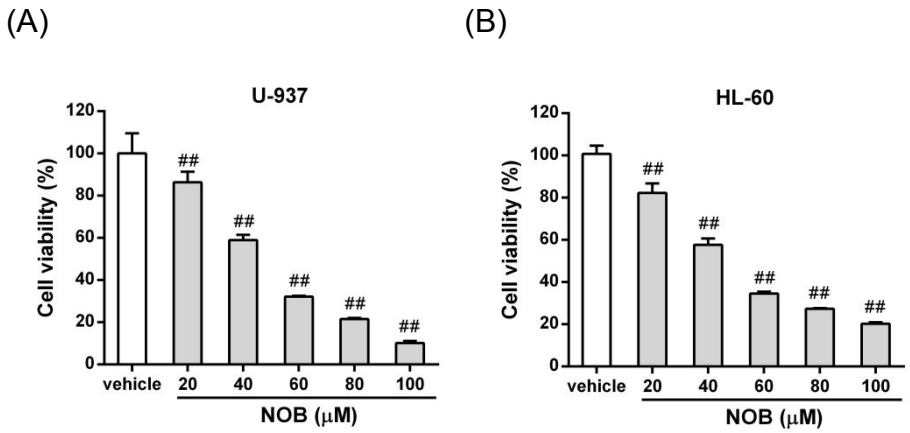

Figure S1. Effects of NOB on the cell growth in U-937 and HL-60 cells. (A) U-937 and (B) HL-60 cells were treated with vehicle (0.1% DMSO) or NOB (20-100 μM) for 48 h. The viability of NOB-treated cells was measured using an MTT assay. The data represent the mean ± SD of three independent experiments. ##p<0.01 represent significant differences compared to the vehicle-treated group.

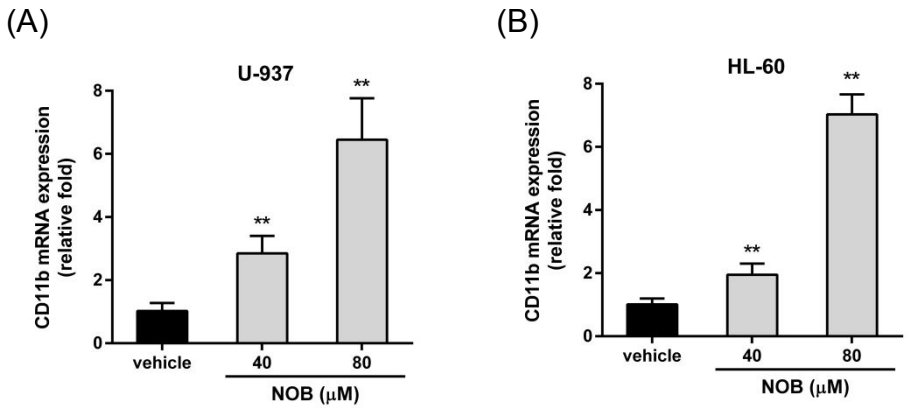

Figure S2. Effects of NOB on myeloid cell differentiation in U-937 and HL-60 cells. (A) U-937 and (B) HL-60 cells were treated with vehicle or NOB (40 and 80 μM) for 48 h. The expression of CD11b mRNA was measured by Q-RT-PCR analysis. The data represent the mean ± SD of three independent experiments. ***p*<0.01 represent significant differences compared to the vehicle-treated group.

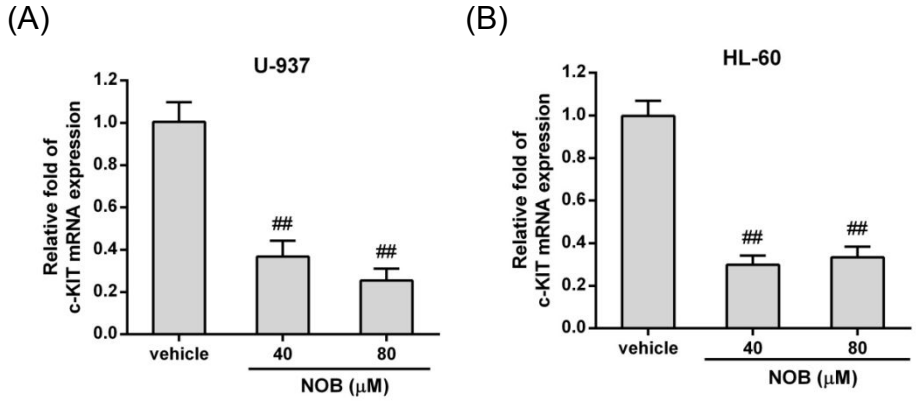

Figure S3. Effects of NOB on c-KIT mRNA expression in U-937 and HL-60 cells. (A) U-937 and (B) HL-60 cells were treated with vehicle or NOB (40 and 80 μM) for 48 h and the c-KIT mRNA levels were determined by Q-RT-PCR analysis. The data represent the mean ± SD of three independent experiments. ##$p<0.01$ represent significant differences compared to the vehicle-treated cells.

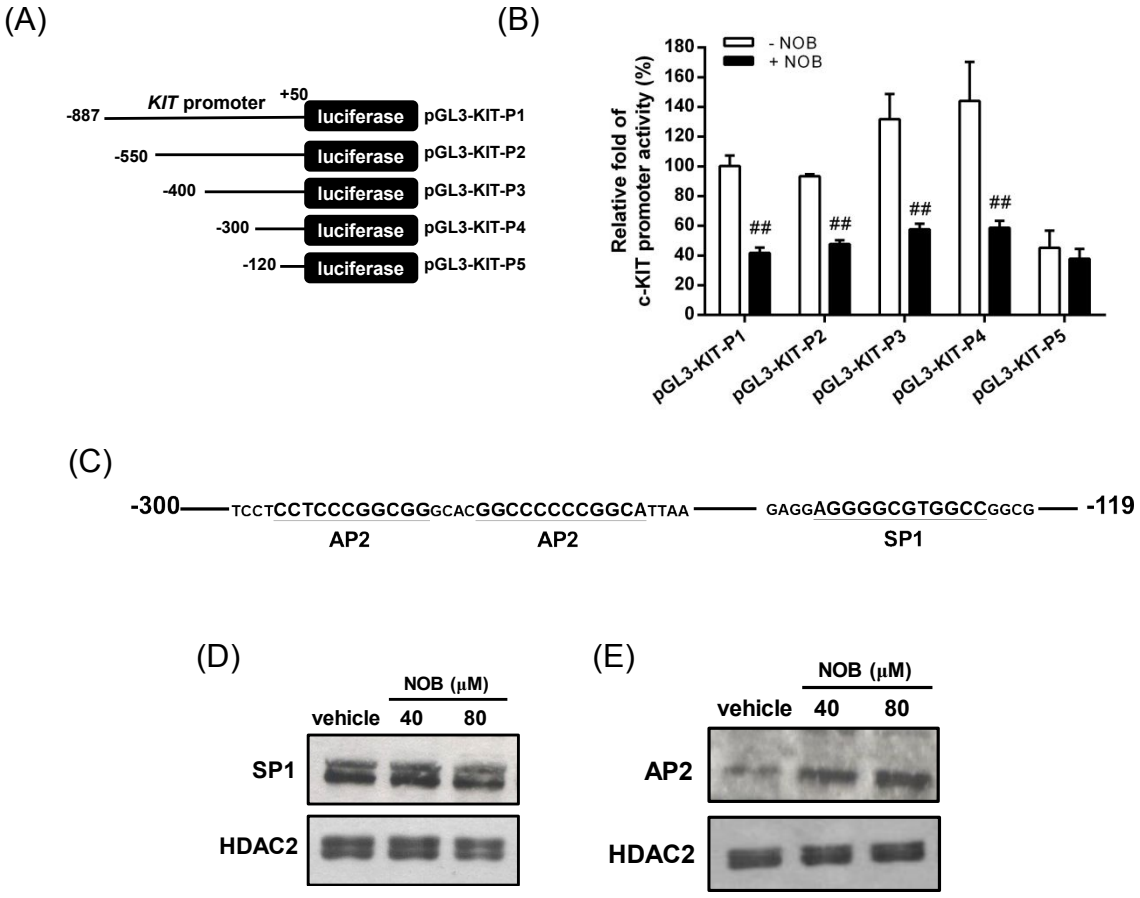

Figure S4. Characterization of the nobiletin-responsive element within human c-KIT promoter. (A) Serially deleted c-KIT promoter reporter plasmids. (B) THP-1 cells were co-transfected with reporter plasmids and Renilla luciferase control plasmid for 24 h and then treated with the vehicle (0.1% DMSO) or NOB (80 μM) for 48 h. The luciferase activities were measured and normalized to their respective Renilla luciferase activities. The data represent the mean ± SD from three independent experiments. ## $p<0.01$ represents a significant difference compared to the respective vehicle-treated cells. (C) The analysis of the NOB-response DNA element within the c-KIT promoter. The DNA elements of AP2 and SP1 binding sequences are indicated. THP-1 cells were

treated with vehicle or NOB (40 and 80 μM) for 48 h and the levels of nuclear (D) SP1 and (E) AP2 protein were determined by Western blot analysis. The immunoblots were performed at three independent experiments. A represent blot is shown.

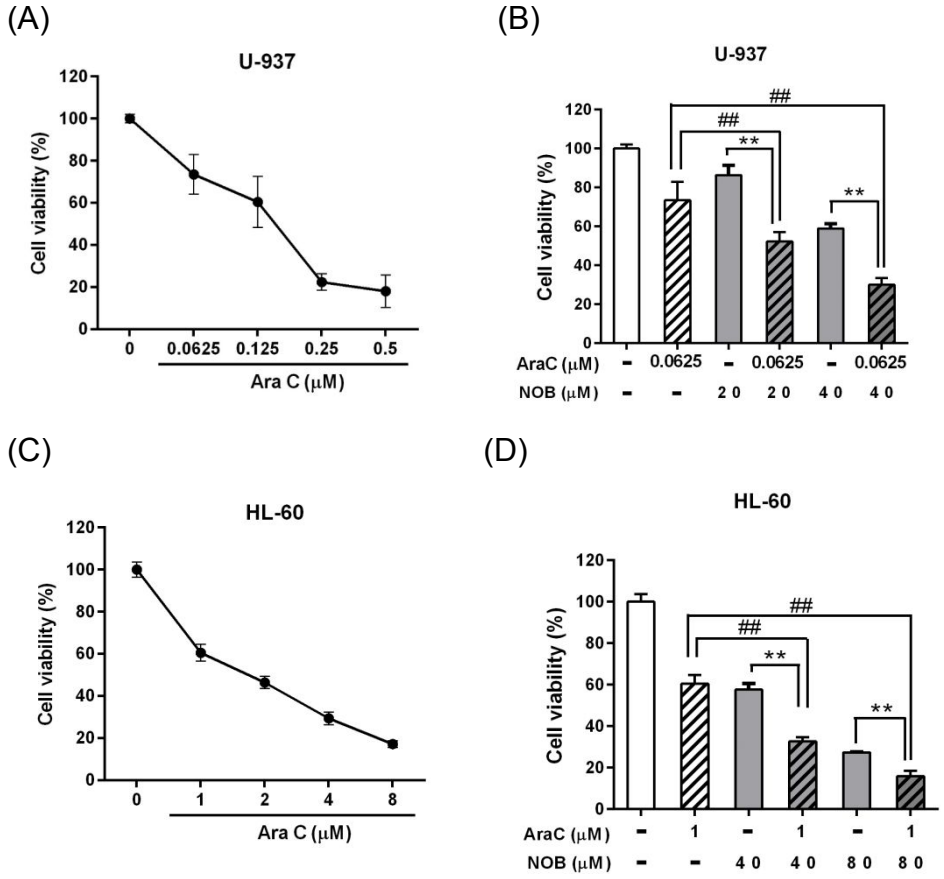

Figure S5. Effects of combination treatment of cytarabine (Ara C) and NOB in U-937 and HL-60 cells. (A) U-937 cells were treated with Ara C (0-0.5 μM) for 48 h. The cell viability was measured by MTT assay. (B) U-937 cells were pre-treated with Ara C (0.0625 μM) and HL-60 cells were pre-treated with Ara C (1 μM) for 12 h followed by treatment of cells with vehicle or NOB (40 or 80 μM) for further 36 h. The cell viability was measured by MTT assay. (C) HL-60 cells were treated with Ara C (0-8 μM) for 48 h. The cell viability was measured by MTT assay. (D) HL-60 cells were pre-treated with Ara C (1 μM) for 12 h followed by treatment of cells with vehicle or

NOB (40 or 80 μM) for further 36 h. The cell viability was measured by MTT assay. The data represent the mean ± SD of three independent experiments. ##p<0.01 represents significant differences compared to the NOB-untreated group. **p<0.01 represents significant differences compared to the Ara C-untreated group.

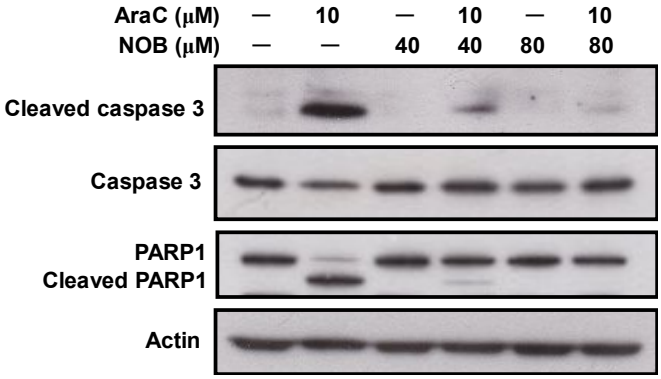

Figure S6. Effects of combination treatment of cytarabine (Ara-C) and NOB on cell apoptosis in THP-1 cells. THP-1 cells were pre-treated with Ara-C (10 μM) for 12 h, followed by treatment of cells with vehicle or NOB (40 or 80 μM) for a further 36 h. The cleaved caspase 3, caspase 3, PARP1 and cleaved PARP1 proteins in total lysates were detected by Western blot analysis. The immunoblots were performed at three independent experiments. A represent blot is shown.