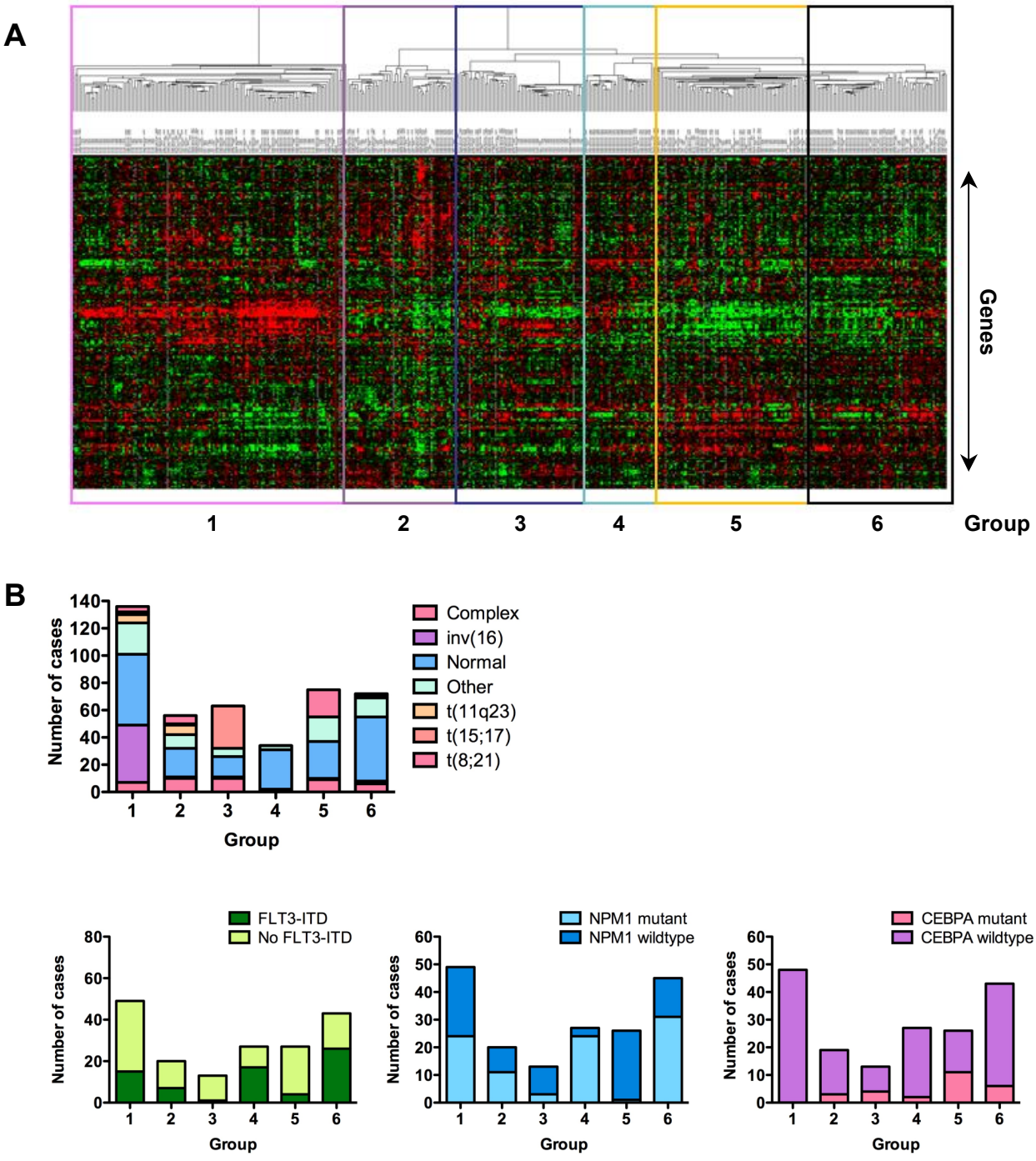
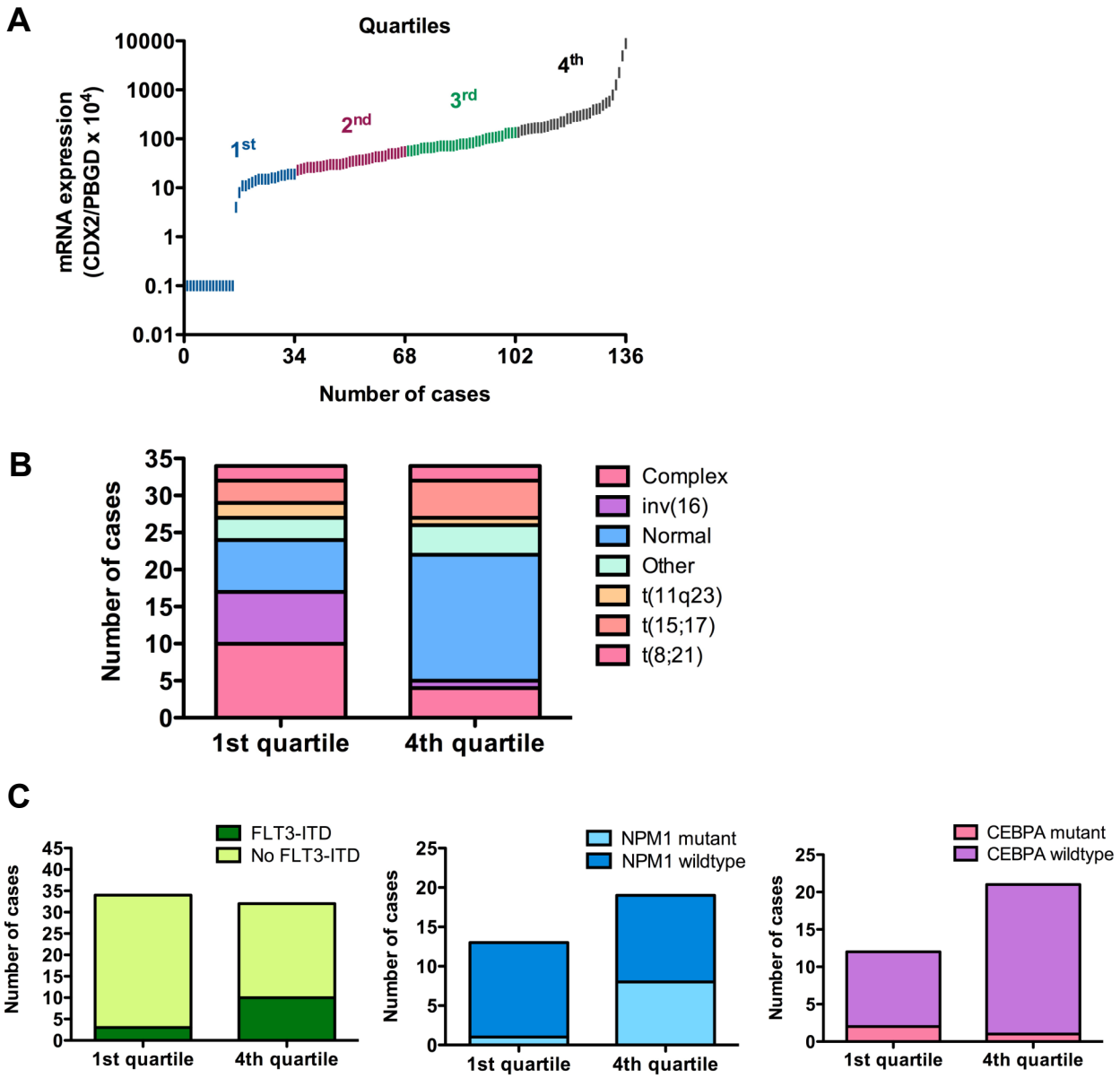
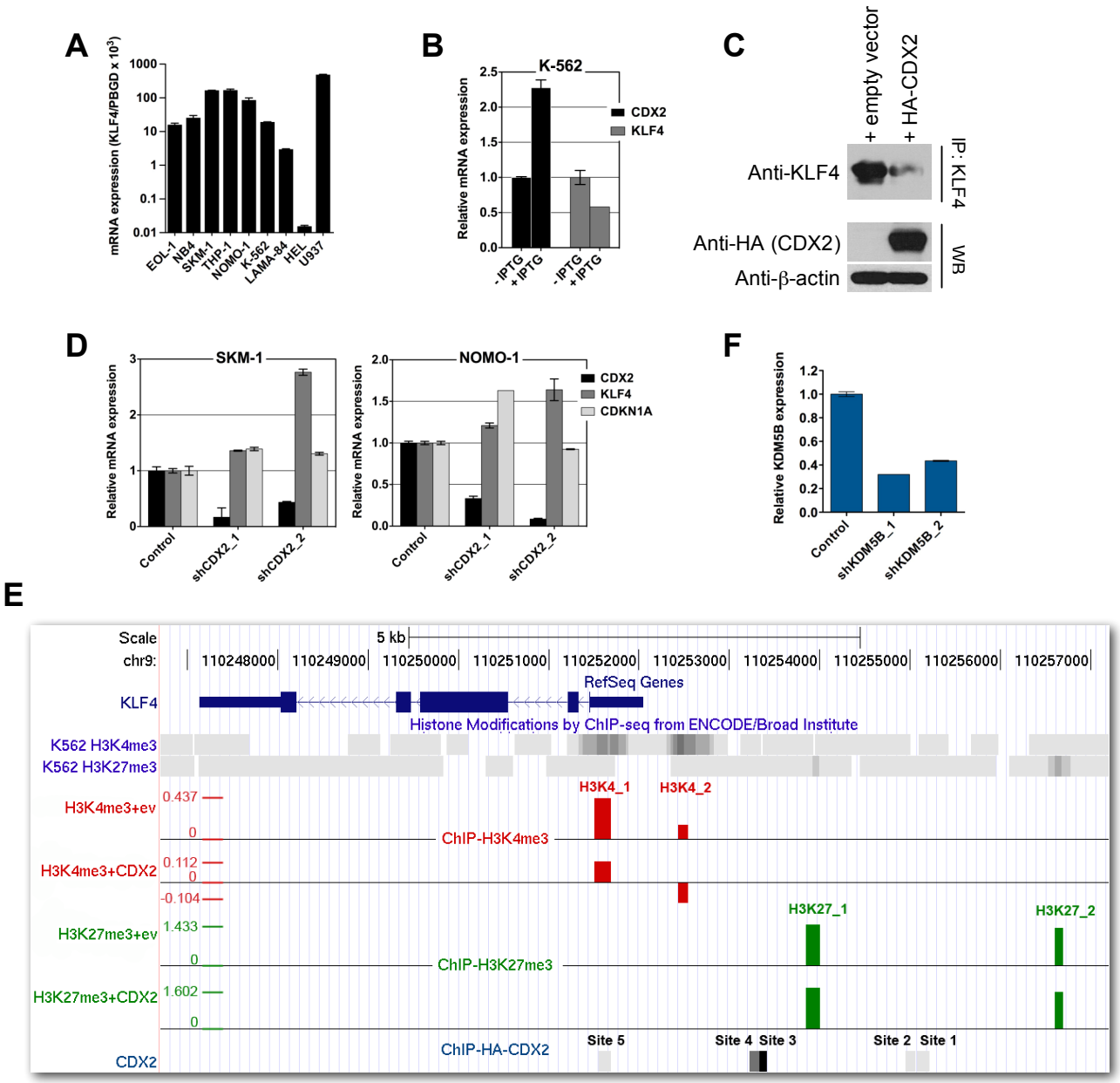


Supplemental Figure 1
Categorization of 436 primary AML patient samples based on the top 200 differentially expressed genes from the murine-derived HSPC Cdx2 signature. **(A)** Identification of 6 patient groups using hierarchical clustering. **(B)** Differential distribution of karyotypic abnormalities ($P < 0.0001$) and mutations in the *FLT3* ($P < 0.0001$), *NPM1* ($P < 0.0001$), and *CEBPA* ($P < 0.0001$) genes among the 6 patient clusters. *P* values were calculated using the chi-square test. ITD, internal tandem duplication.



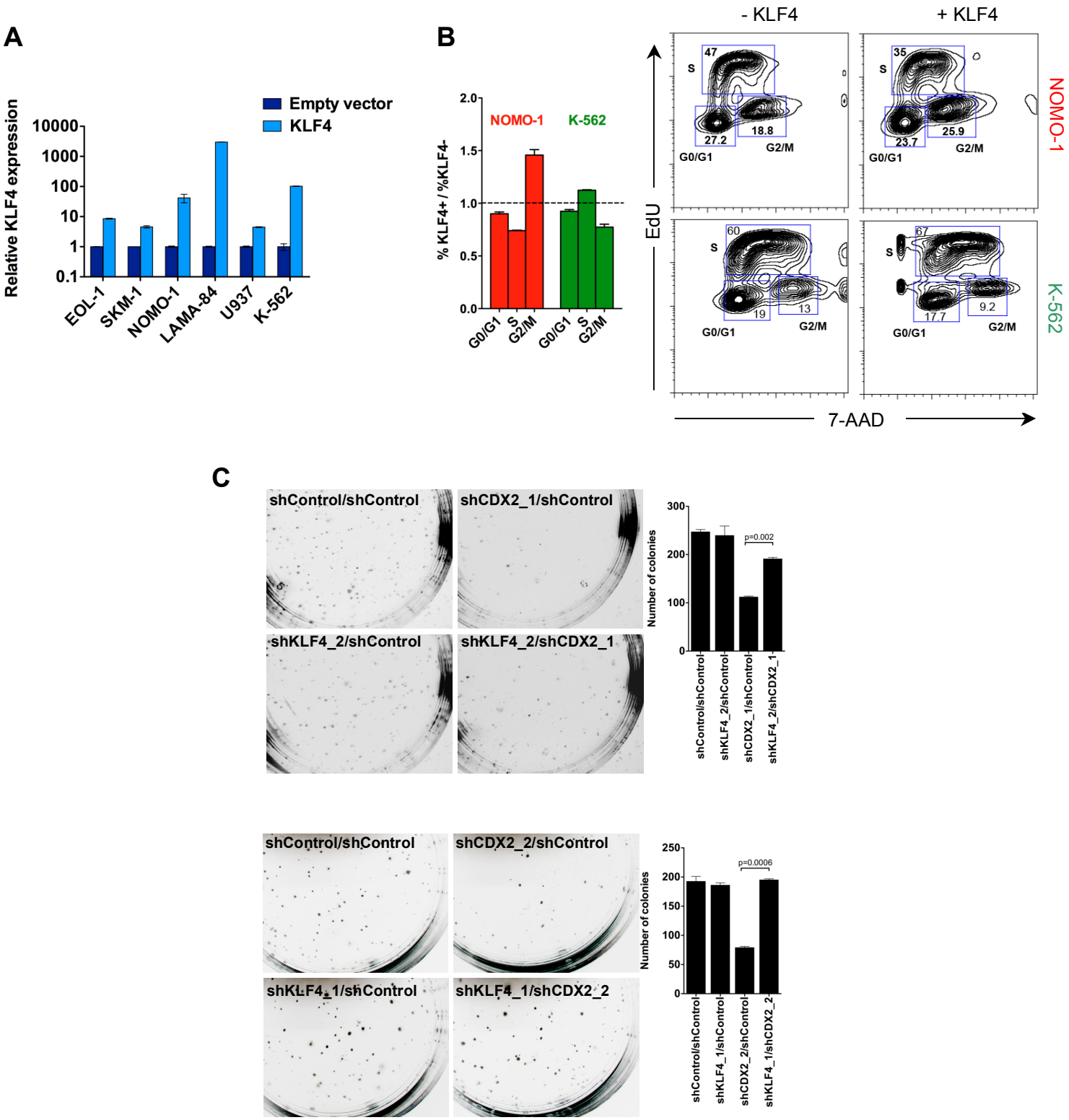
Supplemental Figure 2

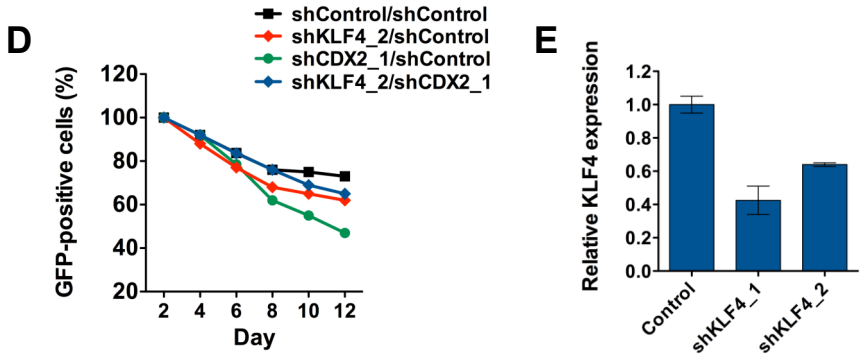
Categorization of 136 primary AML patient samples based on *CDX2* mRNA expression and structural genetic abnormalities. **(A)** *CDX2* mRNA levels as determined by qRT-PCR. Cases were grouped into quartiles according to *CDX2* mRNA expression. Negative qRT-PCR results are indicated by an expression value of 0.1. **(B)** Distribution of karyotypic abnormalities among patients in the first and fourth quartile of *CDX2* mRNA expression. **(C)** Distribution of mutations in the *FLT3*, *NPM1*, and *CEBPA* genes among patients in the first and fourth quartile of *CDX2* mRNA expression. Due to the association of *FLT3*, *NPM1*, and *CEBPA* mutations with specific cytogenetic categories, such as normal karyotype, molecular genetic data were not always available for all cases in the first and fourth quartile of *CDX2* mRNA expression. ITD, internal tandem duplication.



Supplemental Figure 3

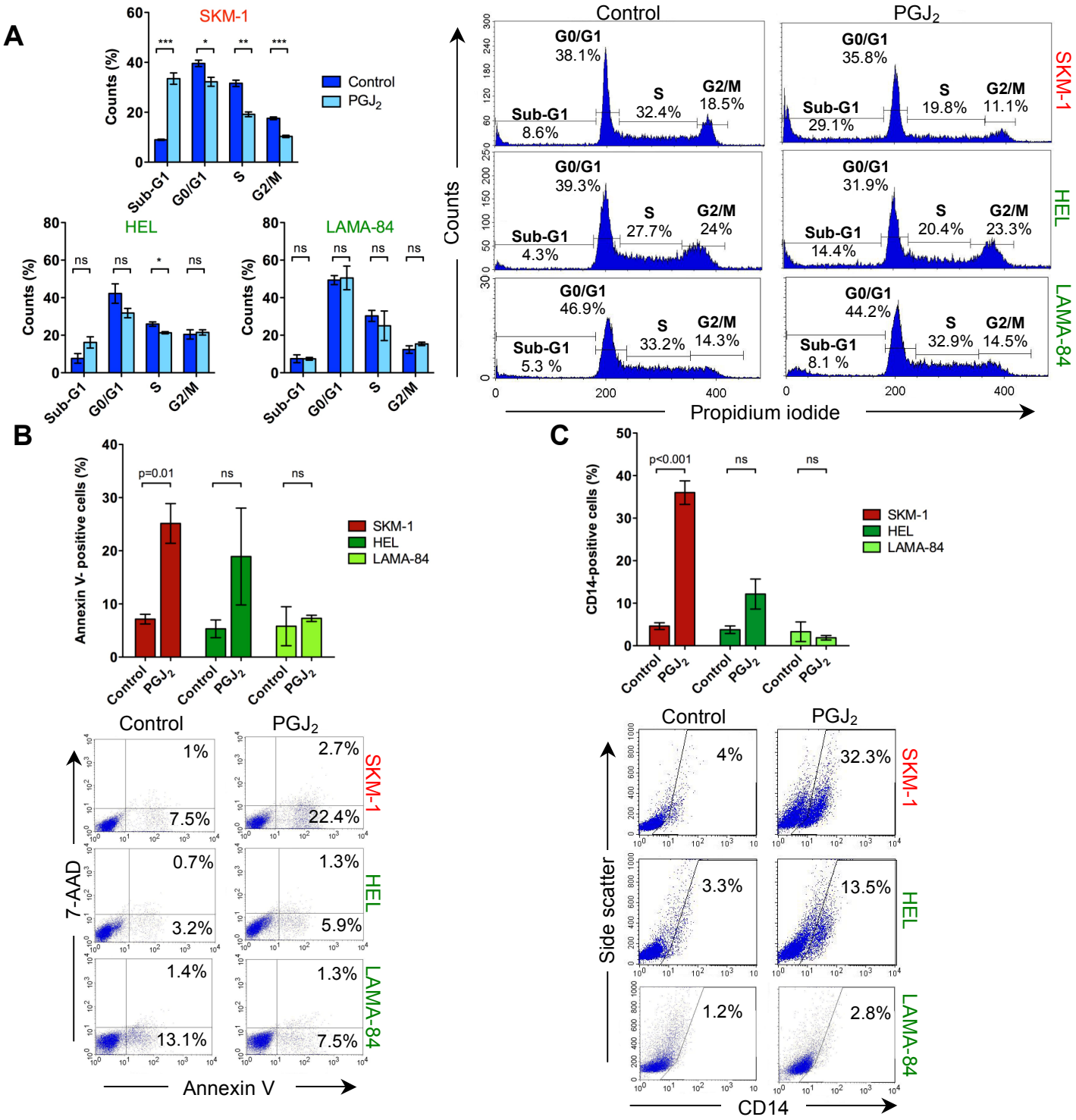
Relationship between CDX2 and KLF4 expression and analysis of the *KLF4* regulatory region in human myeloid leukemia cell lines. **(A)** *KLF4* mRNA expression of myeloid leukemia cell lines. **(B)** Decreased *KLF4* mRNA levels in response to expression of CDX2 using a lentiviral vector (IPTG-inducible pLKO.1puro). **(C)** Decreased KLF4 protein levels in response to expression of CDX2 in K-562 cells. WB, western blot. **(D)** Increased *KLF4* and *CDKN1A* mRNA levels in response to shRNA knockdown of CDX2. **(E)** H3K4me3 and H3K27me3 marks in the *KLF4* upstream regulatory region detected in K-562 cells in the ENCODE Histone Modification ChIP-seq project by the Broad Institute and visualized with the UCSC genome browser (www.genome.ucsc.edu; February 2009 (GRCh37/hg19) Assembly). ChIP results shown in Figure 2, E and G were uploaded into the UCSC genome browser to indicate the location of the histone modifications and CDX2 binding sites studied. **(F)** *KDM5B* mRNA knockdown of K-562 cells stably expressing HA-CDX2 used for the experiment shown in Figure 2I.


A
Relative KLF4 expression
Empty vector
KLF4
10000
1000
100
10
1
0.1
EOL-1
SKM-1
NOMO-1
LAMA-84
U937
K-562
B
% KLF4+ / %KLF4-
NOMO-1
K-562
2.0
1.5
1.0
0.5
0.0
G0/G1
S
G2/M
- KLF4
+ KLF4
EdU
7-AAD
NOMO-1
K-562
47
27.2
18.8
35
23.7
25.9
60
19
13
67
17.7
9.2
C
shControl/shControl
shCDX2_1/shControl
shKLF4_2/shControl
shKLF4_2/shCDX2_1
Number of colonies
p=0.002
shControl/shControl
shKLF4_2/shControl
shCDX2_1/shControl
shKLF4_2/shCDX2_1
shControl/shControl
shCDX2_2/shControl
shKLF4_1/shControl
shKLF4_1/shCDX2_2
Number of colonies
p=0.0006
shControl/shControl
shKLF4_1/shControl
shCDX2_2/shControl
shKLF4_1/shCDX2_2



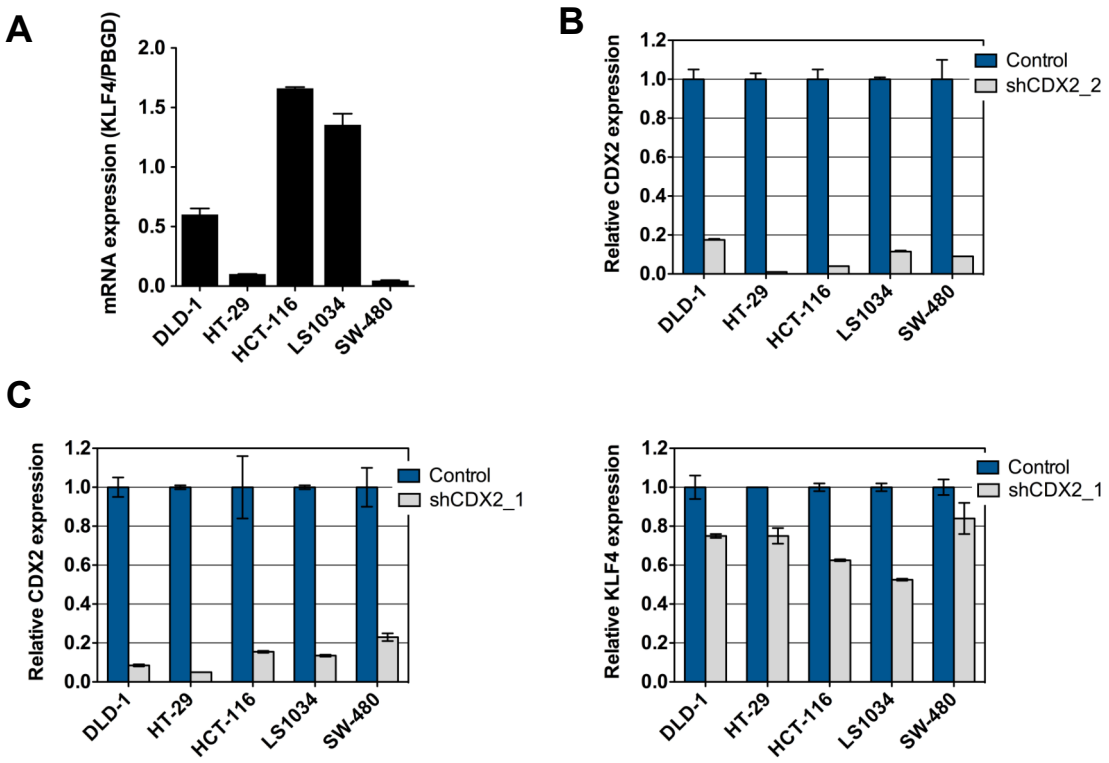
Supplemental Figure 4

Tumor-suppressive effect of KLF4 in human myeloid leukemia cells expressing CDX2. **(A)** *KLF4* mRNA expression relative to *PBGD* of the cell lines shown in Figure 3A. **(B)** G2/M cell cycle arrest in CDX2-expressing myeloid leukemia cell lines in response to KLF4. NOMO-1 and K-562 cells were transduced with a lentiviral vector coexpressing KLF4 and Tomato Red. Cell cycle was analyzed after 3 d by gating on Tomato Red-positive (+KLF4) or Tomato Red-negative (–KLF4) cells after staining with EdU and 7-AAD. KLF4 expression induced G2/M arrest in CDX2-positive NOMO-1 cells, but not in CDX2-negative K-562 cells. **(C)** Colony formation of AML cells in response to CDX2 and KLF4 knockdown. Same experiment as in Figure 3D but with different shRNAs. CDX2-expressing NOMO-1 cells were stably transduced with combinations of shRNAs and plated in methylcellulose. Knockdown of CDX2 (shCDX2/shControl) reduced the number of colonies. Colony formation was partially rescued by concomitant knockdown of KLF4 (shKLF4/shCDX2). Representative photomicrographs of methylcellulose cultures are shown. Original magnification, x 4.7. **(D)** Same experiment as in Figure 3E but with a different KLF4 shRNA. CDX2-expressing NOMO-1 cells stably transduced with an shRNA targeting KLF4 or a non-targeting control shRNA were transduced with a lentiviral vector coexpressing GFP with an shRNA targeting CDX2 or a non-targeting control shRNA. The GFP-positive fraction was measured by flow cytometry at the indicated time points. Knockdown of CDX2 alone (shCDX2/shControl) depleted GFP-positive cells over time. The proportion of GFP-positive cells was rescued by concomitant knockdown of KLF4 (shKLF4/shCDX2). **(E)** *KLF4* mRNA knockdown of NOMO-1 cells used for the experiments shown in Figure 3D and Supplemental Figure 4C.



Supplemental Figure 5

Effects of PPARγ agonist treatment in human myeloid leukemia cell lines. **(A)** PPARγ agonist treatment (PGJ_2, 5 μM) for 48 h increases the proportion of cells in the sub-G1 phase of the cell cycle in CDX2-positive SKM-1 cells to a greater extent than in CDX2-negative LAMA-84 and HEL cells. **P* < 0.05; ***P* < 0.01; ****P* < 0.001. ns, not significant. **(B)** PPARγ agonist treatment (PGJ_2, 5 μM) for 48 h increases the proportion of annexin V-positive cells in CDX2-positive SKM-1 cells, but not in CDX2-negative LAMA-84 and HEL cells. Numbers indicate percentages of cells. **(C)** PPARγ agonist treatment (PGJ_2, 5 μM) for 72 h induces monocytic differentiation in CDX2-positive SKM-1 cells to a greater extent than in CDX2-negative LAMA-84 and HEL cells.



Supplemental Figure 6

Effects of CDX2 in human CRC cell lines. **(A)** *KLF4* mRNA expression of CRC cell lines. **(B)** *CDX2* mRNA knockdown of the CRC cell lines shown in Figure 8B. **(C)** Same experiment as in Figure 8B and Supplemental Figure 7B but with a different CDX2 shRNA. *CDX2* (left) and *KLF4* (right) mRNA levels are shown.

Supplemental Methods

Gene expression profiling of mouse HSPC and C-Map analysis

Total RNA (1 μg) was amplified with the One-Cycle Target Labeling Kit (Affymetrix) and hybridized to GeneChip Mouse Genome 430A 2.0 microarrays according to the manufacturer's protocols (Affymetrix). Fluorescence ratios were normalized by applying the RMA algorithm using the BRB-ArrayTools software package. For subsequent analyses, only probe sets were included whose expression varied as previously determined (1), and supervised analyses (class comparison, pathway comparison) were performed using BRB-ArrayTools. The complete microarray dataset is available at NCBI's GEO under accession number GSEXXX. For the discovery of functional connections between transcriptional patterns and bioactive molecules and hypothesis generation, significantly deregulated genes were converted to human counterparts and uploaded into the current version (build 02) of the C-Map (www.broadinstitute.org/cmap). Perturbagens with negative enrichment scores were considered for further experimental evaluation.

Gene expression profiling of human AML samples and outlier analysis

Gene expression profiles of human AML samples were generated previously using the Stanford cDNA microarray platform, and data normalization and filtering was performed as reported elsewhere (2). The complete gene expression microarray dataset is available at NCBI's GEO under accession number GSE16432. For hierarchical clustering, average linkage clustering was used (distance measure, correlation uncentered), and results were visualized using TreeView (3). For outlier analysis, the BRB-ArrayTools software package was used. To compare experiments of distinct phenotypic classes, the average log-ratio within one class was plotted on the x axis (fourth quartile of *CDX2* mRNA expression) versus the average log-ratio within the other class on the y axis (first quartile of *CDX2* mRNA expression), and averages were taken on a gene-by-gene basis, i.e., each gene was represented by a single point in the resulting scatterplot. Genes that were differentially expressed between the 2 phenotypes were considered as outliers if they fell outside of the pair of outlier lines, which were specified to indicate genes with a greater than 2-fold difference between the geometric mean of the expression ratios within each of the 2 classes.

RNA isolation, cDNA synthesis, and qRT-PCR

Total RNA was isolated using the RNeasy Mini or Micro Kit (Qiagen) and reverse-transcribed (2 μg in a total volume of 30 μl) using TaqMan Reverse Transcription Reagents (Applied Biosystems). Real-time RT-PCR was used to quantify the expression of *CDX2*, *KLF4*, *CDKN1A*, *KDM5B*, *PPARG*, *PPARGC1A*, *Cdx2*, and *Klf4* relative to endogenous *PBGD* or *Gapdh*. Primers and/or probes were combined with TaqMan Universal PCR Master Mix or SYBR Green PCR Master Mix reagents (Applied Biosystems), and cDNA was used as template. Reactions were run on an ABI PRISM 7900HT Sequence Detection System at default thermal cycling conditions. Results were analyzed using the standard curve method.

Primer and probe sequences used for qRT-PCR		
Gene	**Primers/probe**	**Source**
CDX2	Hs01078080_m1	Applied Biosystems
KLF4	Hs01034973_g1	Applied Biosystems
KLF4	5'-cacctggcgagtctgacat-3' 5'-gtcgcttcatgtgggagag-3'	Custom made
CDKN1A	5'-gactctcagggtcgaaaacg-3' 5'-ggattagggcttcctcttgg-3'	Custom made
KDM5B	5'-caatgctgtggacctgtatgt-3' 5'-tacggagggtatagtccctgg-3'	Custom made
PPARG	Hs01115513_m1	Applied Biosystems
PPARG	5'-ttcagaaatgccttgcagtg-3' 5'-ccaacagcttctccttctcg-3'	Custom made
PPARGC1A	Hs01016719_m1	Applied Biosystems
PBGD	5'-ggagccatgtctggtaacggca-3' 5'-ggtacccacgcgaatcactctca-3' 5'-6-FAM-tgcggctgcaacggcggaagaaa-BHQ1-3'	Custom made
Cdx2	Mm01212280_m1	Applied Biosystems
Klf4	Mm00516104_m1	Applied Biosystems
Gapdh	4352932E	Applied Biosystems

ChIP assays

ChIP analysis was performed using K-562 and HT-29 cells stably transduced with N-terminally HA-tagged CDX2, alone or in combination with shRNAs targeting KDM5B, or an empty pLenti6.2/V5-DEST control vector. H3K4me3 and H3K27me3 marks in the *KLF4* regulatory region were derived from the Encyclopedia of DNA Elements (ENCODE) Histone Modification ChIP-seq dataset by the Broad Institute (www.genome.ucsc.edu/cgi-bin/hgGateway). Experiments were carried out according to the ChIP Assay Kit protocol (Upstate). Briefly, $1x10^7$ cells were incubated with 1% formaldehyde (Merck) to allow for protein-DNA crosslinking, and fixed cells were washed with PBS and lysed with ChIP lysis buffer (1% SDS, 10 mM EDTA, 50 mM Tris-HCl [pH 8.1]) containing protease inhibitor (Roche). DNA was sheared by sonication with Bioruptor (Diagenode), yielding genomic DNA fragments with an average size of 0.5 kb and 1 kb. Samples were diluted 1:10, and 1% was removed for input control. Each sample was precleared with Protein A or Protein G/Salmon Sperm DNA beads (Millipore), followed by IP with specific Ab or IgG for background determination and Protein A or Protein G/Salmon Sperm DNA beads. Beads were washed, and DNA-protein complexes were reverse-crosslinked, eluted, and digested with RNase A and proteinase K. DNA was purified from samples using the illustra GFX PCR DNA and Gel Band Purification Kit (GE Healthcare) and quantified by PCR using SYBR Green PCR Master Mix reagents (Applied Biosystems). Expression values were normalized to input chromatin and reported as % input after background subtraction. IP was performed using the following Ab: HA-Tag (Cell Signaling), H3K4me3 (Abcam), H3K27me3 (Millipore), and normal mouse or rabbit IgG (Santa Cruz).

Primer sequences used for ChIP analysis		
Gene	**Forward primer**	**Reverse primer**
KLF4 site 1	5'-aatgcagtttgtgctggaaaaa-3'	5'-gaagaatgaaacactcactggtgact-3'
KLF4 site 2	5'-acagtgcaatttagtcatcaaagg-3'	5'-cagaacaggtagggccagaa-3'
KLF4 site 3	5'-cctttgacctttactggccatttca-3'	5'-ccacgcactctcctttcca-3'
KLF4 site 4	5'-ggcttgaaaagtcatcagtgagtgt-3'	5'-ccactgcctgtaatatttgatgactaa-3'
KLF4 site 5	5'-ttgctgattgtctatttttgcgttt-3'	5'-caaattggccgagatccttct-3'
TATA box	5'-aggcggaggaaaaggctgta-3'	5'-ccgcagacacgttcgttct-3'
H3K4m3_1	5'-gaaccccaaagtcaacgaag-3'	5'-actcgccttgctgattgtct-3'
H3K4m3_2	5'-gaagactggtggggtcagc-3'	5'-ataatcgcgctcttctccag-3'
H3K27m3_1	5'-ggctggtgtcaacctaggaa-3'	5'-agaatcgcagggttcaaaca-3'
H3K27m3_2	5'-agcacagacttaacactcaacaca-3'	5'-gcctcagcctcatcatgttt-3'
AFP	5'-gtttctcgttgcttacacaaag-3'	5'-aggccaatagtttgtcctcact-3'
GAPDH	5'-gcacggaaggtcacgatgt-3'	5'-ccgggattgtctgccctaat-3'
SAT2	5'-catcgatggaaatgaaaggagtc-3'	5'-accattggatgattgcagtcaa-3'

LUC reporter assays

The *KLF4* promoter was predicted using the Gene2Promoter program (Genomatix). The putative *KLF4* promoter fragment was amplified by PCR from human BAC clone RP11-80F13 (imaGenes), confirmed by sequence analysis, and cloned into BglII/HindIII sites of the promoterless vector pGL4.12[*luc2CP*] containing the protein destabilization sequences hCL1 and hPEST (Promega). To generate the reporter construct, a fragment of the *KLF4* upstream regulatory region containing CDX2 binding sites 3 and 4 (nt –1383 to –1169 relative to the *KLF4* TSS) was cloned into KpnI/NheI sites in front of the *KLF4* promoter. Mutant reporter constructs were generated from the respective plasmids via site-directed mutagenesis (QuikChange XL Site-Directed Mutagenesis Kit; Stratagene). Site 5 in the *KLF4* promoter was mutated in all reporter plasmids. The identity of all constructs was confirmed by sequencing. For LUC assays, K-562 cells ($1x10^6$/well) stably transduced with either N-terminally HA-tagged CDX2 or an empty pLenti6.2/V5-DEST control vector were transfected in 6-well plates with 4 µg of the reporter plasmids using Amaxa technology (Lonza). After 72 h of incubation, cells were counted, harvested, and lysed with Passive Lysis Buffer (Promega). Firefly luciferase activities were determined by adding Luciferase Assay Reagent II (Dual-Luciferase Reporter Assay System; Promega). Relative firefly luciferase activities were calculated by normalizing to viable cells as determined using the CellTiter96AQ$_{ueous}$One Solution Proliferation Assay (Promega).

Primer sequences and restriction enzymes used for cloning		
Gene	**Primer sequences**	**Restriction enzymes**
KLF4 promoter	5'-tatagatctctggatgagtcacgcgga-3' 5'-tctaagcttatgtcagactcgccaggtg-3'	5'-BglII 3'-HindIII
KLF4 site 3/4	5'-tctggtaccagccgaaggaacgagttgt-3' 5'-tatgctagcgattacaggcgtgagccact-3'	5'-KpnI 3'-NheI

Primer sequences used for site-directed mutagenesis	
Gene	**Primer sequences**
KLF4 site 3	5'-ctggccatttcacctc**tgcatg**actcaatttctcatc-3' 5'-gatgagaaattgagt**catgca**gaggtgaaatggccag-3'
KLF4 site 4	5'-agtgtttaattcttc**tgcaat**gggtgtcttctatt-3' 5' aatagaagacaccc**attgca**gaagaattaaacact-3'
KLF4 site 5	5'-gacgcttccaagttata**tgcaat**ccaaagaagaaggatc-3' 5'-gatccttcttctttgg**attgca**tataacttggaagcgtc-3'

Cell cycle, apoptosis, and differentiation analysis

NOMO-1 and K-562 cells were transduced with the pRRL.PPT.SF.hKLF4.Tomato.pre lentiviral vector and analyzed after 3 d by gating on Tomato-positive (+KLF4) or Tomato-negative (–KLF4) cells. For cell cycle analysis, cells were cultured in the presence of 10 mM EdU for 45 min. EdU was detected using the Click-iT EdU Pacific Blue Flow Cytometry Assay Kit (Invitrogen). For apoptosis analysis, cells were stained with Annexin V-APC (1:20; BD Pharmingen) and SYTOX Blue (1:500; Invitrogen) in Annexin V Binding Buffer (BD Pharmingen). For cell cycle and apoptosis analysis following PPARγ agonist treatment, NOMO-1 and K-562 cells ($1x10^5$/ml) were incubated with 5 μM PGJ_2 for 48 h. Cells were fixed with 70% ice-cold ethanol for 4 h at –20°C and stained with 10 μg/ml RNaseA and 100 μg/ml propidium iodide (Sigma) at room temperature for 30 min, and cell cycle profiles were analyzed by flow cytometry. Apoptosis was measured using the Annexin V-PE Apoptosis Detection Kit (BD Pharmingen). To determine the expression of surface antigens associated with myelomonocytic differentiation, cells were stained with human PE-conjugated anti-CD14 (BD Biosciences) or murine APC-conjugated anti-CD11b (Invitrogen). Flow cytometric analyses were performed on a BD LSR II or a BD FACS-Calibur flow cytometer (BD Biosciences).

Colony formation and LTC-IC assays

Human AML cell lines ($1x10^3$ to $1x10^4$ cells) transduced with shRNA constructs were plated in methylcellulose medium (MethoCult H4236; StemCell Technologies) with or without 5 μM PGJ_2 or 50 μM telmisartan, and colonies were counted after 10 d. Spleen or whole BM cells ($1x10^4$) from mice with secondary Cdx2-induced leukemia were plated in methylcellulose medium (MethoCult GF M3434; StemCell Technologies), and colonies were counted after 7 d. Secondary, tertiary, and quaternary colony formation were analyzed by replating $1x10^4$ cells obtained by harvesting primary, secondary, and tertiary cultures, respectively. To generate murine Cdx2 leukemia cell lines, cells from the fourth plating were cultured in RPMI-1640 supplemented with 10% FBS and 10% WEHI-conditioned medium as a source of IL-3. For colony formation assays with BM MNC from AML patients or BM CD34-positive cells from healthy donors (StemCell Technologies), $2x10^6$ and $1x10^4$ cells, respectively, were plated in methylcellulose medium (MethoCult H4434 Classic; StemCell Technologies) with or without 5 μM PGJ_2, and colonies were counted after 10 d. For LTC-IC assays, $2x10^6$ or $1x10^4$ cells were resuspended in human LTC medium (MyeloCult H5100 supplemented with 10 μM hydrocortisone; StemCell Technologies) with or without 5 μM PGJ_2 and plated on an irradiated (80 Gy) M2-10B4 stromal layer in collagen-coated tissue culture plates. After 5 wk of culture with half-medium change 3 times a wk, cells were harvested and plated in methylcellulose medium (MethoCult H4435 Enriched; StemCell Technologies) without PGJ_2, and colonies were counted after 12 d. Replating was performed as described above with $2x10^6$ cells in MethoCult H4434 Classic.

Murine BMT assays

Non-competitive transplantation experiments were performed as described previously (4) using the following conditions. For the gene expression profiling experiments shown in Figure 4, E-G, BM cells isolated from C57BL/6 mice were transduced with pMSCV-Cdx2-IRES-GFP or pMSCV-IRES-GFP, and $8x10^5$ GFP-positive cells were injected into lethally irradiated syngeneic recipient mice after 48 h, followed by injection of $1x10^6$ spleen cells from primary leukemic animals into sublethally irradiated secondary recipients. Spleen cells from secondary leukemic animals were harvested, and RNA was extracted and hybridized to GeneChip Mouse Genome 430A 2.0 microarrays (Affymetrix) as described above. For the experiments shown in Figure 3, H and I, $2x10^4$ spleen cells from secondary leukemic animals transduced with KLF4 or empty vector were injected into sublethally irradiated tertiary recipients. Animal experiments were performed after approval and in accordance with the guidelines of the Subcommittee on Research Animal Care of Children's Hospital Boston.

IP and western blotting

IP and western blotting were performed using standard procedures with the following Ab: anti-β-actin (Sigma-Aldrich); anti-HA, anti-KLF4, anti-KDM5B (Cell Signaling); anti-KLF4 (H-180; Santa Cruz). Whole-cell protein extracts for co-IP were prepared using CHAPS lysis buffer containing 40 mM HEPES, 120 mM NaCl, 1 mM EDTA, and 0.3% CHAPS supplemented with Halt Protease and Phosphatase Inhibitor Cocktail (Pierce). Protein extracts were precleared with Protein G Sepharose (GE Healthcare) and incubated at 4°C with anti-KDM5B overnight and with Protein G Sepharose for the last h. Immobilized proteins were washed 5 times, resuspended in Laemmli buffer, and subjected to SDS-PAGE and western blotting. Whole-cell protein extracts for IP were prepared using CHAPS lysis buffer and incubated with anti-KLF4 (H-180; Santa Cruz) at 4°C overnight and with Protein G Sepharose for the last h. Immobilized proteins were washed 5 times, resuspended in Laemmli buffer, and subjected to SDS-PAGE and western blotting with anti-KLF4 (Cell Signaling). Whole-cell protein extracts for direct western blotting were prepared using lysis buffer containing 20 mM Tris, 1% Triton X-100, 150 mM NaCl, 1 mM EDTA, and 10% glycerol supplemented with Halt Protease and Phosphatase Inhibitor Cocktail, and 30 μg of protein were subjected to SDS-PAGE and western blotting.

Quantitative DNA methylation analysis

DNA methylation was quantitatively assessed at single CpG units using MassARRAY EpiTyper technology (Sequenom) as previously described (5). Briefly, genomic DNA (1 μg) was bisulfite-converted (EpiTect Bisulfite Kit; Qiagen), and regions of interest tiling the *KLF4* gene upstream and downstream region were PCR-amplified. PCR products were transcribed in vitro, cleaved by RNase A, and subjected to MALDI/TOF mass spectrometry. Calculations and data display are based on CpG units, defined as cleavage fragments containing one or a few CpG dinucleotides. Measurements were performed in duplicates, averages are displayed in the Results section.

Amplicon name	Primer sequences	Position relative to TSS	Length (bp)	Number of CpGs
KLF4 region 1	aggaaga-gagGTGTTATGTTTTGTTTGTTTAATTG (F) cagtaatacgactcactatagggagaaggctATCCT-TAACTCAACTATATATACC (R)	+1295/+1011	308	33
KLF4 region 2	aggaagagagGGGTATATATAGTTGAGT-TAAGGATA (F) cagtaatacgactcactatagggagaaggctACC-TTCTAA ACCCCCACATTAATA (R)	+1012/+596	441	43
KLF4 region 3	aggaagagagTTTTAT-TAATGTGGGGGTTTAGAAG (F) cagtaatacgactcactataggga-gaaggctTAAAAACTTTTATATA-CAAAAAAACTTTTT (R)	+599/+436	193	6
KLF4 region 4a	aggaagagagT-TAAAATTTAAAATTTTAAATTGGT (F) cagtaatacgactcactatagggagaaggctACAAC-CAATCTCACCTAAC (R)	-509/-240	288	19
KLF4 region 6	aggaagagagTTAAGAGAAGGTTAGAG-GAGTGTTT (F) cagtaatacgactcactatagggagaaggctACC-TACCTAACCAACATAATAAAACC (R)	-696/-1091	421	24
KLF4 region 7	aggaagagagTTTAT-TATGTTGGTTAGGTAGGTTT (F) cagtaatacgactcactatagggagaaggctACACT-TTTTAAAAACTAAAATCCCA (R)	-1094/-1462	393	10
KLF4 region 8	aggaaga-gagGGTGGGATTTTAGTTTTTAAAAAGTG (F) cagtaatacgactcactatagggagaaggctTTCC-TAAATTAACACCAACCTAAAC (R)	-1460/-1795	360	23
KLF4 region 9	aggaagagagGGTTGGTGTTAATTTAG-GAAAGAGA (F) cagtaatacgactcactataggga-gaaggctAAAAATAAAAATCA-CACTCAAAAAC (R)	-1800/-2103	328	13
F, forward; R, reverse				

References

1. Sander S, et al. MYC stimulates EZH2 expression by repression of its negative regulator miR-26a. *Blood*. 2008;112(10):4202-4212.

2. Kharas MG, et al. Musashi-2 regulates normal hematopoiesis and promotes aggressive myeloid leukemia. *Nat Med*. 2010;16(8):903-908.

3. Eisen MB, Spellman PT, Brown PO, Botstein D. Cluster analysis and display of genome-wide expression patterns. *Proc Natl Acad Sci USA*. 1998;95(25):14863-14868.

4. Scholl C, et al. The homeobox gene CDX2 is aberrantly expressed in most cases of acute myeloid leukemia and promotes leukemogenesis. *J Clin Invest*. 2007;117(4):1037-1048.

5. Ehrich M, et al. Quantitative high-throughput analysis of DNA methylation patterns by base-specific cleavage and mass spectrometry. *Proc Natl Acad Sci USA*. 2005;102(44):15785-15790.