Supplementary Table 1. List of siRNA, shRNA and primer sequences used in study.

siRNA	**Sequence**
***MYC* #1**	5'−GGAACUAUGACCUCGACUA−3'
***MYC* #2**	5'−CAGAGAAGCUGGCCUCCUA−3'
***SLC1A4* #1**	5'−GAGAUAGAAGGGAUGAACA−3'
***SLC1A4* #2**	5'−GACAUCAUCGUGCUGGUGA−3'
***SLC1A5* #1**	5'−GUCAGCAGCCUUUCGCUCA−3'
***SLC1A5* #2**	5'−CCAAGCACAUCAGCCGUUU−3'
shRNA	**Sequence**
MYC	5'−CTCGAGAAGGTATATTGCTGTTGACAGTGAGCGCACGACGA GAACAGTTGAAACATAGTGAAGCCACAGATGTATGTTTCAACT GTTCTCCGTCGTTTGCCTACTGCCTCGGAATTC−3'
qPCR Primers	
SLC1A4	Forward: TGCCTATGGGGATCTTTTCA
	Reverse: TGGACTCTTTCCTCGACCTG
SLC1A5	Forward: GGGCAAAGAGTAAACCCACA
	Reverse: CACCATGGTTCTGGTCTCCT
GLS	Forward: GGGAATTCACTTTTGTCACGA
	Reverse: TGACTTTACCCTTTGATCACCAC
GLUL	Forward: ACGCCACTCCAAAAAGAGAA
	Reverse: AGTGGGAACTTGCTGAGGTG
MYC	Forward: CACCGAGTCGTAGTCGAGGT
	Reverse: TTTCGGGTAGTGGAAAACCA
***RPLP0* (36B4)**	Forward: GGACATGTTGCTGGCCAATAA
	Reverse: GGGCCCGAGACCAGTGTT
FKBP5	Forward: TGGGGCTTTCTTCATTGTTC
	Reverse: GCGGAGAGTGACGGAGTC

Supplementary Figure S1

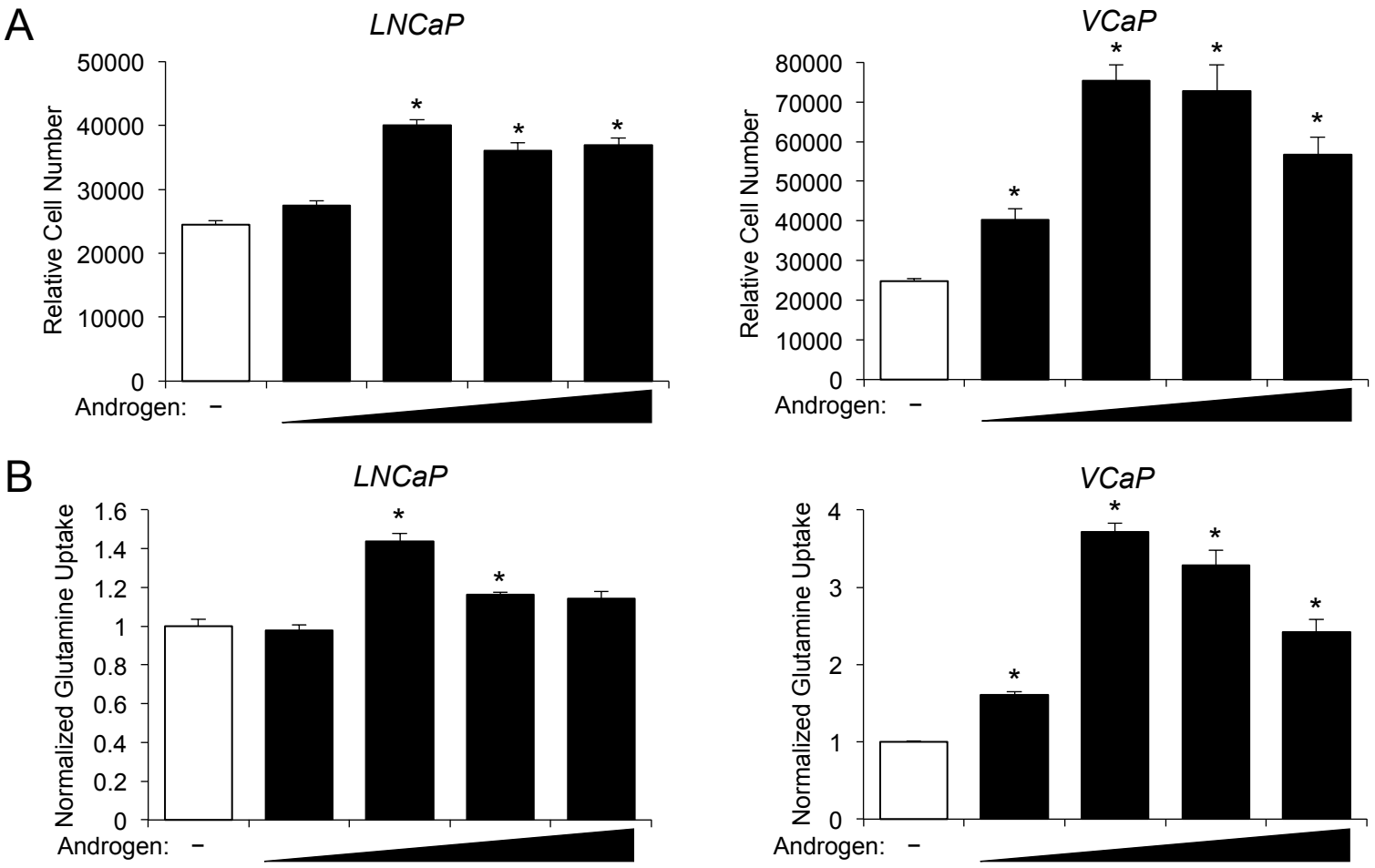

Figure S1. Androgen-mediated cell growth and glutamine uptake is biphasic in LNCaP and VCaP cells. Indicated cells were treated with increasing concentrations of androgen (R1881: 0, 10 pM, 100 pm, 1 nM and 10 nM) for 72 hours. A, cell were lysed and relative cell numbers were assessed using a fluorescent DNA dye. B, Spent medium was collected and analyzed for glutamine levels using a bioanalyzer and normalized to cellular DNA content. *, significant (P<0.05) changes from vehicle.

Supplementary Figure S2

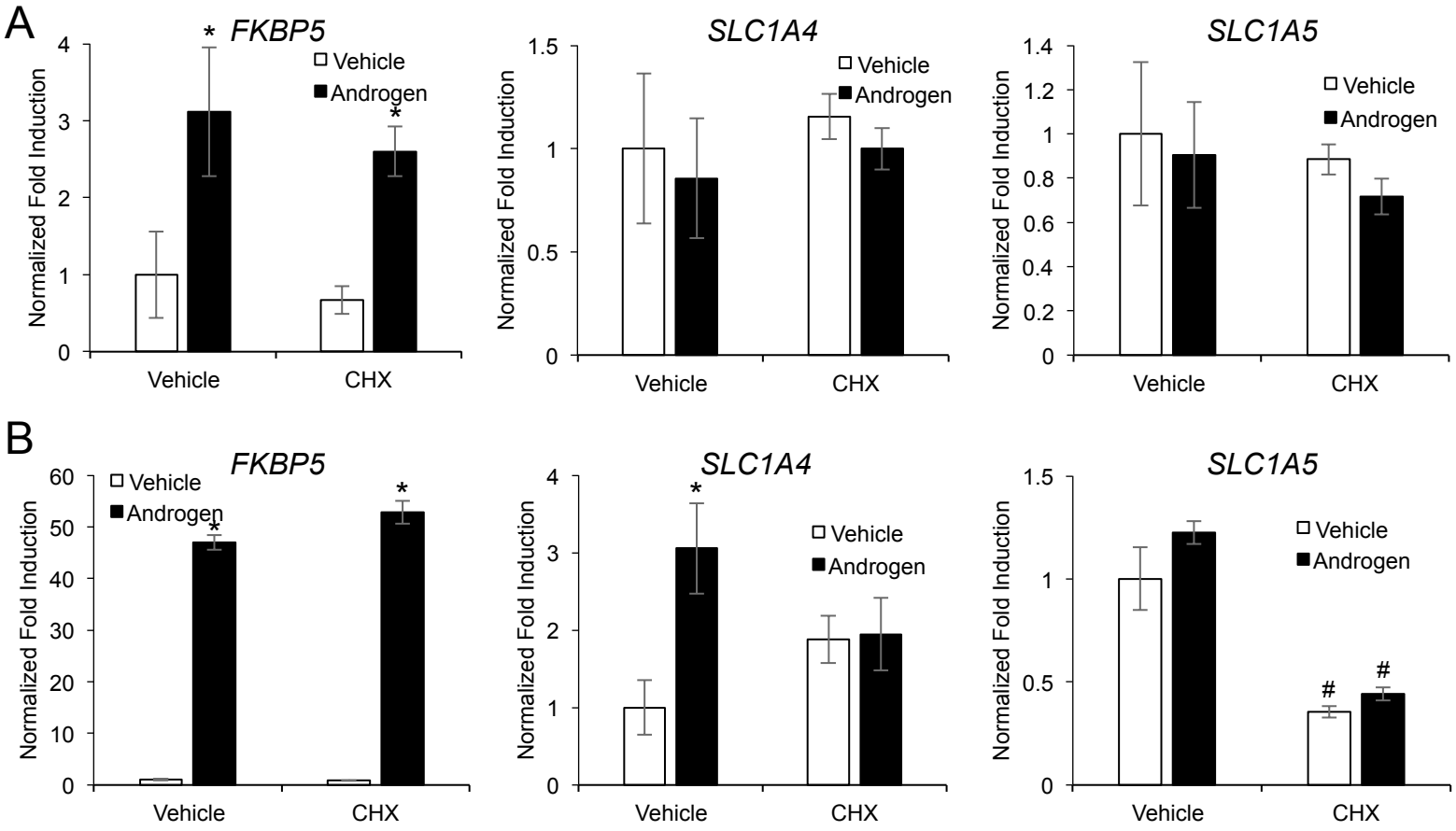

Figure S2. *SLC1A4* and *SLC1A5* are likely secondary targets of AR in prostate cancer cells. LNCaP (A) and VCaP (B) cells were treated with 1 µg/ml cycloheximide (CHX) and either vehicle or androgen (100 pM) for 16 hours. Cells were lysed and RNA collected for qRT-PCR to analyze the mRNA levels of the indicated genes. Results were normalized to 36B4 mRNA levels. *, significant (P<0.05) changes from vehicle. #, significant (P<0.05) changes from no cycloheximide.

Supplementary Figure S3

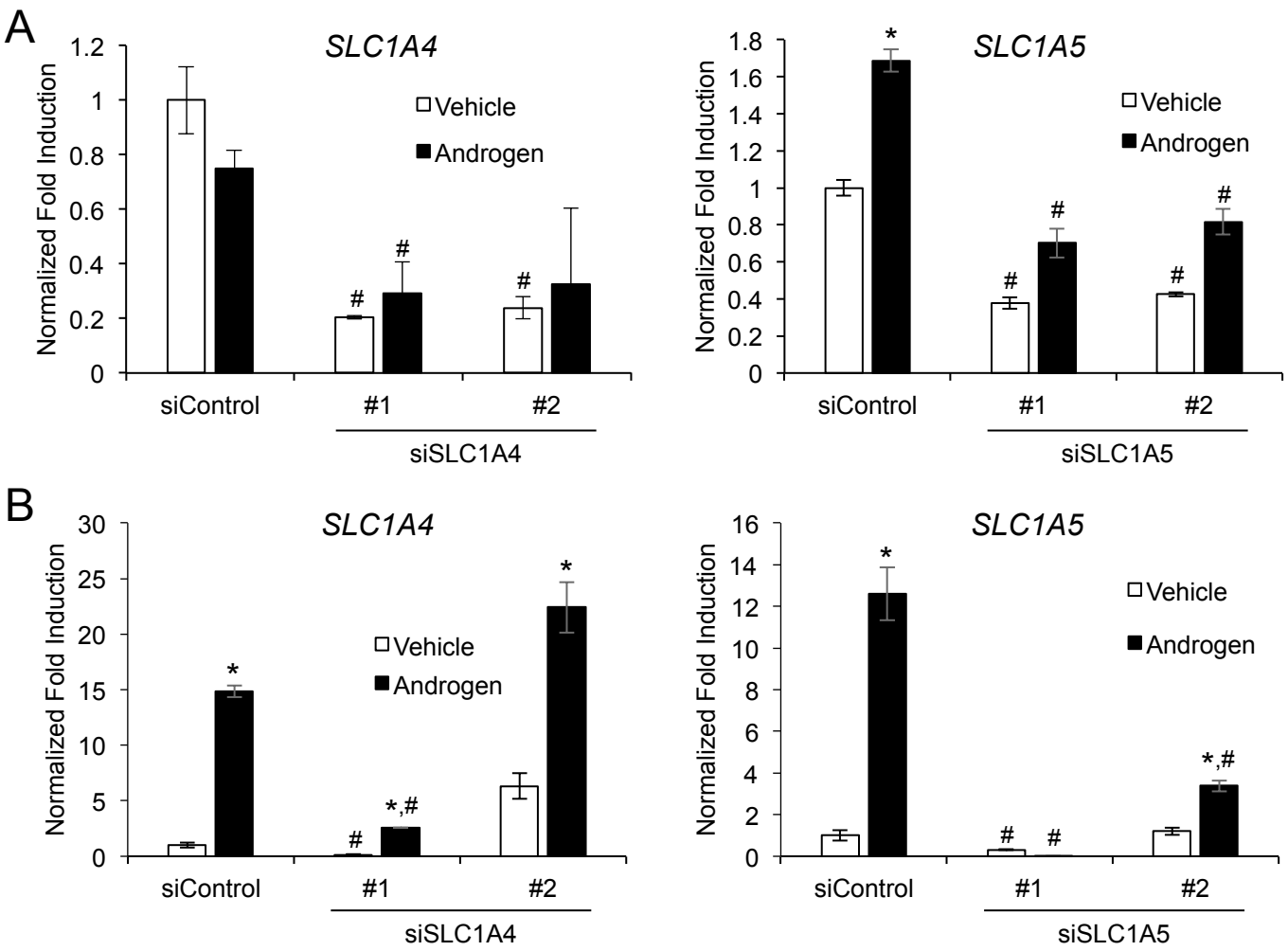

Figure S3. qRT-PCR knockdown controls for Figure 3. LNCaP (A) or VCaP (B) prostate cancer cells were transfected for 3 days with indicated siRNAs and treated ± androgen (100 pM R1881). RNA was then extracted and subjected to qRT-PCR analysis. Results are normalized to 36B4 levels. *, significant (P<0.05) changes from vehicle. #, significant (P<0.05) changes from siControl.

Supplementary Figure S4

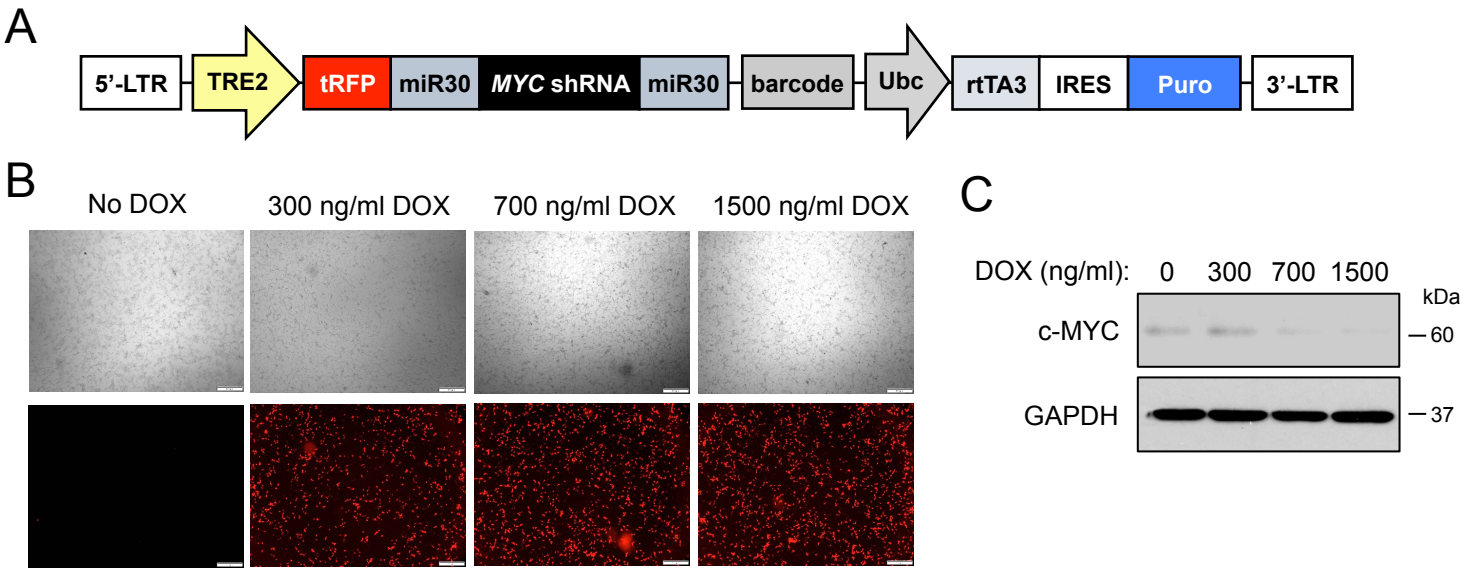

Figure S4. Creation of stable LNCaP cells that express an shRNA targeting *MYC* under the control of a doxycycline (DOX)-inducible promoter. A, construct used to created LNCaP-shMYC stable cell line. B and C, validation of LNCaP-shMYC stable cell line. Cells were cotreated for 3 days with increasing concentrations of doxycycline. Cells were then (B) imaged using phase contrast (*top*) or immunofluorescence (*bottom*) microscopy (scale bars = 500 μm) or (C) lysed and subjected to Western blot analysis.

Supplementary Figure S5

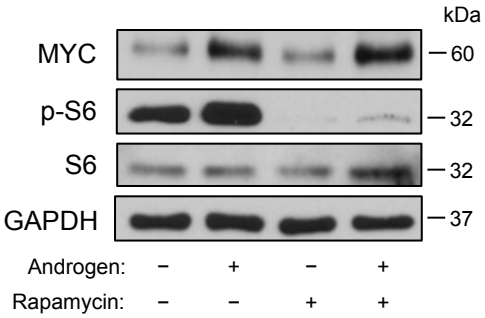

Figure S5. Androgens increase MYC levels independent of mTOR signaling in LNCaP cells. LNCaP cells were cotreated ± 10 nM rapamycin and either vehicle or androgen (100 pM R1881) for 3 days. Whole cell lysates were collected and subjected to Western blot analysis.

Supplementary Figure S6

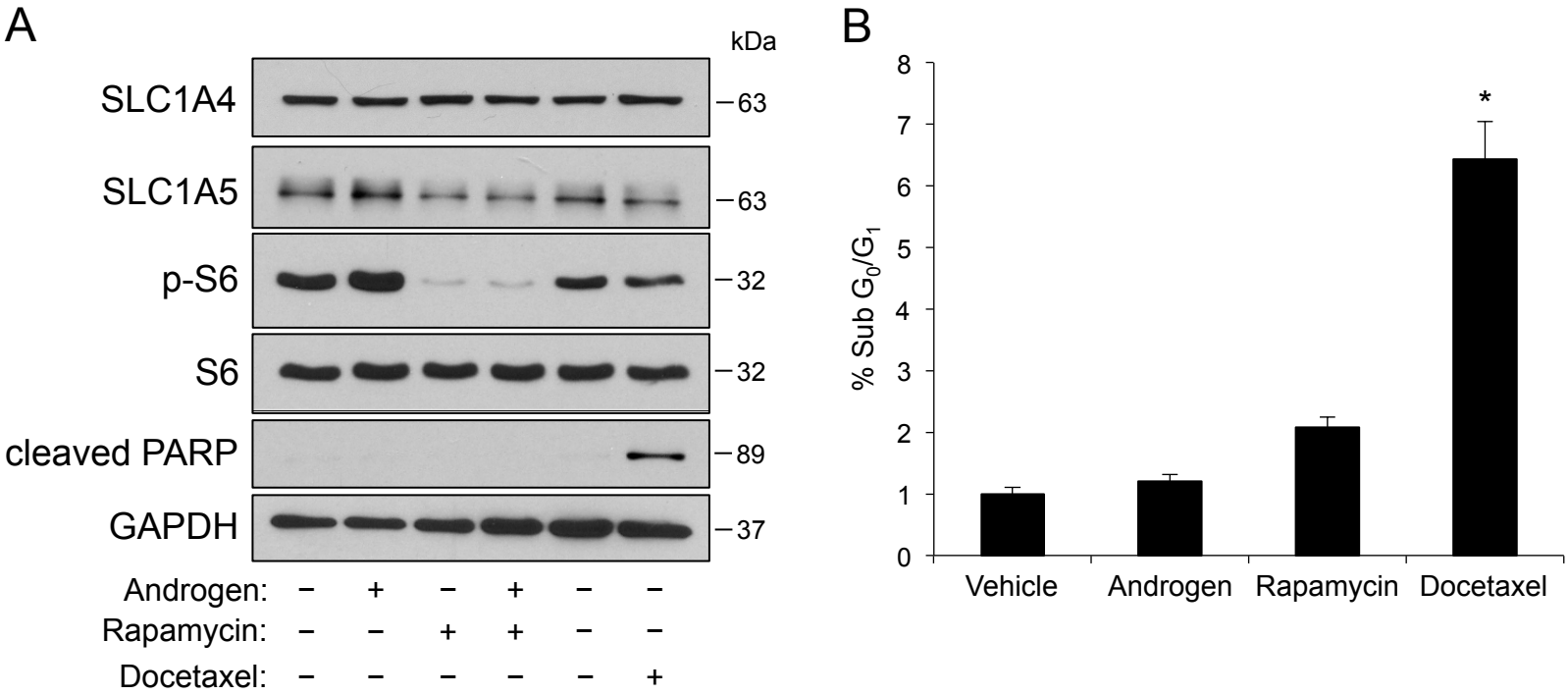

Figure S6. Rapamycin has minimal effects on cell death after 72 hours of treatment in LNCaP cells. LNCaP cells were treated with vehicle, R1881 (androgen), rapamycin or docetaxel (positive control) for 3 days. Whole cell lysates were collected and subjected to (A) Western blot analysis or (B) PI staining and subsequent FACS analysis to assess sub G0/G1 DNA levels. *, significant (P<0.05) changes from vehicle (n = 2).

Supplementary Figure S7

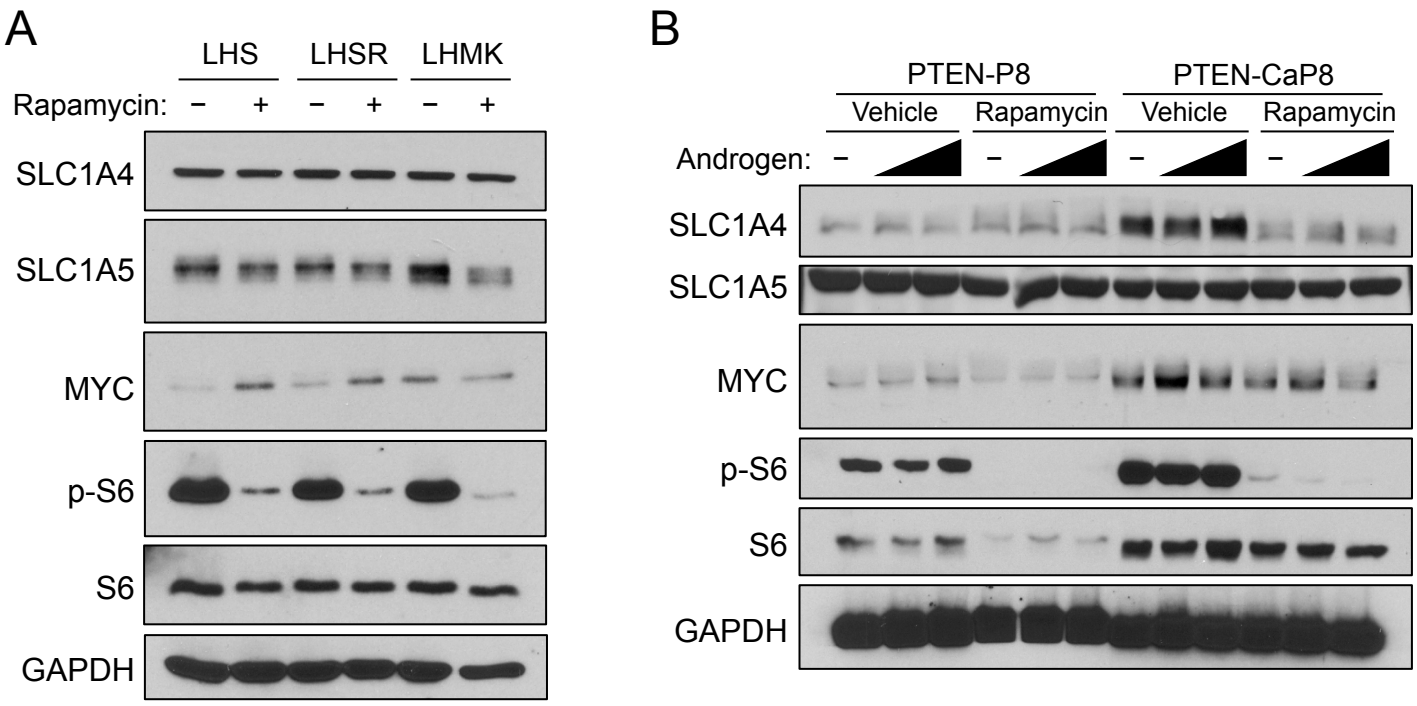

Figure S7. Influence of cancer signaling pathways on SLC1A4 and SLC1A5 expression in genetically defined prostate cell models. A, previously defined PrEC derivative cell models were used to compare the effects of different cancer-related signaling events on SLC1A4 and SLC1A5 levels. LHS (PrEC cells engineered to express the SV40 large T antigen (LT), small t antigen (ST), *hTERT*), LHSR (LHS cells engineered to also express H-*ras*) and LHMK (PrEC cells engineering to express SV40 LT, *hTERT*, *MYC* and PI3K) cells were treated for 72 hours with vehicle or 10 nM rapamycin. B, previously defined isogenic mouse prostate cell lines were used to evaluate the role of *Pten* in SLC1A4 and SLC1A5 expression. PTEN-P8 is heterozygous for *Pten* deletion. Its isogenic partner, PTEN-CaP8, is homozygous for *Pten* deletion. Cells were cotreated for 72 hours with increasing concentrations of androgen (R1881: 0, 0.1 and 10 nM) and vehicle or 10 nM rapamycin. A and B, cells were then lysed and subjected to Western blot analysis.