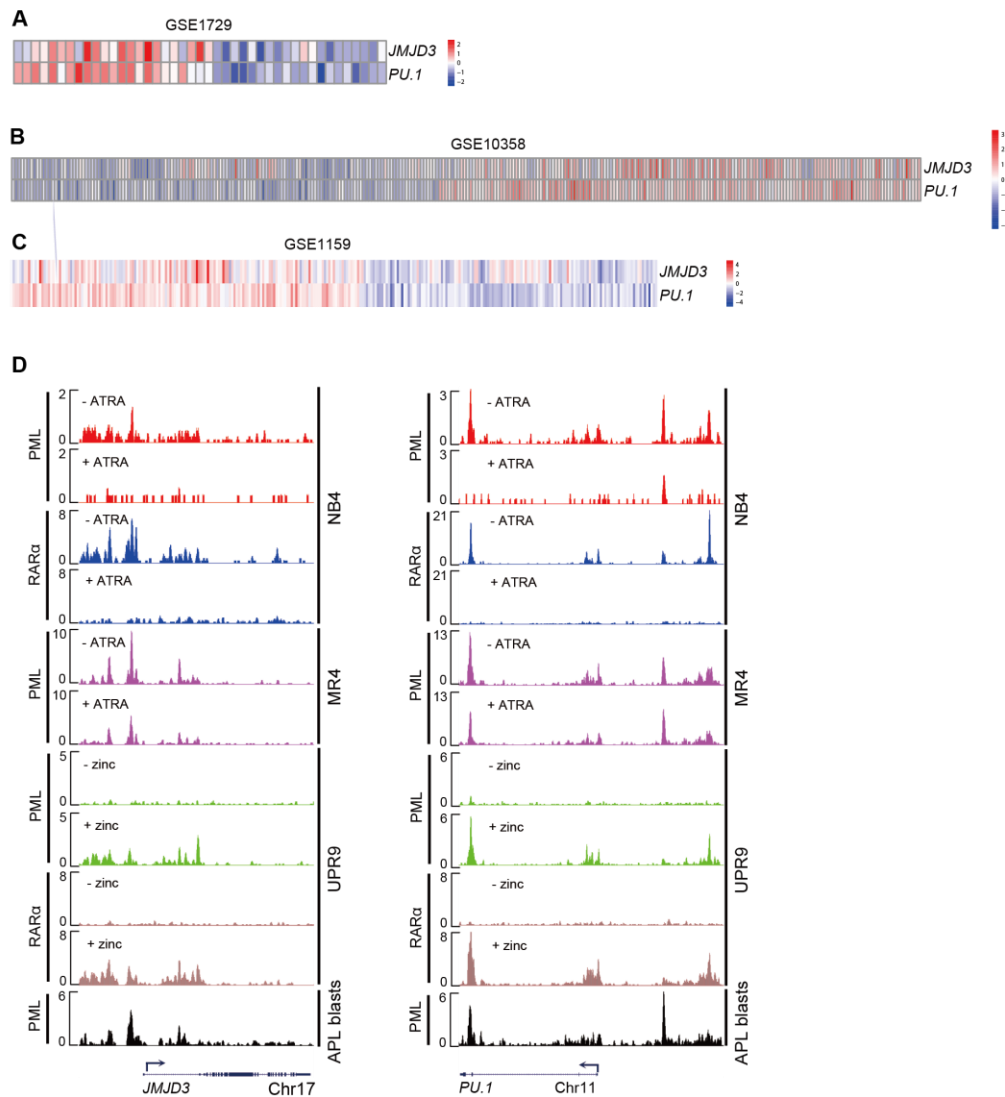


## Supplementary Information

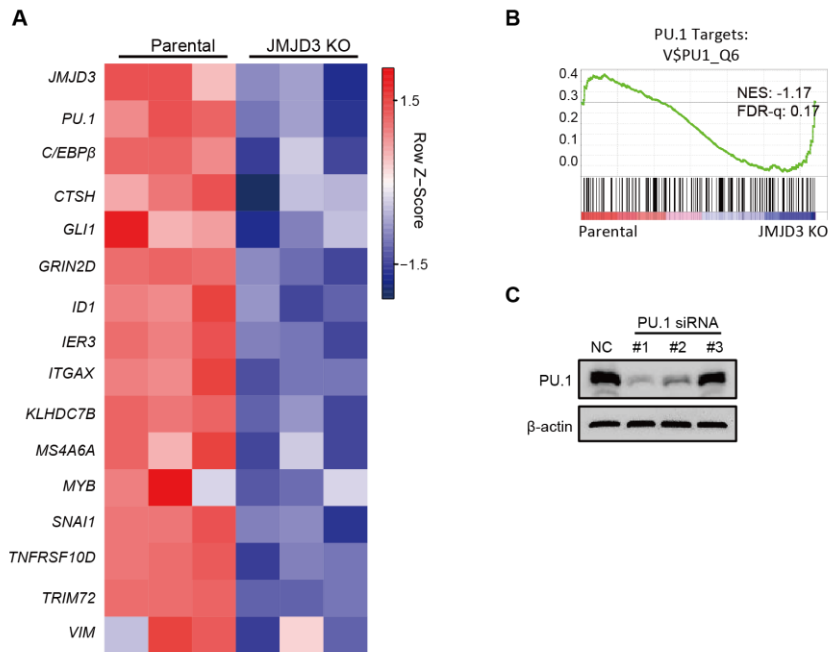
### *JMJD3* exerts oncorepressor activity in acute promyelocytic leukemia by promoting *PU.1* expression

Authors: Meng-Xi Wang, Shan-He Yu, and Juan Chen

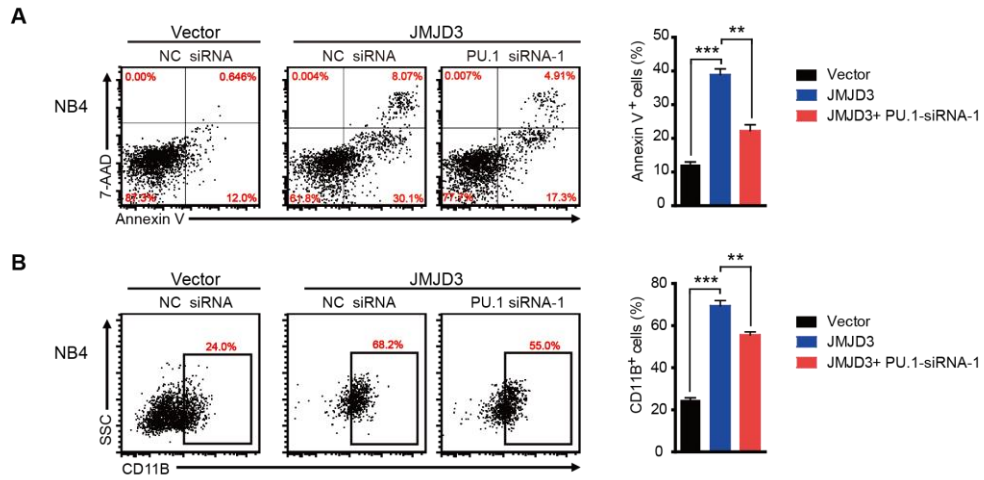


**Figure S1. Correlation between *JMJD3* and *PU.1* in APL.** (A-C) Heatmap of the expression of *JMJD3* and *PU.1* in the BM mononuclear cells of AML patients from GSE1729 (A), GSE10358 (B), and GSE11159 (C). (D) The analyses of published ChIP-seq data revealed that both *JMJD3* and *PU.1*

belong to the target genes of PML and RAR $\alpha$ .



**Figure S2. *PU.1* represents the key target of *JMJD3* in APL.** Differentially expressed genes (DEGs) are classified as those with fold change  $>2$  or  $<0.5$  ( $p < 0.05$ ). (A) Heatmap of DEGs in parental and JMJD3 KO HL-60. (B) GSEA of parental and JMJD3 KO HL-60. The gene set of *PU.1* targets was used. (C) Verification of the effects of *PU.1* siRNAs in HL-60. Western blot was conducted to determine the effect of knockdown.



**Figure S3. JMJD3 exhibited anti-human AML activity in a PU.1-dependent manner.** (A) Flow cytometric analyses of Annexin V (A), and CD11B (B) in NB4 transduced with empty vector, JMJD3-expressing vector, or JMJD3-expressing vector plus PU.1 siRNA-1.