Correction: Adaptive Reprogramming of De Novo Pyrimidine Synthesis Is a Metabolic Vulnerability in Triple-Negative Breast Cancer

In this article (Cancer Discov 2017;7:391–9), which was published in the April 2017 issue of Cancer Discovery (1), an error in Fig. 2D was introduced by the compositor during the production of the paper. Specifically, Western blot data appearing for CAL-51 cells were duplicated to also appear for MDA-MB-231 cells. The online version of the article has been changed to show the correct data for MDA-MB-231 cells, and the amended figure is included below. The publisher regrets this error and apologizes to the authors and community for any inconvenience this may have caused.

**Figure 2.** Chemotherapy exposure alters the phosphorylation state of CAD to stimulate de novo pyrimidine nucleotide synthesis. A, SUM-159PT cells were treated with 0.5 μmol/L doxorubicin for the indicated times, and the phosphorylation states of CAD, ERK, and S6K1 were monitored by immunoblotting. B, SUM-159PT cells were pretreated with 5 μmol/L U0126 for 12 hours before a 4-hour exposure to 0.5 μmol/L doxorubicin, and the phosphorylation states of CAD, ERK, and S6K1 were monitored by immunoblotting. C, SUM-159PT cells were treated with 0.5 μmol/L doxorubicin (dox) for 10 hours, in the absence or presence of 5 μmol/L U0126 or 40 μmol/L PALA, and pyrimidine deoxyribonucleoside triphosphate levels were monitored using a fluorescence-based assay. D, TNBC cell lines (HCC1143, MDA-MB-468, CAL-51, and MDA-MB-231) were treated with 0.5 μmol/L doxorubicin for 4 hours, and changes in CAD phosphorylation were monitored by immunoblotting. E, TNBC cell lines (HCC1143, MDA-MB-468, CAL51, and MDA-MB-231) were treated with 0.5 μmol/L doxorubicin for 10 hours, and pyrimidine deoxyribonucleoside triphosphate levels were monitored using a fluorescence-based assay. All error bars represent SEM. N.S., not significant; *, P < 0.05; **, P < 0.01; ***, P < 0.001 by a Student t-test.

**REFERENCE**
