Supplementary Materials and Methods

Plasmids

The pcDNA3.1-CCNE2 (plasmid 19935) was purchased from Addgene (Addgene, Cambridge, MA). The pcDNA3.1-CCNA2 construct was generated by subcloning the full-length CCNA2 from Rc/CMV-CCNA2 (plasmid 8959, Addgene) into the pcDNA3.1 vector (Invitrogen).

Establishment of LNCaP stable cell lines

LNCaP cells were transfected with the pcDNA3.1-CCNE2 and the pcDNA3.1-CCNA2 constructs. Two days after transfection, cells were selected by G418 for 2 weeks. G418 resistant colonies were picked up and identified by Western Blots analysis.

Correlation of FoxA1 cistrome in LNCaP and abl cells with clinical ADPC and CRPC microarray data.

abl-specific binding sites (3,862) were defined as those regions with significant FoxA1 binding in abl (MAT-score $\geq$ 3.72, equivalent to p-value $\leq 1\times10^{-4}$) while the corresponding binding in LNCaP was insignificant (MAT-score $\leq$ 1.28, p-value $\geq$ 0.1), whereas LNCaP-specific binding sites (717) were defined the other way around. Common binding sites (14,248) are those regions with significant FoxA1 binding (MAT-score $\geq$ 3.72) in both abl and LNCaP. In order to improve signal-to-noise ratio, all the remaining binding sites (grey points in Supplementary Fig. S3A) were not considered in this comparison. As a control, we randomized FoxA1 binding sites by keeping the chromosome and size unchanged while shuffled the genome coordinates using BEDTools

(1). Consequently, each group of binding sites has its matched random control, which was used to estimate the background distribution of number of genes associated with FoxA1 binding sites.

To determine the differential expressed genes in clinical CRPC compared with ADPC, we compared 94 ADPC cases with 39 CRPC cases from three clinical ADPC/CRPC gene expression datasets. We selected top 200 over-expressed and under-expressed genes in CRPC versus ADPC. As a control, we also randomly picked 200 genes from Refseq database (2).

We associated each of the overexpressed, underexpressed and random gene lists with each of the FoxA1 binding site lists (i.e. abl-specific, LNCaP-specific, and common binding sites), respectively. Genes having FoxA1 binding sites within 20 kb of the transcription start site (TSS) were defined as FoxA1 associated genes. Those genes having both cell line-specific and common binding sites will be regarded as cell line-specific FoxA1 associated genes. Genes having both abl-specific and LNCaP-specific binding sites were not identified. To determine the statistical significance of the association, we estimated the background distribution of "number of genes with abl-specific FoxA1 binding sites" by shuffling the binding sites 1000 times. Because the distributions were approximately normal (Supplementary Fig. S3C), we calculated the p-values of abl FoxA1 binding association according to a normal distribution. However, in the case of "number of genes with LNCaP-specific binding sites", the actual distribution is only part of the bell curve (Supplementary Fig. S3C). We thus determine those p-values of LNCaP FoxA1 binding association in a non-parametric manner (i.e. area under

the curve with the number of genes equal to or greater than the one that was actually observed).

Supplementary Table S1: Primers and siRNA sequences

RT-PCR primers	**Sequence (5'-3')**
CCNE2 mRNA+	ACCTCATTATTCATTGCTTCCAA
CCNE2 mRNA-	TCTTCACTGCAAGCACCATC
CCNA2 mRNA+	CAGAAAACCATTGGTCCCTC
CCNA2 mRNA-	CACTCACTGGCTTTTCATCTTC
CCNE1 mRNA+	GAAATGGCCAAAATCGACAG
CCNE1 mRNA-	TCTTTGTCAGGTGTGGGGA
CDK2 mRNA+	CTGAAATCCTCCTGGGCTG
CDK2 mRNA-	GAATCTCCAGGGAATAGGGC
CDK4 mRNA+	TGCAGTCCACATATGCAACA
CDK4 mRNA-	GTCGGCTTCAGAGTTTCCAC
CDK6 mRNA+	ATGCCGCTCTCCACCAT
CDK6 mRNA-	TGTCTGTTCGTGACACTGTGC
CCND1 mRNA+	TCCTCTCCAAAATGCCAGAG
CCND1 mRNA-	GGCGGATTGGAAATGAACTT
CCND2 mRNA+ (3)	AAGAATTCCTCCTCAATAGCCTGCAGCAGTA
CCND2 mRNA- (3)	GCGGGATATCGACCTGTGAGAATTCGAT
CCND3 mRNA+	TGACCATCGAAAAACTGTGC
CCND3 mRNA-	TTGAGCTTCCCTAGGACCAG
CDKN1A mRNA+	AGTCAGTTCCTTGTGGAGCC
CDKN1A mRNA-	CATGGGTTCTGACGGACAT
CDKN1B mRNA+	AAGAAGCCTGGCCTCAGAAG
CDKN1B mRNA-	TTCATCAAGCAGTGATGTATCTGA
RB mRNA+	TGTGAACATCGAATCATGGAA
RB mRNA-	TCAGTTGGTCCTTCTCGGTC
E2F1 mRNA+ (4)	CATCCAGCTCATTGCCAAGAAG
E2F1 mRNA- (4)	GATCCCACCTACGGTCTCCTCA
siRNA sequences	
siFoxA1-1 (5)	GGACUUCAAGGCAUACGAA
siFoxA1-2 (6)	GAGAGAAAAAAUCAACAGC
siMYBL2	AAACGAGCCUGCCUUACAA
(ON-TARGETplus SMARTpool siRNA)	GAGCACACCUGUUAAGACC
	GUAACAGCCUCACGCCCAA
	GAACAGGAGCCCAUCGGUA
siCREB1	GAGAGAGGUCCGUCUAAUG
(ON-TARGETplus SMARTpool siRNA)	UAGUACAGCUGCCCAAUGG
	CAACUCCAAUUUACCAAAC
	GCCCAGCCAUCAGUUAUUC
siE2F1	UCGGAGAACUUUCAGAUCU
(ON-TARGETplus SMARTpool siRNA)	GAGAAGUCACGCUAUGAGA
	GAGCAGAUGGUUAUGGUGA
	GAACAGGGCCACUGACUCU
siCCNE2	GCAGAUAUGUUCAUGACAA
(siGenome SMARTpool siRNA)	CAUAAUAUCCAGACACAUA

	UAAGAAAGCCUCAGGUUUG
	AAGACGAAGUAGCCGUUUA
siCCNA2	GCUGUGAACUACAUUGAUA
(siGenome SMARTpool siRNA)	GAUGAUACCUACACCAAGA
	AAGCUGGCCUGAAUCAUUA
	GGAAAUGGAGGUUAAAUGU

ChIP real-time PCR and FAIRE primers	**Sequence (5'-3')**
UBE2C Enhancer 1+ (7)	TGCCATGTGCCCTAGAAACTG
UBE2C Enhancer 1- (7)	CAAGCTCAGCAAAATGGTGAAA
UBE2C Enhancer 2+ (7)	CCACAAACTCTTCTCAGCTGGG
UBE2C Enhancer 2- (7)	TTCTTTCCTTCCCTGTTACCC
CDK1 Enhancer + (7)	GGGAAAGAGAAGCCCTACACTTG
CDK1 Enhancer - (7)	GGGCTGTGCTACTTCTCTGGG
CCNA2 Promoter +	CCCCAGCCAGTTTGTTTCTC
CCNA2 Promoter -	CGAGTGAAGGGTAAACCA AAGG
CCNE2 Enhancer 1+	ACTGAACATTACTATCCTGTCTCCTG
CCNE2 Enhancer 1-	CTTTCAGGTCAGCATTGAGAACATC
CCNE2 Enhancer 2+	GCAAGTGAGTCCTGTGATGTGAG
CCNE2 Enhancer 2-	TACACAGAGATGCCATTCAGTAAACA
CCNE2 Promoter+	CCGCCAGTTTGCTTTTGCT
CCNE2 Promoter-	TGACACCCCCGA AATCCA
CCNE2 Enhancer 3+	TTCCAACTTGTTTGCTTAGTTATCTTCT
CCNE2 Enhancer 3-	GACACCACCGAAGAGCACTGA
CCNE2 Enhancer 4+	TTAATGTTGTGTGTCTTTCCTTTTAGC
CCNE2 Enhancer 4-	CGAACTAGCTCTTTCACTGTGATTG
E2F1 Promoter +	CTGGGTGCAGGATTAGGATAATG
E2F1 Promoter -	CCATCAGCCACCTCTTCCAT
E2F1 Enhancer 1+	GGGACACGGCCACATTGT
E2F1 Enhancer 1-	TGGTCCCCAAGTCCTTCCA
E2F1 Enhancer 2+	CTCTTGTGGACAGAGCTGGACTT
E2F1 Enhancer 2-	GGAATCTGGCTTAGGGATGGT
LNCaP specific binding site 1+	GCTTGCTTGCCAAATAAATCTTG
LNCaP specific binding site 1-	CAGTGGACATGCCAGTAACTTCTT
LNCaP specific binding site 2+	CCTCATGGAACATGCTGATCTACT
LNCaP specific binding site 2-	AAAAAGGCTGGCAGATCCAA
LNCaP specific binding site 3+	GACTGGCTGTGCCTTATTTTCTC
LNCaP specific binding site 3-	CAACAAAGGAAGAGCTGAATGACTAA
LNCaP specific binding site 4+	AGGATAAGCCAAGCCTGACAAC
LNCaP specific binding site 4-	TCAGGTGCCGGACTGACA
LNCaP specific binding site 5+	CACACCATGCTAGATTCCCTCTT
LNCaP specific binding site 5-	GAGCTAGGATTCCAGGCTGTGA
LNCaP specific binding site 6+	AGAGCTAGGACTACTGAAGTGTGCTTT
LNCaP specific binding site 6-	TGGAGGACAGATTGCATTTCC
LNCaP specific binding site 7+	CCCAGCAAGGGTGATCATAAA
LNCaP specific binding site 7-	GACAAGCATTGAACCAGACCAA
LNCaP specific binding site 8+	CTTACCGCACCTGCCACAT

LNCaP specific binding site 8-	CCTTCTTCATCTCTGCAGAAAGC
LNCaP specific binding site 9+	GGAAAGCAGGGCAACACAGA
LNCaP specific binding site 9-	ACCCTAAGCACACAGGCTGATT
LNCaP specific binding site 10+	GGATTGGTTAATGGGTGTCCAT
LNCaP specific binding site 10-	CCTAAGGCCAGGAGCTATTTTG
abl specific binding site 1+	GAATTTGGGAATAGTGACTGAGCAT
abl specific binding site 1-	TCCCAGCAGTGAGGTTCCA
abl specific binding site 2+	GCATAGGCCGGAGGAGTGA
abl specific binding site 2-	CAAACCCCCAGAGGATGCT
abl specific binding site 3+	CCTGGGAGGCCCAACCT
abl specific binding site 3-	CTGAGGGCTGGTAATTAGGATTTC
abl specific binding site 4+	AAACACCCAGCCCTCTCCTT
abl specific binding site 4-	AACCCCGATCATTCACAAACA
abl specific binding site 5+	TCAAGGAATCGGCATCCAA
abl specific binding site 5-	AACTCTTCCTTTAGATCCTCACTCACA
abl specific binding site 6+	CCATTTTAGCCCTGAGCTTCA
abl specific binding site 6-	ATGCCCTAAAGGTTGGTGATTG
abl specific binding site 7+	CACAGGCTTGGTTCAGAATTCA
abl specific binding site 7-	GAACATCACCGGCCTTCCT
abl specific binding site 8+	CCCTTCGGAGAGCTGAATGA
abl specific binding site 8-	GCAAGACAGGCCCCAAAA
abl specific binding site 9+	CTGGCAGGACAGCCTTCAA
abl specific binding site 9-	ACTTCCGCATTTTGTCAACGT
abl specific binding site 10+	CTTTTTTGCTATGTGGCAGTGAGT
abl specific binding site 10-	ATCTTTATCTTGTTTGATAGCACTTGGT
Common binding site 1+	CCTACTTCTTGCCCCTGTAGATCTC
Common binding site 1-	CCTGTGTGATGCAGATCTCACTAGT
Common binding site 2+	ACCTATGTGCAAAATACCTGGAAGT
Common binding site 2-	TTTGCTGAAAGACTGCTTTTCG
Common binding site 3+	TTCTGGACTGCGGCAACAT
Common binding site 3-	GATAGGTTAAGGCTCTGCAAAGGT
Common binding site 4+	TCCTTCACTCCCAAAGGTGACT
Common binding site 4-	GTTAGCAAAGTGCATGGGAATG
Common binding site 5+	CATATGGCACGTTTCCTGCAT
Common binding site 5-	TCCGTCTCAGAACGTGTCAGA
Common binding site 6+	TTCCAATGATGTCAAATCCCATT
Common binding site 6-	TCCGCCCTCTAGCGGTTA
Common binding site 7+	TTGTCCCCAATGTCTTCTAGCA
Common binding site 7-	TTTGGGTTATGGGCCATTTG
Common binding site 8+	AAGTTCTGAGAAAACAGCAGGTGAT
Common binding site 8-	GCCTCCTGGAAGAGCCAATAT
Common binding site 9+	GGTGAGTGCTAAACAGATTGTATGCT
Common binding site 9-	AGCATATCTCAGGACAGAGTGTCAA
Common binding site 10+	TGCCCACCCTGTGCAAA
Common binding site 10-	CCCCTTGGCCTGGATTCT
Negative control 1+	CCTTTGCCTCTTCCCTTTCC

Negative control 1-	GGATCAATGGCAGCTGTGGTA
Negative control 2+	GCAGTGTCAGGTTTGGAGCAA
Negative control 2-	TCATTCGCCCTCCTCTTGAT
Negative control 3+	CCTCATTGGCTGGACAAGGA
Negative control 3-	CTCTCCACTGCCAGCTGTGA
Negative control 4+	CTGCATGCTCAAGGAGTGTGTT
Negative control 4-	CACAGAATCAGTCTAGGGTGCTCTT
Negative control 5+	AGTTTGGGACAGACGGGAAA
Negative control 5-	GCTGATTCAATTACCTCCCAGAA
Negative control 6+ (7)	CCCATGCCAGCAGTAGCTAGA
Negative control 6- (7)	GCACTCACAGAATGCACAGAAAA
Negative control 7+ (7)	AAGAGGGACCATCTCATTTTGG
Negative control 7- (7)	GCTGTCTCCCCGACCTTTC
Negative control 8+ (7)	TTTGCAGTGAGTGCTATGAGAAACT
Negative control 8- (7)	CCCTGAAAGAAAAGAGCTGTCAGT
Negative control 9+ (7)	GGAGTAAAGCTGCTCAGGGAGAA
Negative control 9- (7)	GCCTGGCTGAGTCGGTCAT
Negative control 10+ (7)	GGTTACACACGTTAGGTATTCATCATG
Negative control 10- (7)	TTGCTGTGCCCGTGTAGCT

Supplementary Table S2: Antibody information

Antibody	**Catalogue number**	**Company**
anti-FoxA1	ab23738	Abcam
anti-CCNE2	ab32103	Abcam
anti-CCNA2	ab7956	Abcam
anti-Rb	ab6075	Abcam
anti-H3K4me1	ab8895	Abcam
anti-phosphorylated RNA pol II (phospho S5)	ab5131	Abcam
anti-acetyl-Histone H3	ab47915	Abcam
anti-CBP(451)	sc-1211	Santa Cruz Biotechnology
anti-CDK2(M2)	sc-163	Santa Cruz Biotechnology
anti-phosphor-CDK2 (Thr160)	sc-101656	Santa Cruz Biotechnology
anti-MYBL2 (N-19)	sc-724	Santa Cruz Biotechnology
anti-E2F1 (KH95)	sc-251	Santa Cruz Biotechnology
anti-MED1 (M-255)	sc-8998	Santa Cruz Biotechnology
anti-CREB1	17-600	Millipore
anti-Phospho-Rb (Ser795)	9301	Cell Signaling Technology

Supplementary References

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Supplementary Figure legends

Supplementary Figure S1. Effects of FoxA1 silencing on CWR22Rv1 and LNCaP cell proliferation. A, FoxA1 silencing decreases CWR22Rv1 cells growth. The cell proliferation was measured after siRNA transfection, using the WST-1 assay. ** $p < 0.01$. B, FoxA1 silencing induces G1-S arrest in CWR22Rv1 cells. Seventy-two hours after siRNAs transfection, cells were fixed, stained with propidium iodide, and analyzed by flow cytometry. * $p < 0.05$, ** $p < 0.01$. C, Effects of FoxA1 silencing on androgen-stimulated cell-cycle entry. Forty-eight hours after siRNA transfection, LNCaP cells were treated with or without 10 nM DHT for 24 hours, and analyzed by flow cytometry. D, FoxA1 silencing does not decrease androgen-dependent prostate cancer cell growth. LNCaP cells were transfected with two independent FoxA1 siRNAs in the absence (-) or presence (+) of 10 nM DHT. The cell proliferation was measured after siRNA transfection, using the WST-1 assay.

Supplementary Figure S2. Expression and function of CCNE2 and CCNA2 in CWR22Rv1, LNCaP and abl cells. A, FoxA1 silencing decreases mRNA and protein expression of CCNE2 in CWR22Rv1 cells. Left panel: Seventy-two hours after siRNA transfection, total RNA was isolated and amplified by real-time RT-PCR using gene specific primers. Expression data are summarized in heat maps. Right panel: Western blots were performed using the antibodies indicated 72 hours after siRNA transfection. B, CCNE2 or CCNA2 silencing decreases CWR22Rv1 cells growth. The cell proliferation was measured after siRNA transfection, using the WST-1 assay. ** $p < 0.01$. C, Comparison of cell-cycle distribution of LNCaP cells stably expressing CCNE2 or

CCNA2 with abl cells. LNCaP cells stably expressing CCNE2 or CCNA2 were treated with or without 10 nM DHT for 24 hours. abl cells were cultured in the absence of DHT. Cell-cycle profiles were analyzed by flow cytometry.

Supplementary Figure S3. Mapping the FoxA1 Cistromes in LNCaP and abl cells and correlation with clinical CRPC/ADPC gene expression data. A, Comparison of FoxA1 whole genome binding in LNCaP and abl cells. FoxA1 binding sites were divided into 3 categories: Common binding sites (blue), abl-specific binding sites (red) and LNCaP-specific binding sites (dark green). Dashed lines indicate two cutting-off MAT score value of 3.72 and 1.28. B, Direct ChIP validation of FoxA1 ChIP-chip. FoxA1 binding at cell type-specific binding sites, common binding sites and negative control regions were determined by ChIP assays in LNCaP and abl cells. ** $p < 0.01$. C, Association between cell type-specific FoxA1-binding sites and differential expressed genes in CRPC versus ADPC. Curves in each panel indicate the distribution of "number of genes with FoxA1 binding sites". Distributions were estimated by shuffling the corresponding FoxA1 binding sites 1,000 times. Red dot in each panel presents our observation. P-values in the first and third rows were calculated according to normal distribution (z-test), whereas p-values in the second row were determined non-parametrically (see "Supplementary Materials and Methods"). D, Correlation between cell type-specific FoxA1-binding sites and differential expressed genes in CRPC versus ADPC. P-values upon each bar show the significance level of association between FoxA1 binding and gene having FoxA1 binding sites with 40 kb of the TSS.

Supplementary Figure S4. FoxA1 functions on the UBE2C and CDK1 enhancers. A, Higher H3K4me1 level and greater recruitment of FoxA1, p-Pol II, CBP and MED1 to the UBE2C and CDK1 enhancers in abl cells. ChIP assays were performed using indicated antibodies. * $p < 0.05$, ** $p < 0.01$. B, Effects of FoxA1 silencing on FAIRE enrichment at the UBE2C and CDK1 enhancer regions. LNCaP and abl cells were transfected with siFoxA1 or siControl. FAIRE-qPCR experiments were performed 72 hours posttransfection. Relative enrichment compared to uncrosslinked DNA is shown. * $p < 0.05$, ** $p < 0.01$.

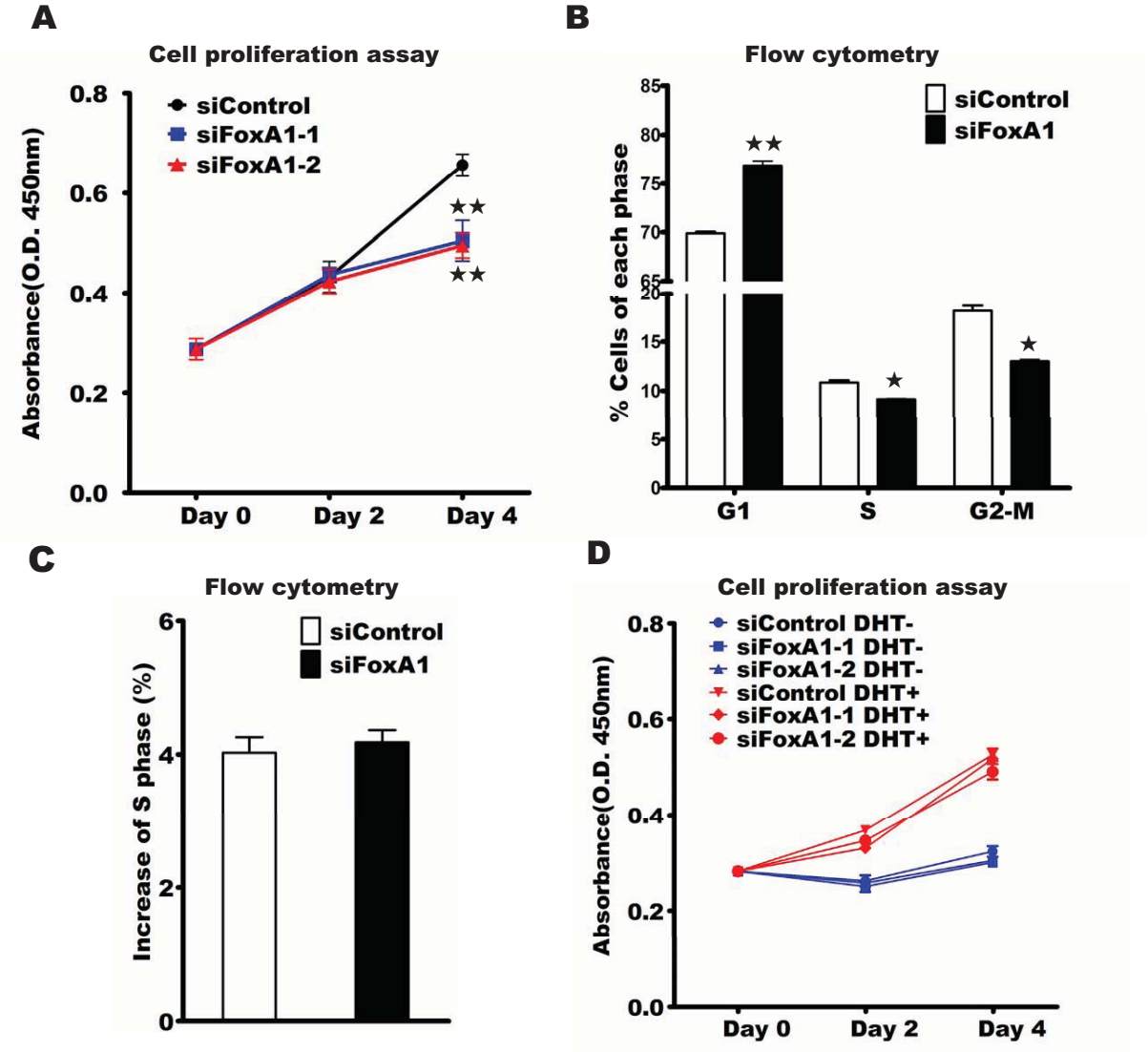
Supplementary Figure S5. Functional studies of FoxA1 collaborating factors MYBL2 and CREB1. A, ChIP analyses of MYBL2 and CREB1 binding at the UBE2C and CDK1 enhancers. * $p < 0.05$, ** $p < 0.01$. B, Effects of MYBL2 or CREB1 silencing on FoxA1 recruitment to the UBE2C and CDK1 enhancers. LNCaP and abl cells were transfected with siMYBL2, siCREB1 or siControl. FoxA1 ChIP assays were performed 72 hours posttransfection. * $p < 0.05$, ** $p < 0.01$. C, Quality controls for ChIP assays after MYBL2 and CREB1 silencing. LNCaP and abl cells were transfected with siMYBL2, siCREB1 or siControl. MYBL2 (Left panel) or CREB1 (Right panel) ChIP assays were performed 72 hours posttransfection * $p < 0.05$, ** $p < 0.01$. D, Effects of MYBL2, CREB1 or FoxA1 silencing on p-Pol II level and/or FoxA1 binding on the CCNE2, E2F1, UBE2C and CDK1 enhancers. Left panel: LNCaP and abl cells were transfected with siRNA targeting each transcription factor or siControl. ChIP assays were performed 72 hours posttransfection using antibodies against p-Pol II. * $p < 0.05$, ** $p < 0.01$. Right panel: Quality controls for ChIP assays after FoxA1 silencing. LNCaP and abl cells were

transfected with siFoxA1 or siControl. FoxA1 ChIP assays were performed 72 hours posttransfection. * $p < 0.05$, ** $p < 0.01$.

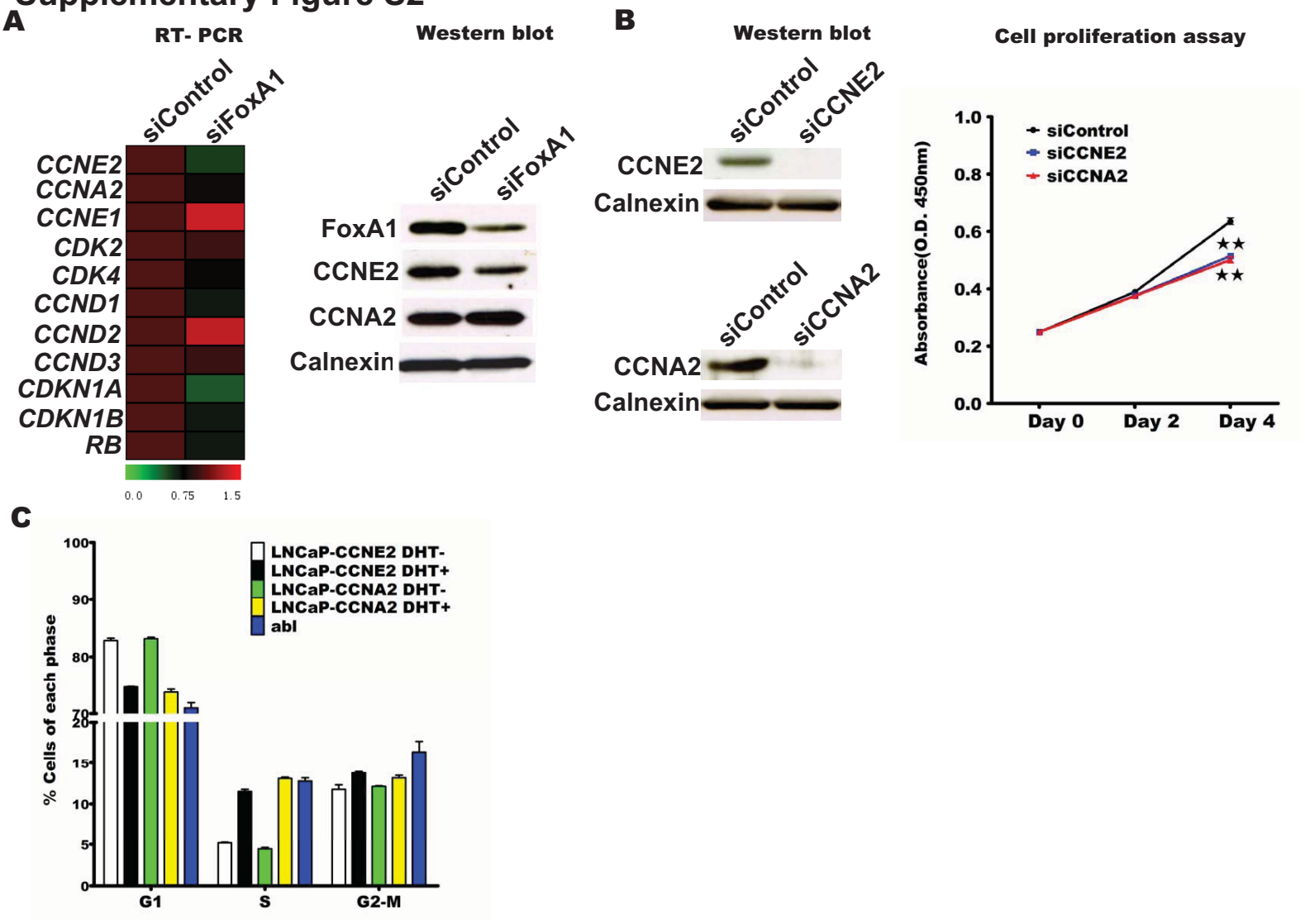
Supplementary Figure S6. FoxA1 collaborates with MYBL2 and CREB1 to regulate target gene expression and cell proliferation. A, Effects of FoxA1 silencing on MYBL2 or CREB1 binding to the UBE2C and CDK1 enhancers. LNCaP and abl cells were transfected with siMYBL2, siCREB1 or siControl. ChIP assays were performed 72 hours posttransfection using an anti-MYBL2 antibody (Left panel) or an anti-CREB1 antibody (Right panel). * $p < 0.05$, ** $p < 0.01$. B, Enhanced interactions between FoxA1 and MYBL2 or CREB1 on regulatory regions of FoxA1 target genes in abl versus LNCaP cells. Re-ChIP assays were performed in LNCaP and abl cells with antibodies against FoxA1 (for first ChIP) and MYBL2 (for re-ChIP) (Left panel), or antibodies against CREB1 (for first ChIP) and FoxA1 (For re-ChIP). C, Effects of FoxA1, MYBL2, CREB1 or CBP silencing on AcH3 level on the UBE2C and CDK1 enhancers. LNCaP and abl cells were transfected with siRNA targeting each factor or siControl. ChIP assays were performed 72 hours posttransfection using an anti-AcH3 antibody. * $p < 0.05$, ** $p < 0.01$. D, Effects of MYBL2 or CREB1 silencing on FAIRE enrichment at the UBE2C and CDK1 enhancers. LNCaP and abl cells were transfected with siMYBL2, siCREB1 or siControl. FAIRE-qPCR experiments were performed 72 hours posttransfection. Relative enrichment compared to uncrosslinked DNA is shown. * $p < 0.05$, ** $p < 0.01$. E, Effects of MYBL2 or CREB1 silencing on UBE2C and CDK1 mRNA expression. LNCaP and abl cells were transfected with indicated siRNA. Real-time RT PCR was performed using UBE2C and CDK1 specific primers 72 hours after siRNA transfection. ** $p < 0.01$. F,

MYBL2 and CREB1 silencing decreases CWR22Rv1 cell growth. The cell proliferation was measured after siRNA transfection, using the WST-1 assay. $^{\star\star}p < 0.01$.

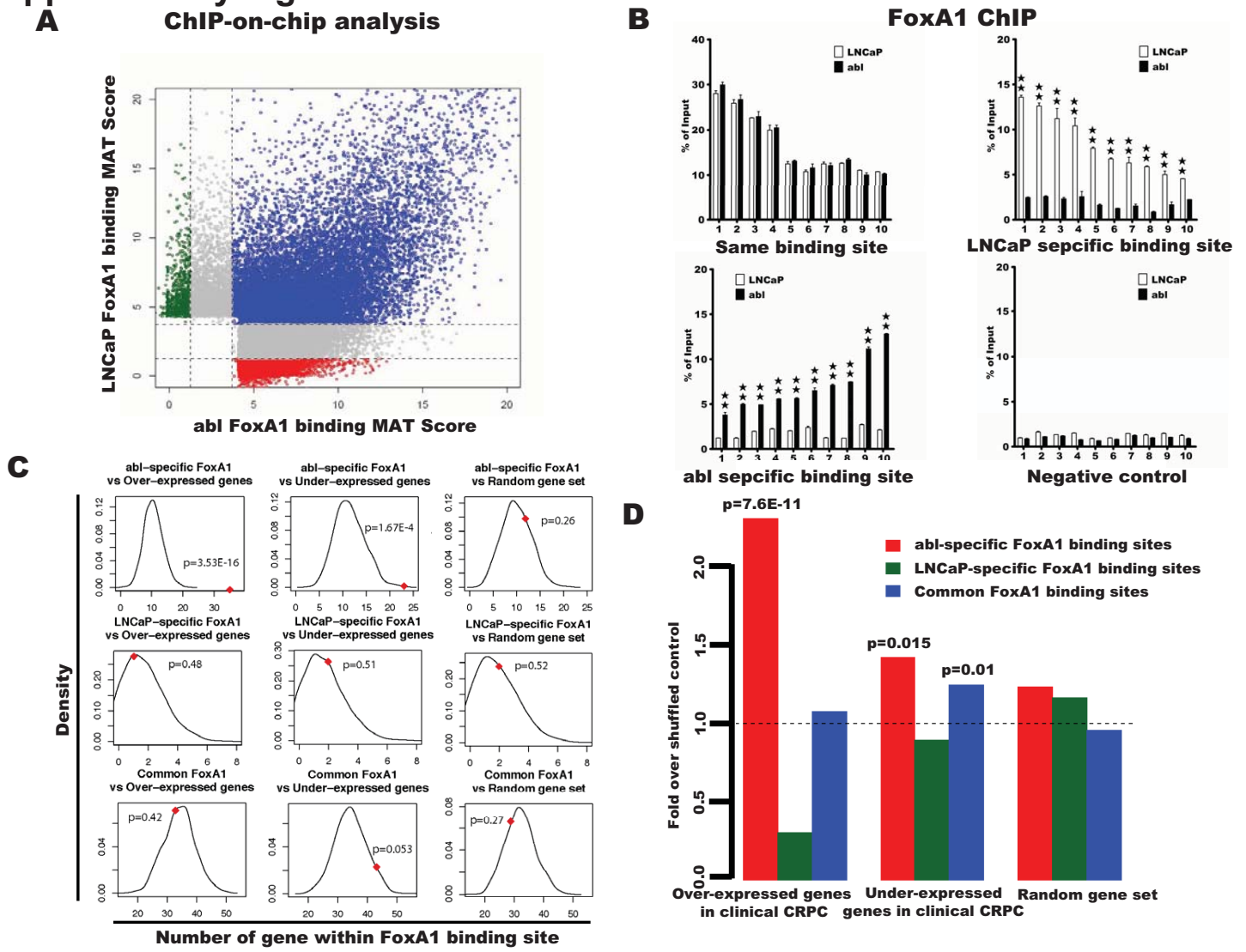
Supplementary Figure S1



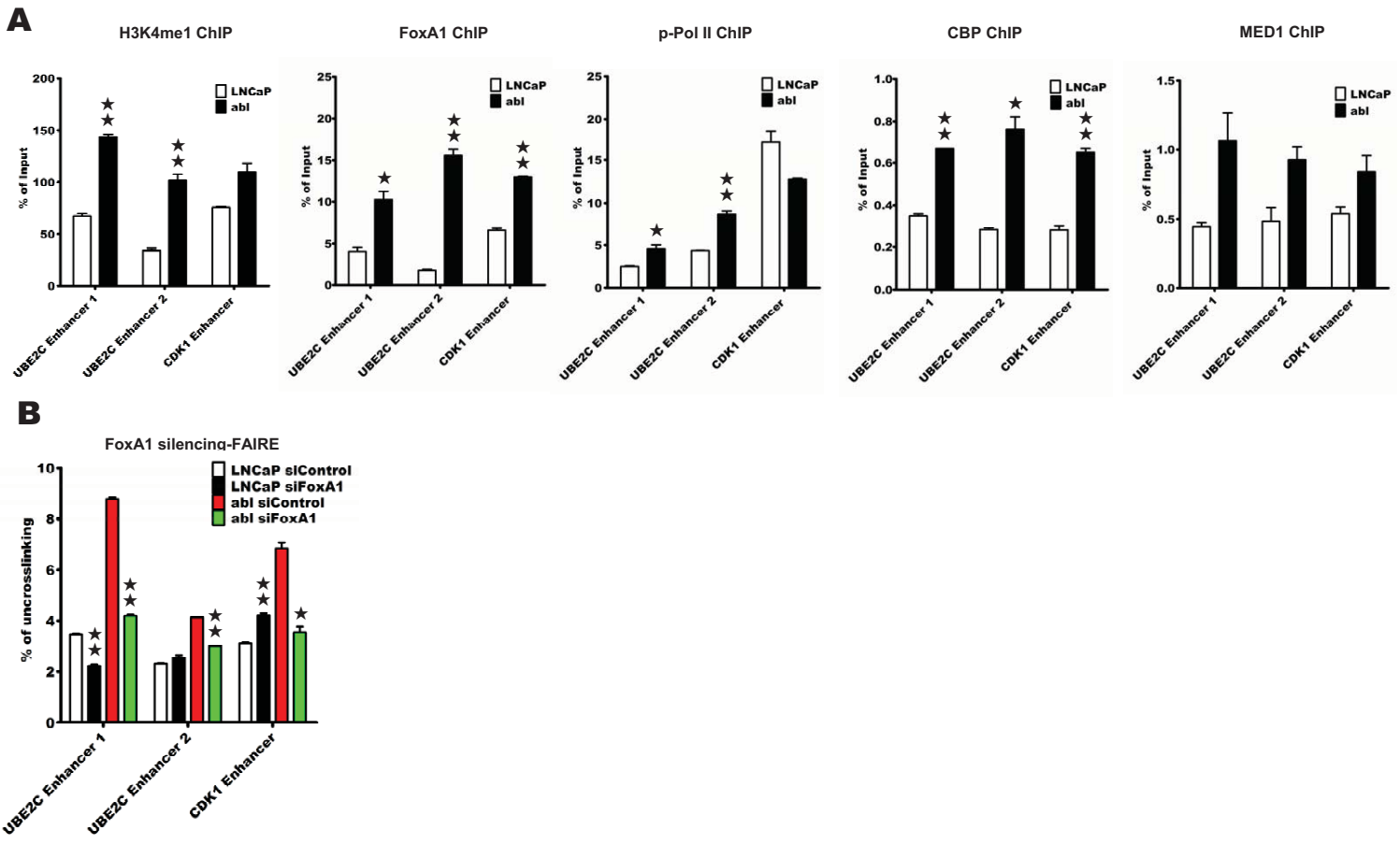
Supplementary Figure S2



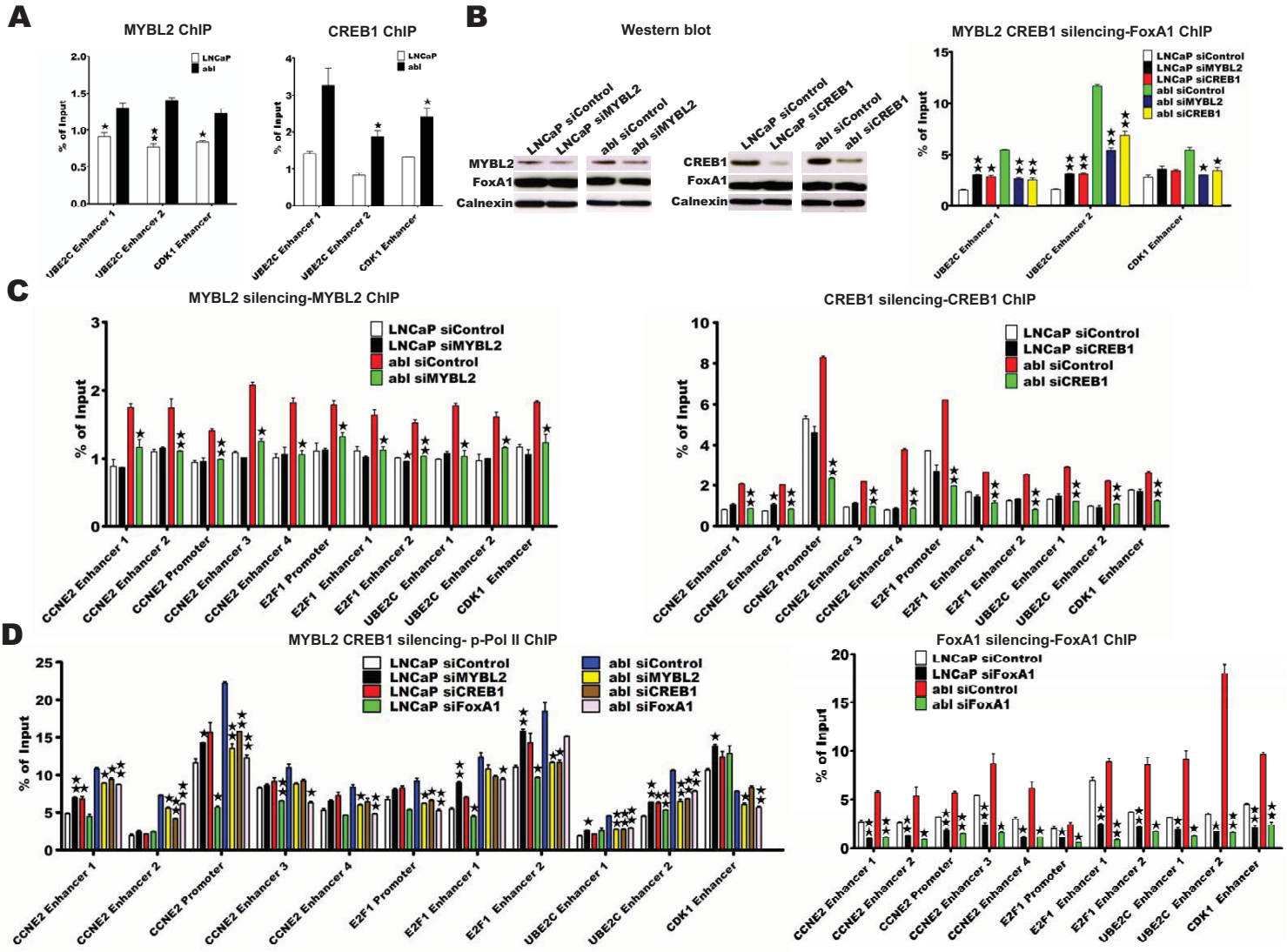
Supplementary Figure S3



Supplementary Figure S4



Supplementary Figure S5



Supplementary Figure S6