


CORRECTION



Correction to: VEGF_{165b}, a splice variant of VEGF-A, promotes lung tumor progression and escape from anti-angiogenic therapies through a β 1 integrin/VEGFR autocrine loop

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Following publication of this article, the authors became aware of errors in several figures.

When preparing the combined Figure 3a, two P-VEGFR2(Tyr1054/1059) bands were inadvertently duplicated. Original P-VEGFR2(-Tyr1054/1059) immunoblots were retrieved. A corrected version of Figure 3a is provided in which the correct PVEGFR2(Tyr1054/1059) immunoblot for H1299 cells is presented.

In Figures 2d (H358 cells) and 5b, the same tubulin immunoblots were inadvertently used to illustrate loading controls. The original tubulin immunoblots of Figures 2d and 5b were retrieved. A corrected version of Figure 5b is provided in which the correct tubulin data is presented.

When preparing the combined Figure 6c, an incorrect BVZ-treated image was inadvertently used. The original BVZ-treated image was retrieved. A corrected version of Figure 6c is provided.

All corrected figures are included below. The results and scientific conclusions of this paper have not been affected by these modifications. The authors apologize to readers for any inconvenience caused.

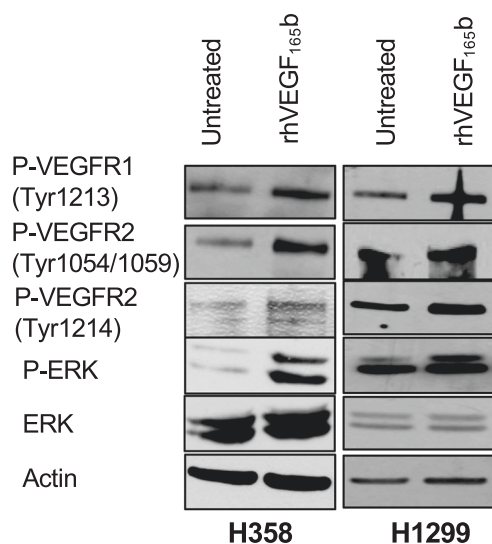


Fig. 3 VEGF165b activates a VEGFR autocrine loop to promote tumor cells proliferation. a Immunoblots for indicated proteins in H358 or H1299 cells treated with rhVEGF165b (0.1 ng/ml) for 72 h (a).

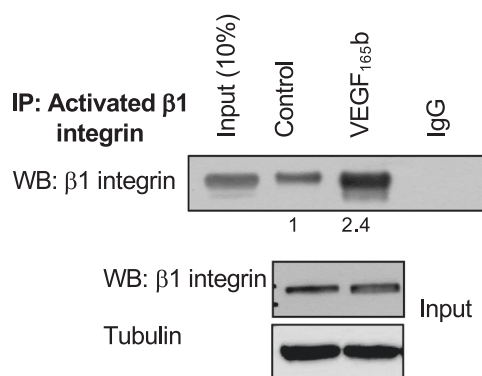


Fig. 5 VEGF165b activates a β 1 integrin signaling pathway to trigger actin cytoskeleton reorganization. b Immunoprecipitation (IP) of activated β 1 integrin in cellular extracts from (b) H358 control or VEGF165b clones.

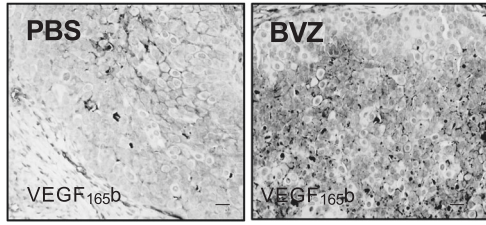


Fig. 6 Anti-angiogenic therapies such as bevacizumab increase VEGF165b protein level and promote the invasive phenotype.
c Representative immunostaining of VEGF165b in H358 subcutaneous xenografts having received, or not, bevacizumab (7.5 mg/kg) once a week. Scale bar = 50 μ m.