SUPPLEMENTARY TABLES

Table S1. Oligonucleotide primers used for real-time PCR quantitation and semi-quantitive PCR of adipocyte gene expression. m, mouse; h, human; f, forward; r, reverse; GPAT1, glycerol 3-phosphate acyltransferase-1; AGPAT2, acylglycerol-3-phosphate acyltransferase-2; DGAT1, diacylglycerol acyltransferase-1; C/EBPα, CCAAT/enhancer-binding protein-α; LPL, lipoprotein lipase; ACBP, Acyl-CoA binding protein; Insig1, Insulin-induced gene 1; FATP1, fatty acid transport protein-1; aP2, fatty acid binding protein 4; PEPCK1, phosphoenolpyruvate carboxykinase-1; 11βHSD, 11 β-hydroxysteroid dehydrogenase type 1; Glut4, glucose transporter4; TBP, TATA box binding protein; HPRT, hypoxanthine phosphoribosyltransferase.

Primer	Sequence
mLipin-1A	f – GGTCCCCCAGCCCCAGTCCTT; r - GCAGCCTGTGGCAATTCA
mLipin-1B	f – CAGCCTGGTAGATTGCCAGA; r - GCAGCCTGTGGCAATTCA
Total mLipin-1	f – CCCTCGATTTCAACGTACCC; r - GCAGCCTGTGGCAATTCA
mGPAT1	f – AGCAAGTCCTGCGCTATCAT; r - CTCGTGTGGGTGATTGTGAC
mAGPAT2	f – GCAACGACAATGGGGACCTG; r - ACAGCATCCAGCACTTGTACC
mDGAT1	f – GCTACGACGAGTTCTTGAG; r - CTCTGCCACAGATTGAGAC
mC/EBPα	f - GAACAGCAACGAGTACCGGGTA; r – GCCATGGCCTTGACCAAGGAG
mLPL	f – GGGCTCTGCCTGAGTTGTAG; r - AGAAATTTCGAAGGCCTGGT
mACBP	f – AGTCACTTCAAACAAGCTACTG; r - CACATAGGTCTTCATGGCACT
mPerilipin	f – TGAAGGGCACCATCTCTACC; r - CTTCTCGATGCTTCCCAGAG
mInsig1	f – TCAACCTGCTGCAGATCCAGC; r - AGGACCAGTGTCTCCACATCC
mFATP1	f – GTGCGTCATCTACGGGTTGAC; r - ACCGGCTGCCTCAGCAGGTA
maP2	f – GAACCTGGAAGCTTGTCTTCG; r - ACCAGCTTGTCACCATCTCG
mPEPCK1	f – TGGACTTCTCTGCCAAGGTT; r - AGTGAGAGCCAGCCAACAGT
m11β-HSD1	f – CCCATGCTGAAGCAGAGCAATG; r - TGGTGGAAAAGAACCCATCCAG
mHPRT	f – CACAGGACTAGAACACCTGC; r - GCTGGTGAAAAGGACCTCT

mTBP	f – ACCCTTCACCAATGACTCCTATG; r - ATGATGACTGCAGCAAATCGC
mGlut4 exon10	f – ACTCTTGCCACACAGGCTCT; r - AATGGAGACTGATGCGCTCT
m18S rRNA	f – ACCGCAGCTAGGAATAATGGA; r - GCCTCAGTTCCGAAAACCA
hLipin-1	f – TGCTGGAGAGCAGCAGAACTC; r - TAGGGTATGAGGCTGACTGAG
hHPRT	f – TATGGCGACCCGCAGCCCT; r - CATCTCGAGCAAGACGTTCAG

Table S2. Oligonucleotide primers used to prepare *Lpin1* promoter-luciferase reporter constructs. All sequences are shown 5' to 3'. The *Kpn*I and *Bgl*II sites used for cloning are underlined. The position of the 5′ nucleotide in the primer relative to the *Lpin1* transcription initiation site is noted.

Primer	**Sequence**	Position
F1	CGATC GGTACCTGCTTAACCAACACCTCCGCTCA	- 2045
F2	CGATCGGTACCGGTAAGGGCTTTGGGCTGGACTT	- 1564
F3	CGATCGGTACCCGCACCCGTCATCATTCTGACCT	- 940
F4	CGATCGGTACCTGTTGGTCTACGAGACCCCAGGT	- 823
F5	CGATCGGTACCTCCTTCCTGGCTCTGCTGACTGG	- 421
F6	CGATCGGTACCTCAAAACAGGGTCAGGCTGTGGA	- 285
F7	CGATCGGTACCCTTTGGCTGCCTACTCCCTGGAA	- 253
R1	CGATCAGATCTCGGGTCCCCATTGCTACTCACC	+ 70

Supplemental Figure Legends

Figure S1. **Lipin-1A is regulated by glucocorticoids at the transcriptional level**. Data in panels A-C are for lipin-1A; the analogous experiments for lipin-1B are presented in Fig. 3, panels A-C. *A*, induction of lipin-1A expression by DEX is inhibited by the RNA synthesis inhibitor actinomycin D (ACTD). Confluent 3T3-L1 preadipocytes were treated with the indicated concentration of DEX in the absence or presence of ACTD for 4 h. *B*, induction of lipin-1A was not inhibited by the protein synthesis inhibitor cycloheximide (CHX). Confluent 3T3-L1 preadipocytes were treated with the indicated concentration of DEX in the absence or presence of CHX for 4 h. *C*, induction of lipin-1A was inhibited by the glucocorticoid receptor antagonist RU486. Confluent 3T3-L1 preadipocytes were treated with the indicated concentration of DEX in the absence or presence of RU486 for 4 h. *D*, human primary adipocytes differentiated for 7 days and then treated with 1μM DEX in the absence or presence of 1μM RU486 for 4 h. Levels of total lipin-1 mRNA are shown. For all studies, values represent the mean ± SD for 3 samples. *, $p < 0.05$; **, $p < 0.01$ *vs*. controls or for comparisons indicated.

Figure S2. **The GRE in the *Lpin1* promoter activates expression in Hepa 1-6 hepatocytes**. ***A***, transcriptional activity of *Lpin1* promoter-luciferase constructs under treatment with vehicle (DMSO) or DEX (1 μM) for 24 h. Experiments were performed as in Figure 5 using Hepa 1-6 cells. Data represent the mean ± SE of 4 samples, expressed as the ratio of the *Lpin1* promoter segment to that of the pGL3-basic vector. ***B***, effect of 1 μM DEX on *Lpin1* promoter-luciferase constructs containing wild-type (wt) and mutant (mt) versions of the GRE sequence. n = 3; *, $p < 0.05$ *vs*. DMSO treated cells.

Figure S1

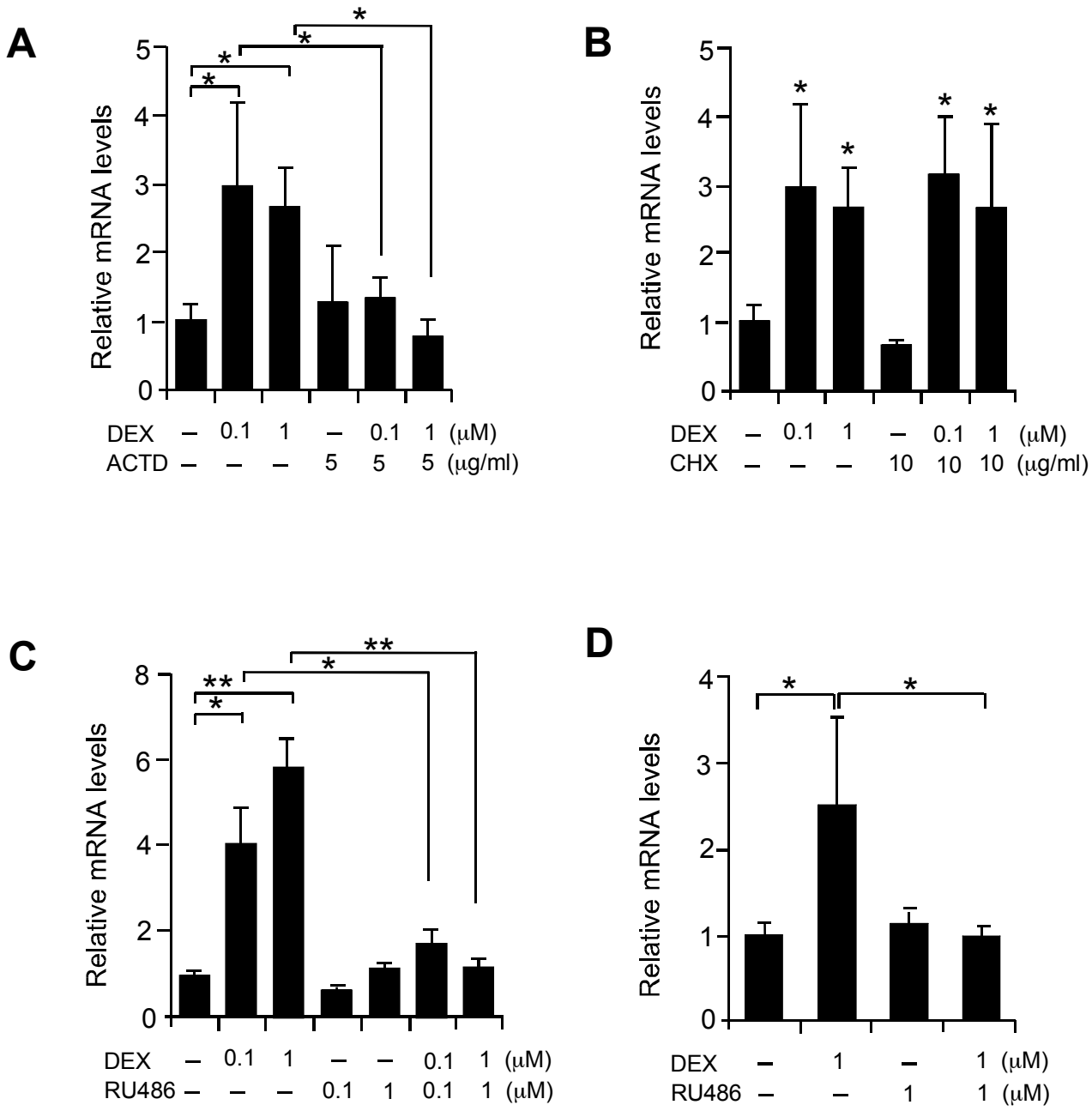
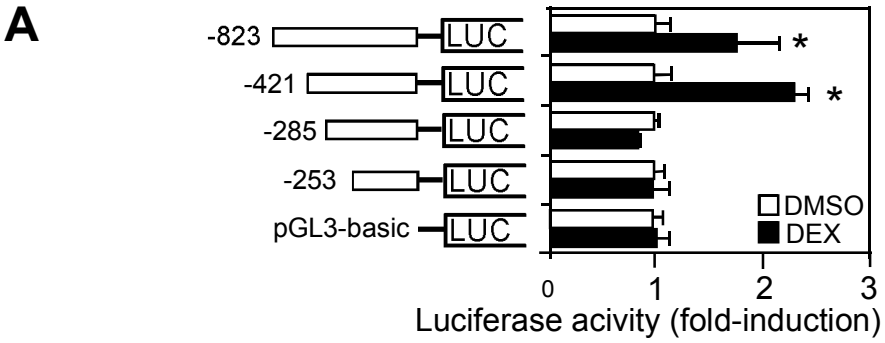
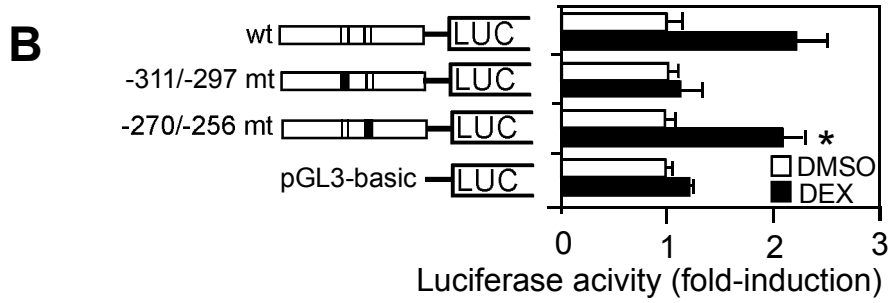

A
Relative mRNA levels
DEX – 0.1 1 – 0.1 1 (μM)
ACTD – – – 5 5 5 (μg/ml)
B
Relative mRNA levels
DEX – 0.1 1 – 0.1 1 (μM)
CHX – – – 10 10 10 (μg/ml)
C
Relative mRNA levels
DEX – 0.1 1 – – 0.1 1 (μM)
RU486 – – – 0.1 1 0.1 1 (μM)
D
Relative mRNA levels
DEX – 1 – 1 (μM)
RU486 – – 1 1 (μM)

Figure S2


A
-823
-421
-285
-253
pGL3-basic
LUC
*
*
DMSO
DEX
0
1
2
3
Luciferase acivity (fold-induction)


B
wt
-311/-297 mt
-270/-256 mt
pGL3-basic
LUC
*
DMSO
DEX
0
1
2
3
Luciferase acivity (fold-induction)