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CORRECTION

Correction to: PRSS8 suppresses colorectal carcinogenesis and metastasis

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In the original published version Fig. 4c and Fig. 5d are incorrect. The correct figures are given below.

The original article was corrected.

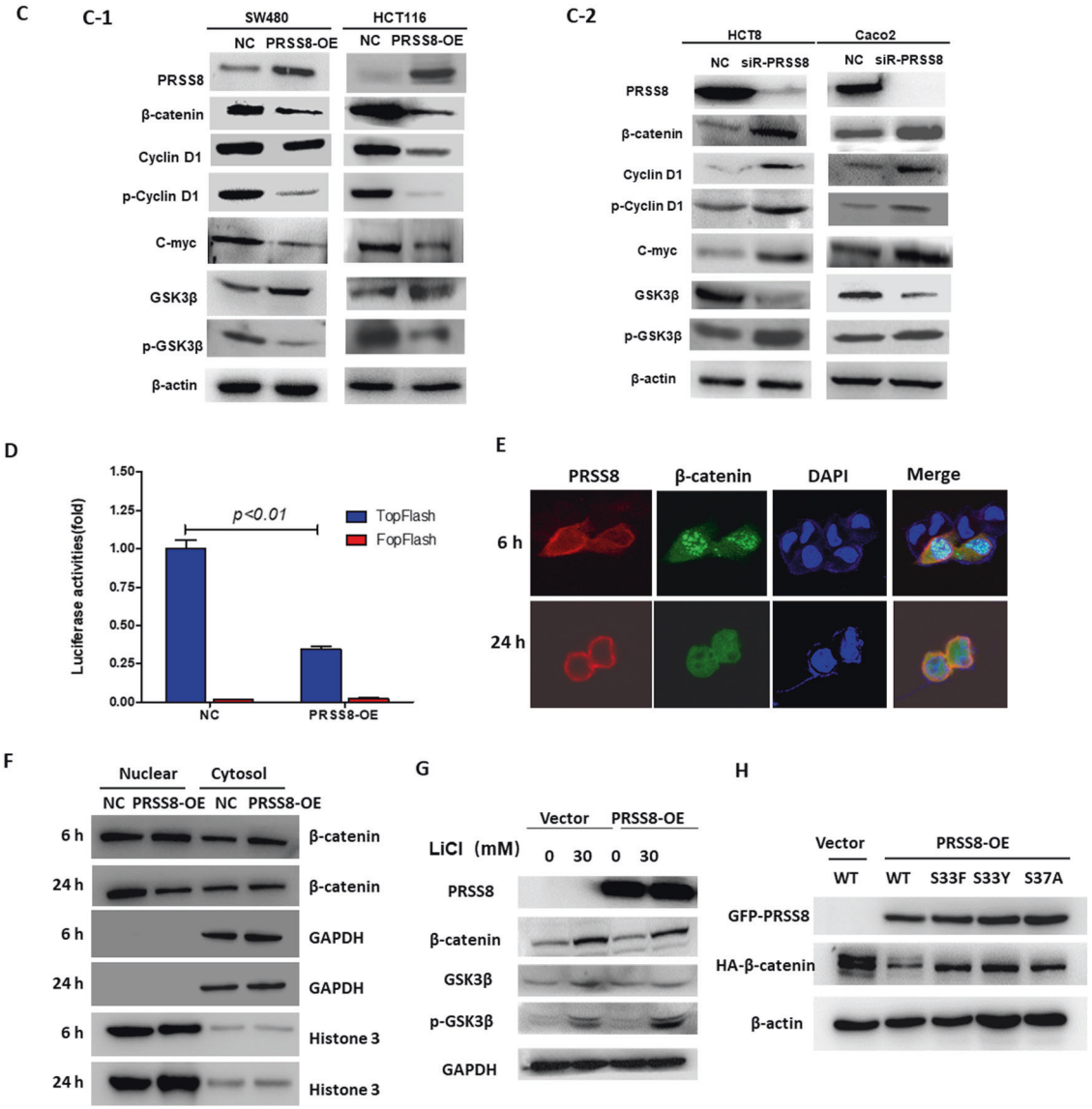

Fig. 4 Molecular mechanisms of PRSS8-mediated suppression of carcinogenesis and metastasis, and crosstalk between PRSS8 and the Wnt/β-catenin pathway. **a** Gene profile on HCt166 cells and gene set enrichment analysis (GSEA) showed that PRSS8 expression is associated with PI3K-AKT, Wnt/β-catenin, epithelial-mesenchymal transition (EMT) and stem cell signaling pathways. **b** RNA sequence on mouse intestinal epithelial cells from *Prss8fl/fl*, *Cre* + and wild-type mice and GSEA showed significant changes in certain major signaling pathways. **c** PRSS8 affected β-catenin signaling at the protein level in SW480, HCT116, HCT8 and Caco2 colon cancer cells transiently transfected with vector only (negative control, NC), PRSS8 over-expression plasmid (PRSS8-OE), or siRNA targeting PRSS8 (siRNA-PRSS8). **d** Increased PRSS8 expression suppressed β-catenin-TCF4 luciferase activity, as assayed by determining TopFlash activity. FopFlash was used as a control. **e**, **f** PRSS8 affected β-catenin cyto-plasmic/nuclear translocation, as determined by immunofluorescence staining and cellular fragment immunoblotting (GAPDH was used as a cytoplasmic loading control, and Histone 3 was used as a nuclear loading control). **g** LiCl caused GSk3bta phosphorylation and β-catenin upregulation (lane 2 vs lane 1), and the phosphorylated GSK3β (p-GSk3β) affected PRSS8-mediated β-catenin degradation (lane 4 vs lane 3), in HCT116 cells. **h** Mutation of GSK3β-mediated phosphorylation sites on the β-catenin protein attenuated PRSS8-induced degradation of the β-catenin protein in HEK293 cells. (WT, wild-type β-catenin; S33F, serine 33 to phenylalanine 33; S33Y, serine 33 to tyrosine 33; S37A, serine 37 to alanine 37).

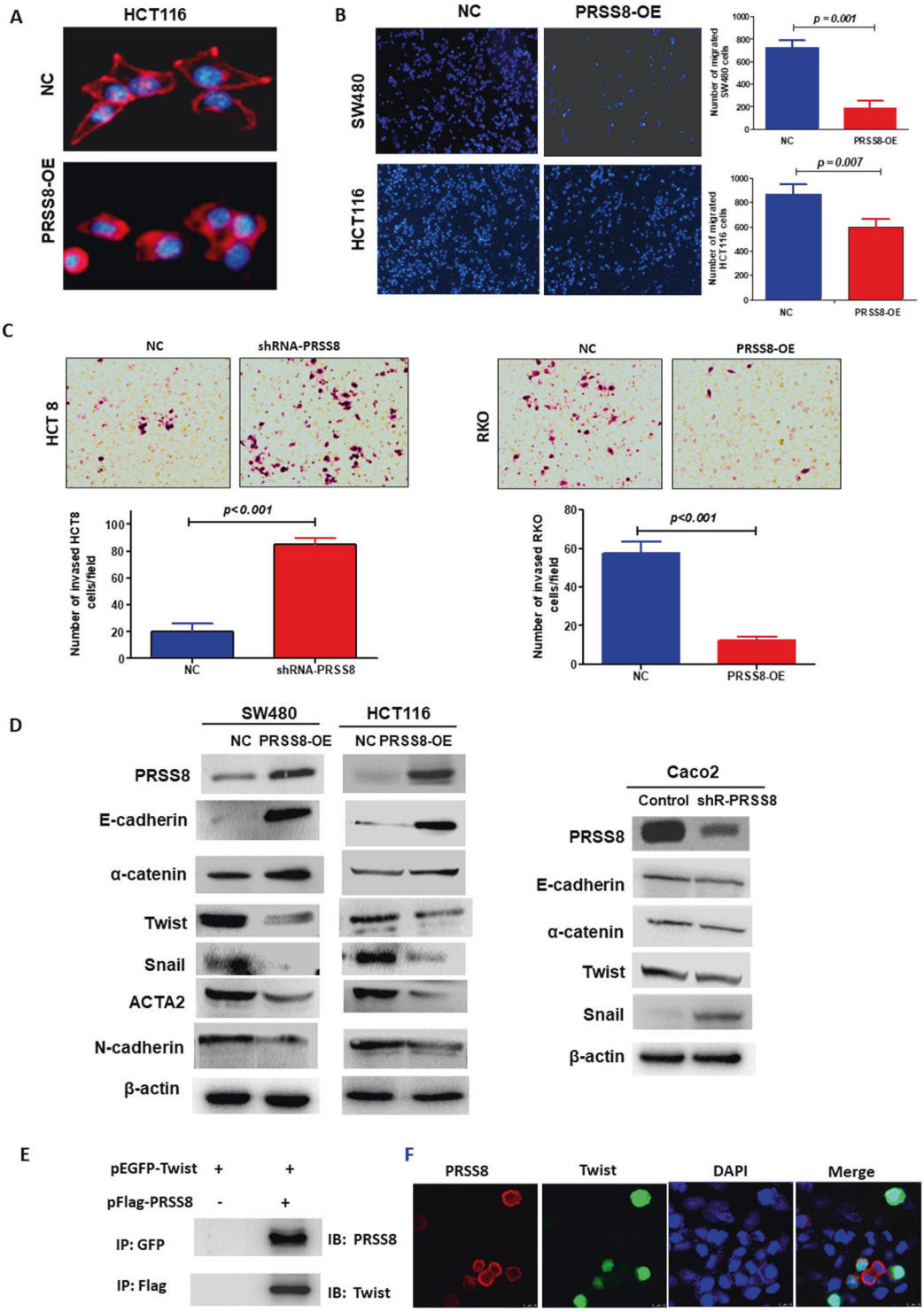

Fig. 5 PRSS8 altered cancer cell morphology and suppressed the epithelial-mesenchymal transition pathway. **a** PRSS8 overexpression induced cytoskeletal changes, converting HCT116 cells from an invasive, spindle-like morphology to a less invasive, rounded pheno-type. **b** Increased PRSS8 expression inhibited migration of SW480 and HCT116 cancer cells. **c** Knockdown of PRSS8 expression promoted invasion of HCT8 cancer cells, and increased PRSS8 expression suppressed invasion of RKO cancer cells. **d** PRSS8 expression level saffected EMT-related protein changes. **e** Co-immunoprecipitation assay showed binding between PRSS8 and TWIST proteins. **f** PRSS8 and Twist were overlapped in subcellular localization in HCT116 cells by immunofluorescence staining. (NC negative control; shRNA-PRSS8, knockdown of PRSS8 expression by a virus-based short hairpin RNA vector targeting the PRSS8 gene; PRSS8-OE,increased expression of PRSS8 induced by a virus-based expression plasmid. IB immunoblotting, IP co-immunoprecipitation).