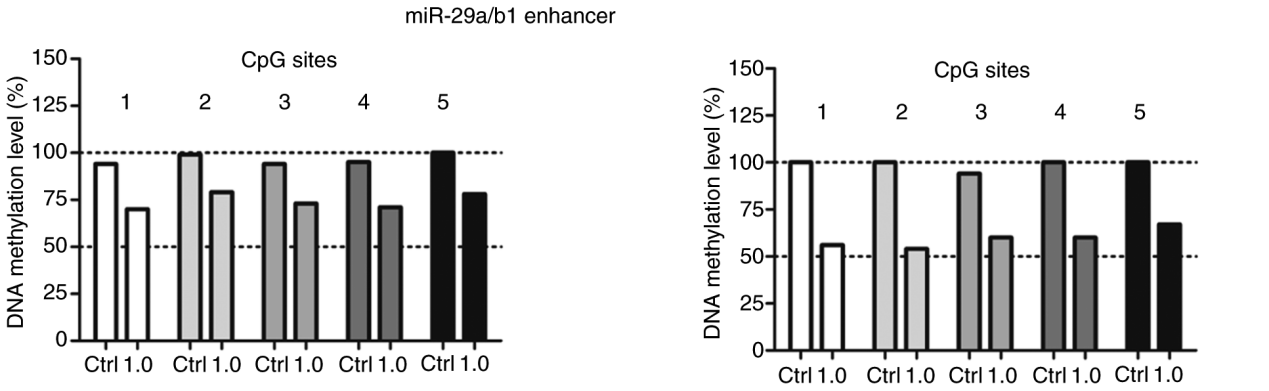
Figure S1. miR-29a/b1 and miR-29b2/c genes are silenced in BL cells by methylation at promoter and enhancer regions. The methylation percentage of each CpG site is depicted in the bar plots followed by the mean and SD of each CpG sites, as presented in the box at the bottom of each graph. The percentage of methylation levels was compared between the control (untreated) and decitabine (1 μM) groups after 24 h in BL41 and Raji cells. (A) Five CpG sites on the miR-29a/b1 enhancer region, (B) six CpG sites on the miR-29b2/c promoter, and (C) five CpG sites flanking the miR-29b2c promoter. BL, Burkitt lymphoma; SD, standard deviation.

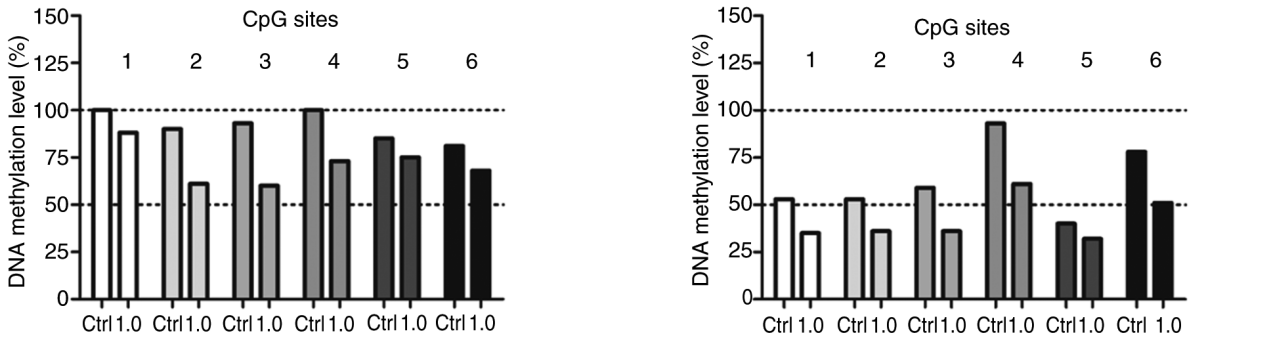
A



BL41 cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	96%±3	74%±4

Raji cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	99%±3	59%±5

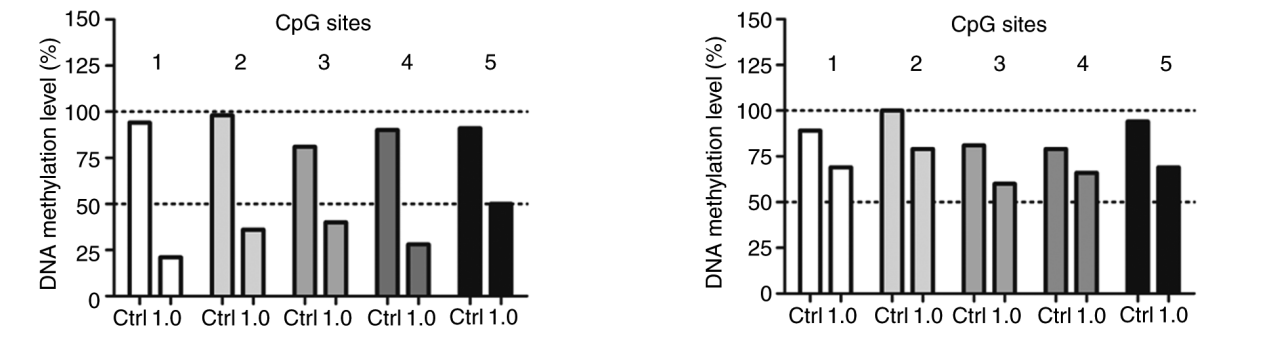
B



BL41 cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	92%±8	71%±10

Raji cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	63%±19	42%±12

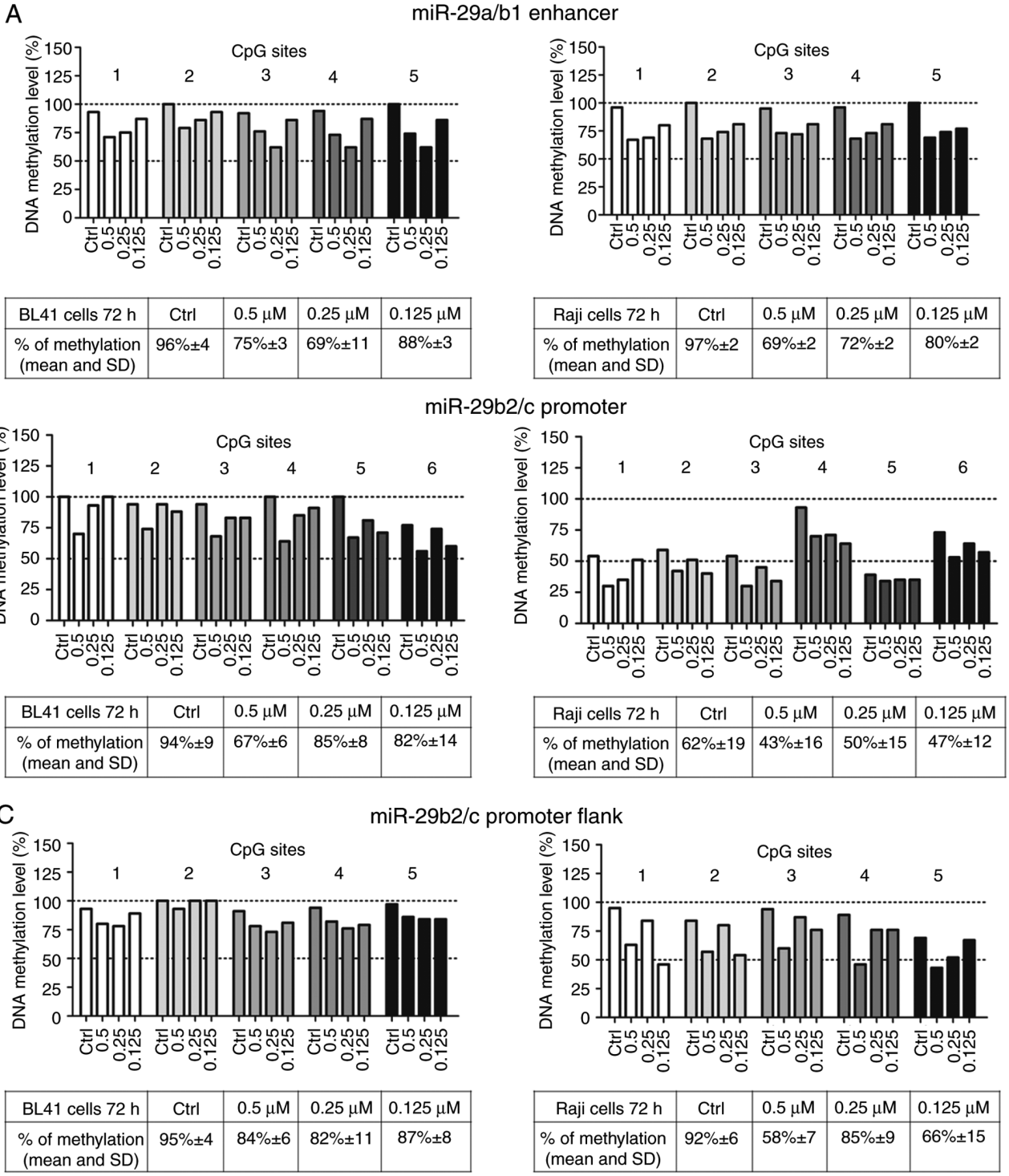
C



BL41 cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	91%±6	35%±11

Raji cells 24 h	Ctrl	1.0 μM
% of methylation (mean and SD)	89%±9	69%±7

Figure S2. Treatment with low doses of decitabine causes miR-29 demethylation in BL cells. Methylation analysis after treatment with low doses of decitabine. The methylation percentage of each CpG site is depicted in the bar plots followed by the mean and SD of the CpG sites, as revealed in the box at the bottom of each graph relative to each decitabine concentration (0.5, 0.25 and 0.125 μM). The percentage of methylation levels were compared between the control (untreated) vs. the decitabine groups after 72 h in BL41 and Raji cells. (A) Five CpG sites in the miR-29a/b1 enhancer region, (B) six CpG sites in the miR-29b2/c promoter, and (C) five CpG sites flanking the miR-29b2c promoter; SD, standard deviation.



BL41 cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	96%±4	75%±3	69%±11	88%±3

Raji cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	97%±2	69%±2	72%±2	80%±2

BL41 cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	94%±9	67%±6	85%±8	82%±14

Raji cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	62%±19	43%±16	50%±15	47%±12

BL41 cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	95%±4	84%±6	82%±11	87%±8

Raji cells 72 h	Ctrl	0.5 μM	0.25 μM	0.125 μM
% of methylation (mean and SD)	92%±6	58%±7	85%±9	66%±15

Figure S3. Transfection efficiency analysis. Flow cytometric analysis of the cell transfection assay was performed using Lipofectamine 2000 or RNAiMAX. Raji ($1x10^5$) cells were transfected with Cy3-labeled siRNA (200 nM) and washed with phosphate-buffered saline. Lipofectamine 2000 was used in subsequent experiments due its more efficient performance in comparison to RNAiMAX. In solid white, cells transfected with Lipofectamine 2000; in solid gray, cells transfected with RNAiMAX; in dashed grey, the negative control of RNAiMAX; in dashed white, the Lipofectamine 2000 negative control.

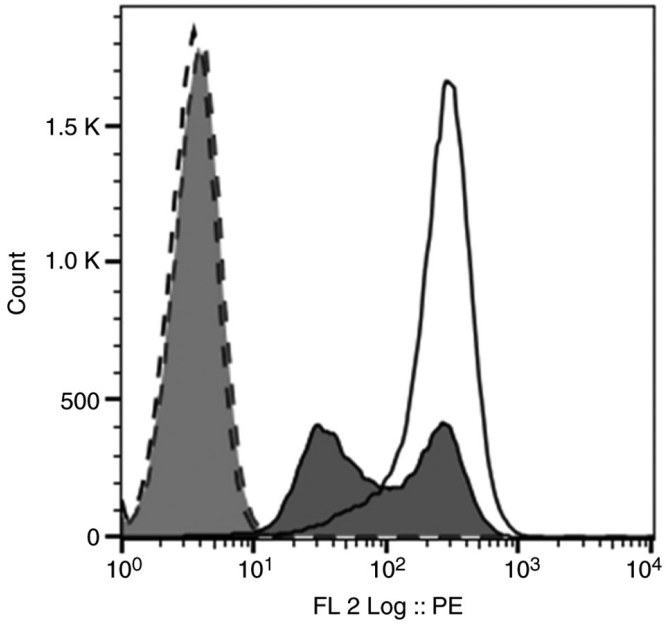


Figure S4. miR-29a/b/c expression in Namalwa, Daudi, Ramos and BL41 cells in comparison to Raji cells. The relative levels of miR-29a/b/c expression between untreated Namalwa, Daudi, Ramos and BL41 cells and untreated Raji cells. The results reveal the fold increases in the miR-29a/b/c expression levels in comparison to Raji cells. The gene RNU6B was used as the endogenous control in the qRT-PCR. The mean of two or three independent experiments plus the standard deviation is presented.