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CORRIGENDUM

PI3K-dependent cross-talk interactions converge with Ras as quantifiable inputs integrated by Erk

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In our previously published study (Wang *et al*, 2009), we offered quantitative measurements and a kinetic model to delineate Ras- and phosphoinositide 3-kinase-dependent pathways that contribute to the activation of extracellular signal-regulated kinase (ERK). As a part of that study, we reported on the perturbation of the network by expression of a constitutively active variant of Ras, G12V H-Ras. The attendant effects on ERK and Akt phosphorylation kinetics were shown in Figure 1D and Figure 2D of the article, respectively.

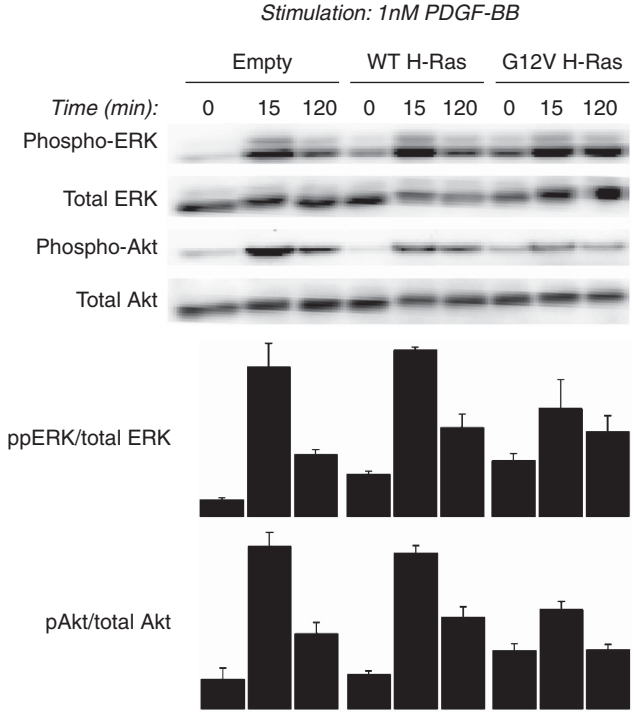

Figure 1 Representative immunoblots and quantification of ERK and Akt phosphorylation in NIH 3T3 cells infected with retrovirus bearing an empty control, wild-type (WT) H-Ras, or G12V H-Ras. The PDGF stimulation conditions were as indicated, and values are quantified as described in the article and reported as mean ± s.e.m. (n=3–6).

Since the publication of the above article, we submitted the vector harboring what was thought to be G12V H-Ras (from the same glycerol stock) for resequencing. The results from multiple sequencing reactions definitively showed the gene to be wild-type H-Ras, not G12V H-Ras. We regret that this error occurred; however, it does not substantively change our interpretation of the data. Overexpression of wild-type Ras results in the same qualitative effect that was desired: an increase in the amount of intracellular Ras-GTP. Perhaps more importantly, the data presented in Figures 1D and 2D were not used in any way in the formulation or parameterization of the kinetic model.

Given that the properties of Ras mutants are of sufficiently broad interest to the cell signaling community, we thought it important to report the effects of expressing wild-type and genuine G12V H-Ras (generously provided by Carla Mattos, Molecular and Structural Biochemistry, NCSU) in our cells, using the same methods as in our published article. The results are presented in Figure 1 below. Relative to the empty vector control, expression of wild type and G12V H-Ras elicit a progressive increase in the basal ERK phosphorylation level and, in the case of G12V H-Ras, a modest reduction in the peak PDGF-stimulated ERK phosphorylation level. As shown in Figure 1, Akt phosphorylation is similarly affected. These quantitative effects of G12V H-Ras expression await further characterization.

References

Wang C-C, Cirit M, Haugh JM (2009) PI3K-dependent crosstalk interactions converge with Ras as quantifiable inputs integrated by Erk. *Mol Syst Biol* **5**: 246