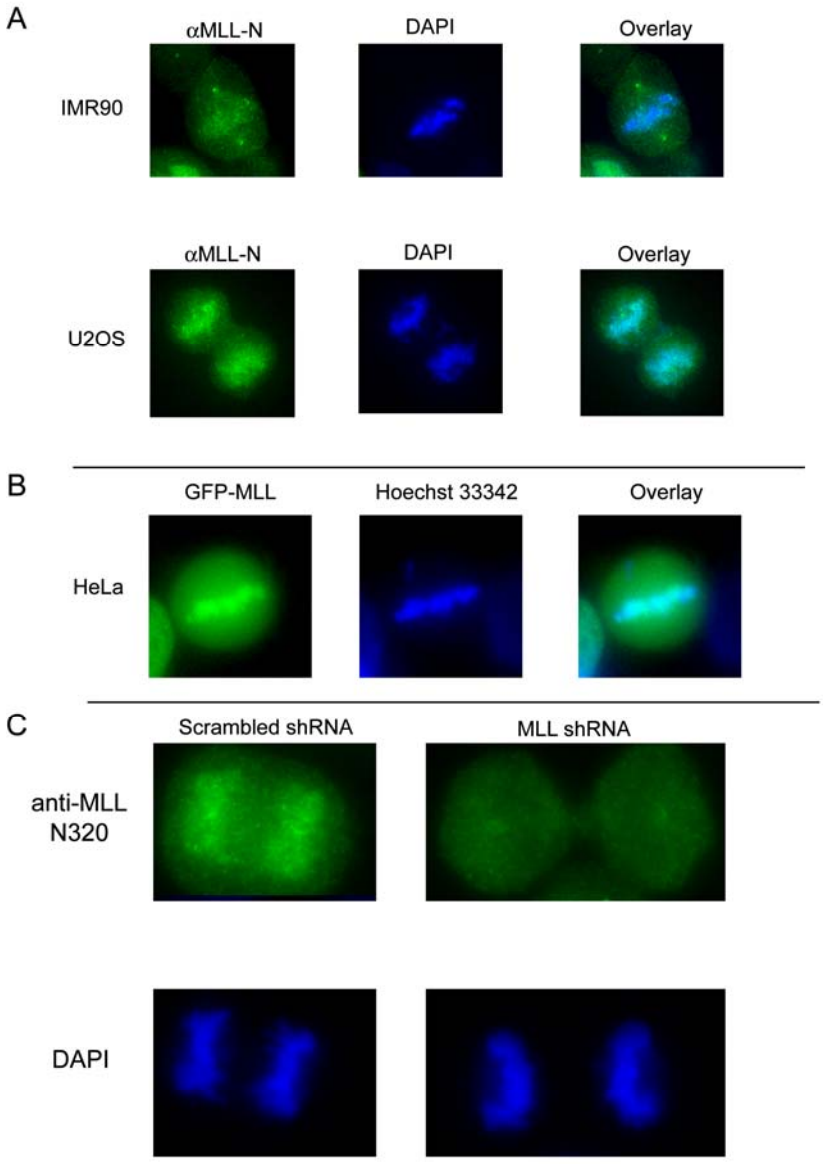
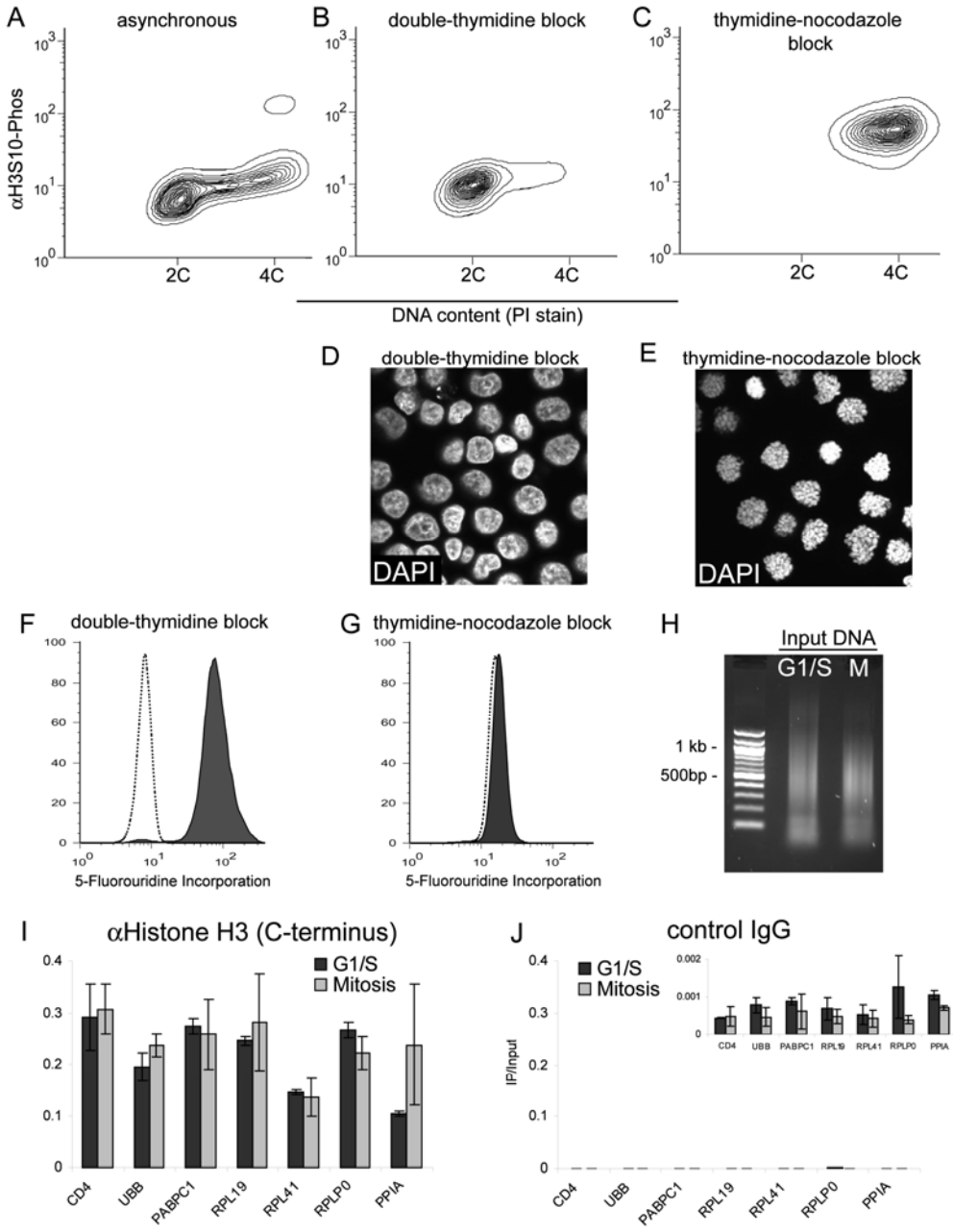
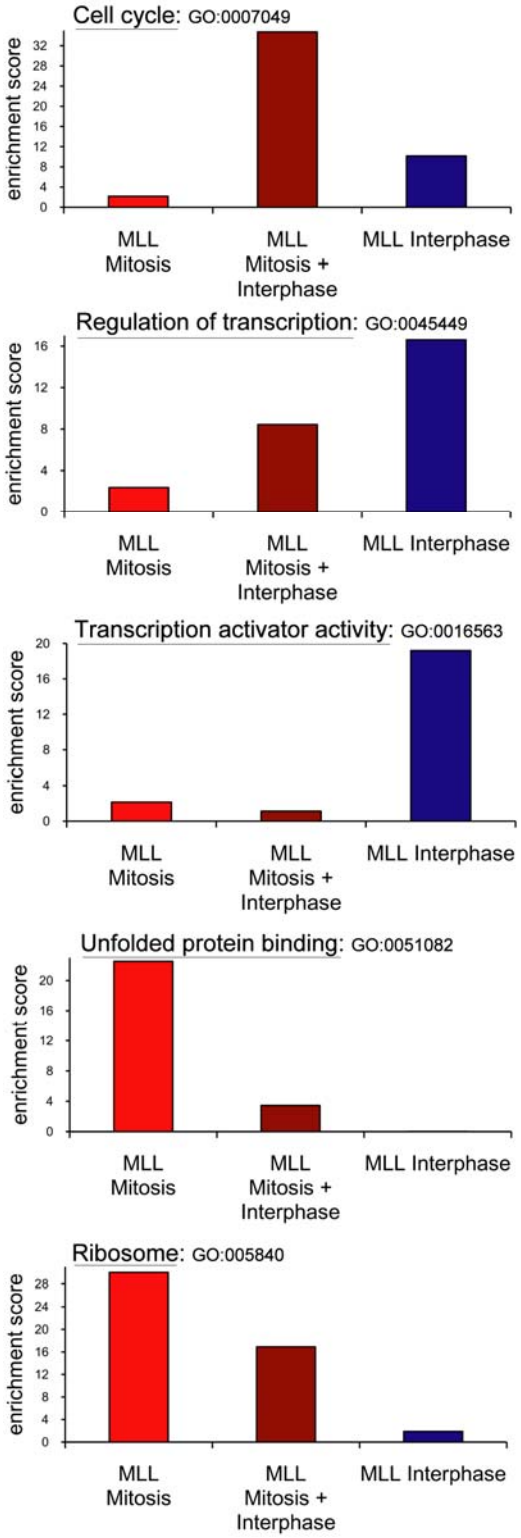


Supplemental Figure 1. A) Immunofluorescence of MLL localization in mitotic IMR90 and U2OS cells. Asynchronous cells stained with anti-MLL-N (457). Secondary antibodies were conjugated with Cy2. All cells were counterstained with DAPI. These are representative images of patterns seen in all mitotic phases. B) Live cell image of GFP-MLL localization in HeLa cells (the GFP-MLL PCDNA3 plasmid was kindly provided by James Hsieh, Wash U, St Louis) C) Control immunofluorescence for MLL in cells expressing an MLL shRNA, verifying specificity of the antibodies used in this study.



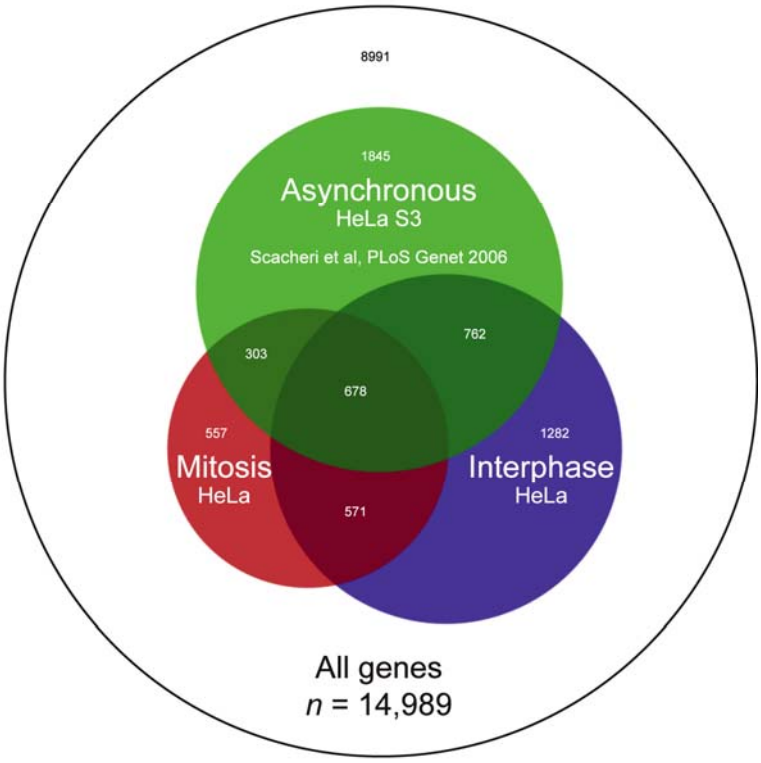
Supplemental Figure 2. Preparation of pure synchronized populations of mitotic and interphase cells for chromatin immunoprecipitation. A – C) Flow cytometry analysis of H3S10-Phos (mitotic marker) and DNA content (PI) of asynchronous, double-thymidine blocked, or thymidine-nocodazole blocked cells. D – E) DAPI staining of synchronized cells confirming condensed pro-metaphase chromatin morphology in thymidine-nocodazole arrested cells. F – G) 5-fluoruridine incorporation in arrested cell populations, confirming the absence of nascent transcription in nocodazole arrested cells. H) Effective sonication of crosslinked mitotic chromatin. Following ChIP procedure, purified input DNA was resolved on a 1% agarose gel, confirming similar fragmentation in both synchronized populations. I) Similar H3 occupancy in both arrested cell populations. J) Similar levels of background in mitotic and interphase populations, as detected with control (pre-immune) IgG.



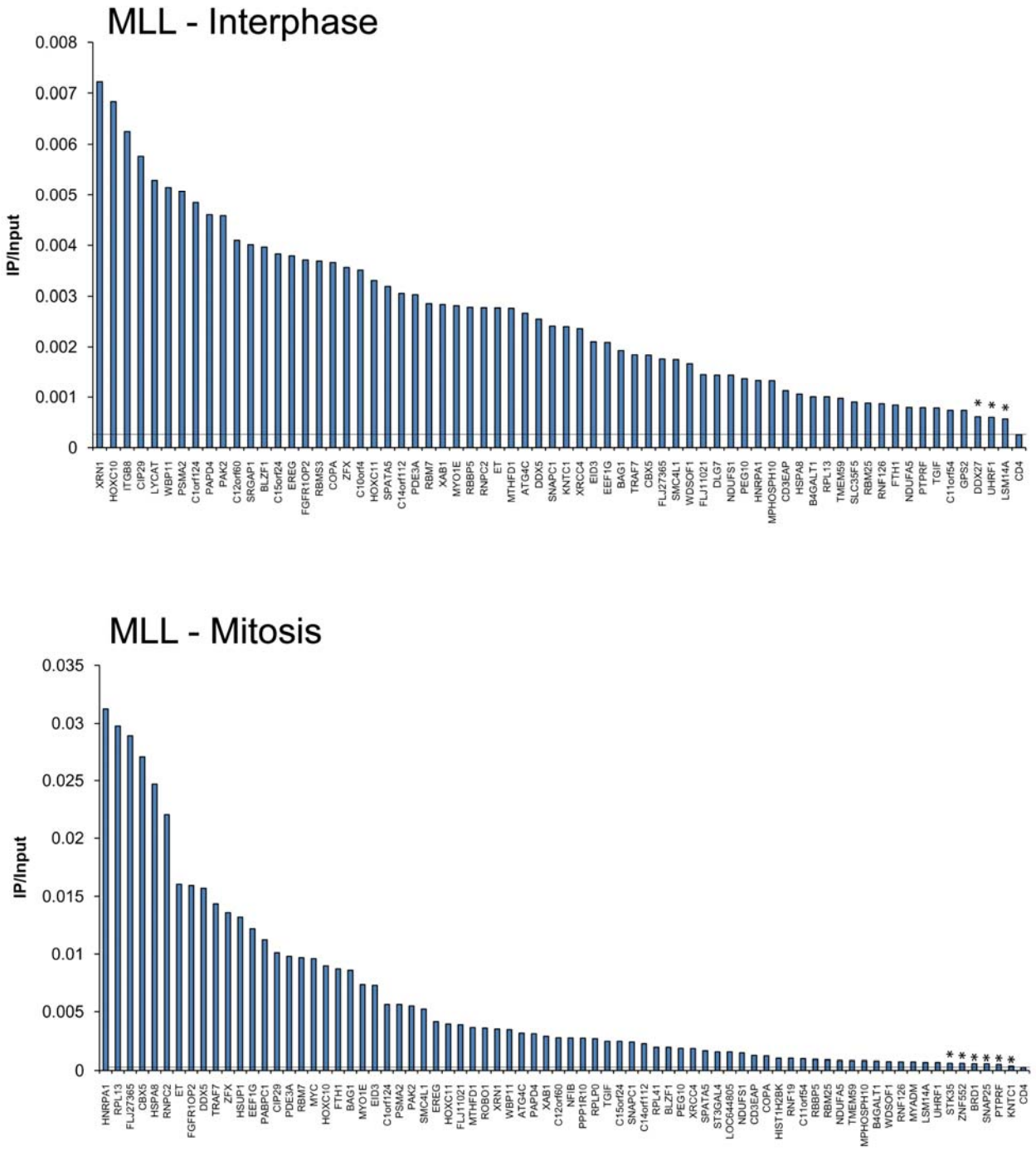
Supplemental Figure 3. Gene ontology analysis of MLL bound genes in each occupancy category. Analysis was performed using DAVID software (Dennis et al, Genome Biology 2003). Enrichment score was calculated as the –log(p value) calculated from Fisher's exact test.

Motif	Hits	Fold Change	pValue
M00098.Pax-2	3277	2.585319	0
M00090.Abd-B	3086	2.18607	4.9E-324
M00913.MYB	3419	1.9799902	1.83E-282
M00961.VDR	3011	2.07995	8.24E-282
M00448.Zic1	3061	2.0313458	2.86E-270
M00492.STAT1	2794	2.0721855	2.46E-259
M00484.Ncx	2660	2.0304177	6.08E-235
Elk-1	1349	2.6237614	1.24E-205
AP2alpha	1966	2.1651695	4.26E-203
M00427.E2F	692	3.4658551	7.98E-163
SPI-1	3851	1.6020057	1.73E-162
M00468.AP-2rep	3117	1.6897777	2.21E-160
M00792.SMAD	3481	1.6178355	9.24E-153
M00395.HOXA3	2051	1.8768134	1.75E-147
M00470.AP-2gamma	1227	2.3049645	3.36E-146
M00695.ETF	814	2.8286862	5.11E-142
M00493.STAT5A	2584	1.71699	8.32E-141
M00940.E2F-1	466	4.185456	8.17E-139
M00147.HSF2	2613	1.6888419	1.36E-134
M00456.FAC1	2512	1.6986021	4.55E-132

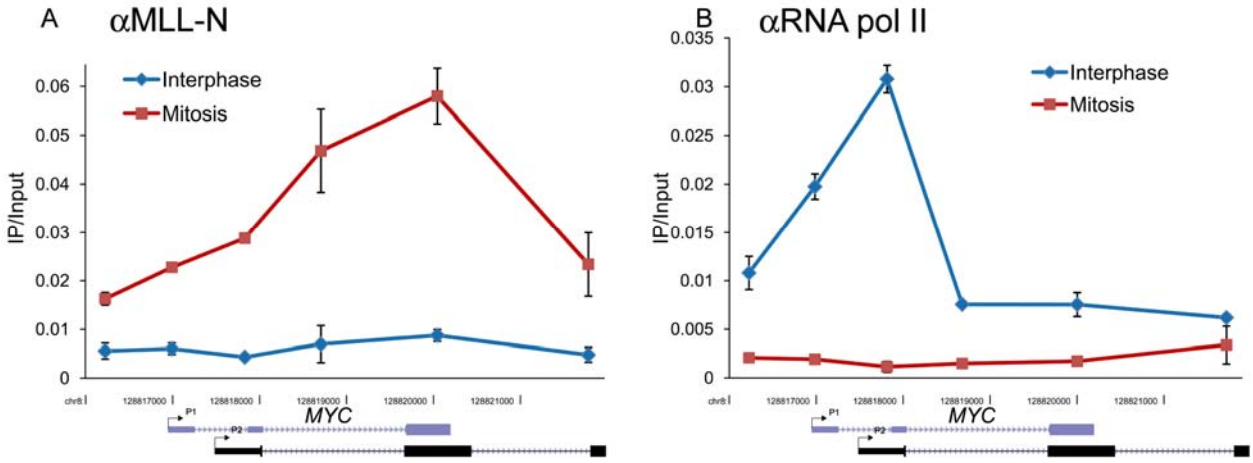
Supplemental Figure 4. Sequence motifs correlated with MLL occupancy in interphase. MLL interphase peak coordinates evaluated using cis-regulatory element annotation system (CEAS, Ji et al, Nucleic Acids Research 2006).



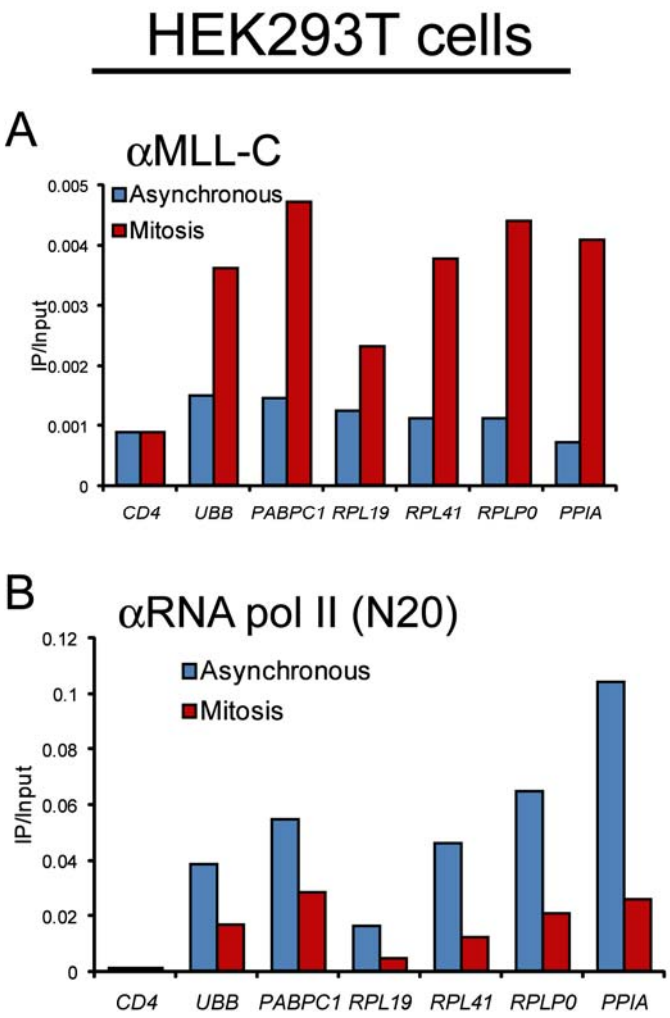
Supplemental Figure 5. Comparison of genome-wide MLL binding in interphase and mitosis to a published dataset of MLL binding in asynchronous HeLa S3 cells. Genes that were not covered on both platforms were excluded, yielding a total of 14,989 genes in the analysis. Binding thresholds were chosen that yielded roughly equal numbers of MLL-positive genes in both studies (Collins: $p = 0.05$; Vakoc: FDR = 0.1). The open circle represents all genes covered in the analysis. The Venn diagram shows the level overlap between the three data sets. Discrepancies between the data sets could be related to different array platforms (the nimblegen array tiling included 500 bp downstream of the TSS whereas Scacheri et al tiled the promoter), analytical approaches, or differences between HeLa vs HeLa S3 cell lines.



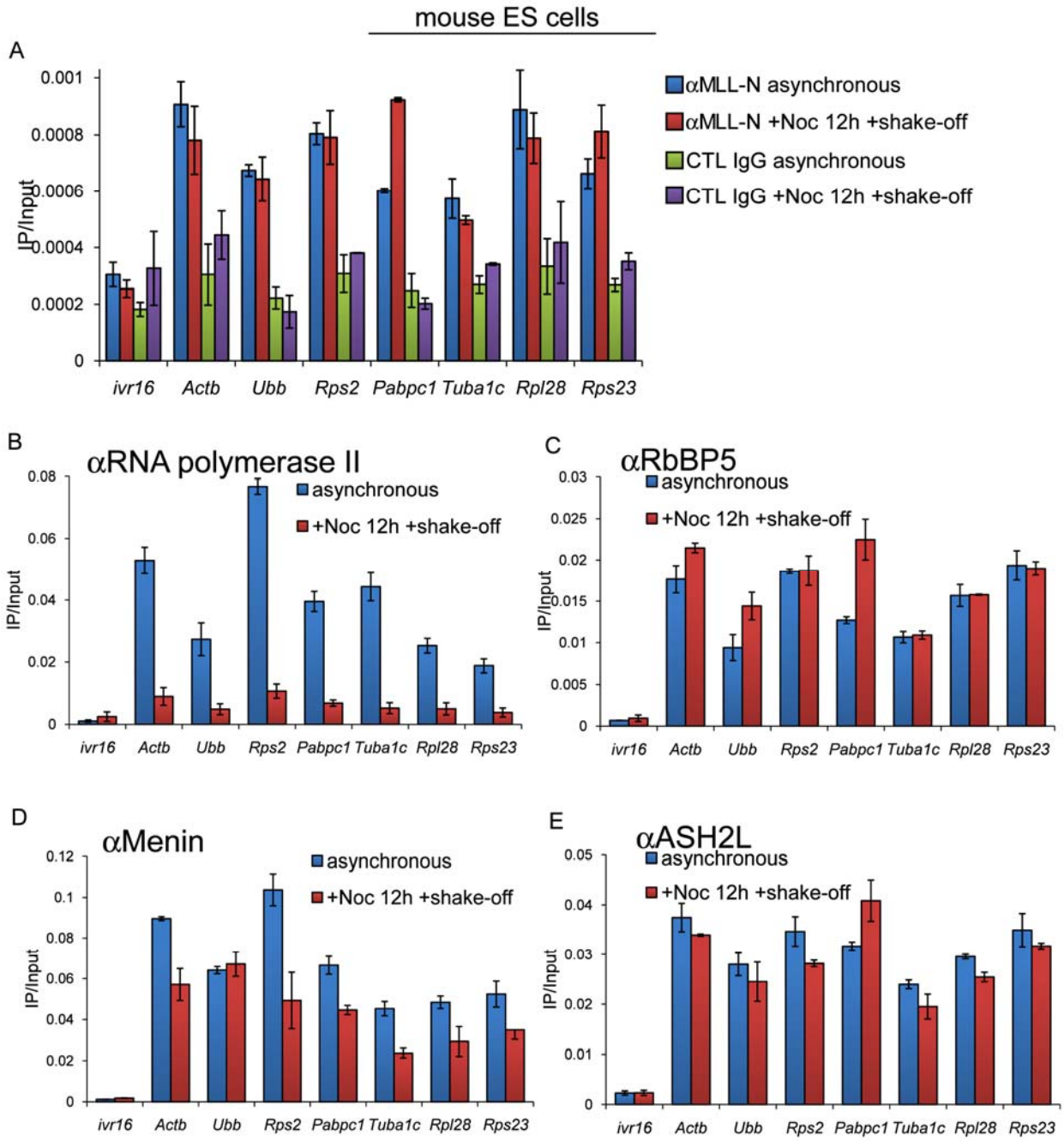
Supplemental Figure 6. ChIP-qPCR validation of MLL peaks identified using ChIP-Chip. Results are the average of two biological replicates. CD4 was included as our negative control. A horizontal line is drawn at the CD4 level, which we have extensively verified as background in comparison with control IgG. Peaks were randomly chosen from the interphase or mitotic dataset below the FDR 0.2 cutoff. qPCR primers were designed that amplify the genomic interval called as containing a peak by the Nimblescan algorithm. Asterisks indicate regions scoring below 2.5 fold enrichment above background. Primer sequences are available upon request.



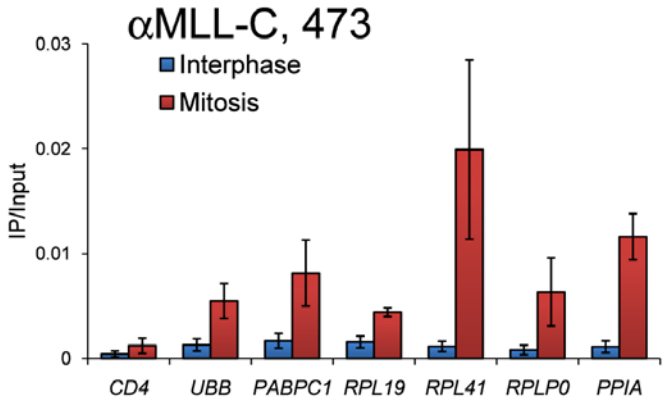
Supplemental Figure 7. MLL preferentially occupies the MYC locus in mitosis (A) whereas RNA polymerase II occupies the locus in interphase (B). ChIP-qPCR analysis using the indicated antibodies comparing interphase and mitotic preparations of chromatin. Primer-pairs amplify the indicated genomic positions relative to the promoter regions (P1 and P2) Interphase (blue) refers to cells arrested by double-thymidine block. Mitosis (red) refers to cells arrested by sequential thymidine-nocodazole treatment. Error bars denote S.E.M.



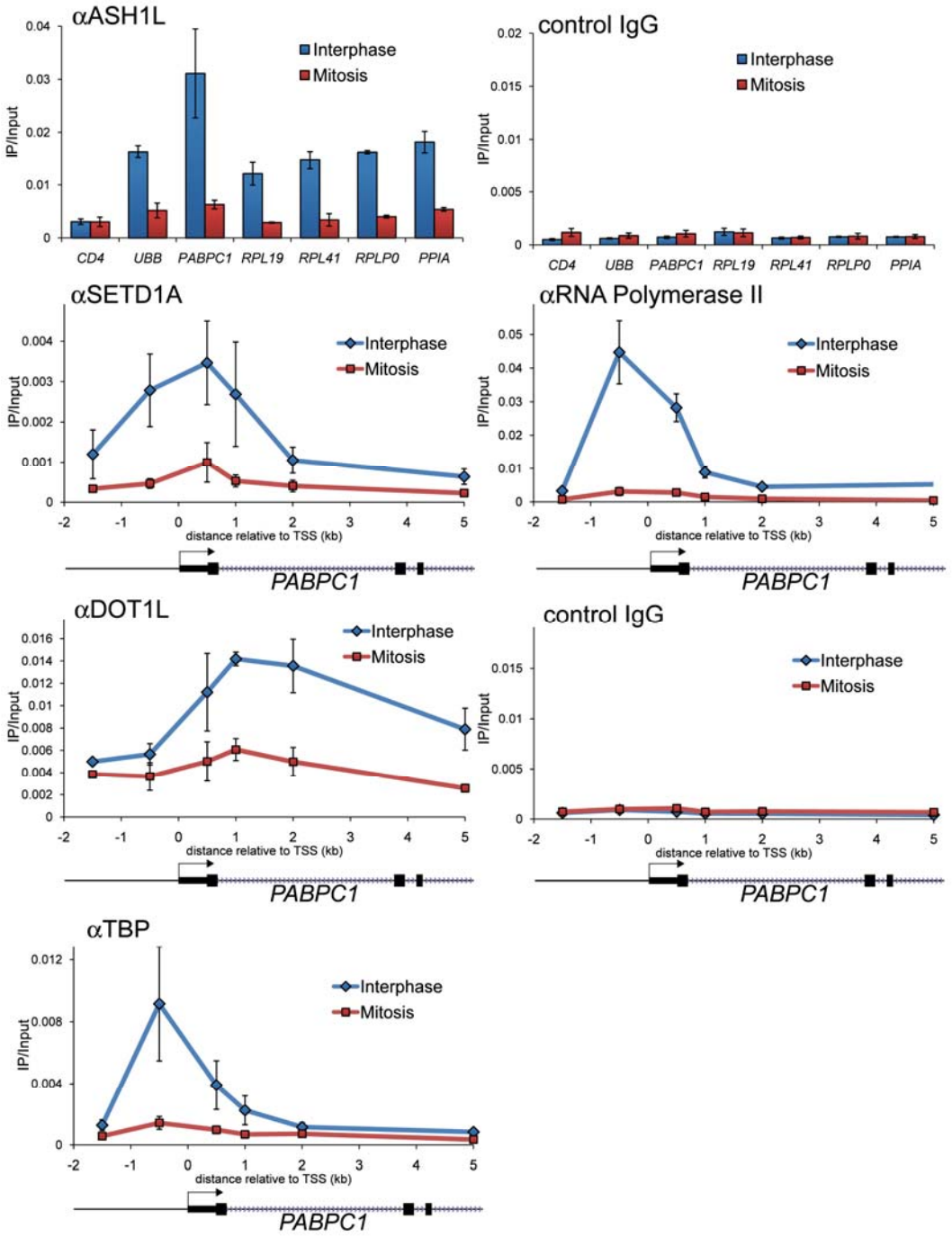
Supplemental Figure 8. MLL is retained during mitosis at highly-expressed genes in HEK293T cells. ChIP-qPCR analysis using the indicated antibodies comparing largely interphase and mitotic preparations of chromatin. The anti-MLL-C (468) antibody was used. Primer-pairs amplify at +0.5 kb relative to the TSS. Mitosis (red) refers to cells arrested with nocodazole treatment for 16 hours. *CD4* is a transcriptionally silent control region. Representative experiment is shown.



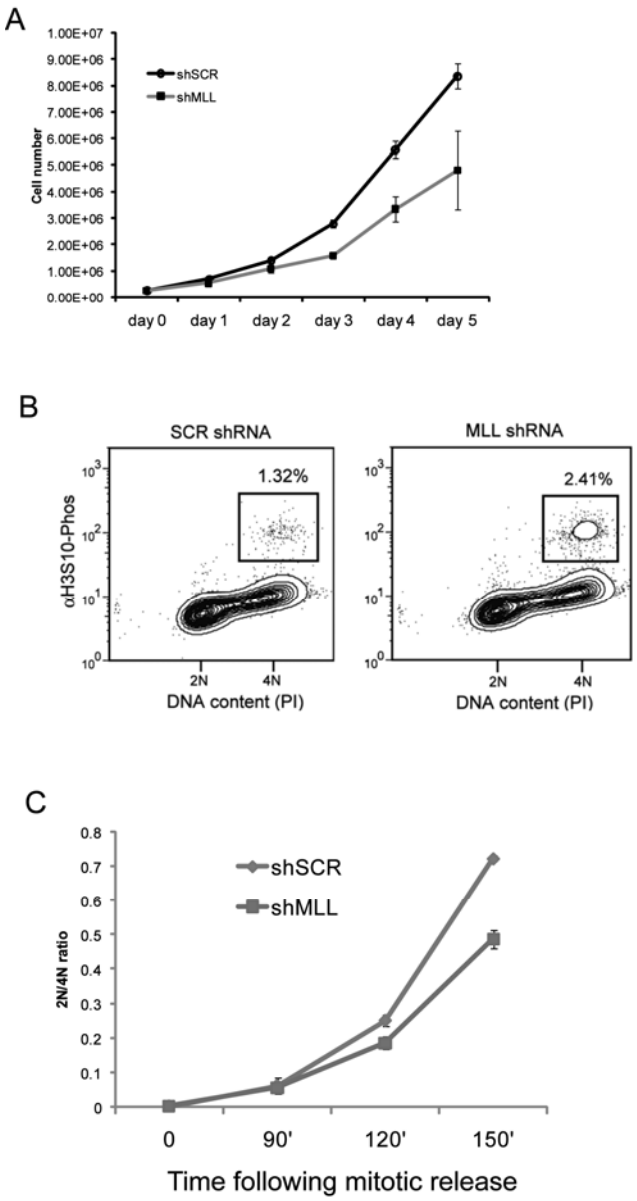
Supplemental Figure 9. MLL is retained during mitosis at highly-expressed genes in mouse ES cells. ChIP-qPCR analysis using the indicated antibodies comparing interphase and mitotic preparations of chromatin. The anti-MLL-N (456) antibody was used. Primer-pairs amplify at +0.5 kb relative to the TSS. Mitosis (red and purple) refers to cells arrested by nocodazole treatment for 12 hours followed by shake-off. *Ivr16* is a non-coding silent control region within the beta-globin locus. Error bars denote S.E.M. Note: the weak MLL signals in these cells might reflect lower occupancy than seen in human cells or a weaker reactivity of the antibody against the mouse MLL protein.



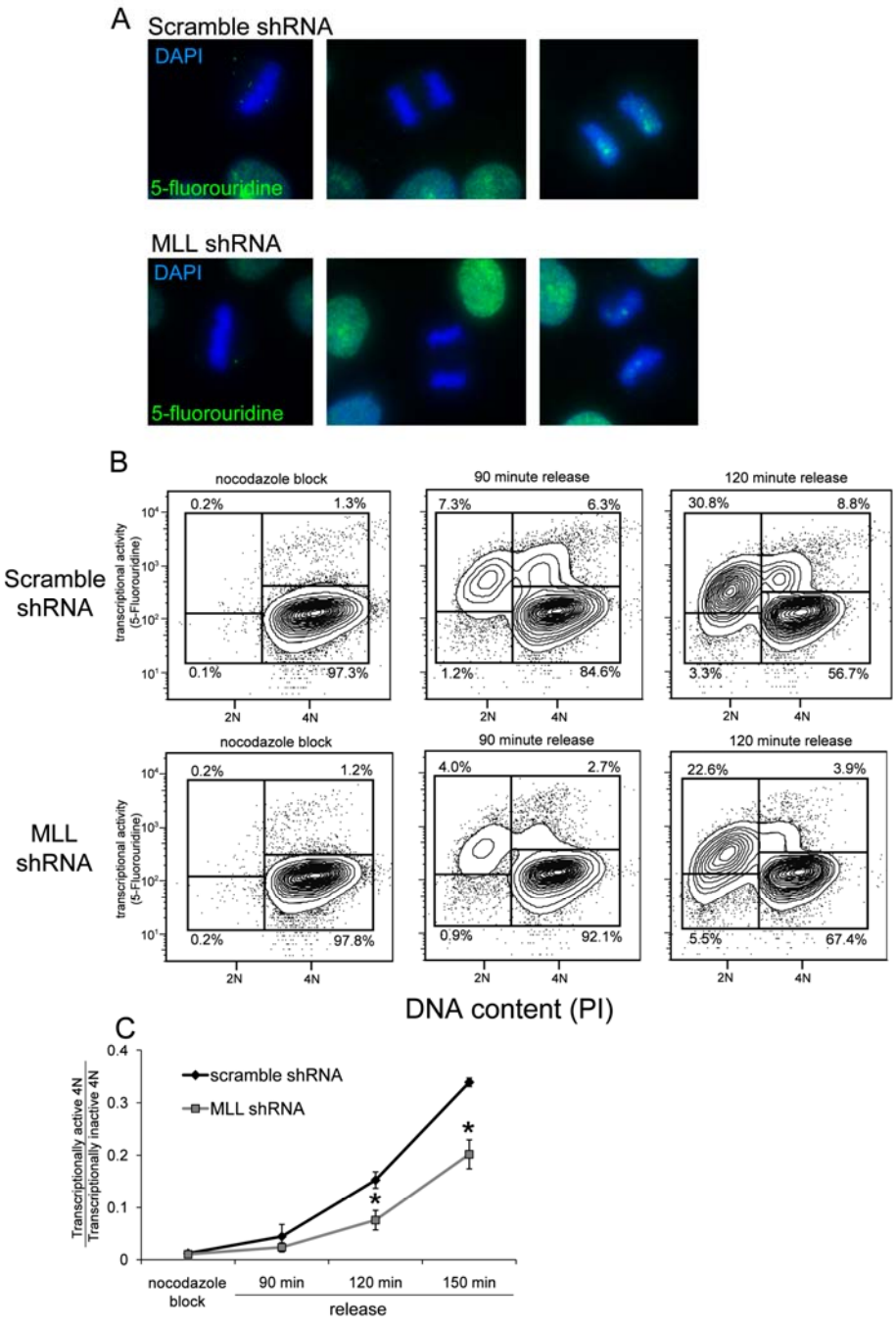
Supplemental Figure 10. Confirmation of MLL retention on mitoic chromosomes with an additional antibody recognizing the C-fragment. ChIP-qPCR analysis using the indicated antibody comparing interphase and mitotic preparations of chromatin. Primer-pairs amplify at +0.5 kb relative to the TSS. Interphase (blue) refers to cells arrested by double-thymidine block. Mitosis (red) refers to cells arrested by sequential thymidine-nocodazole treatment. *CD4* is a transcriptionally silent control region. Error bars denote S.E.M.



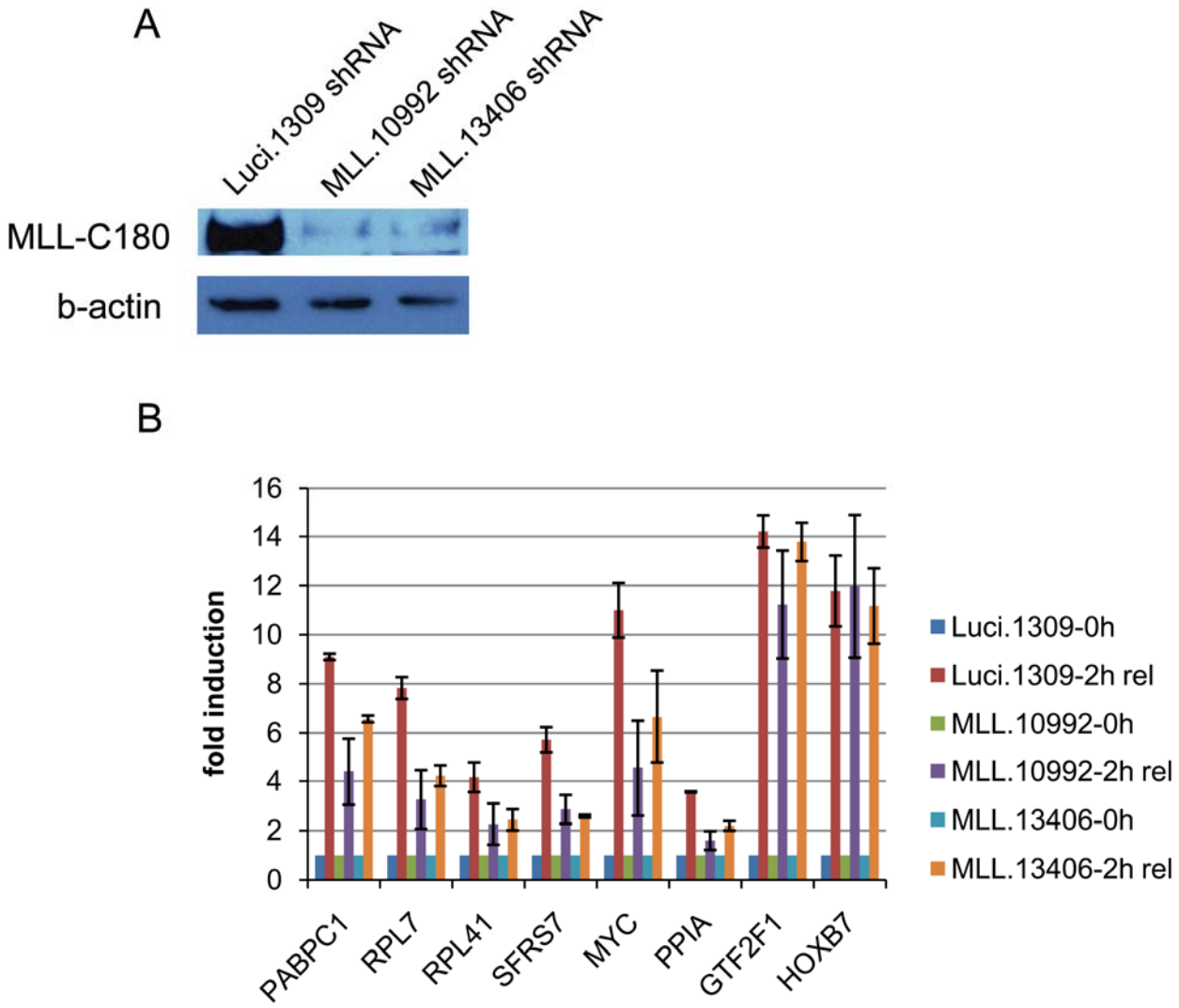
Supplemental Figure 11. TBP, DOT1L, ASH1L, SETD1A, and RNA polymerase II are displaced from highly expressed genes during mitosis. ChIP-qPCR analysis using the indicated antibodies comparing interphase and mitotic preparations of chromatin. The anti-TBP (sc-204) antibody was used (similar results obtained with sc-273). For the top panels, primer-pairs amplify at +0.5 kb relative to the TSS. For the lower panels, primers amplify at the indicated locations relative to the *PABPC1* TSS. Interphase (blue) refers to cells arrested by double-thymidine block. Mitosis (red) refers to cells arrested by sequential thymidine-nocodazole treatment. *CD4* is a constitutively silent control gene. Error bars denote S.E.M.



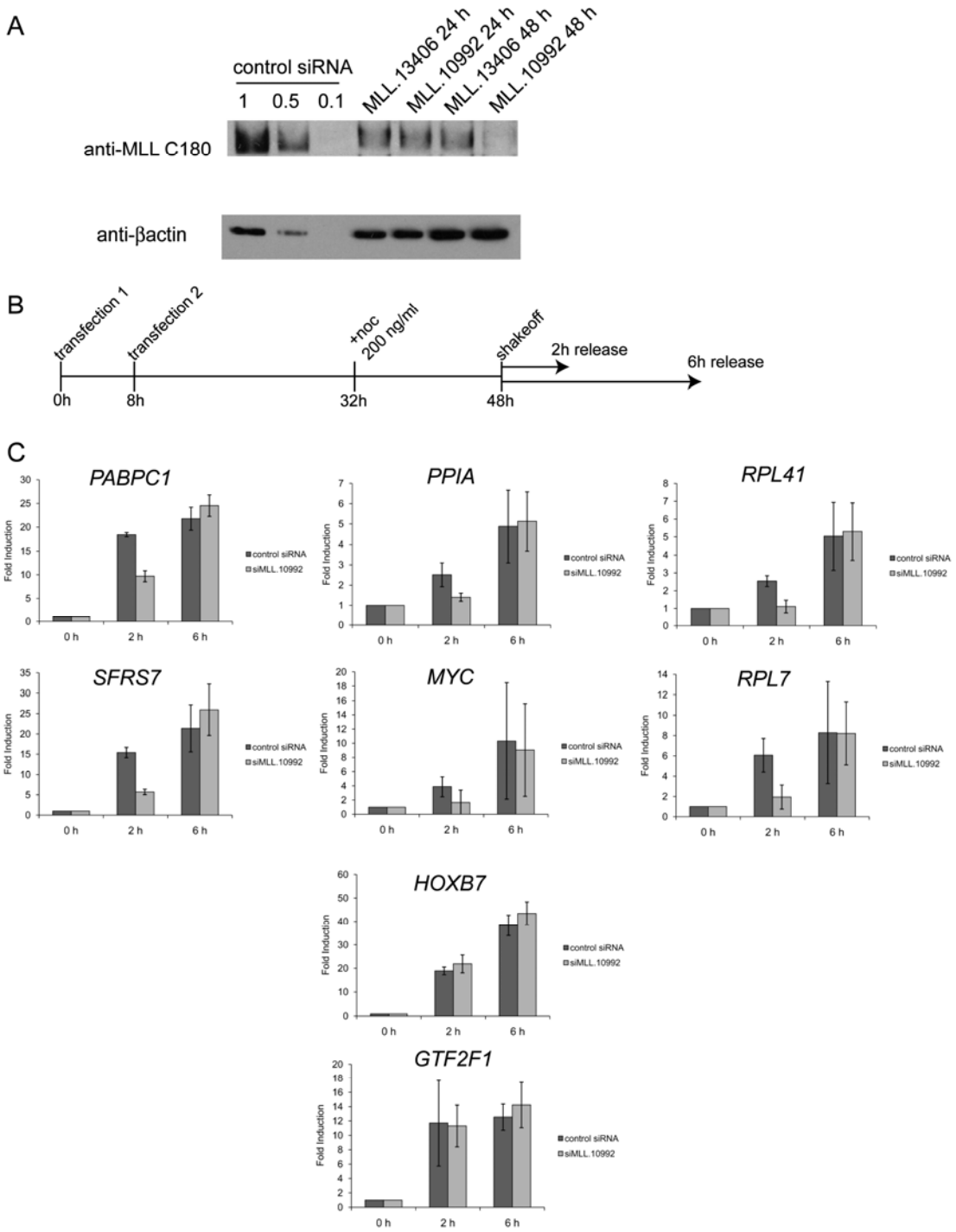
Supplemental Figure 12. MLL knockdown results in a delayed progression through mitosis. A) Comparison of cell growth between HeLa cells cultures expressing a control (shSCR) or MLL (shMLL) hairpin (pSUPER.retro.puro vector). B) Flow cytometry analysis of H3S10-Phos (mitotic marker) and DNA content (PI) of control and MLL knockdown cell cultures grown asynchronously (representative experiment shown of 5 performed). C) Mitotic progression quantified in control and MLL-deficient cells. Cells were arrested by sequential thymidine-nocodazole, shaken-off, washed 3X in PBS, and re-plated in fresh media. Harvested cells analyzed by PI staining of methanol-fixed cells. Plotted is the ratio of percentages of cells having 4N (pre-cytokinesis) to 2N (post-cytokinesis) at the indicated timepoints following release.



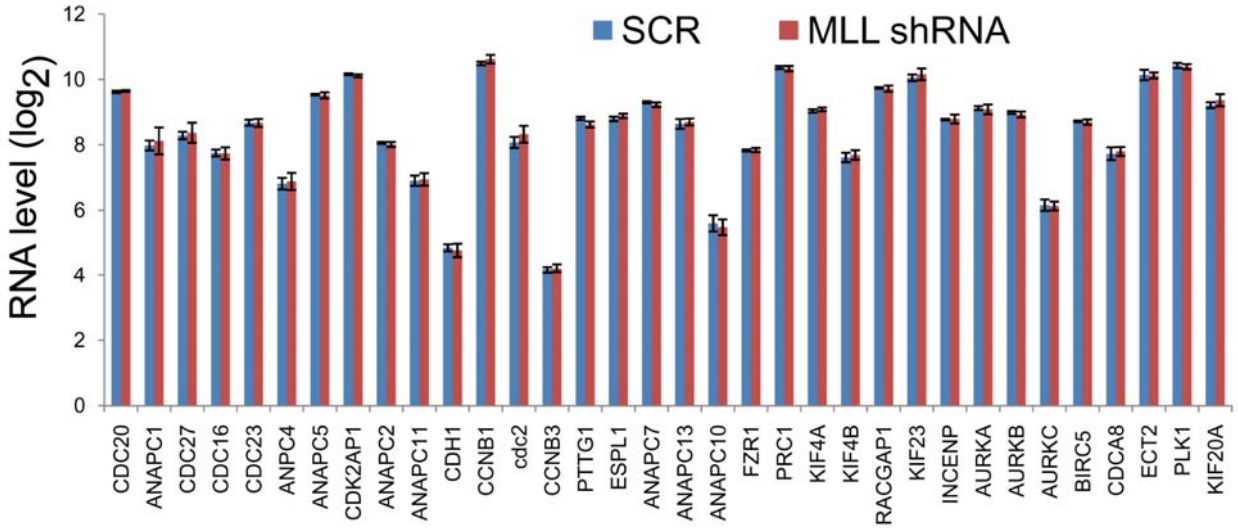
Supplemental Figure 13. MLL-deficient cells displayed delayed restoration of transcriptional activity following mitotic exit. A) Immunofluorescence of HeLa cells pulsed with 5-fluorouridine (FUrd) following by antibody staining (green) overlaid with counterstain with DAPI. Interphase cells display incorporation of FUrd whereas mitotic cells do not B) Flow cytometry analysis of 5-fluorouridine incorporation (antibody stain) and DNA content (PI) in cells released from nocodazole arrest. C) Quantitation of the ratio of 5-fluorouridine positive:negative 4N cells at the indicated timepoints. Asterisks indicate $p < 0.05$ calculated using a two-tailed student's t-test.



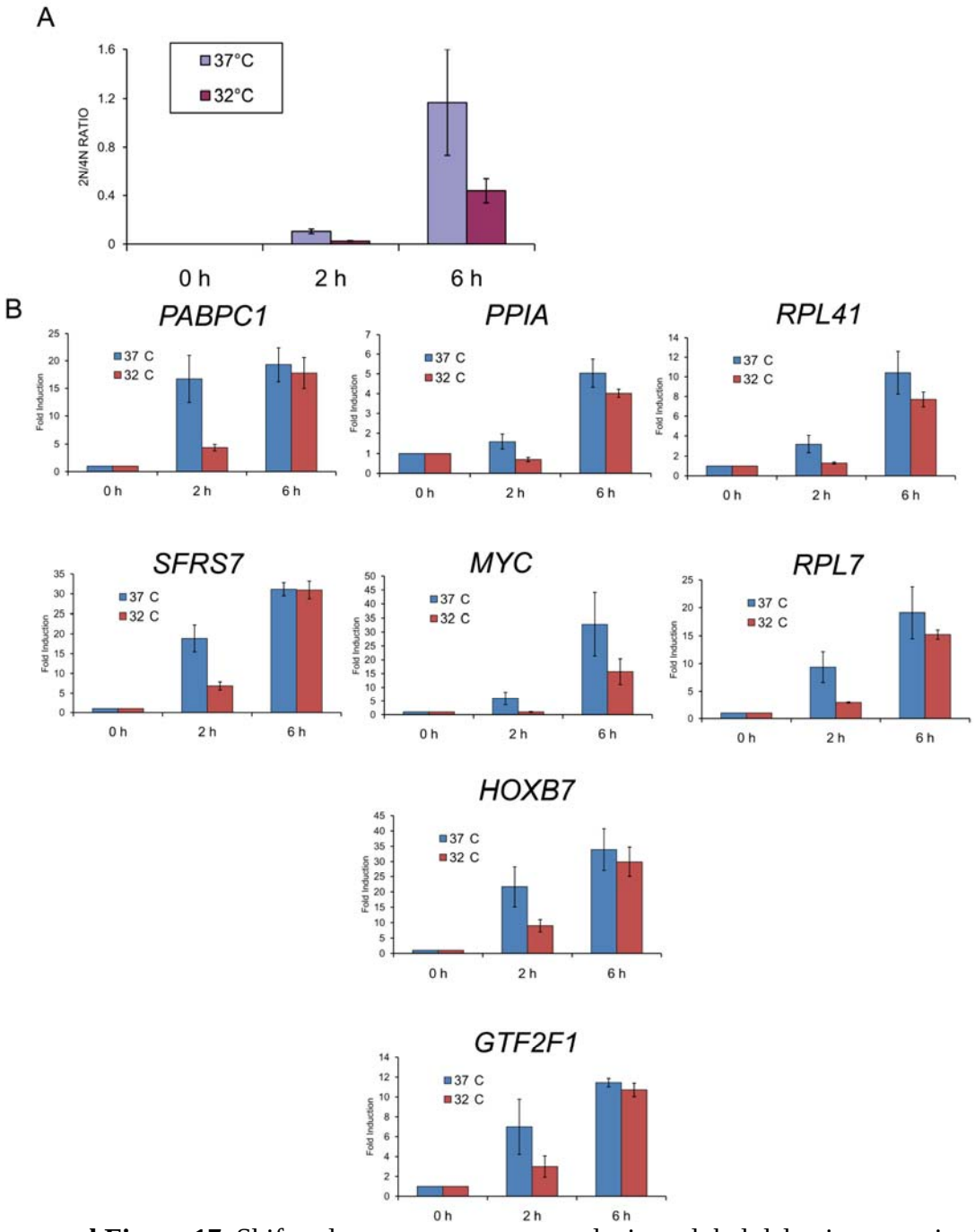
Supplemental Figure 14. MLL knockdown with multiple shRNAs results in gene-specific delays in post-mitotic reactivation. Hairpins were expressed from the LMP retroviral vector infected into HeLa cells, followed by puromycin selection A) Western blot performed with RIPA whole cell extracts prepared from cells selected for 1 week in puromycin. B) Primary transcript RT-qPCR analysis, as performed in Figure 4 comparing blocked and 2 hour released cells. Error bars denote S.E.M.



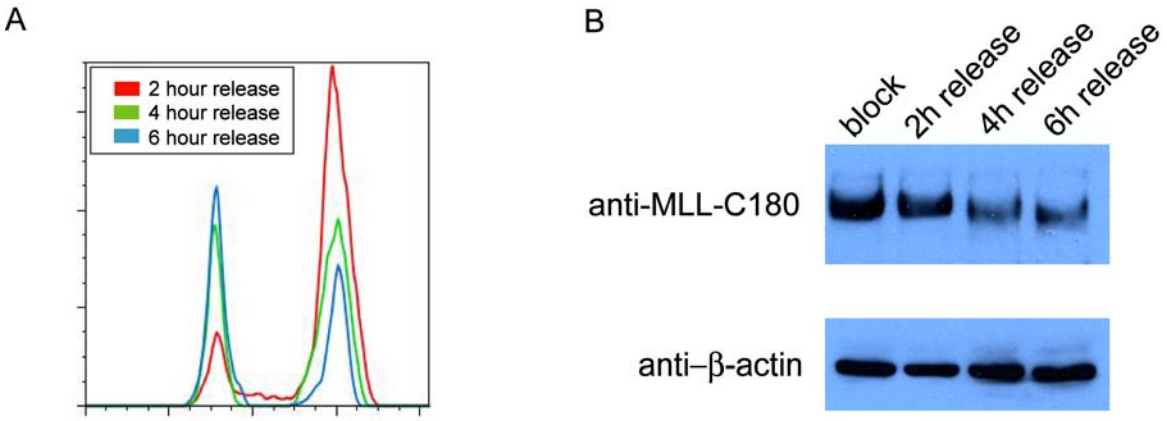
Supplemental Figure 15. MLL knockdown via siRNA transfection results in delayed post-mitotic gene reactivation following mitotic release. A) Western blot performed with RIPA whole cell extracts prepared from cells transfected with the indicated siRNAs for the indicated timepoints. The control siRNA-transfected cell extract was diluted by ½, or1/10 to gauge knockdown efficiency. C) Primary transcript qPCR analysis, as performed in Figure 4. Note: MLL.13406 siRNA was not potent enough to observe delays.



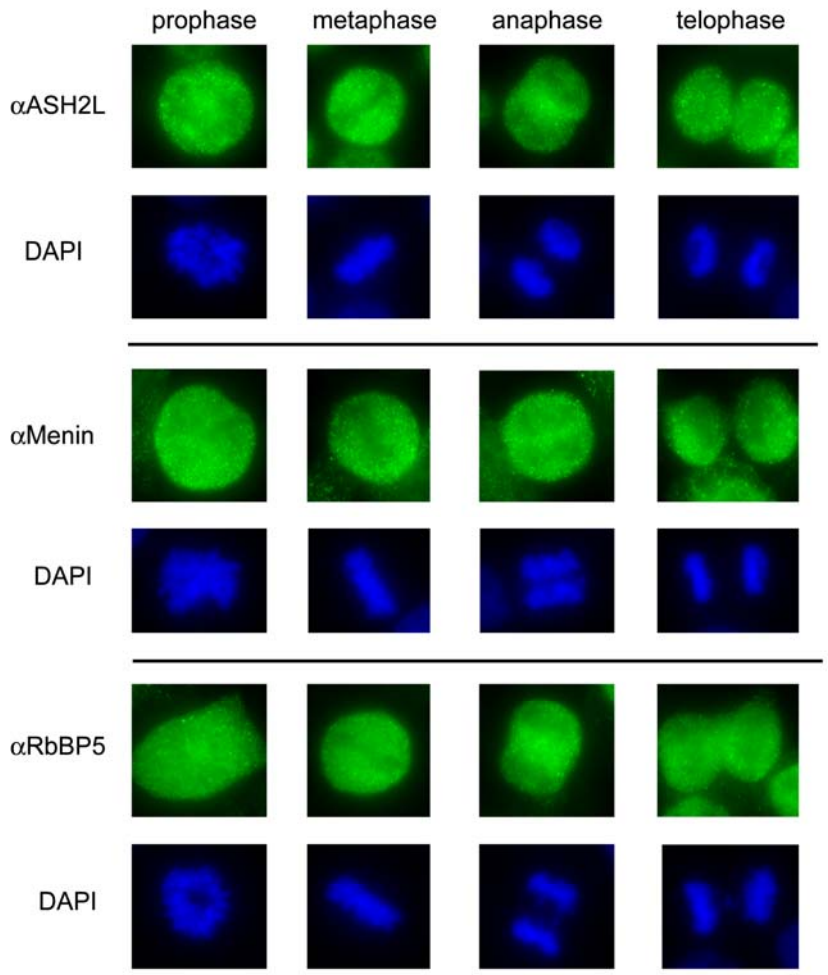
Supplemental Figure 16. Expression microarray data demonstrating that several known regulators of mitotic progression are expressed normally in MLL-deficient cells.



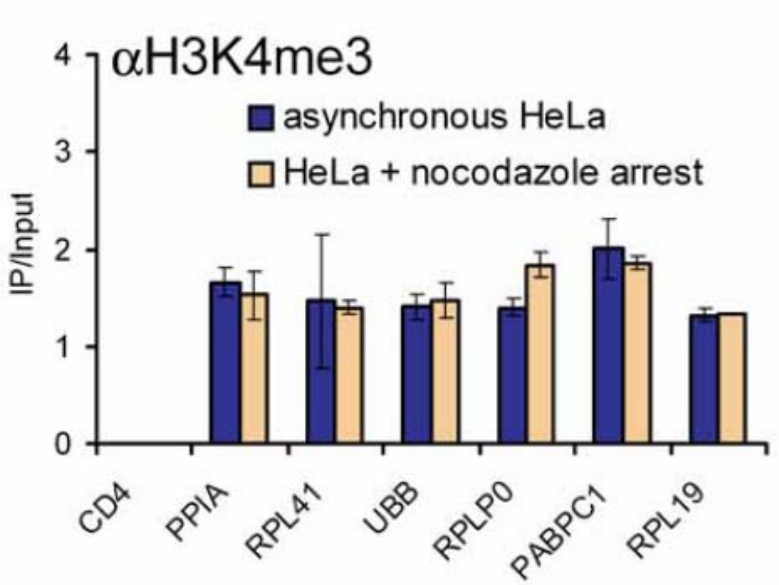
Supplemental Figure 17. Shift to lower temperature results in a global delay in post-mitotic gene reactivation, distinct from that observed in MLL-deficient cells. Cells were thymidine-nocodazole arrested, washed 3X in PBS, then release into fresh medium in either a 37 or a 32 C incubator. A) Quantitation of PI stain for DNA content following mitotic release. B) Primary transcript-qPCR analysis measuring fold activation compared to blocked cells. All primary transcript quantitation was normalized to the level of mature GAPDH mRNA. Note: the delay in gene reactivation is of lesser magnitude than the delay observed by PI staining. This could be due to gene reactivation occurring in late telophase prior to cytokinesis.



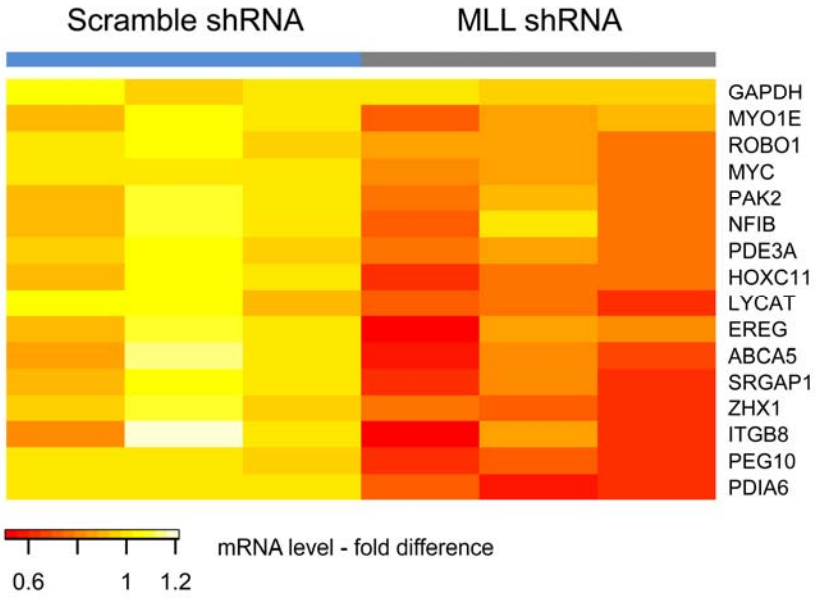
Supplemental Figure 18. Downregulation of MLL C180 protein levels following mitotic release. A) PI stain for DNA content following mitotic release. B) Western blot of whole cell extracts were prepared using RIPA buffer at the various timepoints following mitotic release.



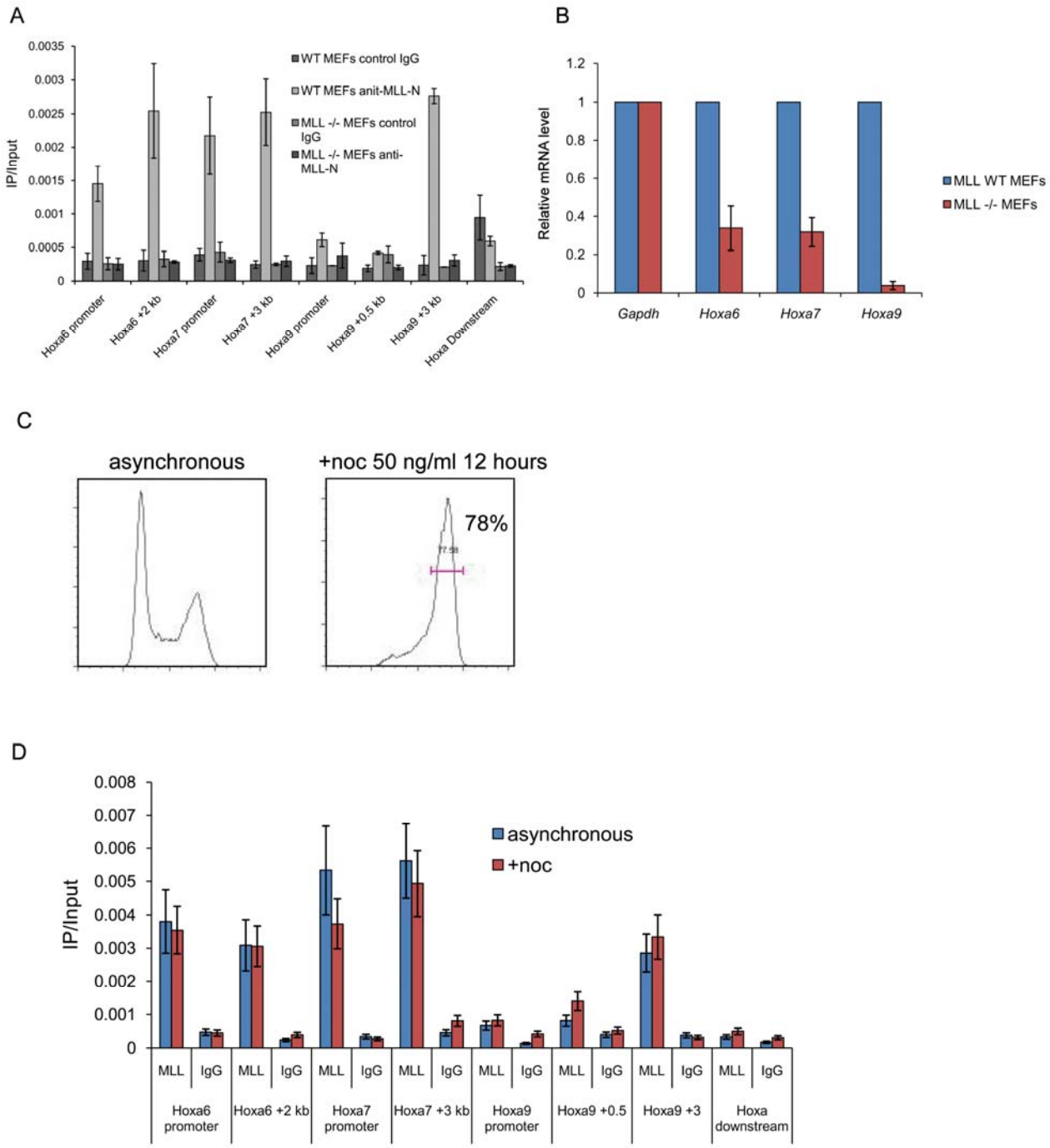
Supplemental Figure 19. Menin, ASH2L, and RbBP5 display a largely cytoplasmic localization during mitosis by immunofluorescence. Staining was performed on fixed HeLa cells grown asynchronously. Cy2 secondary antibody. Counterstained with DAPI.



Supplemental Figure 20. H3K4me3 levels at highly expressed genes are similar in interphase and mitotic chromatin. ChIP-qPCR performed with the indicate antibody. Primers amplify at +0.5 kb relative to the TSS.



Supplemental Figure 21. Heatmap representation of expression microarray data for the indicated genes. Each vertical column represents an independent biological replicate.



Supplemental Figure 22. MLL-dependent genes in murine embryonic fibroblasts (MEFs) display MLL occupancy in both interphase and mitosis. A) ChIP with the indicated antibodies and primers performed in wild-type or MLL -/- MEFs. MLL displays occupancy at *Hoxa6*, *Hoxa7*, and *Hoxa9*. B) RT-qPCR with primers detecting the indicated genes, comparing expression between WT and MLL -/- cells. Expression is normalized to *Gapdh*. C) PI stain of WT MEFs treated with 50 ngl/ml nocodazole for 12 hours. Note: higher concentrations of nocodazole were found to result in loss of cell viability. D) ChIP performed in untreated or nocodazole-arrested wild-type MEFs. Note: we were unable to perform nocodazole arrest-release experiments in MEFs due to elevated toxicity following nocodazole washout.

Supplemental Experimental Procedures

Primary transcript RT-qPCR

RNA was prepared using Trizol reagent (Invitrogen). cDNA synthesis was performed using Superscript II (Invitrogen) with random hexamer primers. Primers were designed that spanned intron-exon junctions. All signals were normalized to the levels of mature (spliced) GAPDH.

GFP-MLL HeLa cell lines

HeLa cells were transfected with GFP-MLL PCDNA3 using PEI reagent. Cells were selected with 400 ug/ml G418 for 3 weeks. GFP positive cells were enriched via two rounds of FACS sorting. Live cell imaging was performed on a DeltaVision RT microscope (Applied Precision).

siRNA transfection

HeLa cells were transfected at 30% confluency using RNAiMAX reagent (invitrogen), according to manufacturer's protocol. control siRNA target sequence (targets the EBV-encoded EBNA gene; not present in HeLa cells) AUGAAAUAACAGACAAUGGAC. MLL.10992 siRNA target sequence: AAGGAAAGCAUUACUGAGAAA . MLL.13406 siRNA target sequence: CCGCGGUAUUAUCCUAAUUUA.

Flow cytometry

For cell-cycle analysis, cells were fixed in 90% methanol/10% PBS for 20 min on ice. Cells were then washed in PBS-T with 2% BSA twice. Anti-H3S10 antibody was then added (1:100) and incubated for 1 hour at room temp. Cells were washed in PBS-T twice then incubated with goat anti-rabbit FITC antibody (1:100) (Jackson Immunoresearch). Cells were washed twice in PBS-T then stained with 50 micrograms/ml propidium iodide (Sigma) in PBS.

FUrd Assay

For 5-fluorouridine incorporation experiments, cells were pulsed with 1.5 mM 5-fluorouridine (Sigma) in the growth media for 30 min before harvest. Cells were washed in PBS, followed by fixation in 3.7% formaldehyde/ 10 mM HEPES 7.5/100 mM NaCl for 12 minutes. Fixation was quenched with 0.125 M glycine in PBS. All subsequent solutions used DEPC water. Cells were permeabilized with PBS containing 0.2% Triton X-100 (PBS-T) for 5 min and blocked with PBS-T containing 2% BSA for 10 minutes. anti-BrdU antibody was added at 2 micrograms/ml for 1 hour at room temperature. Cells were washed twice in PBS-T then incubated with goat anti-mouse FITC secondary antibody (Jackson). Cells were washed twice in PBS-T then stained with 50 micrograms/ml propidium iodide (Sigma) in PBS. RNase and DRB treatment were used as controls to verify that signals were generated from nascent transcription (data not shown). Flow cytometry was performed on a FACSCalibur (Beckton Dickinson) and analyzed with FlowJo software.

Expression microarrays

RNA was prepared using Trizoll reagent from shSCR or shMLL expressing HeLa cells. Expression microarrays were performed at the University of Pennsylvania Microarray facility using a Human Gene 1.0 ST Array (Affymetrix). The data was extracted using Expression Console and analyzed using RMA.

shRNA validation hairpins

Additional MLL hairpins used in Supplemental Figure 16 were expressed using the LMP retroviral backbone (Dickins et al, Nature Genetics 2005). Cells were selected with 2

microgram/ml puromycin for 1 week prior to performing experiments. Cloning was performed using the following oligos as PCR templates, as previously described (Paddison et al, Nature Methods 2004).

Luci.1309 shRNA
TCGAGAAGGTATATTGCTGTTGACAGTGAGCGCCCGCCTGAAGTCTCTGATTAATAG
TGAAGCCACAGATGTATTAATCAGAGACTTCAGGCGGTTGCCTACTGCCTCGG

MLL.10992 shRNA
TCGAGAAGGTATATTGCTGTTGACAGTGAGCGAAAGGAAAGCATTACTGAGAAATA
GTGAAGCCACAGATGTATTTCTCAGTAATGCTTTCCTTCTGCCTACTGCCTCGG

MLL.13406 shRNA
TCGAGAAGGTATATTGCTGTTGACAGTGAGCGCCCGCGGTATTATCCTAATTTATAG
TGAAGCCACAGATGTATAAATTAGGATAATACCGCGGTTGCCTACTGCCTCGG

Human Primary transcript RT-qPCR primers (written 5' to 3')
PABPC1: TGCCCTTTATAACATCAAAATTCATG and GTAATATTGACTCTGAAAATGTCCTTCCT
SFRS7: TGCTCACTACACCCACACCAA and GGCGGTACGGAGGAGGTAA
RPL7: TTGACGAAGGCGAAGAAGCT and CGTTTTCACCACAGTATCAATGG
HOXB7: GCCTCGTTTGCGGTCAGT and GCCGGCGTTTTCTTTACTCA
GAPDH (mature): CCTGACCTGCCGTCTAGAAAA and CTCCGACGCCTGCTTCAC
PPIA: TTGGCACACTTCATGGTTATGTT and CCATTATGGCGTGTGAAGTCA
RPL41: TTGGCACACTTCATGGTTATGTT and CCATTATGGCGTGTGAAGTCA
MYC: CACCACCAGCAGCGACTCT and CAATGAAAATGGGAAAGGTATCCA
GTF2F1: CATCAGTGAGATTGTCCCCAGTAG and CCCAACAGCGGGTAAGTTGA

Mouse ChIP-qPCR primers *(written 5' to 3')*
Ivr16: TGGCCATTTTTACTATGTTAATTTTGC and TAGACTTGTCATGGTTATGGATTGG
Actb: AAAGCCGTATTAGGTCCATCTTGA and GGCCATTGAGGCGTGATC
Ubb: GGATGCCCCGCAGTTG and TCAAACTGCTAAACCTTTCCAAAA
Rps2: TGGACATCCGGACAGACACA and GGAAAAAAAGGAAATCAGCAGAAC
Pabpc1: GCCGGAGACTTTTATTTTTTTTCTT and TCTCTGGGTTCCTGCTTGGA
Tuba1c: GGGAGATCTTCGTCACCCTTT and GCCATACTCACCATATTTAGTCCTTGA
Rpl28: GCCTCCTTTGCTCACCCTTT and GCAGCACCGTTGGGAAAA
Rps23: GCGGGCTCGGTTTGC and TTCGGAGACCACGACACTTG

Human ChIP-qPCR primers *(written 5' to 3')*
CD4: TGTGCTCTGCCCAGTTGTCT and GCTCATGACCAGTTCCAAGAGAA
UBB: GCGCGTTGACGGAAACTAAC and CCTTCCCCGCGAAGTAAAC
PABPC1: CAGCGGCAGTGGATCGA and GGACAAAAATCAACCGGAATTG
RPL19: GAGCAAACGGAGCGAACAAG and CCGGGTGTGCCAAGCA
RPL41: CGCCATGAGAGCCAAGGT and TCGCTACTACTCGCCAACTCCTA
RPLP0: GCTCCCTGTCTCTCCTCAGTGA and GCGGTGCGTCAGGGATT
PPIA: GCTCGTGCCGTTTTGCA and GGGTTGACCATGGCTAATAGTACA
PDE3A: CTCGGCGCTGCTCTTCA and GCGCGCAGGAGGTACAAG
MYC: CCGCATCCACGAAACTTTG and GGGTGTTGTAAGTTCCAGTGCAA
MYO1E: GCAACCCGGCCACAGA and GGGCCAAGGAAAACTTTATTCC
ROBO1: AAGAACCATCCAAAGCGAACA and GAGGCAGGAAACGGCACTAC

PAK2: GGCGGTGGCTCATTGTCT and GAGCTTCCCATTTCTGGCTAAA
NFIB: CCAGTCCTCCTTAAATAGCCAAAG and TGGATTTCACCCCTGGAAATC
PEG10: CCTGGTACACGCGCTTCAA and TCCTCAGGGCGCAGTCA
ZHX1: GCCGCCATCTTGCATTTC and GGTTTAGGGTTCACAGGTCAGAGT
HOXC11: GAGAACGATGTTTAACTCGGTCAAC and ATCTGCGCCCCTCTCCTT
EREG: CTGCTCTGCCTGGGTAAGTTCT and CCTTCCTTATTTGTCTCCTCTTTGG
ABCA5: CTGTTCAGACGGAAATTTACATCAA and GTCGTTCCACCCTCAGCAA
PDIA6: CCGGCTTAACATTTCAGTTTTTAAA and CCTGCACTGCTGCTTGTTTC
SRGAP1: GGGCTTCCTACAAGCCAGTTAG and GCTCCCCGTGGGTTTGA
ITGB8: CGGGCTTTGTTTGGGTTTG and GGGCTTCTGCTGCATAAATCA
LYCAT: CGTGCCCTGCTTGTCACA and TTTTCCTCGCGCCGTAAC

Human *PABPC1* ChIP profile primers *(written 5' to 3')*
PABPC1 -1.5 kb: AAAGTGCTGGAATTACAGGAGTGA and ATCCCACATAAATACTATTTTGCTCCTAT
PABPC1 -0.5 kb: TCCTCTCTGCTCTTTCCTCCTGTT and TCCTGCACCCTCTACTTATACCC
PABPC1 +0.5 kb: CAGCGGCAGTGGATCGA and TCCTGCACCCTCTACTTATACCC
PABPC1 +1 kb: TCCTCCTTTGCGACCATTTT and GGCGCGTCATCACCCTAA
PABPC1 +2 kb: TGACACAGAGCCAGAAGTTGTAAAA and TGGAGTTACTGCTTACAGAGCAAAA
PABPC1 +5 kb: CCCAGCCACTGGGAAGCT and TCACAGGCACAATCACAACACA

Human *RT-qPCR primers (mature transcripts) (written 5' to 3')*
PDE3A: GCATCAAACTGCAGGAAGCA and GTGAGGGTCATCATCACTGGATT
MYC: CGTCTCCACACATCAGCACAA and TCTTGGCAGCAGGATAGTCCTT
MYO1E: AGAGGTATGCCATTCTGACCAAA and GCAGGACGCCTTGCTTCTC
ROBO1: AATTAGAAGAGGAGGAAGATGAACGA and CACGGCAGCTGGAGAAGAAG
PAK2: GACTGCTCCTCCCGTTATTGC and GGGTCAATTACAGACCGTGTGTAA
NFIB: CCCTTGGAAAGTACCGATGGA and TGGACACAAAGTGCTGGGTTT
PEG10: TGTCATCGACTACTCCAATGCTTT and AATCAGCGCAGGCTCGTT
ZHX1: AACATGAAACTGCATTGGTAAACC and TTCTGCCAGTTGCTCTTTTGTC
HOXC11: GGCCGACGAGCTTATGCA and GCCTTCGTTTTTCATGAGGATCT
EREG: TGACATGAATGGCTATTGTTTGC and CCACTTCACACCTGCAGTAGTTTT
ABCA5: TGAAGATGTCAAAGCTGAAAGACTAAA and TGGATGGTTTCTCCTCACAACA
PDIA6: TGTGGACACTGCAAAAACCTAGA and CCTTTCGTCTGCTCTTTTACTTCTG
SRGAP1: TCCGCAAACTCCTCCTGACT and TGATTGAGGAAGGCAAAGAGGTA
ITGB8: GAGAGTTGCAAGTCACACAAGGA and TGAACATTTCCCACAAACACAAA
LYCAT: CTGTGGCGTATCCTCACAACA and TGGATTTCCCTGGGAAAGTCT
GAPDH: CCTGACCTGCCGTCTAGAAAA and CTCCGACGCCTGCTTCAC

Human *HOXC* primers *(written 5' to 3')*
A (chr 7 control region): GGCCTGTGCCTTGCCTTT and GCAGCACCTCAAGCCATTTC
B (HOXC11 upstream region): CCCTGGCAGCTCCTCTTTG and GAGCACCACAGGACACATG
C (HOXC11 +0.5 kb region): GAGAACGATGTTTAACTCGGTCAAC and ATCTGCGCCCCTCTCCTT
D (HOXC11 +2 kb region): TGCTCTCGCCAGATTTCAC and GCCGCCCTCCTAGTGGTT
E (HOXC10 upstream region): GCAAACCAGGTGTCTGAAAGG and TCGAGAAATTAGCCCAAGGTAGT
F (HOXC10 +0.5 kb region): CGCCTATCGCCTGGAACA and GCAGACATTCTCCTCCTTGACA
G (HOXC10 +2 kb region): GCAAGGGCAGAGCCTCATC and GCGCTCATCTTCCAGTCAGT
H (HOXC9 promoter 1 region): AAGGGCGCTTTGCTTCTGTT and CCTGGAGCCGGAAACTCA
I (HOXC9 +0.5 kb region): GGTTTGGTGCCGGACTGTAG and CCACGACGTGCTGAACAC
J (HOXC9 +2 kb region): ACGCTGGAACTGGAGAAGGA and CCGCTCGGTGAGATTGAGAAC
K(HOXC9 downstream): CAGTGCTGGTGGCAGACTT and GGTGCTTTGAGCAGTTCTTGTG
L (HOXC distal downstream): CAGTGCTGGTGGCAGACTT and GGTGCTTTGAGCAGTTCTTGTG

Mouse HoxA ChIP primers *(written 5' to 3')*
Hoxa6 promoter: TCAGCTGGTGCGATTTGCT and AGTCATCGAACTGGTTTGTTTCTG
Hoxa6 +2 kb: AGACACTGGAGCTTGAGAAGGAAT and GCGCTCGGTGAGGCAAAG
Hoxa7 promoter: CCATTGTGAAGTCGGGTTTATGA and CAGGCCATGCTGGAAGACT

Hoxa7 +3 kb: GGGACGCCAGACCTACAC and CGTCAGGTAGCGGTTGAAATGG
Hoxa9 promoter: GCACTGGACTTGAGCTGTAGTTTG and CCCCTGCCTTGGTTATCCTT
Hoxa9 +0.5 kb: CGCCCTGGCCCACTTC and ACCCGCTGGCTAGGGAATA
Hoxa9 +3 kb: GAGATGAGGCCTGGGATTTAGA and TGTCTCCTCTCCCCCAAACC
HoxA downstream: GAGGAGAATGACGCCCTTTCATAC and GTGTCACGAGTTTGTGCATATGTG

Mouse HoxA RT-qPCR primers *(written 5' to 3')*
Hoxa6: TCTCCCGAGCAGCAGTACAA and CTTCCTCATGGAGGGCTTTG
Hoxa7: AGTTCAGGACCCGACAGGAA and CAGGTAGCGGTTGAAATGGAA
Hoxa9: CCGAAAACAATGCCGAGAA and CCGGGTTATTGGGATCGAT
Gapdh: TTCACCACCATGGAGAAGGC and CCCTTTTGGCTCCACCCT

Human MYC ChIP profile primers *(written 5' to 3')*
MYC upstream: TCTTTGATCAGAATCGATGCATTT and TCCTTTCCAGGTCCTCTTTCC
MYC P1 promoter +0.5: GCGCCCATTAATACCCTTCTT and AGGGCCGCGCTTTGAT
MYC P2 promoter +0.5: CCGCATCCACGAAACTTTG and GGGTGTTGTAAGTTCCAGTGCAA
MYC intron: AAAGGGAGGCGAGGATGTG and TCGCCCTGGTTTTTCCAA
MYC middle exon: AAAACATCATCATCCAGGACTGTATG and GGAGGCCAGCTTCTCTGAGA
MYC last: CCTTTCTTAAAGAGGAGGAACAAGAA and GGAGCCTGCCTCTTTTCCA