Supplemental Figure Legends:

Supplemental Figure S1. ***Brm is expressed in the luminal prostatic epithelia.*** **A.** Validation of the Brm antisera was performed using SW13 (Brm null, negative control) xenografts or human tonsil specimens (positive control). No primary IgG control is provided in left panels. To generate SW13 xenografts, $5X10^6$ cells were inoculated into athymic mice and allowed to grow until reaching approximately 0.5 cm^3 at which time xenografts were harvested, fixed in 10% buffered formalin and imbedded in paraffin blocks. Unidentified tonsil specimens were obtained from the University of North Carolina Anatomic Pathology Translational Core Laboratory in accordance with Institutional Review Board approval. **B.** Dorsolateral (DLP) ventral (VP), and anterior (AP) lobes of the murine prostate were immunostained as in panel A.

Supplemental Figure S2. ***Brm-/- prostates show lobe specific hyperplasia without nuclear atypia.*** **A.** H&E stained sections of the DLP, VP, and DLP lobes from 6 month old littermates (Brm +/+ or Brm -/-) are shown for both low magnification. **B.** 60x magnification of the AP revealed no significant nuclear atypia.

Supplemental Figure S3. Strategy for analyses of androgen dependence and responsiveness. A. Schematic for assessment of androgen dependence and androgen responsiveness of the prostatic epithelia of 12 week old animals. B. Serum testosterone levels were monitored before and after castration in Brm positive (Brm+/-) vs. Brm negative (Brm-/-) animals. As shown, testosterone levels were indistinguishable between the Brm+/- and Brm-/- animals in the unchallenged, age matched mice, both of which were in the range of what has been reported for wild-type animals. As also shown, testosterone levels from the serum of castrated animals of both genotypes are at undetectable levels. As such, it is apparent that the castration resistant proliferation

observed in Brm-/- prostatic epithelia cannot be attributed to altered serum testosterone levels.

Supplemental Figure S4. Tumor-specific attenuation of Brm mRNA expression is observed in human prostate cancer. Cancer-specific reductions in SMARCA2 expression were confirmed through analyses of a second dataset (34). Heat maps are provided to demonstrate relative reductions in expression (top panel). Increasing blue intensity is reflective of downregulated gene expression, increasing red intensity of upregulated gene expression. Data using all 4 probe sets against the 5' end of SMARCA2 are plotted in the lower panel.

Supplemental Figure S5. Brm loss is associated with cell cycle perturbations and E2F1 deregulation. A. Heat map analyses of genes that correlated (cluster C2, 498 features) or anti-correlated (cluster C1, 422 features) with Brm mRNA levels using the data described in Figure 5A. Lower bar indicates specimens. B. Gene lists from overrepresented pathways described in Figure 5C. **C.** Representation of the major gene pathways identified in panel A that are directly associated with proliferative control. Genes that correlate with Brm downregulation are denoted by a green star, and those that anti-correlate are denoted by a red star.

Supplemental Figure S6. Complete list of genes that are significantly altered as a function of Brm status. Genes that positively or negatively correlate with Brm reduction in human tumors, corresponding to the heat map shown in Supplemental Figure 5A.

Supplemental Figure S7. E2F1 analyses in multiple tissues. Immunblot analyses of E2F1 expression in 3T3 immortalized fibroblasts derived from Brm +/+ and Brm -/-

animals (panel A), or in tissues obtained from 6 month old littermates (panel B). B-actin was included as a loading control.