

Supporting Information

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SI Materials and Methods

Leukemic and Normal Control Samples. All of the acute myeloid leukemia (AML) patient samples were obtained at the time of diagnosis or relapse and with informed consent at the University of Chicago Hospital (UCH) or other collaborative hospitals, and were approved by the institutional review board of the institutes/hospitals. All patients were treated according to the protocols of the corresponding institutes/hospitals. The samples were stored in liquid nitrogen until used. Blasts and mononuclear cells (MNCs) were purified by use of Nycoprep 1.077A (Axis-Shield) according to the manufacturer's manual. All cell lines were maintained in the laboratory.

Exiqon microRNA Array Assay. As described previously (1, 2), our microRNA (miRNA) expression profiling assay of 85 [10 mixed lineage leukemia (*MLL*)-rearranged and 75 non-*MLL*-rearranged] AML samples and 15 human normal bone marrow (BM) samples was performed by Exiqon using the miRCURY LNA arrays (v10.0; covering 757 human miRNAs). Total RNA (0.5 μ g) was used for each sample. The global Lowess (LOcally WEighted Scatterplot Smoothing) regression algorithm (3) was used for data normalization. When calling a particular miRNA failed on an array, its expression value was indicated by the acronym "NA." The criteria for deciding that a miRNA had failed on a particular array, was that three or more of the four replicated measures of this miRNA were flagged (i.e., the signal is below background) by the image analysis software. The expression values are log₂ (Hy3/Hy5) ratios, which were obtained on the basis of the normalized data where replicated measurements on the same slide have been averaged. Median-centering genes for each array and median-centering each gene across all arrays were conducted for heatmap illustration.

Agilent Custom-Design Gene Arrays of Human Samples. Gene expression profiling of 79 human samples (9 *MLL*-rearranged AML, 61 non-*MLL*-rearranged AML, and 9 normal controls) were analyzed using Agilent's custom-design microarrays (Agilent Technologies) as described previously (1, 2). RNA quality control, cRNA amplification, hybridization, and image scans were conducted in the Functional Genomics Facility of the University of Chicago. Total RNA (0.5 μ g) per sample was used. Partek Genomics Suite (Partek Inc.) was used for the data normalization. Briefly, background adjustment, quantile normalization, and log-transformation were used to normalize and treat gene expression intensities, and then median-centering genes for each array and median-centering each gene across all arrays were conducted for heatmap illustration. Pearson correlation was used to assess expression correlation between miR-9 and predicted target genes by use of the Partek Genomics Suite. The complete microarray data set has been submitted to the Gene Expression Omnibus (GEO) database (www.ncbi.nlm.nih.gov/geo/) and the accession no. is GSE30258.

Affymetrix Exon Arrays of Human Samples. As described previously (1), a total of 13 *MLL*-rearranged AML samples and 9 human normal BM samples (including 3 each of CD34⁺ hematopoietic stem/progenitor, CD33⁺ myeloid, and MNC samples) were analyzed by use of Affymetrix GeneChip Human Exon 1.0 ST arrays (Affymetrix). The QC test and array assays were done in the core facility of National Human Genome Research Institute, National Institutes of Health. One microgram total RNA was used for each sample. After hybridization and background

correction according to the standard protocols, the quantified signals were then normalized using robust multiarray average (RMA), which is a robust algorithm of background adjustment, quantile normalization, and log-transformation (4). Partek Genomics Suite (Partek Inc.) was used for the analysis of the normalized data. The complete microarray data set has been submitted to the GEO database and the accession number is GSE34184.

Affymetrix Gene Arrays of Mouse Samples. As described previously (1, 2), a total of 15 mouse BM samples including 6 primary (including 3 each of negative control and *MLL-AF9*) and 9 secondary (including 3 negative control and 6 *MLL-AF9*) obtained from the in vivo mouse BM reconstitution assays were analyzed by use of Affymetrix GeneChip Mouse Gene 1.0 ST Array (Affymetrix). The RNA quality control, cDNA amplification, hybridization, and image scan were conducted in the Functional Genomics Facility of University of Chicago. After hybridization and background correction according to the standard protocols, the quantified signals were then normalized using RMA, which is a robust algorithm of background adjustment, quantile normalization, and log-transformation (4). Then median-centering genes for each array and median-centering each gene across all arrays were also conducted for heatmap illustration. The complete microarray data set has been submitted to the GEO database and the accession number is GSE34185.

Target Gene Analysis. Four major miRNA target prediction programs/databases including TargetScan (www.targetscan.org) (5), PITA (<http://genie.weizmann.ac.il/pubs/mir07/>) (6), miRanda (www.microrna.org) (7), and miRBase Targets (<http://microrna.sanger.ac.uk>) (8) were used for the identification of putative target genes of miR-9.

RNA Extraction and quantitative RT-PCR. Total RNA was extracted with the miRNeasy extraction kit (Qiagen) and was used as template to synthesize cDNA for quantitative RT-PCR (qRT-PCR) analysis in a ViiA7 real-time PCR system (Applied Biosystems). TaqMan qRT-PCR assay was performed to validate the expression pattern of miR-9 using kits from Applied Biosystems. qRT-PCR with SYBR Green dye (Qiagen) was used to determine expression of mRNA genes. *GAPDH* and *snoRNA202* or *RNU6B* was used as an endogenous control for qRT-PCR of miRNA and mRNA, respectively. Each sample was run in triplicate. Primers for qPCR of the two target genes are as follows:

<i>RHOH</i>	Forward	5'-GGA AGC CAG GAA AGC TTG GTG TTT-3'
	Reverse	5'-TCC AGT GGC CAA GCC TCA TCA ATA-3'
<i>RYBP</i>	Forward	5'-AAA TGC AGC ATC TGC GAT GTG AGG-3'
	Reverse	5'-TAC TGT TGT GCC ACT TGT TGT GCC-3'
<i>Rhoh</i>	Forward	5'-ACT CTG TGG CCA ACC ATA ACT CGT-3'
	Reverse	5'-TCT CAC ATC CTG GGC AAG TCT CTT-3'
<i>Rybp</i>	Forward	5'-TTA GGA ACA CGC CCG AAG CCT TTA-3'
	Reverse	5'-ACC AGC TGA GAA TTG ATG CGA GGT-3'

Plasmid Construction. The miR-9 fragment was amplified by PCR using the following primers: forward, 5'-TACTCGAGGTGTGCG-TGTGTCTGTCCAT-3'; reverse, 5'-ATGAATTCGCAAGTGTCCAGAGAGAG-3'. This fragment was subsequently cloned into the XhoI and EcoRI sites of the retrovirus vector MSCVpig (i.e., MSCV-Puro-IRES-GFP vector; bearing the *GFP* gene), which was a kind gift from Gregory Hannon (Watson School of Biological Sciences, Cold Spring Harbor, NY), Scott Hammond (University of North Carolina, Chapel Hill, NC), and Lin He (University of California, Berkeley, CA) (9, 10). The MSCVneo-*MLL-AP9* plasmid was provided by Scott Armstrong (Memorial Hospital, New York). All of the insertions were confirmed by DNA sequencing.

Cell Apoptosis and Viability Assay. Cell apoptosis and viability were assessed 48 h after transfection using the ApoLive-Glo Multiplex Assay Kit (Promega) following the manufacturer's manuals. For cell proliferation assays, 2×10^6 cells were electroporated with 100 μ M of hsa-miR-9 microRNA inhibitor or inhibitor control (miRCURY LNA; Exiqon) or 2 μ g of plasmid. Twenty-four hours after transfection, cells were seeded in 96-well plates at a concentration of 10,000 cells per well. Cell numbers were counted for the indicated number of days. Analysis of cell viability was also performed using the 3-(4,5-dimethylthiazol-2-yl)-5-(3-carboxymethoxyphenyl)-2-(4-sulfophenyl)-2H-tetrazolium, inner salt (MTS) assay (Promega) according to the manufacturer's manuals. Cell apoptosis was confirmed by staining with the APC-Annexin V+ Propidium Iodide (PI) Apoptosis Detection Kit (BD Biosciences) by flow cytometry according to the manufacturer's recommendations.

Cell Culture and Transfection. MONOMAC-6 and THP-1 cells were grown in RPMI-1640 medium and transfected using the Amaxa Nucleofector Technology (Amaxa Biosystems), according to the manufacturer's manuals. MONOMAC-6 cells were maintained in RPMI-1640 supplemented with 2 mM of L-glutamine, nonessential amino acid, 1 mM of sodium pyruvate, 9 μ g/mL of human insulin, 1% (vol/vol) penicillin-streptomycin, and 10% (vol/vol) FBS. THP-1 cells were maintained in RPMI-1640 containing 0.05 mM of 2-mercaptoethanol, 1% (vol/vol) penicillin-streptomycin, and 10% (vol/vol) FBS (Invitrogen). The *MLL-ENL-ERtm* cell line was kept in RPMI-1640 supplemented with 10 ng/mL of IL-3, 10 ng/mL of IL-6, 10 ng/mL of GM-CSF, 100 ng/mL of stem cell factor (SCF; R&D Systems), 1% (vol/vol) penicillin-streptomycin, and 10% (vol/vol) FBS. 4-Hydroxy-tamoxifen (4-OHT; Sigma-Aldrich) was added at a 100 nM final concentration as a 1 mM stock solution in ethanol. HeLa, HEK293T, and Rat1a cells were cultured in DMEM supplemented with 10% (vol/vol) FBS, 1% (vol/vol) Hepes, and 1% (vol/vol) penicillin-streptomycin (Invitrogen). For transfection, HEK293T cells were plated in 60-mm dishes at a concentration of 4×10^5 per dish and transfected with Effectene Transfection Reagent (Qiagen).

ChIP. ChIP assay was performed with the ChampionChIPTM One-Day kit (SABiosciences Corporation, Qiagen) following the manufacturer's protocol with some modifications. Cells (10×10^6) were cross-linked in 1% (wt/vol) formaldehyde at 37 °C for 10 min. The reaction was terminated by the addition of stop buffer and incubated at room temperature for 5 min. After cell lysis, the cross-linked chromatin was sonicated to an average size of ~500 bp and

was immunoprecipitated with antibodies against the amino-terminal portion of MLL (Abcam), H3K79, or IgG (Abcam). The purified ChIP DNA was amplified by real-time qPCR using specific primers targeting the human miR-9 CpG island. The primers used for the ChIP-qPCR are as follows: miR-9-1, forward 5'-TTCGCTTCCAACGCCTTTCTGAGT-3' and reverse 5'-TCTCCACTGCCCTTCTCTGGAAGAT-3'; miR-9-2, forward 5'-GCAGGGAGATCAAGTGAGTATCGT-3' and reverse 5'-ACAGGTTTAACATCCGAGGCACAC-3'; miR-9-3, forward 5'-AGATCTACTGCAAGTGCTGGGCAT-3' and reverse 5'-TGGGAGACAAGAATGCTCGAGTGA-3'.

Colony-Forming/Replating Assay. Retrovirus vectors were cotransfected with pCL-Eco packaging vector (IMGENEX) into 293T cells using Effectene Transfection Reagent (Qiagen) to produce the retroviruses. BM cells were harvested from a cohort of 4- to 6-wk-old C57B6/L (CD45.2) donor mice after 5 d of 5-fluorouracil (5-FU) treatment, and primitive hematopoietic progenitor cells were enriched with the Mouse Lineage Cell Depletion Kit (Miltenyi Biotec). An aliquot of enriched hematopoietic progenitor cells was added to retroviral supernatant together with polybrene in a conical tube, which were centrifuged at $2,000 \times g$ for 2 h at 32 °C, and then the media were replaced with fresh media and incubated for 20 h at 37 °C. The next day, the same procedure was repeated once. Then, on the day following the second spinoculation, an equivalent of 2×10^4 cells was plated onto a 35-mm Petri dish in 1.5 mL of Methocult M3230 methylcellulose medium (Stem Cell Technologies) containing 10 ng/mL each of murine recombinant IL-3, IL-6, and GM-CSF and 30 ng/mL of murine recombinant SCF, along with 1.0 mg/mL of G418 and/or 2 μ g/mL of puromycin. For each transduction, there were two duplicate dishes. Cultures were incubated at 37 °C in a humidified atmosphere of 5% CO₂. The colonies were replated every 7 d under the same conditions.

Flow Cytometry. Cells from BM, spleen (SP), and peripheral blood (PB) were harvested for analysis of immunophenotypes. After washing with PBS, blocking unspecific binding with affinity purified anti-mouse CD16/32 (eBioscience), cells were stained in 4 °C with various antibodies diluted in flow cytometry staining buffer (eBioscience) for 30 min. Subsequently, cells were washed with PBS and resuspended in intracellular (IC) fixation buffer (eBioscience) for flow cytometry analysis. Antibodies (eBioscience, San Diego) were used for the flow cytometry analysis, including anti-mouse CD11b APC (17-0112), anti-mouse CD117 (c-Kit) APC (17-1171), anti-mouse Ly-6G (Gr-1) eFluor 450 (48-5931), and anti-mouse CD11b eFluor 450 (48-0112).

Statistical Software. The miRNA and gene/exon array data analyses were conducted using Partek Genomics Suite (Partek Inc.), TIGR Multiple Array Viewer software package (TMeV version 4.6; TIGR), and/or Bioconductor R packages. The miRNA-gene expression correlation was analyzed using Partek Genomics Suite (Partek Inc.). The heatmaps were constructed using the TIGR Multiple Array Viewer software package. The *t* test, Kaplan-Meier method, and log-rank test were performed with WinSTAT (R. Fitch Software) and/or GraphPadPrism (GraphPad Software).

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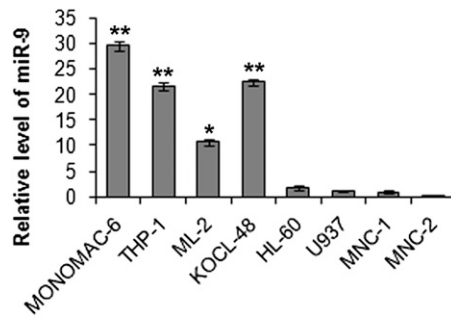


Fig. S1. miR-9 is significantly up-regulated in human *MLL*-rearranged leukemia cell lines. Relative expression levels of miR-9 in four human *MLL*-rearranged cell lines (i.e., MONOMAC-6, THP-1, ML-2, and KOCL-48), two non-*MLL*-rearranged cell lines (i.e., HL-60 and U937), and two human normal BM MNC (MNC-1 and -2) samples were detected by qRT-PCR analysis. RNU6B was used as endogenous control. * $P < 0.05$; ** $P < 0.01$, two-tailed t test.

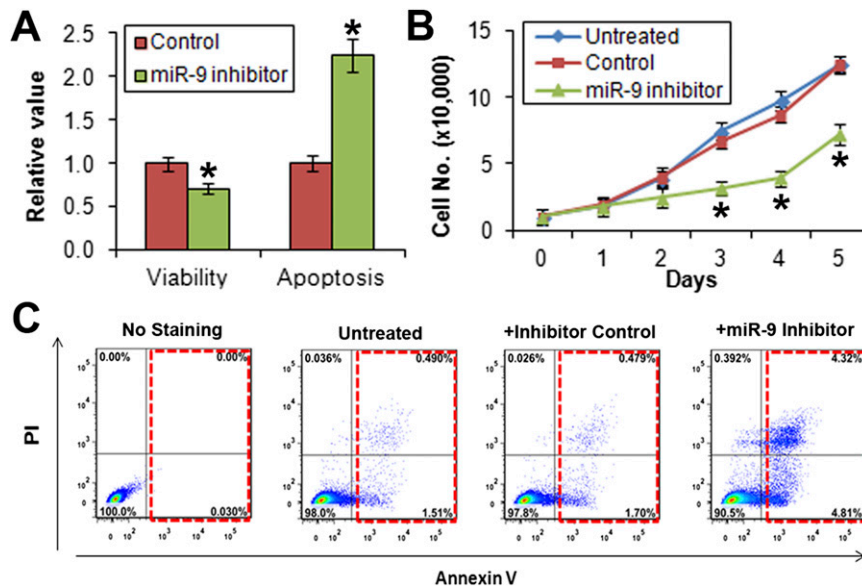


Fig. S2. Blocking expression of miR-9 decreases cell viability, promotes cell apoptosis, and inhibits cell growth/proliferation of THP-1 cells. Cells were transfected with miR-9 inhibitor or inhibitor control (as control). (A) Cell viability and apoptosis were assessed 48 h after transfection by the ApoLive-Glo Multiplex Assay Kit (Promega). (B) Cell numbers were counted every day after transfection for 6 d. (C) Representative flow cytometry analysis of cell apoptosis by APC-Annexin V (a mark of apoptotic cells) + PI staining.

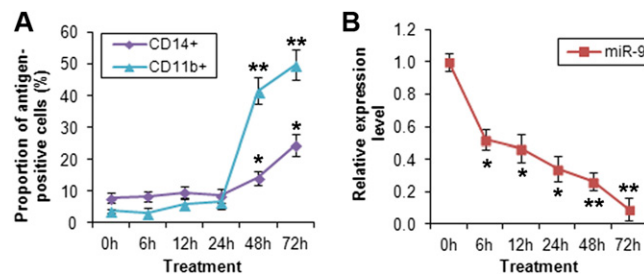


Fig. S3. Expression of miR-9 is significantly down-regulated during differentiation of THP-1 cells induced by phorbol-12-myristate-13-acetate (PMA). THP-1 cells were treated with 100 nM PMA. (A) The proportion of CD14⁺ or CD11b⁺ (two markers of differentiated myeloid cells/macrophages) cells were detected by flow cytometry at different time points after the treatment. (B) Relative expression of miR-9 was detected by qPCR. * $P < 0.05$; ** $P < 0.01$; two-tailed t test, compared with the values at 0 h (i.e., when cells were untreated).

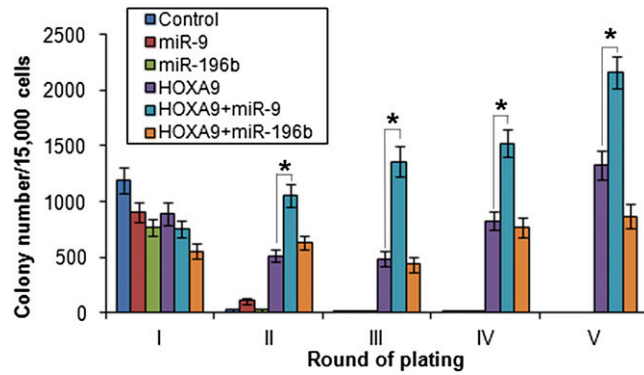


Fig. S4. Forced expression of miR-9 significantly promotes HOXA9-mediated cell transformation as detected by in vitro colony-forming/replating assays. Normal BM progenitor cells from CD45.2 mice were retrovirally transfected with MSCVneo+MSCVpig (i.e., Control), MSCVneo+MSCVpig-miR-9 (i.e., miR-9), MSCVneo+MSCVpig-miR-196b (i.e., miR-196b), MSCVneo-HOXA9 (containing only CDS coding region)+MSCVpig (i.e., HOXA9), MSCVneo-HOXA9+MSCVpig-miR-9 (i.e., HOXA9+miR-9), or MSCVneo-HOXA9+MSCVpig-miR-196b (i.e., HOXA9+miR-196b). The cells were then plated into methylcellulose medium under double selection of puromycin and G418. The colonies were replated every 7 d for a total of five passages, and the colony numbers were counted and compared. Mean \pm SD values of colony numbers are shown. * $P < 0.05$; two-tailed t test.

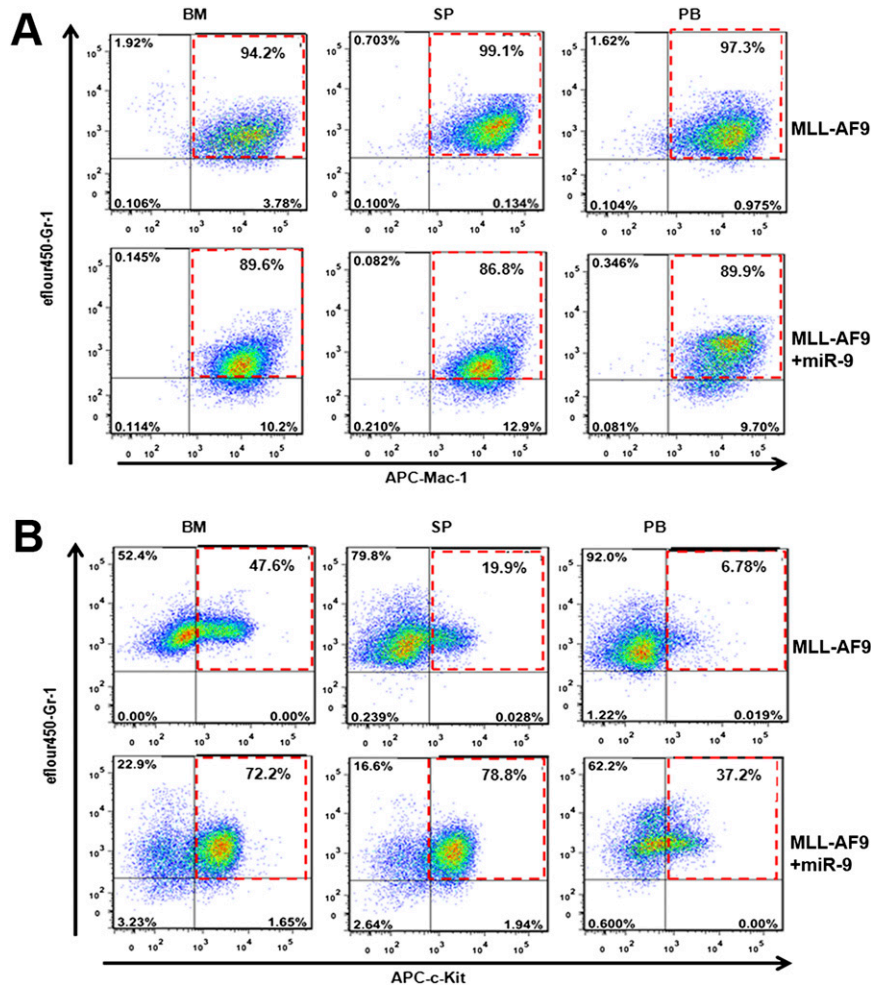


Fig. S5. Representative of flow cytometric analysis of BM, SP, and PB from *MLL-AF9* and *MLL-AF9*+miR-9 representative mice. (A) Cells stained for Mac-1/Gr-1, showing the development of AML in recipient mice. (B) Cells stained for c-Kit/Gr-1, showing increased percentage of c-Kit/Gr-1 double-positive cells in *MLL-AF9*+miR-9 mice compared with *MLL-AF9* mice.

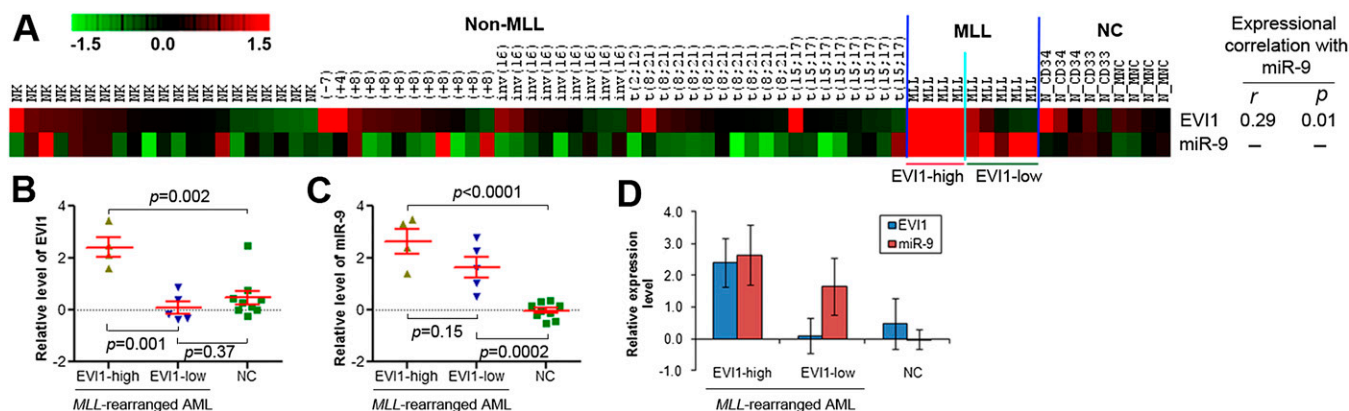


Fig. S6. Expression of miR-9 is not repressed by EVI1 in *MLL*-rearranged AML. (A) Expression profiles of *EVI1* and miR-9 across 79 human samples including 9 *MLL*-rearranged AML (i.e., *MLL*), 61 non-*MLL*-rearranged AML (i.e., non-*MLL*), and 9 normal control (i.e., NC) samples. Based on the expression level of *EVI1*, 9 *MLL*-rearranged AML samples were classified into EVI1-high ($n = 4$; the expression level of *EVI1* in these four samples is higher than the average level of *EVI1* expression in the entire set of nine samples) or EVI1-low ($n = 5$) groups. The correlation coefficient (r) and P value of the correlation between miR-9 and *EVI1* are shown. Expression data are mean centered, and the relative value for each sample is represented by a color; high expression is represented with red and low expression is represented with green (scale shown in the upper left). (B and C) Scott plots show the relative expression levels of *EVI1* (B) or miR-9 (C) in EVI1-high *MLL*-rearranged AML, EVI1-low *MLL*-rearranged AML, and normal control samples. The P value of comparison between each two groups is shown. (D) Mean \pm SD values of relative expression levels of *EVI1* and miR-9 in the above three groups are shown.

Table S1. Chromosomal translocations of the 85 AML patients

Characteristic	<i>MLL</i> -rearranged ($n = 10$)	Non- <i>MLL</i> -rearranged ($n = 75$)
t(11q23)	t(9;11)(p22;q23)/ <i>MLL-AF9</i> ($n = 5$; 50%) t(11;19)(q23;p13.3)/ <i>MLL-ENL</i> ($n = 2$; 20%) t(11;19)(q23;p13.1)/ <i>MLL-ELL</i> ($n = 2$; 20%) t(4;11)(q21 q23)/ <i>MLL-AF4</i> ($n = 1$; 10%)	0
t(15;17)	0	9 (12%)
t(8;21)	0	10 (13%)
inv(16)	0	9 (12%)
t(2;12)	0	2 (3%)
t(1;16)	0	1 (1%)
+8	0	12 (16%)
+4	0	1 (1%)
-7	0	2 (3%)
Normal karyotype (NK)	0	29 (39%)

Table S2. Comparison of c-Kit⁺ blast cell proportions in BM, SP, and PB cells between *MLL-AF9* mice and *MLL-AF9*+miR-9 mice

Cell population	<i>MLL-AF9</i> mice ($n = 3$) vs. <i>MLL-AF9</i> +miR-9 mice ($n = 3$)		
	BM	SP	PB
c-Kit ⁺ /Mac-1 ⁺	39.4 \pm 7.8% vs. 70.8 \pm 9.4%*	18.0 \pm 4.4% vs. 78.3 \pm 8.5%*	6.7 \pm 1.8% vs. 38.6 \pm 2.6% [†]
c-Kit ⁺ /Gr-1 ⁺	37.6 \pm 6.1% vs. 67.7 \pm 7.9%*	22.4 \pm 8.1% vs. 69.6 \pm 14.3%*	7.9 \pm 2.8% vs. 39.8 \pm 4.3% [†]

* $P < 0.05$, t test.

[†] $P < 0.01$, t test.

Table S3. Cont.

Potential target genes	Correlation with miR-9 expression in the set of 79 human samples (9 <i>MLL</i> -rearranged AML, 61 non- <i>MLL</i> -rearranged AML, and 9 normal controls)		Correlation with miR-9 expression in the set of 18 human samples (9 <i>MLL</i> -rearranged AML and 9 normal controls)		SAM analysis of the set of 22 human samples (13 <i>MLL</i> -rearranged AML samples vs. 9 normal controls)		SAM analysis of the set of 15 mouse samples (9 <i>MLL</i> -rearranged AML samples vs. 6 normal controls)	
	<i>r</i>	<i>P</i>	<i>r</i>	<i>P</i>	Fold	q-value	Fold	q-value
<i>NECAP1</i>	-0.28	0.011	-0.48	0.046				
<i>ZSWIM6</i>	-0.28	0.011	-0.57	0.013				
<i>ELF1</i>	-0.28	0.012	-0.56	0.017				
<i>CDYL</i>	-0.28	0.012	-0.48	0.042				
<i>MGEA5</i>	-0.28	0.013	-0.69	0.002				
<i>PIGH</i>	-0.27	0.017	-0.74	0.000				
<i>ITGB1</i>	-0.26	0.019	-0.52	0.026				
<i>SLC4A7</i>	-0.26	0.020	-0.62	0.006				
<i>RIMS3</i>	-0.26	0.021	-0.54	0.019				
<i>TNFRSF21</i>	-0.26	0.023	-0.66	0.003				
<i>TMEM64</i>	-0.25	0.024	-0.59	0.009				
<i>MTMR6</i>	-0.25	0.027	-0.57	0.014				
<i>YBX1</i>	-0.25	0.027	-0.47	0.047				
<i>NUP160</i>	-0.25	0.029	-0.57	0.014				
<i>PHF20L1</i>	-0.24	0.030	-0.54	0.021				
<i>AZ12</i>	-0.24	0.032	-0.53	0.023				
<i>CUGBP2</i>	-0.24	0.035	-0.48	0.046				
<i>CDKN1B</i>	-0.23	0.038	-0.71	0.001				
<i>NOPE</i>	-0.23	0.039	-0.64	0.005				
<i>SFRS1</i>	-0.23	0.039	-0.52	0.026				
<i>TMSB4X</i>	-0.23	0.040	-0.49	0.039				
<i>SLC7A1</i>	-0.23	0.041	-0.51	0.032				
<i>STK24</i>	-0.23	0.042	-0.59	0.009				
<i>SMNDC1</i>	-0.22	0.047	-0.59	0.009				
<i>LEMD3</i>	-0.22	0.049	-0.50	0.034				
<i>ACSL1</i>	-0.22	0.050	-0.61	0.008				
<i>FAM117A</i>	-0.39	0.000						
<i>CLDND1</i>	-0.39	0.000						
<i>SAPS3</i>	-0.37	0.001						
<i>CSDA</i>	-0.36	0.001						
<i>UBR5</i>	-0.35	0.002						
<i>6-Mar</i>	-0.34	0.002						
<i>LXN</i>	-0.34	0.002						
<i>NUP153</i>	-0.34	0.003						
<i>BCR</i>	-0.33	0.003						
<i>TMEM49</i>	-0.33	0.003						
<i>BCAT1</i>	-0.31	0.005						
<i>MAP2K3</i>	-0.31	0.005						
<i>NR2C2</i>	-0.31	0.006						
<i>AFF4</i>	-0.31	0.006						
<i>FAM13A1</i>	-0.30	0.007						
<i>USP9X</i>	-0.30	0.008						
<i>CLIC4</i>	-0.30	0.008						
<i>AK3L1</i>	-0.29	0.009						
<i>PTX3</i>	-0.29	0.010						
<i>DUSP2</i>	-0.29	0.010						
<i>F13A1</i>	-0.29	0.011						
<i>CDK8</i>	-0.29	0.011						
<i>FAM46C</i>	-0.28	0.011						
<i>BIN1</i>	-0.28	0.012						
<i>STOM</i>	-0.28	0.012						
<i>MREG</i>	-0.28	0.012						
<i>GRB10</i>	-0.28	0.013						
<i>TFDP2</i>	-0.28	0.013						

