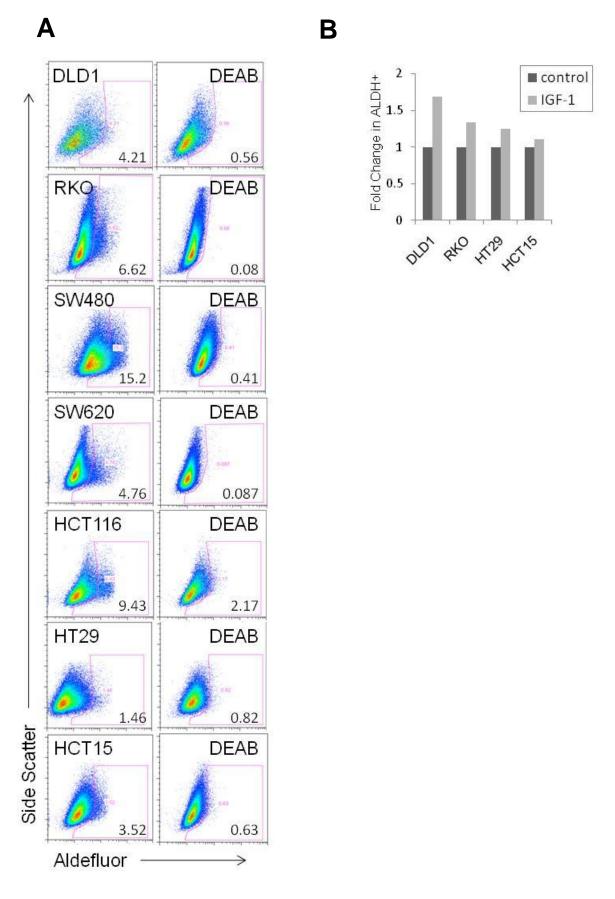
Supplementary Figure 1. Putative CSC analysis in the panel of human colon cancer cell lines. (A) Aldefluor activity across the panel of cell lines (DEAB is the ALDH inhibitor used as a gating control), and (B) ALDH activity following IGF-1 treatment,

Supplementary Figure 2. Putative CSC analysis in the panel of human colon cancer cell lines. (A) immunoblot analysis of P-glycoprotein and IGF-1R-β, (B) cell surface expression of CD133 as assessed by flow cytometry, (C) side population analysis and CD133 cell-surface expression in live DLD1 cells (upper panel) and immunoblot analysis of CD133 in sorted SP and non-SP cells (lower panel), and (D) mitoxantrone efflux assay in RKO, SW480, and DLD1 cells under control (FBS cultures), SFM (serum starved), or IGF (serum starved cells treated with IGF-1 at 100 ng/ml for 24 hours).

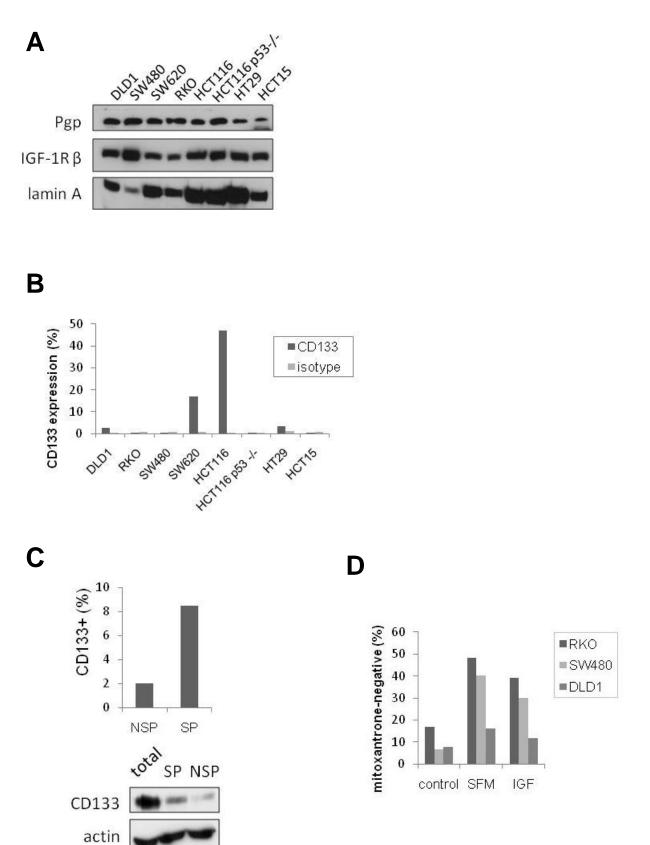
Supplementary Figure 3. IGF-1 enrichment of putative CSCs is dependent on β -catenin but independent of MAPK activity. (A) pBabe and CA-Akt cells transfected with β -catenin siRNA followed by serum starvation and IGF-1 treatment at 100ng/ml for 24 hours (all values normalized to serum starved levels to rule out the effect of CA-Akt and β -catenin), and (B) side population analysis of DLD1 and SW480 cells pretreated with PD-098,059 (10 μ M) prior to IGF-1 at 100ng/ml for 24 hours (all values were normalized to control in the absence of IGF-1 to compare IGF-1 enrichment. Note:PD-098,059 alone caused a 10-20% decrease in the SP fraction).

Supplementary Figure 4. In vivo delivery of CP-751,871 to subcutaneous tumors and decreased ALDH+ cells. (A) immunohistochemistry of human IgG levels in tumors (top row SW480, bottom row HCT-116) from mice treated with PBS or CP-751,871

(counterstained with hematoxylin), and **(B)** immunohistochemical staining of ALDH1 in HT29 tumors (counterstained with hematoxylin).

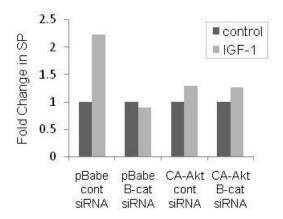


Supplementary Figure 1

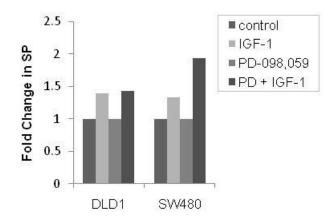


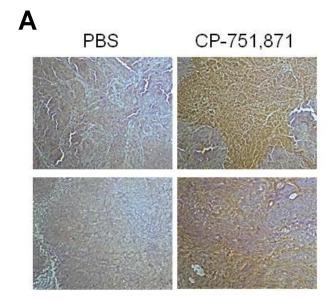
Supplementary Figure 2

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