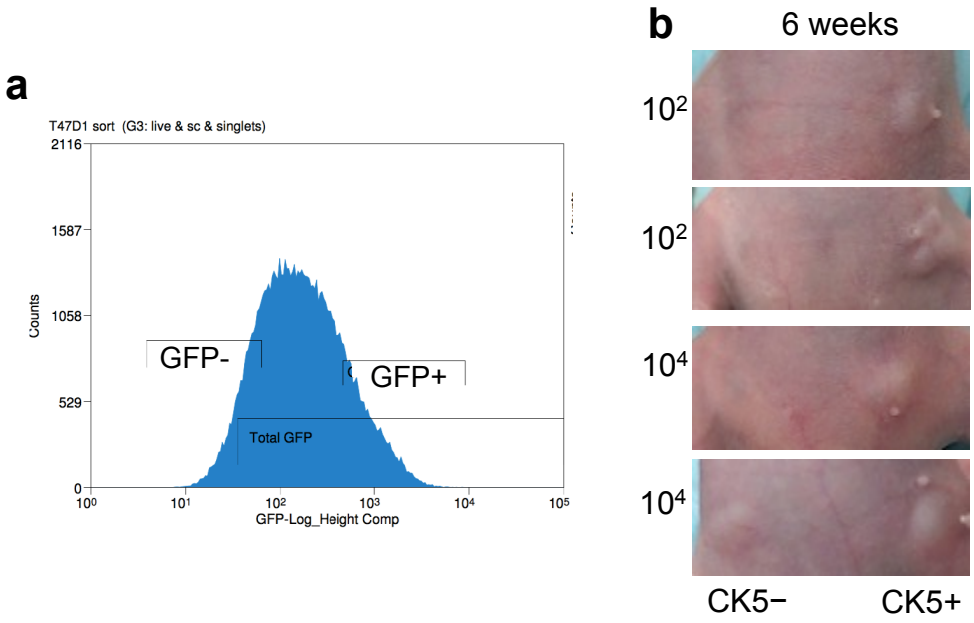
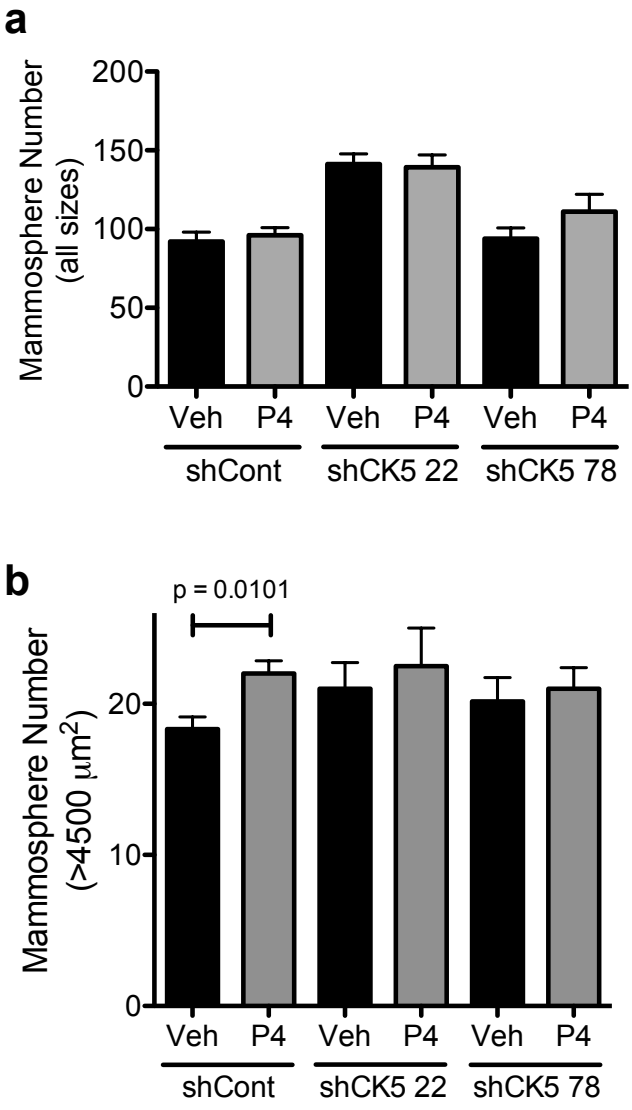
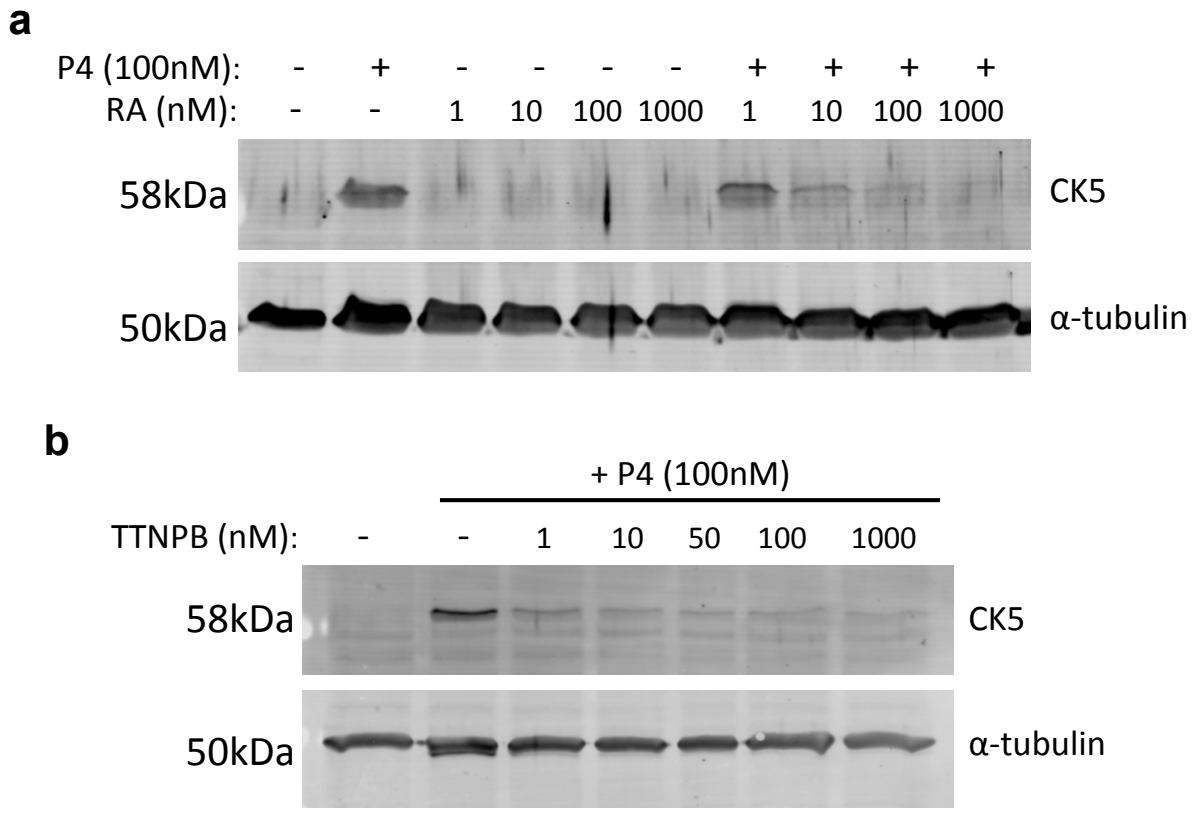


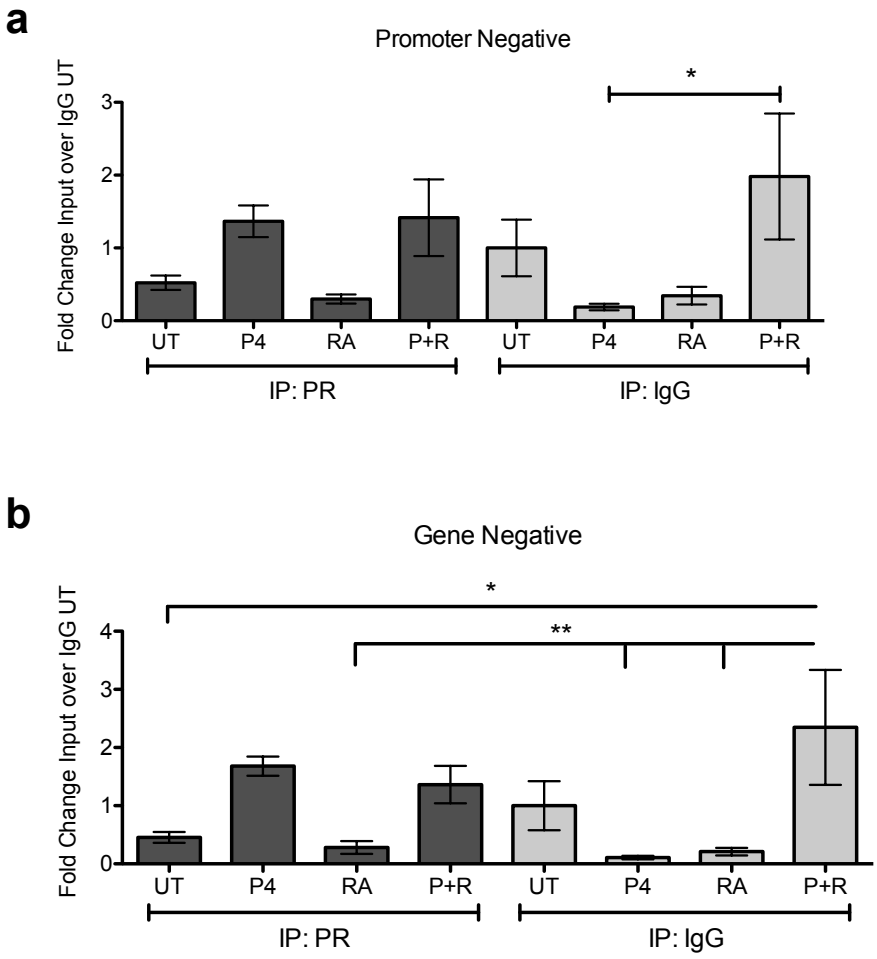
Supplementary Figure 1. CK5+ cells are more tumor initiating. (**a** and **b**) T47D breast cancer cells stably integrated with a CK5 promoter-driven GFP reporter were treated with 100 nM P4 for 24 h to induce CK5 expression. CK5+ and CK5- cell populations were sorted based on GFP expression via FACS (**a**) and injected into fourth mammary fat pads of nude mice (**b**). Tumor growth was assessed by palpation after 6 weeks.



Supplementary Figure 2. Mammosphere number. While no significant change in mammosphere number is detected when no size cutoff is selected (**a**), a significant increase in the sphere size can be seen in control cells with P4 treatment at very large sphere sizes (>4500 μm^2) (**b**). This difference is attenuated, however, with loss of CK5 expression. Data represent mean ± s.e.m. Significance was assessed using Student's T-test.



Supplementary Figure 3. RAR agonist dose curves. (**a** and **b**) To determine lowest effective concentrations of 9-cis RA (**a**) and RAR-specific agonist TTNPB (**b**), T47D cells were treated with the indicated increasing concentrations of RA or TTNPB with or without P4 for 24 h. Cell lysates were harvested using RIPA buffer and analyzed via immunoblot. α-tubulin was used as a loading control.



Supplementary Figure 4. CK5 negative control primers. (**a** and **b**) To assess the specificity of binding in the CK5 promoter, negative control primer sets were designed to target 200 bp regions more than 500 bp upstream of the putative PRE in the CK5 promoter (**a**) or downstream of the TSS (**b**). All samples had signals equal to or lower than IgG negative control IPs. Data represent ± s.e.m. *P<0.05 **P<0.01

Promoter construct	Restriction enzyme(s)
4.6 kb	ClaI, SnaBI
3.9 kb	ClaI, PshAI
3.4 kb	ClaI, PmlI
2.0 kb	ClaI, SmaI
1.2 kb	ClaI, NheI
1.0 kb	ClaI, SbfI
0.8 kb	ClaI, SmaI
0.2 kb	ClaI, NheI
Δ0.2-1.2 kb	NheI
Δ1.0-4.3 kb	SbfI
Δ3.4-4.6	SnabI, PmlI

Supplementary Table 1. Table of restriction enzymes used to create CK5 promoter deletion constructs in the previously described pCDH1-luciferase vector[16].