SUPPLEMENTAL DATA

Supplementary Figure 1. Association of BAF57 and BAF155 with PRMT7 target DNA repair genes. Cross-linked chromatin from control NIH 3T3/sh-GFP and NIH 3T3/sh-PRMT7-1 cells was immunoprecipitated with either pre-immune (PI), anti-BAF57, or anti-BAF155 antibody, and the purified DNA was checked for enrichment of promoter sequences of PRMT7 target genes using specific primers. Fold enrichment was determined relative to the PI sample.

Supplementary Figure 2. PRMT1 and PRMT4 are not recruited to PRMT7 target genes. (A-D) ChIP assays were carried out using chromatin from control NIH 3T3/sh-GFP and sh-PRMT7-1 cells as described in supplementary figure 1, and the immunoprecipitated DNA was analyzed by real time PCR using gene-specific primers. BIRC5 and MYC were used as positive controls.

Supplementary Figure 3. Anti- H2A(Me_2)R3 and anti-H4(Me_2)R3 antibodies are highly specific and do not cross-react with other histone peptides. Approximately 1 or 2 μg of either symmetrically methylated H2A (A) or H4 (B) peptides were slot-blotted on nitrocellulose, and methylation was detected using anti-H2A(Me_2)R3. To show specificity of the anti-H4(Me_2)R3 antibody, either 1 or 2 μg of symmetrically methylated H4 (C) or H2A (B) peptides were slot-blotted and detected by Western blot analysis using anti-H4(Me_2)R3 antibody. Unmethylated H2A, H4, and BSA were used as controls.

Supplementary Figure 4. NIH 3T3/sh-PRMT7-2 knock down cells are resistant to DNA damaging drugs. Drug treatments were carried out as described in figure 6 using an equal number (2×10^5) of control NIH 3T3/sh-GFP and NIH 3T3/sh-PRMT7-2 cells, and proliferation was monitored every two days for 6 days. Viability was determined by trypan blue staining cells, and each drug treatment experiment was conducted twice in triplicate.

Supplementary Figure 5. PRMT5 knock down cells do not show resistance to DNA-damaging drugs. (A-D) Drug treatments were carried out as described in experimental procedures using an equal number (2×10^5) of control NIH 3T3 and PRMT5 knock down cells (AS-PRMT5). Proliferation was monitored every two days for 6 days, and each experiment was conducted twice in triplicate.

Supplementary Figure 6. PRMT7 knock down cells are more resistant to cisplatin-induced DNA damage. An equal number (5×10^6) of control NIH 3T3/sh-GFP or NIH 3T3/sh-PRMT7-1 cells were treated with cisplatin (10 μg/ml) and harvested at days 0, 4 and 6. Cells were stained with FITC-Annexin V antibody and propidium iodide before they were analyzed by flow cytometry.

Supplementary Figure 7. Knock down of the polymerase delta catalytic subunit POLD1 re-sensitizes NIH 3T3/sh-PRMT7-2 knock down cells to DNA damage. (A-D) Growth rates of control NIH 3T3/sh-GFP, NIH 3T3/sh-PRMT7-2, and NIH 3T3/sh-PRMT7-2 cells, where expression of individual DNA repair genes has been knocked down, were measured by seeding 2×10^5 cells in each plate and treating cells with cisplatin as described in experimental procedures. The number of viable cells was determined by trypan blue staining every 2 days for 6 days. Each experiment was conducted in triplicate and repeated twice.

Figure 1

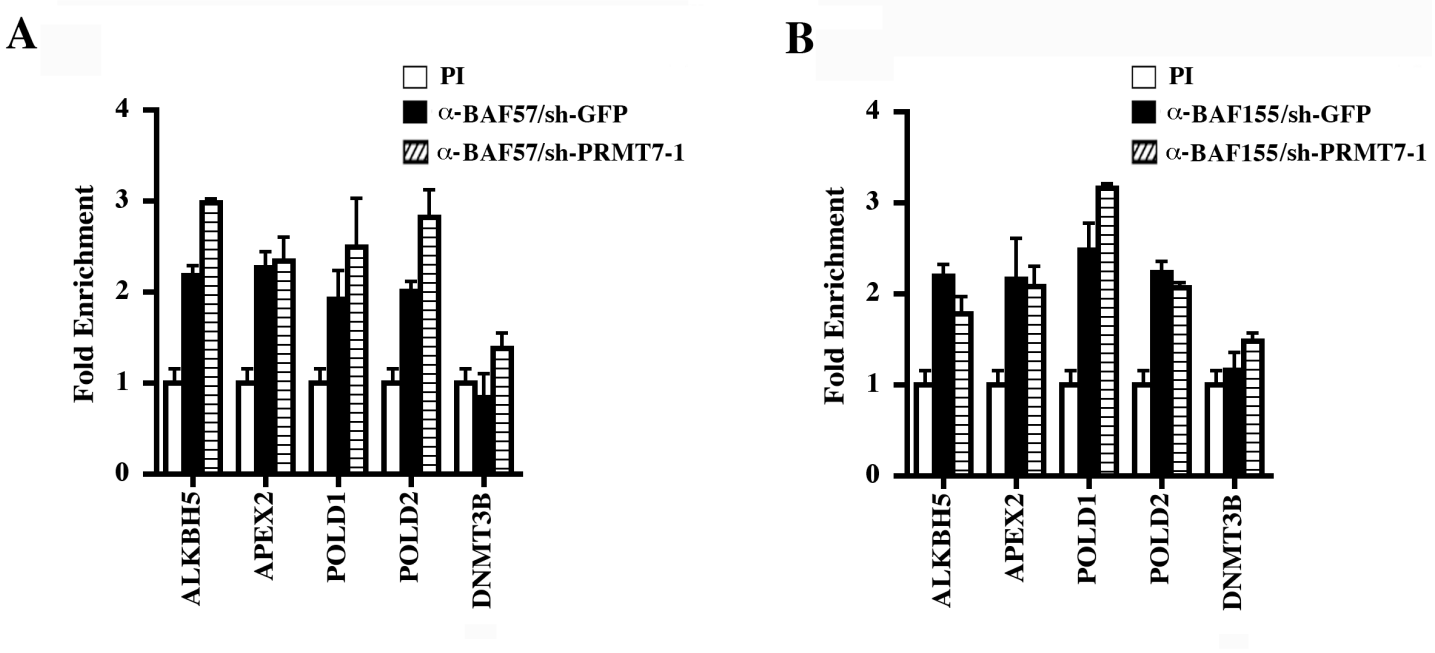

A
PI
α-BAF57/sh-GFP
α-BAF57/sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
ALKBH5
APEX2
POLD1
POLD2
DNMT3B
B
PI
α-BAF155/sh-GFP
α-BAF155/sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
ALKBH5
APEX2
POLD1
POLD2
DNMT3B

Figure 2

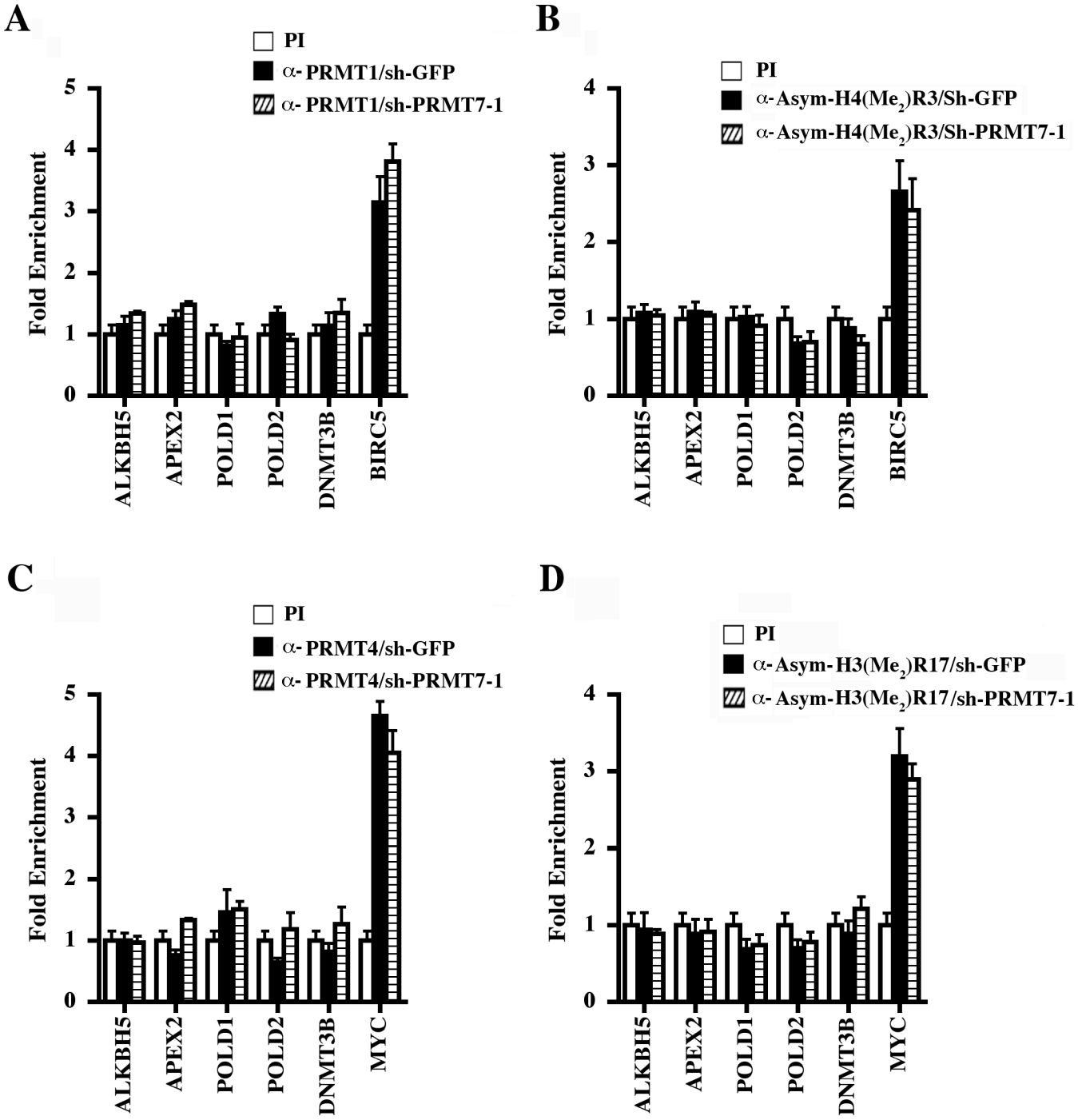

A
PI
α- PRMT1/sh-GFP
α- PRMT1/sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
5
ALKBH5
APEX2
POLD1
POLD2
DNMT3B
BIRC5
B
PI
α-Asym-H4(Me2)R3/Sh-GFP
α-Asym-H4(Me2)R3/Sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
ALKBH5
APEX2
POLD1
POLD2
DNMT3B
BIRC5
C
PI
α- PRMT4/sh-GFP
α- PRMT4/sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
5
ALKBH5
APEX2
POLD1
POLD2
DNMT3B
MYC
D
PI
α-Asym-H3(Me2)R17/sh-GFP
α-Asym-H3(Me2)R17/sh-PRMT7-1
Fold Enrichment
0
1
2
3
4
ALKBH5
APEX2
POLD1
POLD2
DNMT3B
MYC

Figure 3

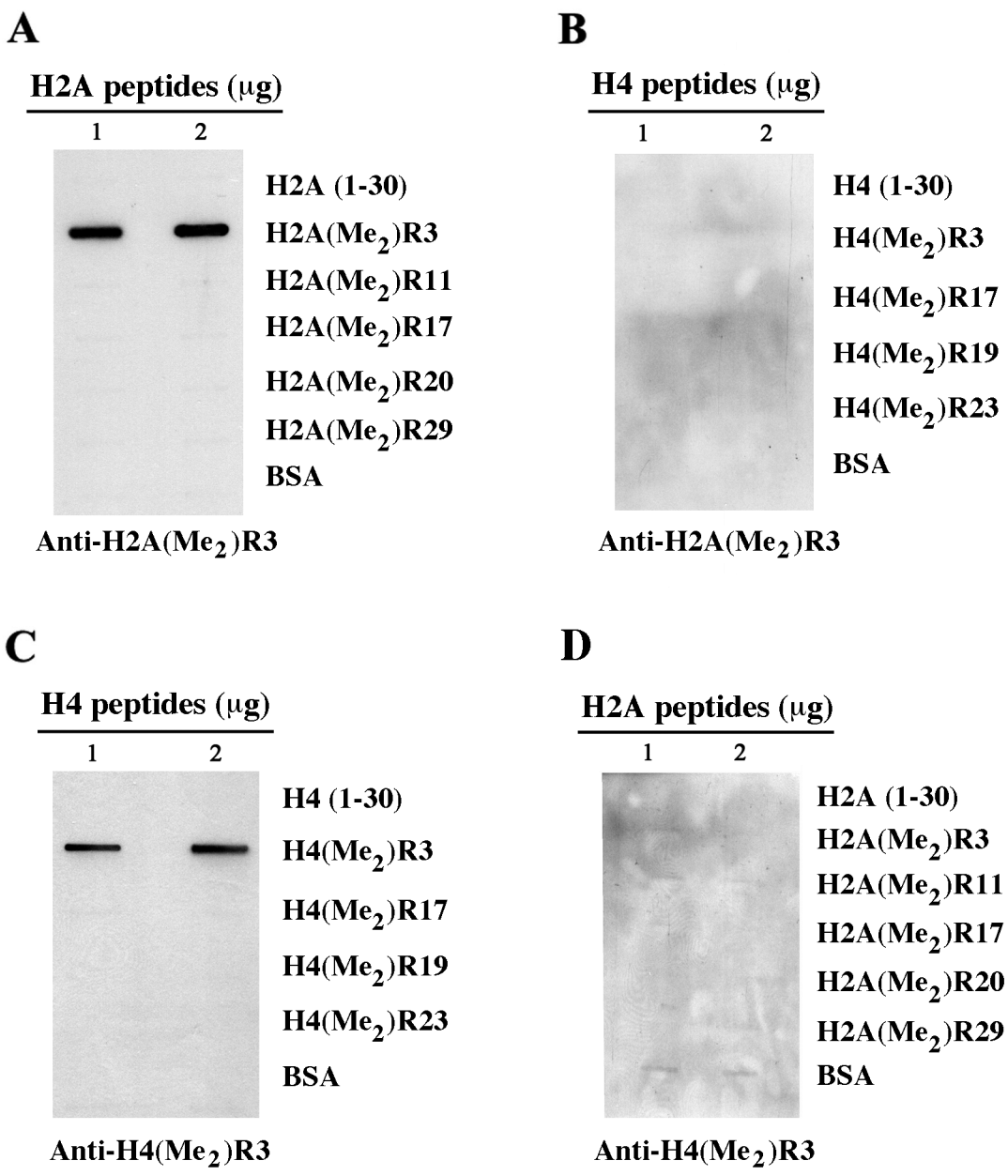

A
H2A peptides (μg)
1
2
H2A (1-30)
H2A(Me2)R3
H2A(Me2)R11
H2A(Me2)R17
H2A(Me2)R20
H2A(Me2)R29
BSA
Anti-H2A(Me2)R3
B
H4 peptides (μg)
1
2
H4 (1-30)
H4(Me2)R3
H4(Me2)R17
H4(Me2)R19
H4(Me2)R23
BSA
Anti-H2A(Me2)R3
C
H4 peptides (μg)
1
2
H4 (1-30)
H4(Me2)R3
H4(Me2)R17
H4(Me2)R19
H4(Me2)R23
BSA
Anti-H4(Me2)R3
D
H2A peptides (μg)
1
2
H2A (1-30)
H2A(Me2)R3
H2A(Me2)R11
H2A(Me2)R17
H2A(Me2)R20
H2A(Me2)R29
BSA
Anti-H4(Me2)R3

Figure 4

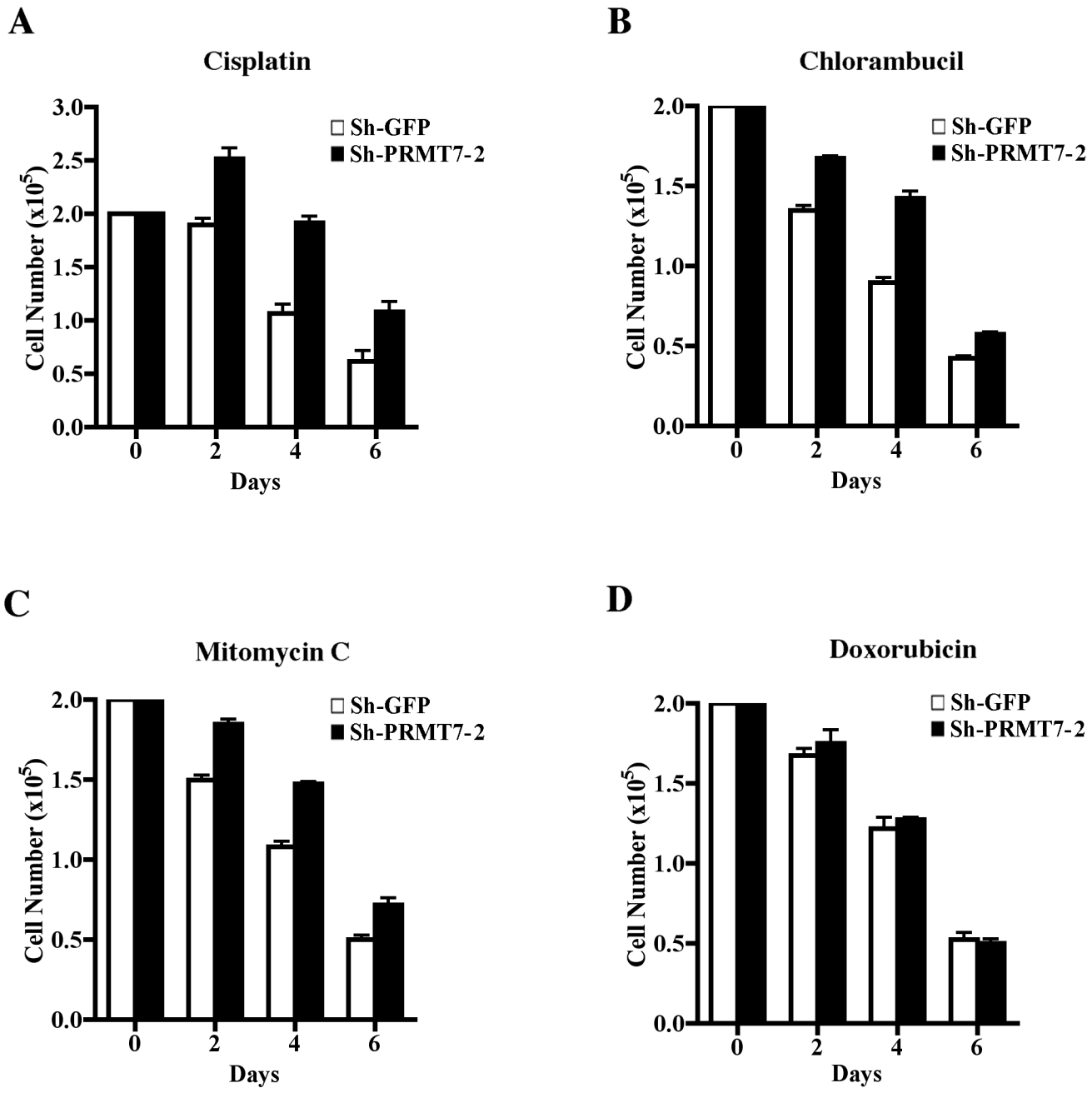

A
Cisplatin
Cell Number (x10^5)
3.0
2.5
2.0
1.5
1.0
0.5
0.0
0
2
4
6
Days
Sh-GFP
Sh-PRMT7-2
B
Chlorambucil
Cell Number (x10^5)
2.0
1.5
1.0
0.5
0.0
0
2
4
6
Days
Sh-GFP
Sh-PRMT7-2
C
Mitomycin C
Cell Number (x10^5)
2.0
1.5
1.0
0.5
0.0
0
2
4
6
Days
Sh-GFP
Sh-PRMT7-2
D
Doxorubicin
Cell Number (x10^5)
2.0
1.5
1.0
0.5
0.0
0
2
4
6
Days
Sh-GFP
Sh-PRMT7-2

Figure 5

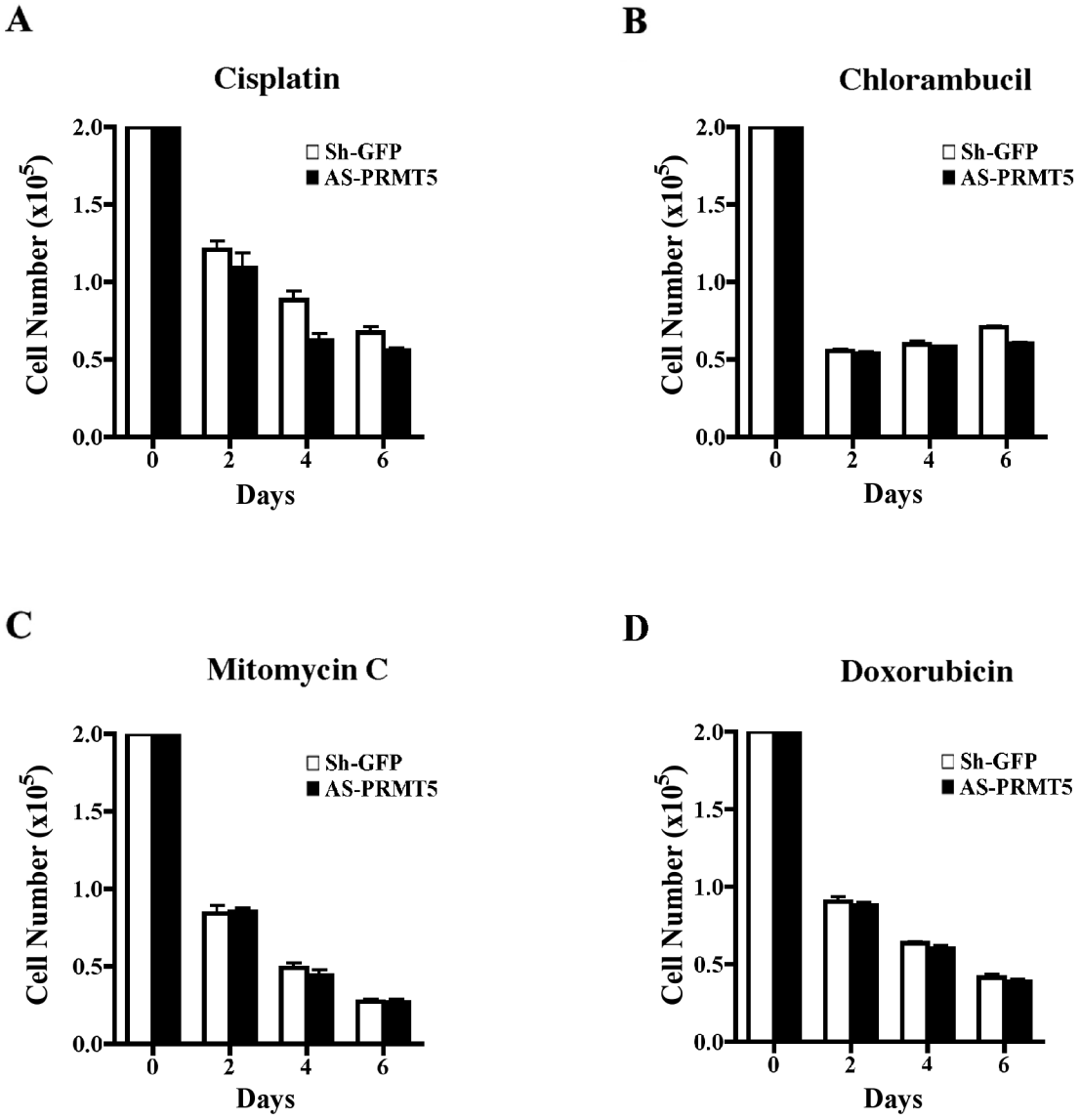

A
Cisplatin
Cell Number (x10^5)
2.0
1.5
1.0
0.5
0.0
0
2
4
6
Days
Sh-GFP
AS-PRMT5
B
Chlorambucil
Cell Number (x10^5)
Days
Sh-GFP
AS-PRMT5
C
Mitomycin C
Cell Number (x10^5)
Days
Sh-GFP
AS-PRMT5
D
Doxorubicin
Cell Number (x10^5)
Days
Sh-GFP
AS-PRMT5

Figure 6

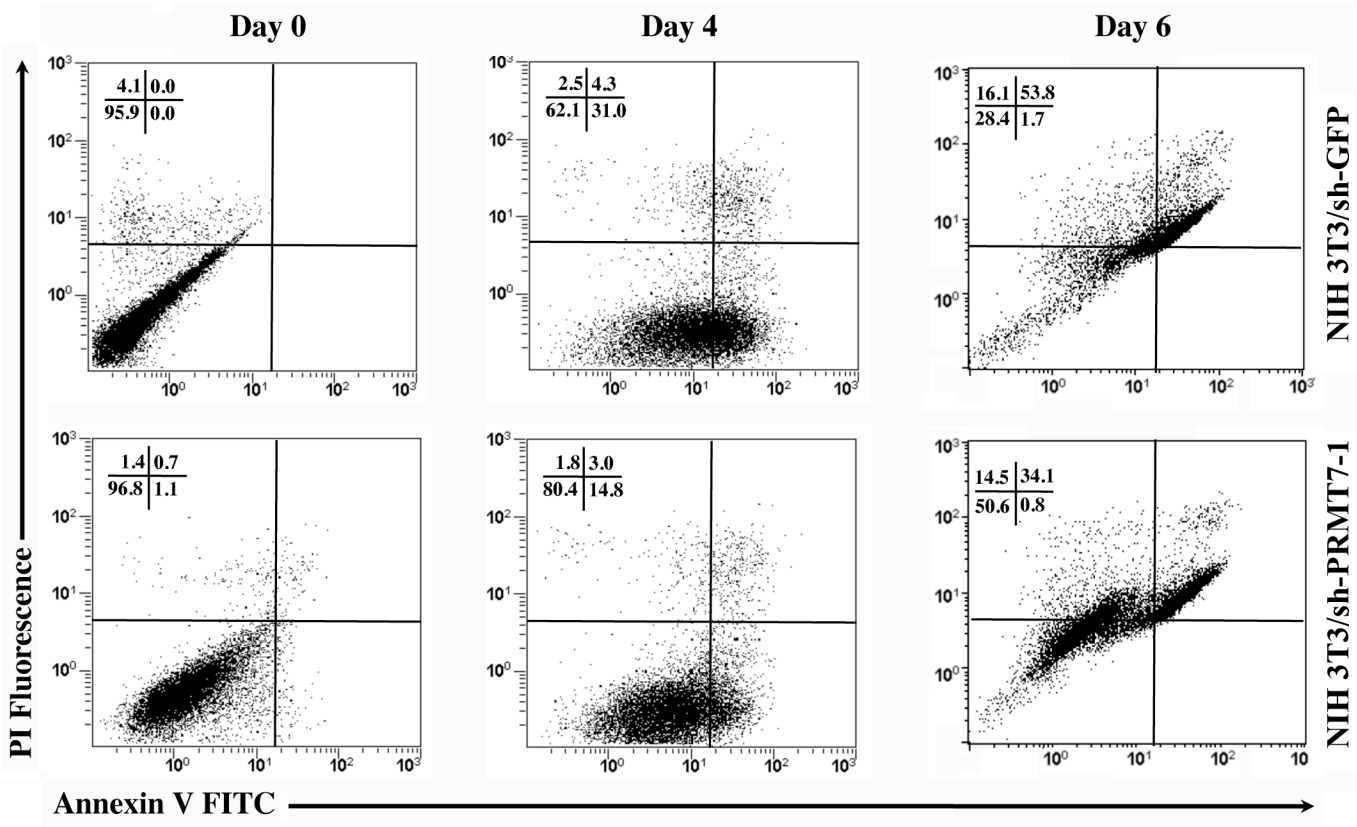
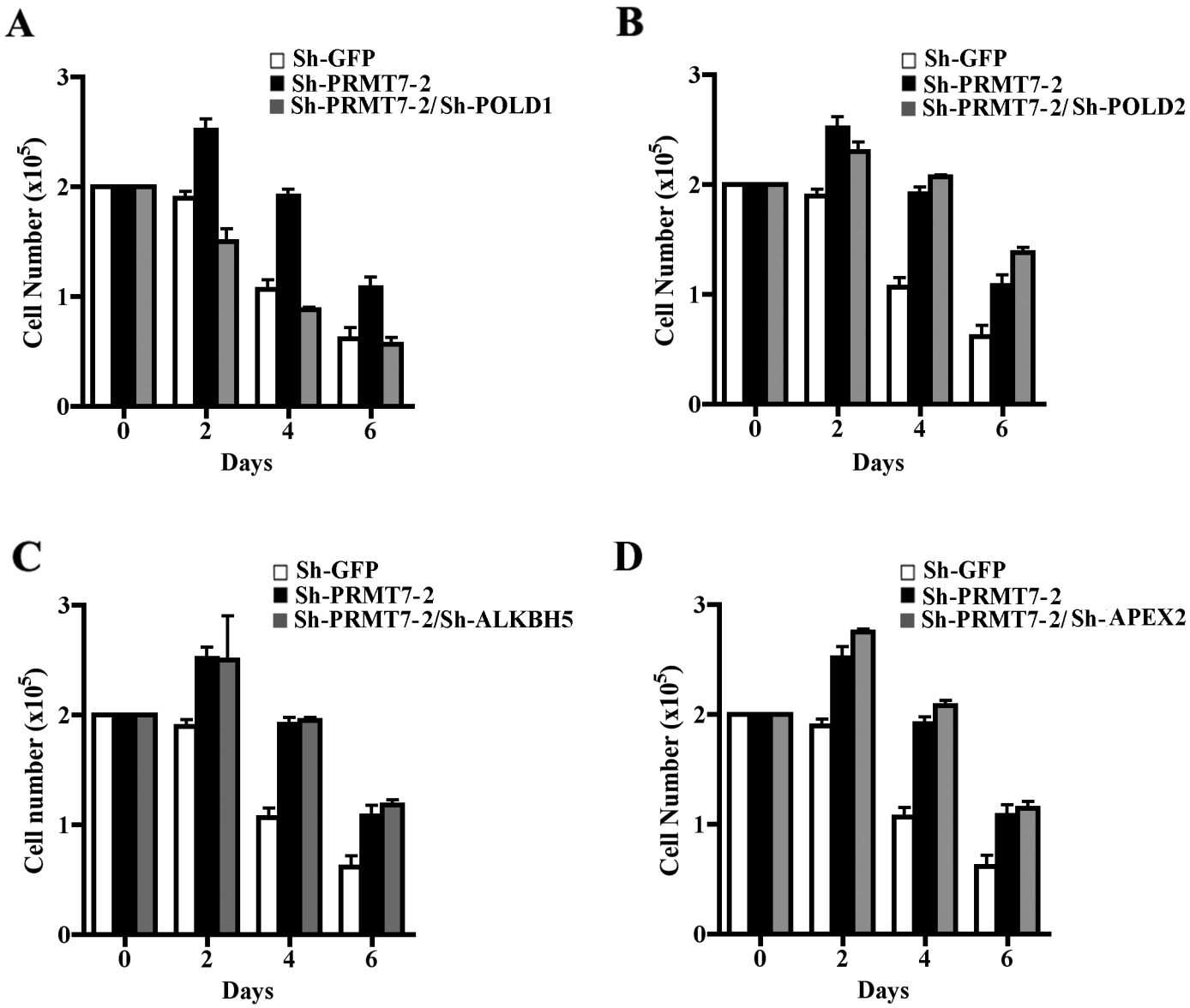

Day 0
Day 4
Day 6
NIH 3T3/sh-GFP
NIH 3T3/sh-PRMT7-1
PI Fluorescence
Annexin V FITC
4.1 0.0
95.9 0.0
2.5 4.3
62.1 31.0
16.1 53.8
28.4 1.7
1.4 0.7
96.8 1.1
1.8 3.0
80.4 14.8
14.5 34.1
50.6 0.8

Figure 7


A
Sh-GFP
Sh-PRMT7-2
Sh-PRMT7-2/ Sh-POLD1
Cell Number (x10^5)
Days
B
Sh-GFP
Sh-PRMT7-2
Sh-PRMT7-2/ Sh-POLD2
Cell Number (x10^5)
Days
C
Sh-GFP
Sh-PRMT7-2
Sh-PRMT7-2/Sh-ALKBH5
Cell number (x10^5)
Days
D
Sh-GFP
Sh-PRMT7-2
Sh-PRMT7-2/ Sh-APEX2
Cell Number (x10^5)
Days