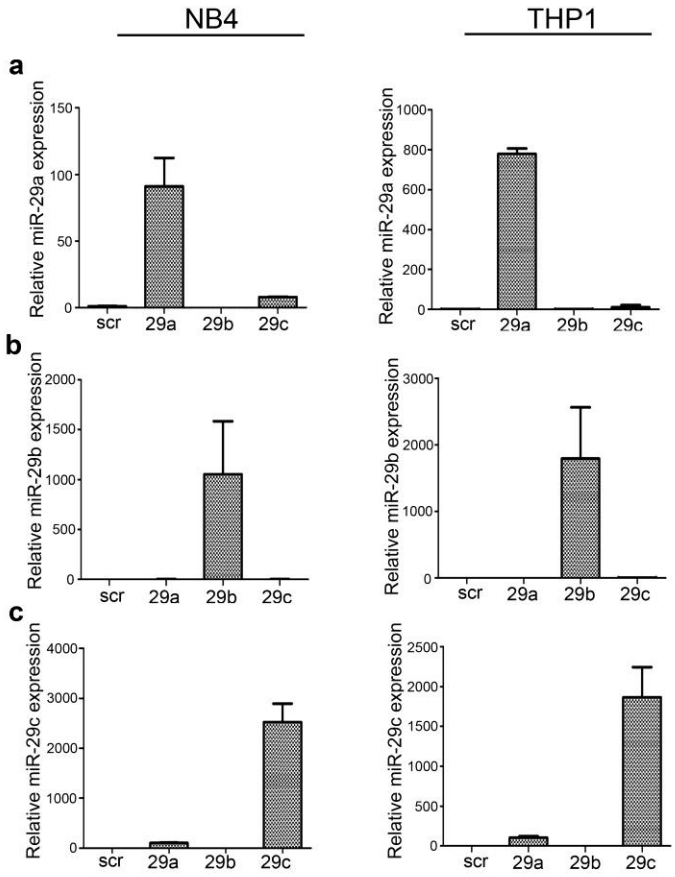
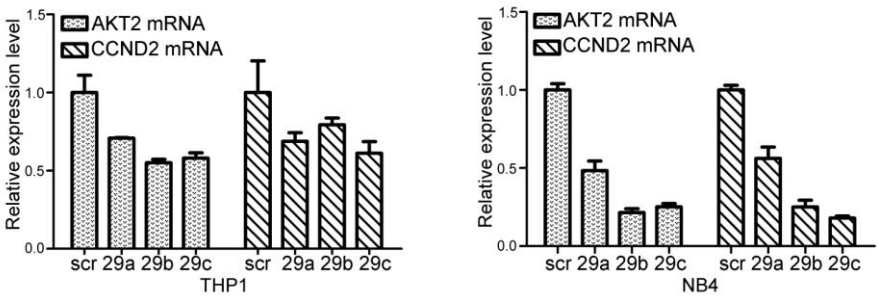
Supplementary Figure S1-S9 and the Relative Legends

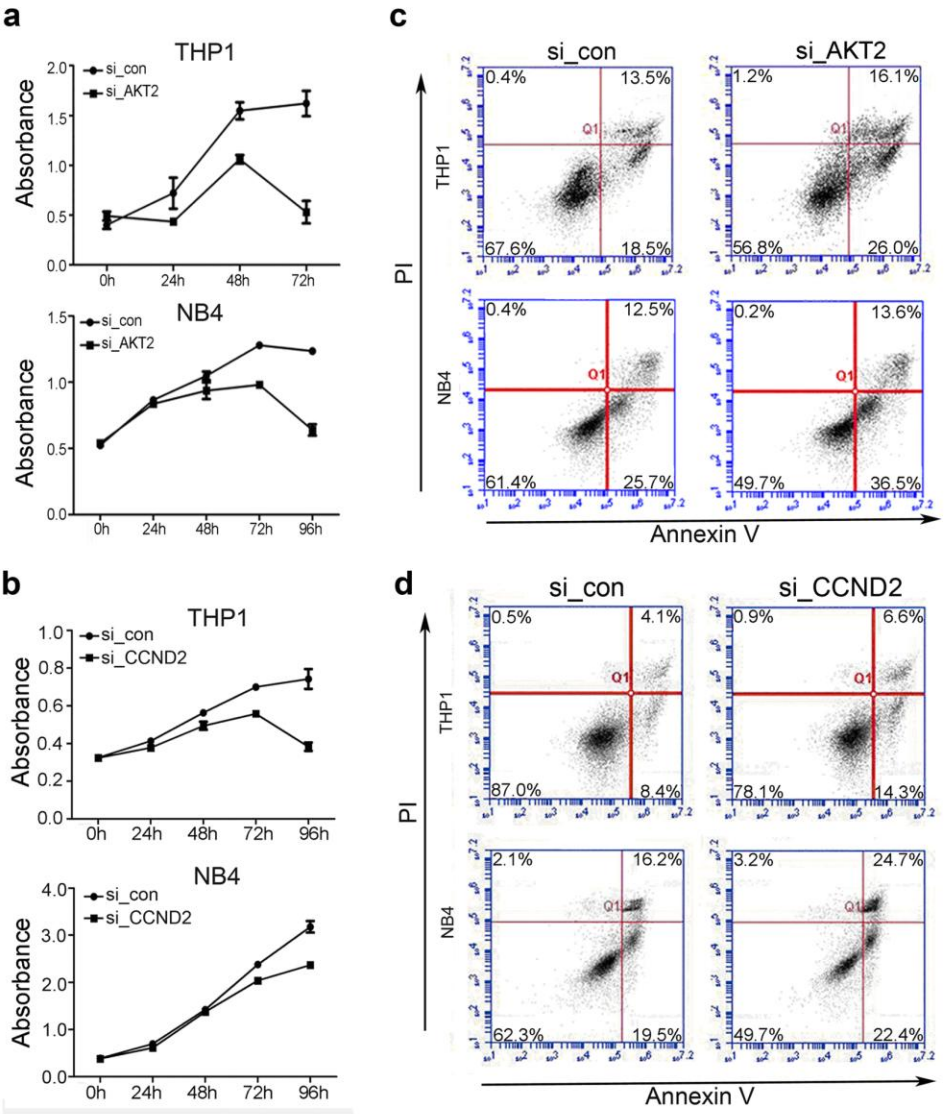


Supplementary Figure S1. A specificity validation of Taqman probes and primers for detecting miR-29a, miR-29b and miR-29c. Taqman stem-loop RT-PCR was performed in THP1 and NB4 transfected with miR-29a, miR-29b, miR-29c and mimic control (scr) for 48 h. (**a**) Detection of miR-29a in THP1 and NB4 cells transfected with miR-29a, miR-29b, miR-29c and scr. (**b**) Detection of miR-29b in THP1 and NB4 cells transfected with miR-29a, miR-29b, miR-29c and scr. (**c**) Detection of miR-29c in THP1 and NB4 cells transfected with miR-29a, miR-29b, miR-29c and scr.

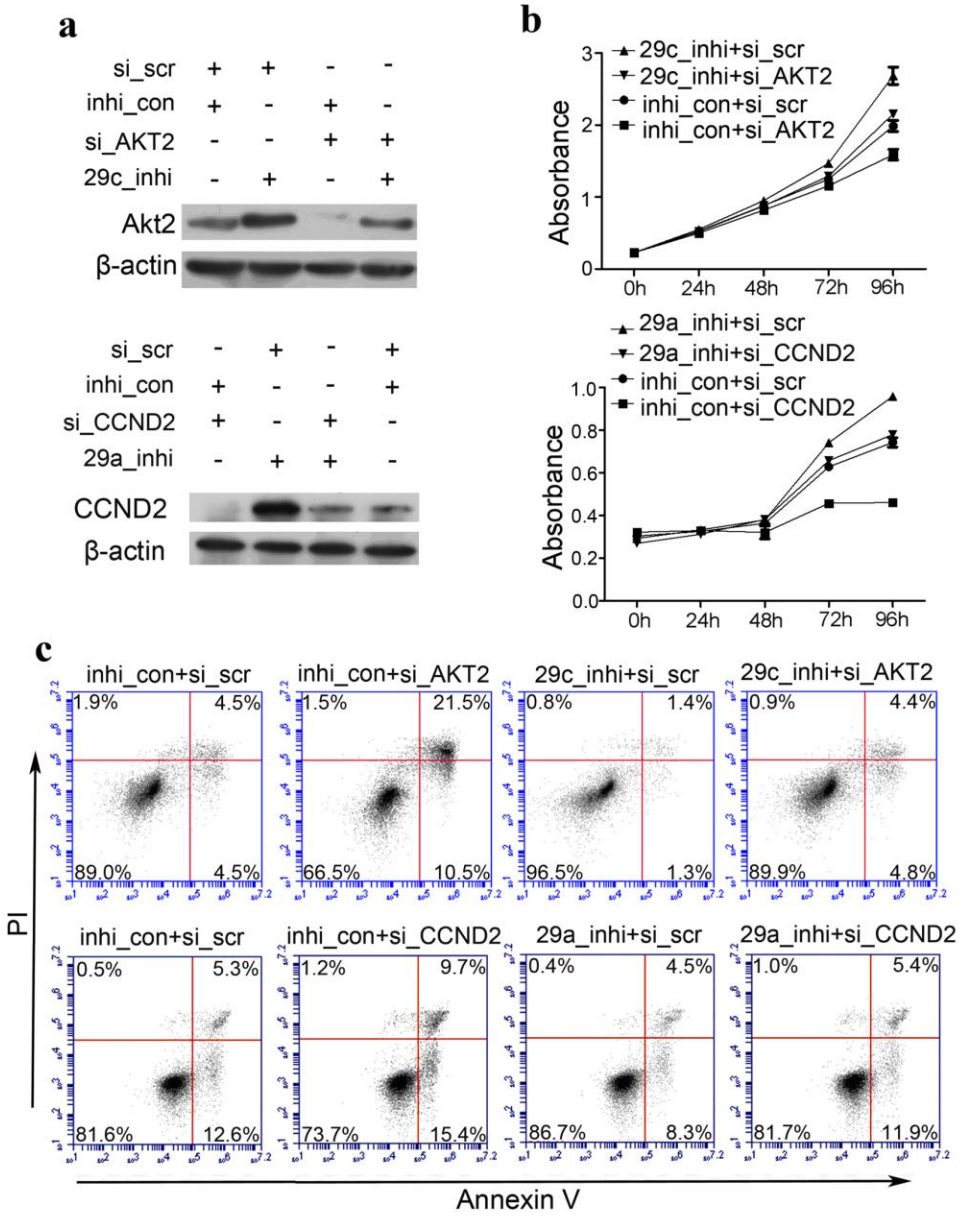


Supplementary Figure S2. Significantly decreased AKT2 mRNA and CCND2 mRNA

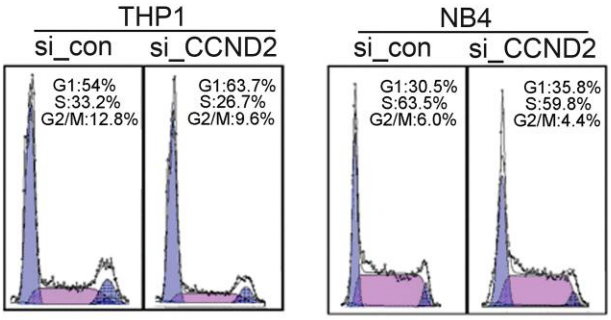
expression levels were detected in THP1 and NB4 cells transfected with miR-29a, -29b or -29c mimic as compared with the cells transfected with mimic scramble control (scr). The mRNA levels were measured by real-time PCR at 48 h after transfection and β-actin was used as the internal control. Each real-time PCR assay was performed in triplicate and the results were shown as Mean±SD.



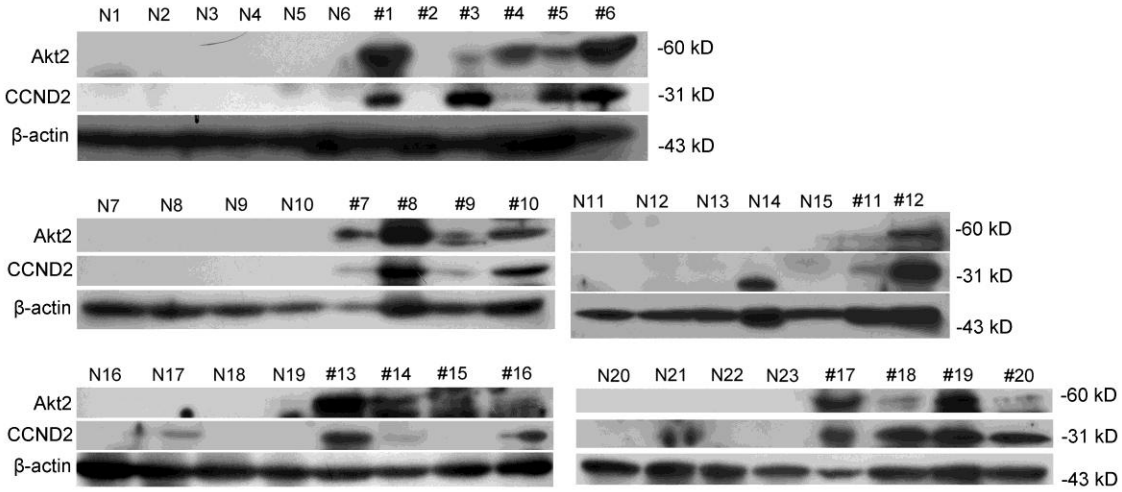
Supplementary Figure S3. Function of Akt2 and CCND2 in regulation of cell amplification and cell apoptosis inTHP1 and NB4 cells. (**a, b**) Cell growth curves of THP1 and NB4 after knockdown of *AKT2/CCND2* through siRNA. NB4 and THP1 cells were transfected with the *AKT2* siRNA（or *CCND2* siRNA） or control siRNA and the cell viability was measured at the indicated time point using CCK-8. The optical density was read at 450 nm with a microplate spectrophotometer. Each experiment was carried out in triplicate. (**c, d**) Cells at early apoptosis stage and late apoptosis stage were analyzed in THP1 and NB4 by using FITC Annexin Ⅴ Apoptosis detection kit 1 after transfection with si_AKT2/CCND2 and si_con for 72h.



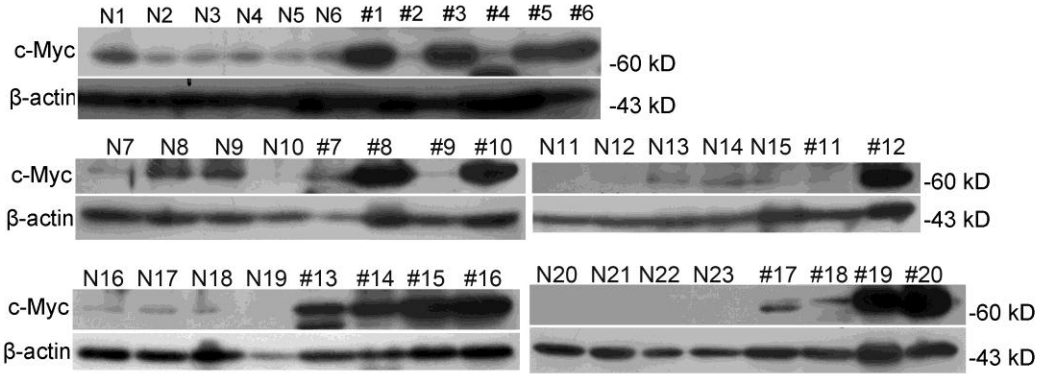
Supplementary Figure S4. MiR-29s function through targeting AKT2 and *CCND2* directly. (**a**) Immunoblotting of Akt2 and CCND2 in THP1 that were transfected with inhibitor control or miR-29c inhibitor (or miR-29a inhibitor) for 24 h and then subsequently treated for another 48 h with si_AKT2 (or si_CCND2) and si_con. (**b, c**) Cell growth curves (**b**) and Annexin V/PI assays (**c**) in THP1 cells transfected with different combinations.



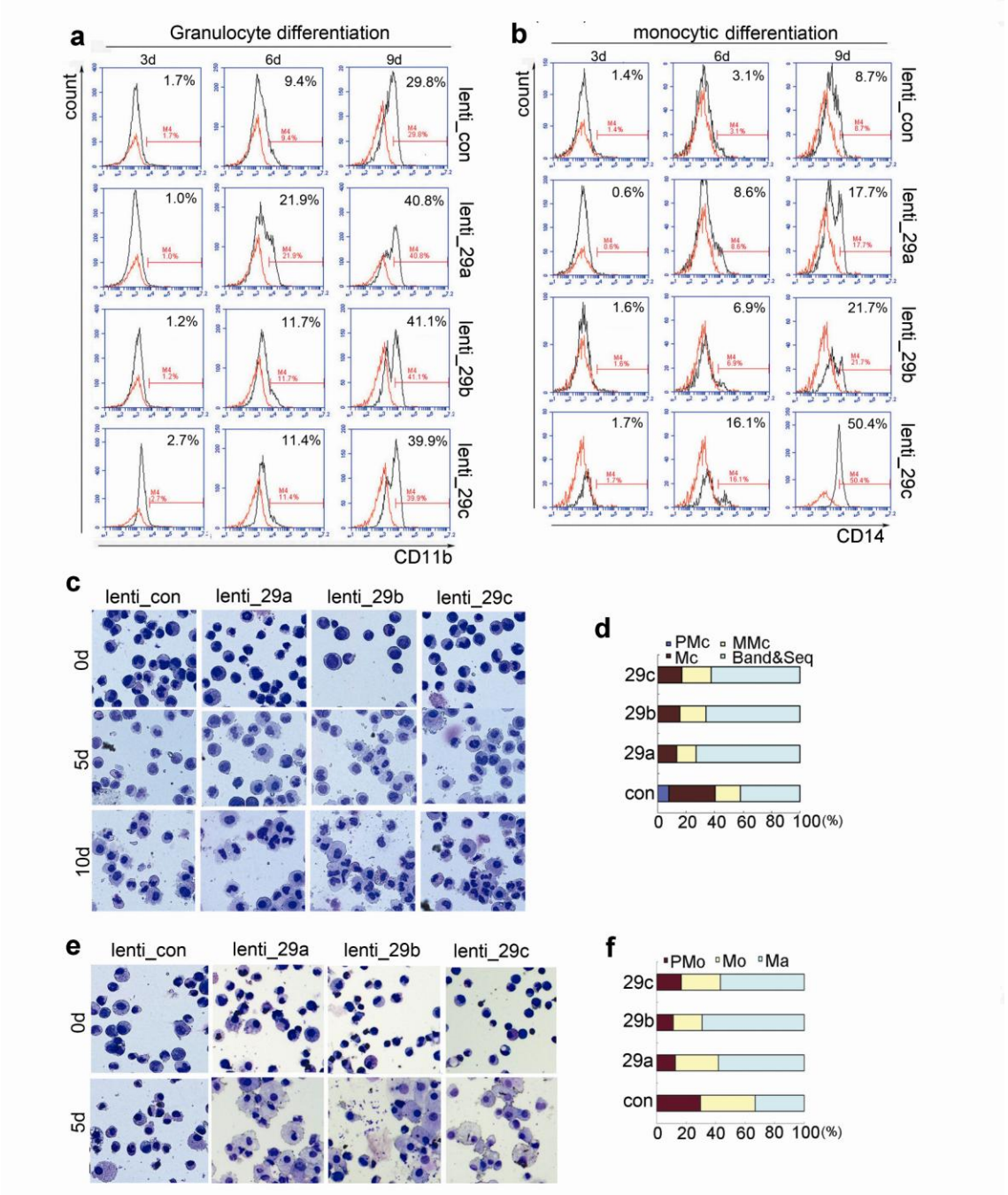
Supplementary Figure S5. Cell cycle distribution in THP1 and NB4 cells after transfection with si_CCND2 and si_con for 48 h.



Supplementary Figure S6. Western blot analysis of Akt2 and CCND2 expression in the PBMNCs derived from 23 healthy donors and 20 AML patients.

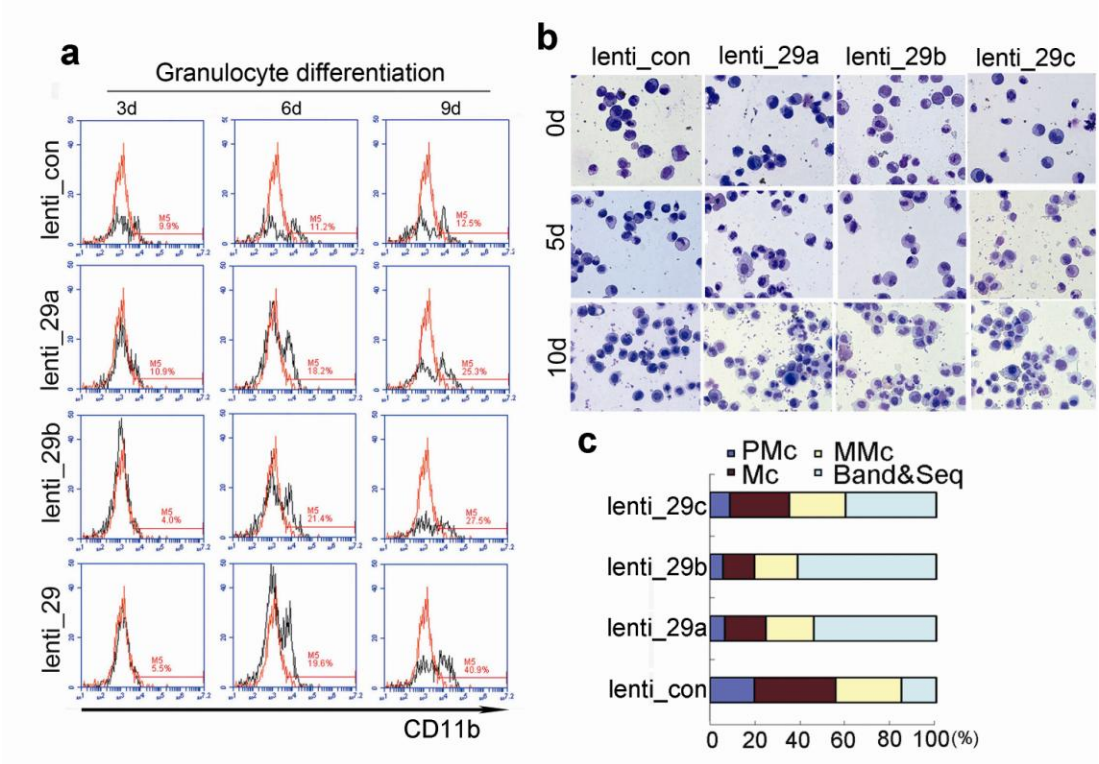


Supplementary Figure S7. Western blot analysis of c-Myc expression in the PBMNCs derived from 23 healthy donors and 20 AML patients.



Supplementary Figure S8. Re-introduction of the miR-29 members into BM CD34+ cells derived from an AML M4 patient (#6) can partially overcome monocytic differentiation and granulocytic differentiation arrest simutaneously. BM CD34+ cells from the patient were infected with lenti_29a, lenti_29b, lenti_29c and lenti_con respectively, and then induced to monocytic and granulocytic differentiation simultaneously after infection for 24 h. Cells were obtained at the indicated times and the GFP-positive cells were collected for myeloid differentiation analysis. The expression levels of CD11b (**a**) and CD14 (**b**) were analyzed by FACS. The cell morphology of granulocytic (**c**) and monocytic (**e**) differentiation at different stages were detected by May-Grünwald-Giemsa staining. The cell numbers at different stages during granulocytic differentiation (**d**, at day 10) and monocytic differentiation (**f**, at day

5) were counted. In brief, 100 cells from three different scopes were counted independently for per sample and the average was shown. Mb: myeloblasts; PMc: promyelocytes; Mc: myelocytes; MMc: metamyelocytes; Band and Seg; band neutrophils and segmented neutrophils for granulocytic differentiation, and Mob: monoblasts; PMo: promonocytes; Mo: monocytes; Ma: macrophage for monocytic differentiation.



Supplementary Figure S9. Re-introduction of the miR-29 members into BM CD34+ cells derived from an AML M2 patient can partially overcome granulocytic differentiation arrest. BM CD34+ cells from an AML patient (#1) who was diagnosed as FAB M2 were infected with lenti_29a, lenti_29b, lenti_29c and lenti_con respectively, and induced to granulocytic differentiation after infection for 24 h. Cells were obtained at the indicated times and the GFP-positive cells were collected. (**a**) Expression level of CD11b was analyzed by FACS. (**b**) May-Grünwald-Giemsa staining for detecting the cells at different stages of granulocytic differentiation. (**c**) Statistic analysis of cell numbers at different differentiation stages at day 10 from the differentiation induction was shown.