Mithramycin A suppresses basal triple-negative breast cancer cell survival partially via down-regulating Krüppel-like factor 5 transcription by Sp1

Rong Liu^{1#}, Xu Zhi^{2#}, Zhongmei Zhou¹, Hailin Zhang¹, Runxiang Yang³, Tianning Zou⁴, Ceshi Chen^{1*}

¹Kunming Institute of Zoology, Chinese Academy of Sciences, Key Laboratory of Animal Models and Human Disease Mechanisms of Chinese Academy of Sciences & Yunnan Province, Kunming, 650223, China

²Center for Reproductive Medicine, Department of Obstetrics and Gynecology, Peking University Third Hospital, Beijing 100191, China

³Department of Oncology, Yunnan Tumor Hospital, The Third Affiliated Hospital of Kunming Medical University, Kunming, Yunnan, 650118, China

⁴Department of Breast Surgery, Yunnan Tumor Hospital, The Third Affiliated Hospital of Kunming Medical University, Kunming, Yunnan, 650118, China

Supplementary Figure legends

Figure S1. MIT suppresses HCC1937 xenograft growth and induces apoptosis *in vivo*.

A. MIT suppressed HCC1937 tumor growth in Balb/c nude mice. HCC1937 cells were injected into the fat pat of female Balb/c nude mice. When the average tumor size reached approximately 100 mm³ after inoculation, the mice were randomly and equally distributed into two groups (n=6/group): saline control and 0.05 or 0.15 mg/kg MIT/d. Tumor size were measured twice per week for 7 weeks. Tumors were collected 7 weeks after MIT treatment.

B. MIT significantly decreased tumor weights compared to the saline control group (**, p<0.01, t-test).

C. MIT did not decrease the body weight of mice. The mice were weighed at the end of the experiment.

D-E. MIT suppressed HCC1937 cell proliferation and promoted apoptosis *in vivo*. Tumors collected from saline control and MIT groups were paraffin-fixed, sliced and stained with anti-ki-67 (D) or cleaved-caspase 3 (E). (*, p<0.05, t-test). The quantitative results are shown on the right.



Figure S1