Supporting Information

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SI Materials and Methods

Plasmids and Recombinant Proteins. The *PCGEM1* clone (1) was subcloned into pcDNA3 and pLenti4/CMV-TO vectors (Invitrogen) to make *PCGEM1*-expressing plasmid. DNA encoding shRNA specifically targeting *PCGEM1* at sequence GGGCATAAATGATAATGGA was cloned into pLenti4 vector carrying Tet-operating H1 promoter. The *PCGEM1* deletion mutant (Δ761–849) was generated from pcDNA3-*PCGEM1* by QuikChange site-directed mutagenesis. c-Myc, p53, and HIF-1α were constructed into pcDNA3 vector with N-terminal HA epitope tag. Recombinant c-Myc protein was purchased from Active Motif.

Cell Culture. Cell lines LNCaP, PC3, and HEK293T (293T) were purchased from ATCC and were cultured according to the manufacturer's instructions. For hormone deprivation, 10% (vol/vol) charcoal dextran-treated (CDT)-FBS was used to make RPMI-CDT medium. LNCaP cells were washed by PBS and then cultured in the CDT medium for 3 d to achieve complete hormone deprivation. For androgen treatment, cells cultured in CDT medium for 3 d were subjected to 1 nM dihydrotestosterone (DHT) treatment for 24 h.

Cell Proliferation, Flow Cytometry, and Caspase Activity. LNCaP cells were seeded in triplicate in 48-well plates, and the proliferation was monitored using MTT Cell Proliferation Kit I (Roche Applied Science) on days 2, 4, 6, 8, and 10 according to the manufacturer's instructions. LNCaP/shPCGEM1 cells treated with or without DOX for 3 d were harvested and fixed by 70% (vol/vol) ethanol for >4 h at −20 °C, followed by propidium iodide (Sigma Aldrich) staining. The DNA content was analyzed by Becton Dickinson FACScan flow cytometry, and the cell-cycle population at different stages was quantified by WinMDI 2.9. The caspase activity was measured by the Caspase-Glo 3/7 Assay System (Promega) following the manufacturer's instructions.

Metabolic Enzyme Reactions. The cell-culture media and cell pellets were harvested after 3 d of DOX treatment to LNCaP/shPCGEM1 cells for knockdown, or after 3 d of lentiviral infection for *PCGEM1* overexpression (LNCaP-PCGEM1). Concentration of glucose and lactate in the culture medium was determined using commercial enzyme-based kits (BioVision, Inc.) to estimate glucose consumption and lactate production. The G6PD activity and concentration of citrate and NADPH in the cell pellets were determined by enzyme-based kits (BioVision, Inc.) following the manufacturer's instructions.

qRT-PCR. Total cellular RNAs were isolated using RNeasy Plus kits (Qiagen), and cDNA was prepared using SuperScript III first-strand synthesis reagents (Invitrogen). cDNA was quantified by the Bio-Rad CFX Real-Time PCR detection system using SYBR Green Supermix (Ferments) and the primers listed in Table S1. The expression levels were quantified using the comparative *Ct* method and were normalized against *actin* level.

Luciferase Assay. The FK506 binding protein 5 (FKBP5)-promoter and PSA-enhancer luciferase plasmids used in the transactivation assays are as described previously (2). The Myc-responsive luciferase construct was purchased from Qiagen. PC3 or LNCaP/shPCGEM1 cells were seeded to 24-well plates at a density of 1×10^5 per well and incubated overnight before transfection. For *PCGEM1* knockdown, doxycycline was added to medium upon seeding. On the following day, cells were cotransfected with empty vector or *PCGEM1* constructs with HA-c-Myc or HA-AR, together with Myc-responsive Luc or PSA enhancer-Luc plasmid, respectively. pRL-SV40 *Renilla* luciferase plasmid (Promega) was also cotransfected. Then, 48 h posttransfection, cells were lysed and assayed for luciferase and *Renilla* production using the Dual-Luciferase Assay Kit (Promega). All sample groups were tested in triplicate (mean values are reported), and the luciferase relative light units (RLUs) were normalized against the *Renilla* values acquired for each sample.

ChIP. ChIP assays (2×10^7cells per assay) were performed following the University of California Davis Genome Center ChIP protocol (genomics.ucdavis.edu/farnham). LNCaP cells were cross-linked with 1% formaldehyde for 15 min followed by incubation with 125 mM glycine for 5 min. The cross-linked chromatin was sonicated to achieve the majority of DNA fragments with 200–500 bp. Supernatants were precleared with Protein A/G and were subsequently used for ChIP with specific antibody for overnight incubation at 4 °C. The antibodies used are as follows: anti-c-Myc (N-262) and anti-PolII (Santa Cruz); anti-acetylation-Histone H3 and anti-acetylation-Histone H4 (Millipore); and anti-AR, anti-Histone H3, and anti-Histone H4 (Abcam). The immunoprecipitated complexes were washed with the lysis buffer and eluted in 0.1 M $NaHCO_3$, 1% SDS, followed by overnight reverse cross-linking. The DNA was recovered by a PCR purification kit (Qiagen) and analyzed by qPCR using the primers listed in Table S1. The ChIP-reChIP assay was performed as described (3).

RIP. RIP was performed as previously described (4). Briefly, nuclei were obtained from 2×10^7 of 293T cells expressing *PCGEM1* and HA-tagged proteins. The nuclei were lysed in lysis buffer (100 mM KCl, 5 mM $MgCl_2$, 10 mM Hepes, pH 7.0, 0.5% Nonidet P-40, 1 mM DTT plus RNaseOut, protease inhibitors and phosphatase inhibitor) followed by sonication on ice. The nuclear lysates were then treated with DNase for 30 min and diluted in the NT2 buffer to obtain the final buffer concentration of 50 mM Tris·HCl, pH 7.4, 150 mM NaCl, 1 mM $MgCl_2$, and 0.05% Nonidet P-40. Protein A/G precleared lysates were incubated with α-HA antibody (Covance, Inc.) for IP, and the RNA–antibody complexes were subsequently washed following the standard protocol. Coprecipitated RNA was extracted by TRizol (Invitrogen) according to the manufacturer's protocol and subjected to qRT-PCR analysis.

Biotinylated RNA Pull-Down and Dot Blot Assay. An RNA pull-down assay was performed as previously described (4). *PCGEM1* RNA was in vitro transcribed coupling with the Biotin labeling using T7 RNA polymerase (Ambion) and purified with an RNeasy Mini Kit (Qiagen). The biotinylated RNAs were incubated with LNCaP nuclear extract or purified proteins in the presence of RNaseOut, protease, and phosphatase inhibitors, followed by incubating with streptavidin magnetic beads (GE). The pull-down complexes were resolved in SDS/PAGE and analyzed by Western blotting. The RNA pull-down coupling with dot blot assay was performed as described (5). Briefly, the pull-down biotin–*PCGEM1* complexes were subjected to UV cross-linking and RNA partial digestion by RNaseI. The partially digested *PCGEM1*–Myc complex was subjected to proteinase treatment to remove the protein, followed by phenol-chloroform RNA purification. This purified RNA was then used for hybridization with the nitrocellulose membrane dotted with 27 PCGEM1 probes (Table S2). Following standard washing procedures for nucleic acid blotting, the binding of probe and biotin-RNA was detected by streptavidin-HRP chemiluminescence agents.

ChIRP. ChIRP was performed as described (6) with minor modifications. Briefly, 60-mer antisense DNA probes targeting *PCGEM1* full-length sequences were designed at www.singlemoleculefish.com/designer.html. A set of probes complementary of lacZ RNA was also designed as the negative control. All probes were biotinylated. LNCaP cells were cross-linked with 1.25% (vol/vol) glutaldehyde for 15 min at room temperature and then quenched with 125 mM glycine for 5 min. The cross-linked chromatin was then isolated and hybridized with the probes, followed by streptavidin magnetic beads capturing and wash/elution steps. The ChIRP captured chromatin was then reverse cross-linked and analyzed by qPCR using the primers listed in Table S1.

1. Srikantan V, et al. (2000) PCGEM1, a prostate-specific gene, is overexpressed in prostate cancer. *Proc Natl Acad Sci USA* 97(22):12216–12221.
2. Shi XB, Ma AH, Xia L, Kung HJ, de Vere White RW (2002) Functional analysis of 44 mutant androgen receptors from human prostate cancer. *Cancer Res* 62(5):1496–1502.
3. Furlan-Magaril M, Rincón-Arano H, Recillas-Targa F (2009) Sequential chromatin immunoprecipitation protocol: ChIP-reChIP. *Methods Mol Biol* 543:253–266.
4. Tsai MC, et al. (2010) Long noncoding RNA as modular scaffold of histone modification complexes. *Science* 329(5992):689–693.
5. Yang L, et al. (2013) lncRNA-dependent mechanisms of androgen-receptor-regulated gene activation programs. *Nature* 500(7464):598–602.
6. Schulze A, Harris AL (2012) How cancer metabolism is tuned for proliferation and vulnerable to disruption. *Nature* 491(7424):364–373.

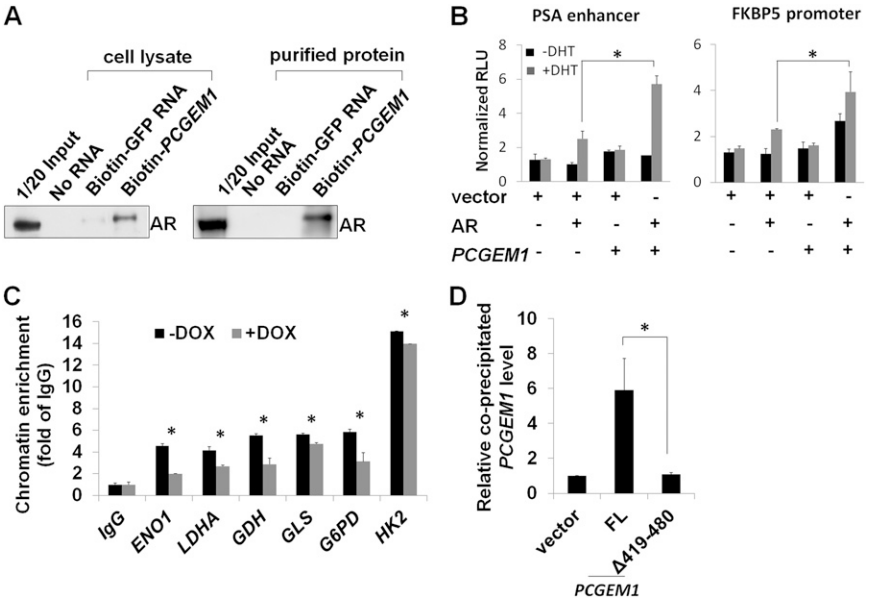

Fig. S1. *PCGEM1* functions as an AR coactivator. (*A*) RNA pull-down of the in vitro transcribed biotinylated-*PCGEM1* incubated with LNCaP cell lysate (*Left*) or purified recombinant AR protein (*Right*). (*B*) The luciferase activity driven by PSA enhancer (*Left*) or FKBP5 promoter (*Right*) was tested in PC3 cells expressing AR, *PCGEM1*, or both. PC3 cells were cultured in CDT medium for 3 d, followed by 1 nM DHT treatment. (*C*) AR ChIP analysis in LNCaP/shPCGEM1 cells. Recovered DNA in the precipitated complexes was analyzed by qPCR, and the DNA levels were normalized against IgG nonspecific bound DNA to obtain the enrichment folds, indicated by fold of IgG. (*D*) Association of HA-tagged AR with full-length (FL) or mutant (Δ419–480) *PCGEM1* was detected by RIP assay (*$P < 0.05$).

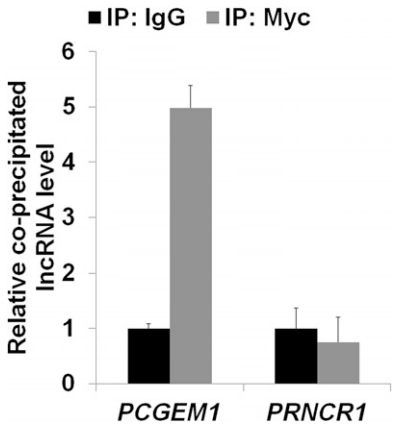

Fig. S2. Specific association of endogenous c-Myc with *PCGEM1* in LNCaP cells. Association of c-Myc with *PCGEM1* or *PRNCR1* was detected by RIP assay using c-Myc antibody. The relative levels of coimmunoprecipitated lncRNAs were calculated as fold difference compared with IgG.

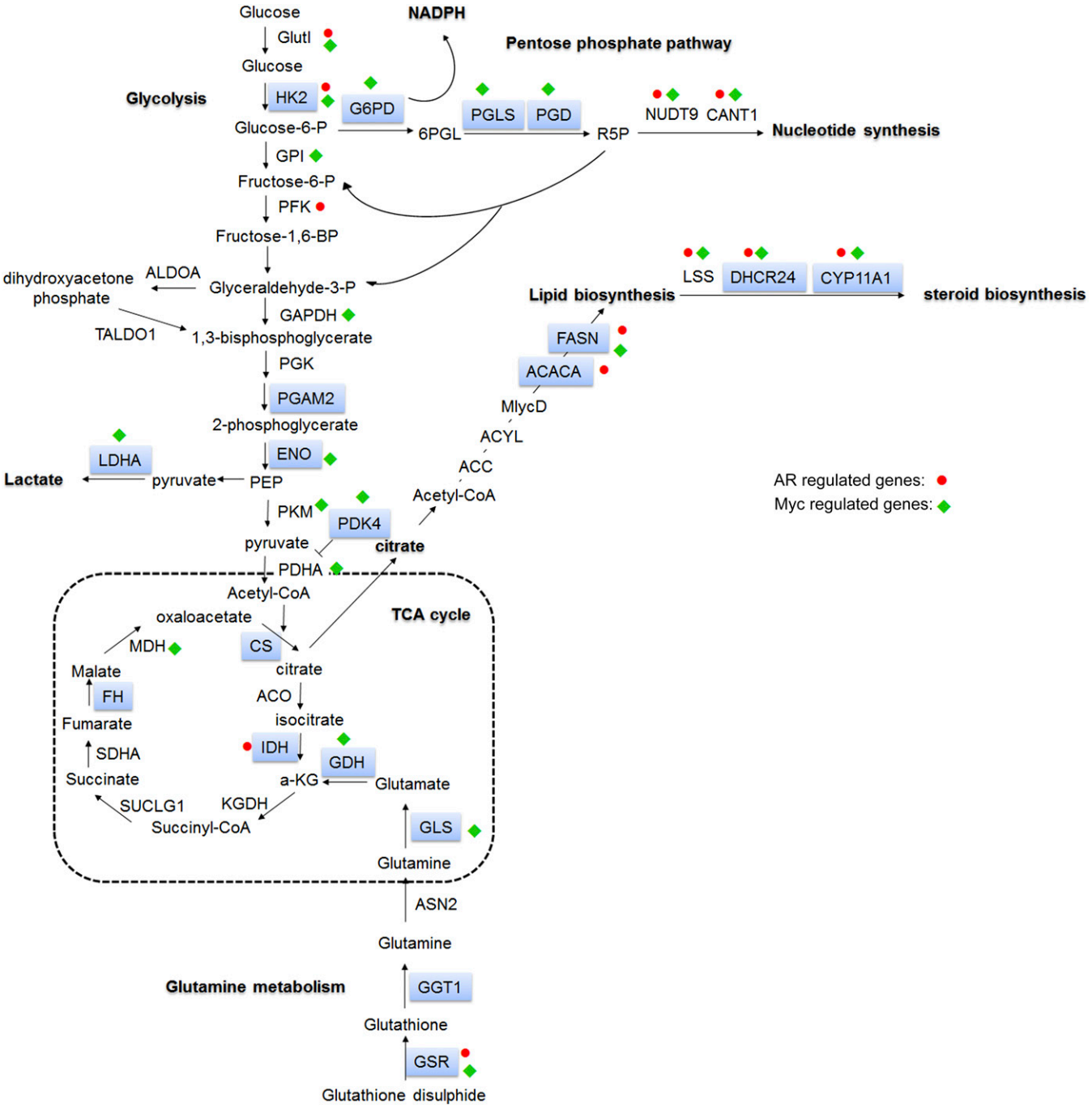

Fig. S3. Schematic metabolic pathways regulated by *PCGEM1*. Genes down-regulated with more than twofold differences in *PCGEM1* knockdown cell are highlighted in blue boxes. Known AR (1) and c-Myc (ENCODE) targets are indicated.

1. Massie CE, et al. (2011) The androgen receptor fuels prostate cancer by regulating central metabolism and biosynthesis. *EMBO J* 30(13):2719–2733.

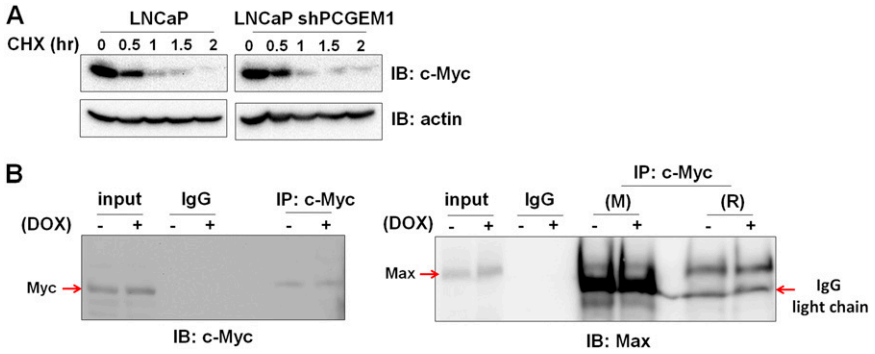

Fig. S4. c-Myc protein stability and Myc-Max dimerization. (*A*) The protein stability of c-Myc was analyzed in control and *PCGEM1* knockdown LNCaP cells with the indicated cycloheximide treatment (20 μg/mL). To knockdown *PCGEM1*, the LNCaP/shPCGEM1 cells were treated with DOX for 3 d before the cycloheximide treatment. (*B*) Interaction of c-Myc and Max in the presence or absence of *PCGEM1* (−DOX or +DOX, as described in *Materials and Methods*) was detected by coimmunoprecipitation. Two different Myc antibodies, one originated from rabbit (Santa Cruz Biotech N-262), and one from mouse (Santa Cruz Biotech 9E10), were used to perform IP independently. Normal rabbit and mouse IgG were used as negative controls. For the immunoblotting, rabbit anti-Myc (N-262) and mouse anti-Max (H-2) antibody were used. (*Left*) Mouse anti-Myc antibody was used for IP, followed by IB with rabbit c-Myc antibody. (*Right*) Mouse and rabbit c-Myc antibodies were used for IP as indicated. Rabbit normal IgG was used as control. IB was carried out using mouse anti-Max antibody.

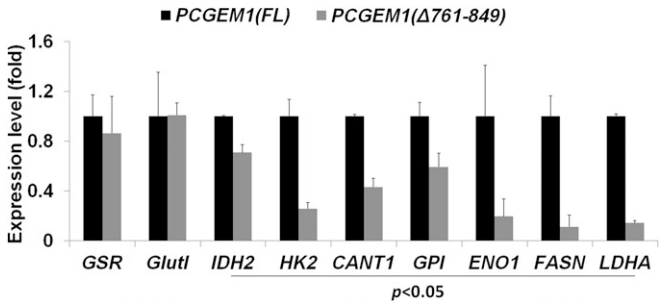

Fig. S5. Metabolic gene expression in LNCaP cell overexpressing full-length (FL) or mutant (Δ761–849) *PCGEM1* was detected by qRT-PCR. The normalized expression levels are shown by fold difference.

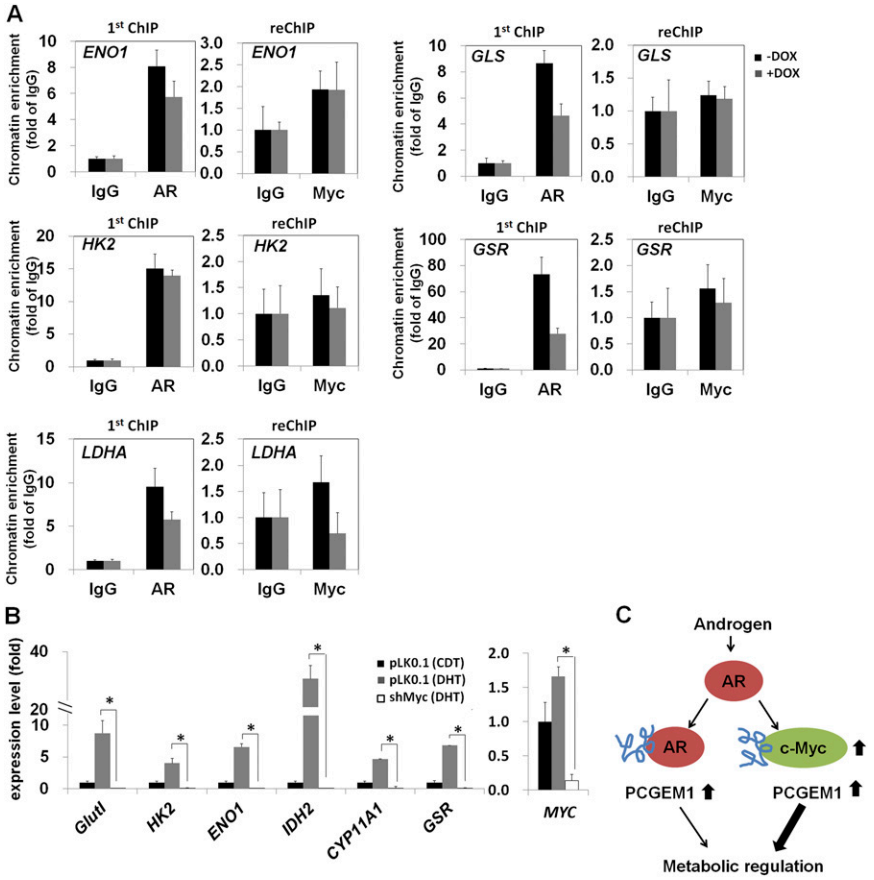

Fig. S6. (*A*) ChIP-reChIP analysis of the AR-chromatin complex in LNCaP/shPCGEM1 cells. The AR-bound chromatin was obtained by ChIP assay (1st ChIP) using AR antibody with IgG as control. A portion of the precipitated DNA was analyzed by qPCR to detect AR enrichment on the indicated promoters. The other portion of the AR-bound chromatin complex was subjected to a subsequent ChIP assay (reChIP) using c-Myc antibody. The DNA levels were normalized against IgG to obtain the enrichment folds, indicated by fold of IgG. *PCGEM1* knockdown was achieved by DOX treatment as described in *Materials and Methods*. (*B*) qRT-PCR comparison of the metabolic gene expression in control LNCaP cells (pLKO.1) cultured in CDT- or DHT-treated medium, and that in c-Myc knockdown cell (shMyc) treated with DHT. The normalized expression levels were compared with pLKO.1 (CDT), shown by fold (*$P < 0.05$). *MYC* mRNA expression level is shown *Right*. (*C*) The working model of androgen-induced tumor metabolic regulation. Androgen treatment induces *PCGEM1* and c-Myc expression. The overexpressed *PCGEM1* forms complex with and coactivate c-Myc and AR and in turn corporately reprogram metabolic gene expression.

Table S1. Cloning primers and PCR primers used in the present study

Primer name	Sequence
Cloning primers	
PCGEM1-CpoF	GGCCGGTCCGAAGGCACTCTGGCACCCAGTTTTGGAAC
PCGEM1-F400	GGCCGGTCCGCTAGGGCAGCAAAAAGTGGC
PCGEM1-R800	GGCCGGACCGTGCAACAAGGGCATTTCAGAAT
PCGEM1-F800	GGCCGGTCCGCTGCCCTTGTTGCAAATATTGGTT
PCGEM1-R1200	GGCCGGACCGATGCTGGATTGTTCCCACAATTT
PCGEM1-CpoR	GGCCGGACCGCCAGGTGCTTTTTTTTTTTTTTTCTATTTCGAAATCT
PCGEM1-del761-849-antisense	GGGCATTTCAGAATGCAAAGACTCTGCAAAGGTTGTATCTTT
PCGEM1-del761-849	AAAGATACAACCTTTGCAGAGTCTTTGCATTCTGAAATGCCC
qPCR primers	
rtACACA	F:TCACACCTGAAGACCTTAAAGCC
	R:AGCCCACACTGCTTGTACTG
rtACO	F:GATATGGGCGCTTACCATTTTCG
	R:TGTGCTGCGTGACATTCCAA
rtactin	F:GTACCACTGGCATCGTGATGGACT
	R:CCGCTCATTGCCAATGGTGAT
rtACYL	F:TGCTCGATTATGCACTGGAAGT
	R:ATGAACCCCATACTCCTTCCCAG
rtALDOA	F:ATGCCCTACCAATATCCAGCA
	R:GCTCCCAGTGGACTCATCTG
rtCANT1	F:CTGGGTGTCCAACTACAACG
	R:ACTCCAGCAGGCAGACTCAT
rtCS	F:GGTGGCATGAGAGGCATGAA
	R:TAGCCTTGGGTAGCAGTTTCT
rtCYP11A1	F:GCAGTGTCTCGGGACTTCG
	R:GGCAAAGCGGAACAGGTCA
rtDHCR24	F:GCCGCTCTCGCTTATCTTCG
	R:GTCTTGCTACCCTGCTCCTT
rtENO1	F:GTGTGGCTCTAACCCTCTGG
	R:TCTGTGACGTTCAGTTTCTTGC
rtFASN	F:TTCTGGGACAACCTCATCGG
	R:CTCCGAAGAAGGAGGCATCA
rtFH	F:GGAGGTGTGACAGAACGCAT
	R:CATCTGCTGCCTTCATTATTGC
rtG6PD	F:ACAGAGTGAGCCCTTCTTCAA
	R:GGAGGCTGCATCATCGTACT
rtGDH	F:GGAGATGTCCTGGATCGCTG
	R:GTCCATGGATTCCCCCTTGG
rtGGT1	F:CTTCTACAACGGCAGCCTCA
	R:TCAGCTCAGCACGGTAGTTG
rtGLS	F:GACATGGAACAGCGGGACTAT
	R:TGTCCTTGGGGAAAGGGTTT
rtGLUT1	F:GATTGGCTCCTTCTCTGTGG
	R:TCAAAGGACTTGCCCAGTTT
rtGPI	F:CAAGGACCGCTTCAACCACTT
	R:CCAGGATGGGTGTGTTTGACC
rtGSR	F:ACGGCATGATAAGGGGATTCA
	R:AGTTTTCGGCCAGCAGCTAT
rtHK2	F:GTGACGCCAAAATCACGTCT
	R:TGGTCAACCTTCTGCACTTG
rtIDH1	F:AGAAGCATAATGTTGGCGTCA
	R:CGTATGGTGCCATTTGGTGATT
rtIDH2	F:GAAGGTGTGCGTGGAGAC
	R:CCGTGGTGTTCAGGAAGT
rtKGDH	F:TTGGCTGGAAAACCCCAAAAG
	R:TGTGCTTCTACCAGGGACTGT
rtLDHA	F:GGAGGACCCAGCAATTAGTCT
	R:GTTCACCCATCGCGGTTTAT
rtLSS	F:GCACTGGACGGGTGATTATGG
	R:TCTCTTCTCTGTATCCGGCTG
rtMDH1	F:TTTGGATCACAACCGAGCTAAAG
	R:ACATCTGGATACTGAGTCGAGG
rtNUDT9	F:AGGCACCAACTAAGAGCGAC

Table S1. Cont.

Primer name	Sequence
	R:AGGAGACGTCCGAGCCTTAT
rtPCGEM1	F:CAAGCATAACAATCTGCTCA
	R:GCACACAACGTAAATGAATG
rtPDHA1	F:GGGACGTCTGTTGAGAGAGC
	R:TGTGTCCATGGTAGCGGTAA
rtPDHB1	F:GGCCACAGTTTGGAGTAGGA
	R:AGCACCAGTGACACGAACAG
rtPFK	F:AGATGCGCACCAGCATCAACG
	R:GAACCCGGCACATTGTTGGA
rtPGAM2	F:AGAAGCACCCCTACTACAACTC
	R:TCTGGGGAACAATCTCCTCGT
rtPGD	F:GTACCCGTCACCCTCATTGG
	R:AGAGTGCCTTCCGAATGTCC
rtPGK1	F:GAACAAGGTTAAAGCCGAGCC
	R:GTGGCAGATTGACTCCTACCA
rtPGLS	F:GAGACAGGCATTCCAAGGGG
	R:GTCACTGATGGGAGCCACAA
rtPRNCR1	F:CCAGATTCCAAGGGCTGATA
	R:GATGTTTGGAGGCATCTGGT
rtSDHA	F:TGGGAACAAGAGGGCATCTG
	R:CCACCACTGCATCAAATTCATG
rtSUCLG1	F:GAGCAACGGCTTCTGTCATTT
	R:TGCTTGACTCGTACCATGTCC
rtTALDO1	F:TCGGTCTTGCTATGTCGAGC
	R:TGTACTCGTCGATGGCGTG
rtPDK4	F:GACCCAGTCACCAATCAAAATCT
	R:GGTTCATCAGCATCCGAGTAGA
rtPKM2	F:GAACTTCTCTCATGGAACTCAT
	R:GATCTCAGGTCCTTTAGTGTCTA
rtGAPDH	F:AATCCCATCACCATCTTCCAG
	R:CCTTCTCCATGGTGGTGAAGAC
rtMlycD	F:CCTCATGGTCAACTACCGCTACT
	R:CTTGGAGCCCAGGTAGGAGAT
Myc-ChIP-ACACA	F:GTGTGTGCCATTCTCCCTCA
	R:ACAGGACAGCCCCTATTTGC
Myc-ChIP-CANT1	F:TCTCTATTTGCCATGTTCCTGTC
	R:TGACAATTCCTCAGACCAGTG
Myc-ChIP-CYP11A1	F:CTTTTCTCCCTTTCCAGTCTCC
	R:GTGATTTGGGCACACTGAAAG
Myc-ChIP-DHCR24	F:CAGAGAAGGTCCTGACCACG
	R:CTGGATCCCCACGGGTAGTA
Myc-ChIP-ENO1	F:GAAGGGCCTGGCCTGAGT
	R:GGGAGTTGGGAGTGGAAGTTG
Myc-ChIP-FASN	F:AACCCAGACCCGAGACGGA
	R:TTTGTCCGCACCACACCAGG
Myc-ChIP-G6PD	F:GGATCTGCCCAAGGACACAAGGTGAC
	R:GCATCGTGCTCTACCATGAAACCCTC
Myc-ChIP-GDH	F:CTGACACGTGGGCAGGAAAG
	R:CCAGCCACGCATAATCCAAC
Myc-ChIP-GLS	F:GGATGAAGCTTGTCCGGGG
	R:ATTCGAGCTGTTGGGGAGAC
Myc-ChIP-GLUT1	F:GAAACTTGGAAAGCAACTCGG
	R:GGAGGTGATCGTTAGGACTTAAG
Myc-ChIP-GPI	F:TGTACCTTCTAGTCCCGCCA
	R:CTTGTTGGCATCGAAGAGGC
Myc-ChIP-GSR	F:TACTCGCCAAAACCACAGGA
	R:TGGTTCGCTTCTCTGCCTAC
Myc-ChIP-HK2	F:GAGCACGTGGAGAGAATCGT
	R:GAACCGCTCGTCTCCTACAC
Myc-ChIP-IDH1	F:AGGTGGTGGTCTTGGAAACAG
	R:AGCATTTTCTCCCCCAAACCA
Myc-ChIP-IDH2	F:CAAGCTGGAGAGCGAACGAG

Table S1. Cont.

Primer name	Sequence
	R:CCCGGCAAGGCCCAATG
Myc-ChIP-LDHA	F:TTACTTAGACTCCCAGCGCAC
	R:AGTGGAACAGCTATGCTGACG
Myc-ChIP-NUDT9	F:AACGCACGACCTTTGCTTTG
	R:AGCCCTCCCACGATAAGAGT
Myc-ChIP-PGLS	F:CTCGTCTCGATGCTAGCCC
	R:CCGTAGCTCTCACCCGGTA
Myc-ChIP-PGD	F:TCCTGCGTGAGTTGCTATGG
	R:CCTGTTAGACCATCCGAGGC
ChIP-actin-1F	F:TTCTACGTTTCCATCCAAGCCGT
	R:TTTCTTGTTCGAAGTCCAAGTCCAAGG
AR-ChIP-ENO1	F:AGATAGGACCGGTGAGCCGAACT
	R:AAAGTTGTCAGCAAGGTCGAGGG
AR-ChIP-LDHA	F:TTACTTAGACTCCCAGCGCAC
	R:AGTGGAACAGCTATGCTGACG
AR-ChIP-G6PD	F:GGATCTGCCCAAGGACACAAGGTGAC
	R:GCATCGTGCTCTACCATGAAACCCTC
AR-ChIP-HK2	F:GCACACTCAACTTGGCACTC
	R:CAGGAGATGGGCATTTGGGA
AR-ChIP-GSR	F:GAACCCAGGACCGCAAGTT
	R:ACACACGTGATGGGAGAGTC
AR-ChIP-GDH	F:CTGACACGTGGGCAGGAAAG
	R:CCAGCCACGCATAATCCAAC
AR-ChIP-GLS	F:GGATGAAGCTTGTCCGGGG
	R:ATTCGAGCTGTTGGGGAGAC

Table S2. Probe sequences used in the ChIRP and dot blot assay

Probe name	Sequence
PCGEM1-ChIRP1	CTGCAGTTCCAAAACTGGGT
PCGEM1-ChIRP2	AAGTTGCAGACACAGGTTAC
PCGEM1-ChIRP3	CCAGTGCATCTCGTAGTACA
PCGEM1-ChIRP4	AGTTCCACTTTGGCTGATAT
PCGEM1-ChIRP5	CTAGTCCTTTTCCAAAGGGT
PCGEM1-ChIRP6	CACTCTGAAATGCCTCATGA
PCGEM1-ChIRP7	CTCAGAAATCTCAGGGCTTG
PCGEM1-ChIRP8	GCTCTCTGCAAAGGTTGTAT
PCGEM1-ChIRP9	CCAATATTTGCAACAAGGGC
PCGEM1-ChIRP10	CCAGTTACACAGAAAATATG
PCGEM1-ChIRP11	AAGAATTTGTAGATGGGCCC
PCGEM1-ChIRP12	TTGAATTGATCAGCACAGCT
PCGEM1-ChIRP13	AAAAGACTTCAAGGCACCAA
PCGEM1-ChIRP14	GCCATAATTAGCATCATGTA
ChIRP lacZ-1	TAGCCAGCTTTCATCAACAT
ChIRP lacZ-2	AGCAGCAGACCATTTTCAAT
ChIRP lacZ-3	GTGTGGGCCATAATTCAATT
ChIRP lacZ-4	CGGCAGCCGTTATTATTATT
ChIRP lacZ-5	GAAACTGTTACCCGTAGGTA
ChIRP lacZ-6	CACGGCGTTAAAGTTGTTCT
ChIRP lacZ-7	GGATCGACAGATTTGATCCA
ChIRP lacZ-8	GTAGTTCAGGCAGTTCAATC
ChIRP lacZ-9	CAACGGTAATCGCCATTTGA
ChIRP lacZ-10	TGCAAGGCGATTAAGTTGGG
PCGEM1-binding probe-1	TTTTCATTCAATTTATGACTTTTAAAACTGCAGTTCCAAAACTGGGTGCCAGAGTGCCTT
PCGEM1-binding probe-2	AAAAAGATGCTGTCCAGGGACCTATAAATAGCTCTTTAAAAACCTCCACCTTTGCTATCA
PCGEM1-binding probe-3	ATTAGAGGAAGTTGCAGACACAGGTTACGGCATAGGGCAAAAAGGTTGCTGCCTAATTGA
PCGEM1-binding probe-4	AGTGCATCTCGTAGTACAATTTTATCATTCAGCTCTATGAATCTGCTTAACTATTTCCCA
PCGEM1-binding probe-5	GATATCATGAAGTGTCAAATGCACCAAGCCTGCTCACTTGATAAGGTCACGTTGAGTCCC
PCGEM1-binding probe-6	CTCCCAACCTGATGATGTCATAGTCCTCTTCCAGGAGCTGTTTTTAGTTCCACTTTGGCT
PCGEM1-binding probe-7	GAAGACTAATAGATTTCACACTTTCTAGTCCTTTTCCAAAGGGTCCGCTGTCCCTGGAGA
PCGEM1-binding probe-8	GGAGTAGGCCTGTGTCTTGTAAATATGAAATGCTTTTACAGAGACAGAGAATTTCATATC
PCGEM1-binding probe-9	CTGAAATGCCTCATGATCTCTTTTCCCTCTGCTTGCCTGTTGCCACTTTTTGCTGCCCTA
PCGEM1-binding probe-10	GCTTGGCCAAAATAAAACCAAACATACGGCATTGAGAAATATATGAAAAGACAGTGCACT
PCGEM1-binding probe-11	TAACATTGGCAAAGGCACCTTTGTTTTCTCCAGATTTTTTTTTCTTGAGCAGATTGTTAT
PCGEM1-binding probe-12	GACCTGATCCCATTTATGTGAATTCCCCTCAGAAATCTCAGGGCTTGTCAAAAAGAAACA
PCGEM1-binding probe-13	AAGCATGCTCTCTGCAAAGGTTGTATCTTTAAATCATATTTGCACACAACGTAAATGAAT
PCGEM1-binding probe-14	TTGATTAACTGATCTTGAAGAATGCTTTACCCTTAGTCCTCCACGTGCCTACCCTTAGGA
PCGEM1-binding probe-15	TTTAATCAATATAACCAATATTTGCAACAAGGGCATTTCAGAATGCAAAGAGCACCTTTC
PCGEM1-binding probe-16	TTTTTTGCTTTTGTGGGTTTGTTCATCTGTAAGTTAAAGGTTGTTTCCATTAAGTGTAAA
PCGEM1-binding probe-17	AAGCAGGAATCCTTTAATCCAGTTACACAGAAAATATGAAATCATAGGTAGGGCTTTTGA
PCGEM1-binding probe-18	GATGGGCCCTCATTTTAAACAATACCTGGAAATATTCCATTATCATTTATGCCCAAAAGC
PCGEM1-binding probe-19	ACAATTAGACAATGTCCAGCTGAATTTTAGAATTATCCAAAGTATTGCTAAGAATTTGTA
PCGEM1-binding probe-20	GCTAATTAACTAAATTCACATACTTAAAATTTGAAATTCTAGCAAAGATGTATATAAAAA
PCGEM1-binding probe-21	AATTTATGACCTTCATAGTCTAAAATTTAGGAAAGTAATGTTTTTGAATTGATCAGCACA
PCGEM1-binding probe-22	TAAGATGAAGACATTATAATGAAAAACAATCTATAATTGTATGTGTAGATATATTTGTTG
PCGEM1-binding probe-23	TCAAGTAATTATGAATTAAAACTCTCAGTTTTCTAAAAACAATCACAAAGACAATTCTGT
PCGEM1-binding probe-24	CTTATGTACATAAAAACAATCACATACAATTAATGCTGGATTGTTCCCACAATTTTTTGA
PCGEM1-binding probe-25	GTAGTAATTTTAAACATGGGACTAAGTACAAAAGACTTCAAGGCACCAAGCTTAAGACTC
PCGEM1-binding probe-26	AATTGCCATAATTAGCATCATGTAAATTGAATTGAAAAACATAAATGCTTTAGATATAAA
PCGEM1-binding probe-27	TTTTTTTTTTTTTTTCTATTTCGAAATCTTTAATATTTGTTAT