MOLECULAR PHARMACOLOGY

Correction to: "Human Chorionic Gonadotropin Modulates Prostate Cancer Cell Survival after Irradiation or HMG CoA Reductase Inhibitor Treatment"

The above article [Yacoub A, Hawkins W, Hanna D, Young H, Park MA, Grant M, Roberts JD, Curiel DT, Fisher PB, Valerie K, Grant S, Hagan MP, Dent P (2007) *Mol Pharm* **71**:259–275], contained the following errors:

It has been pointed out to us that the control 0 min time point bands in Figure 2E were duplicated. The control lanes were purposefully placed as duplicates for illustrative purposes, and the Figure Legend inadvertently neglected to mention this. To remedy this issue, these and related experiments corresponding to Figure 2D(i) and Figure 2E of the original manuscript were repeated and the new data are shown here as Panel A and Panel B, respectively.

In Panel A (corresponding to Figure 2D(i)), we observe that hCG increased ERBB1 Y1173 phosphorylation over a 2h time course that was very similar to the originally published data. The ERBB1 Y845 phosphorylation also exhibited a similar pattern of phosphorylation, though the level of increased phosphorylation did not exhibit as high a peak at 60 min and at 90 min as was presented in the originally published data. The hCG –stimulated phosphorylation of ERBB1 Y1068 and ERBB1 Y1173 in the present studies and those in our previously published data were very similar. However, repeat data for P-ERB1(Y992) was omitted due to a poor antibody that failed to recognize the substrate. It should be noted that the data in this revised Figure 2D(i) does not impact the major findings of the paper.

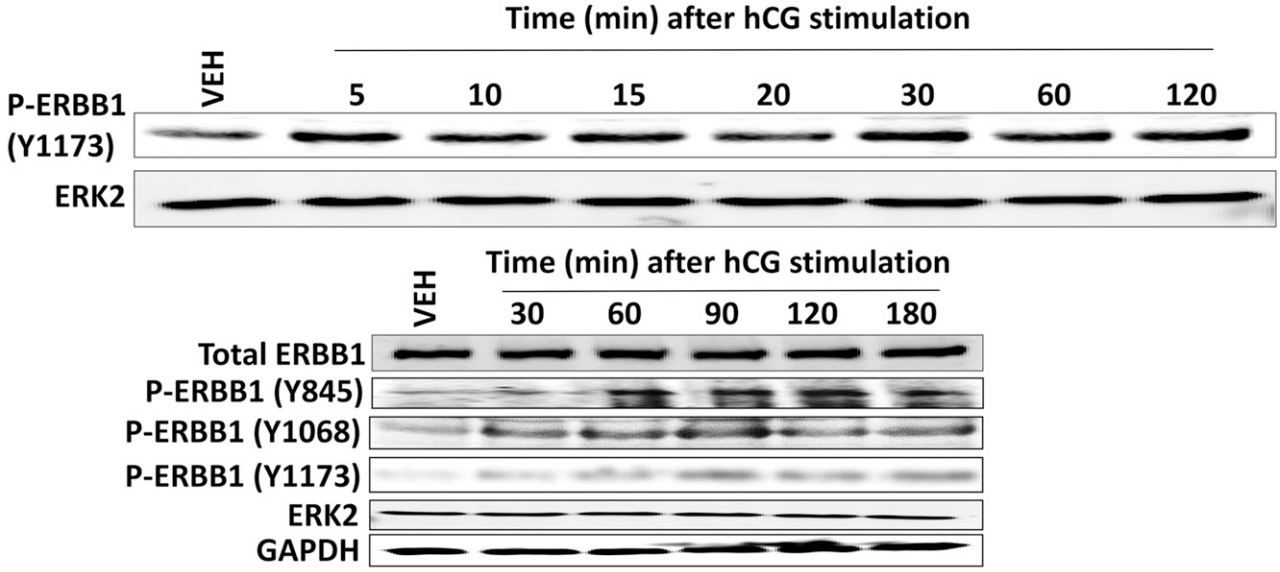
Panel B presents new data corresponding to original Figure 2E with independent vehicle controls at each time point. LNCaP cells, 24 hours after plating, were treated with 2 mU/ml hCG or its vehicle (phosphate-buffered saline). After hCG treatment (0-180 min, as indicated), cells were isolated, lysed in SDS-PAGE sample buffer, and equal protein concentrations were subjected to SDS-PAGE followed by immunoblotting to determine the phosphorylation status of ERBB1 Tyr1068/Tyr1173/ Tyr845 and of ERK1/2 and the expression level of ERK2 and ERBB1 protein. Repeat data for P-ERB1(Y992) was omitted due to a poor antibody that failed to recognize the substrate. The data presented herein are representative of multiple experiments, n = 3.

In Panel B we previously observed that both hCG and ionizing radiation individually increased ERBB1 Y1173 and ERBB1 Y1068 phosphorylation after 30 min and 180 min, and similar data were obtained in the present studies. In the prior studies combined treatment with hCG and radiation caused a modest further increase that was not observed in our present assays. Similar to the original studies, we observed that hCG and radiation promoted an increase in ERBB1 Y845 phosphorylation after 30 min, but not at 180 min.

For treatments with radiation that were also associated with AKT T308 phosphorylation, present studies observed greater levels of AKT S473 phosphorylation compared to the modest increase observed in the original studies. The changes in ERK1/2 and in JNK1/2 phosphorylation in the present studies and those previously published were very similar though in the present studies the synergy of interaction for ERK1/2 activation was less apparent. It should be noted that the data in this revised Figure 2E does not impact on the major findings of the paper.

The authors regret these errors and any inconvenience it may have caused.

Panel A, corresponding to Fig. 2D(i)



Panel B, corresponding to Fig. 2E