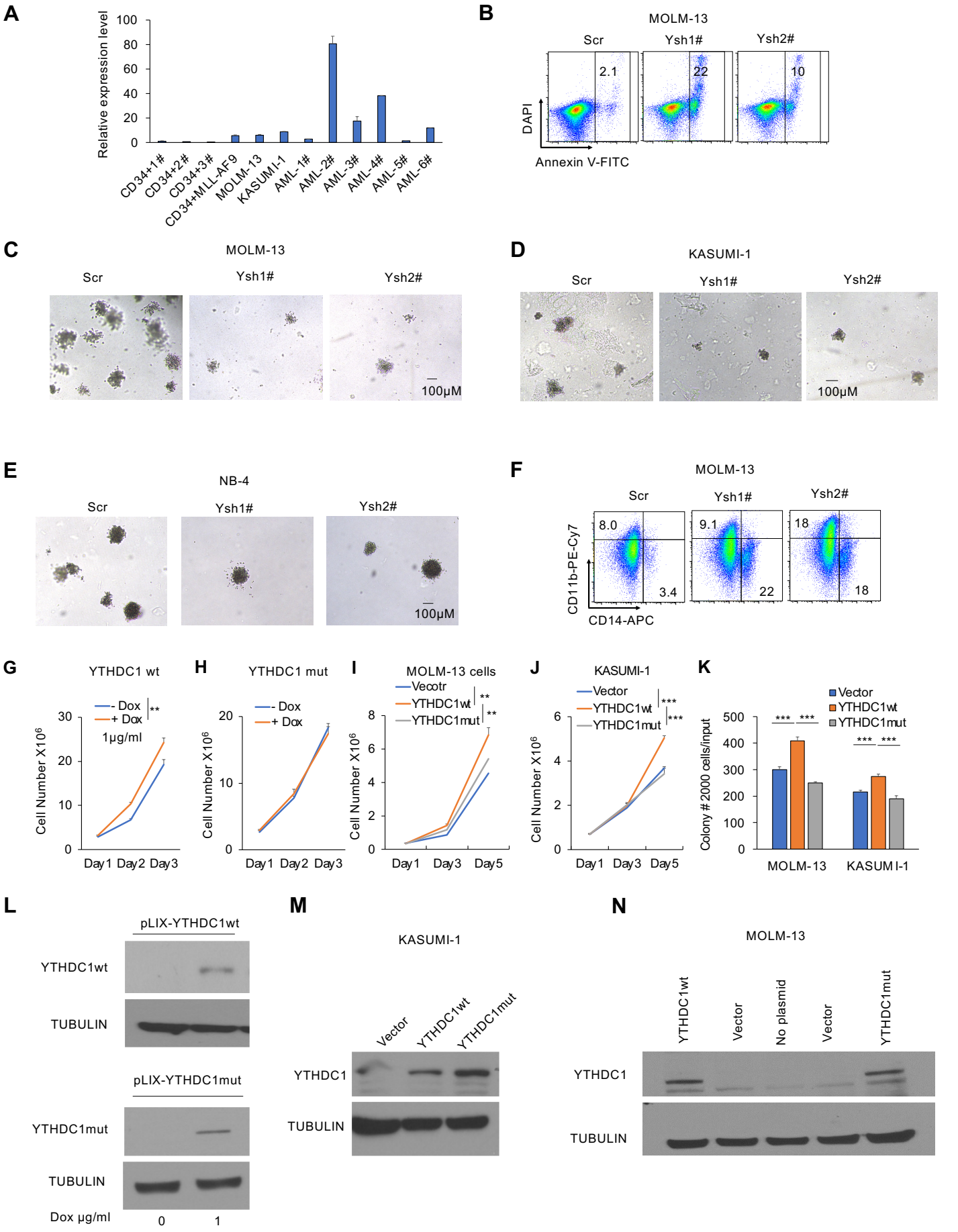
Supplemental Figure 1



Supplemental Figure 1. YTHDC1 KD inhibits proliferation and promotes apoptosis and differentiation of human AML cells.

(A) Relative YTHDC1 expression level in $CD34^+$ cells,$CD34^+$ cells expressed MLL-AF9, AML patient cells, MOLM-13 and KASUMI-1 cells, as determined by quantitative(q) RT-PCR. $CD34^+$ cells were collected from three independent individuals; AML cells were collected from six independent individuals. $CD34^+$ 1# cell expression was set as 1.

(B) Gating strategy for flow cytometry analysis of apoptosis frequency of MOLM-13 cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).

(C-E) Colony morphology of MOLM-13 (C), KASUMI-1 (D) and NB-4 (E) cells expressed Scramble shRNA(Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#). Colony photos were taken after 7 days of plating.

(F) Gating strategy for flow cytometry analysis of differentiated cell frequency of MOLM-13 cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).

(G-H) Growth curve of MOLM-13 cells transduced pLIX-402-Tet on- YTHDC1 wildtype (wt) (G) or YTHDC1 W377A W428A mutant (mut) (H) with or without 1 μg/ml Doxycycline (Dox), p value was calculated at Day 3.

(I-J) Growth curve of MOLM-13 (I) and KASUMI-1 (J) cells expressed vector, YTHDC1 wt or YTHDC1 mut, p value was calculated at Day 5.

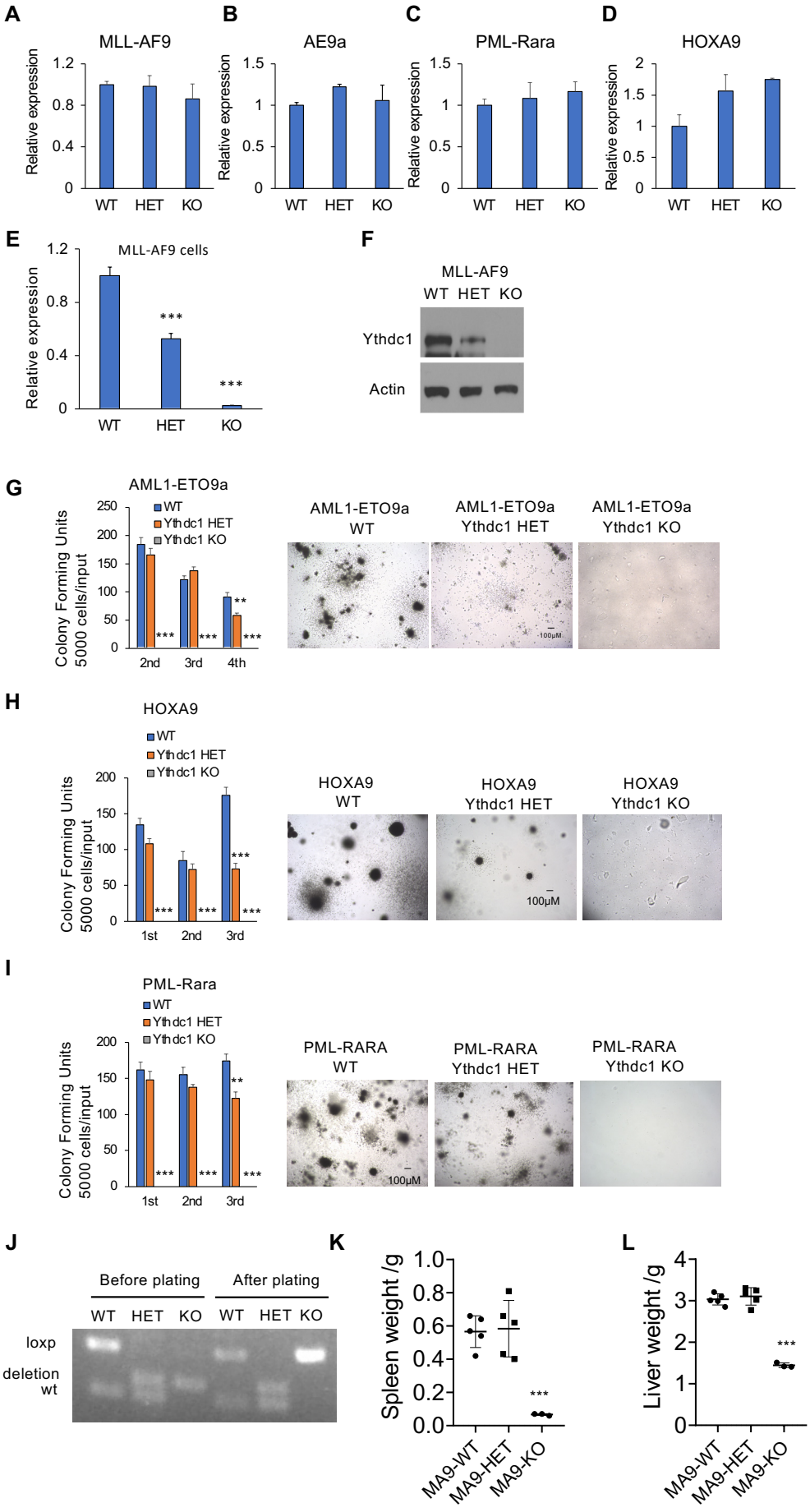
(K) Colony forming units of MOLM-13 and KASUMI-1 cells expressed vector, YTHDC1 wt or YTHDC1 mut. Colony number was counted 7 days after plating.

(L) Western blot analysis of Flag-YTHDC1 in MOLM-13 cells transduced pLIX-402-Tet on- YTHDC1 wildtype (wt) or YTHDC1 W377A W428A mutant (mut) with or without 1 μg/ml Doxycycline (Dox).

(M-N) Western blot analysis of Flag-YTHDC1 in KASUMI-1 (H) or MOLM-13 (I) cells expressed vector, YTHDC1 wt or YTHDC1 mut. TUBULIN was used as a loading control.

$^{**}p < 0.01$, $^{***}p < 0.001$, mean $\pm$ s.d., t-test.

Supplemental Figure 2



Supplemental Figure 2. Ythdc1 deficiency reduces colony-forming ability of AML cells driven by different fusion genes.

(A-D) Quantitative real time-PCR analysis of MLL-AF9(A), AML1-ETO9a (AE9a)(B), PML-Rara(C) and HOXA9(D) in WT, Ythdc1 HET and Ythdc1 KO cells expressed MLL-AF9, AML1-ETO9a (AE9a), PML-Rara and HOXA9, respectively.

(E-F) Quantitative real time-PCR (E) and western blot (F) analysis of Ythdc1 expression in WT, Ythdc1 HET and Ythdc1 KO cells expressed MLL-AF9.

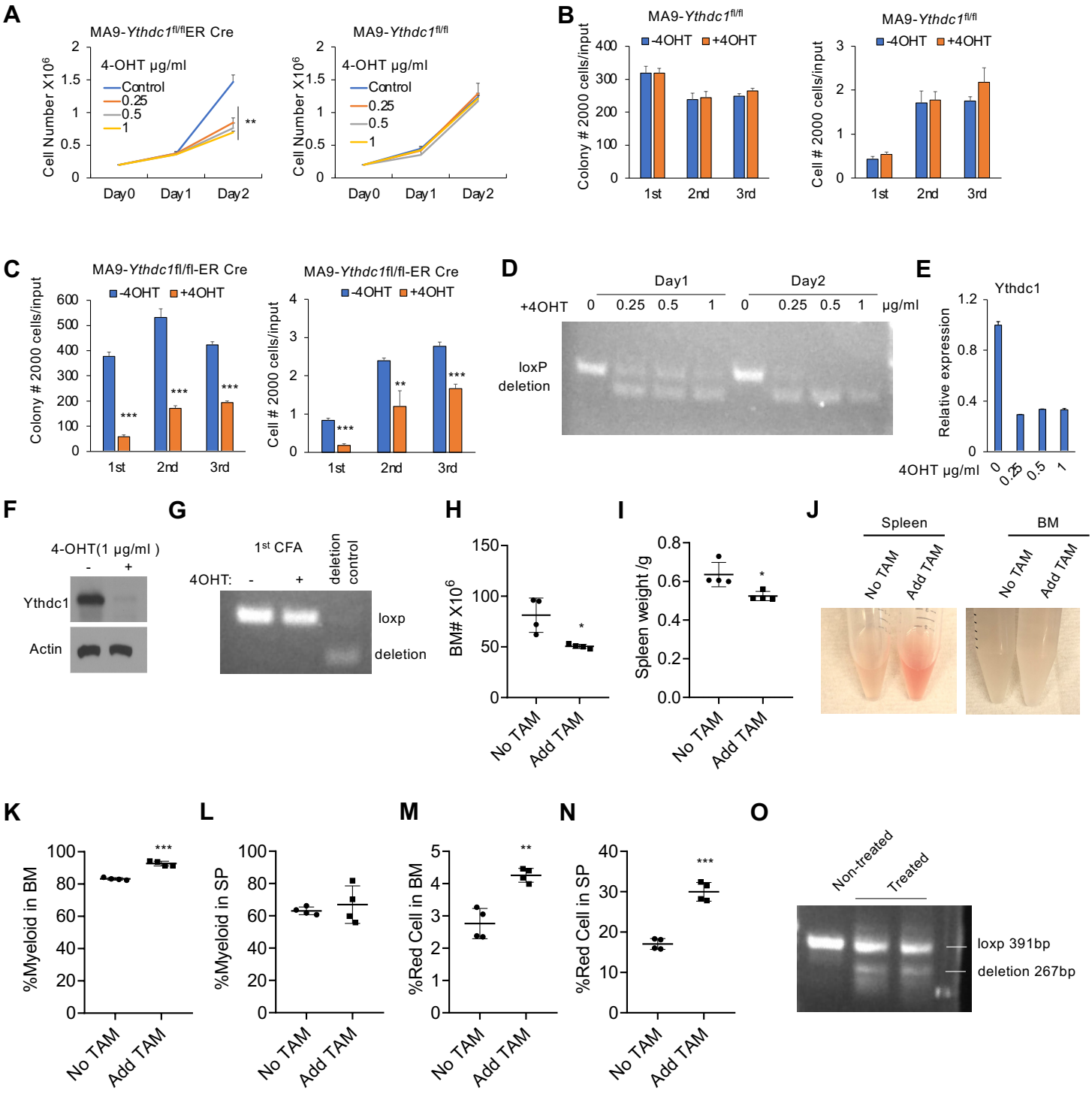
(G-I) Colony forming units of Lin^- BM cells from WT, Ythdc1 HET and Ythdc1 KO mice expressed AML1-ETO9a (G), HOXA9 (H) or PML-Rara (I), representative images of the colonies were displayed. Cells were resuspended and replated weekly in Methocult™ medium containing cytokines, bar = 100 μM.

(J) Semiquantitative PCR analysis of the deletion of Ythdc1 and loxP-flanked Ythdc1 allele (loxP) from MLL-AF9 colonies isolated from 1st plating. The cells before plating served as control.

(K-L) Spleen (D) and liver (E) weight of MLL-AF9-WT (MA9-WT), MLL-AF9-*Ythdc1* HET (MA9-HET) and MLL-AF9-*Ythdc1* KO (MA9-KO) mice, n = 5 mice for WT and HET group, n = 3 mice for KO group.

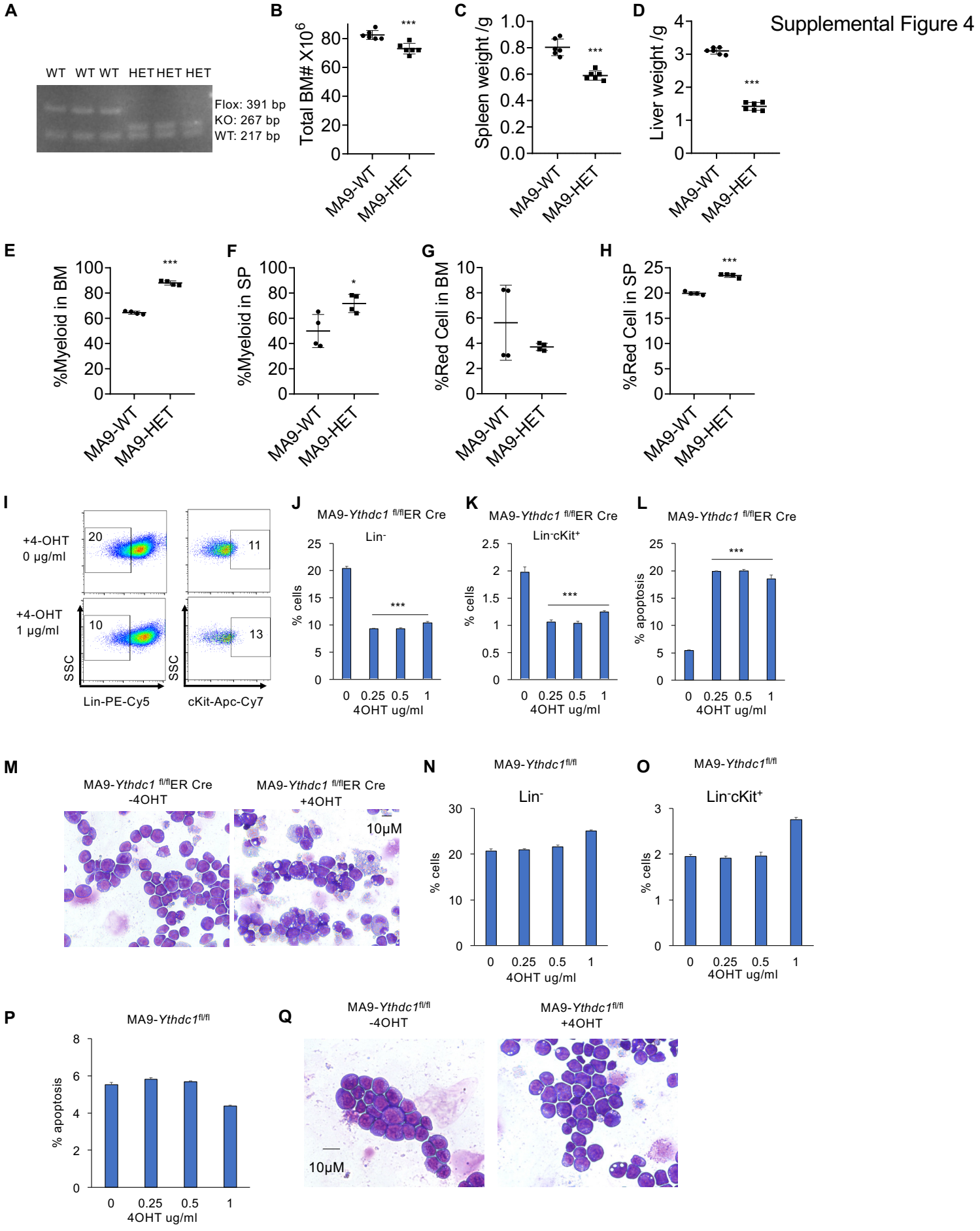
**p < 0.01, ***p < 0.001, mean ± s.d., t-test.

Supplemental Figure 3



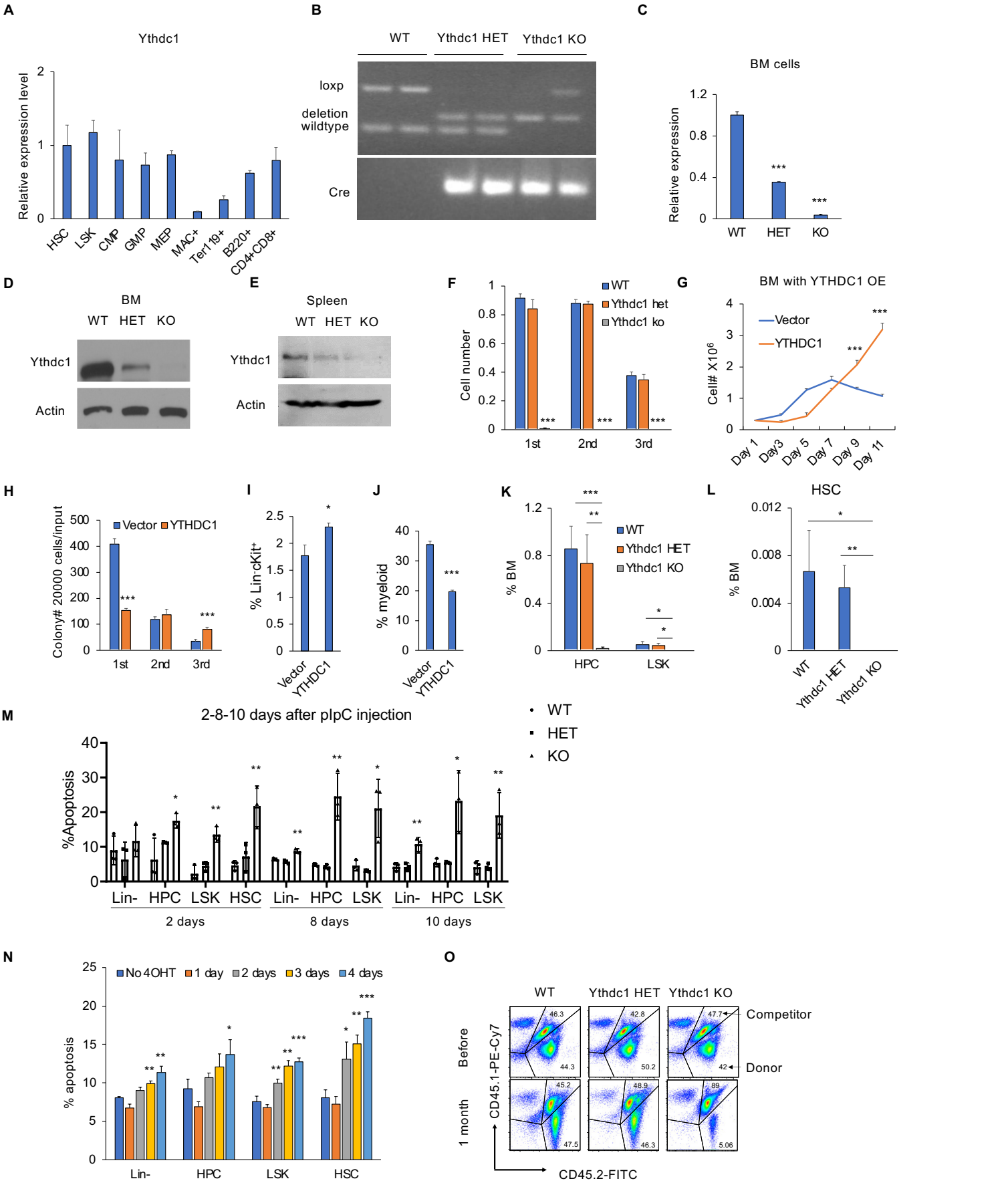
Supplemental Figure 3. Genetic deletion of Ythdc1 inhibits AML cell growth in vitro and in vivo.
(A) Growth curve of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells with or without ER Cre treated by 0.25, 0.5, 1 ug/ml 4-hydroxytamoxifen (4-OHT) or vehicle control, p value was calculated at Day 2 comparing to Control group.
(B) Colony forming units and cell number of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells without ER Cre treated by 1 ug/ml 4-OHT or vehicle control, cells were resuspended and replated weekly in methocult™ medium containing cytokines.
(C) Colony forming units and cell number of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells with ER Cre treated by 1 ug/ml 4-OHT or vehicle control, cells were resuspended and replated weekly in methocult™ medium containing cytokines.
(D) Semiquantitative PCR analysis of the deletion of Ythdc1 and loxP-flanked Ythdc1 allele (loxP) from of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells with ER Cre treated by 0, 0.25, 0.5, 1 ug/ml 4-OHT.
(E-F) Ythdc1 expression level in MLL-AF9-*Ythdc1*$^{fl/fl}$ cells with ER Cre treated by 0, 0.25, 0.5, 1 ug/ml 4-OHT after two days treatment, as determined by quantitative(q) RT-PCR (E) and western blot (F).
(G) Semiquantitative PCR analysis of the deletion of Ythdc1 and loxP-flanked Ythdc1 allele (loxP) from 1st colony forming assay of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells with ER Cre treated by 1 ug/ml 4-OHT or vehicle control.
(H) Total BM cell number of MLL-AF9-*Ythdc1*$^{fl/fl}$ ERCre mice with or without Tamoxifen (TAM) treatment, n = 4 for each group.
(I) Spleen weight of MLL-AF9-*Ythdc1*$^{fl/fl}$ ERCre mice with or without Tamoxifen (TAM) treatment, n = 4 for each group.
(J) Suspension cells of spleen and BM from MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre mice with or without Tamoxifen (TAM) treatment.
(K-L) Flow cytometric analysis of myeloid cell (Mac^{+}Gr1^{+}) percentage in BM (K) and spleen (SP) (L) from of MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre mice with or without Tamoxifen (TAM) treatment, n = 4 for each group.
(M-N) Flow cytometric analysis of red cell (Ter119^{+}) percentage in BM (M) and spleen (SP) (N) from MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre mice with or without Tamoxifen (TAM) treatment, n = 4 for each group.
(O) Semiquantitative PCR analysis of the deletion of Ythdc1 and loxP-flanked Ythdc1 allele (loxP) from BM cells isolated from MLL-AF9-*Ythdc1*$^{fl/fl}$ ERCre mice with or without Tamoxifen (TAM) treatment when mice became moribund.
*$p < 0.05$, **$p < 0.01$, ***$p < 0.001$, mean $\pm$ s.d., t-test.

Supplemental Figure 4



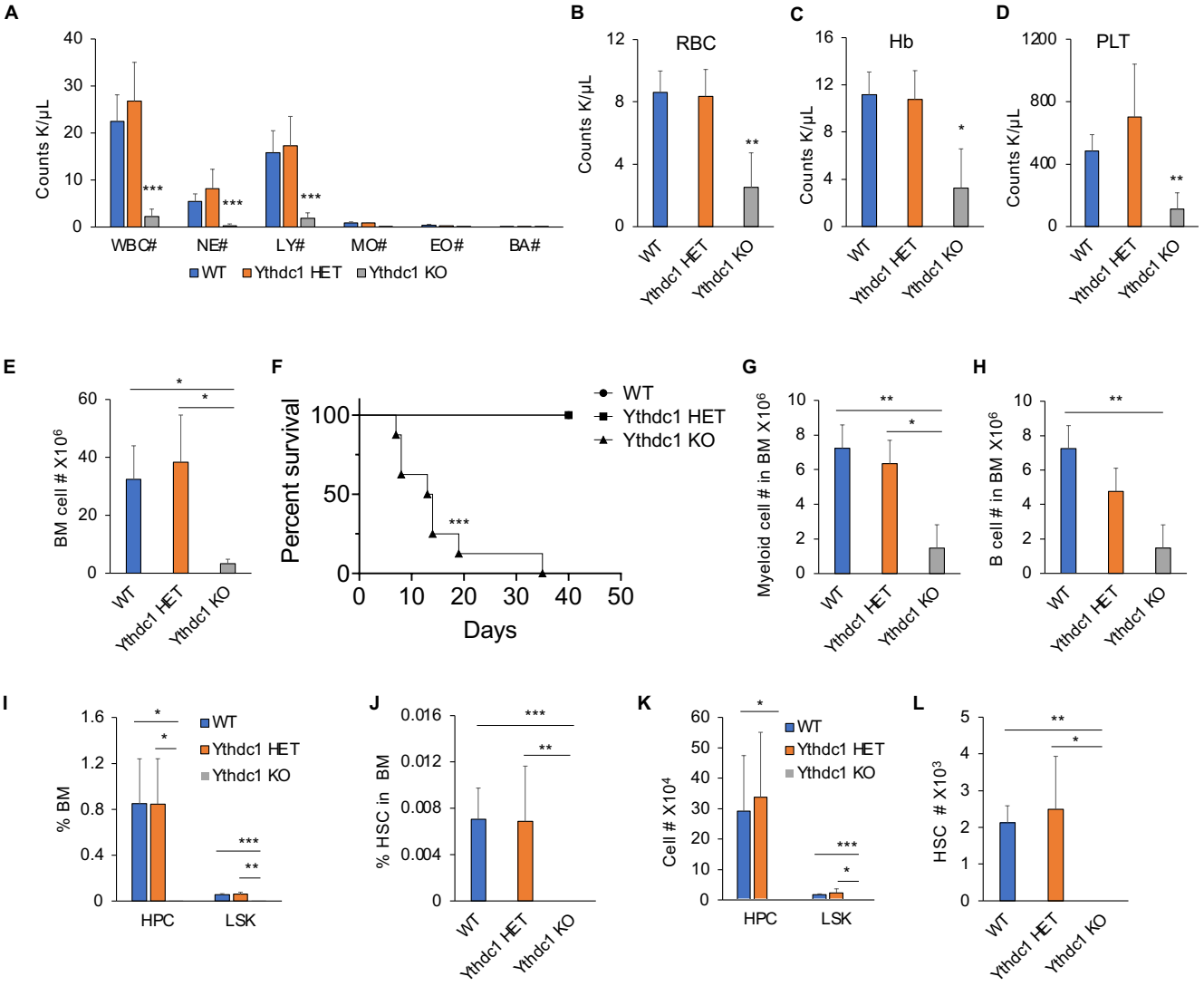
Supplemental Figure 4. Ythdc1 is required for AML maintenance in vivo.
(A) Semiquantitative PCR analysis of the deletion of Ythdc1 and loxP-flanked Ythdc1 allele (loxP) from BM cells isolated from MLL-AF9-WT (MA9-WT) and MLL-AF9-*Ythdc1* HET (MA9-HET) leukemic mice.
(B-D) Total BM number (B), spleen weight (C) and liver weight (D) of the secondary MLL-AF9-WT (MA9-WT) and MLL-AF9-*Ythdc1* HET (MA9-HET) mice, n = 6 mice for each group.
(E-F) Flow cytometric analysis of myeloid cell (Mac^+Gr1^+) percentage in BM (E) and SP (F) from the secondary MLL-AF9-WT (MA9-WT) and MLL-AF9-*Ythdc1* HET (MA9-HET) mice, n = 4 mice for each group.
(G-H) Flow cytometric analysis of red cell ($Ter119^+$) percentage in BM (G) and SP (H) from the secondary MLL-AF9-WT (MA9-WT) and MLL-AF9-*Ythdc1* HET (MA9-HET) mice, n = 4 mice for each group.
(I-K) Flow cytometric analysis of percentage of Lin^- (J) and Lin^-cKit^+ (K) in MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated with 0, 0.25, 0.5, 1 ug/ml 4-OHT after 2 days. Gating strategy was shown in I.
(L) Flow cytometric analysis of apoptosis frequency in MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated with 0, 0.25, 0.5, 1 ug/ml 4-OHT after 2 days.
(M) Wright-Giemsa-staining of MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT after 2 days.
(N-O) Flow cytometric analysis of percentage of Lin^- (N) and Lin^-cKit^+ (O) in MLL-AF9-*Ythdc1*$^{fl/fl}$ cells treated with 0, 0.25, 0.5, 1 ug/ml 4-OHT after 2 days.
(P) Flow cytometric analysis of apoptosis frequency in MLL-AF9-*Ythdc1*$^{fl/fl}$ cells treated with 0, 0.25, 0.5, 1 ug/ml 4-OHT after 2 days.
(Q) Wright-Giemsa-staining of MLL-AF9-*Ythdc1*$^{fl/fl}$ cells treated 1 ug/ml 4-OHT or vehicle control after 2 days.
*$p < 0.05$, ***$p < 0.001$, mean $\pm$ s.d., t-test.

Supplemental Figure 5

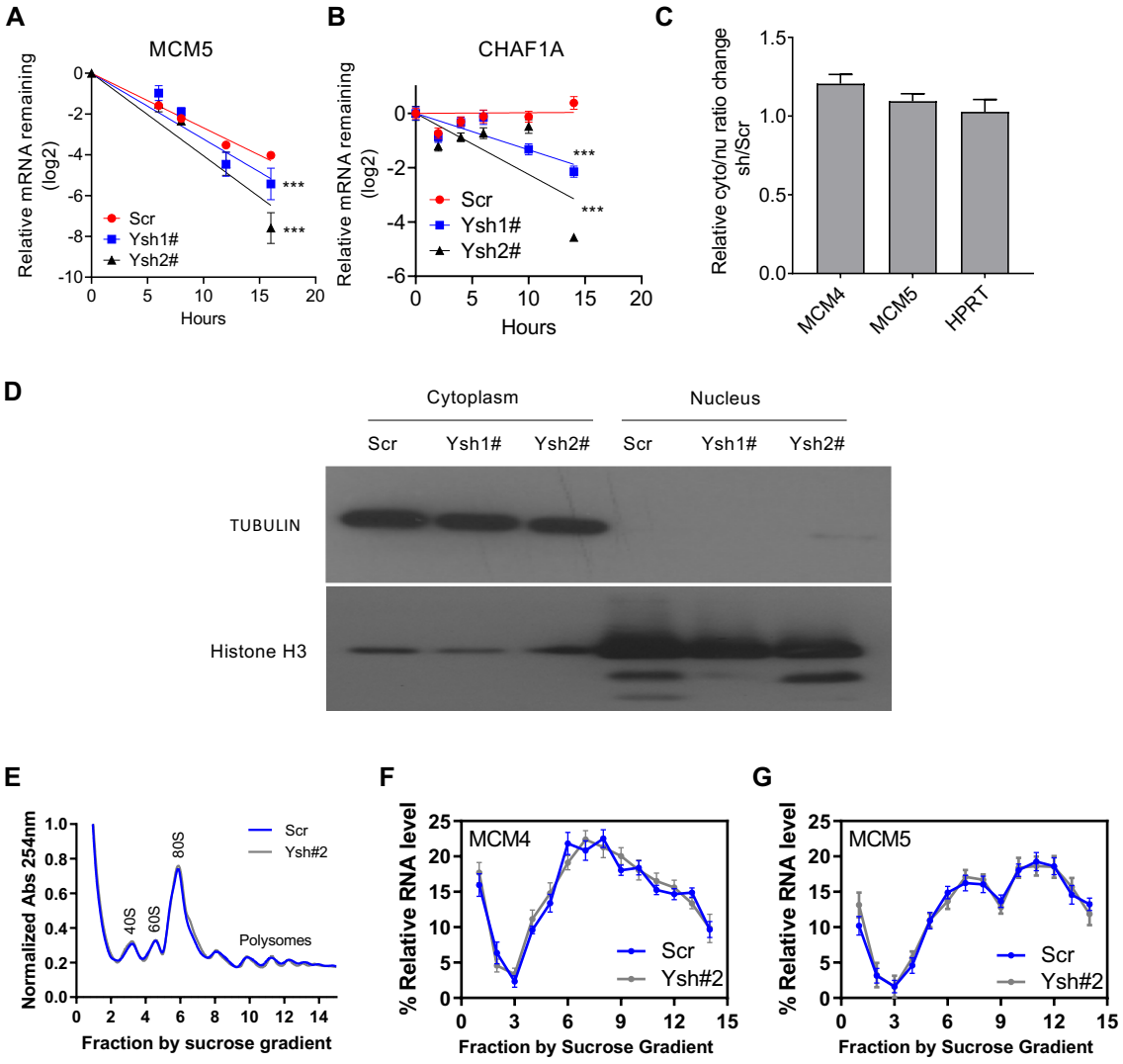


Supplemental Figure 5. Ythdc1 regulates HSC self-renewal.
(A) Ythdc1 expression level in stem cell and mature cell populations, as determined by quantitative(q) RT-PCR.
(B) Semiquantitative PCR analysis of Cre and the deletion of Ythdc1, loxP-flanked Ythdc1 allele (loxP) and wildtype (wt) among genomic DNA in BM cells from *Ythdc1*$^{fl/+}$ (WT), *Ythdc1*$^{fl/+}$Mx1-Cre (*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, deletion was induced by pIpC injection.
(C) Quantitative real time-PCR analysis of Ythdc1 expression in WT, HET and KO BM cells.
(D-E) Western blot analysis of Ythdc1 in BM (D) or spleen (E) cells isolated from *Ythdc1*$^{fl/+}$ (WT), *Ythdc1*$^{fl/+}$Mx1-Cre (HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (KO) mice, deletion was induced by pIpC injection. Actin was served as inner control.
(F) Cell number of the colonies of BM cells from *Ythdc1*$^{fl/+}$ (WT), *Ythdc1*$^{fl/+}$Mx1-Cre (*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, cells were resuspended and replated weekly in methocult™ medium containing cytokines, 10000 cells for 1st input, 50000 for 2nd and 3rd input.
(G) Cell growth curve of Lin$^-$ BM cells expressed vector or YTHDC1, cell number was counted every two days.
(H) Colony forming units of Lin$^-$ BM cells expressed vector or YTHDC1, cells were resuspended and replated weekly in methocult™ medium containing cytokines, 20000 cells for each plating.
(I) Flow cytometric analysis of the percentage of Lin$^-$cKit$^+$ population in vector or YTHDC1 overexpressed cells harvested from 1st plating colonies.
(J) Flow cytometric analysis of the percentage of myeloid population (Mac$^+$Gr1$^+$) in vector or YTHDC1 overexpressed cells harvested from 1st plating colonies.
(K-L) Flow cytometric analysis of HPC, LSK (K) and HSC (L) ratio in bone marrow (BM) cells from *Ythdc1*$^{fl/+}$ (WT), *Ythdc1*$^{fl/+}$Mx1-Cre (*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice 8-10 days after pIpC injection, n= 4 mice for each.
(M) Flow cytometric analysis of apoptosis rate in HSPCs from WT, HET and KO mice 2, 8 and 10 days after pIpC injection.
(N) Flow cytometric analysis of apoptosis rate BM-*Ythdc1*$^{fl/fl}$ ERCre cells with or without 1μg/ml 4-OHT treatment for indicated time points.
(O) Gating strategy for the competitive assay, donor cells: CD45.2$^+$CD45.1$^-$; competitor: CD45.2$^+$CD45.1$^+$; recipient cells: CD45.2$^-$CD45.1$^+$.
*p < 0.05, **p < 0.01, ***p < 0.001, mean ± s.d., t-test.

Supplemental Figure 6

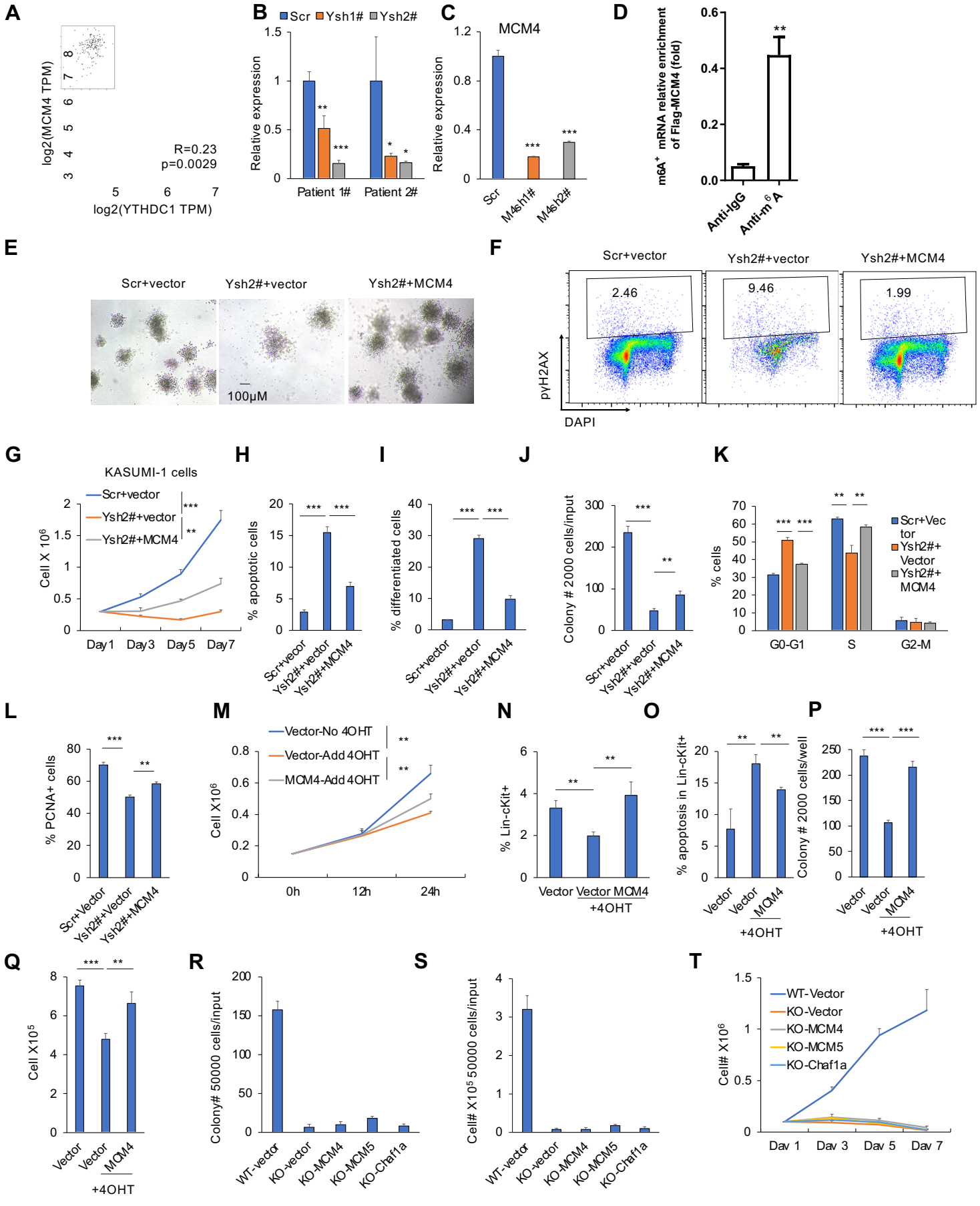


Supplemental Figure 6. Ythdc1 regulates HSC functions in an intrinsic manner.
(A-D) Absolute number of white blood cells (WBC), neutrophils (NE), lymphocytes (LY), monocytes (MO), eosinophils (EO) and basophils (BA) (A), as well as red blood cells (RBC) (B),and concentration of hemoglobin (C) and platelets (PLT) (D) in peripheral blood from the recipient mice reconstituted with *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) BM cells from the primary mice, the deletion was induced by pIpC one-month post-transplantation, n= 6 mice for WT and HET group, n = 7 mice for KO group.
(E) BM cell number of the transplanted *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, n = 3 mice for each group.
(F) Kaplan–Meier survival analysis of the transplanted *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, n= 10 mice for WT and HET group, n = 8 mice for KO group.
(G-H) Myeloid cell (Mac^{+}Gr1^{+}) (G) and B cell (B220^{+}) (H) number of the transplanted *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, n= 3 mice for each group.
(I-J) Flow cytometric analysis of HPC, LSK (I) and HSC (J) ratio in bone marrow (BM) cells from the transplanted *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, n= 3 mice for each group.
(K-L) Cell number of HPC, LSK (K) and HSC (L) in bone marrow (BM) cells from the transplanted *Ythdc1*$^{fl/+}$(WT), *Ythdc1*$^{fl/+}$Mx1-Cre(*Ythdc1* HET) and *Ythdc1*$^{fl/fl}$Mx1-Cre (*Ythdc1* KO) mice, n= 3 mice for each group.
*p < 0.05, **p < 0.01, ***p < 0.001, mean ± s.d., t-test or Log-rank (Mantel-Cox) Test for survival curve.

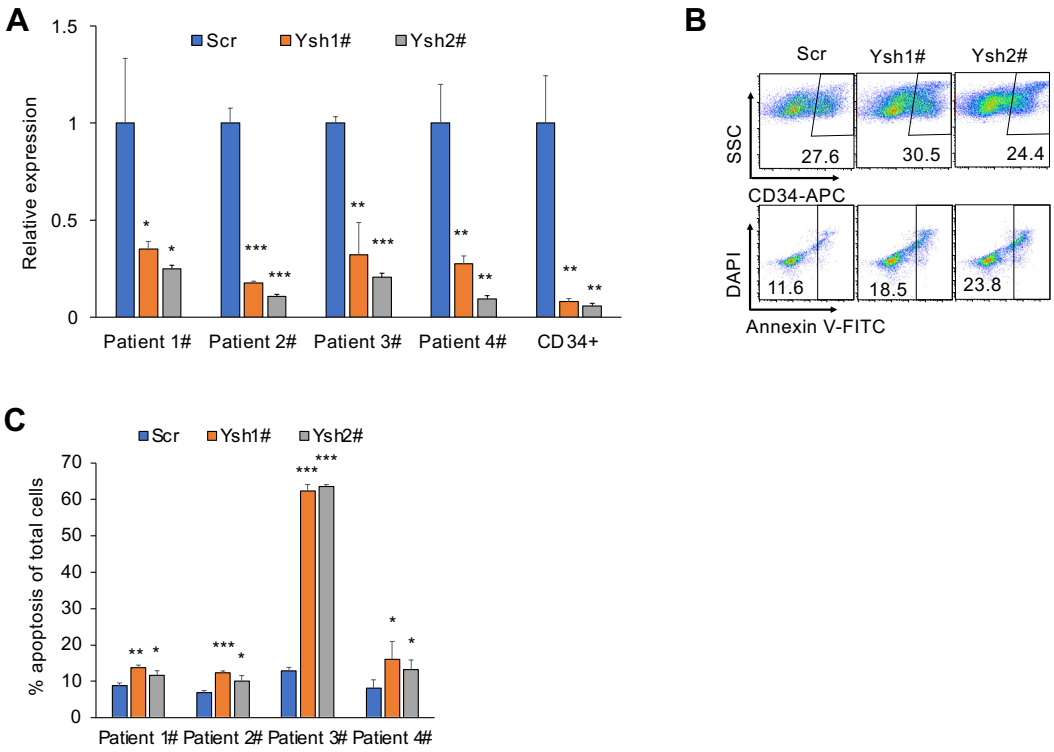


Supplemental Figure 7. YTHDC1 regulates stability of MCM5 and CHAF1A transcripts.
(A-B) The RNA stability of MCM5 (A) and CHAF1A (B) in MOLM-13 cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#). Experiments were performed in triplicate. The p value was detected by One-way ANOVA followed by multiple comparisons to Scr group 16 hours (for MCM5) or 14 hours (for CHAF1A) after actinomycin D treatment, ***$p < 0.001$, mean ± s.d..
(C) Quantitative RT-PCR analysis of MCM4 expression ratio of cytoplasm/nucleus in MOLM-13 cells expressed YTHDC1 shRNA2# (Ysh2#) compared to Scr group.
(D) Western blot to determine the protein levels of TUBULIN and Histone H3 in cytoplasm and nucleus separated from MOLM-13 cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).
(E-G) Polysome profiling assays. (E) Absorbance of different fractions of MOLM-13 cell lysates. (F-G) Quantitative RT-PCR analysis of MCM4(F) and MCM5(G) in each ribosome fraction and plotted as a percentage of the total. Data are from three biological repeats. Mean ± s.e.m. are shown.

Supplemental Figure 8



Supplemental Figure 8. MCM4 mediates YTHDC1 functions in AML cells.
(A) The expression correlation between MCM4 and YTHDC1 in AML patient cells from TCGA database.
(B) Quantitative RT-PCR analysis of MCM4 expression in AML patient cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).
(C) Quantitative RT-PCR analysis of MCM4 expression in MOLM-13 cells expressed Scramble shRNA (Scr), MCM4 shRNA1# (M4sh1#) or MCM4 shRNA 2# (M4sh2#).
(D) mRNA m6A methylation analysis of exogenously expressed MCM4 in MOLM-13 cells.
(E) The morphology of colony units of MOLM-13 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression. Photos was taken after 7 days of plating.
(F) The gating strategy for flow cytometric analysis of the percentage of p-γH2AX^{+} cells in MOLM-13 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression.
(G) Cell growth curve of KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression. The p value was detected by t-test between Scr+vector and Ysh2#+vector or Ysh2#+vector and Ysh2#+MCM4 at Day 7.
(H) Flow cytometric analysis of apoptosis frequency of KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression.
(I) Flow cytometric analysis of differentiated cell frequency of KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression.
(J) Colony forming units of KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression.
(K) Flow cytometric analysis of cell cycle in KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression. Cells were stained with BrdU (determine the S phase) and DAPI (determine the DNA content).
(L) Flow cytometric analysis of the percentage of PCNA^{+} cells in KASUMI-1 cells expressed Scramble shRNA (Scr) or YTHDC1 shRNA2# (Ysh2#) with or without MCM4 expression.
(M) Cell growth curve of MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT or vehicle control for indicated time with or without MCM4 expression, p value was calculated at 24 hours after 4-OHT treatment.
(N) Flow cytometric analysis of the percentage of Lin^{-}cKit^{+} population in MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT or vehicle control for 2 days with or without MCM4 expression.
(O) Flow cytometric analysis of the apoptosis rate of Lin^{-}cKit^{+} population in MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT or vehicle control for 2 days with or without MCM4 expression.
(P) Colony forming units of MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT or vehicle control for 7 days with or without MCM4 expression.
(Q) Cells number from colonies of MLL-AF9-*Ythdc1*$^{fl/fl}$ ER Cre cells treated 1 ug/ml 4-OHT or vehicle control for 7 days with or without MCM4 expression.
(R) Colony forming units of WT and Ythdc1 KO BM cells expressed vector, MCM4, MCM5 or Chaf1a.
(S) Cell number from colonies of WT and Ythdc1 KO BM cells expressed vector, MCM4, MCM5 or Chaf1a.
(T) Cell growth curve of WT and Ythdc1 KO BM cells expressed vector, MCM4, MCM5 or Chaf1a.
*$p < 0.05$; **$p < 0.01$, ***$p < 0.001$, mean $\pm$ s.d., t-test.



Supplemental Figure 9. YTHDC1 KD inhibits survival of human primary AML cells.
(A) Quantitative RT-PCR analysis of YTHDC1 expression in AML patient cells and CD34⁺ cells from healthy donor expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).
(B) Gating strategy for the flow cytometric analysis of apoptosis frequency of CD34⁺ cells in AML patients expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).
(C) Flow cytometric analysis of apoptosis frequency of AML patient cells expressed Scramble shRNA (Scr), YTHDC1 shRNA1# (Ysh1#) and YTHDC1 shRNA2# (Ysh2#).
*p < 0.05, **p < 0.01, ***p < 0.001, mean ± s.d., t-test.

Supplemental Table 1: Patient karyotype information

Patient karyotype information			
#	SOURCE	Barcode	karyotype
1	Northwestern University	17-0047	46,XX,t(9;11)(p22;q23)[20]
2	Northwestern University	15-1756	51,XX,+6,+8,t(9;11)(p22;q23),+der(9)t(9;11)(p22;q23),+14,+19[18]
3	City of Hope	2017_63	46, t(10;11), t(15;21)
4	University of Florida	LPP-1	46,XY(3),FLT3 pos, NPM-1 pos
5	University of Florida	LPP-3	MPO(-); weakly/partially NSE(+).
6	University of Florida	LPP-4	MLL rearrangment， (-) for FLT3 ITD and NPM-1 mutation

Supplemental Table 2: Primer list.

Genotyping		
Ythdc1 loxp-1	CATCTCTCCAGCCCGGTAAA	Flox: 391 bp
Ythdc1 loxp-2	GTGCTACACTAAGTCCTGTGAC	WT: 217 bp
Ythdc1 loxp-3	AGCTCAGACCATAACTTCGT	KO: 267 bp
Cre1	CTGCATTACCGGTCGATGCAAC	
Cre2	GCATTGCTGTCACTTGGTCGTG	Detect Cre allele (301bp)
shRNA		
hYTHDC1 scramble-F	CCGGAAATCGCTGATTTGTGTAGGGCTCGAGCCCTACACAAATCAGCGATTTTTTTTG	
hYTHDC1 scramble-R	AATTCAAAAAAAAATCGCTGATTTGTGTAGGGCTCGAGCCCTACACAAATCAGCGATTT	
Real-Time PCR		
m-rt-Ythdc1-F	GTCCACATTGCCTGTAAATGAGA	
m-rt-Ythdc1-R	GGAAGCACCCAGTGTATAGGA	
m-rt-Actin-F	TGTGATGGTGGGAATGGGTCAG	
m-rt-Actin-R	TTTGATGTCACGCACGATTTCC	
h-rt-YTHDC1-F	GAGGGCCAAATCTCCTACGC	
h-rt-YTHDC1-R	GTCTCATGGTCAGAGCCATATTC	
h-rt-CHAF1A-F	TTAGACCGAAACTTGTCAACGG	
h-rt-CHAF1A-R	GTCTGGCTGCTCATTCGAGT	
h-rt-MCM4-F	TGAACCTCTATACATGCAACGAC	
h-rt-MCM4-R	CAGGGTAACGGTCAAAGAAGATT	
h-rt-MCM2-F	ATGATCGAGAGCATCGAGAACC	
h-rt-MCM2-R	GCCAAGTCCTCATAGTTCACCA	
h-rt-MCM5-F	GGAAGTGCAACACAGATCAGG	
h-rt-MCM5-R	AGGGACGACCTTGTCACACA	
h-rt-BCL2-F	GGTGGGGTCATGTGTGTGG	
h-rt-BCL2-R	CGGTTCAGGTACTCAGTCATCC	
h-rt-APC-F	AAAATGTCCCTCCGTTCTTATGG	
h-rt-APC-R	CTGAAGTTGAGCGTAATACCAGT	
h-rt-RFC1-F	TTGAACGAGATGAGGCCAAGT	
h-rt-RFC1-R	ACTATCACGACCCATGACAAGAT	
h-rt-GAPDH-F	GAAATCCCATCACCATCTTCCAGG	
h-rt-GAPDH-R	GAGCCCCAGCCTTCTCCATG	