

Corrections

CELL BIOLOGY. For the article “Activation of the PTEN/mTOR/STAT3 pathway in breast cancer stem-like cells is required for viability and maintenance,” by Jiangbing Zhou, Julia Wulfkuhle, Hao Zhang, Peihua Gu, Yanqin Yang, Jianghong Deng, Joseph B. Margolick, Lance A. Liotta, Emanuel Petricoin III, and Ying Zhang, which appeared in issue 41, October 9, 2007, of *Proc Natl*

Acad Sci USA (104:16158–16163; first published October 2, 2007; 10.1073/pnas.0702596104), the authors note that the legend for Fig. 1 appeared incorrectly in part. The figure and its corrected legend appear below. This error does not affect the conclusions of the article.

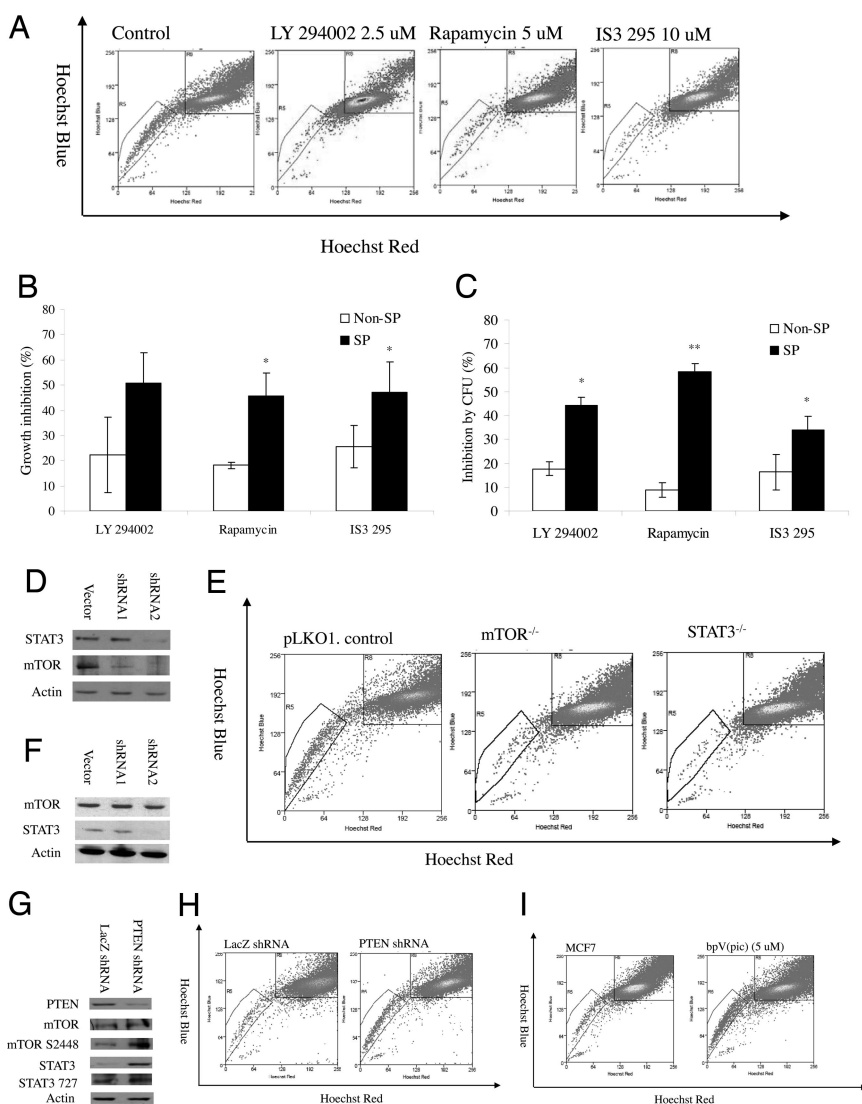


Fig. 1. Importance of PI3K/mTOR, STAT3, and PTEN signaling for the SP cells. (A) Decrease of the SP fraction within MCF7 cells by pathway-specific inhibitors. (B) Proliferation inhibition effects of LY294002 (2.5 μ M), rapamycin (5 μ M), and IS3 295 (50 μ M) on MCF7 SP and non-SP cells. (C) Colony formation inhibition effects of LY294002 (2.5 μ M), rapamycin (5 μ M), and IS3 295 (25 μ M) on MCF7 SP and non-SP cells. (D) Western blot analysis of expression of STAT3 and mTOR in mTOR knockdown cells. (E) Decrease of SP fraction in stable mTOR and STAT3 knockdown MCF7 cells. (F) Western blot analysis of expression of STAT3 and mTOR in STAT3 knockdown cells. (G) Negative regulation of STAT3 and mTOR expression by PTEN, shown by Western blot. (H) Increase of SP fraction in MCF7 cells by PTEN knockdown. (I) Increase of SP fraction in MCF7 cells treated with PTEN-specific inhibitor bpV(pic).

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