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Supplemental data contents

Table S1, related to Figure 1B.

Differential stem cell signaling between CP70 and CP70sps were compared using an RT-PCR pathway-focused arrays (Stem signaling/Wnt signaling). Red indicates gene upregulation while green indicates gene downregulation, in CP70sps cells. Numbers indicate fold changes. Table S1A is a general focus array containing genes in Wnt, Notch, Hedgehog signaling pathways. Table S1B is a focus array on Wnt signaling.

Figure S1, related to Figure 1A and B.

Confirmation of dye-exclusion by the ABCG2 transporter and sphere formation assay. A, pretreatment of cells with the ABCG2 inhibitor GF120918 essentially eliminated the SP fraction of CP70 ovarian cancer cells (right panel). B, ovarian cancer CP70 SP spheroid-forming cells ("CP70sps cells"). CP70 SP cells were cultured in ultra low coating culture dishes, with anchorage-independent cells forming a spheroid morphology (CP70sps, 400×).

Figure S2, related to Figure 4B.

Inhibition of OTIC induced tumor formation by niclosamide *in vivo.* Niclosamide treatment (10mg/kg/daily) was given intraperitoneally every week one day after OTICs inoculation in NOD/SCID mice. Furthermore, Body weights remained constant during niclosamide treatment demonstrating a lack of systemic toxicity.

Figure S3, related to Figure 4C.

Tumor growth inhibition of CP70, non-stem parental cells, by niclosamide. One day after inoculation of 1×10^6 CP70 cells in NOD/SCID mice, niclosamide (10mg/kg/day) was administered intraperitoneally for three weeks, then twice a week for 2 weeks. After 9 weeks, mice were sacrificed, and tumor size/incidence assessed. (A) Red lines outline the peritoneal tumors. (B) Comparison of tumor formation after dissecting all tumors.