**Supplemental Data Figure Legends**

**Supplemental Figure 1:** Doxorubicin, paclitaxel, and staurosporine induce a transient increase in bioluminescence in luciferase expressing prostate carcinoma cells. 22Rv1-CMVluc, PC3-CMVluc, and 22Rv1–SV40luc cells were treated with either 4 μmol/L of staurosporine (STS) for 4 h; 90 nmol/L paclitaxel (PAC) for 6 days; or doxorubicin (DOX) (400 nmol/L in 22RV1-CMVluc and 22RV1-SV40luc and 700nmol/L in PC3-CMVluc cells) for 5 days. Bioluminescent images, caspase-3 activation assays and cell viability assays were performed daily throughout the time course for each treatment. The Y axis represents the percent of control values for each parameter being measured. Staurosporine induces a rapid and significant increase in bioluminescence in all three cell lines after 1 h of treatment, after which signal begins to decline as cells die (A, D, G). Paclitaxel induces a significant increase in bioluminescence in both CMVluc lines from 24 - 48 h; after 75 h, bioluminescence signal begins to decline as cells lose viability and die (B, E, H). Doxorubicin induces a significant increase in bioluminescence only in 22Rv1-CMVluc cells (C) at 24 h; after 75 h, signal begins to decline as cells die (C,F,I). Significant increases in photon flux relative to controls are denoted by asterisks; **p<0.01 ***p<0.001

**Supplemental Figure 2:** Trichostatin-A (TSA) induces bioluminescence and AR gene expression in cells engineered to express luciferase and AR from the CMV promoter. A, 22Rv1-CMVluc and PC3-CMVluc cells were treated with 100 ng/mL TSA for 48 h and bioluminescence values (photons/sec/cm2/sr) were compared to untreated control cells. TSA induces a significant increase in bioluminescence. Significant increases in photon flux relative to controls are denoted by asterisks; *p<0.05, **p<0.01. B, LNCaP (# 1),
PC3-CMV-AR (#2) and two different PC3-SFFV-AR lines (# 3 and 4) were treated for 24 h with TSA (400 ng/mL) and expression of AR was examined via western blot. TSA induces AR expression only in cell lines engineered to express AR from the CMV promoter (lane 2).

**Supplemental Figure 3**: Doxorubicin treatment increases luciferase mRNA and protein in 22Rv1-CMVluc cells but not 22Rv1-SV40luc cells. 22Rv1-CMVluc and 22Rv1-Sv40luc cells were either treated with 400 nmol/L doxorubicin or left untreated for 48 h. A, RNA was prepared from treated and untreated cells and qRT-PCR was performed for luciferase mRNA. Doxorubicin treatment significantly increased luciferase mRNA levels in 22Rv1-CMVluc cells but not 22Rv1-SV40 luc cells. B, Cell lysates from above were also analyzed for luciferase protein using an *in vitro* luciferase activity assay (see material and methods). Doxorubicin treatment significantly increased luciferase protein in 22Rv1-CMVluc cells but not 22Rv1-SV40luc cells. Significant increases in photon flux relative to controls are denoted by asterisks; ***p<0.001

**Supplemental Figure 4**: Doxorubicin treatment of 22RV1-SV40luc subcutaneous xenograft tumors does not affect luciferase expression *in vivo*. Subcutaneous 22Rv1-Sv40luc xenografts were initiated in *nu/nu* mice (see Materials and Methods). Groups of 6 randomly assigned animals were injected once via the tail vein with either 200μL PBS or 200μl 1mg/mL doxorubicin (8mg/kg). Tumor volumes were recorded and BLI was performed daily for a period of 10 days. The Y axis represents bioluminescence normalized to tumor volume (photons/sec/cm²/sr/mm³). In contrast to the 22Rv1-CMVluc xenografts, the bioluminescent signal of doxorubicin treated 22Rv1–SV40luc cells remained similar to the PBS controls over a period of 6 days following treatment.
Supplemental Figure 5: Full length blot represented in cropped form in Figure 5C.