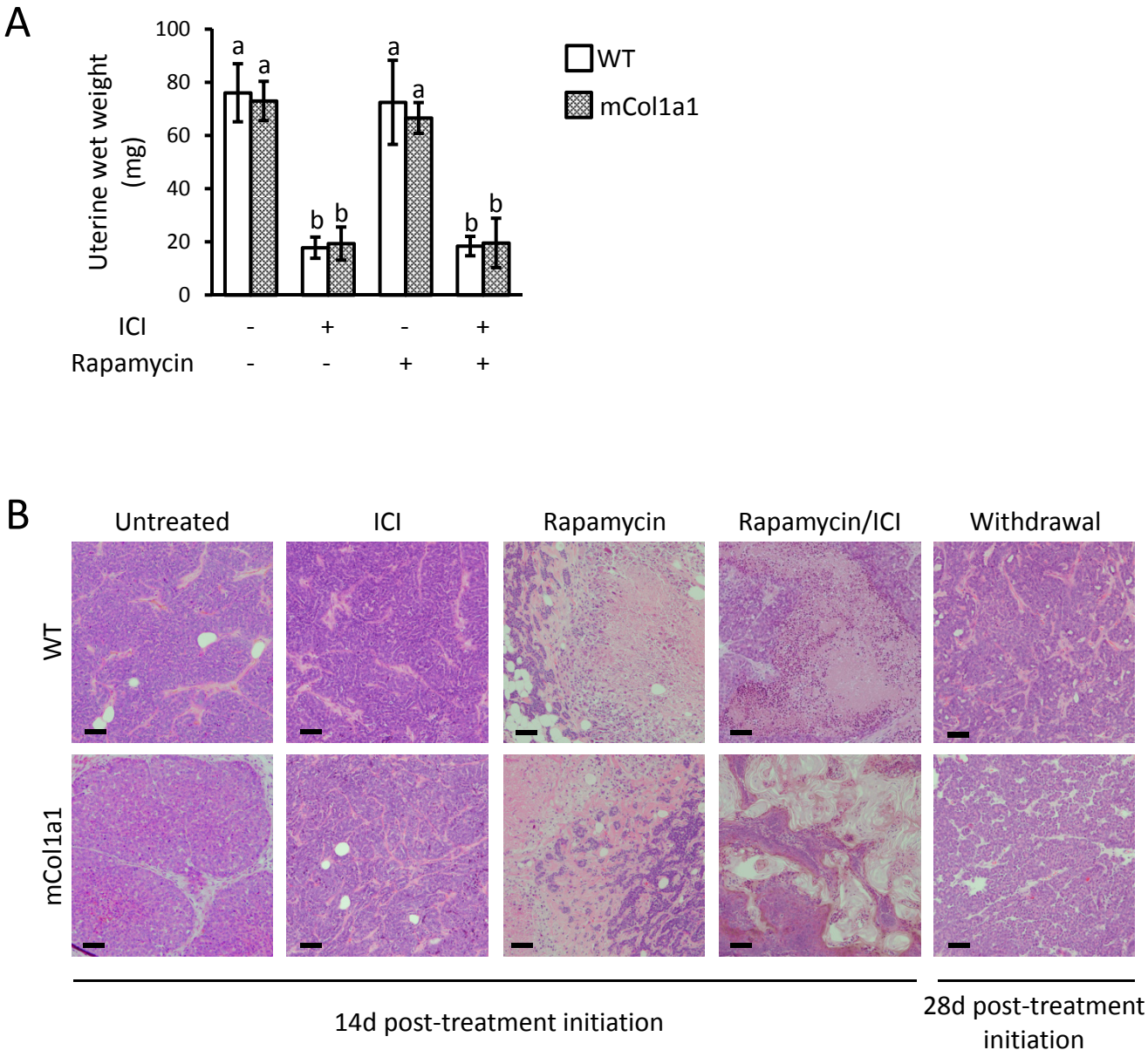
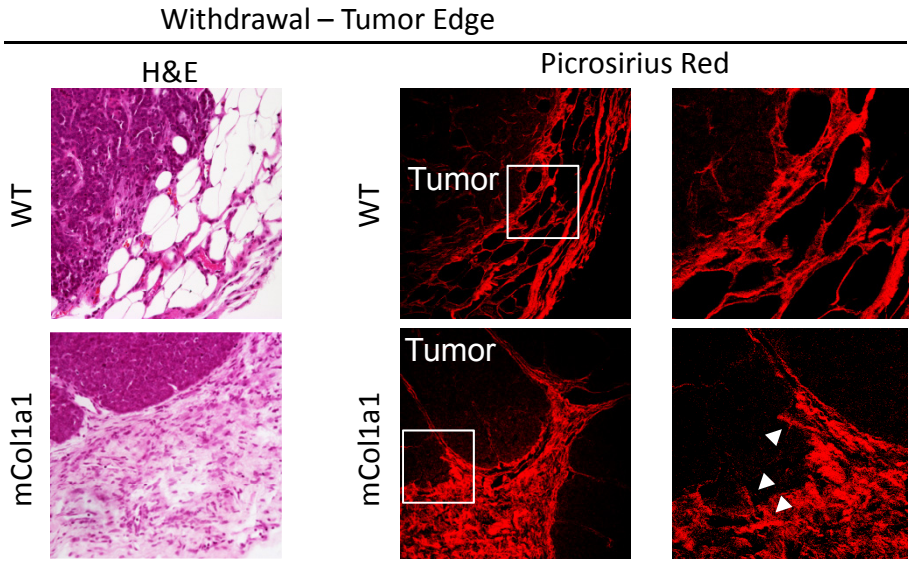
Supplementary Fig. S1A,B



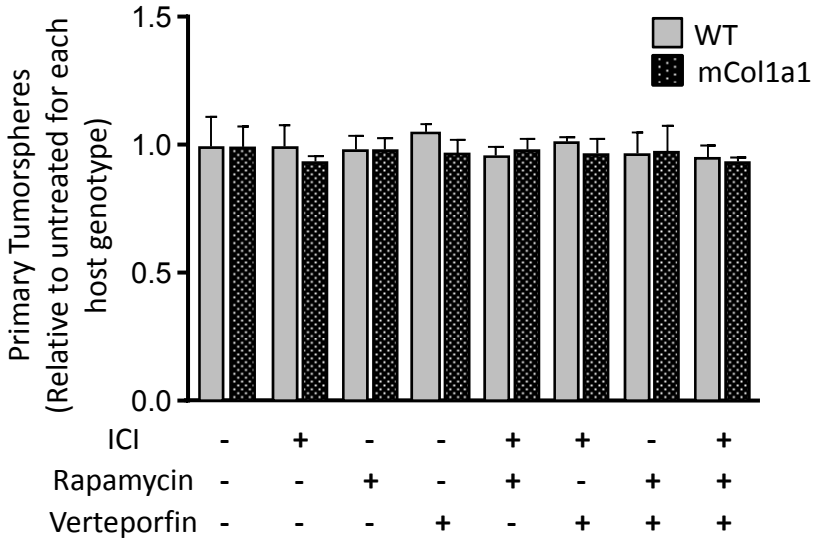
Supplementary Fig. S1C

C



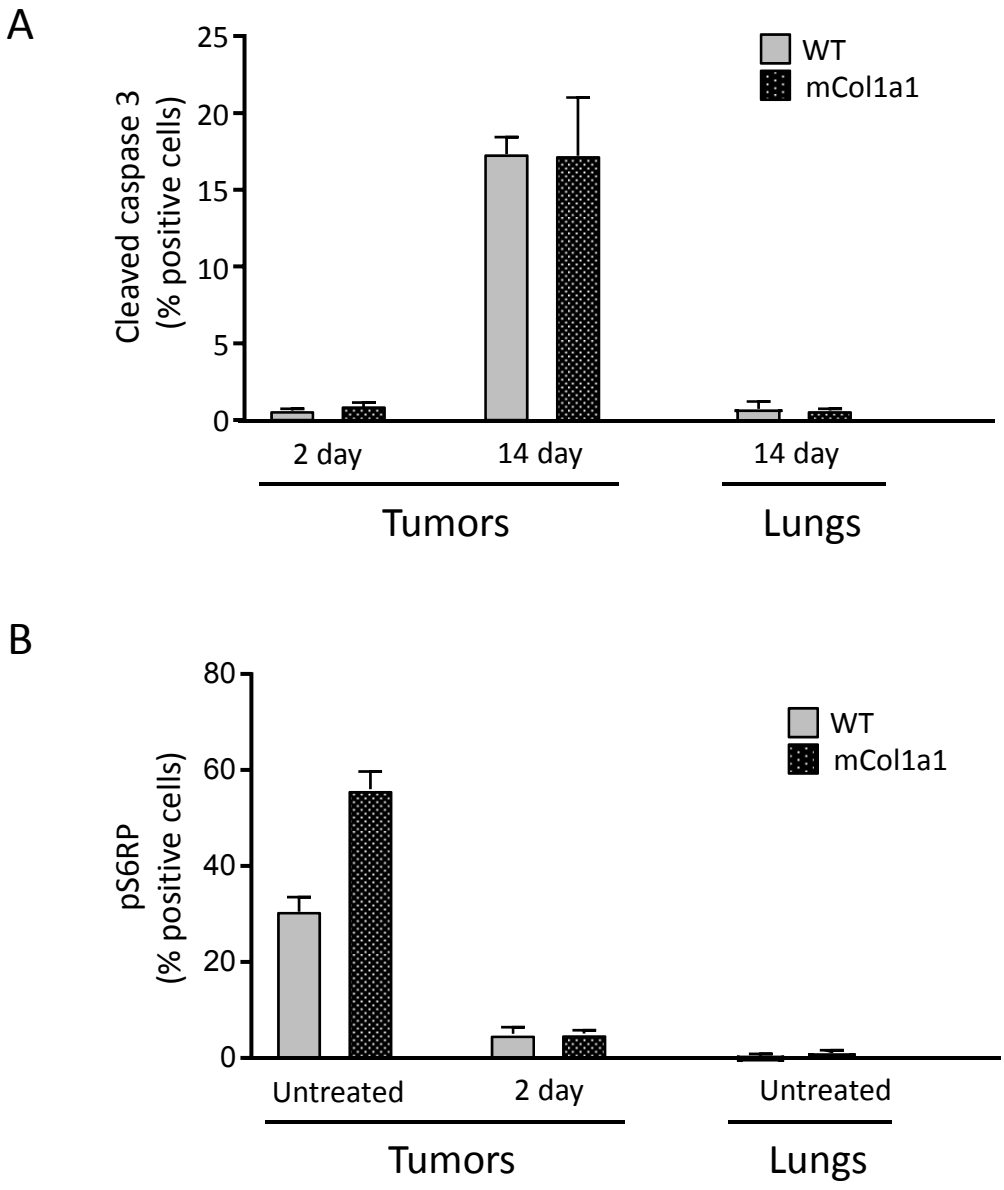
Supplementary Fig. S1: (A) Uterine wet weights confirm bioactivity of the anti-estrogen, ICI 182,780 (ICI) (N=4; mean ± S.D.). (B) Representative H&E stained sections of tumors from control, ICI-, Rapamycin-, Rapamycin/ICI-treated hosts after 14 days of treatment, and from Rapamycin/ICI-treated hosts from whom treatment had been withdrawn for 14 more days. Scale bars, 100 µm. (C) Representative H&E- and picrosirius red-stained tumor boundaries from Rapamycin/ICI-treated recipients from whom treatment had been withdrawn for 14 days. Arrowheads mark fibers perpendicular to tumor boundary.

Supplementary Fig. S2



Supplementary Fig. S2: Effects of *in vitro* treatments on ability of tumor cells harvested from untreated WT and mCol1a1 hosts to form primary tumorspheres (N=4 independent experiments; mean ± S.D.). Data are normalized to the number of tumorspheres that develop in untreated cells from tumors from each host genotype, respectively.

Supplementary Fig. S3



Supplementary Fig. S3: (A) Frequency of cleaved-caspase-3-labeled cells in primary tumors and lung metastases from WT and mCol1a1 hosts treated with ICI/ Rapamycin for 2 or 14 days as shown. (N= 4 mice; Mean ± S.D.). (B) Frequency of pS6RP-labeled cells in primary tumors and lung metastases in WT and mCol1a1 hosts, untreated or ICI/ Rapamycin-treated for 2 days as shown. (N= 4 mice; Mean ± S.D.).