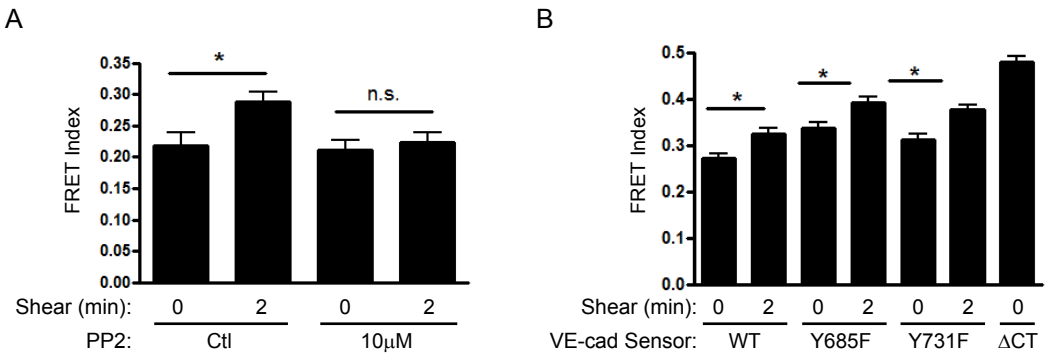

Figure S1. VE-cadherin tyrosine phosphorylation in shear responses, Related to Figure 1. (A) BAECs expressing the VE-cadherin tension sensor were pretreated with or without the src family tyrosine kinase inhibitor PP2 for 30 min at 10μM prior to treatment with or without shear. FRET at cell-cell junctions was imaged and quantified as described in Methods. (B) BAECs expressing WT VE-cadherin tension sensor or the indicated Y-to-F mutants were subject to shear and junctional FRET analyzed as in (A). Results for both A and B were quantified from an experiment with n> 20 junctions per condition. Similar results were obtained in two additional experiments. *$p < 0.05$.

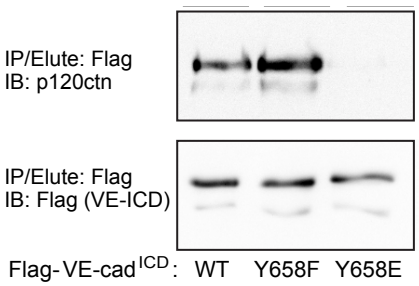

Figure S2. VE-cadherin Tyr658 phosphorylation and p120ctn binding, Related to Figure 3.
(A) Recombinant Flag-tagged VE-cadherin WT, Y658F, or Y658E intracellular domain constructs were purified, bound to anti-Flag resin and used to pull-down p120ctn from HUVEC lysates. Eluates were immunoblotted to detect bound p120ctn.

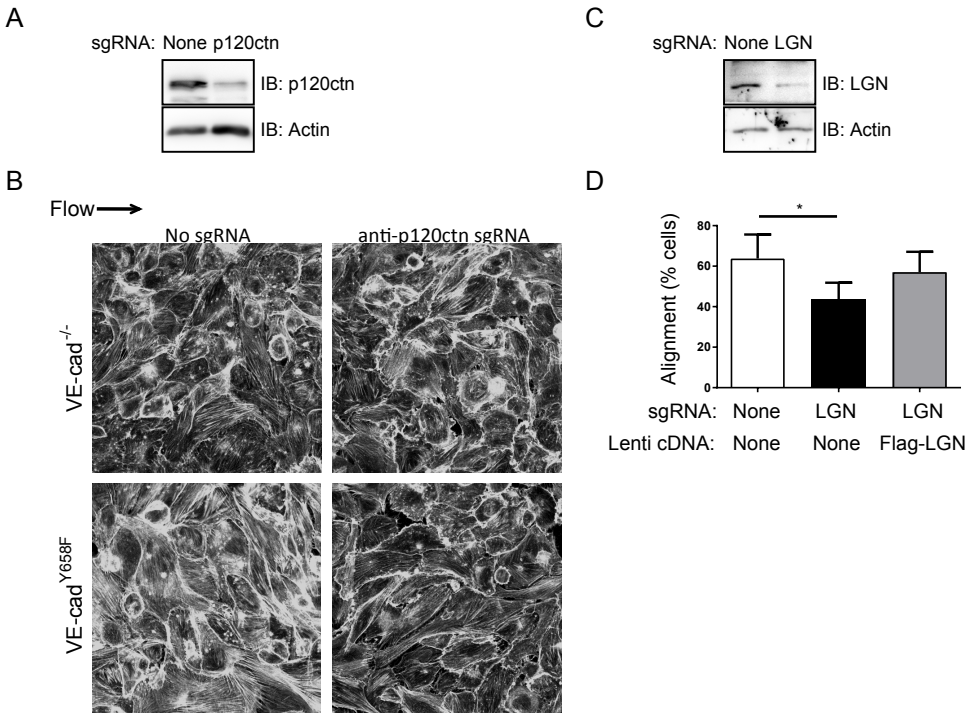

Figure S3. p120ctn and LGN control cell alignment in flow, Related to Figure 4. (A) VE-cadherin-/- cells with or without p120ctn sgRNA lentiviral construct were immunoblotted for p120ctn antibodies to confirm gene disruption. (B) Cells from (A) were transduced with VE-cadY658F and analyzed for shear-induced alignment. Representative images are shown. (C) HUVEC with or without expression of Cas9 and sgRNA targeting LGN immunoblotted for LGN to confirm mutation. (D) Cells from (C) were reconstituted with sgRNA-resistant Flag-tagged LGN and assayed for shear-induced alignment. Values are means ± SEM from 3 independent experiments. *$p < 0.05$.