Supporting Information

Icardi et al. 10.1073/pnas.1206458109

SI Materials and Methods

Plasmids and Cytokine Treatment. The generation of the pXP2d2-rat pancreatitis-associated protein 1 (rPAP1)-luciferase reporter and its specificity to activated STAT3 were reported previously (1, 2). pCMV6-GFP-*SIN3A* plasmid was purchased from OriGene. Plasmids were cloned using the primer pairs reported in Table S2. The generation of Flp-In T-Rex Hek293 cells stably expressing the pXP2d2-rPAP1-luciferase reporter is described elsewhere (3). Recombinant human IFN-α2 (PBL IFN) and recombinant human leukemia inhibitory factor (LIF) (Millipore) were used at 10 ng/mL unless specified otherwise. Recombinant human IL-6 (Peprotech) was used at 100 ng/mL

Genome-Wide RNAi Screen, siRNA, shRNAmir, and Plasmid Transfection. For the genome-wide RNAi screen, Flp-In T-Rex Hek293 cells stably expressing the pXP2d2-rPAP1-luciferase reporter were transfected with prespotted siRNAs (50 nM) in 384-well plates using Dharmafect 1 (Dharmacon RNAi Technology) transfection reagent following the manufacturer's instructions. After 72 h, the cells were stimulated with IFN-α2 for 24 h before luciferase activity was measured. In every plate, the first four columns were used for internal screening controls: control siRNA (*Renilla* luciferase)-transfected nonstimulated cells (background control), control siRNA-transfected IFN-α2–stimulated cells (basal rPAP1-luciferase expression control), control siRNA-transfected LIF-stimulated cells (rPAP1-luciferase induction control), and *JAK1* siRNA-transfected LIF-stimulated cells (siRNA transfection efficiency control). Luciferase activity was measured after 24 h of cytokine treatment by chemiluminescence with a Mithras LB940 plate reader (Berthold Technologies). Screening data were analyzed with the CellHTS2 software (4) and quantified by *z*-scoring. A *z*-score of 0 indicates no effect of the gene silencing on the rPAP1-luciferase reporter activity, whereas a positive *z*-score indicates genes whose silencing permits IFN-α2–induced reporter activation as such highlighting potential STAT3 transcriptional repressors. During the analysis, one outlayer candidate was identified as an artifact and discharged. The screen was performed in duplicate.

For individual silencing experiments, *Renilla* luciferase (*RL*), *JAK1*, *STAT3*, *SAP130*, *SUDS3*, *SFPQ* and *TGIF2* siGENOME SMARTpools were purchased from Thermo Scientific; *SIN3A* siRNA Silencer Select Predesigned siRNA was purchased from Ambion. siRNA transfection (50 nM) was performed using Dharmafect 1 transfection reagent following the manufacturer's instructions. Western blot analysis to verify silencing efficiency was performed 72 h after transfection.

For stable silencing, the pGIPZ-GFP-*SIN3A*-shRNAmir and the pGIPZ-GFP-scrambled-shRNAmir lentiviral vectors were purchased from Open Biosystems. Lentiviral packaging (pCMVR8.74) and pseudotyping (pMD2.G) constructs were provided by D. Trono (Tronolab, CH-1015, Lausanne, Switzerland) through Addgene (plasmid 22036 and plasmid 12259, respectively). Lentiviral particles were produced using a classic calcium phosphate transfection (5). Briefly, 3.2 μg of the pGIPZ-shRNAmir vectors were cotransfected with 2.4 μg of pCMVR8.74 and 1 μg of pMD2.G on Hek293T cells seeded the day before in six-well plates. The supernatant containing the viral particles was harvested 48 and 72 h after transfection. Target cells were transduced with the supernatant after centrifugation of cellular debris and filtration through a 0.45-μm filter. Transduced cells were then selected with 2 μg/mL puromycin.

All plasmid transfections were performed using a standard calcium phosphate precipitation procedure (5).

Western Blot Analysis, Immunoprecipitation, and Luciferase Assays. For Western blot analysis, total lysates from six-well plates were performed as previously described (6). For immunoprecipitation of endogenous proteins, the nuclear extracts from two pulled confluent 10-cm Petri dishes were used for each condition. For immunoprecipitation of overexpressed proteins, Hek293T cells were plated in 10-cm Petri dishes and transfected with 5 μg of Etag-STAT3 mutants and 7 μg of GFP-Sin3a. Immunoprecipitation was performed 48 h after transfection using 1 μg of antibody. Immune complexes were precipitated using protein A-conjugated Sepharose beads (GE Healthcare). The following primary antibodies were used: anti-STAT3 (124H6), anti–P-Y_{705}STAT3 (9131), and antiacetylated lysines (9441) antibodies were purchased from Cell Signaling Technology; Sin3a antibody (sc-994) was purchased from Santa Cruz Biotechnology; anti-actin mAb antibody (A2228) was purchased from Sigma Aldrich; anti–E-tag antibody (A5441) was purchased from GE Healthcare. As negative control, the equivalent amount of normal rabbit immunoglobulins (sc-2027; Santa Cruz) was used. For endogenous immunoprecipitation, HPR-conjugated secondary antibodies (Jackson Laboratories) were used and revealed by autoradiography. For the other Western Blots, either Dylight 800- or Dylight 680-conjugated secondary antibodies (Pierce) were used. Targeted proteins on the blots were visualized using the Odyssey infrared imaging system (LI-COR Biosciences).

For the luciferase assays, cells were transfected with the molar ratio (1:5) of reporter vector to expression vector. The next day, cells were washed with PBS, trypsinized, transferred to a 96-well plate, and left nonstimulated or stimulated with IFN-α2 or LIF for 24 h. Luciferase activity from triplicate samples was measured by chemiluminescence with a TopCount luminometer (Canberra-Packard) and expressed as fold-induction (stimulated/nonstimulated).

GST-Pull-Down Assay. Flag-Sin3a was produced in vitro from the pcDNA3.1-T7-Flag-*SIN3A* construct with the TnTT7 Quick Coupled Transcription/Translation System (Promega) following the manufacturer's instructions. GST-tagged constructs were transformed in the *Escherichia coli* BL21DE3 cells and grown overnight under Ampicillin selection. Next, cells in exponential growth phase were incubated with 2 mM isfopropyl-β-D-thiogalactopyranoside (IPTG) and 3% (vol/vol) ethanol for 20 h at 25 °C. Cells were then resuspended in NETN buffer [20 mM Tris-HCl pH8, 100 mM NaCl, 6 mM MgCl2, 1 mM EDTA, 10% (vol/vol) Nonidet P-40, 1% DTT, proteinases inhibitors], sonicated, and supernatant was loaded on Glutathione Sepharose beads (GE Healthcare) for 90 min. The in vitro produced Flag-Sin3a protein was added and incubated overnight. Beads containing the bound complexes were extensively washed in NETN buffer and complexes were revealed with anti-FLAG (F3165; Sigma Aldrich) and anti-GST antibody (ab9085; Abcam).

NanoPro-Based Analysis of STAT3 Posttranslational Modification Pattern. Hek293T cells were transfected with a control siRNA (*Renilla* luciferase) or *SIN3A* siRNA (50 nM) and LIF-stimulated 72 h after transfection. Lysates were prepared using Bicine/CHAPS Lysis Buffer (ProteinSimple) and analyzed for STAT3 posttranslational modification pattern using NanoPro technology. Samples were separated using a 10% 5-8, 90% 5-6 separa-

tion gradient (ProteinSimple) and then probed using an anti-STAT3 antibody (9132; Cell Signaling Technology) followed by a goat anti-rabbit biotin-labeled secondary antibody. STAT3 was detected using a streptavidin-HRP molecule activated when luminol/peroxide was flushed through the capillary. Data analysis was performed using Compass software (ProteinSimple).

ChIP and Quantitative RT-PCR Assays. Hek293T cells stably transfected with GFP-scrambled or GFP-*SIN3A* shRNAmir were cultured 4 h without FCS and then LIF-stimulated for the indicated time points. ChIP experiments and precipitated DNA purification were performed as previously described (2). The fold-changes of promoter occupancy were calculated by normalizing the relative values to the input and comparing with untreated cells, using the ΔΔCT method. Quantitative RT-PCR was performed as previously described (2). The fold-change of mRNA levels was calculated by normalizing the relative amount to the internal control GAPDH and compared with the non-stimulated condition using the ΔΔCT method. The primers and the probes used were selected with the online tool Universal Probe Library Assay Design Center (Roche Applied Science) and are summarized in Table S2. Chromatin-associated proteins were precipitated using the following antibodies: rabbit IgG (sc-2027; Santa Cruz), anti-Sin3a and anti-STAT3 (AK-11 and sc-482, respectively, Santa Cruz Biotechnology), anti-STAT3 (9132; Cell Signaling), anti–phospho-pol II (ab5095 and ab5131, Abcam), and H3K27 (ab4729; Abcam).

Affymetrix Microarray. MCF7 cells were transfected with control (*Renilla* luciferase) or *SIN3A* siRNA. After 72 h, cells were cultured 4 h without FCS, then left nonstimulated or stimulated 1 h with LIF. RNA was isolated using RNeasy columns, as described by the manufacturer (Qiagen). RNA concentration and purity were determined spectrophotometrically using the Nanodrop ND-1000 (Thermo Scientific) and RNA integrity was assessed using a Bioanalyzer 2100 (Agilent). Per sample, an amount of 100 ng of total RNA spiked with bacterial RNA transcript positive controls (Affymetrix) was amplified and labeled using the GeneChip 3′ IVT express kit (Affymetrix). All steps were carried out according to the manufacturers protocol (Affymetrix). A mixture of purified and fragmented biotinylated antisense RNA and hybridization controls (Affymetrix) was hybridized on Affymetrix HG U133 Plus 2.0 arrays followed by staining and washing in a GeneChip fluidics station 450 (Affymetrix) according to the manufacturer's procedures. To assess the raw probe signal intensities, chips were scanned using a GeneChip scanner 3000 (Affymetrix). Analysis of the data were performed in the R programming environment, in conjunction with the packages developed within the Bioconductor project (7). The analysis was based on the RMA expression levels of the probe sets that had at least once a present MAS 5.0 detection call. Differential expression was assessed via the moderated *t*-statistic (8). To control the false-discovery rate, multiple testing correction was performed and probes with a corrected *P* value below 0.05 were selected.

Confocal Microscopy. Cells were seeded on cover-slips and grown in serum-free medium for 4 h. Cell fixation, methanol permeabilization, and staining was performed according to the Cell Signaling guidelines. STAT3 was visualized with the STAT3 monoclonal antibody (Cell Signaling Technology), followed by probing with Alexa Fluor 594 (Invitrogen). Sin3a was stained with anti-Sin3a antibody (Santa Cruz Biotechnology) followed by probing with Alexa Fluor 488 (Invitrogen). A motorized inverted IX81 FluoView FV1000 laser scanning confocal microscope (Olympus) was used to record high-resolution images.

Influenza A Virus and Hepatitis C Virus Infection. Influenza virus PR8 (A/Puerto Rico8/34, H1N1 subtype) was grown and titrated on Madin-Darby canine kidney (MDCK) cells. Three days after siRNA transfection, Hek293T and MCF7 cells were left non-stimulated or stimulated with IFN-α2 in OptiMEM (Invitrogen). Twenty-four hours later, cells were infected with PR8 virus [multiplicity of infection (MOI) 0.05 for Hek293T cells and MOI 0.5 for MCF7 cells) for 14 h. IFN-α2 was not removed from the medium throughout infection. Cells were then fixed with paraformaldehyde and infection efficiency was evaluated by staining nucleoprotein (NP) (anti-RNP antibody, National Institutes of Health Biodefense and Emerging Infections Research Resources Repository) and M2 (anti-M2 mAb directed against the extracellular part of M2) followed by Alexa Fluor 633 (Invitrogen) and Alexa Fluor 488 (Invitrogen), respectively. NP staining was revealed by confocal microscopy, and M2 staining was analyzed by flow cytometry.

The cell-culture–produced hepatitis C virus (HCV) Jc1 was generated as previously described (9, 10). Three days after control (*Renilla* luciferase) or *SIN3A* siRNA transfection, HuH7.5 cells were seeded 1.2×10^4 cells per well in a 96-well imaging plate (BD Biosciences) and stimulated with IFN-α2. One day later, HCV Jc1 was added at MOI 0.0033. IFN-α2 was not removed from the medium throughout infection. Infection was allowed to proceed for 2 d before PFA fixation and stain of HCV-infected cells with anti-NS5A mAb (9E10) in combination with Alexa Fluor 647 antibody (Invitrogen). Images were acquired using a BD Pathway 435 High Content Bioimager (BD Biosciences) with a 10× objective.

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8. Smyth GK (2004) Linear models and empirical bayes methods for assessing differential expression in microarray experiments. *Stat Appl Genet Mol Biol* 3:Article3.
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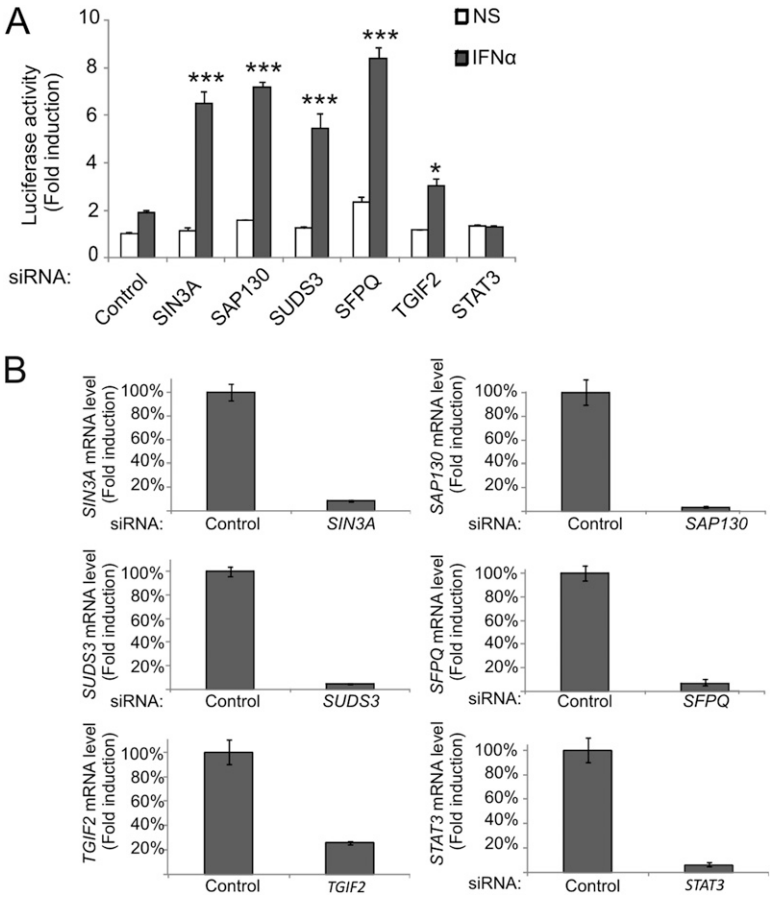

Fig. S1. The SIN3 transcription regulator homolog A (Sin3a) complex represses STAT3-dependent transcription. (*A*) Hek293T cells were transiently transfected with the indicated siRNA and with the rPAP1-luciferase reporter after 24 h. *Renilla* luciferase siRNA was used as control. After 48 h, cells were left non-stimulated (NS) or stimulated with IFNα2. Luciferase readout is expressed as a ratio over the control-silenced NS condition. ***$P < 0.001$, *$P < 0.01$; one-way ANOVA with Bonferroni test. SIN3A/HDACs knockdown increases STAT3 activity. (*B*) siRNA-transfected Hek293T cells from *A* were assessed for silencing efficiency in quantitative RT-PCR assay 48 h after transfection. Graphs represent the percentage of mRNA levels relative to the control siRNA condition. Error bars indicate SD from triplicates.

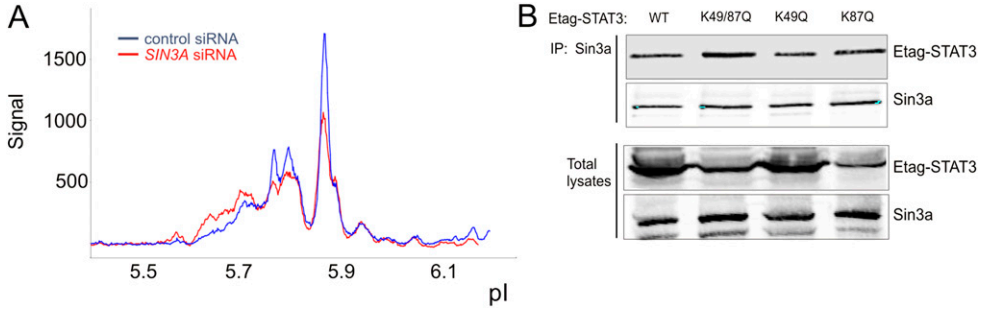

Fig. S2. (*A*) *SIN3A* silencing results in pI-shift of STAT3 isoforms toward more acidic values. Hek293T cells were transfected with a control (*Renilla* luciferase) or a *SIN3A*-specific siRNA and total lysates were analyzed using NanoPro technology, as described in the *SI Materials and Methods*. The spectrum shows a shift toward more acidic protein isoelectric point (pI) values in the *SIN3A*-silenced condition indicating an increase in a posttranslation event, such as acetylation. (*B*) Hek293T cells were transfected with plasmids coding for GFP-Sin3a and Etag-STAT3 WT or acetyl-mimicking mutants (K49/87Q, K49Q, or K87Q). Sin3a was immunoprecipitated and coprecipitated Etag-STAT3 mutants were revealed with an anti-Etag antibody. Total lysates were blotted as loading control.

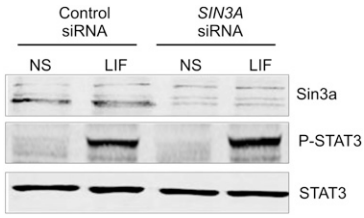

Fig. S3. *SIN3A* knockdown and STAT3 phosphorylation prior microarray analysis. MCF7 cells were transfected with *Renilla* luciferase (control) siRNA or *SIN3A* siRNAs. Seventy-two hours after transfection, cells were cultured 4 h without FCS and then left nonstimulated or stimulated with LIF for 1 h. Total lysates were blotted with anti-Sin3a, anti-phospho STAT3, and anti-STAT3 antibodies.

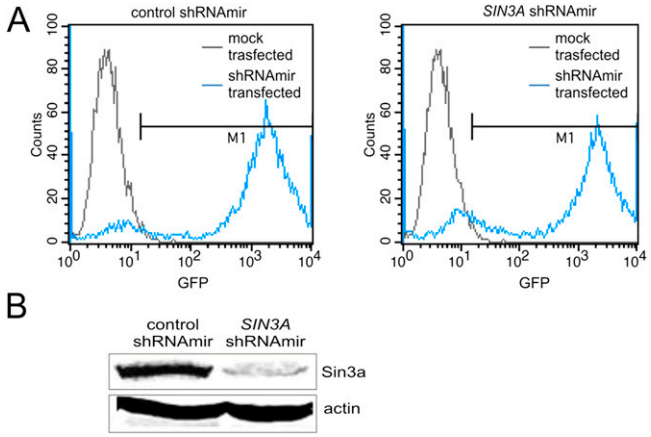

Fig. S4. Generation of a stable *SIN3A*-silenced Hek293T cell line. Transduction of Hek293T cells with lentiviral particles containing pGIPZ-GFP-scrambled-shRNAmir (control shRNAmir) or pGIPZ-GFP-*SIN3A*shRNAmir vectors and selection of stably transduced cells was performed as described in *SI Materials and Methods*. (*A*) GFP expression levels were evaluated in flow cytometry and indicate comparable efficiency of transduction. (*B*) Silencing efficiency was determined by blotting the total cell lysates with an anti-Sin3a antibody and antiactin as loading control.

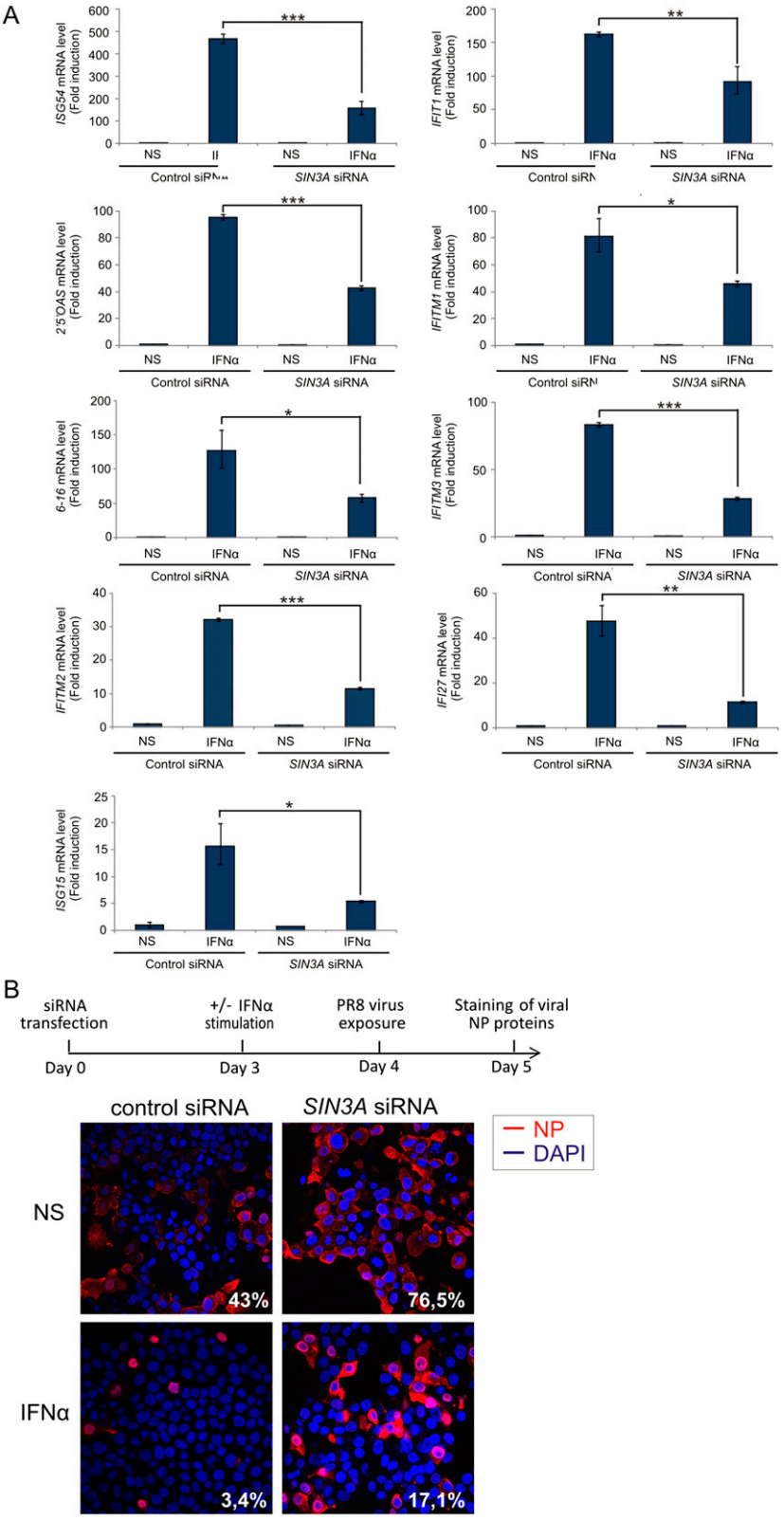

Fig. S5. Silencing of *SIN3A* impairs IFN-trigged IFN-stimulated gene (ISG) transcription and antiviral activity. (*A*) Hek293T cells were transfected with a control siRNA (*Renilla luciferase*) or an alternative *SIN3A* siRNA. Seventy-two hours after transfection, cells were left nonstimulated or stimulated with IFN-α2 for 24 h and ISG transcription was evaluated. The quantitative RT-PCR analysis represents the relative mRNA levels of a subset of ISGs, compared with the nonstimulated control-silenced condition. Error bars indicate SD from triplicates. ***$P < 0.001$, **$P < 0.005$, *$P < 0.01$; one-way ANOVA with Bonferroni test. (*B*) MCF7 cells were transfected with a control siRNA (*Renilla* luciferase) or *SIN3A* siRNA. After 72 h, cells were left nonstimulated or stimulated with IFN-α2 for 24 h before 14-h exposure to influenza A/PR8/34 virus. The efficiency of the infection was evaluated by immunostaining of the viral NP. *SIN3A* silencing increased influenza infection in both untreated cells and, at higher extent, in IFN-α2 pretreated cells. Confocal immunofluorescence of representative cell fields is shown. (Magnification: 40×.)

Table S1. *SIN3A* silencing did not affect the induction of APR genes in IL-6 or LIF-stimulated Huh7 cells

		RL siRNA						SIN3A siRNA					
		1 h IL-6			24 h IL-6			1 h IL-6			24 h IL-6		
Gene symbol	Gene ID	Fold-induction	SD+	SD−	Fold-induction	SD+	SD−	Fold-induction	SD+	SD−	Fold-induction	SD+	SD−
AGT	183	**1.35**	0.0	0.0	**2.40**	0.0	0.0	**1.43**	0.0	0.0	**2.25**	0.6	0.4
FGG	2266	**1.66**	0.0	0.0	**3.46**	0.2	0.1	**2.32**	0.1	0.1	**4.45**	0.4	0.4
CD14	929	**1.11**	0.1	0.1	**1.51**	0.3	0.2	**1.31**	0.2	0.1	**1.27**	0.1	0.1
		1 h LIF			24 h LIF			1 h LIF			24 h LIF		
AGT	183	**1.09**	0.3	0.3	**1.25**	0.1	0.1	**0.56**	0.0	0.0	**0.84**	0.0	0.0
FGG	2266	**1.46**	0.1	0.1	**1.80**	0.1	0.1	**1.39**	0.1	0.1	**1.67**	0.1	0.1
CD14	929	**1.53**	0.1	0.1	**1.06**	0.1	0.1	**3.36****	0.2	0.2	**1.27**	0.1	0.1

Huh7 cells were transfected with control (*Renilla* luciferase) or *SIN3A* siRNA. After 72 h, cells were left nonstimulated or stimulated with LIF. Fold-inductions represent mRNA levels relative to NS sample and are highlighted in bold. Results are representative of three independent experiments. $^{**}P < 0.05$.

Table S2. Primer pairs used in plasmid cloning, quantitative RT-PCR, and ChIP-PCR assays (in combination with Roche UPL probe system)

Plasmids	Application	Primer sequences	Strategy
E-tag STAT3 FL	Cloning	5′-GCGGCGGCCGCAGCCCAATGGAATCAGCTACAG-3′ 5′- CGCCTCGAGTCACATGGGGGAGGTAGCGCA-3′	PCR and insertion in pMet7-Etag vector
E-tag STAT3 131-770	Cloning	5′-CGATGCGGCCGCtCACCCCACAGCAGCCGTGG-3′ 5′- GCTGCTCGAGTCACATGGGGGAGGTAGCGC-3′	
E-tag STAT3 321-770	Cloning	5′-CGTAGCGGCCGCATTTGTGGTGGAGCGGCAGCCC-3′ 5′- GCTGCTCGAGTCACATGGGGGAGGTAGCGC-3′	
E-tag STAT3 465-770	Cloning	5′-CGTAGCGGCCGCATCCAACATCTGTCAGATGCC-3′ 5′- GCTGCTCGAGTCACATGGGGGAGGTAGCGC-3′	
E-tag STAT3 585-770	Cloning	5′-CGTAGCGGCCGCAATCATGGGCTTTATCAGTAAGG-3′ 5′- GCTGCTCGAGTCACATGGGGGAGGTAGCGC-3′	
E-tag STAT3 1-130	Cloning	5′-CCAGCAAGGGGGCCAGGCCAACTAACCCACAGCTG CCGTGGTGACGGAGAAGCAGC-3′ 5′- GCTGCTTCTCCGTCACCACGGCAGCTGTGGGTTAGTT GGCCTGGCCCCCTTGCTGG-3′	Mutagenesis of pMet7-Etag-STAT3 FL
E-tag STAT3 1-320	Cloning	5′-GAAACTTAATGAAAAGTGCCTGAGTGGTGGAG CGGCAGCC-3′ 5′- GGCTGCCGCTCCACCACTCAGGCACTTTTCA TTAAGTTTC-3′	
E-tag STAT3 1-465	Cloning	5′- CCAGTTGTGGTGATCTCCAACTAGCTGTCAGA TGCCGAATGCC-3′ 5′- GGCATTCGGCATCTGACAGCTAGTTGGAGATCA CCACAACTGG-3′	
E-tag STAT3 1-585	Cloning	5′- GGAACGAAGGGTACATCATGTGATTTATAA GTAAGGAGC-3′ 5′- GCTCCTTACTTATAAATCACATGATGTACCCTTCGTTCC-3′	
E-tag STAT3 K49Q	Cloning	5′- GCATATGCGGCCAGCCAAGAATCACATGCC-3′ 5′- GGCATGTGATTCTTGGCTGGCCGCATATGC-3′	
E-tag STAT3 K49R	Cloning	5′- GCATATGCGGCCAGCCGAGAATCACATGCC-3′ 5′- GGCATGTGATTCTCGGCTGGCCGCATATGC-3′	
E-tag STAT3 K685Q	Cloning	5′- GGAGGCATTCGGACAGTATTGTCGGCC-3′ 5′- GGCCGACAATACTGTCCGAATGCCTCC-3′	
E-tag STAT3 K685R	Cloning	5′- GGAGGCATTCGGAAGGTATTGTCGGCC-3′ 5′- GGCCGACAATACCTTCCGAATGCCTCC-3′	
E-tag STAT3 KK49/87QQ	Cloning	5′- GCACAATCTACGAAGAATCCAGCAGTTTCTTCAGAGC-3′ 5′- GCTCTGAAGAAACTGCTGGATTCTTCGTAGATTGTGC-3′	Mutagenesis of pMet7-Etag-STAT3K49Q
E-tag STAT3 KK49/87RR	Cloning	5′- GCACAATCTACGAAGAATCAGGCAGTTTCTTCAGAGC-3′ 5′- GCTCTGAAGAAACTGCCTGATTCTTCGTAGATTGTGC-3′	Mutagenesis of pMet7-Etag-STAT3 K49R
GST-STAT3 1-130	Cloning	Digest pMet7-Etag-Stat3 1–130	Ligate inserts in pGEX-4T-2 vector
GST-STAT3 131-770	Cloning	Digest pMet7-Etag-Stat3 131–770	
GST-MAD1	Cloning	Digest pMet7-Etag-MAD1	
SOCS3	RT-PCR	5′-CTTCGACTGCGTGCTCAA-3′ 5′-GTAGGTGGCGAGGGGAAG-3′	UPL probe #1
FGG	RT-PCR	5′-CCACTATGAAGATAATCCCATTCA-3′ 5′-AACGGTCTTTTAAACGTCTCCA-3′	UPL probe #56
AGT	RT-PCR	5′-TCAACACCTACGTCCACTTCC-3′ 5′-GCTGTTGTCCACCCAGAACT-3′	UPL probe #7

Table S2. Cont.

Plasmids	Application	Primer sequences	Strategy
GADD45G	RT-PCR	5′-CAGCCAAAGTCTTGAACGTG-3′ 5′-CCTGGATCAGCGTAAAATGG-3′	UPL probe #71
CEBPD	RT-PCR	5′-GGACATAGGAGCGCAAAGAA-3′ 5′-GCTTCTCTCGCAGTTTAGTGG-3′	UPL probe #64
KLF10	RT-PCR	5′-AGCCAACCATGCTCAACTTC-3′ 5′-CTCTTTTGGCCTTTCAGAAATC-3′	UPL probe #67
CD14	RT-PCR	5′-GTTCGGAAGACTTATCGACCAT-3′ 5′-ACAAGGTTCTGGCGTGGT-3′	UPL probe #74
SOX9	RT-PCR	5′-GTACCCGCACTTGCACAAC-3′ 5′-TCGCTCTCGTTCAGAAGTCTC-3′	UPL probe #61
RND1	RT-PCR	5′-GAAAATTACACAGCCTGTTTGGA-3′ 5′-CGGACATTATCGTAGTAGGGAGA-3′	UPL probe #6
STAT3	RT-PCR	5′-CCCTTGGATTGAGAGTCAAGA-3′ 5′-AAGCGGCTATACTGCTGGTC-3′	UPL probe #14
FOS	RT-PCR	5′-CTACCACTCACCCGCAGACT-3′ 5′-AGGTCCGTGCAGAAGTCCT-3′	UPL probe #67
EGR1	RT-PCR	5′-AGCCCTACGAGCACCTGAC-3′ 5′-GGTTTGGCTGGGGTAACTG-3′	UPL probe #22
ISG54	RT-PCR	5′-TGGTGGCAGAAGAGGAAGAT-3′ 5′-GTAGGCTGCTCTCCAAGGAA-3′	UPL probe #27
IFIT1	RT-PCR	5′-AGAACGGCTGCCTAATTTACAG-3′ 5′-GCTCCAGACTATCCTTGACCTG-3′	UPL probe #9
2′5′OAS	RT-PCR	5′-GACGGATGTTAGCCTGCTG-3′ 5′-TGGGGATTTGGTTTGGTG-3′	UPL probe #43
IFITM1	RT-PCR	5′-CACGCAGAAAACCACACTTC-3′ 5′-TGTTCCTCCTTGTGCATCTTC-3′	UPL probe #60
6–16	RT-PCR	5′-CTGTGCCCATCTATCAGCAG-3′ 5′-GGGCTCCGTCACTAGACCTT-3′	UPL probe #41
IFITM3	RT-PCR	5′-TCAAGGAGGAGCACGAGGT-3′ 5′-GATGTGGATCACGGTGGAC-3′	UPL probe #32
IFITM2	RT-PCR	5′-CTCCGTGCCTGACCATGT-3′ 5′-GCCAACCATCTTCCTGTCC-3′	UPL probe #62
IFI27	RT-PCR	CCAAGCTTAAGACGGTGAGG CCGTGGCCTAGAGAGTAAGAGA	UPL probe #41
ISG15	RT-PCR	GCGAACTCATCTTTGCCAGT AGCATCTTCACCGTCAGGTC	UPL probe #76
ISG20	RT-PCR	5′-CACCCCTCAGCACATGGT-3′ 5′-TGGAAGTCGTGCTTCAGGT-3′	UPL probe #17
SOCS3	ChIP-PCR	5′-AAAAGGGGAAGGGGAACC-3′ 5′-GGAGAGCGGGCAGTTCTA-3′	UPL probe #85

List of oligonucleotides. For each construct used in this study, primer pairs and cloning strategies are reported. For quantitative RT-PCR and ChIP-PCR assays, both primer pairs and corresponding Universal Probe Library (UPL) probe [UPL, Roche] numbers are indicated.

Dataset S1. Top 250 repressor candidates identified in the RNAi screen

Dataset S1 (xls)

STAT3 repressor candidates identified in the RNAi screen and relative z-score. Sin3a complex components are highlighted in bold. Detailed description of the screening procedure and the analysis can be found in *SI Materials and Methods*.