obese mice infected with either Ad-Luc or Ad-Elov/5 was prepared and assayed for GPAT1 and cyclophilin mRNA by qRT-PCR. GPAT1 mRNA abundance is represented as mRNA Abundance-Fold Change from fasted lean (LFD) Ad-Luc infected mice; mean \pm SD, n=6; *a*, p<0.05 versus fasted lean LFD-fed mice infected with Ad-Luc; *b*, p<0.05 versus fasted lean LFD-fed mice infected with Ad-Elov/5; *c*, p<0.05 versus refed lean LFD-fed mice infected with Ad-Luc; one-way ANOVA plus post-hoc HSD.

FIGURE 2S. *Diet and ElovI5 effects on hepatic microsomal transfer protein (MTTP) expression.* Mouse liver RNA from fasted lean (LFD) and obese (HFD) mice infected with either Ad-Luc or Ad-ElovI5 was assayed for MTTP and cyclophilin mRNA by qRT-PCR. MTTP mRNA was represented relative to cyclophilin as mean <u>+</u> SD, n=6.

FIGURE 3S. Hepatic nuclear abundance of PPAR subtypes in fasted lean and obese mice. Panels A & B: Hepatic nuclear extracts from fasted lean and obese mice infected with Ad-Luc or Ad-ElovI5 were assayed for PPAR α and TATA-binding protein (TBP) [Methods]. Panel A is the representative immunoblot with extracts from 2 separate mice per group. Panel B is the quantified level PPAR α relative to the loading control TBP; mean <u>+</u> S.D., n=4. **a**, p<0.05 versus LFD fed mice infected with Ad-Luc. Panels C & D. The nuclear abundance of PPAR α , PPAR δ , PPAR γ 1 and PPAR γ 2 was quantified as

described above. Livers from obese (HFD-fed) mice infected with Ad-Luc or Ad-ElovI5 were used in this analysis. <u>Panel C</u> is the representative immunoblot with extracts from 3 mice/group. <u>Panel D</u> is the quantified level of the PPAR subtypes. Results are from 2 separate studies and are expressed as mean \pm SD, n=6. *a*, *p*≤0.05 versus high fat (HF) diet fed Ad-Luc infected mice; one-way ANOVA with post-hoc HSD.

FIGURE 4S. *Fatty acid oxidation of saturated, mono- and polyunsaturated fatty acids in AML12 cells.* AML12 cells were treated with ¹⁴C-label fatty acids at 100 μ M in serum-free media containing 33 μ M bovine serum albumin (BSA) for 6 hours. Cells and media were harvested; cells were assayed for total protein and media was assayed for acid soluble material (ASM), an indicator of FAO [Methods]. Results are expressed as ASM, nmoles/mg protein and represented as the average <u>+</u> range of duplicate samples.

Figure 5S. Regulation of PDK4, ATGL and Rictor mRNA by fatty acids, PPAR agonist and antagonist in HepG2 cells. <u>Panels A & B</u>. HepG2 cells were treated with EPA (100 μ M), GW0742 (1 μ M) [Panels A & B] or cis-VA (100 μ M) [Panel C) in serum free media containing 33 μ M BSA for the times indicated in the figure. RNA was extracted and PDK4, ATGL, rictor and cyclophilin mRNA was quantified by qRT-PCR. Results are expressed as mRNA abundance-Fold Change for PDK4 (<u>Panel A</u>), ATGL (<u>Panel B</u>); and Rictor (<u>Panel C</u>); mean <u>+</u> SD, n=3. <u>Panel D</u>. HepG2 cells were treated with vehicle (Veh) or cis-VA (100 μ M) with BSA as above in the absence or presence of PPAR subtype specific antagonist GW6471 (PPARα, 1 μ M), GSK3787 (PPARβ, 1 μ M) or T0070907 (PPARγ, 4 nM) for 48 hrs. ATGL and cyclophilin RNA was quantified as above and represented as the mean \pm S.D., n=6. *a*, p<0.05 versus vehicle-treated cells. Student's t-test.

Supplemental Fig. 1S



Supplemental Fig. 2S



Supplemental Fig. 3S



Supplemental Fig. 4S



Supplemental Figure 5S:

