

Targeting the PI3K/AKT pathway via GLI1 inhibition enhanced the drug sensitivity of acute myeloid leukemia cells

Hui Liang¹, Qi-Li Zheng¹, Peng Fang¹, Jian Zhang², Tuo Zhang⁷, Wei Liu⁵, Min Guo⁶, Christopher L. Robinson⁴, Shui-bing Chen⁴, Xiao-Ping Chen^{3, 8*}, Fang-Ping Chen^{2*}, and Hui Zeng^{1*}

¹ Department of Hematology, Xiangya Hospital, Central South University, Changsha, Hunan, China; ² Department of Hematology, Third Xiangya Hospital, Central South University, China; ³ Department of Clinical Pharmacology, Xiangya Hospital, Central South University, China; ⁴ Department of Surgery Genomic Core, Weill Cornell Medical College, NY, USA; ⁵ Department of Oncology, Xiangya Hospital, Central South University, China; ⁶ Department of Endocrinology, Xiangya Hospital, Central South University, China; ⁷ Genomic Core, Weill Cornell Medical College, NY, USA; ⁸ Institute of Clinical Pharmacology, Central South University, China.

Supplementary Figures with captions

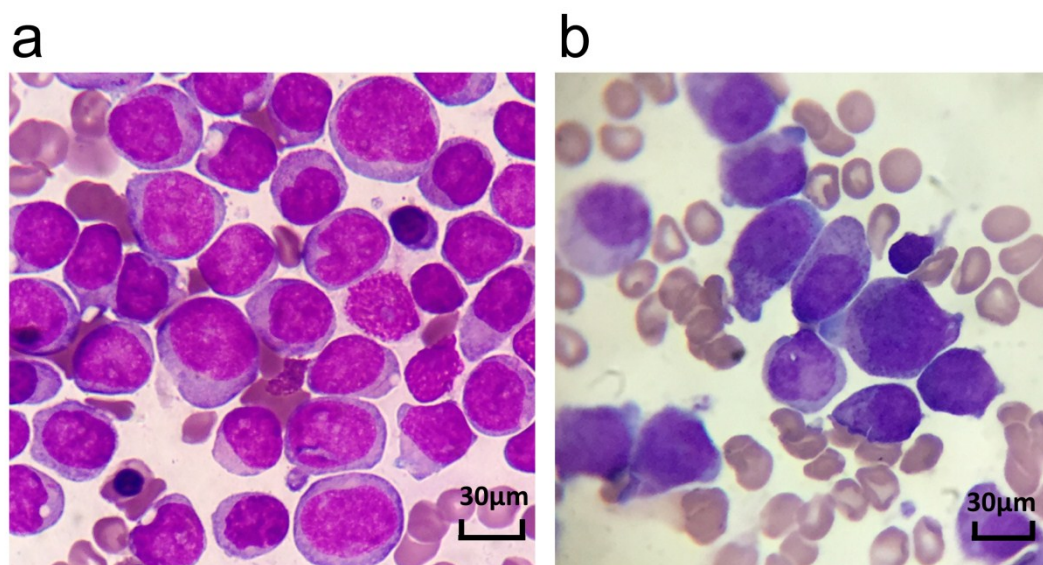


Figure S1. Bone marrow biopsies for AML patients (Wright–Giemsa staining).10×100 (a) Bone marrow biopsy for M2 AML patient. (b) Bone marrow biopsy for M3 AML patient.

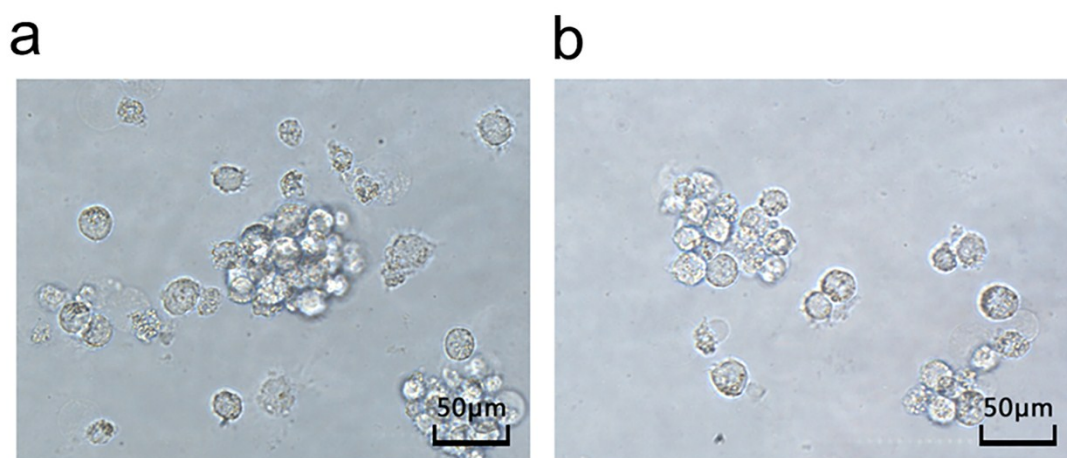


Figure S2. Growth of HL60 (a) and NB4 (b) cells.10 × 40

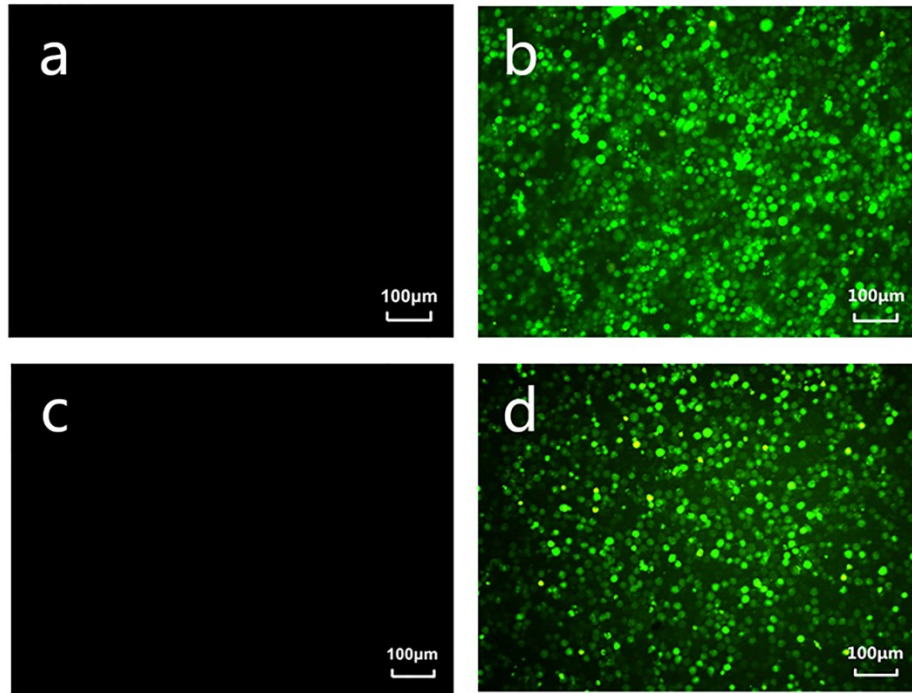


Figure S3. Growth of HL60 (ab) cells and NB4 (cd) under an inverted microscope by using fluorescence microscopy after transfection for 72h. 10×10

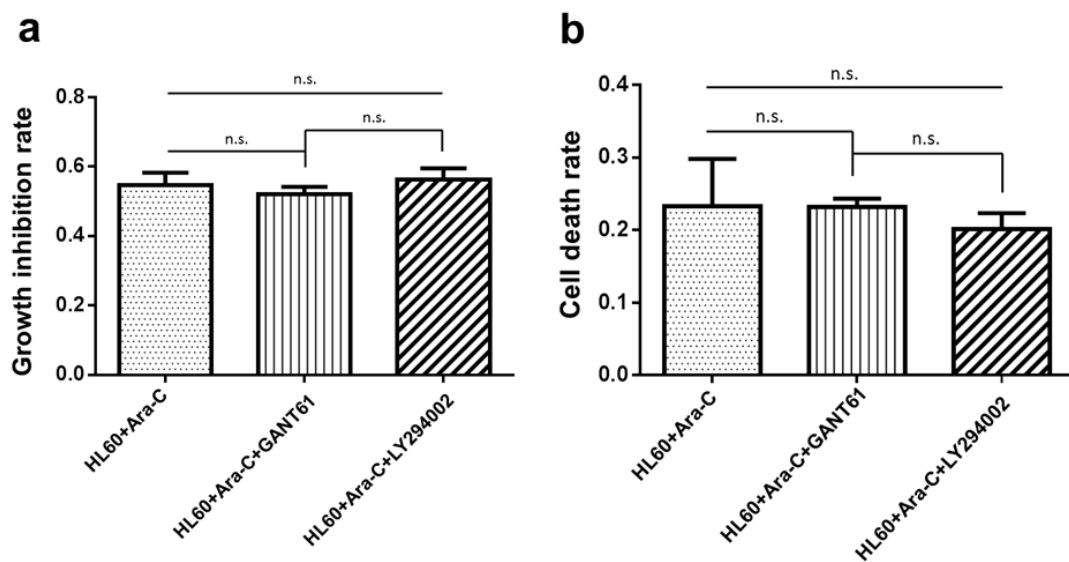


Figure S4. The growth inhibition rate (a) and cell death rate (b) in wild-type HL60 cells treated with Ara-C and combined with GANT61 or LY294002