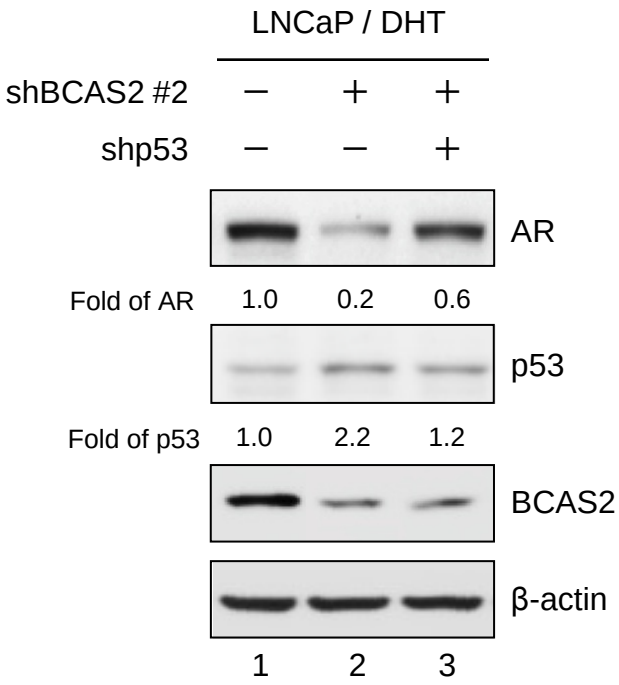
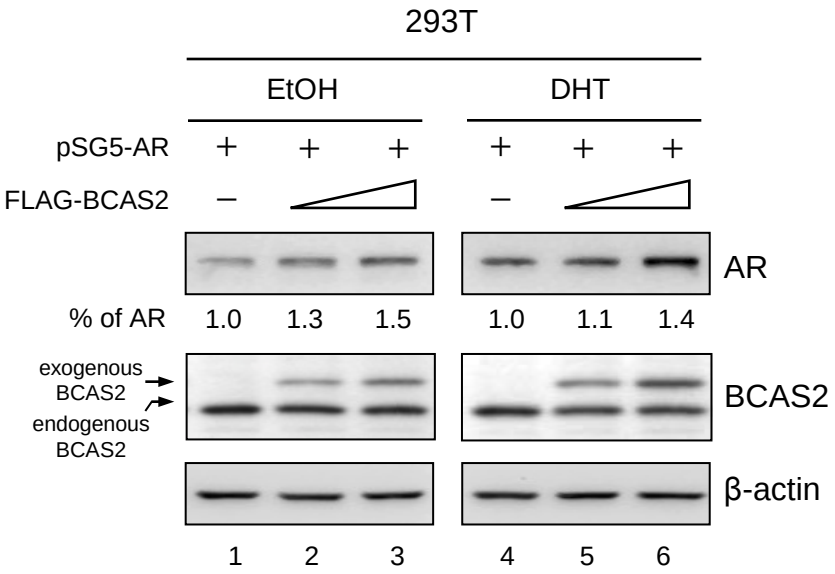
Supplementary Figure 1



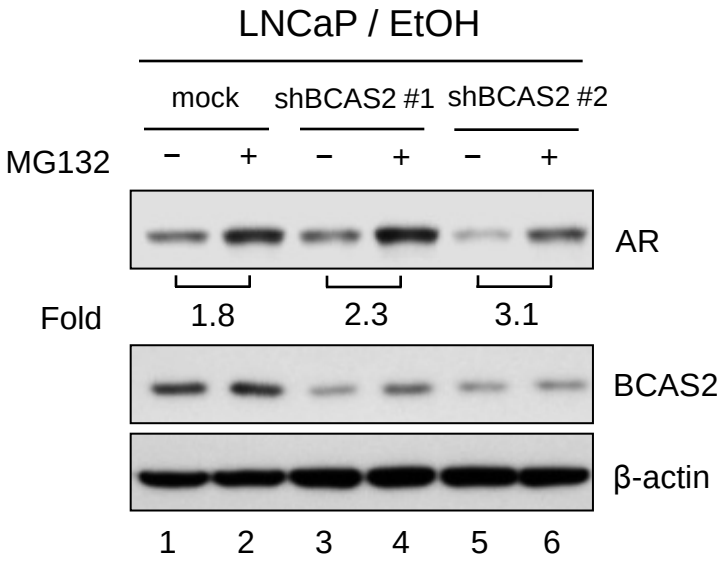
Supplementary Figure 1. Depletion of BCAS2 decreases AR through a p53-dependent pathway. In the presence of DHT, LNCaP cells transfected with shBCAS2#2 along, with shp53 or shCtrl, were subjected to western blotting with the antibodies indicated.

Supplementary Figure 2



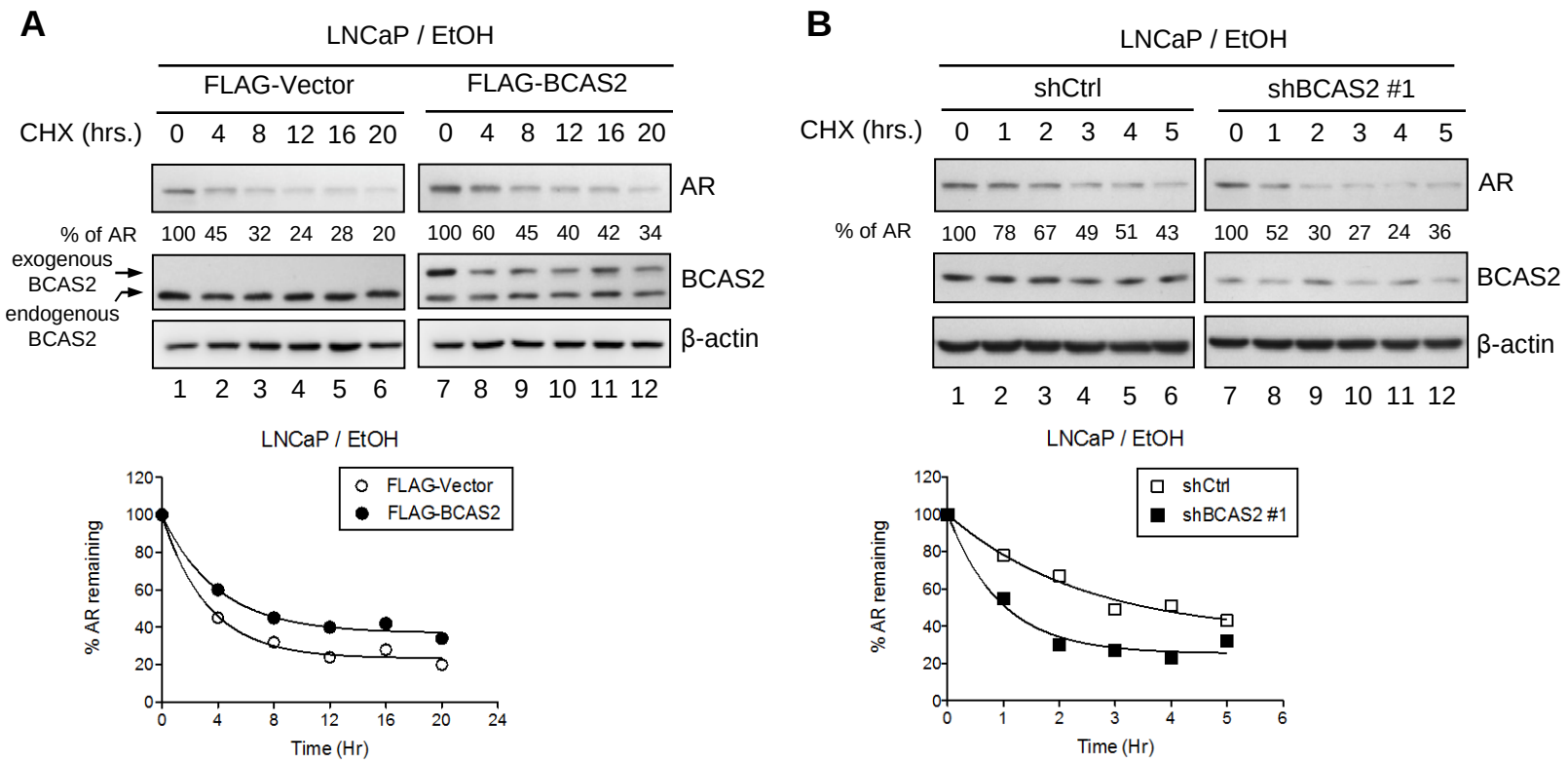
Supplementary Figure 2. BCAS2 enhances AR protein expression in 293T cells. 293T cells (p53 is blocked by SV40 T Ag) were transiently transfected with pSG5-AR (AR gene expression driven by a heterologous SV40 promoter) along with increasing doses of FLAG-BCAS2 plasmid. The expression levels of AR protein were quantified by UVP BioSpectrum-AC imaging software and data were normalized to β-actin.

Supplementary Figure 3



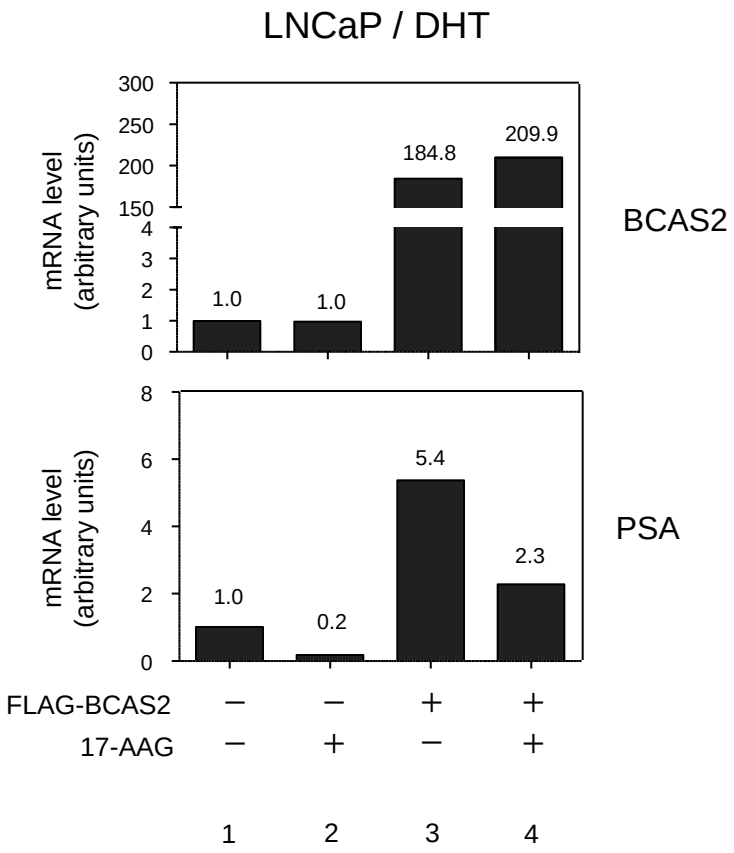
Supplementary Figure 3. BCAS2 protects AR from proteasome degradation without ligand treatment. LNCaP cells transfected with shCtrl, shBCAS2#1 or shBCAS2#2 were treated with MG132 (20 μM) or DMSO in the absence of ligand. The levels of AR protein were quantified by UVP BioSpectrum-AC imaging software and data were normalized to β-actin. The fold of increase in AR level were indicated.

Supplementary Figure 4



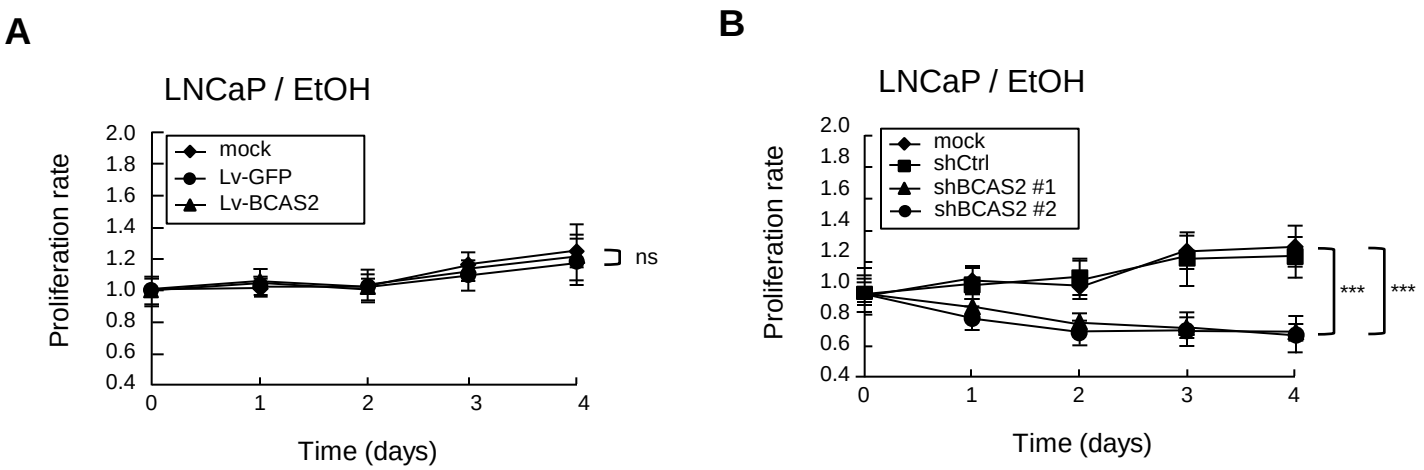
Supplementary Figure 4. BCAS2 enhances AR protein stability without ligand treatment. (**A**) Overexpression of BCAS2 increases AR protein stability. In the absence of DHT, LNCaP cells transfected with FLAG-BCAS2 or FLAG-Vector were incubated with CHX (200 μg/ml) for the times indicated. The levels of AR protein were quantified using UVP BioSpectrum-AC imaging software and data were normalized to β-actin. The % of AR indicates the amount of AR at each time point, relative to the control (time 0 set as 100%). Upper panel, Western blot. Lower panel, the calculated regression curves. (**B**) Depletion of BCAS2 reduces AR protein stability. LNCaP cells transfected with shCtrl or shBCAS2#1 in the absence of ligand were subjected to CHX-chase assay as described in panel **A**.

Supplementary Figure 5



Supplementary Figure 5. Overexpression of BCAS2 restores PSA expression level from 17-AAG treatment in LNCaP cells. LNCaP cells transfected with FLAG-BCAS2 were treated with 17-AAG (0.2 μM) in the presence of DHT. BCAS2 and PSA mRNA levels were determined by qPCR.

Supplementary Figure 6



Supplementary Figure 6. BCAS2 affects the LNCaP cell growth rate without ligand treatment (**A**) Overexpressed BCAS2 cannot alter the growth rate of AR-containing prostate cancer cells without ligand treatment. LNCaP cells infected with Lv-BCAS2 or Lv-GFP virus were growth in phenol red-free RPMI medium containing 5% CDS with EtOH treatment. The growth rates of LNCaP cells were counted using the MTT assay method. ns, no significance, Student's *t*-test. (**B**) Depletion of BCAS2 reduces the growth rate of AR-containing prostate cancer cells without ligand treatment. The growth rates of LNCaP cells transfected with shBCAS2#1, shBCAS2#2, or shCtrl plasmids were also determined using MTT assay. Means are shown from three independent experiments. ***: Student's *t*-test, $p<0.001$.

Supplementary Table 1

Supplementary Table 1. Identification of a new siRNA construct against BCAS2

Target Sequence for BCAS2 RNAi Constructs		
shBCAS2#2	5’-AGACTGGCTGCTCGACAAC-3’	Position 214 – 232
shCtrl	5’-CATGCCTGATCCGCTAGTC-3’	–

Supplementary Table 2

Supplementary Table 2. Real-time PCR primers used in this study.

Primers for Real-time PCR	
AR	F 5'-GGAATTCCTGTGCATGAAA-3'
	R 5'-CGAAGTTCATCAAAGAATT-3'
BCAS2	F 5'-CACAGCATGGATGTAATGCC-3'
	R 5'-TCCAGCTGTGAGTTGCATGT-3'
PSA	F 5'-AGACACTCACAGCAAGGATGGA-3'
	R 5'-CTCCTTGGCTCACAGCCTTCT-3'
GAPDH	F 5'-TGCACCACCAACTGCTTAGC-3'
	R 5'-GGCATGGACTGTGGTCATGAG-3'