Supporting Information

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SI Materials and Methods

Oral Glucose Tolerance Test. For the oral glucose tolerance tests, 16-h fasted mice were challenged with a 1.5-mg glucose per gram body weight glucose load. Blood glucose was measured from tail-vein blood collected at the designated times, using an automatic glucose monitor (OneTouch; LifeScan).

Cell Culture and Primary Hepatocyte Isolation. Alpha mouse liver (AML) 12 hepatocytes (ATCC) were grown at 37 °C in DMEM-F-12 (Gibco); 1× insulin, transferrin, and selenium (Invitrogen); 100 nM dexamethasone; and 10% (vol/vol) FBS. Primary mouse hepatocytes were isolated by the two-step collagenase perfusion method from the livers of male C57BL/6 and C3H/HeJ mice (7 wk old) as previously described (1). Hepatocytes were plated on six-well dishes at 1.0×10^6 cells per well and incubated for 12 h in DMEM containing 10% (vol/vol) FBS to allow cells to attach. Cell counts and viability (Adam cell counter; Digital Bio) were confirmed before use; viability was routinely >80%. After attachment, cells were infected with various adenoviruses, indicated in Figs. 2–4 and Figs. S4 and S5.

Western Blot. For protein preparation from liver tissues, mouse livers (50 mg) were placed in a glass homogenizer containing 0.6 mL Pro-Prep Protein Extraction Solution (Intron Biotechnology). Completely homogenized tissues were incubated on ice for 20 min and centrifuged at 13,400 × *g* at 4 °C for 10 min. Tissue or cell lysates were separated by electrophoresis on SDS/PAGE gels. Gels were transferred onto nitrocellulose membranes and the blots were incubated with blocking buffer [5% (wt/vol) nonfat dried milk in TBST] before incubation with antibodies. Primary antibodies used for blotting were anti-peroxisome proliferator-activated receptor γ (anti-PPARγ), anti–β-actin, anti–α-tubulin, anti-FLAG (Santa Cruz), anti-aP2/422 (2), and anti-adipose differentiation-related protein (anti-ADRP) (Abcam). A mouse monoclonal antibody against monoacylglycerol *O*-transferase 1 (MGAT1) was generated using a synthetic peptide (residues 228–240, GENDLYKQINNPK) of murine MGAT1 by AbClon. Bands were detected with anti-rabbit or anti-mouse IgG conjugated with horseradish peroxidase (Pierce), using the ECL-PLUS detection system (Amersham).

RNA Isolation and PCR Analysis. Total RNA was isolated using TRIzol reagent (Invitrogen) according to the manufacturer's instructions. First-strand cDNA synthesis from 5 μg total RNA was performed using SuperScript II reverse transcriptase (Invitrogen) primed by random hexamer primers. Real-time qPCR was performed using SYBR Green Master mix (Applied Biosystems) in a total volume of 20 μL with a Step One instrument (Applied Biosystems). A standard curve was used to calculate mRNA level relative to that of a control gene, RPLP0. The primer sequences used in real-time qPCR are as follows: PPARγ2, 5′-CTCTGGGAGATTCTCCTGTTGA-3′, 5′-GGTGGGCCAGAATGGCATCT-3′; aP2/422, 5′-TCTCCAGTGAAAACTTCGAT-3′, 5′-TTACGCTGATGATCATGTTG-3′; FSP27, 5′-TCCAGGACATCTTGAAACTT-3′, 5′-GGCTTGCAAGTATTCTTCTGT-3′; Cd36, 5′-TGCACCACATATCTACCAAA-3′, 5′-TTGTAACCCCACAAGAGTTC-3′; SREBP1a, 5′-AGATGTGCGAACTGGACACA-3′, 5′-CATCTTTAAAGCAGCGGGTG-3′; SREBP1c, 5′-GGAGCCATGGATTGCACATT-3′, 5′-CATCTTTAAAGCAGCGGGTG-3′; ACCα, 5′-GAGGAAGTTGGCTATCCAGT-3′, 5′-GTTGAACCTGTCTGAAGAGG-3′; FAS, 5′-AAGCCGTTGGGAGTGAAAGT-3′, 5′-CAATCTGGATGGCAGTGAGG-3′; ACL, 5′-ACATTGCAGACCTGGATGCCA-3′, 5′-TTAAGGAGGAAGTTGGCAGT-3′; AGPAT2, 5′-AGCGGACAGAAGAAACTGGA-3′, 5′-TGAAGTAGACACCCCCAAGG-3′; MGAT1, 5′-CTGGTTCTGTTTCCCGTTGT-3′, 5′-TGGGTCAAGGCCATCTTAAC-3′; DGAT1, 5′-TTCCGCCTCTGGGCATT-3′, 5′-AGAATCGGCCCACAATCCA-3′; and Rplp0, 5′-GCAGGTGTTTGACAACGGCAG-3′, 5′GATGATGGAGTGTGGCACCGA-3′. Also, data in Fig. S1*C* were carried out by RT-PCR with agarose gel electrophoresis, using the following primers: PPARγ1, 5′-ACAGGACTGTGTGACAGACA-3′, 5′-GCTGGAGAAATCAACTGTGG-3′; PPARγ2, 5-TATGGGTGAAACTCTGGGAG-3′, 5′-GCTGGAGAAATCAACTGTGG-3′; and GAPDH, 5′-ACCACAGTCCATGCCATCAC-3′, 5′-TCCACCACCCTGTTGCTGTA-3′.

Gene Silencing with siRNA. The siRNA was designed to target PPARγ mRNA. Its target sequence was 5′-GAAGUUCAAUGCACUGGAATT-3′ (siPPARγ). The scrambled siRNA was purchased from GenePharma. They were introduced into primary mouse hepatocytes using Lipofectamine RNAiMAX (Invitrogen).

Preparation of Recombinant Adenovirus. Murine PPARγ2 and MGAT1 cDNAs were cloned into pcDNA3.0 vector or FLAG-tagged pcDNA3, respectively. Recombinant adenoviruses expressing PPARγ2 and MGAT1-FLAG were prepared as follows. Briefly, we first constructed an E1 shuttle vector expressing murine PPARγ2 and MGAT1. The newly constructed E1 shuttle vector was linearized with PmeI digestion. The linearized shuttle vector was then cotransformed into *Escherichia coli* BJ5183 along with pAdEasy1 vector. All viruses were propagated in 293 cells and purified by CsCl density purification, dissolved in 1× HBSS (Invitrogen), and stored at −80 °C. Viral particle numbers were calculated from measurements of absorbance at 260 nm. The multiplicity of infection was calculated from viral particle numbers. Recombinant adenovirus containing the GFP gene was used as a control. Similarly, ad-shRNA for MGAT1 was constructed as previously described (3). On the basis of the design principles for shRNA construction, we selected RNAi target sites that efficiently suppressed the MGAT1 expression among three RNAi samples tested: sh-MGAT1-a, 5′-UUUCACCCUCAUGGAAUAUUCGUGCCU-3′; and sh-MGAT1-b, 5′-CAAGACGCAAUGUAUGAUUCAAUGGGA-3′. To prepare recombinant adenovirus expressing shRNA against MGAT1, annealed shRNA DNA nucleotides were cloned into pBS-U6 and subcloned into the AdTrack(ΔCMV) vector, using an adenoviral vector system. The AdTrack(ΔCMV)-U6-shMGAT1 vector and the adenovirus vector AdEasy were linearized by restriction enzyme. The linearized AdTrack(ΔCMV)-U6-shMGAT1 was cotransformed into *E. coli* BJ5183 with AdEasy for homologous recombination. The complete Ad-shMGAT1 virus was recovered by transfection of PacI-digested DNA into human embryonic kidney (HEK) 293 cells by a lipofectamine-based procedure. Large-scale purification of all adenoviruses was performed by ultracentrifugation with cesium chloride according to standard techniques. Ad-US control RNAi was used as a control (3).

Histological Analysis. Livers and white adipose tissue (WAT) were removed and fixed with 10% (vol/vol) formalin and embedded in paraffin. Tissue sections were stained with H&E.

Triglyceride (TG) Assay in the Liver and Cultured Hepatocytes. Liver extracts were prepared by homogenization in chloroform:meth-

anol (2:1). TG level was measured using TG assay reagents (Thermo Scientific).

Electrophoretic Mobility Shift Assays (EMSA). Nuclear extracts from C57BL/6 (B6) and C3H mice fed with a chow diet (CD) or a high-fat diet (HFD) were prepared as described in ref. 4. As a control, we used nuclear extract from fully differentiated 3T3-L1 cells. In another experiment, in vitro-translated PPARγ2 and RXRα protein was used. RXRα and PPARγ2 expression vectors were constructed by insertion of cDNAs into pcDNA3.0 (Invitrogen) and used to synthesize RXRα and PPARγ2 protein following the TNT T7 quick master mix (Promega). EMSAs were performed using in vitro-transcribed RXRα and PPARγ2 protein. Double-stranded probes were labeled with [γ-^{32}P]ATP, using T4 polynucleotide kinase. The binding reactions were previously described (4). For supershift assay, we added 1 μg of anti-PPARγ antibody (Santa Cruz). Protein–DNA complexes were resolved from the free probe by electrophoresis at 4 °C on a 4% (wt/vol) polyacrylamide gel in 0.25 × TBE buffer. The dried gels were exposed to X-ray film at −70 °C with an intensifying screen.

Transfection and Luciferase Assay. Cells (293T cells) were transfected with 0.8 μg pGL3-MGAT1 plasmid, 0.1 μg expression vector plasmid, and 10 ng pRL-CMV (Promega), using Lipofectamine (Invitrogen), according to the manufacturer's instructions. After 24 h, the luciferase activities were measured using the Dual Luciferase Reporter Assay System (Promega), according to the manufacturer's instructions. Firefly luciferase activities were standardized to Renilla activities.

Microarray Analysis. DIO-prone C57BL/6 mice and DIO-resistant C3H/HeJ mouse strains were maintained on a CD and a HFD for 12 wk. A total RNA sample was prepared and the gene expression was analyzed by the Agilent Mouse Genome 4×44k oligo chip. Similarly, total RNA was prepared from Ad-PPARγ2–injected mice and subjected to microarray analysis. The preparation of hybridization and the scanning of mouse chips were performed according to the manufacturer's protocols (Genocheck).

TLC. TLC was performed as previously described (5). Briefly, primary hepatocytes were labeled with 1 μCi/mL of [^{14}C]glycerol (ARC) in DMEM containing 5% (vol/vol) FBS, 1.5 mM glycerol, 100 units/mL penicillin, and 100 μg/mL streptomycin at 37 °C in 5% (vol/vol) CO_2. After 4 h, lipids were extracted twice with 1 mL of isopropanol:hexane (2:3) at room temperature for 30 min. Delipidated cells were digested with 1 N NaOH for protein extraction. Lipid extracts were dried under a N_2 stream and redissolved in 300 μL of chloroform. Neutral lipid standards (Sigma) were applied (20 μg) onto a 0.2-mm silica G plate (Macherey-Magel). The chromatograms were developed in hexane:diethyl ether:acetic acid (80:20:1) and exposed to iodine vapor, and the visualized bands indicating diacylglycerol (DAG) and TG were scraped into scintillation vials containing 5 mL of counting mixture.

Chromatin Immunoprecipitation (ChIP) Assay. Formaldehyde was used to cross-link the cells for 10 min. Cells or liver tissue were transferred and rinsed with PBS, lysed, and subjected to chromatin shearing by sonication. Lysate was washed and chromatin immunoprecipitations were performed using the ChIP Assay Kit (Upstate) according to the manufacturer's protocol with anti–acetyl-Histone H3 or anti–dimethyl-Histone H3 antibody (Millipore) or anti-PPARγ antibody (Santa Cruz). Input control or ChIP samples were used as a template in PCR, using the following primers to amplify the PPARγ or MGAT1 promoter. Primers were as follows: PPARγ1 pair primers, 5′-GAAGTCACACTCTGACAGG-A-3′, 5′-CTTGTCTGTCACACAGTCCT-3′; PPARγ2 pair primers, 5′-CCAAATACGTTTATCTGGTGTTTC-3′, 5′-CGTTGC-TACATTGTCTCGC-3′; and MGAT1 pair primers, 5′-GCCAA-GAAGTTACAAATGGTGCCC-3′, 5′-CTGTCTTTCCAGCCC-TTGAG-3′.

Methylation Analysis. Genomic DNA was purified with the Wizard genomic DNA purification kit (Promega). Methylation analysis was performed by bisulfite conversion of genomic DNA, using the MethylDetector kit (Zymo Research). The primer sequences to amplify the PPARγ1 promoter region are sense 5′-TTTTA-GYGGTTGTGAGGAGTAAGG-3′ and antisense 5′-TCAAA-ATATAACTTC TCCTCAACCC-3′. The PCR product was cloned using the TOPO TA Cloning kit (Invitrogen).

1. Kim JW, Ahn YH (1998) CCAAT/enhancer binding protein regulates the promoter activity of the rat GLUT2 glucose transporter gene in liver cells. *Biochem J* 336:83–90.
2. Kim JW, Tang QQ, Li X, Lane MD (2007) Effect of phosphorylation and S-S bond-induced dimerization on DNA binding and transcriptional activation by C/EBPbeta. *Proc Natl Acad Sci USA* 104:1800–1804.
3. Lee MW, et al. (2010) Regulation of hepatic gluconeogenesis by an ER-bound transcription factor, CREBH. *Cell Metab* 11:331–339.
4. Park SK, et al. (2004) CCAAT/enhancer binding protein and nuclear factor-Y regulate adiponectin gene expression in adipose tissue. *Diabetes* 53:2757–2766.
5. Cortés VA, et al. (2009) Molecular mechanisms of hepatic steatosis and insulin resistance in the AGPAT2-deficient mouse model of congenital generalized lipodystrophy. *Cell Metab* 9:165–176.

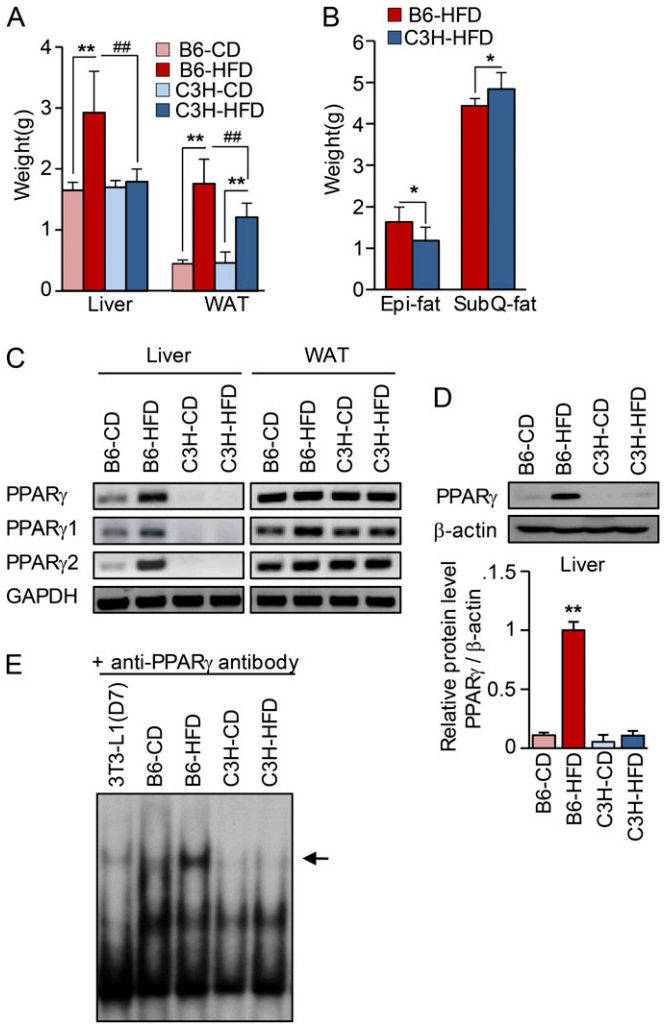

Fig. S1. Changes of liver and fat weight and hepatic PPARγ expression and its activity in B6 and C3H mice fed a CD or a HFD. (*A*) Liver and epididymal white adipose tissue (WAT) weight of B6 and C3H mice fed a CD or a HFD (n = 8 and 9, respectively). Data were analyzed by two-way ANOVA (**$P < 0.01$ vs. CD-fed mice, respectively, $^{\#\#}P < 0.01$ vs. B6-HFD). (*B*) Epididymal and s.c. fat weight of B6 and C3H mice fed a HFD (n = 8 and 9, respectively, *$P < 0.05$). (*C*) RT-PCR analysis of PPARγ, PPARγ1, and PPARγ2 mRNA levels in liver and epididymal adipose tissue from B6 and C3H mice fed a CD or a HFD at 12 wk. (*D*) Western blot analysis of transcription factors in livers from B6 and C3H mice fed a CD or a HFD at 12 wk. (*Lower*) Densitometry results from two different mouse groups. Data represent the mean ± SD. **$P < 0.01$. (*E*) EMSA experiment showing PPARγ binding activity using nuclear extracts prepared from B6 and C3H mice fed a CD or a HFD at 12 wk. Nuclear extract from differentiated 3T3-L1 adipocytes (day 7) was used as a positive control for PPARγ bindng to PPRE on the CD36 promoter. The bands shifted by anti-PPARγ antibody (arrow) indicate the specific binding.

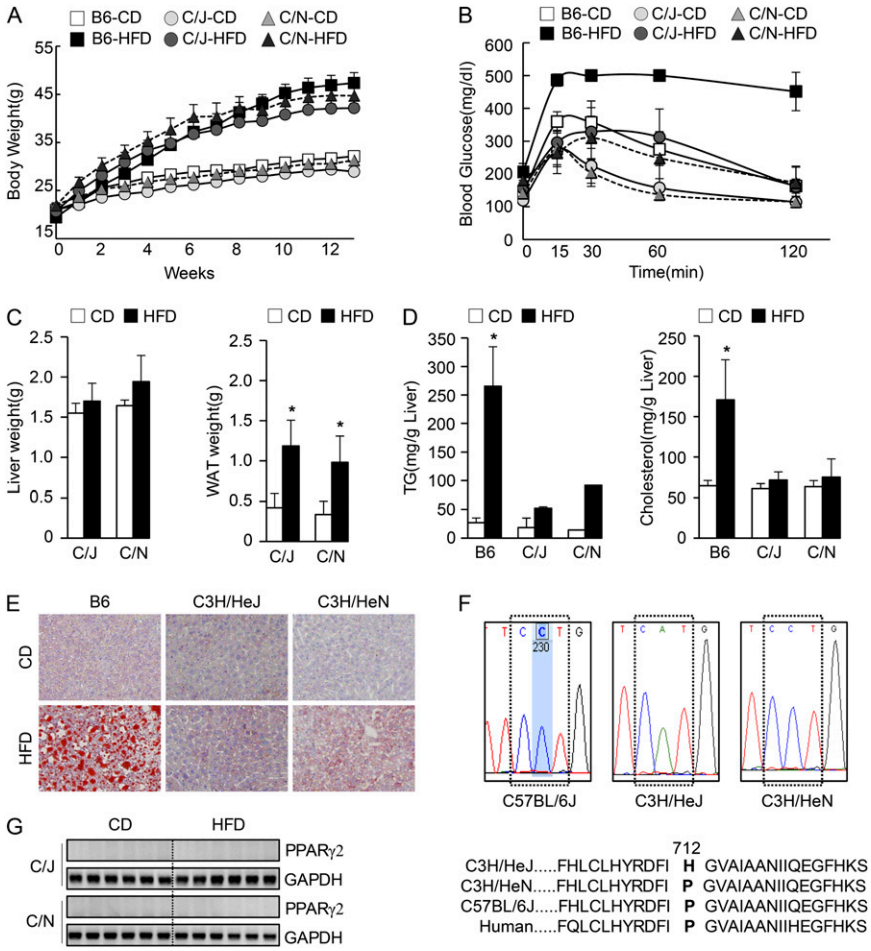

Fig. S2. C3H/HeN and C3H/HeJ mice showed a similar phenotype in response to a HFD. (*A*) Body weights of B6, C3H/HeJ, and C3H/HeN mice fed a CD or a HFD (CD and HFD: $n = 6$, respectively). (*B*) Blood glucose during a glucose tolerance test ($n = 6$, respectively). (*C*) Liver and epididymal white adipose tissue weight of C3H/HeJ and C3H/HeN mice fed a CD or a HFD ($n = 6$, respectively, $^*P < 0.01$). (*D*) Total TG and cholesterol contents in B6, C3H/HeJ, and C3H/HeN mice ($n = 6$, respectively, $^*P < 0.01$). (*E*) Oil-Red-O lipid staining in liver sections from the mice in CD and HFD groups at 12 wk. Red staining indicates lipid deposition. (*F*) The TLR4 cDNA was amplified by RT-PCR from liver RNA derived from B6, C3H/HeJ, and C3H/HeN mice. The mutation, at position 712, lies in the most conserved portion of the TLR4 sequence and is located within the cytoplasmic domain. (*G*) PPARγ expression in liver tissues of C3H/HeJ and C3H/HeN mice fed a CD or a HFD. Total RNA of liver tissue was prepared and subjected to RT-PCR.

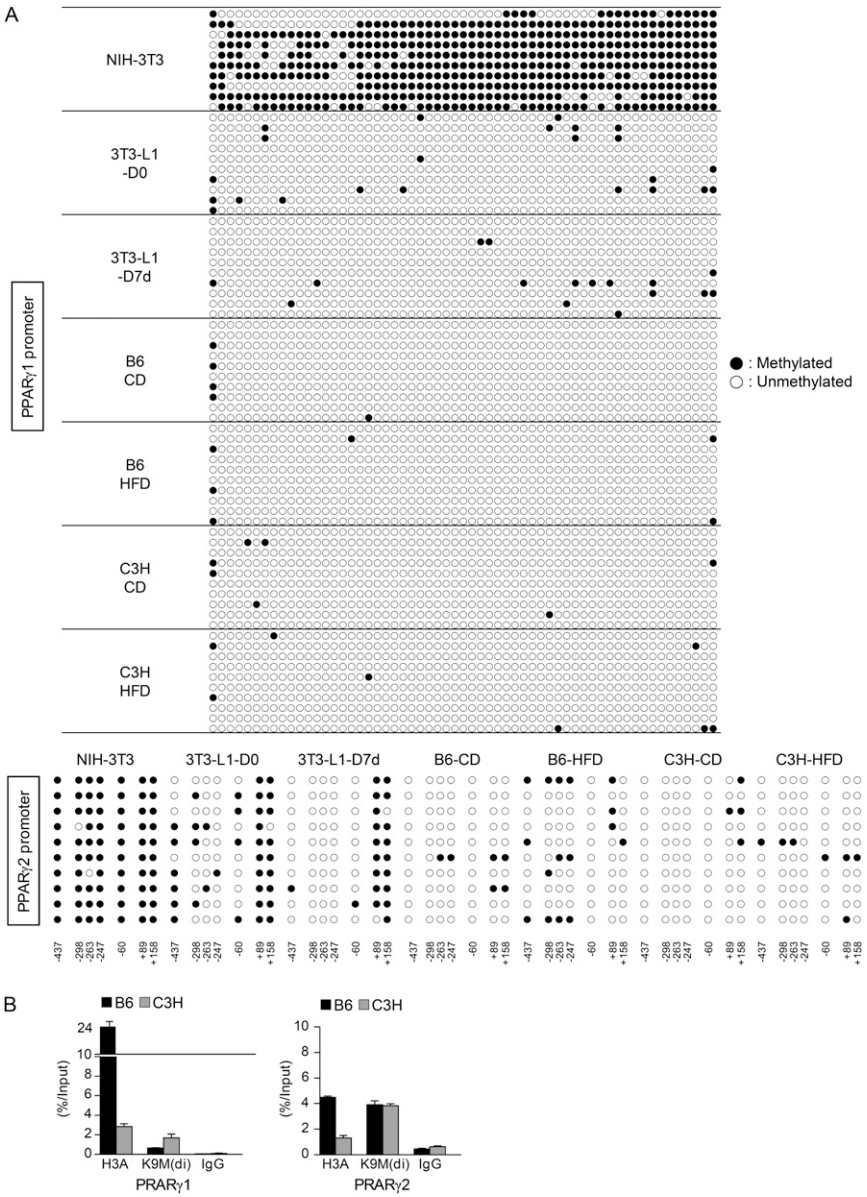

Fig. S3. Hepatic PPARγ expression is associated with acetylation status of histone H3. (*A*) The DNA methylation status for PPARγ1 and PPARγ2 promoter was determined from B6 and C3H/HeJ mice by bisulfite sequencing analysis. The DNA methylation profile of the individual CpG sites in the PPARγ promoter in NIH/3T3, 3T3-L1 preadipocytes (day 0) and 3T3-L1 adipocytes (day 7) was used for control status. Each PCR product was subcloned, and 59 (PPARγ1) or 10 (PPARγ2) clones were subjected to sequencing analysis. Each line represents one DNA strand. The methylation status of each site, either methylated (solid circle) or unmethylated (open circle), is aligned corresponding to their genomic order. (*B*) Primary hepatocytes of B6 and C3H/HeJ mice were subjected to a ChIP experiment. Formaldehyde cross-linked chromatin was immunoprecipitated with antibodies specific for acetylated H3, dimethylated H3-K9, and preimmune IgG. The purified DNA was amplified with gene-specific primers. Results of quantitative analysis of fold change in association of H3A and K9M (di) to the PPARγ1 and PPARγ2 promoter are normalized to input DNA from three different ChIP assays. Data are expressed as mean ± SD.

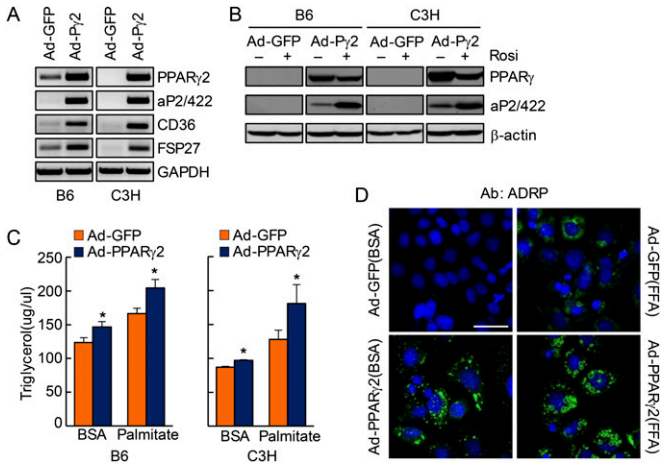

Fig. S4. Overexpression of PPARγ using primary hepatocytes isolated from B6 and C3H mice. Mouse primary hepatocytes were isolated from B6 and C3H mice and infected with 100 multiplicities of infection of either Ad-GFP or Ad-PPARγ2. (*A*) RT-PCR analysis of PPARγ2 and its target genes in the primary hepatocytes after 48 h of adenoviral infection. (*B*) Primary hepatocytes were infected with adenoviruses, and after 24 h, cells were treated with or without 1 μM rosiglitazone for another 24 h. Equal amounts of total protein were prepared and analyzed by Western blot analysis for expression of PPARγ2 and aP2/422. (*C*) After 36 h of infection, hepatocytes were incubated with or without palmitate (0.5 mM final concentration) in 1% FBS-DMEM for 24 h, and intracellular TG content was measured (*$P < 0.05$ compared with GFP). Data represent the mean ± SD. (*D*) After 36 h of infection, hepatocytes were incubated with or without palmitate (0.5 mM final concentration) in 1% FBS-DMEM for 24 h. Hepatocytes were then fixed in MeOH/acetone and incubated with rabbit anti-ADRP for 2 h at room temperature. After three washes in PBS, cells were incubated for 1 h with FITC-conjugated anti-rabbit. Images of cells were generated by confocal laser microscopy. (Scale bar, 50 μm.)

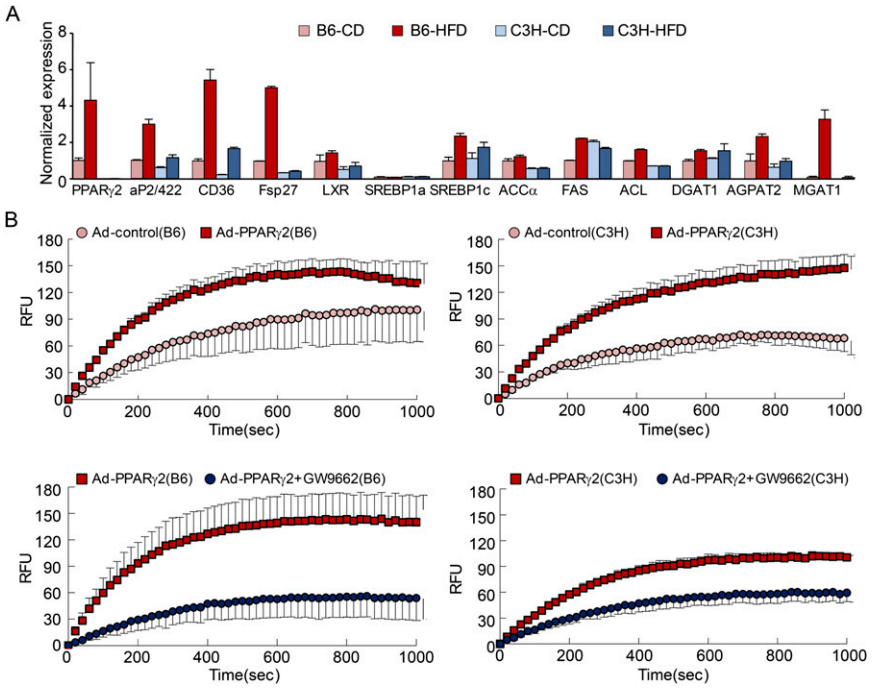

Fig. S5. Effect of PPARγ on fatty acid transport and TG accumulation by the regulation of target genes. (*A*) Real-time Q-PCR analysis showing hepatic gene expression involved in lipid metabolism in B6 and C3H mice fed a CD or a HFD ($n = 3–4$). (*B*) Fatty acid uptake was measured with a QBT Fatty Acid Uptake Assay kit, using primary hepatocytes isolated from B6 and C3H mice. Hepatocytes were plated on 96-well plates and real-time quantification of fatty acid uptake using a fluorescence assay was carried out after 48 h. (*Lower*) GW9662 (10 μM), a specific PPARγ inhibitor, was used. Data in *A* and *B* represent the mean ± SD.

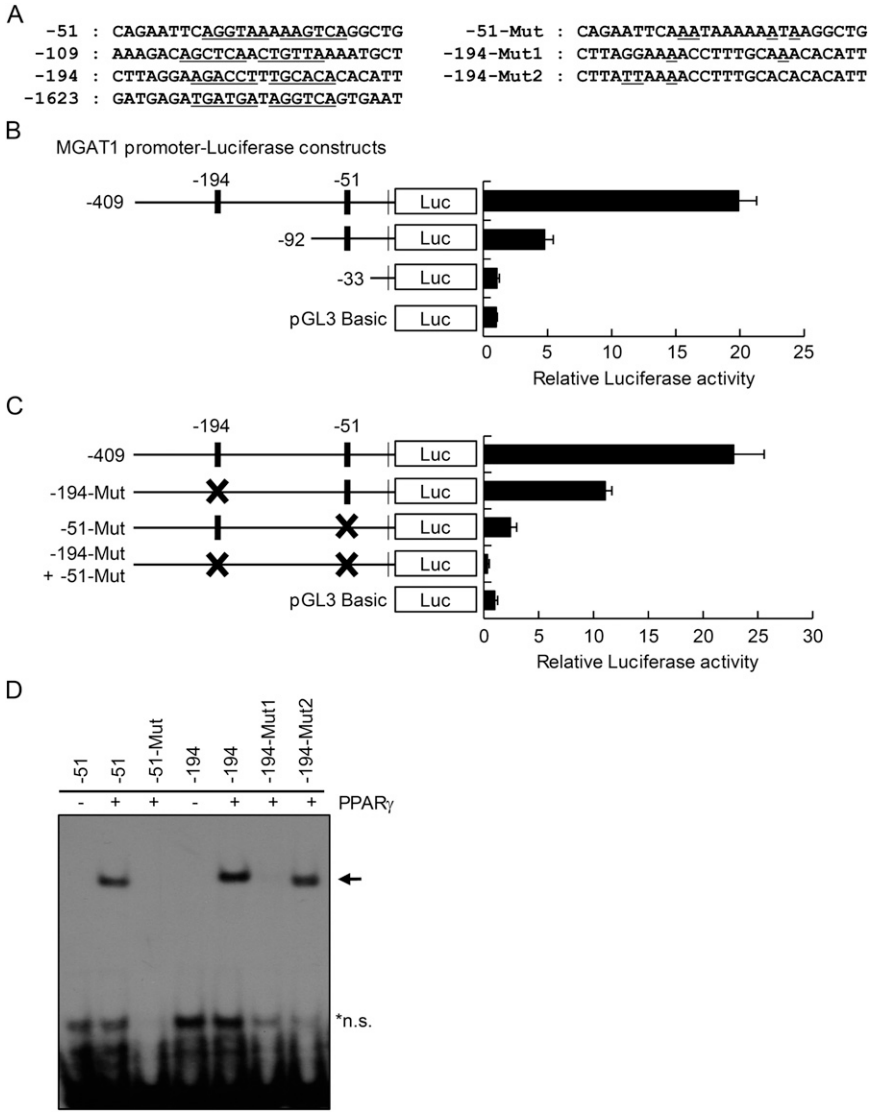

Fig. S6. Deletion and mutation analysis of MGAT1 promoter. (*A*) The sequence of wild-type and mutation of putative PPREs. (*Left*) The putative DR-1 sequence for PPRE is underlined; (*Right*) mutated sequences are underlined. The −194-Mut2 was also tested for unrelated mutation for PPRE by EMSA. (*B*) Deleted constructs of mouse MGAT1 promoter were generated and subjected to luciferase assay, with overexpression of retinoid X receptor (RXR)α and PPARγ2 in 293T cells. (*C*) Mutation of the −194 and/or the −51 region was introduced by site-directed mutagenesis and subjected to luciferase assay, with overexpression of RXRα and PPARγ2 in 293T cells. (*D*) EMSA experiment of mutated PPREs on the mouse MGAT1 promoter. The oligonucleotides shown in *A* were labeled and incubated with TNT-translated mouse PPARγ and RXRα proteins (*n.s., nonspecific bands). Data in *B* and *C* represent the mean ± SD.

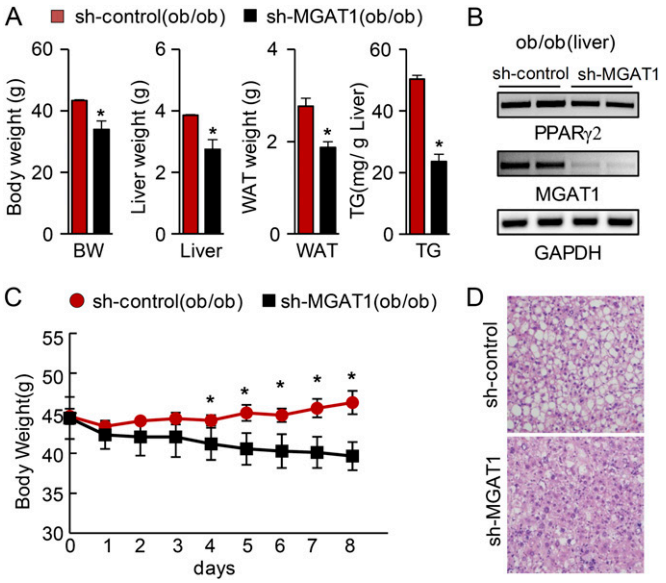

Fig. S7. Knockdown of MGAT1 improves hepatic steatosis in ob/ob mice. Leptin-deficient obese (ob/ob) mice were injected with either Ad-US or Ad-sh-MGAT1 virus via tail veins. After 1 wk, the mice were killed and then assays were performed. (*A*) Body weight, liver weights, epididymal fat weights, and hepatic TG contents were determined. Data represent the mean ± SD; $n = 3/3$. *$P < 0.05$. (*B*) RT-PCR analysis showing efficient knockdown of MGAT1 in livers from ob/ob mice. (*C*) Body weight changes were measured from the day of viral injection (day 0). *$P < 0.05$. (*D*) H&E staining of liver sections from ob/ob mice treated with Ad-US or Ad-sh-MGAT1.

Table S1. Biochemical parameters of C57BL/6J and C3H/HeJ mice fed with chow diet and high-fat diet

	Chow diet				High-fat diet			
Parameter	C57BL/6J	C3H/HeJ	*n*	*P* value	C57BL/6J	C3H/HeJ	*n*	*P* value
Triglyceride	60.33	95.67	8	0.0002	34.00	40.67	8	0.2198
Total cholesterol	71.67	92.67	8	0.0004	160.33	163.00	8	0.4337
HDL cholesterol	52.33	53.00	8	0.3838	72.00	89.00	8	0.0044
LDL cholesterol	4.00	5.33	8	0.0081	17.33	7.00	8	0.0057
AST	35.67	69.67	8	0.1072	74.33	83.67	8	0.3795
ALT	26.33	45.67	8	0.1502	81.67	80.00	8	0.4792

ALT, alanine aminotransferase; AST, aspartate aminotransferase; HDL, high-density lipoprotein; LDL, low-density lipoprotein. The mice were subjected to 4 h of fasting before being killed.

Table S2. Microarray of differential gene expression profile in livers of B6 and C3H mice

Gene	GenBank accession no.	Fold HFD/CD B6	Fold HFD/CD C3H	Fold Ad-PPARg2/GFP B6	Fold Ad-PPARg2/GFP C3H
Fatty acid synthesis					
Acetyl-CoA carboxylase α	NM_133360	1.06	0.70	2.52	1.93
Acetyl-CoA carboxylase β	NM_133904	1.23	1.14	1.73	1.86
ATP citrate lyase	NM_134037	0.29	1.04	1.10	1.13
ELOVL family member 6	NM_130450	1.97	0.64	2.00	1.83
Fatty acid synthase	NM_007988	1.31	0.58	1.67	2.38
Malic enzyme	NM_008615	1.15	0.77	3.55	1.92
Stearoyl-CoA desaturase 1	NM_009127	0.39	1.09	1.16	1.57
Stearoyl-CoA desaturase 2	NM_009128	1.05	1.37	0.65	1.68
TG metabolism					
1-Acylglycerol-3-phosphate *O*-acyltransferase 1	NM_018862	0.91	1.44	2.02	1.45
1-Acylglycerol-3-phosphate *O*-acyltransferase 2	NM_026212	1.87	1.23	1.61	1.30
1-Acylglycerol-3-phosphate *O*-acyltransferase 3	NM_053014	1.06	0.99	1.15	1.13
1-Acylglycerol-3-phosphate *O*-acyltransferase 6	NM_018743	2.38	0.87	1.80	1.19
Diacylglycerol *O*-acyltransferase 1	NM_010046	1.62	0.84	4.15	2.47
Diacylglycerol *O*-acyltransferase 2	NM_026384	0.97	1.28	1.19	1.08
Glycerol-3-phosphate acyltransferase	NM_008149	5.50	0.75	1.45	1.59
Monoacylglycerol *O*-acyltransferase 1	NM_026713	2.48	—	4.19	5.22
Monoacylglycerol *O*-acyltransferase 2	NM_177448	0.99	1.05	0.93	0.71
Phosphatidic acid phosphatase 2a (LPP1)	NM_008903	0.59	1.20	—	—
Phosphatidic acid phosphatase 2a (LPP1)	NM_008247	—	—	0.99	1.29
Phosphatidic acid phosphatase type 2B (LPP3)	NM_080555	0.96	1.36	1.54	1.12
Phosphatidic acid phosphatase type 2c (LPP2)	NM_015817	2.35	0.97	0.81	1.27
Transport protein					
Adipose differentiation-related protein	NM_007408	1.74	1.48	1.96	1.71
Apolipoprotein B	AK147540	0.48	0.88	0.88	0.93
CD36 antigen	NM_007643	2.71	0.98	2.57	7.97
CD68 antigen	NM_009853	1.76	0.86	0.53	0.67
Cell death-inducing DFFA-like effector c	NM_178373	13.0	7.36	19.15	12.16
Fatty acid-binding protein 1, liver	NM_017399	1.12	1.02	1.23	1.08
Fatty acid-binding protein 2, intestinal	NM_007980	1.72	2.13	1.76	1.72
Fatty acid-binding protein 3, muscle and heart	NM_010174	4.23	1.11	1.83	1.00
Fatty acid-binding protein 4, adipocyte	NM_024406	2.36	3.38	20.48	26.37
Fatty acid-binding protein 5, epidermal	NM_010634	—	0.27	2.24	2.13
Fatty acid-binding protein 7, brain	NM_021272	1.08	0.91	0.58	0.75
Fatty acid-binding protein 9, testis	NM_011598	0.73	0.64	1.17	0.73
Fatty acid transporter, member 1	NM_011977	1.26	0.78	0.97	0.99
Fatty acid transporter, member 2	NM_011978	1.13	0.89	1.14	1.04
Fatty acid transporter, member 4	NM_011989	1.66	0.98	2.84	1.75
Fatty acid transporter, member 5	NM_009512	0.84	1.13	0.52	0.79
Perilipin	NM_175640	0.52	1.01	1.11	0.90
Facilitated glucose transporter, member 4	NM_009204	3.65	1.21	1.31	0.54
Transporter 1, ATP-binding cassette	NM_013683	3.90	1.08	0.97	0.75

—, undetermined.