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Cancer Research

Correction

Correction: Interaction between FGFR-2, STAT5, and Progesterone Receptors in Breast Cancer

In this article (Cancer Res 2011;71:3720–31), which was published in the May 15, 2011, issue of *Cancer Research* (1), there are several typographical errors in the references called out in the Results and Discussion sections. The applicable passages including the correct reference callouts are provided below:

Results, page 3724, last paragraph and page 3725, the first two lines:
To investigate whether the overlapping functions of MPA and FGF2 were observed for endogenous gene products, we evaluated on T47D cells, the mRNA levels of 2 progestin-regulated genes: *CCND1* (45) and *MYC* (29). MPA and FGF2 induced a time course–dependent increase in *CCND1* and in *MYC* mRNA expression (Fig. 5A). The increase of the latter was abolished when cells were cotreated with MPA and the FGFR inhibitor or with FGF2 and the anti-progestin (Fig. 5A, bottom). The expression of CCND1, MYC, and several progestin-regulated proteins such as BCL-XL (30), tissue factor (TF; 31),. . .

Discussion, page 3728, first column, second paragraph:
We report that FGF2 and MPA induced ERK, AKT, and STAT5 activation, in agreement with a recent report showing that FGF2 activates STAT5 in endothelial cells by a SRC- and JAK2-dependent mechanism (9).

Discussion, page 3728, second column, last paragraph:
When we moved to a more physiologic setting, CCND1 and MYC mRNA were also increased by both ligands as well as all the MPA/STAT5-regulated proteins studied (45, 29).

Reference

1. Cerliani JP, Guillardoy T, Giulianelli S, Vaque JP, Gutkind JS, Vanzulle SI, et al. Interaction between FGFR-2, STAT5, and progesterone receptors in breast cancer. Cancer Res 2011;71:3720–31.

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