Figure S1. (A and B) Reverse transcription-quantitative PCR analysis of the mRNA expression levels of HMGB1 and ACSL4 in HL60, K562 and Kas-1 cells. *P<0.05, **P<0.01. HMGB1, high mobility group box-1 protein; Kas-1, Kasumi-1.

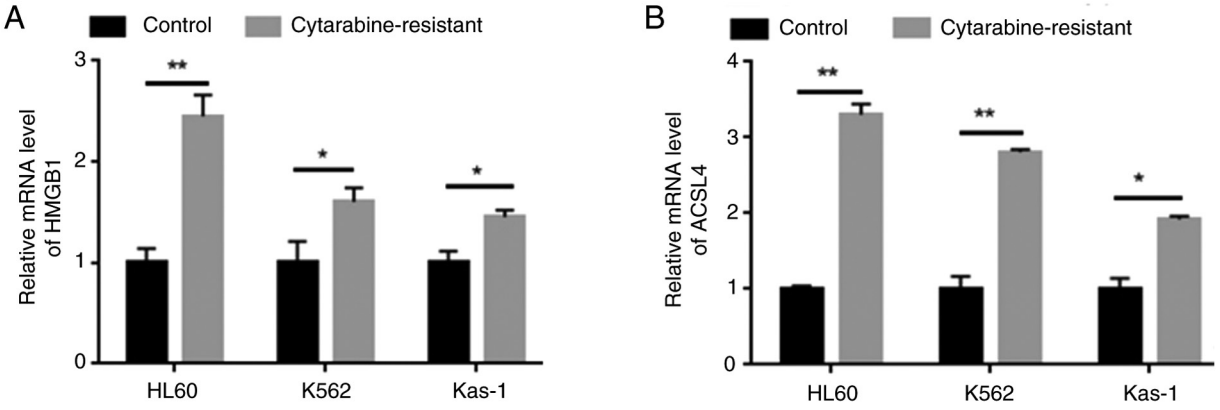

Figure S2. Cell Counting Kit-8 assay detected cell viability after treatment with different concentrations of cytarabine. *P<0.05. NC, negative control; si, small interfering; SIRT1, sirtuin 1.

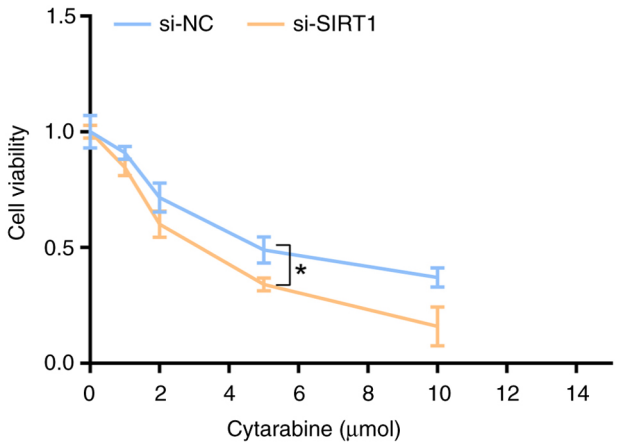

Figure S3. Knockdown of SIRT1 did not affect the viability of HL60 cells. (A) Reverse transcription-quantitative PCR analysis of the knockdown efficiency of si-SIRT1 in HL60 cells. (B) Cell Counting Kit-8 assay was used to detect cell viability after knocking down SIRT1. *P<0.05, **P<0.01. HL60/C, cytarabine-resistant HL60; NC, negative control; si, small interfering; SIRT1, sirtuin 1.

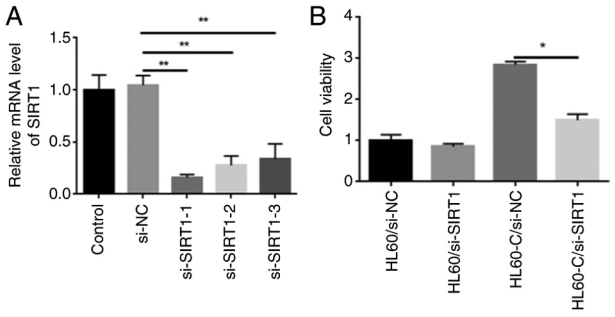

Figure S4. Apoptosis was detected by flow cytometry in HL60 and HL60/C cells. **P<0.01. Control, HL60 cells; NC, negative control; si, small interfering; SIRT1, sirtuin 1.

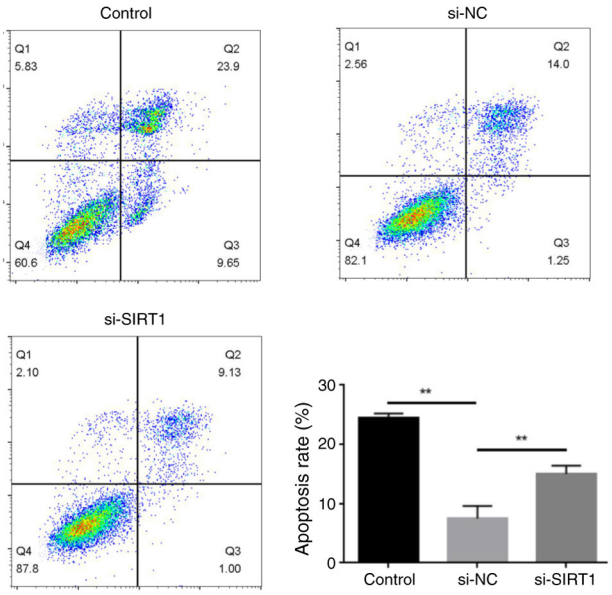

Figure S5. Efficiency of SIRT1 overexpression was detected by reverse transcription-quantitative PCR in 293T,HL60 and HL60/C cells. **P<0.01. SIRT1, sirtuin 1.

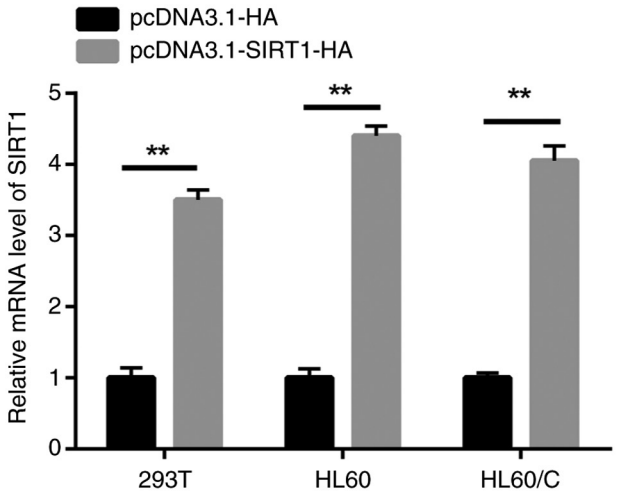

Figure S6. Efficiency of HMGB1 overexpression was detected by reverse transcription-quantitative PCRin 293T,HL60 and HL60/C cells. **P<0.01. HMGB1, high mobility group box-1 protein.

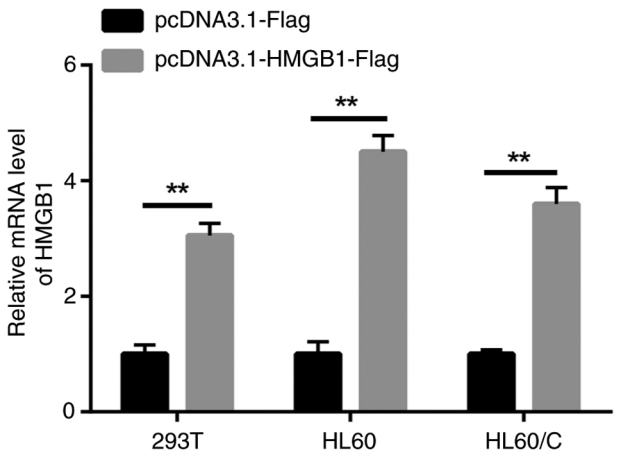

Figure S7. Western blot analysis of the knockdown efficiency of HMGB1 siRNA sequences. **P<0.01, ***P<0.001. HMGB1, high mobility group box-1 protein; NC, negative control; si, small interfering.

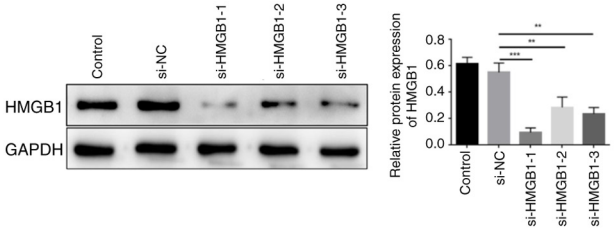

Figure S8. Reverse transcription-quantitative PCR determining the knockdown efficiency of ACSL4. **P<0.01. NC, negative control; si, small interfering.