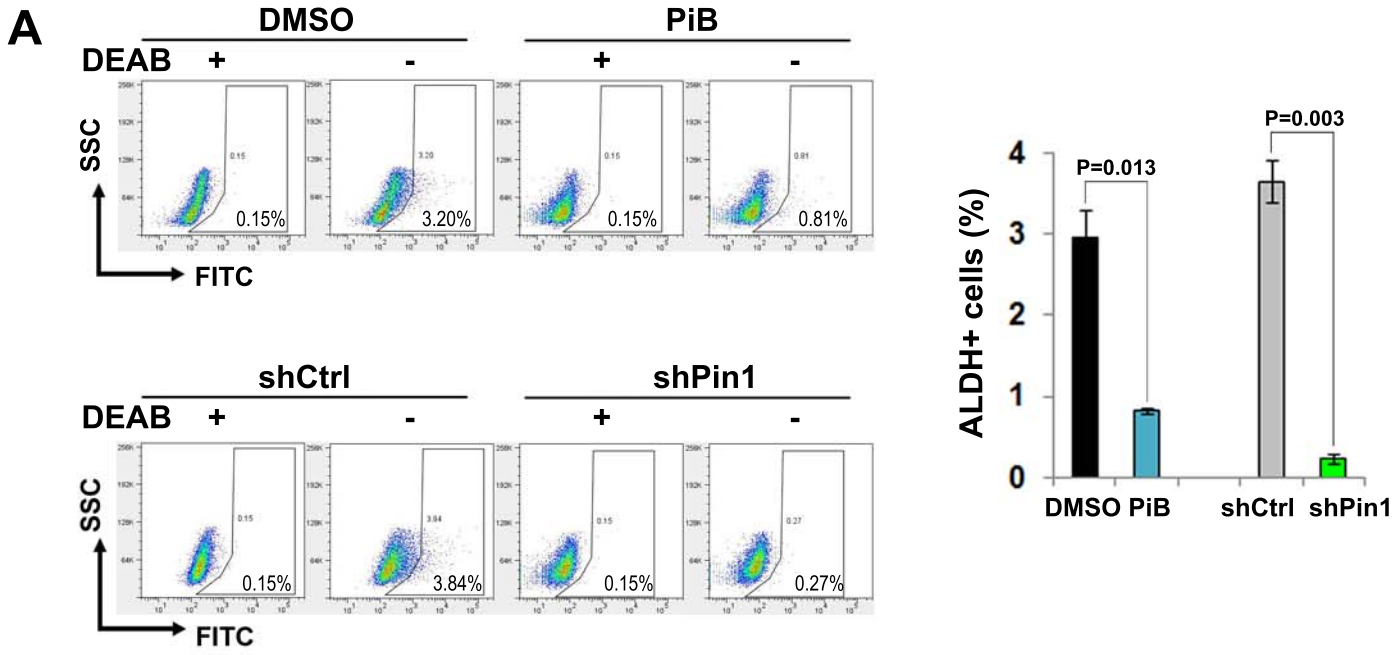
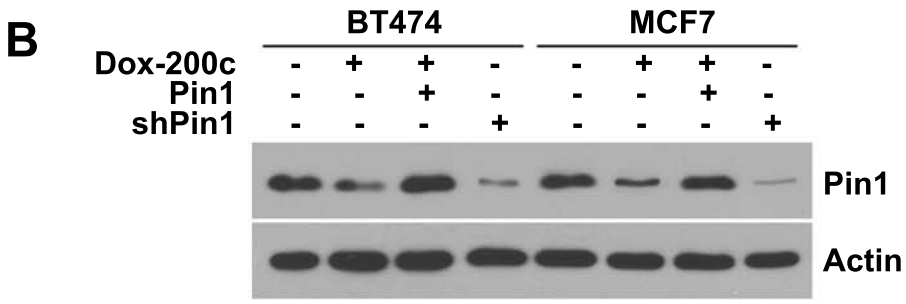
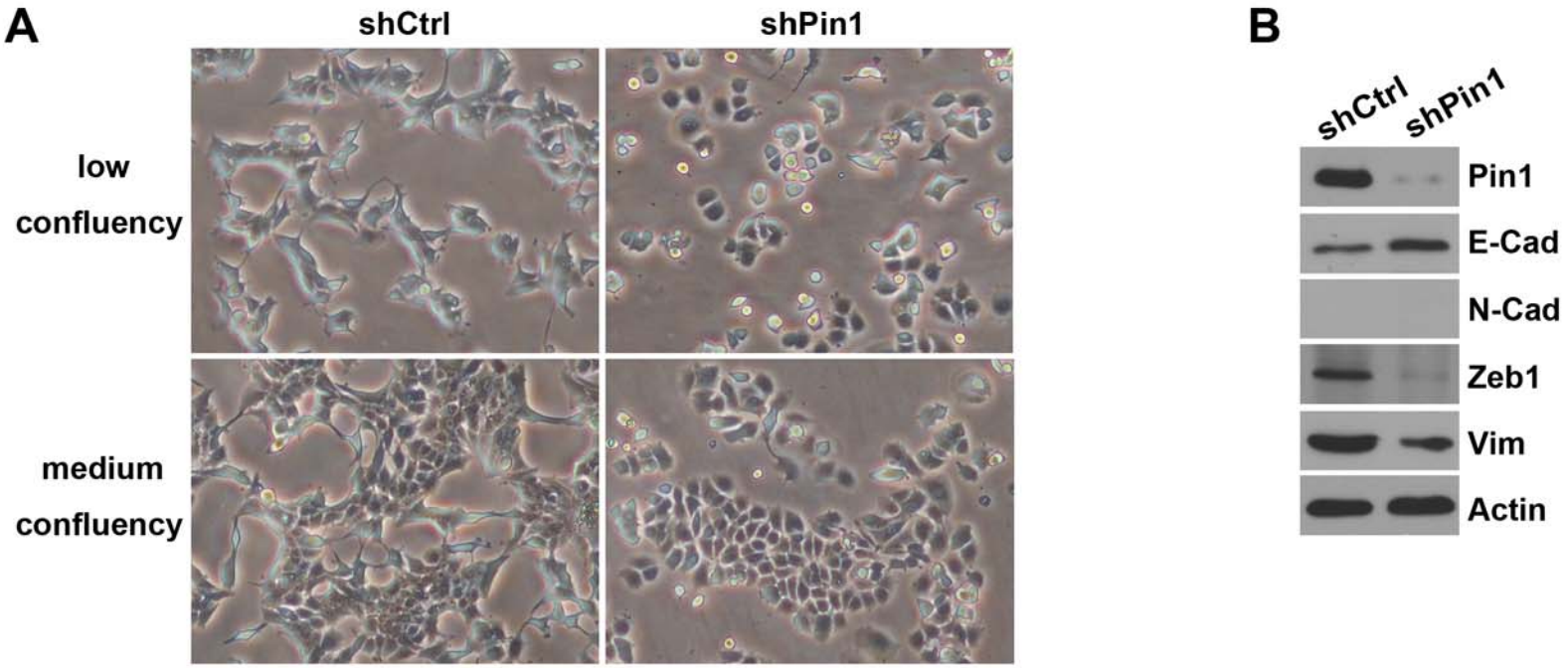
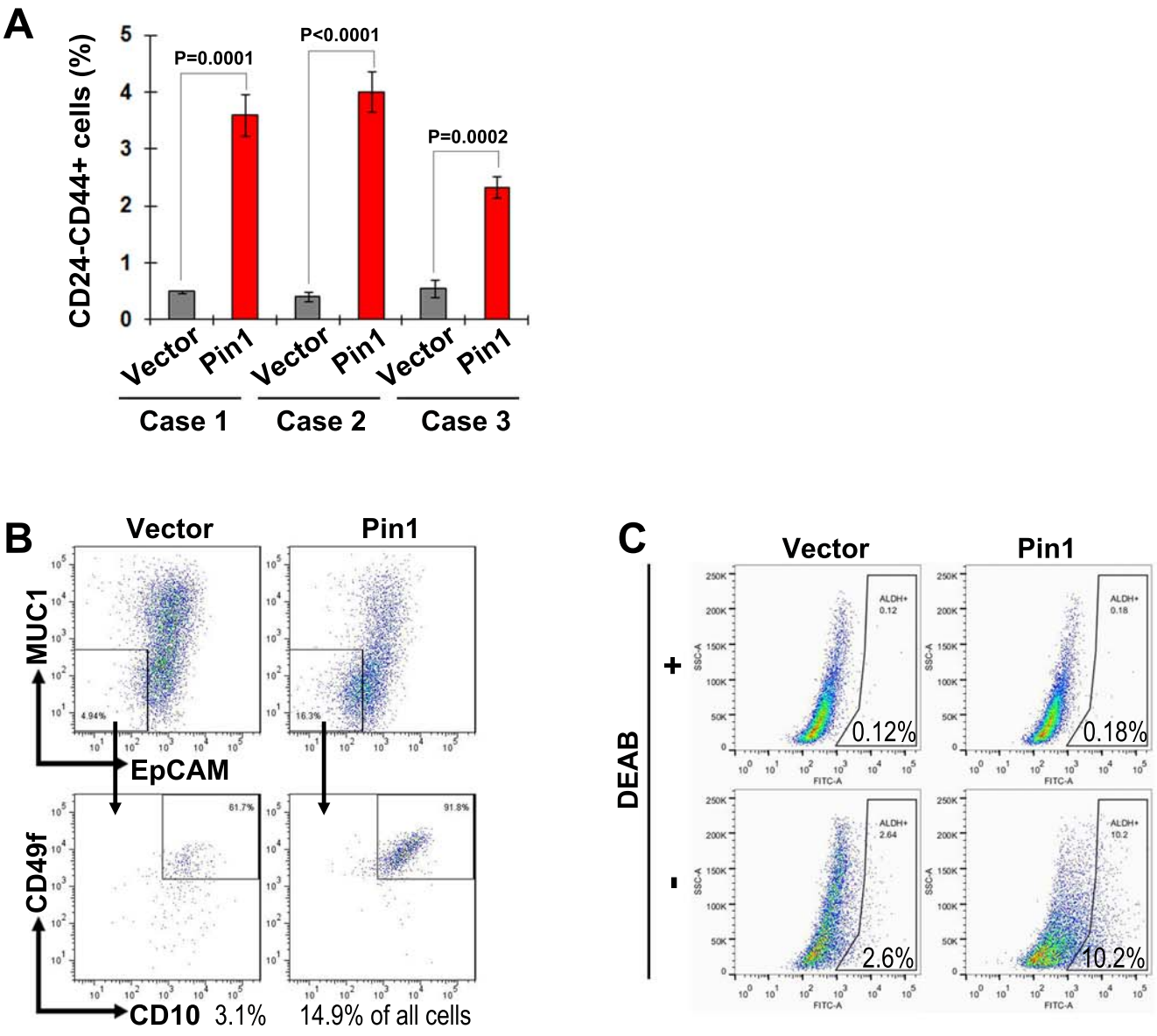




Luo et al., Supplementary Figure S1. Pin1 inhibition suppresses the expansion of BCSC-enriched populations. A, Pin1 inhibition by PiB treatment or shRNA decreased the ALDH+ population in HMLE cells. Cells treated with DEAB, a specific ALDH inhibitor were used as a negative control to establish the baseline fluorescence and define the ALDEFLUOR-positive population. The bar graph presents mean±SD of three independent experiments. B, BT474 and MCF7 cells were infected with lentiviruses expressing tetracycline-inducible miR-200c with or without miR-200c-resistant Pin1, and Pin1 shRNA, as detected by immunoblotting analysis.



Luo et al., Supplementary Figure S2. Pin1 knockdown in MCF7 cells reverts EMT phenotype. A, Silencing of Pin1 resulted in a morphological change in MCF7 cells. Pin1 knockdown cells exhibited more epithelial-like cobblestone morphology, whereas the control cells displayed an elongated spindle shape. B, Pin1 knockdown increased the expression of epithelial marker (E-Cad) and decreased the expression of mesenchymal markers (Zeb1 and Vimentin), as detected by immunoblotting analysis. N-Cad expression was not detected in these cells.



Luo et al., Supplementary Figure S3. Pin1 promotes the expansion of BCSC-enriched populations, as well as basal/myoepithelial and luminal progenitors in primary normal human MECs. A, Pin1 overexpression increased the CD24-CD44+ population in primary human MECs. B, Pin1 over-expression increased the basal/myoepithelial progenitor-enriched population in primary human MECs. Basal/myoepithelial progenitor-enriched MUC1-EpCAM-CD10+CD49f+ cell were first analyzed for MUC1-EpCAM- fraction, and then CD10 and CD49f expression. C, Pin1 over-expression increased the luminal progenitor-enriched population in primary human MECs. ALDH+ cells were analyzed without adding DEAB, the inhibitor of ALDH enzyme, which was used as a negative control.