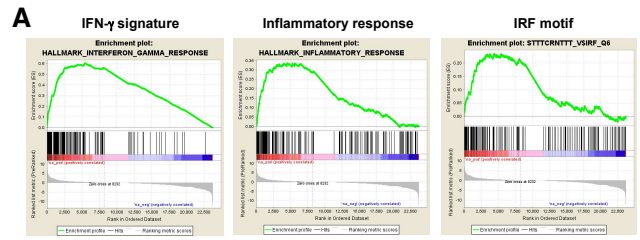
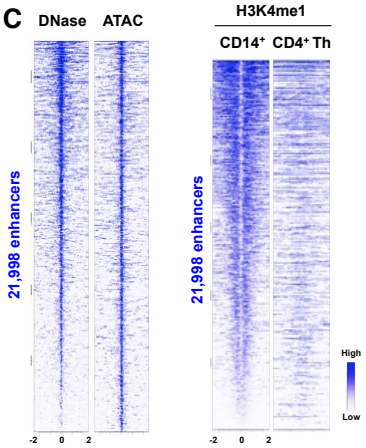
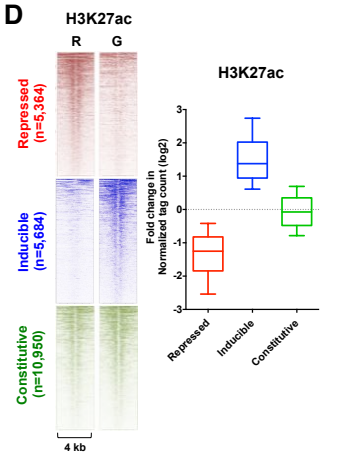


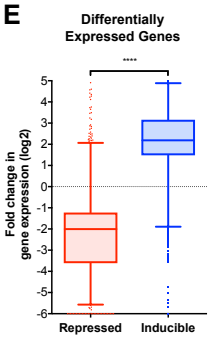
B

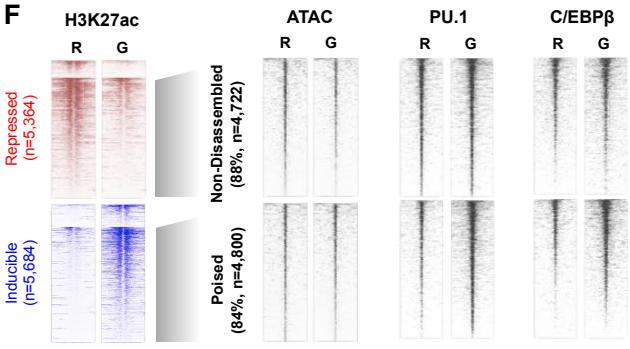
IFN-γ-repressed genes

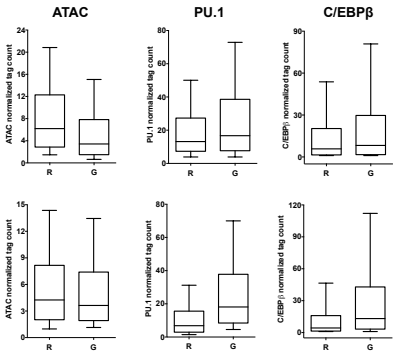
Pathway	Gene Set Name	p-value
REACTOME	HEMOSTASIS	2.14E-17
HALLMARK	COAGULATION	6.57E-15
HALLMARK	EPITHELIAL_MESENCHYMAL_TRANSITION	9.16E-13
REACTOME	PLATELET_ACTIVATION_SIGNALING_AND_AGGREGATION	9.32E-10
HALLMARK	ADIPOGENESIS	1.55E-07
KEGG	LYSOSOME	7.68E-06
KEGG	ENDOCYTOSIS	1.09E-03
KEGG	PPAR_SIGNALING_PATHWAY	2.71E-03

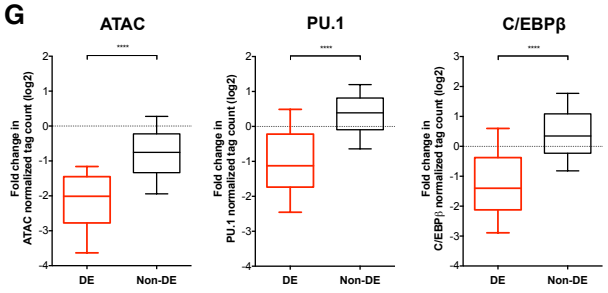


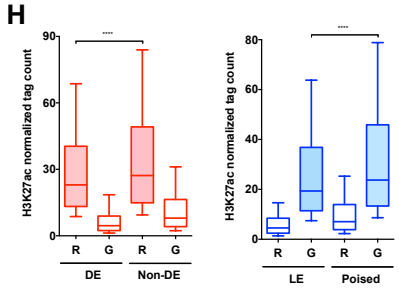












I

	ATAC-seq	PU.1	C/EBPβ	Pol2	SMC1
Resting	91,225	54,236	32,089	21,769	44,039
IFN-γ	72,306	104,987	41,194	23,242	64,293
Donor #	1+1	2+2	2+3	2	5+3

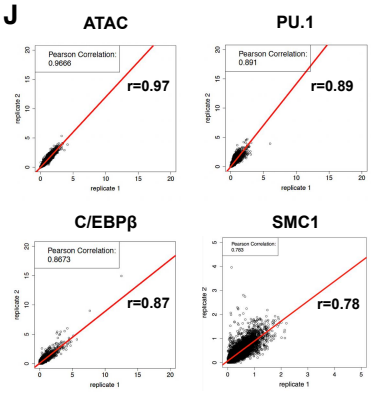


Figure S1. Analysis of RNA-seq Data and of Enhancers that were Suppressed but not Disassembled by IFN-γ, Related to Figure 1

(A) Gene set enrichment analysis (GSEA) of IFN-γ-regulated genes reveals that IFN-γ-inducible genes in this study are enriched in defined gene sets from MSigDB (Broad Institute) of interferon gamma response, inflammatory response and IRF promoter motifs.

(B) The significantly enriched GSEA/MSigDB pathways (HALLMARK, KEGG and REACTOME) for IFN-γ-repressed genes.

(C) Left shows congruence of ATAC-seq peaks and DNase-seq peaks from ENCODE project. Heat maps of normalized tag densities for DNase-seq ($CD14^+$ monocytes from ENCODE, GSM1024791) and ATAC-seq (monocyte-derived macrophages used in this project) at the 21,998 enhancers defined and analyzed in this project. Right shows heat maps of normalized tag densities for H3K4me1 ChIP-seq from $CD14^+$ monocytes (left, GSM1102793) and $CD4^+$ T cells (right, GSM1220567) at the 21,998 enhancers defined in this project. Four-kilobase windows are shown centered at the midpoints of the ATAC-seq peak.

(D) Heat maps of normalized tag densities for the H3K27-Ac histone mark at IFN-γ-repressed, IFN-γ-inducible and constitutive enhancers in resting (R) and IFN-γ-primed (G) macrophages. The boxplots show the fold changes in normalized tag counts for the three distinct sets of enhancers (repressed, inducible, and constitutive). All enhancers are shown ordered by H3K27-Ac signal; Figure 1E shows the same data but breaks out the disassembled and latent enhancers. Boxes encompass the 25th to 75th percentile changes. Whiskers extend to 10th and 90th percentiles. The central horizontal bar indicates the median fold change.

(E) Box plots of the fold change in gene expression after IFN-γ treatment for the nearest differentially expressed genes (within 100 kb) from IFN-γ-repressed enhancers (red) or IFN-γ-inducible enhancers (blue). ****$P < 0.0001$ by Welch's *t*-test.

(F) Heat maps of H3K27ac, ATAC-seq, PU.1 and C/EBPβ signals at enhancers that were regulated by IFN-γ. The left panels show H3K27ac signals for all regulated enhancers, defined by >2-fold change in H3K27ac between resting (R) and IFN-γ-primed (G) macrophages. The right 3 panels show a breakout of ATAC-seq, PU.1, and/or C/EBPβ signals for enhancers where these signals changed by less than or equal to 2-fold. The upper panels show enhancers that were suppressed by IFN-γ; suppressed enhancers that maintained open chromatin and PU.1 or C/EBPβ binding are termed 'non-disassembled enhancers'. Enhancers that exhibit open chromatin prior to activation correspond to poised enhancers as previously described. The box plots indicate normalized tag counts at non-DEs (upper right panels) and poised enhancers (lower right panels).

(G) Box plots of the fold change in normalized tag counts for ATAC-seq (left), PU.1 (middle) and C/EBPβ (right) at disassembled (DE) *versus* non-disassembled enhancers (non-DE). DEs exhibited significantly decreased chromatin accessibility and PU.1 and C/EBP binding than did non-DEs; ****$P < 0.0001$ by Welch's *t*-test.

(H) Box plots of normalized H3K27ac tag counts for DEs versus non-DEs (left) and for latent versus poised enhancers (right). R = unstimulated, G = IFN-γ-stimulated human macrophages. ****$P < 0.0001$ by Welch's *t*-test.

(I) Peak numbers detected by HOMER version 4.7.2. Each ChIP-seq replicate used pooled samples from independent experiments with different donors.

(J) Correlation of ATAC-seq and ChIP-seq replicates.

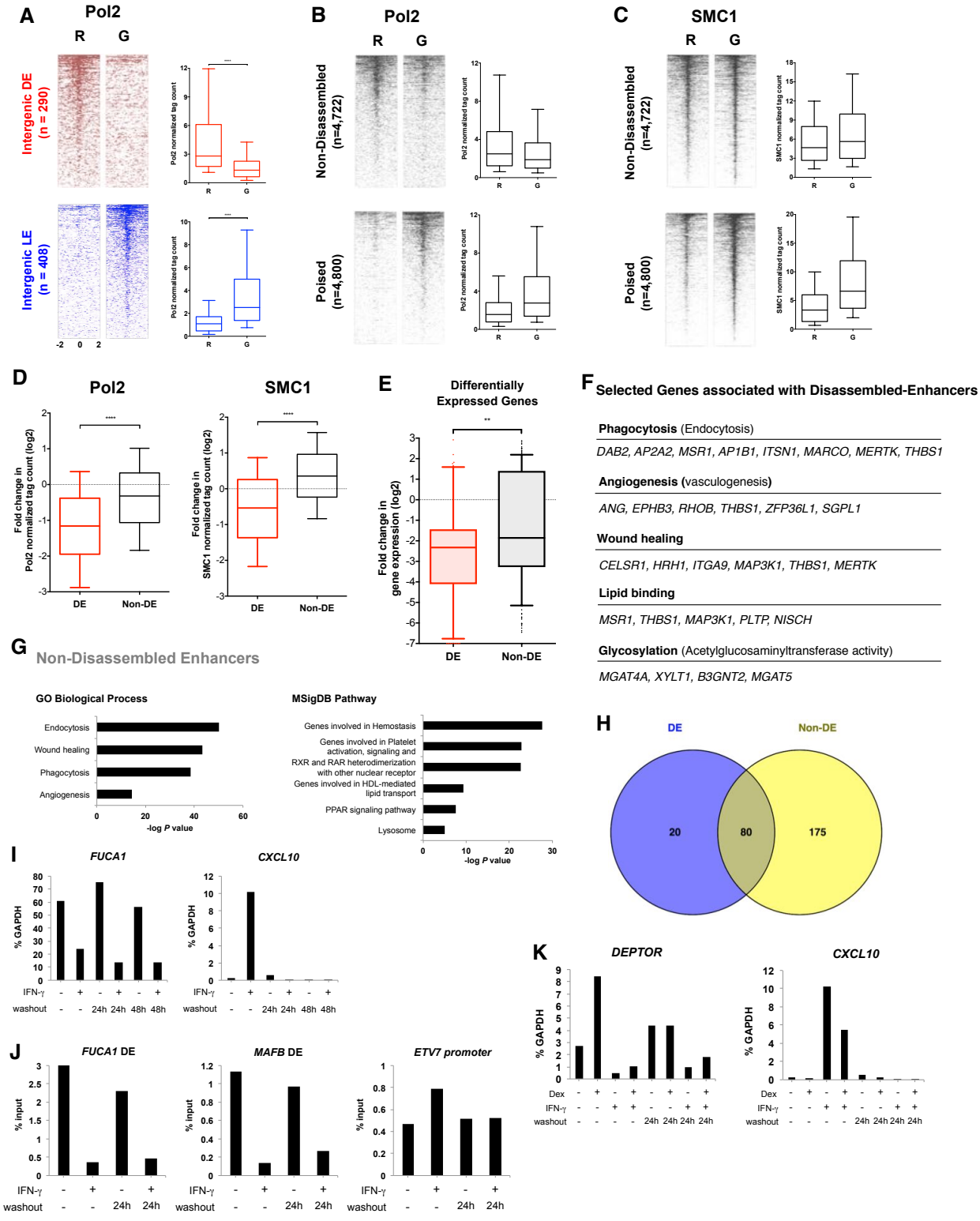

A
Pol2
R
G
Intergenic DE (n = 290)
Intergenic LE (n = 408)
Pol2 normalized tag count
B
Pol2
Non-Disassembled (n=4,722)
Poised (n=4,800)
C
SMC1
SMC1 normalized tag count
D
Pol2
SMC1
Fold change in Pol2 normalized tag count (log2)
Fold change in SMC1 normalized tag count (log2)
DE
Non-DE
E
Differentially Expressed Genes
Fold change in gene expression (log2)
F
Selected Genes associated with Disassembled-Enhancers
Phagocytosis (Endocytosis)
DAB2, AP2A2, MSR1, AP1B1, ITSN1, MARCO, MERTK, THBS1
Angiogenesis (vasculogenesis)
ANG, EPHB3, RHOB, THBS1, ZFP36L1, SGPL1
Wound healing
CELSR1, HRH1, ITGA9, MAP3K1, THBS1, MERTK
Lipid binding
MSR1, THBS1, MAP3K1, PLTP, NISCH
Glycosylation (Acetylglucosaminyltransferase activity)
MGAT4A, XYLT1, B3GNT2, MGAT5
G
Non-Disassembled Enhancers
GO Biological Process
Endocytosis
Wound healing
Phagocytosis
Angiogenesis
-log P value
MSigDB Pathway
Genes involved in Hemostasis
Genes involved in Platelet activation, signaling and
RXR and RAR heterodimerization with other nuclear receptor
Genes involved in HDL-mediated lipid transport
PPAR signaling pathway
Lysosome
H
DE
Non-DE
20
80
175
I
FUCA1
CXCL10
% GAPDH
IFN-γ
washout
24h
48h
J
FUCA1 DE
MAFB DE
ETV7 promoter
% input
K
DEPTOR
CXCL10
Dex

Figure S2. Regulation of Non-Disassembled Enhancers by IFN-γ, Related to Figure 2

(A) Heat maps of normalized tag densities for the Pol II ChIP-seq at 290 intergenic disassembled enhancers (DE) and 408 intergenic latent enhancers (LE) in resting (R) and IFN-γ-primed (G) macrophages. Four-kilobase windows are shown centered at the midpoints of the ATAC-seq peak. The box plots indicate normalized tag counts at intergenic DEs (upper right panels) and intergenic LEs (lower right panels). ****$P < 0.0001$ by paired-samples Wilcoxon signed-rank test.

(B) Heat maps of normalized tag densities for Pol II ChIP-seq at non-disassembled enhancers and poised enhancers in resting (R) and IFN-γ-primed (G) macrophages. The box plots indicate normalized tag counts at non-DEs (upper right panels) and poised enhancers (lower right panels).

(C) Heat maps of normalized tag densities for SMC1 ChIP-seq at non-disassembled enhancers and poised enhancers in resting (R) and IFN-γ-primed (G) macrophages. The box plots indicate normalized tag counts at non-DEs (upper right panels) and poised enhancers (lower right panels).

(D) Pol II and SMC1 occupancy are more strongly downregulated by IFN-γ at DEs relative to non-DEs. Box plots of the fold change in normalized tag counts for Pol II (left) and SMC1 (right) ChIP-seq at disassembled (DE) or non-disassembled enhancers (non-DE). ****$P < 0.0001$ by Welch's *t*-test.

(E) Genes associated with DEs are more strongly downregulated by IFN-γ than genes associated with non-DEs. Box plots show fold change in gene expression after IFN-γ stimulation. **$P = 0.0017$ by Welch's *t*-test.

(F) List of selected genes associated with disassembled enhancers that play important roles in M2-like macrophages identified using DAVID and GSEA. The top 100 DE-associated genes are shown in Table S1.

(G) Functionally enriched Gene Ontology (GO) and MSigDB pathway categories of genes associated with non-disassembled enhancers.

(H) Venn diagram showing number of genes associated with only DE (20, blue), only non-DE (175, yellow), and with both DE and non-DE (80, overlap).

(I) RT-qPCR analysis of the DE-associated gene *FUCA1* in macrophages cultured with or without IFN-γ for 48 hr, followed by washout and incubation in fresh medium for 24 hr or 48hr. *CXCL10* expression serves as a control indicating the termination of IFN-γ signal after washout.

(J) FAIRE analysis of disassembled enhancers in macrophages cultured with or without IFN-γ for 48 hr, followed by washout and incubation in fresh medium for 24 hr. *ETV7* promoter serves as a specificity control.

(K) RT-qPCR analysis of the DE-associated, GC-inducible *DEPTOR*. Cells were cultured as in (I), except dexamethasone (Dex) was added 3 hr prior to harvesting.

Data (I – K) are representative of two experiments.

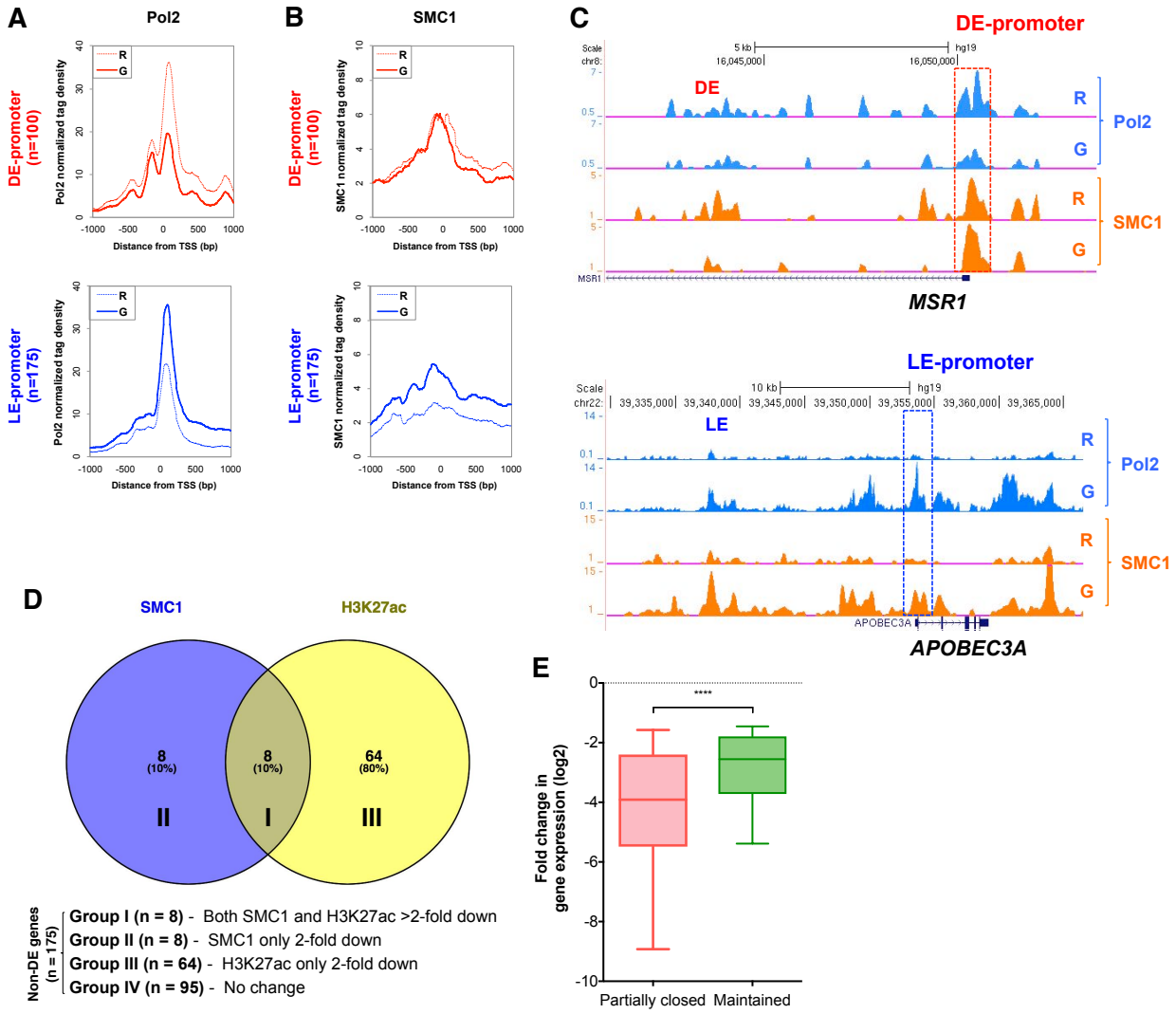

A
Pol2
DE-promoter (n=100)
LE-promoter (n=175)
Pol2 normalized tag density
Distance from TSS (bp)
R
G
B
SMC1
SMC1 normalized tag density
C
DE-promoter
DE
LE-promoter
LE
Pol2
SMC1
MSR1
APOBEC3A
D
SMC1
H3K27ac
8 (10%)
II
8 (10%)
I
64 (80%)
III
Non-DE genes (n = 175)
Group I (n = 8) - Both SMC1 and H3K27ac >2-fold down
Group II (n = 8) - SMC1 only 2-fold down
Group III (n = 64) - H3K27ac only 2-fold down
Group IV (n = 95) - No change
E
Fold change in gene expression (log2)
Partially closed
Maintained
****
F
Partially closed Promoters (n=62)
Motif
TF
P-value
TBP
1e-4
NFY
1e-4
Maintained Promoters (n=596)
NFY
1e-19
Sp1
1e-19
KLF
1e-15
TBP
1e-8

Figure S3. Regulation of Pol II and Cohesin Occupancy at Promoters by IFN-γ, Related to Figure 3

(A) Distribution of average ChIP-seq signal of Pol II at promoters of DE-associated genes (red, upper panel) or LE-associated genes (blue, lower panel) in resting (R, dotted line) and IFN-γ-primed (G, solid line) macrophages.

(B) Distribution of average ChIP-seq signal of SMC1 at promoters of DE-associated genes (red, upper panel) or LE-associated genes (blue, lower panel) in resting (R, dotted line) and IFN-γ-primed (G, solid line) macrophages.

(C) Representative UCSC Genome Browser tracks showing the change of RNA polymerase II (Pol2) and cohesin (SMC1) occupancy at promoters of *MSR1* and *APOBEC3A* genes in resting (R) and IFN-γ-activated (G) macrophages. Boxes enclose promoter of gene associated with disassembled enhancer (DE, top) and latent enhancer (LE, bottom).

(D) Regulation of H3K27ac and SMC1 at promoters of IFN-γ-repressed genes associated with non-disassembled enhancers (n=175 genes). Venn diagram showing the number of promoters of non-DE genes that exhibited > 2-fold decreases in normalized tag counts for SMC1 ChIP-seq alone (8, blue), H3K27ac alone (64, yellow) or both SMC1 and H3K27ac (8, overlap).

(E) Box plot showing $\log_2$ fold change in gene expression of genes associated with partially closed promoters (n=62) and promoters who chromatin accessibility was preserved after IFN-γ stimulation (n=596). ****$P < 0.0001$ by Welch's *t*-test.

(F) Motif enrichment analysis of promoters with partially closed or maintained chromatin accessibility after IFN-γ treatment.

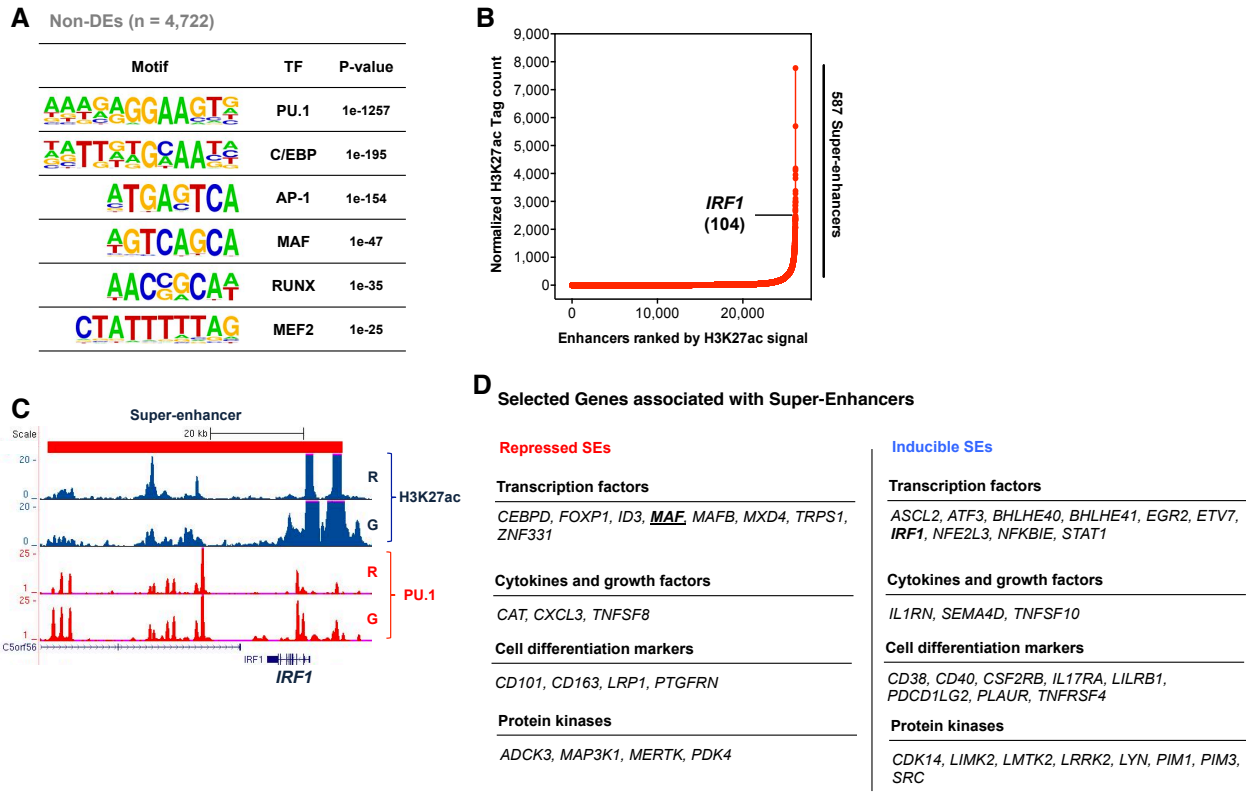


Figure S4. Dynamic Super-Enhancers Associated with Candidate Regulators of Macrophage Function During IFN-γ Stimulation, Related to Figure 4

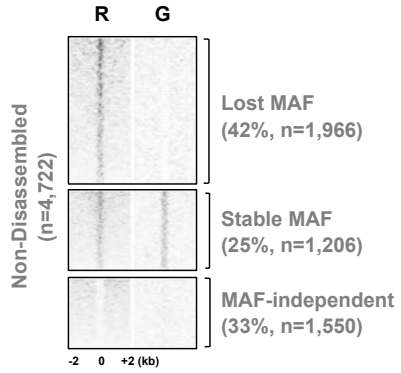
(A) Most significantly enriched transcription factor (TF) motifs identified by *de novo* motif analysis using HOMER at non-disassembled enhancers.

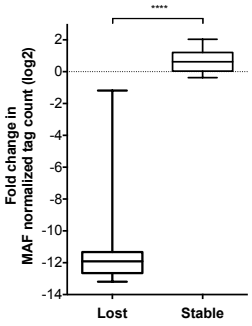
(B) Induced super-enhancers (based on >2-fold increase in H3K27ac tag counts in response to IFN-γ) are plotted in an increasing order of H3K27ac signal. Super-enhancers (SEs) are defined as the population of enhancers above the inflection point of the curve.

(C) A highly ranked inducible SE at the *IRF1* locus.

(D) List of selected genes associated with repressed- (left) or induced- (right) SEs. Genes are grouped according to gene families using GSEA.

A





B

Lost MAF Non-DE (n = 1,966)

Motif	TF	P-value
	PU.1	1e-510
	C/EBP	1e-97
	AP-1	1e-85
	MAF	1e-27

Stable MAF Non-DE (n = 1,206)

Motif	TF	P-value
	PU.1	1e-323
	C/EBP	1e-38
	AP-1	1e-32
	MAF	1e-28

MAF independent Non-DE (n = 1,550)

Motif	TF	P-value
	PU.1	1e-368
	C/EBP	1e-57
	RUNX	1e-13

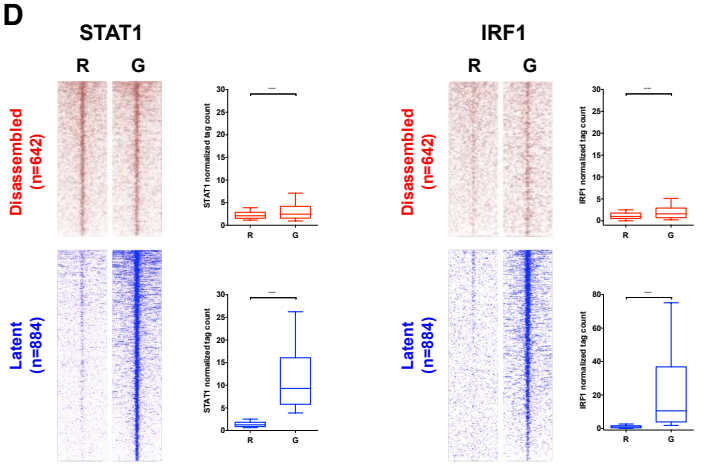
C

MAF-bound	Lost	Stable	Sum
DE	338	154	492
Non-DE	1,966	1,206	3,172
Other Enhancers	744	371	1,115
Total Enhancers	3,048	1,731	4,779

Enhancers	IFN-γ-repressed genes
DE	100
MAF+lost	66 (66%)
MAF+stable	16 (16%)
MAF-independent	18 (18%)

Enhancers	IFN-γ-repressed genes
Non-DE	175
MAF+lost	101 (58%)
MAF+stable	42 (24%)
MAF-independent	32 (18%)

D



E

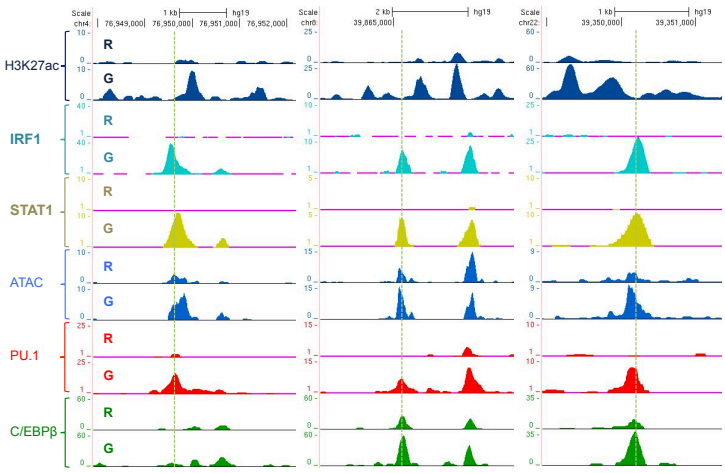


Figure S5. IFN-γ-inducible STAT1 and IRF1 Binding to Latent Enhancers but not to Disassembled Enhancers, Related to Figure 5

(A) Heat map of normalized tag counts for MAF ChIP-seq at non-disassembled enhancers where MAF binding was lost or stable after IFN-γ treatment, or was not detected (MAF-independent). The boxplot shows the fold changes in normalized tag counts for the two distinct groups of non-disassembled enhancers (lost and stable). **** $P < 0.0001$, by Welch's *t*-test.

(B) Most significantly enriched transcription factor (TF) motifs identified by *de novo* motif analysis using HOMER (Heinz et al., 2010) at non-disassembled enhancers where MAF binding was lost (upper panel), stable (middle panel) or not detected (lower panel). Enrichment of the MAF motif was not detected in enhancers that did not bind MAF in ChIP-seq.

(C) Upper table summarizes MAF binding and its loss after IFN-γ treatment at various types of enhancers. Left lower table summarizes the number of IFN-γ-repressed genes associated with three different subsets of disassembled enhancers. Lower right table summarizes the number of IFN-γ-repressed genes associated with three different subsets of non-disassembled enhancers.

(D) Heat maps of normalized tag densities for STAT1 and IRF1 ChIP-seq at DEs and LEs in resting (R) and IFN-γ-primed (G) macrophages. Four-kilobase windows are shown centered at the midpoints of the ATAC-seq peak. The box plots indicate normalized tag counts at DEs (upper right panels) and LEs (lower right panels).

(E) Representative UCSC Genome Browser tracks displaying normalized profiles for active histone mark (H3K27ac), IFN-γ-activated TFs (IRF1 and STAT1), open chromatin (ATAC-seq), and lineage-determining transcription factors (PU.1 and C/EBPβ) signals at latent enhancers (LEs) of *CXCL10*, *IDO2*, and *APOBEC3A* genes in resting (R) and IFN-γ-primed (G) macrophages.

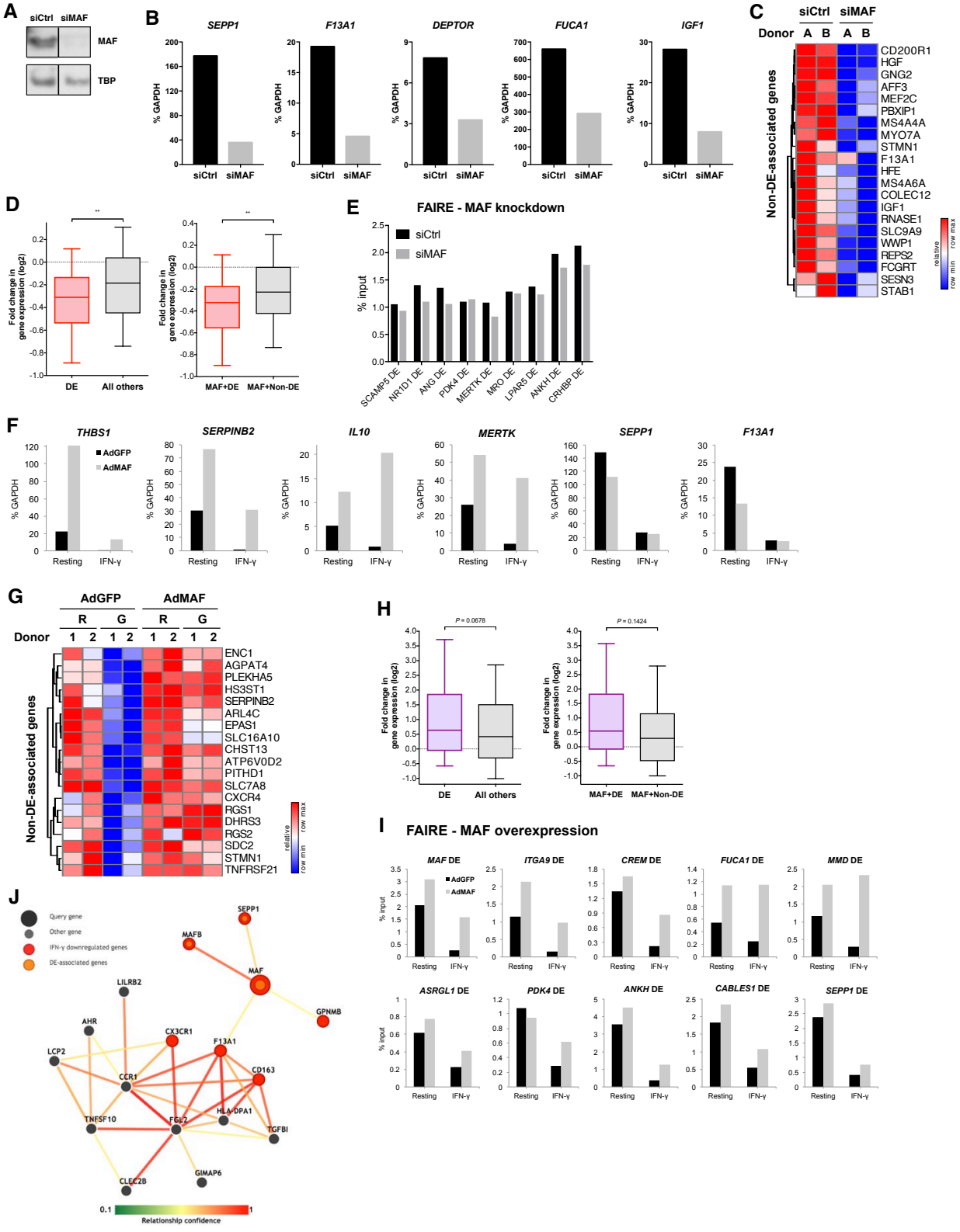

A
siCtrl
siMAF
MAF
TBP
B
SEPP1
F13A1
DEPTOR
FUCA1
IGF1
% GAPDH
siCtrl
siMAF
C
siCtrl
siMAF
Donor
A
B
A
B
Non-DE-associated genes
CD200R1
HGF
GNG2
AFF3
MEF2C
PBXIP1
MS4A4A
MYO7A
STMN1
F13A1
HFE
MS4A6A
COLEC12
IGF1
RNASE1
SLC9A9
WWP1
REPS2
FCGRT
SESN3
STAB1
relative
row min
row max
D
Fold change in gene expression (log2)
DE
All others
MAF+DE
MAF+Non-DE
E
FAIRE - MAF knockdown
siCtrl
siMAF
% input
SCAMP5 DE
NR1D1 DE
ANG DE
PDK4 DE
MERTK DE
MRO DE
LPAR5 DE
ANKH DE
CRHBP DE
F
THBS1
SERPINB2
IL10
MERTK
SEPP1
F13A1
AdGFP
AdMAF
% GAPDH
Resting
IFN-γ
G
AdGFP
AdMAF
R
G
Donor
1
2
Non-DE-associated genes
ENC1
AGPAT4
PLEKHA5
HS3ST1
SERPINB2
ARL4C
EPAS1
SLC16A10
CHST13
ATP6V0D2
PITHD1
SLC7A8
CXCR4
RGS1
DHRS3
RGS2
SDC2
STMN1
TNFRSF21
relative
row min
row max
H
P = 0.0678
P = 0.1424
Fold change in gene expression (log2)
DE
All others
MAF+DE
MAF+Non-DE
I
FAIRE - MAF overexpression
MAF DE
ITGA9 DE
CREM DE
FUCA1 DE
MMD DE
ASRGL1 DE
PDK4 DE
ANKH DE
CABLES1 DE
SEPP1 DE
AdGFP
AdMAF
% input
Resting
IFN-γ
J
Query gene
Other gene
IFN-γ downregulated genes
DE-associated genes
SEPP1
MAFB
MAF
LILRB2
GPNMB
AHR
CX3CR1
F13A1
LCP2
CD163
CCR1
TNFSF10
FGL2
HLA-DPA1
TGFBI
CLEC2B
GIMAP6
0.1
1
Relationship confidence

Figure S6. MAF associates with a Subset of Non-Disassembled Enhancers, Related to Figure 6.

(A) Immunoblot of lysates from macrophages transfected with control or *MAF*-specific siRNAs probed with MAF and TBP antibodies. Two lanes from the same gel were juxtaposed (marked by vertical black line).

(B) Expression of mRNA of *SEPP1*, *F13A1*, *DEPTOR*, *FUCA1* and *IGF1* was measured by RT-qPCR and normalized relative to GAPDH mRNA in resting human macrophages transfected with scrambled control siRNA (siCtrl) or *MAF*-specific siRNA (siMAF). Data are representative of at least three independent experiments.

(C) Heat map showing relative expression of representative non-DE-associated genes that are significantly downregulated by RNAi of *MAF* ($p < 0.05$).

(D) RNAi of *MAF* preferentially affects expression of MAF-bound and DE-associated genes. Box plots show fold change in gene expression after RNAi of *MAF* in resting human macrophages. $^{**}P = 0.0068$ and $^{**}P = 0.0062$ by Welch's *t*-test.

(E) FAIRE analysis of disassembled enhancers in resting macrophages treated with a control siRNA (siCtrl) or with the MAF-specific siRNA (siMAF). Signal is shown as percent of input. Data are representative of two independent experiments.

(F) RT-qPCR analysis of mRNA of *THBS1*, *SERPINB2*, *IL10, MERTK, SEPP1 and F13A1* in IFN-γ-stimulated macrophages transduced with adenoviral particles encoding MAF (AdMAF) or GFP (AdGFP). Data are representative of at least three independent experiments.

(G) Heat map showing relative expression of representative non-DE-associated genes that are significantly upregulated by ectopic MAF expression ($p < 0.05$).

(H) Ectopic expression of MAF preferentially affects expression of DE-associated genes. Box plots show fold change in gene expression after ectopic MAF expression in IFN-γ-stimulated human macrophages. The p values were generated by Welch's *t*-test.

(I) FAIRE analysis of disassembled enhancers (DEs) in resting and IFN-γ-stimulated macrophages transduced with control (AdGFP) or with MAF-expressing (AdMAF) adenovitral particles. Signal is shown as percent of input. Data are representative of two independent experiments.

(J) The functional network analyzed by querying the ImmuNet database (Toll-like receptor signaling pathway network) with MAF, IFN-γ-repressed gene sets (red circles), and DE-associated gene sets (orange circles). The visualization parameters used to generate the graph shown are minimum relationship confidence = 0.5 and maximum number of genes = 20.

Table S1. The top 100 DE-associated genes

SEPP1	ITGA9	KLF2	RAPH1
CD28	RNASE4	LDLRAD4	TNFSF8
KLHL13	BMP2	MERTK	ZBTB4
MRO	LTBP2	NISCH	STARD13
TMEM37	KIF26B	FOXP1	XYLT1
ROR2	PLTP	TESK2	THADA
GPR34	PSCA	AP2A2	C20orf194
ZNF704	PMP22	DCBLD1	SOCS6
CRHBP	ITSN1	MGAT4A	ZFP36L1
THBS1	CELSR1	MAML3	DEF6
ARMC9	RHOB	MAF	MAFB
DEPTOR	EEPD1	TMEM173	MAP3K1
FBLIM1	ADAM11	PPM1L	LY86
PDK4	PEX11G	SSBP2	LPAR6
LGMN	LPAR5	TANC2	SWAP70
MARCO	HRH1	SGPL1	AP1B1
CNRIP1	SLMO1	MGAT5	MSR1
CABLES1	ANKH	B3GNT2	ADAM9
MTSS1	PDGFC	CREM	ZADH2
EPHB3	PIK3IP1	CPQ	ALOX5
SCAMP5	CRYL1	FAM198B	C22orf29
PTGFRN	FUCA1	APMAP	KIAA0195
PID1	MMD	NTAN1	DAB2
KIAA1147	ASRGL1	TRPS1	ADCY9
KIAA1671	ZC3H12D	SMS	RAPH1

Table S2. The top 100 LE-associated genes

HSD11B1	CD80	SEMA4D	CIR1
CXCL10	DGKG	CDK14	PLEK
GBP5	CCND2	NFE2L3	SPATA13
IDO2	EDN1	STX11	RAPGEF2
APOBEC3A	MGC12916	CLCN4	ZNF366
P2RY14	NEDD9	CD226	JAK2
CD274	GPC4	TRIB2	ELOVL5
FAM26F	MREG	TNFSF10	GDPD5
MYO7B	F3	MGAM	USP6NL
FLT1	KCNE1	PLEKHG3	GLIPR2
KREMEN1	ASAP2	PRKAR2B	GK
TGM2	LIF	P2RX7	ANKRD33B
MYOF	RMI2	IL15	LYN
GPR88	FBP1	MX1	ZCCHC2
DCSTAMP	SLIT2	PPARGC1B	ATOX1
CD38	SOD2	GP1BA	HIVEP2
MYEOV	ALAS1	VOPP1	KLF9
IL1RN	SERPINA1	DRAM1	SLC1A3
TRIM46	SH2B2	TAGAP	EXT1
CXCL11	UBE2L6	ISG20	KLF10
TMEM217	PDE4B	TMSB10	KIAA0247
EGR3	RHBDF2	FAM213A	RAB39A
GCH1	NLRC5	IRG1	NFKBIA
SNX10	LMTK2	OPTN	CSF2RB
BCL2A1	C4orf32	TRAF2	NMI

Table S3. Primers for mRNA expression

hMAF_F	CTGGCAATGAGCAACTCCGA
hMAF_R	AGCCGGTCATCCAGTAGTAGT
hSEPP1_F	AAAGCTCCTTATGTAAGCAACCC
hSEPP1_R	ACAGTCACTGAACCATTGGAGT
hF13A1_F	CCCAATAACTCTAATGCAGCGG
hF13A1_R	ACCTTGTTAGTGTCCCATCTCTC
hDEPTOR_F	TTAGCAGACCGGGGCATTATT
hDEPTOR_R	GAAGGTGCCGTCATCCTTTCT
hFUCA1_F	GAAGCCAAGTTCGGGGTGTT
hFUCA1_R	GGGTAGTTGTCGCGCATGA
hTHBS1_F	AGACTCCGCATCGCAAAGG
hTHBS1_R	TCACCACGTTGTTGTCAAGGG
hSERPINB2_F	CAGCACCGAAGACCAGATGG
hSERPINB2_R	CCTGCAAAATCGCATCAGGATAA
hIL10_F	GACTTTAAGGGTTACCTGGGTTG
hIL10_R	TCACATGCGCCTTGATGTCTG
hMERTK_F	CCACAATTTCTTGGTGGAAAGA
hMERTK_R	AGATGGGATCAGACACGATCTC
hCXCL10_F	GTGGCATTCAAGGAGTACCTC
hCXCL10_R	TGATGGCCTTCGATTCTGGATT

Table S4. Primers for ChIP-qPCR and FAIRE assay

ChIP_control_F	GGTTGTGGTGGTAAGAAGTTGA
ChIP_control_R	TGAAAACACTCCAAGGCAGA
SEPP1_DE_F	ACATTGCCAGGCAGTAAAGG
SEPP1_DE_R	CGCAATTCCATTTTGTTTCC
ITGA9_DE_F	GACCAGGAAGCTCTTGAGGT
ITGA9_DE_R	TTTGCCCCAAGAAAGCAGAC
MMD_DE_F	AAGTTCCCCATTTCCGAGGG
MMD_DE_R	GCAGAGCAGGAGACAAATGG
MAF_DE_F	CTTGGCCTGGAGAATCTTGC
MAF_DE_R	AAGGGGAAGGAAGCTAACCC
FUCA1_DE_F	GAAATTAGAAGAAGGGCCATGG
FUCA1_DE_R	CAGCCAGCTTTGACTTTGTC
CABLES1_DE_F	GCTGACCAAGTAGAATCACGT
CABLES1_DE_R	GAGATTCAGGAGGCCAGGAG
STARD13_DE_F	AAGCCCTGTGTCAGCTTTTG
STARD13_DE_R	GGACTGCCGGACCTATGAAA
MTSS1_DE_F	CGTGATGCAGAAGGAAGCC
MTSS1_DE_R	CCCAGTTCCCCTTCGTTGAA
DAB2_DE_F	GGAGACTCAAGAAGAAAGCAACA
DAB2_DE_R	ATTCCGCACTTTAGCTGAGG
MAFB_DE_F	GGGGCAGAGAAGAGGCTACT
MAFB_DE_R	CGACTTCCTTTCCCTGACTG
CREM_DE_F	CTGCTTCCTCCACTTCCTGA
CREM_DE_R	TGAAACACTCAGGACAGGGG
THBS1_DE_F	AGGCAGAATAACCAGGAAGCT
THBS1_DE_R	GATTGGGTAGAGCTGTCCGA
HRH1_DE_F	AGGCCTAAAACTAGCCCAGG
HRH1_DE_R	CTGCGTCCACACTTTCATCC
CRHBP_DE_F	AGCCAAGAACCCCTATCAACT
CRHBP_DE_R	AGGGAAAATGCTACCAGTGC
MERTK_DE_F	TCAAACCAGCAGAGGACCAA
MERTK_DE_R	GGTCGTCCCTCTTAGAGCAA

MRO_DE_F	GGAGACCCAGGGAAGAGTTG
MRO_DE_R	ATAAGAACTGACCTCCCCTGA
LPAR5_DE_F	ACGCTTAGGTTGCTGTTACT
LPAR5_DE_R	GCTGTGAACATGCCTGGAAC
ARMC9_DE_F	ACCAACTGTGAGGAGCTAGG
ARMC9_DE_R	CGCAGTGCCTTAAATTTCTGAC
ZNF704_DE_F	TTTCAGTCTGAGGAGGGCTG
ZNF704_DE_R	CCTCTCTCCGCTCATGTGAT
DEPTOR_DE_F	AACCAGGGGAAATCTCTTCAA
DEPTOR_DE_R	CAAACTCCTCCCTGAATAAACG
GPR34_DE_F	TGTGTGGCATTTAAGGAAGTGG
GPR34_DE_R	GTTCAGCTCTTGTGTTCCTCA
SCAMP5_DE_F	CCATTGGTTTAATCAGTCCCCTA
SCAMP5_DE_R	CCCATACACAGGTGCATTAATTG
ANG_DE_F	ACACTGCAAGAATCAAGCCG
ANG_DE_R	CTCTCTCATCCCACACCCAA
PDK4_DE_F	TGCAAAAGGGAGGGGAAAGA
PDK4_DE_R	AATGTCCATCTCCTCCCCAG
ANKH_DE_F	TCAAACACAGCACATTCCCC
ANKH_DE_R	ATAGGACTGGGCAAAGGAGG
ASRGL1_DE_F	TGCAGAAGTCCAGTTTATCAGT
ASRGL1_DE_R	GCAATGCTGGGTTAGGCAAA
F13A1_E_F	AGCCAAATAAAGGGGCAAGT
F13A1_E_R	CTTTCACAGGAAGGGCTCTG
CXCL10_LE_F	GTGAGCAGGAGGACATCAGT
CXCL10_LE_R	TGTGAGCTCGGGGAATCTTT
ETV7_pro_F	CATCACCCTTTCCATCTGTCTC
ETV7_pro_R	TCTTCGGTGTCTTCTGGTTTGT