Supplementary materials

Table 1. Nucleotide sequences of the forward primers, reverse primers and amplicon size for the genes quantified by RT-PCR.

Gene	RefSeq accession number	Forward primer (5' ⟶ 3')	Reverse primer (5' ⟶ 3')	Product size (bp)
p15	NM_004936	ATT CGA TGA AGG CTG CCA AC	TGA AAA GCA AAC GAC CCC TG	219
CDH-1	NM_004360	CGT AGC AGT GAC GAA TGT GG	CTG GGC AGT GTA GGA TGT GA	175
DNMT1	NM_001130823	GAG CTA CCA CGC AGA CAT CA	CGA GGA AGT AGA AGC GGT TG	161
DNMT3a	NM_022552	CCG GAA CAT TGA GGA CAT CT	CAG CAG ATG GTG CAG TAG GA	162
DNMT3b	NM_006892	CTC AGA GGC AGT GAC AGC AG	TGT CTG AAT TCC CGT TCT CC	166

Table 2. Dose-effect analysis and calculated CI values using CalcuSyn.

Cell type	Treatment	CI value
CEM	250 nM DAC + 250 nM DMC	1.79
	250 nM DAC + 500 nM DMC	2.06
	250 nM DAC + 1000 nM DMC	1.25
	500 nM DAC + 250 nM DMC	0.69
	500 nM DAC + 500 nM DMC	1.01
	500 nM DAC + 1000 nM DMC	0.71
	1000 nM DAC + 250 nM DMC	1.17
	1000 nM DAC + 500 nM DMC	1.81
	1000 nM DAC + 1000 nM DMC	1.15
Jurkat	250 nM DAC + 250 nM DMC	1.17
	250 nM DAC + 500 nM DMC	2.21
	250 nM DAC + 1000 nM DMC	1.13
	500 nM DAC + 250 nM DMC	0.82
	500 nM DAC + 500 nM DMC	0.86
	500 nM DAC + 1000 nM DMC	0.68
	1000 nM DAC + 250 nM DMC	1.15
	1000 nM DAC + 500 nM DMC	1.27
	1000 nM DAC + 1000 nM DMC	1.59
Primary MDS	100 nM DAC + 250 nM DMC	0.92
	100 nM DAC + 500 nM DMC	10.96
	250 nM DAC + 250 nM DMC	2.83
	250 nM DAC + 500 nM DMC	1.92
Primary AML	100 nM DAC + 250 nM DMC	1.52
	100 nM DAC + 500 nM DMC	1.39
	250 nM DAC + 250 nM DMC	1.13
	250 nM DAC + 500 nM DMC	1.11

Figure 1. ViaCount reagent assessment of the viability of BMNC in an AML sample. The upper left corner of the dot plot shows nucleated life cells and the upper right corner shows apoptotic and dead cells.

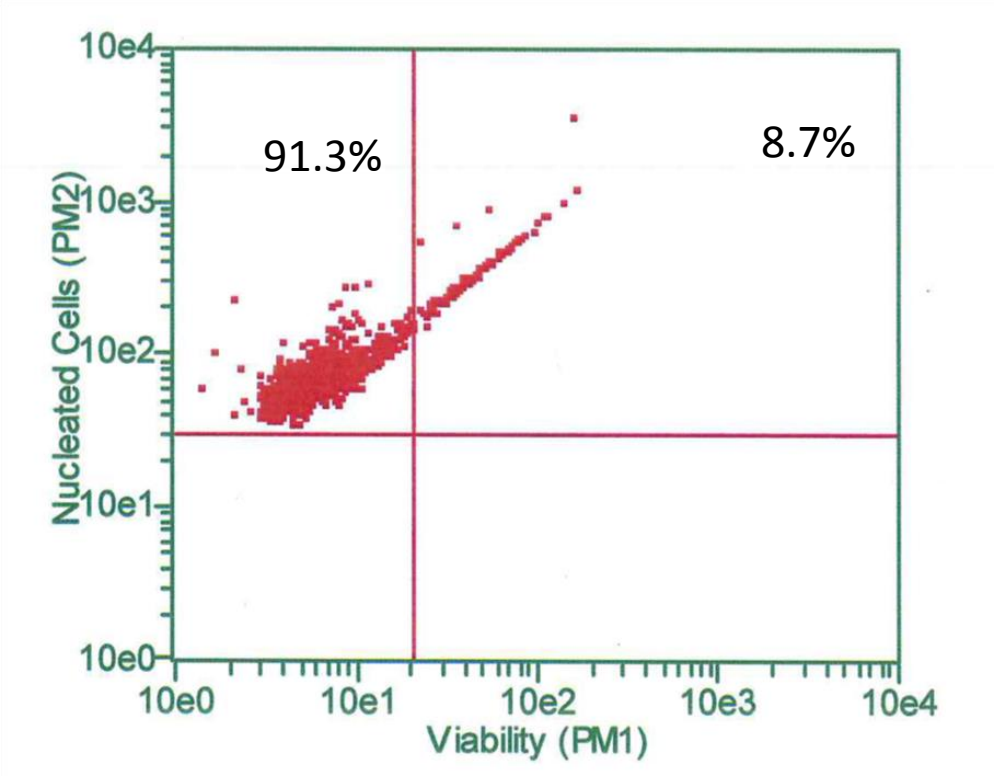


Figure 2. Verification of the amplicon size for the p15 and CDH-1 genes prior to DNA pyrosequencing using 2% agarose gel electrophoresis. Lane 1: 100 bp ladder; lanes 2-4: *p15* amplicon (170 bp) from 3 representative samples. Lanes 5-7: *CDH1* amplicon (207 bp) from 3 representative samples. Lanes 8 and 9 are negative controls (water) for the p15 and CDH1 PCR reaction, respectively.

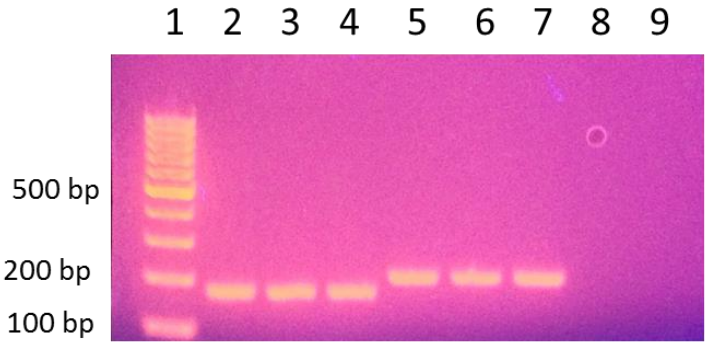


Figure 3. Covaris shearing of Input ChIP samples. The average fragment size (bp) after shearing is shown for each cell line for the control cells, DAC treated, DMC treated and the combination treatment (D+M).

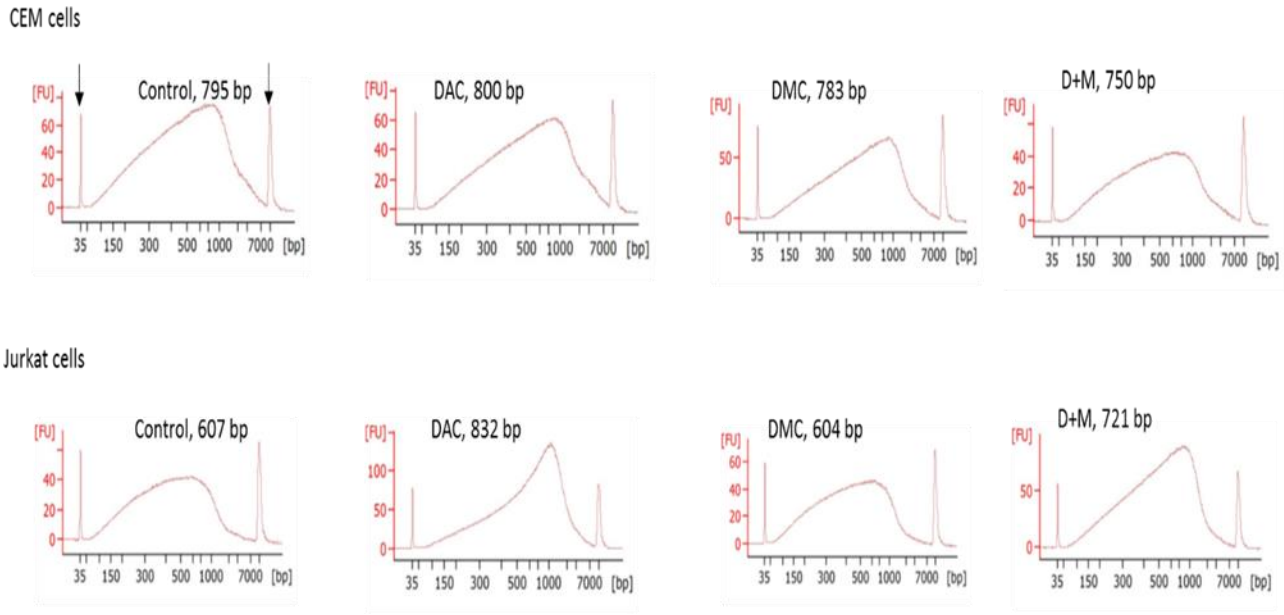
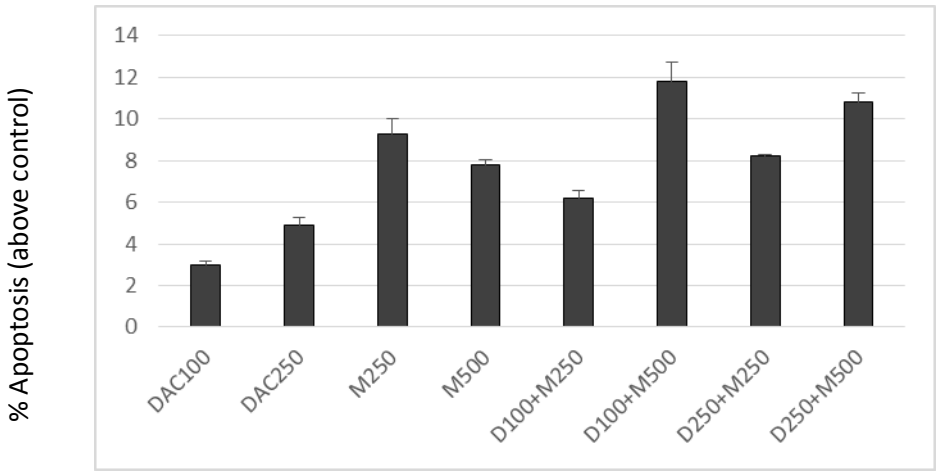


Figure 4. AML (a) and MDS (b) samples treated with single agents or the combination for 24 h. Apoptosis was quantitated as described under methods. Data represent the mean of 3 replicates ± SD. “M” indicates DMC, D100 indicates DAC 100 nM, D250 indicates DAC 250 nM, M250 indicates DMC 250 nM and M500 indicates DMC 500 nM.

a.



b.

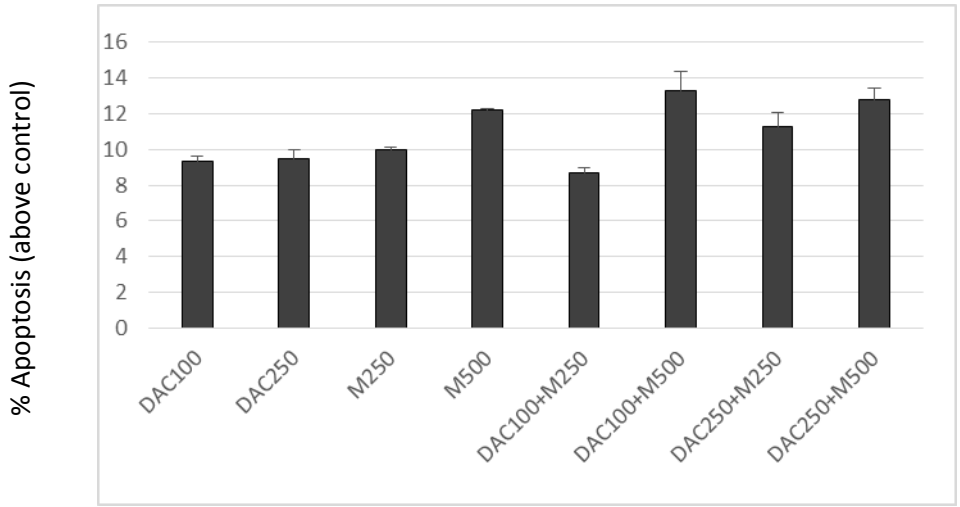


Figure 5. Jurkat cells treated with single agents or the combination for 24 h followed by quantitative analysis of the gene expression of *p15* (a) and *CDH-1* (b) genes. “M” indicates DMC, D100 indicates DAC 100 nM, D250 indicates DAC 250 nM, M250 indicates DMC 250 nM and M500 indicates DMC 500 nM.

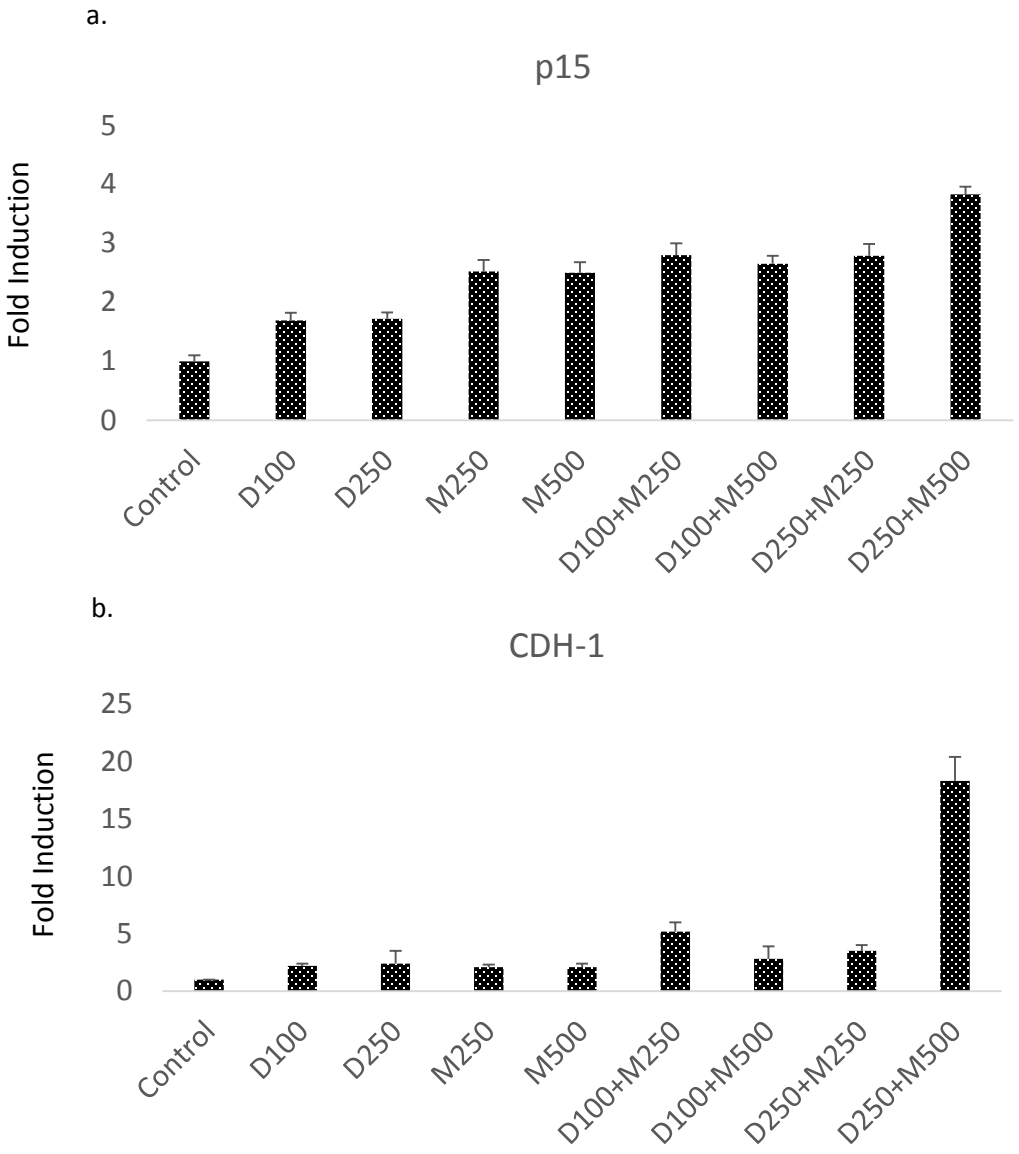


Figure 6. Jurkat cells treated with the single drugs and the combination for 24 h followed by quantitating the protein expression of p15 and CDH-1 by Western blotting. D250 indicates DAC 250 nM, M250 indicates DMC 250 nM and M500 indicates DMC 500 nM.

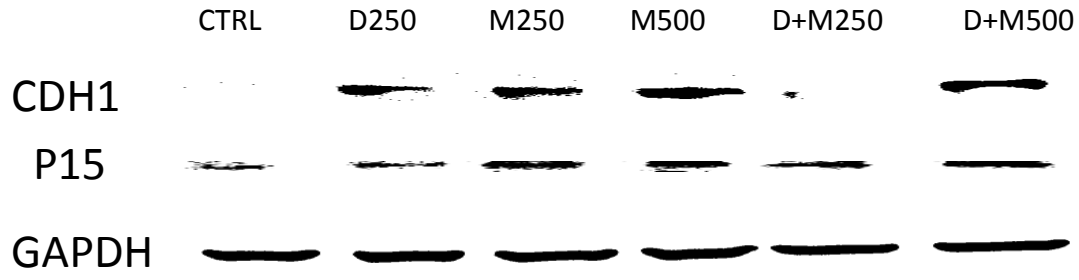


Figure 7. Sequential ChIP – DNA pyrosequencing of 7 CpG sites in the CpG island of p15. CEM cells were treated with the single agents or the combination for 24 h followed by ChIP using H3K27Ac antibody. IgG was used as a negative control. DNA pyrosequencing of the immunoprecipitated DNA using sequencing primers for the p15 gene to analyze 7 CpG sites was performed. Data represent the mean ± SD of duplicate runs. D+M indicates 250 nM DAC + 500 nM DMC.