CORRECTION

Correction: MiR-16 mediates trastuzumab and lapatinib response in ErbB-2-positive breast and gastric cancer via its novel targets CCNJ and FUBP1

L. Venturutti, R. I. Cordo Russo, M. A. Rivas, M. F. Mercogliano, F. Izzo, R. H. Oakley, M. G. Pereyra, M. De Martino, C. J. Proietti, P. Yankilevich, J. C. Roa, P. Guzmán, E. Cortese, D. H. Allemand, T. H. Huang, E. H. Charreau, J. A. Cidlowski, R. Schillaci and P. V. Elizalde

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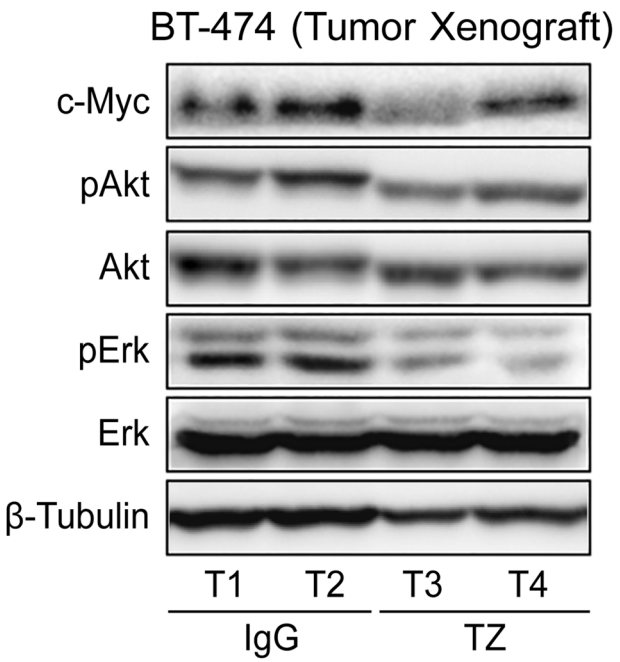
Following publication of this article, mistakes were noted in the panels of the Western blot (WB) in Fig. 3c (right), which shows results in the model of BT-474 cells grown as xenografts in nude mice (Fig. 1b of the article). Specifically, we made the mistakes of placing the results of the WB revealed with CCNE in the panels corresponding to the expression of c-Myc, the phosphorylation state of Akt (pAkt) and the levels of beta tubulin (the loading control) from two representative tumors of mice treated with TZ and two control tumors of mice treated with IgG.

In the preclinical models the amount of protein obtained in extracts from small tumors is low. Therefore, in the experiments of Fig. 3c (right) membranes in the WBs were cut into pieces according to the MW of the protein of interest. Next, the middle part of one membrane, where CCNE (MW 60 kDa) was fist revealed, was stripped and revealed with a c-Myc (MW 62 kDa) antibody. The membrane was again stripped and revealed with a beta tubulin (MW 50 kDa) antibody. Likewise, the middle part of other membrane was revealed with pAkt (MW 56–62 kDa), stripped and revealed with a total Akt antibody, stripped again and revealed with a beta tubulin (MW 50 kDa) antibody. These consecutive incubations of the membranes with three antibodies, the stripping among incubations and the simultaneous incubation with beta tubulin of the membranes which were first revealed with different antibodies, led to the mistakes in Fig. 3c (right). The results on TZ regulation of c-Myc expression in the tumors (Fig. 1b) were not presented in this manuscript. The authors apologize for the mistake. While Fig. 3c (right) shows only two representative tumors from each treatment, the authors have provided the correct raw data on the expression of c-Myc, CCNE, Akt and on the levels of Akt phosphorylation (pAkt) in protein extracts of six tumors from mice treated with TZ and six tumors from mice injected with IgG from the in *vivo* experiment in Fig. 1b. Results on CCNE and its loading control, beta tubulin, were correctly presented in the Supplementary Fig. S1e (right panel BT-474 Tumor Xenografts) and described under the Results Section: "TZ induces miR-16 upregulation in ErbB-2-positive BC and GC sensitive to its antiproliferative effects". The authors note that in the left and middle panels of Fig. 3c the same results as those in BT-474 tumors (right panel) were found in two different breast cancer models: the cell lines BT-474 and SKBR-3.

In this article, the results on the mechanisms of TZ regulation of c-Myc expression (Fig. 3c) were also validated using several experimental strategies in multiple cell lines. They were confirmed by the findings that TZ inability to inhibit Erk1/2 and PI3K/AKT activation in BC cells with intrinsic or acquired resistance (JIMT-1, HCC1569 and a TZ-resistant BT-474 clone) is associated with TZ failure to modulate c-Myc expression (Fig. 3e). Furthermore, they were validated by the evidence that TZ inability to block Erk1/2 and PI3K/AKT prevents the downregulation of c-Myc levels in TZ-resistant GC cell lines NCI-N87 and SNU-I (Fig. 3f).



The mistake in Fig. 3c does not affect the results or conclusions of the article.

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