## Supplemental Methods

### Cell culture

HEK293FT (Invitrogen) cells were cultured in DMEM supplemented with 10% FBS and 1% antibiotics. Human leukemia cell lines MOLM-13, MV4-11, OCI-AML2, THP-1, HEL, U937, SET-2, and K562 were cultured in RPMI 1640 supplemented with 10% FBS and 1% antibiotics. Puromycin-resistant Cas9-expressing AML cell lines, including MOLM-13, MV4-11, HEL, U937, SET-2, and K562, were kindly provided by Christopher R. Vakoc.[1] Blasticidin-resistant Cas9-expressing OCI-AML2, THP-1, and MV4-11 cell lines were established by lentiviral delivery of lentiCas9-Blast (Addgene, 52962). All cell lines were tested free of mycoplasma contamination using MycoAlert PLUS Mycoplasma Detection Kit (Lonza).

### Plasmids

Human MEF2D cDNA (NM_001271629.2) was a kind gift from Charles Mullighan (St. Jude) and was cloned into pCDH-EF1α-MCS-IRES-Neo/Puro lentiviral vectors (System Biosciences, CD533A-1/CD532A-1). Murine Hoxa9 cDNA (NM_010456.3) was cloned into pCDH-EF1α-MCS-IRES-Neo vector with an N-terminal HA tag. Retroviral vector MSCV_Cas9_puro (Addgene, 65655) or lentiviral vector lentiCas9-Blast (Addgene, 52962) were used to generate stable Cas9-expressing cells. Lentiviral vectors LRCherry2.1 (Addgene, 108099) or LRNeo2.1 were used to express sgRNAs. LRNeo2.1 is a home-made vector constructed by replacing the mCherry cassette of the LRCherry2.1 vector with a Neomycin resistance gene. The sgRNA target sequences used in this study are as follows: sg*MEF2D* e2.1: TCT AGG ACT TCC CTA CCT GT, sg*MEF2D* e4.1: CAA GTA CCG ACG CGC CAG CG; sg*MEF2D* e5.1: CAT CAT CCC TCA CGG ACC CG; sg*Luc*, CCC GGC GCC ATT CTA TCC GC; sg*PRS19*, GTA GAA CCA GTT CTC ATC GT; sg*CEBPE*.1, ACA CTC GTA GTA GGT CCC GT, sg*CEBPE*.2, AGT GGC TGC TGG CCA CCC CG; sg*ROSA26*, ACC TAC CAC ACT AGC CCG A. All plasmids were verified by Sanger sequencing before use.

**Antibodies**

The primary antibodies used in this work include: MEF2D rabbit polyclonal antibody (Proteintech, 14353-1-AP), GAPDH rabbit polyclonal antibody (Proteintech, 10494-1-AP), Tubulin mouse monoclonal antibody (Sigma, T6199), CEBPE rabbit polyclonal antibody (Proteintech, 14271-1-AP), Vinculin rabbit polyclonal antibody (Proteintech, 26520-1-AP), H3K27ac rabbit polyclonal antibody (Abcam, ab4729). The HRP-conjugated secondary antibodies, including the anti-mouse IgG (7076S) and the anti-rabbit IgG (7074S), were obtained from Cell Signaling Technology. Antibodies used for flow cytometry were FITC anti-human CD14 (BioLegend, 301804), FITC anti-human CD33 (BioLegend, 303304), FITC anti-human CD11b (BioLegend, 301330), and FITC anti-human CD45 (BioLegend, 304054).

**Virus production, infection and CRISPR/Cas9-mediated gene knockout**

Lentiviral particles were prepared using HEK293FT packaging cells (Invitrogen) via Nanofect (ALSTEM, NF100)-mediated transfection of gene delivery vector co-transfected with lentiviral packaging vectors psPAX2 and pMD2.G as previously described.[2] Virus-containing supernatant was harvested 48 or 72 hr after transfection and pooled. For infection, virus-containing supernatant was mixed with target cells supplied with 8 μg/mL polybrene (Sigma-Aldrich, TR-1003-G), and then centrifuged at 1,500 rpm for 60 min at room temperature. Fresh media was changed 24 hr post-infection. Appropriate antibiotics were added 24 hr post-infection when selection was needed. In order to prevent overgrowth of atypical variants due to prolonged cell culture, all CRISPR knockout cells were freshly made by lentiviral delivery of sgRNAs and used for each downstream experiment without subcloning. High infection efficiency was confirmed by flow cytometry or achieved by selection with antibiotics. Knockout efficiency was confirmed by Western blotting for each experiment.

**Inhibitor treatment**

MOLM-13 cells were seeded at a density of 70,000 cells/mL in medium supplemented with DMSO vehicle or different doses of the DOT1L inhibitor SGC0946 (MedChemExpress). Medium was replaced every

three days, and fresh inhibitor was added. After 4- or 6-day treatment, cells were collected for gene expression and phenotypic analyses. Cell viability was determined by ATP-based luminescent assay using CellTiter-Glo 2.0 (Promega).

**Quantitative real-time PCR**

Total RNA was isolated using TRI Reagent and Direct-zol RNA Miniprep Kits (Zymo Research, R2053). First strand cDNA was synthesized using the High-Capacity cDNA Reverse Transcription Kit (Applied Biosystems). Quantitative real-time PCR was performed in triplicate using the iTaq Universal SYBR Green Supermix (Bio-Rad) on the Quant Studio 6 Flex Real-Time PCR System (Applied Biosystems). Expression levels were determined using the ΔΔCt method normalized to the housekeeping gene GAPDH. All real-time PCR primer sequences are provided in Supplementary Table 4.

**Immunoblotting**

Cells lysate was prepared by using RIPA buffer followed with SDS-PAGE and transferred to a PVDF membrane according to the manufacturer's instructions (Bio-Rad). After blocking with 5% non-fat milk in TBST (10 mM Tris, pH 8.0, 150 mM NaCl, 0.5% Tween-20) for 1 hour at room temperature, the membrane was incubated with primary antibodies at 4 °C overnight with gentle shaking. Membranes were then washed three times for 30 mins and incubated with HRP-conjugated secondary antibodies for 2 hours at room temperature. Blots were washed with TBST three times for 30 mins and developed with chemiluminescent HRP substrate (Thermofisher). Images were acquired and processed using the G: BOX Chemi XRQ system (Syngene).

**Flow cytometry**

All flow cytometry analyses were performed on the LSR Fortessa X-20 flow cytometer (BD Biosciences) and all data were analyzed with FlowJo (v10). Cell sorting was performed on a FACSAria (BD Biosciences) at the Flow Cytometry Core at the University of Alabama at Birmingham. For cell apoptosis

assay, cells were washed once with PBS and resuspended in 100 µL of Annexin V Binding Buffer (BioLengend, 422201) containing 5 µL of FITC-conjugated Annexin V (BioLengend, 640906) with 15-minute incubation in the dark. DAPI (Sigma, D9542) was added to each sample prior to flow cytometry analyses. For cell cycle analysis, cells were washed once with PBS and fixed with 70% ethanol overnight at −20°C. Fixed cells were then washed with PBS and incubated with 1 µg/mL DAPI in PBS for 30 min at 4°C. For expression of surface markers, cells were washed once with PBS and stained with fluorescence-conjugated antibodies for 30 min at 4°C.

**_In vivo_ leukemia growth in xenograft models**

All animal experiments were approved by the Institutional Animal Care and Use Committees (IACUC) at the University of Alabama at Birmingham. Cas9 and Luciferase-expressing MOLM-13 cells were transduced with LRcherry2.1 vector encoding sg*Rosa26* (negative control) or s*gMEF2D* e2.1. Infected cells were sorted by mCherry expression on day 3 post infection. 0.2 million sorted viable cells were transplanted into sublethally (1.5 Gy) irradiated NOD scid gamma (NSG) immunocompromised mice by tail-vein injection. *In vivo* bioluminescence imaging was acquired by using the IVIS Spectrum Imaging System on day 16 post injection. The percentage of human CD45-positive cells in peripheral blood were measured at indicated time points by flow cytometry.

**RNA-seq and data analysis**

RNA-seq libraries were generated using the NEBNext Ultra RNA Library Prep Kit for Illumina (New England Biolabs), following the manufacturer's protocol. The quality and concertation of the libraries were examined by Bioanalyzer High Sensitivity DNA Chip (Agilent). The multiplexed RNA-Seq libraries were paired-end sequenced for 150 bp on the Illumina HiSeq 4000 platform. The paired-end reads were mapped to the human genome (hg19) using STAR (v2.5.1b) with default parameters. The read counts for genes with average FPKM > 1 in either control or knockout samples were used for DESeq2 (v3.11)

to define differentially expressed genes (DEGs) between MEF2D knockout (a combination of sg*MEF2D* e2.1 and e5.1) and control (sg*Luc*) samples.

**Gene Set Enrichment Analysis (GSEA)**

GSEA analysis was performed for testing enrichment of curated gene sets (C2) or customized gene sets using GSEA (v4.0.3) software.[3]

**Chromatin immunoprecipitation (ChIP) and sequencing**

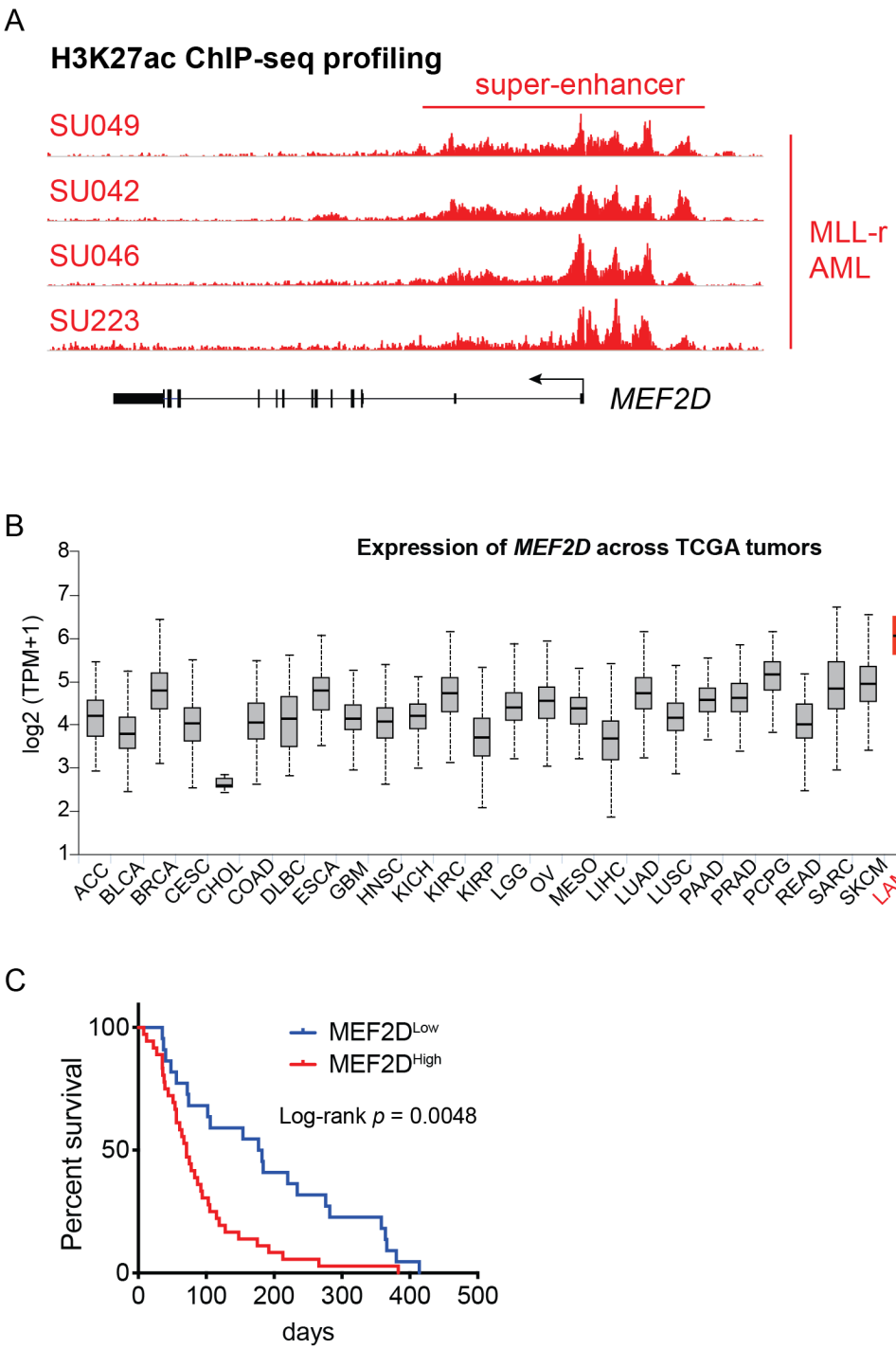
ChIP was performed as previously described with modifications.[4,5] In brief, 20 million MOLM-13 or MV4-11 cells were cross-linked with formaldehyde (1%) for 10 minutes followed by quenching with 0.125 M glycine for 5 minutes at room temperature. After washing with cold PBS, cells were suspended in lysis buffer (50 mM HEPES pH 7.5, 150 mM NaCl, 1 mM EDTA, 0.5 mM EGTA, 1% TritonX-100, 0.1% SDS, 0.1% N-lauroylsarcosine, and protease inhibitor) and subjected to sonication to shear the chromatin DNA into sizes ranging from 200 to 500 bp. The fragmented chromatin was pre-cleared by centrifugation at 14,000 rpm for 15 min at 4°C. The supernatant was incubated with primary antibody and Protein A or G Dynabeads magnetic beads (Invitrogen) at 4°C overnight. After four times wash with high-salt buffer (20 mM Tris-HCl pH 8.0, 2 mM EDTA, 0.1% SDS, 500 mM NaCl, and 1% TritonX-100) and twice wash with TE buffer (10 mM Tris-HCl pH 8.0, 1 mM EDTA), chromatin was eluted from beads in elution buffer (50 mM Tris-HCl pH 8.0, 10 mM EDTA, 1% SDS). After reverse crosslinking, ChIP DNA was purifiied using DNA purification columns (Zymo Research) and subjected to downstream quantitative real-time PCR analysis (ChIP-qPCR) or library preparation for deep sequencing (ChIP-seq). ChIP-seq library was prepared using NEBNext Library Prep Kit following the instructions and subjected to paired-end, 150-cycle sequencing on the Illumina HiSeq 4000 platform. All ChIP-qPCR primer sequences are provided in Supplementary Table 4.

**ATAC-seq**

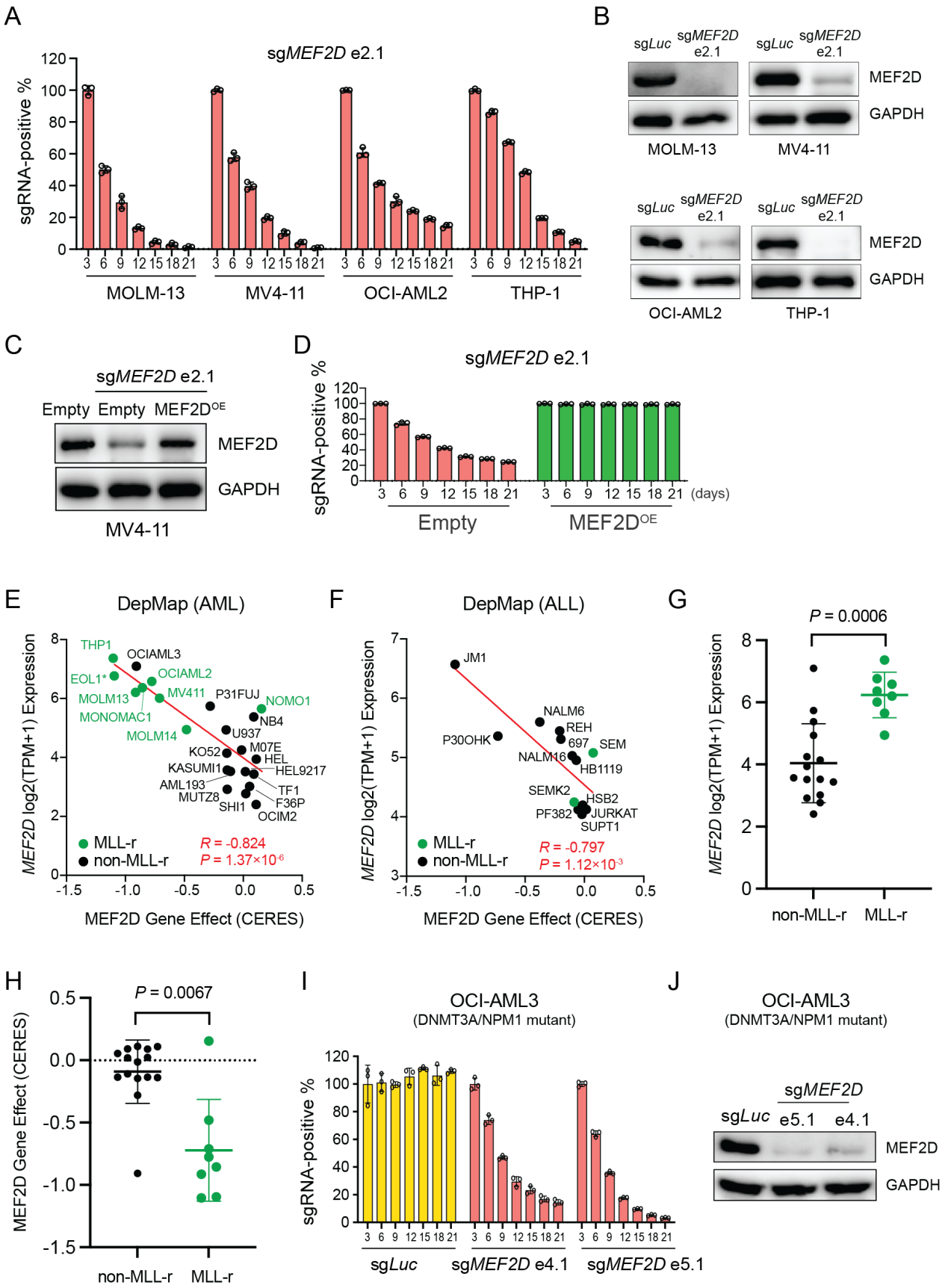
ATAC-seq was performed as previously described.[6] 50,000 MOLM-13 cells were washed once with cold phosphate-buffered saline (PBS) and resuspended with 50 µl cold ATAC-Resuspension Buffer (10 mM Tris-HCl pH 7.5, 10 mM NaCl, 3 mM Mg2Cl, 0.1% NP-40, 0.1% Tween-20, 0.01% Digitonin, and protease inhibitor). After incubation on ice for 3 minutes, the cell nuclei were collected by centrifugation at 500 g for 5 mins at 4°C. Tn5 transposition of nuclei pellets was carried out at 37°C for 30 min using the DNA Sample Preparation Kit from Nextera (Illumina). The transposed DNA was purified using DNA purification columns (Zymo Research, D4003) and subjected to PCR amplification for library generation. ATAC-seq libraries were sequenced on the Illumina HiSeq 4000 platform for paired-end 150 cycles.

**ChIP-Seq and ATAC-seq data analysis**

All sequencing reads were mapped to the human genome (hg19) using the Bowtie2 (v2.2.3) and reads with a mapping score > 20 were selected for data analysis, after duplicated reads removed by SAMtools (v1.9). MACS2 were used to call ChIP-seq peaks with input as controls and ATAC-seq peaks, both using default parameters in paired-end mode. The overlapped peaks called from both replicates were selected as final peaks and their genomic locations related to genes were annotated as described before.[5,7] Bigwig files were generated by bamCoverage and used for visualized in IGV. Heatmaps were generated by Deeptools. For ATAC-seq, peaks from control and MEF2D knockout samples were merged and then used for counting reads in each sample to determine chromatin regions with differential accessibility using DESeq2. Differential ATAC-seq peaks were annotated by the Genomic Regions Enrichment of Annotations Tool (GREAT) for enriched functions.[8] Motif discovery was performed using the Homer software, with both default setting and the input of HOCOMOCO human transcription factor database.




**Supplemental Figure 1. MEF2D is a super-enhancer gene overexpressed in human MLL-r AML.** (**A**) H3K27ac ChIP-seq tracks in patient AML samples. Identified super-enhancer regions were highlighted in samples carrying MLL translocation. (**B**) Expression of MEF2D is the highest in AML among various TCGA tumor types. (**C**) Survival analysis in AML patients (GSE5122) with high MEF2D expression (n=36), comparing to those with low MEF2D expression (n=22).


A
sgMEF2D e2.1
sgRNA-positive %
MOLM-13
MV4-11
OCI-AML2
THP-1
B
sgLuc
sgMEF2D e2.1
MEF2D
GAPDH
MOLM-13
MV4-11
OCI-AML2
THP-1
C
sgMEF2D e2.1
Empty Empty MEF2D^OE
MEF2D
GAPDH
MV4-11
D
sgMEF2D e2.1
sgRNA-positive %
Empty
MEF2D^OE
(days)
E
DepMap (AML)
MEF2D log2(TPM+1) Expression
MEF2D Gene Effect (CERES)
MLL-r
non-MLL-r
R = -0.824
P = 1.37×10^-6
F
DepMap (ALL)
MEF2D log2(TPM+1) Expression
MEF2D Gene Effect (CERES)
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non-MLL-r
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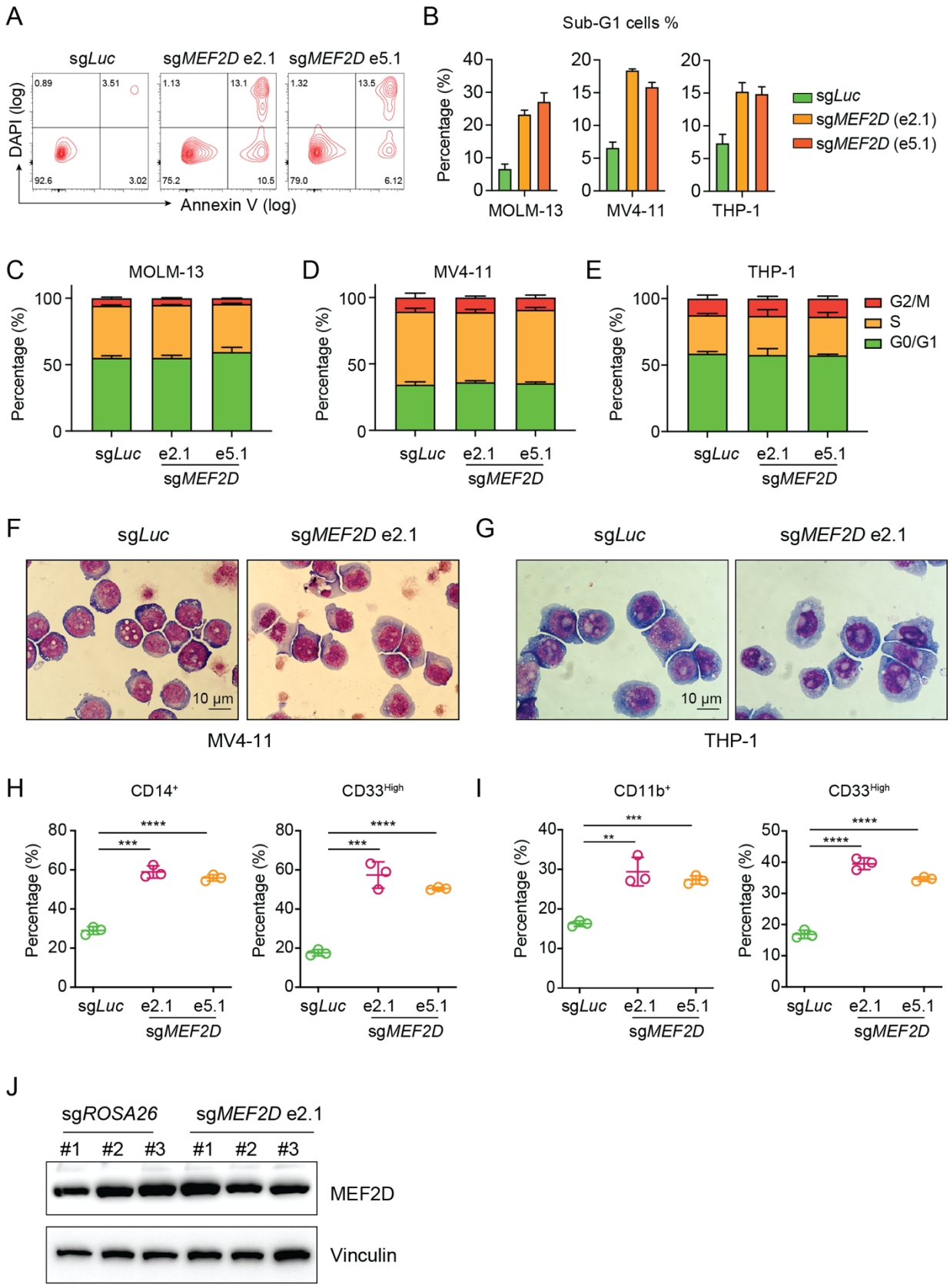
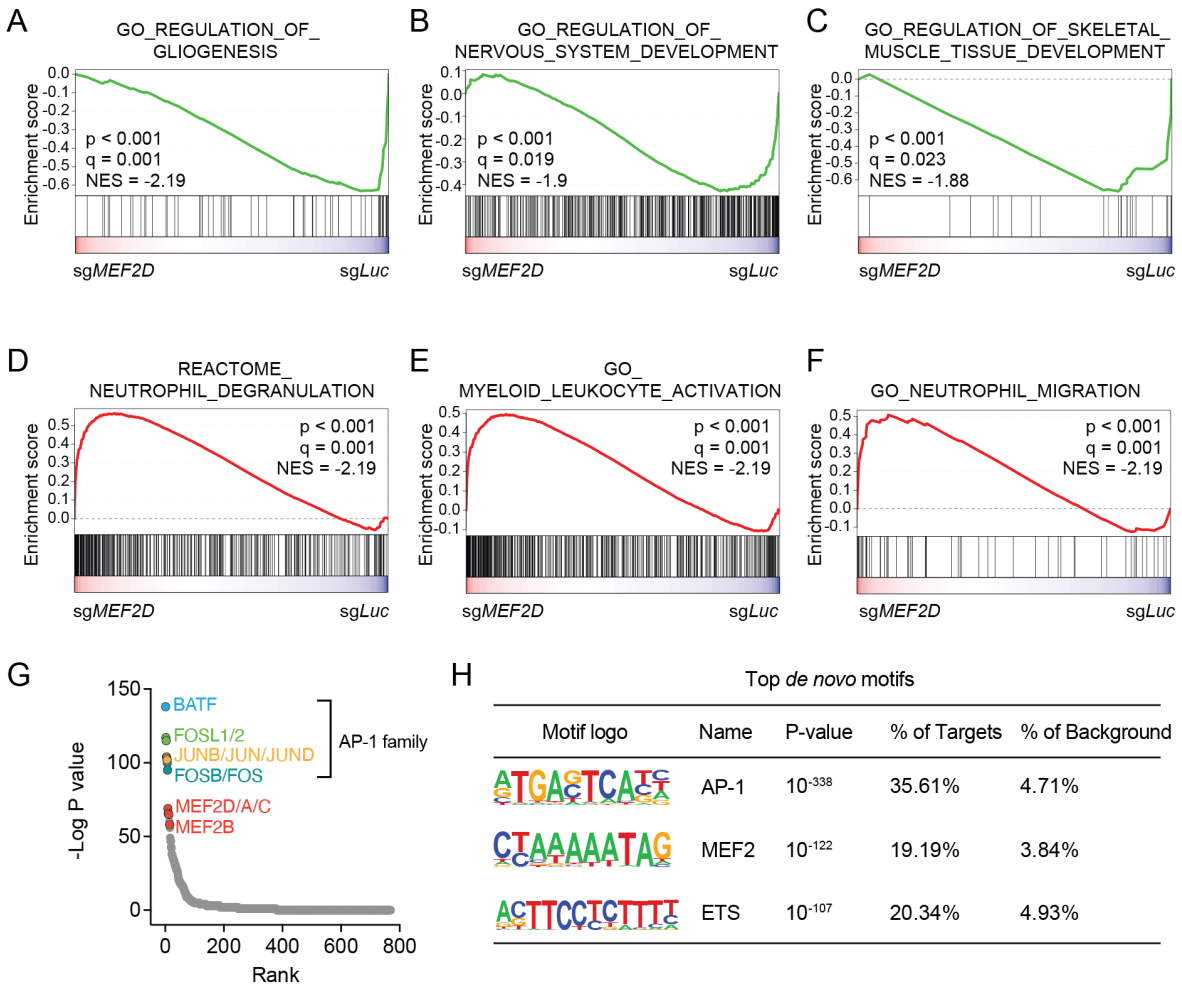
P = 1.12×10^-3
G
P = 0.0006
MEF2D log2(TPM+1) Expression
non-MLL-r
MLL-r
H
P = 0.0067
MEF2D Gene Effect (CERES)
non-MLL-r
MLL-r
I
OCI-AML3
(DNMT3A/NPM1 mutant)
sgRNA-positive %
sgLuc
sgMEF2D e4.1
sgMEF2D e5.1
J
OCI-AML3
(DNMT3A/NPM1 mutant)
sgMEF2D
sgLuc
e5.1
e4.1
MEF2D
GAPDH

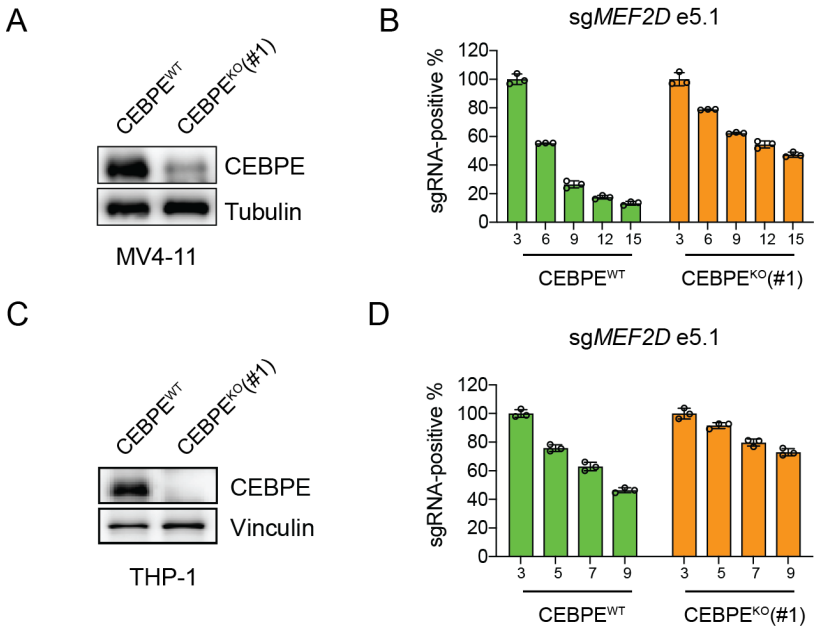
**Supplemental Figure 2. MEF2D is required for MLL-rearranged AML.** (**A**) Competitive proliferation assay showing that MEF2D is required for cell proliferation in indicated MLL-r AML cells. Cells were infected with indicated guide RNA, which is linked with an mCherry gene. The mCherry positive percentage was normalized to the day 3 measurement. A guide RNA targeting MEF2D exon 2 (e2.1) was used for MEF2D knockout. (**B**) Western blot analysis showing CRISPR/Cas9-mediated MEF2D gene knockout in four MLL-r AML cells. A guide RNA targeting MEF2D exon 2 (e2.1) was used for MEF2D knockout. GAPDH serves as a loading control. (**C**) Western blot analysis showing MEF2D protein levels in MV4-11 cell with overexpression of either MEF2D or an empty vector. (**D**) Competitive proliferation assay in indicated MV4-11 Cas9-expressing cell lines showing that the effects of MEF2D loss can be recused by exogenous expression of MEF2D. OE, overexpression. (**E-F**) Correlation analysis of MEF2D gene expression and dependency in AML (n=23) and ALL (n=14) cell lines in Broad Institute Cancer Dependency Map datasets. Cell lines with MLL rearrangements are labeled in green. *, EOL1 carries partial tandem duplication of the MLL gene (MLL-PTD). A lower CERES score indicates a higher likelihood that the gene is essential in a given cell line. A CERES score that >0 indicates the gene is not essential. (**G-H**) MEF2D expression (G) and gene knockout effect (H) in MLL-r (n=8) and non-MLL-r (n=15) AML cell lines. Data were analyzed by Mann-Whitney U test. (**I**) Competitive proliferation assay showing that MEF2D is required for cell proliferation in non-MLL-r OCI-AML3 AML cells. Cells were infected with indicated guide RNA, which is linked with an mCherry gene. The mCherry positive percentage was normalized to the day 3 measurement. Two independent guide RNAs targeting MEF2D (e4.1 and e5.1) were used for MEF2D knockout. (**J**) Western blot analysis showing CRISPR /Cas9-mediated MEF2D gene knockout in OCI-AML3 AML cells. Two independent guide RNAs targeting MEF2D (e4.1 and e5.1) were used for MEF2D knockout. GAPDH serves as a loading control.


A
sgLuc
sgMEF2D e2.1
sgMEF2D e5.1
DAPI (log)
Annexin V (log)
0.89
3.51
92.6
3.02
1.13
13.1
75.2
10.5
1.32
13.5
79.0
6.12
B
Sub-G1 cells %
Percentage (%)
MOLM-13
MV4-11
THP-1
sgLuc
sgMEF2D (e2.1)
sgMEF2D (e5.1)
C
MOLM-13
D
MV4-11
E
THP-1
Percentage (%)
G2/M
S
G0/G1
sgLuc
e2.1
e5.1
sgMEF2D
F
sgLuc
sgMEF2D e2.1
10 μm
MV4-11
G
sgLuc
sgMEF2D e2.1
10 μm
THP-1
H
CD14+
CD33High
Percentage (%)
****
***
sgLuc
e2.1
e5.1
sgMEF2D
I
CD11b+
CD33High
Percentage (%)
***
**
****
sgLuc
e2.1
e5.1
sgMEF2D
J
sgROSA26
sgMEF2D e2.1
#1
#2
#3
#1
#2
#3
MEF2D
Vinculin

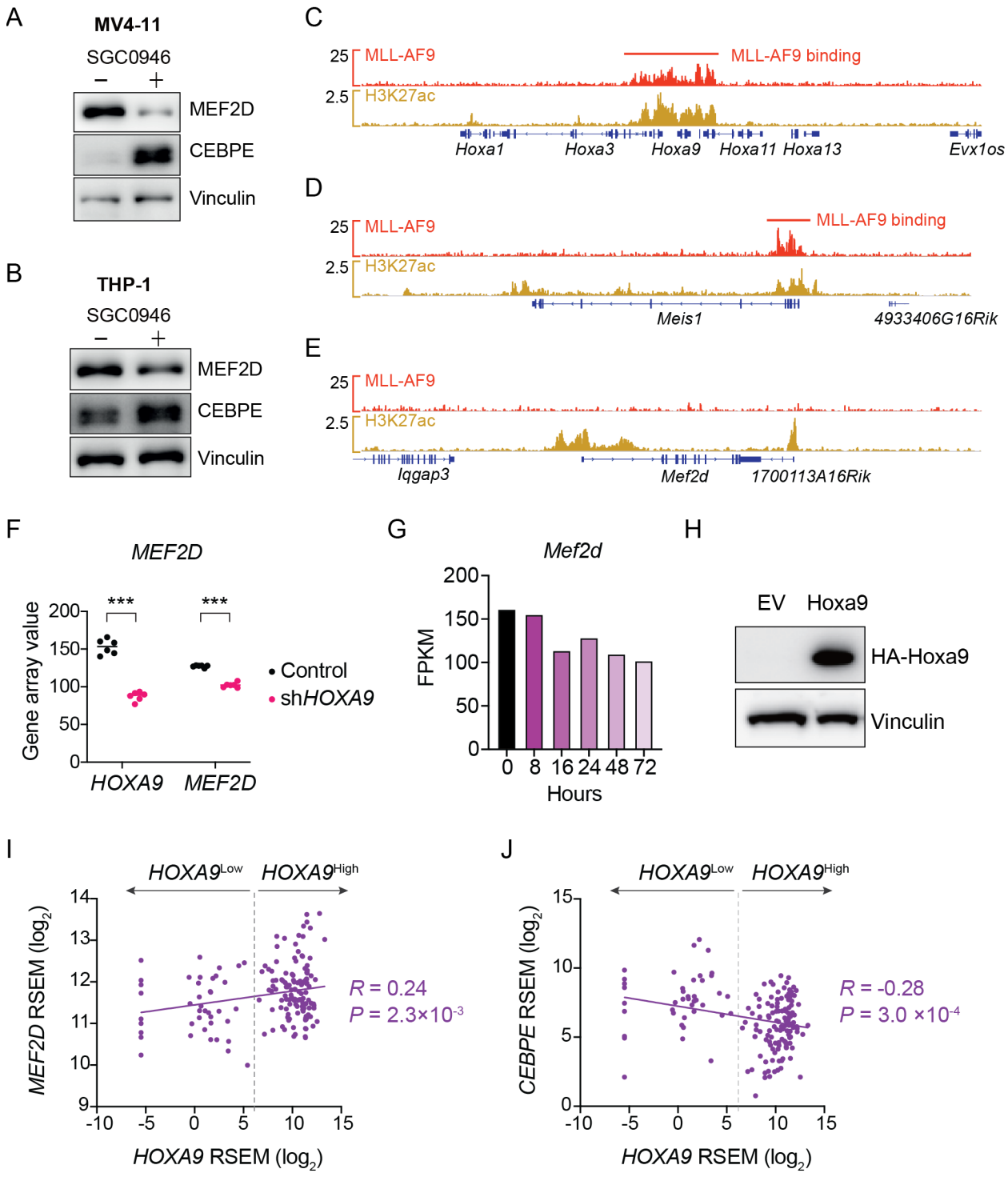
**Supplemental Figure 3. MEF2D knockout promotes apoptosis and differentiation of MLL-r AML cells.** (**A**) Analysis of cell apoptosis in MOLM-13 cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. **(B)** Analysis of sub-G1 population in indicated AML cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**C-E**) Analysis of cell cycle profiles of MOLM-13 (C), MV4-11 (D), and THP-1 (E) cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**F-G**) Wright-Giemsa of MV4-11 (F) and THP-1 (G) cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**H**) Flow cytometry analysis of populations with myeloid differentiation markers CD14 (left) and CD33 (right) in MV4-11 cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**I**) Flow cytometry analysis of populations with myeloid differentiation markers CD11b (left) and CD33 (right) in THP-1 cells 6 days after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**J**) Western blot of sorted human CD45-positive leukemia cells from moribund mice (n = 3) receiving cells transduced with control or MEF2D sgRNA.




**Supplemental Figure 4. Transcriptome signatures of MEF2D-deficient AML cells.** (**A-C**) GSEA analysis showing downregulation of genes associated with development processes including gliogenesis (A), nervous system development (B), and skeletal muscle development (C) in MOLM-13 cells after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**D-F**) GSEA analysis showing upregulation of genes associated with myeloid processes such as neutrophil degranulation (D), myeloid leukocyte activation (E), and neutrophil migration (F) in MOLM-13 cells after sgRNA-mediated knockout of MEF2D, relative to transduction of the sgLuc control sgRNA. (**G**) Motifs enriched in reduced ATAC-seq peaks against 769 human transcription factors. Top motifs were highlighted and labeled. (**H**) Top *de novo* motifs identified from reduced ATAC-seq peaks in MOLM-13 cells post knockout of MEF2D.




**Supplemental Figure 5. CEBPE depletion can partially rescue phenotypes of MEF2D loss.** (**A**) Western blot analysis demonstrating efficient deletion of CEBPE in MV4-11 cells. (**B**) Competitive proliferation assay demonstrating CEBPE knockout partially rescues MEF2D loss-induced proliferation defect in MV4-11 cells. MEF2D knockout was performed in CEBPE wildtype (WT) and knockout (KO) lines by using sgMEF2D e5.1 linked with an mCherry reporter. The mCherry positive percentage was monitored every 3 days post infection and normalized to the day 3 measurement. (**C**) Western blot analysis demonstrating efficient deletion of CEBPE in THP-1 cells. (**D**) Competitive proliferation assay demonstrating CEBPE knockout partially rescues MEF2D loss-induced proliferation defect in THP-1 cells. MEF2D knockout was performed in CEBPE wildtype (WT) and knockout (KO) lines by using sgMEF2D e5.1 linked with an mCherry reporter. The mCherry positive percentage was monitored every 3 days post infection and normalized to the day 3 measurement.


A
MV4-11
SGC0946
–
+
MEF2D
CEBPE
Vinculin
B
THP-1
SGC0946
–
+
MEF2D
CEBPE
Vinculin
C
25
MLL-AF9
MLL-AF9 binding
2.5
H3K27ac
Hoxa1
Hoxa3
Hoxa9
Hoxa11
Hoxa13
Evx1os
D
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2.5
H3K27ac
Meis1
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E
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F
MEF2D
Gene array value
200
150
100
50
0
***
***
Control
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HOXA9
MEF2D
G
Mef2d
FPKM
200
150
100
50
0
0
8
16
24
48
72
Hours
H
EV
Hoxa9
HA-Hoxa9
Vinculin
I
HOXA9Low
HOXA9High
MEF2D RSEM (log2)
14
13
12
11
10
9
-10
-5
0
5
10
15
HOXA9 RSEM (log2)
R = 0.24
P = 2.3×10-3
J
HOXA9Low
HOXA9High
CEBPE RSEM (log2)
15
10
5
0
-10
-5
0
5
10
15
HOXA9 RSEM (log2)
R = -0.28
P = 3.0 ×10-4

**Supplemental Figure 6. MEF2D is transcriptionally regulated by DOT1L and HOXA9 in MLL-r AML.** (**A**) Western blot analysis of MEF2D and CEBPE protein levels in MV4-11 cells post 4-day treatment of 1 μM SGC-0946 or DMSO. (**B**) Western blot analysis of MEF2D and CEBPE protein levels in THP-1 cells post 6-day treatment of 1 μM SGC-0946 or DMSO. (**C-E**) MLL-AF9 ChIP-seq profiles at HOX-A, MEIS1, MEF2D gene loci. (**F**) Microarray signal values of HOXA9 and MEF2D in MOLM-14 MLL-r AML cells harboring control or HOXA9 shRNA (GEO: GSE13714). (**G**) RNA-seq FPKM values of Mef2d in HoxA9-ER leukemia cells at indicated time points following Hoxa9 inactivation by tamoxifen withdrawal (EBI: E-MTAB-7107). (**H**) Western blot analysis showing expression of HA-Hoxa9 in MOLM-13 AML cells. (**I-J**) Gene expression correlation analyses between HOXA9 and MEF2D (I) or CEBPE (J) in TCGA AML patient samples. Samples with low (n = 41, left) and high (n = 124, right) HOXA9 expression were categorized for group analysis in Figure 7F.

**Supplemental References**


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