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CORRECTION

Upon Wnt stimulation, Rac1 activation requires Rac1 and Vav2 binding to p120-catenin

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There was an error published in *J. Cell Sci.* **125**, 5288–5301.

In Fig. 2B, panels labelled as the actin input and p120-catenin IP:p120-catenin panels were inadvertently assembled to show the same blots. Concerns were also raised about possible duplications and/or splices in Figs 4A, 4E, 5D, 6A and 6C. The authors were unable to obtain the original data in order to address these concerns and therefore repeated the experiments. The new data are shown in the figures below and have been verified by the corresponding author's institute as supporting the original conclusions of the study. There are no changes to the figure legends, which are accurate.

The authors acknowledge the increased contribution of Beatriz Del Valle-Pérez in repeating the experiments and apologise to the readers for any confusion that these errors might have caused.

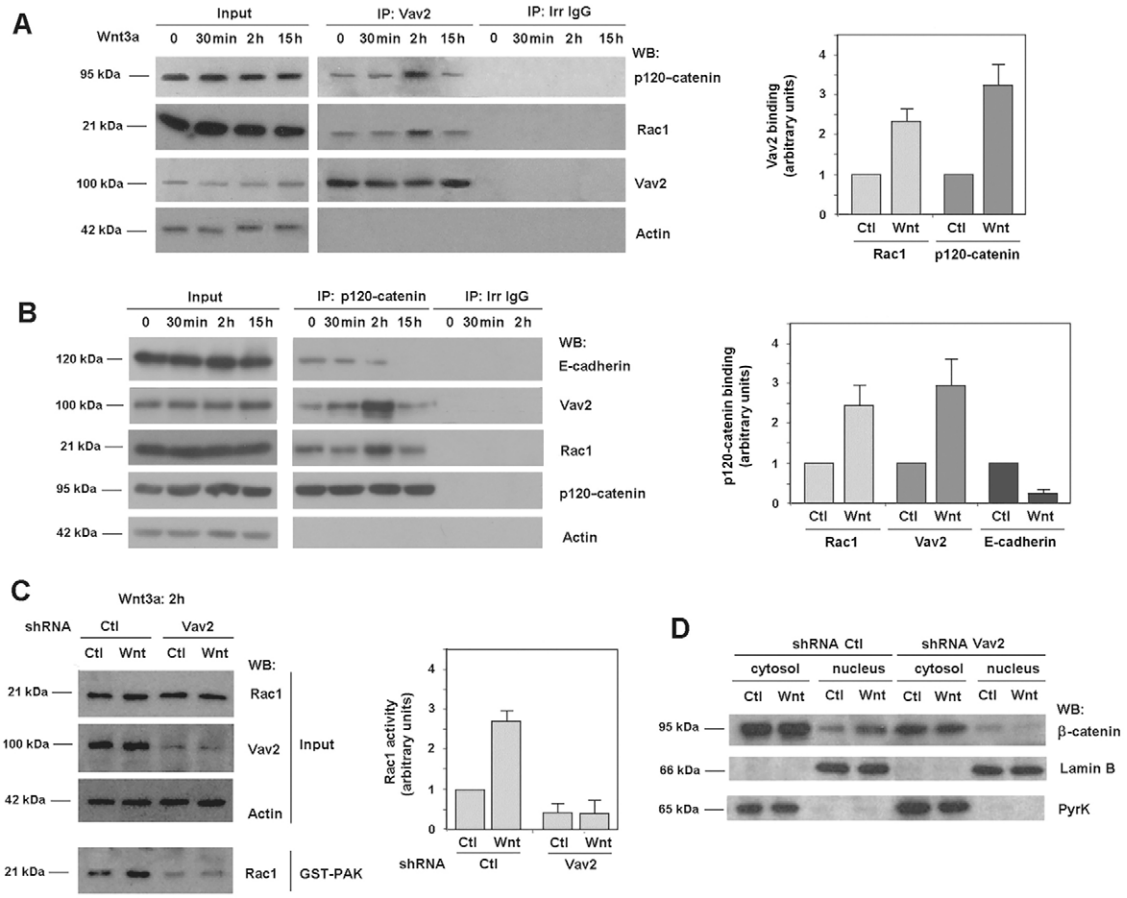

Fig. 2. Wnt-induced Rac1 activation is dependent on Vav2 interaction with p120-catenin. Vav2 (A) and p120-catenin (B) were immunoprecipitated from 500 µg SW-480 whole-cell extracts treated with control or Wnt3a-conditioned medium for the times indicated. Protein complexes were analyzed by western blot (WB) with anti-p120-catenin, anti-Vav2, anti-E-cadherin and anti-Rac1. 10 µg of SW-480 whole-cell extracts were included as internal reference (input). The graphs on the right are autoradiograms from four different experiments that were quantified and the mean ± s.d. obtained after 2 hours of incubation with Wnt3a medium. Each value is presented relative to that obtained in cells treated with control medium and normalized with respect to the amount of immunoprecipitated Vav2 or p120-catenin. (C) SW-480 cells stably expressing scrambled or shRNA specific for Vav2 were treated with control or Wnt3a-conditioned medium for 2 hours. GST–PAK pull-down assays were performed and active Rac1 was determined by WB. Autoradiograms from five different experiments performed in triplicate were quantified and the mean ± s.d. was obtained for each condition. Each value is presented relative to that obtained in nondepleted cells treated with control medium. (D) Cytosolic and nuclear lysates were obtained from control and Vav2-depleted SW-480 cells treated with control or Wnt3a-conditioned medium for 15 hours. β-catenin distribution between the two cell compartments was analyzed by WB.

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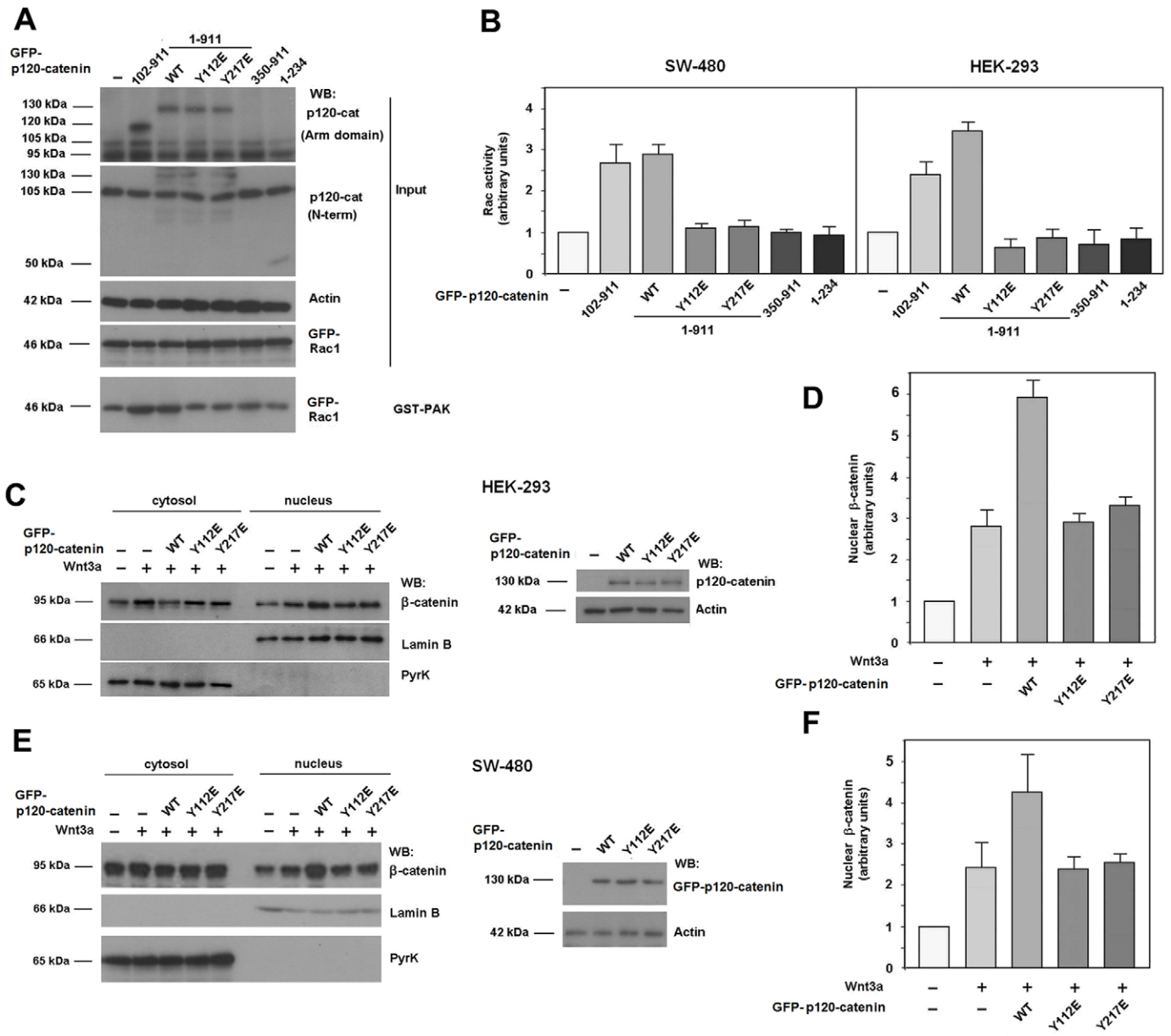

Fig. 4. Only p120-catenin mutants able to interact with Rac1 and Vav2 induce Rac1 activation. (A) GST–PAK pull-down assays were performed in SW-480 cell extracts overexpressing either GFP–p120-catenin (p120-cat) wild-type (wt) isoforms 1 or 3, GFP–p120-catenin 1 harboring point mutants Y112E or Y217E, GFP–p120-catenin deletion mutants (350–911) or (1–234) or the empty vector phrGFP. Active Rac1 was detected by western blot (WB). (B) Autoradiograms from five different experiments performed in triplicate in SW-480 (left panel) and HEK-293 cells (right panel) were quantified and the mean ± s.d. was obtained for each condition. Each value is presented relative to that obtained in nonstimulated cells. (C–F) Cytosolic and nuclear lysates were obtained from HEK-293 (C,D) or SW-480 (E,F) cells overexpressing either GFP–p120-catenin wt isoform 1, GFP–p120-catenin point mutants Y112E or Y217E or the empty vector phrGFP, treated when indicated with Wnt3a-conditioned medium for 15 hours. The nuclear fraction was separated from the cytosolic and membrane-associated fraction as detailed in Materials and Methods. β-catenin levels in each cellular fraction were analyzed by WB. Lamin-β1 was used as a nuclear marker and pyruvate kinase as a marker for the cytosolic-plus-membrane fraction. In the right panel of C, p120-catenin wt, Y112E and Y217E expression levels were analyzed by WB. (D,F) Autoradiograms from four (F) or five (D) different experiments were quantified and the mean ± s.d. was obtained for each condition. Each value is presented relative to that obtained in nonstimulated and nontransfected cells.

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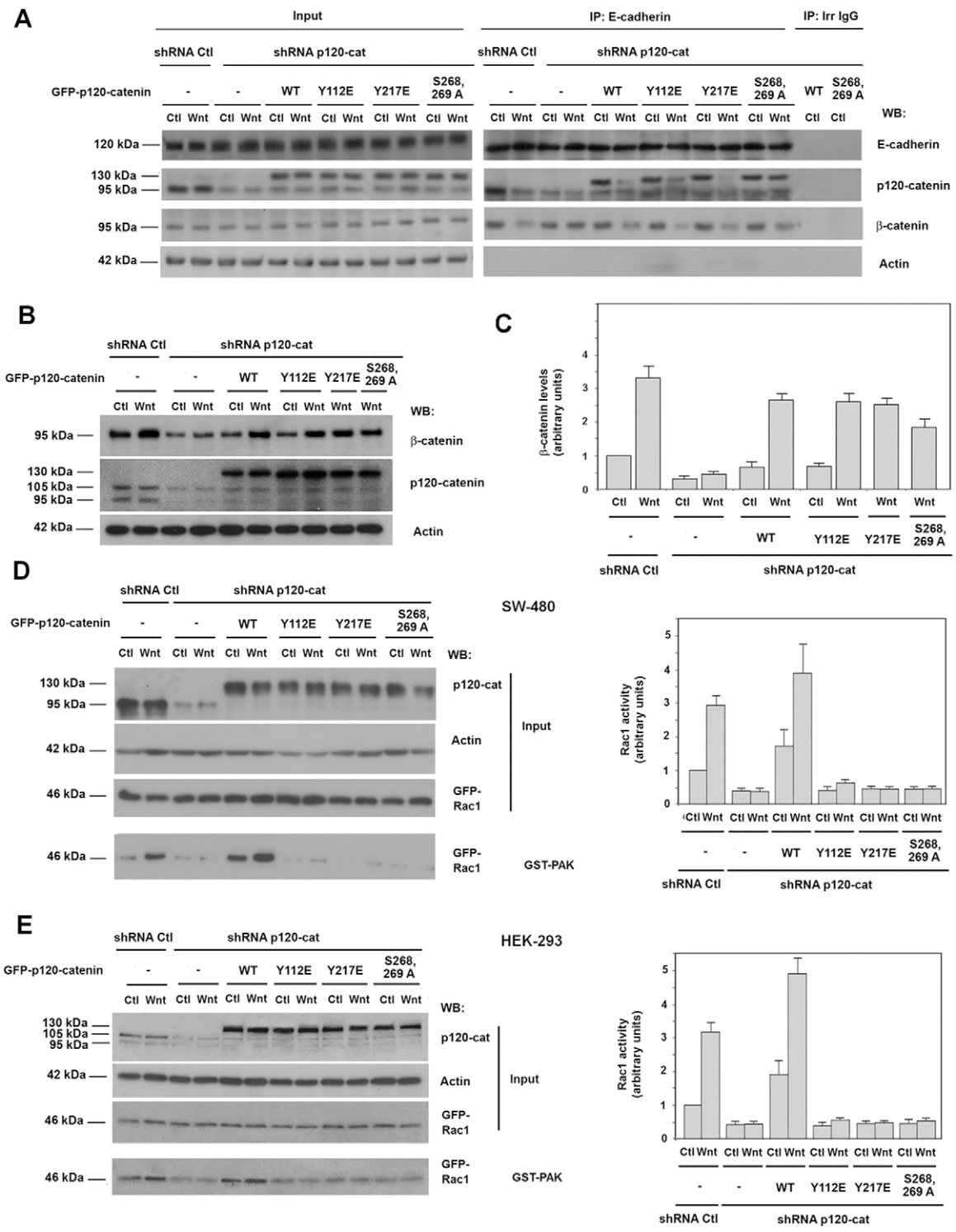

Fig. 5. p120-catenin mutants unable to interact with Rac1 and Vav2 compromise Rac1 activation but not earlier steps of Wnt signaling. (A) E-cadherin was immunoprecipitated from control and p120-catenin (p120-cat)-depleted SW-480 cells overexpressing GFP–p120-catenin wild-type (wt) isoform 1, GFP–p120-catenin 1 point mutants Y112E, Y217E, S268,269A or the empty vector phrGFP and E-cadherin, and treated with control or Wnt3a-conditioned medium for 2 hours. Protein complexes were analyzed by western blot (WB) with the antibodies indicated. (B) Control and p120-catenin depleted HEK-293 cells overexpressing GFP–p120-catenin wt isoform 1, GFP–p120-catenin point mutants Y112E, Y217E and S268,269A or the empty vector phrGFP were treated with control or Wnt3a-conditioned medium for 9 hours and total β-catenin levels were analyzed by WB. (C) Autoradiograms from five different experiments performed as in B were quantified and the mean ± s.d. was obtained for each condition. Each value is presented relative to that obtained in nondepleted and nonstimulated cells. (D,E) p120-catenin-depleted SW-480 (D) or HEK-293 (E) cells overexpressing GFP–p120-catenin wt isoform 1, GFP–p120-catenin 1 harboring point mutants Y112E, Y217E, S268,269A and E-cadherin or the empty vector phrGFP were treated with control or Wnt3a-conditioned medium for 2 hours. GST–PAK pull-down assays were performed and active Rac1 was detected by WB. The graphs on the right show autoradiograms from five different experiments performed in triplicate) that were quantified and the mean ± s.d. obtained for each condition. Each value is presented relative to that obtained in nondepleted and nonstimulated cells.

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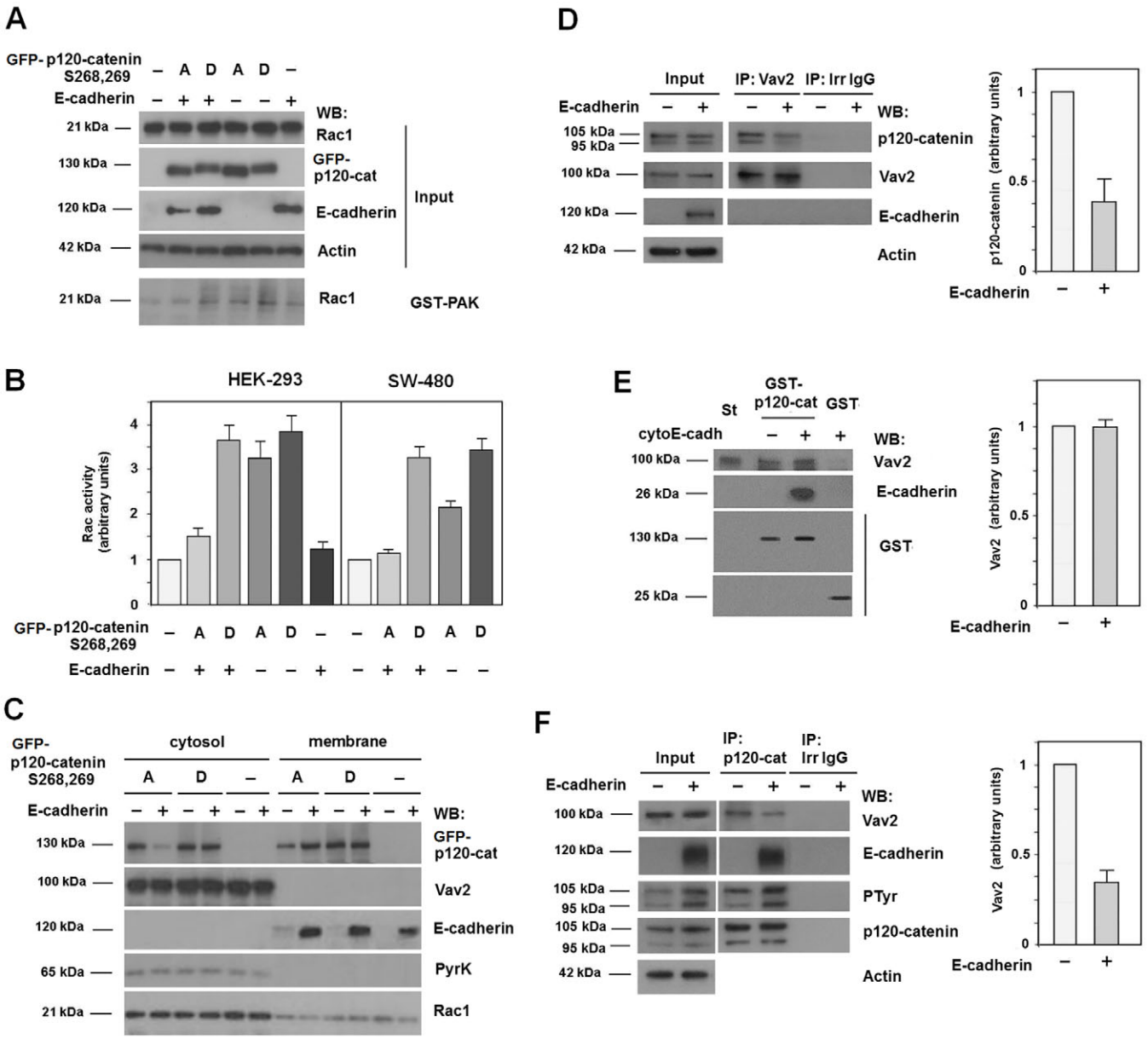

Fig. 6. Only cytosolic p120-catenin interacts with Vav2 and activates Rac1. (A) HEK-293 cells were transfected with GFP–p120-catenin (p120-cat) isoform 1 point mutants S268,269A, S268,269D and E-cadherin or the empty vector phrGFP. GST–PAK pull-down assays were performed and active Rac1 was detected by western blot (WB). (B) Autoradiograms from five different experiments performed in triplicate as in A in SW-480 and HEK-293 cells were quantified and the mean ± s.d. was determined for each condition. Each value is presented relative to that obtained in nontransfected cells. (C) Cytosolic and membrane lysates were prepared from HEK-293 cells coexpressing GFP–p120-catenin 1 harboring point mutants S268,269A or S268,269D and E-cadherin or the empty vector. The subcellular distributions of p120-catenin mutants, Vav2, E-cadherin and Rac1 were analyzed by WB. Pyruvate kinase was used as a marker for the cytosolic fraction. (D) Vav2 was immunoprecipitated from 500 µg HEK-293 whole-cell extracts transfected with either E-cadherin or the empty vector. Protein complexes were analyzed by WB with anti-p120-catenin and anti-Vav2. 5 µg of HEK-293 whole-cell extracts was included as input. (E) GST–p120-catenin isoform 1 or GST (1.5 pmol) were incubated with recombinant Vav2 (3 pmol) in the presence of a tenfold molecular excess of cyto-E-cadherin (15 pmol) when indicated. Protein complexes were affinity purified and analyzed by WB with the indicated mAbs. Vav2 (0.3 pmol) was included as an internal reference standard (St). (F) p120-catenin was immunoprecipitated from 500 µg HEK-293 whole-cell extracts transfected with either E-cadherin or the empty vector. Protein complexes were analyzed by WB with anti-Vav2, anti-E-cadherin, anti-phospho-tyrosine and anti-p120-catenin. 5 µg of HEK-293 whole-cell extracts was included as input. The autoradiograms in D–F correspond to the four different experiments performed; the autoradiograms were quantified and the mean ± s.d. was obtained for each condition. The result obtained after E-cadherin expression was represented relative to the control.

Author contributions

G.V. and M.C. carried out the study and performed the cell culture experiments, participated in the analysis and interpretation of data and prepared the figure panels. B.d.V.-P. and M.V. participated in the biochemical experiments and data analysis. R.M. and P.D.M. participated as collaborators in the design and interpretation of the experiments involving *Xenopus* embryos that were carried out by G.V. (while on an extended visit to Houston) and R.M. C.C. provided essential tools for this study. A.G.d.H. and M.D. conceived the study, participated in the study design and analysis of the data, and wrote the manuscript. To make the needed corrections to this manuscript, B.d.V.-P. repeated the indicated cell culture experiments.