Corrigendum



VE-PTP and **VE-cadherin** ectodomains interact to facilitate regulation of phosphorylation and cell contacts

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In the above paper, the induction of the mutant VE-PTP R/A in triple-transfected CHO cells expressing VE-cadherin and Flk (clone RA-7) increased the function of VE-cadherin similarly, as did induction of the wild-type form of VE-PTP (Figure 9D). We have found now that clone RA-7 had erroneously been transfected with wt VE-PTP instead of the mutant form. Analysing new triple transfectants expressing VE-PTP R/A revealed that this mutant did not stimulate the adhesive activity and did not affect phosphorylation of VE-cadherin, as shown in the figure. We wish to stress that the major findings of the paper (association of VE-PTP and VE-cadherin as well as enhancement of VE-cadherin adhesive function by VE-PTP) are not affected by this mistake.

The authors would like to apologize for their mistake.

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Figure 9 VE-PTP, but not VE-PTP-R/A, reduces paracellular permeability in VE-cadherin/Flk-1/VE-PTP triple-transfected CHO and inhibits tyrosine phosphorylation of VE-cadherin. Paracellular permeability for FITC-dextran was analysed with monolayers of various stably transfected CHO cells. Left graph: Analysis of triple-transfected CHO-F12, transfected with VE-cadherin, VEGFR-2 and mifepristone-inducible FLAG-VE-PTP. Stimulation with mifepristone for 10 h resulted in a $45\pm4\%$ reduction of cell permeability (black) as compared to mock-stimulated cells (light grey). Right graph: Analysis of triple-transfected CHO-RA25.5, transfected with VE-cadherin, VEGFR-2 and mifepristone inducible FLAG-VE-PTP-R/A. Stimulation with mifepristone for 10 h resulted in no significant reduction of cell permeability (black) as compared to mock-stimulated cells (light grey). Upper panel: CHO-F12 and CHO-RA25.5 cells, either induced (+) or not induced (-) with mifepristone were analysed by immunoprecipitations with an it VE-cadherin antibody against VE-cadherin, demonstrating equal loading of VE-cadherin.