CORRECTION

Correction: Targeting ErbB-2 nuclear localization and function inhibits breast cancer growth and overcomes trastuzumab resistance

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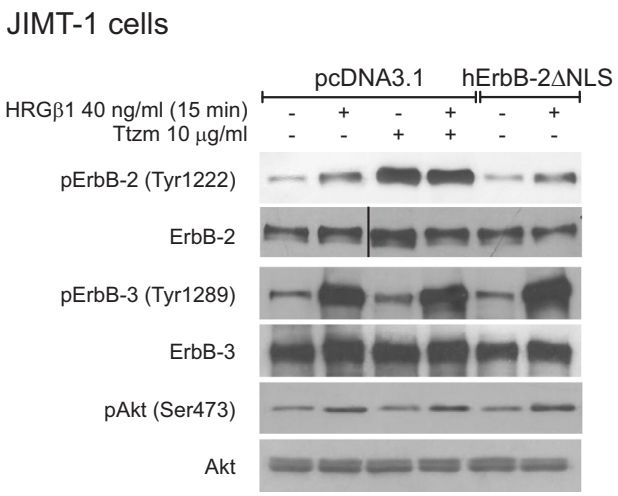
Following the publication of this article, it was noted a mistake in the Western blot (WB) shown in Figure 3f. The band corresponding to JIMT-1 cells stimulated with HRGβ1 and treated with trastuzumab in the second panel of Figure 3f (WB revealed with the ErbB-2 antibody) was inadvertently duplicated. This mistake occurred when bands in the original WB were spliced to assemble Figure 3f. The authors have provided the correct raw data and a revised Figure 3f which contains a black line indicating that lanes that were non-adjacent in the gel were re-arranged. Therefore, in the revised second panel of Figure 3f it is shown that: (i) protein extracts present in lanes 1 and 2 were adjacent in the gel, (ii) lanes in the middle of the gel were spliced, which is highlighted by a black line (iii) the adjacent lanes 3 to 6 in the gel were assembled along with lanes 1 and 2 to present data in said second panel.

The legend in Figure 3f was modified to duly acknowledge the non-adjacent lanes in the original gel. The revised version reads as:

Revised Legend to Figure 3. Inhibition of ErbB-2 nuclear localization blocks proliferation in Ttzm-resistant BC. (a) Cells transfected with pcDNA3.1 (left) or hErbB-2ΔNLS (right) vectors were treated as indicated. ErbB-2 (red) and GFP-tagged-hErbB-2ΔNLS (green) were localized as in Figure 2. Solid arrows: hErbB-2ΔNLS-transfected cells; dashed arrows: wild-type cells that did not uptake the hErbB-2ΔNLS mutant. Nuclei were stained with 4′,6-diamidino-2-phenylindole (DAPI) (blue). (b–d) Quantitative analysis of ErbB-2 subcellular localization. Fluorescence intensities of MErbB-2 and NErbB-2 were quantified in 50 cells from each group and are plotted as the ratio of MErbB-2/NErbB-2 (b), the percentage of MErbB-2 (c) or the percentage of NErbB-2 (d) (mean ± s.d.). For # vs *$P < 0.001$. For b–e vs a, c–e vs b, and d vs c: $P < 0.001$. For c and d vs e: $P < 0.05$. (e) Blockade of NErbB-2 localization inhibits in vitro growth. Cells were transfected and treated as indicated. Proliferation and data were analyzed in Figure 2. For b and c vs a, and d vs b: $P < 0.001$. (f and g) Effect of hErbB-2ΔNLS on ErbB signaling. Cells were transfected and treated as in (e). WB analysis (f) and immunoprecipitation analysis (g) were performed as in Figure 2. *The black line in the second panel of (f) was added to show that lanes 1 and 2 were adjacent in the gel, lanes in the middle of the gel were spliced and lanes 3 to 6 were adjacent in the gel.* (h) Cell proliferation in intrinsic and acquired Ttzm-resistant cells was evaluated by [^{3}H]thymidine incorporation or cell count. Data are presented as in (e). For b and c vs a, and d vs b: $P < 0.001$. Experiments in (a–h) were repeated three times, with similar results. (i) hErbB-2ΔNLS blocks in vivo growth. Left panel: Cells were inoculated subcutaneously in mice. When JIMT-1 tumors reached 40 mm3, animals were divided into three groups and injected with Ttzm, IgG, or remained untreated. JIMT-1-hErbB-2ΔNLS-injected mice developed tumors with a latency of 40 days. Each point represents the mean tumor volume ± s.e.m. Right panel: Decrease in tumor mass. (j) Tumor growth. [a]Growth rates were calculated as in Figure 1. Volume, percentage of growth inhibition, and delay in tumor growth from the different groups with respect to tumors from JIMT-1 cells were calculated at day 64. # vs *$P < 0.001$. [b]With respect to JIMT-1 cells, $P < 0.001$ (see Supplementary Fig. S2).



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The authors emphasize that under "Results" they have shown the same results as those presented in Fig 3f for JIMT-1 cells in multiple BC cell lines and tumor models: A. Murine BC C4HD cells, which mimic ErbB-2-positive BC ("Nuclear ErbB-2 drives HRGβ1-induced BC growth"); B. An in vivo BC model induced by HRGβ1 ("Nuclear ErbB-2 drives HRGβ1-induced BC growth") and C. BT-474 and SK-BR-3 human BC cells treated with HRG β1 that represent a model comparable to JIMT-1 cells. Particularly, SK-BR-3 cells treated with HRGβ1 are a model closer to JIMT-1, since they lack estrogen and progesterone receptor ("Comparison between hErbB-2ΔNLS and Ttzm effects in ErbB-2 activation and BC growth").

The authors apologize for the mistake in the assembly of Figure 3f, which does not affect the conclusions of the article.