Supplementary Figure S1. Oridonin induces BIM-S in multiple leukemia cell lines.
(A) HL60/ADR cells were treated with VP-16 (10 μM), oridonin (ori, 20 μM) or DMSO for 24 hours and lysed for western blot analysis. (B) NB4 and (C) Jurkat cells were treated with oridonin (20 μM for NB4, 10 μM for Jurkat) or DMSO for 24 hours and lysed for western blot analysis. Images showing the expression of the same protein in NB4 cells were cropped from the same blot.

Supplementary Figure S2. Overexpression of BIM induces apoptosis in K562 cells.
(A, B) K562 cells were transfected with empty or Bim-expression vectors as indicated. Twenty-four hours later, cells were subjected to western blot analysis (A) or apoptosis analysis by flow cytometry (B).

Supplementary Figure S3. Response of miRNAs to drug treatment in multiple leukemia cell lines.
(A) Histogram showing changes of miRNAs in K562 cells after treating with Ara-C (10 μM), VP-16 (10 μM), and oridonin (20 μM) for 24 hours. *, $P < 0.05$; **, $P < 0.01$. (B) Histogram showing changes of miRNAs in HL60/ADR cells after treating with VP-16 (10 μM) or oridonin (20 μM) for 24 hours. (C) NB4 and (D) Jurkat cells were treated with oridonin (20 μM for NB4, 10 μM for Jurkat) or DMSO for 24 hours and lysed for qPCR analysis. GAPGH was used as an internal control.

Supplementary Figure S4. Effects of miRNA inhibitors of the miR-17-19b cluster on the expression of BIM-S.
K562 cells were transfected with negative control (NC) or miRNA inhibitors of the miR-17-19b cluster as indicated. Forty-eight hours later, cells were subjected to western blot analysis.

Supplementary Figure S5. miR-17 directly targets the 3'UTR of BIM-S.
293T cells were cotransfected with negative control (NC)/ miR-17 miRNA mimics, pRL-TK Renilla luciferase plasmid, and BimS-3'UTR-WT-pGl3/ BimS-3'UTR-MUT-pGl3 firefly luciferase plasmid, and dual luciferase reporter assays were performed 24 hours later. The BimS-3'UTR-WT-pGl3 plasmid harbors the wild-type 3'UTR sequence of *Bim-S*, which is predicted to basepair with miR-17 or miR-20a, within the 3' terminus of the firefly luciferase gene, whereas this sequence was mutated in the BimS-3'UTR-MUT-pGl3 plasmid. Data were analyzed by normalizing firefly luciferase activity to Renilla luciferase expression for each sample. NC-transfected samples were set to 100%. **, $P < 0.01$.

Supplementary Figure S6. Inhibition of miR-17 or miR-20a has little effect on the sensitivity of K562 to Ara-C.
(A, B) Histograms showing the efficacy of Ara-C in K562 cells transiently transfected with miR-17 or miR-20a miRNA inhibitors, as determined by MTT assays (A) or FITC-Annexin V/PI staining (B). The inhibition rate or apoptotic rate of cells in each transfection group without Ara-C treatment was set to 0.

Supplementary Figure S7. Oridonin has no synergic effect with Ara-C.

K562 cells were treated with Ara-C at different concentrations and with or without oridonin (10 μM) for 24 hours. The effect on the percentage of viable cells was determined by MTT assays. The inhibition rate of cells without Ara-C treatment was set to 0.

Supplementary Figure S8. Oridonin sensitizes leukemia cells to VP-16.

(A, B) Histograms showing the synergistic effects of VP-16 and oridonin in K562/ADR and HL60/ADR cells as determined by MTT assays 24 hours after drug treatment. (C) Histograms showing the synergistic effects of VP-16 (5 μM) and oridonin (2.5 μM) in Jurkat cells as determined by MTT assays 24 hours after drug treatment. The inhibitory effect of oridonin alone was subtracted from the final results and thus the inhibition rate of cells without VP-16 treatment was set to 0. *, $P < 0.05$; **, $P < 0.01$.