Supplementary Data Figure Legends

Supplemental Figure 1. BAF57 expression is in prostate cancer. (A) Specificity of the BAF57 antibody was determined by IHC analysis utilizing tissue sections from paraffin embedded BT549 and LNCaP cell pellets. Antibody was used at a dilution of 1:2000. **(B)** No primary antibody control for the data shown in Figure 1B. **(C)** Gene expression profiling of BAF57 from two independent, publicly available prostate cancer gene expression datasets containing the indicated number of normal, cancer, and metastatic samples. Datasets were analyzed using the Oncomine microarray database and were selected based on having adequate "n" numbers for normal, cancer, and metastatic samples for comparison. P-values are provided based on comparison of non-neoplastic to cancer (including both primary and metastatic lesions).

Supplemental Figure 2. SWI/SNF ATPases and BAF57 are not required for bicalutamide mediated repression of AR activity. (A) LNCaP cells were transfected with limiting amounts of H2B-GFP and pHTP-scrambled control, pHTP-BRMi, or pHTP-BRG1i. Following transfection and rapid selection, cells were harvested and subjected to immunoblot analyses for BRM, BRG1, or CDK4 (control). Band quantification is indicated, as determined using the LiCoR Odyssey Infrared system (left panels). Cells transfected in parallel were treated with 0.1% EtOH or 1μM bicalutamide as indicated for 48h. RNA was then isolated and cDNA generated. Representative PCR amplifications (reflective of at least 3 independent experiments) to detect for PSA and GAPDH levels are shown. Band quantification was performed by densitometry and relative expression level provided (right panels). These data indicate that depletion of the Brm of Brg1 ATPase does not diminish the transcriptional response to bicalutamide. **(B)** LNCaP cells ($3x10^6$) were cultured in steroid-free medium for 72hrs and then treated with 1μM bicalutamide for 2 hrs. After treatment, the cells were cross-linked with formaldehyde

and the chromatin recovered subsequently was subjected to immunoprecipitation with antibodies directed against AR, BAF57 or preimmune serum. DNA fragments purified were used to analyze the PSA promoter (PSA A/B, left panel) or the TMPRSS2 enhancer (right panel) regions by real-time PCR, as indicated, using strategies described in the Materials and Methods section. The PSA promoter region was utilized (A/B, as previously described by Shang et al., 2002), as this region is the primary site of AR recruitment in the presence of bicalutamide. Results are representative data of the ChIP analyses from at least two independent experiments. **(C)** LNCaP prostate cancer cells were transfected with H2B-GFP and pcDNA3-vector or pBabe-BAF57ΔN-flag expression plasmid. Cells were treated with either 0.1% EtOH or 1μM bicalutamide as indicated for 48h (with re-stimulation after 24h). Cells were harvested and RNA was isolated, and then subjected to RT-PCR. cDNA was amplified for PSA or GAPDH and relative quantification is shown (lower panel). Representative data from at least two independent experiments is shown. These data indicate that abrogation of BAF57 activity does not compromise the transcriptional response to bicalutamide (i.e. suppression of PSA expression). **(D)** LNCaP cells were transfected with H2B-GFP and pcDNA3-vector, pBabe-BAF57ΔN-flag (Dominant negative BAF57 expression constuct), or pCMV-p16ink4a (positive control for cell cycle arrest). Following transfection, cells were treated with 0.1% EtOH or 1μM bicalutamide as indicated for 48h (with re-stimulation after 24h). Bromodeoxyuridine (BrdU) was added for a 16h pulse, cells were fixed, and staining for BrdU incorporation was performed. Results from at least two independent experiments are plotted as percent BrdU incorporation relative to vector plus vehicle. * = $p < 0.05$. These data indicate that abrogation of BAF57 activity does not impair growth suppression in the presence of AR antagonists.

Supplemental Figure 3. Schematic and expression data for binding assays. (A) Schematic representation of androgen receptor functional domains and mutant AR constructs used (left panel). Representative Coomassie stained gel of purified GST or GST-BAF57 from Figure 3A is shown in the right panel. **(B)** Schematic representation of GST-AR fusion proteins utilized in GST pull-down analyses for Figure 3B (left panel). Coomassie stained gel of purified GST or GST-AR deletions is shown in the right panel.

Supplemental Figure 4. Expression and functional data to assess the CTE requirement for BAF57 responsiveness (A) Representative Coomassie stained gel of purified GST or GST-BAF57 used for the binding assays in Figure 4A is shown. **(B)** BT549 cells were transfected with 0.5μg H2B-GFP and 1.5μg of pcDNA3 vector, pSG5AR, or pSG5AR∆629-636, in parallel with studies shown in Figure 4B. Immunoblot analysis was performed for AR to detect relative expression. CDK4 is included as a loading control. As shown, both wildtype and mutant AR are expressed at equal levels **(C).** BT549 cells were transfected using strategies outlined for Figure 4B, but with increasing amounts of pBabe-wtBAF57-flag (2.25-4.25μg), as described in the main text. Reporter analysis was carried out using the Dual Luciferase Assay Reporter System (Promega). In these studies, increasing dosage of BAF57 failed to further support ligand-dependent activation of the AR∆629-636 mutant, indicating loss of the AR residues crucial for BAF57 interaction.

Supplemental Figure 5. Schematic and expression data for identification of the AR binding site. (A) Schematic representation of BAF57 domains and deletion constructs employed. **(B)** Representative Coomassie stained gel of purified GST or GST-AR deletions is shown. Stars indicate appropriate bands. **(C)** Immobilized GST or GST-AR

fusions were incubated with [^{35}S]-Met-labeled BAF57, BAF57ΔN, BAF57ΔPR, or BAF57ΔHMG overnight at 4°C, washed, and subjected to SDS-PAGE. 5% input and bound protein was detected by autoradiography. Binding as a percentage to wtBAF57 is shown (right panel). In the absence of the HMG domain alone, little effect on AR binding was observed (bottom panel and quantification at right, 98% of full-length BAF57). Removal of the extreme N-terminal PR domain demonstrated a 56% reduction in AR binding compared to full-length BAF57 binding (set at 100%; right panel). Deletion of both regions ablates AR association. In brief, the N-terminal portion of BAF57 encompassing the proline rich region and the HMG domains can be concluded to be vital for interaction with AR.