



Spindle pole body component 25 in the androgen-induced regression of castration-resistant prostate cancer

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Background: Androgen plays a critical role in the development and growth of prostate cancer (PCa) by binding to the androgen receptor, a steroid receptor for testosterone and dihydrotestosterone (DHT). Androgen deprivation therapy, a clinical endocrine therapy, has resulted in increases in the occurrence of castration-resistant prostate cancer (CRPC); however, the mechanisms of CRPC have not yet fully been determined. We previously showed that spindle pole body component 25 (SPC25), a component of the NDC80 complex that is critical in kinetochore formation and chromosome segregation during the cell cycle, plays a critical role in PCa tumorigenesis and cancer stemness. However, it is not yet known whether SPC25 plays a role in CRPC; thus, we sought to address this question in the current study.

Methods: SPC25 levels were detected in androgen-insensitive PCa cells using the public database and bioinformatics tools. In vitro, SPC25 levels were determined in androgen-sensitive and androgen-insensitive PCa cells treated with or without DHT. The growth of the PCa cells was assessed by the Cell Counting Kit-8 assay. The invasiveness and migratory potential of the PCa cells were assessed by the transwell cell invasive assay and migratory assay, respectively. Gain-of-function and loss-of-function experiments examined the transfection of androgen-sensitive and androgen-insensitive PCa cells by plasmids carrying small-interfering ribonucleic acids for SPC25 or SPC25, respectively.

Results: SPC25 levels were significantly reduced in the androgen-insensitive PCa cells treated with DHT in the Public database. In vitro, PCa cell growth, invasion, and metastasis was reduced in androgen-insensitive PCa cells but increased in androgen-sensitive PCa cells treated with DHT, partially through DHT-regulated expression of SPC25 at transcriptional but not at translational levels.

Conclusions: Androgen treatment reduces CRPC growth, invasion, and metastasis partially through its regulation of SPC25. SPC25 represents a promising target in the treatment of CRPC.

Keywords: Prostate cancer (PCa); androgen receptor (AR); androgen deprivation therapy (ADT); castration-resistant prostate cancer (CRPC); spindle pole body component 25 (SPC25)

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Introduction

Prostate cancer (PCa) occurs commonly in elderly men and is the 2nd most lethal malignant cancer in males (1). Androgen plays a critical role in the development and growth of PCa by binding to the androgen receptor (AR),

a steroid receptor for testosterone and dihydrotestosterone (DHT), the latter of which is the catalyzed product of testosterone by the enzyme 5 α -reductase and is a much more potent agonist of the AR than testosterone (2).

Androgen deprivation therapy (ADT), a clinical

endocrine therapy that includes castration therapy by either surgery or medicine, androgen antagonist therapy or combined therapy, has been applied in patients with local tumor expansion, tumor metastasis, and tumor relapse (3). However, the use of castration therapy has resulted in increases in the occurrence of castration-resistant prostate cancer (CRPC) (4). The exact mechanisms of CRPC remain unclear; however, research has shown that under ADT, some PCas alter their ARs to survive the castration therapy (5). Alterations in ARs include point mutations, the overexpression of ARs, increases in androgen biosynthesis, the generation of alternative constant AR splice variants without the need for ligand binding, and even the activation of alternative pathways (6). These findings have led to the development of new-generation therapies for CRPC, and some promising new AR-targeted medicines, such as Cabazitaxel, Sipuleucel-T, Abiraterone, and Enzalutamide (7), have been developed. However, it should be noted that these new medicines could quickly induce new resistant cancer types. Thus, the molecular mechanisms underlying the development of CRPC need to be urgently elucidated. Conversely, androgen treatment has been shown to reduce cancer growth and metastasis in some CRPC (8), but the mechanisms by which this occur remain unclear.

We previously showed that spindle pole body component 25 (SPC25), a component of the NDC80 complex that is critical in kinetochore formation and chromosome segregation during the cell cycle (9), plays a critical role in PCa tumorigenesis (10) and cancer stemness (11). However, it is not yet known whether SPC25 plays a role in CRPC; thus, this study sought to address this question. We present the following article in accordance with the MDAR reporting checklist (available at <https://tau.amegroups.com/article/view/10.21037/tau-22-214/rc>).

Methods

PCa cell lines and transduction

Human PCa cell lines (LNCap and PC346) were obtained from the American Type Culture Collection (ATCC, Rockville, MD, USA), maintained in Dulbecco's Modified Eagle Medium (DMEM) (Invitrogen, Rockville, MD, USA) with 8% fetal bovine serum (FBS; Sigma-Aldrich, Rockville, MD, USA) in a humidified incubator at 37 °C with a 5% carbon dioxide (CO₂). The PC346 cells express high levels of normal ARs and are very sensitive to androgen treatment. The LNCaP cells have abnormal ARs with broad steroid

binding specificity and represent CRPC.

Plasmids and transfection

The complete coding sequence for SPC25 was amplified and obtained through polymerase chain reaction (PCR) using human PCa cell line PC346 as a template. The sequence for the small-interfering ribonucleic acid (siRNA) targeting SPC25 was 3'-CCGGCCATCAAAGCATTTG CAGAAACTCGAGTTTCTGCAAATGCTTTGATGG TTTTTG-5', and the scrambled sequence for the siRNA of SPC25 was 3'-CCGATGCCTTCCAGAAAGATGATG CACAGACAGAATGTT-5', which was used as a negative control. A pCDNA3 plasmid (GenePharma, Shanghai, China) was used as a backbone plasmid that also carries a green-fluorescent reporter (GFP) co-expressed with the transgene. The transfection of the cells was performed with a Lipofectamine 2000 transfection kit (Invitrogen). The transfected cells were determined by the direct fluorescence of GFP. Nearly 90% transfection efficiency was reached.

Immunocytochemistry

The immunocytochemistry of SPC25 was performed with a rabbit polyclonal anti-SPC25 antibody (Abcam, ab121295, Waltham, MA, USA). We used 4',6-diamidino-2-phenylindole (DAPI) to stain the nuclei.

RNA extraction and quantitative real-time polymerase chain reaction (qRT-PCR)

Total RNA was extracted with a RNeasy mini-kit (Qiagen, Shanghai, China). Complementary deoxyribonucleic acid (DNA) was generated using a reverse transcription kit (Qiagen). The qRT-PCR was applied with the commercial SYBR Green PCR Kit (Qiagen, Shanghai, China), and the commercial primers were all purchased from Qiagen. Duplicated reactions were conducted for the qRT-PCR to reduce variation. The 2^{-ΔΔCt} method was used for quantification. The relative levels of gene expression were quantified and normalized sequentially with β-actin and the experimental controls.

ELISA

A ristocetin-induced platelet aggregation buffer (Sigma-Aldrich) was used to extract the protein from the cultured cells, after which the protein concentration was determined

with a bicinchoninic acid protein assay (Sigma-Aldrich). An enzyme-linked immunoassay (ELISA) was performed using a human-specific SPC25 ELISA kit (MyBiosource, MBS9328421, San Diego, CA, USA) as described previously (10).

Cell proliferation assay

Cell proliferation was assessed using cell counting kit-8 (CCK-8, Sigma-Aldrich), and the quantification was performed based on duplicates.

Cell invasion and migration assay

For the cell invasion assay, the upper chamber of a 24-well plate (Millipore, Bedford, MA, USA) pre-coated with Matrigel (Sigma-Aldrich) was seeded with cells, and the lower chamber was filled with DMEM with 8% FBS. After 24 hours of culturing, the invasive cells were stained with 0.1% crystal violet (Sigma-Aldrich) for 15 minutes before quantification. For the cell migration assay, the upper chamber of a 24-well transwell insert (Millipore) was seeded with cells suspended in serum-free medium, and the lower chamber was filled with DMEM with 8% FBS. After 36 hours, the cells that had migrated from the upper chamber to the lower surface were fixed with methanol, stained with 0.1% crystal violet (Sigma-Aldrich), and quantified.

Statistical analysis

No criteria were used to exclude any experimental units during this experiment, and no data were excluded from the analysis. The data were statistically analyzed with GraphPad Prism 7 (GraphPad, Chicago, IL, USA) using a 1-way analysis of variance with a Bonferroni correction, followed by a Fisher's exact test. The values are expressed as the mean \pm standard deviation (SD). A P value <0.05 was considered statistically significant. For the bioinformatics analyses, transcriptome RNA-sequencing (RNA-seq) data on human LNCap cells were obtained from the Gene Expression Omnibus (GEO) data portal (No. GSE187413; <https://www.ncbi.nlm.nih.gov/geo/>). The RNA-seq data of 3 specimens in vehicle-treated (control) or DHT-treated groups were used for the analysis using the R software Linear Models for Microarray and RNA-Seq Data (Limma) package. Cuffdiff was applied to assess pairwise differential expression and determine gene expression levels. The transcription data were then analyzed with a setting of \log_2 |fold change| >1

and a false discovery rate <0.05 as the cutoff values.

Results

The public database shows that DHT treatment inhibits SPC25 in LNCap cells

A study of CRPC showed that androgen treatment reduced cancer growth and metastasis in some CRPCs (8), but the mechanisms by which this occurs remain unclear. As SPC25 plays a critical role in PCa growth, we explored the GEO database and performed bioinformatics analyses to examine whether the values of SPC25 altered during androgen treatment in androgen-insensitive PCa cells. After the careful selection of related databases, we chose GSE187413 for the analysis. The database provided data of LNCap cells treated with a control vehicle or DHT. We conducted a pathway enrichment analysis and examined the protein-protein interaction network using Metascape, and found that many pathways related to cell-cycle control were significantly affected by DHT, which showed the importance of androgen treatment in the cell growth of CRPC (Figure 1A). Notably, we found that SPC25 mRNA level was significantly downregulated by DHT treatment in LNCap cells (see the volcano map in Figure 1B and the heat map in Figure 1C). Together, these bioinformatic analyses of data from the public database suggest that DHT treatment inhibits SPC25 in LNCap cells, which may contribute to the reduced growth of CRPC by androgen.

DHT treatment transcriptionally inhibits SPC25 in LNCap cells

Next, we used DHT to treat 2 different PCa cell lines. PC346 cells express high levels of normal ARs and are very sensitive to androgen treatment; thus, we used these as controls. LNCaP cells have abnormal ARs with broad steroid binding specificity and represent CRPC. Thus, we used LNCaP cells to examine SPC25 expression in response to androgen. The qRT-PCR and ELISA analyses showed that compared to the control dimethyl sulfoxide (DMSO)-treated PC346 cells, DHT increased both SPC25 messenger RNA (mRNA) (Figure 2A) and protein (Figure 2B) by about 5-fold, respectively. Additionally, the upregulation of SPC25 in PC346 cells by DHT was confirmed by an immunocytochemistry analysis (Figure 2C). Our findings are consistent with the general understanding that androgen increases AR-positive PCa cell growth, and

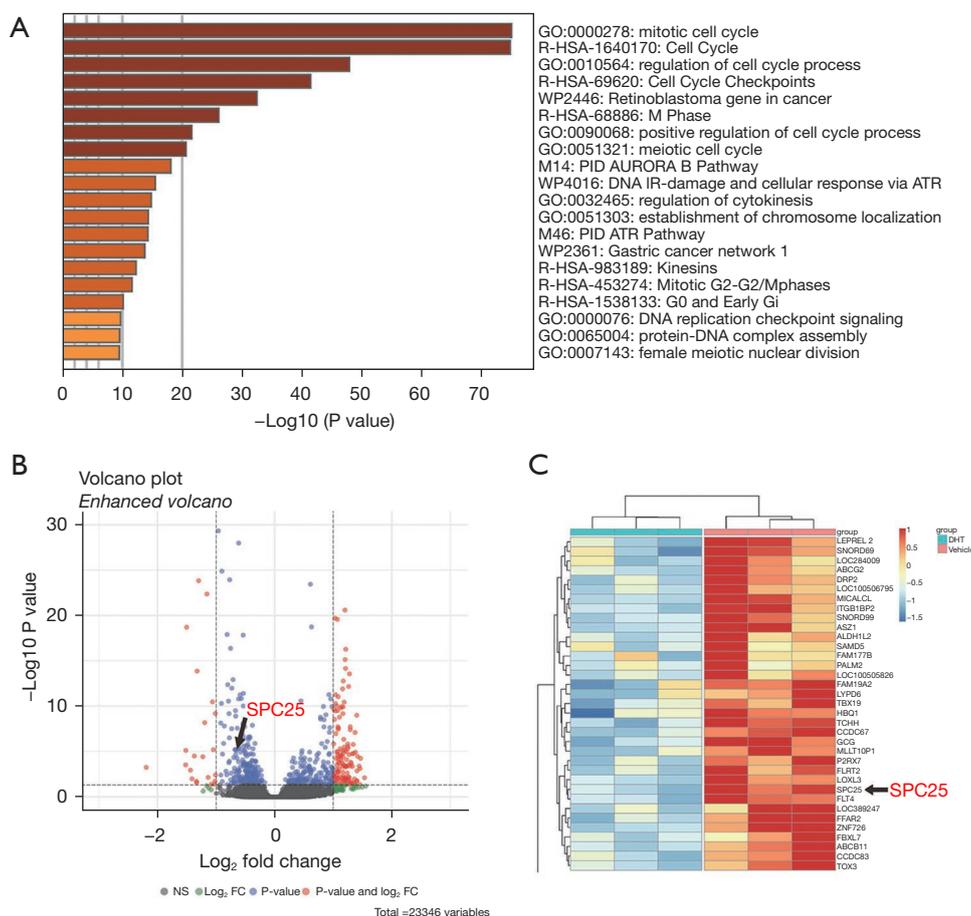


Figure 1 The public database shows that DHT treatment inhibits SPC25 in prostate cancer LNCap cells. The GSE187413 data set comprised LNCap cells treated with a control vehicle or DHT. (A) A pathway enrichment analysis was performed using Metascape, and showed that many of the pathways related to cell-cycle control were significantly affected by DHT. (B,C) The significantly altered genes are shown in the volcano map (B) and the heat map (C). SPC25 was significantly reduced by DHT treatment. DHT, dihydrotestosterone; SPC25, spindle pole body component 25.

SPC25 is activated during and is important for cell growth. However, the qRT-PCR and ELISA analyses also showed that compared to the control DMSO-treated LNCap cells, DHT decreased both SPC25 mRNA (*Figure 2A*) and protein (*Figure 2B*) by about 80%, respectively. Additionally, the downregulation of SPC25 in LNCap cells by DHT was confirmed by an immunocytochemistry analysis (*Figure 2C*). Our findings are consistent with other recent findings that androgen treatment inhibits cancer growth and metastasis in CRPC (8) and our analysis of the public database (*Figure 1*). As the altered degree of the SPC25 mRNA was comparable to that of the SPC25 protein, it appears that DHT treatment inhibits SPC25 in LNCap cells transcriptionally rather than translationally.

DHT treatment inhibits LNCap cell growth, invasion, and migration

Next, we examined the effects of androgen treatment on LNCap cell growth, invasion, and migration. The CCK-8 assay showed that DHT significantly increased viable PC346 cells (*Figure 3A*), but significantly decreased viable LNCap cells (*Figure 3B*). Additionally, DHT significantly increased the invasiveness of PC346 cells, but significantly decreased the invasiveness of LNCap cells in a transwell cell invasion assay (*Figure 3C, 3D*). Further, the transwell cell migration assay showed that DHT significantly increased the migratory potential of PC346 cells, but significantly decreased the migratory potential of LNCap cells (*Figure 3E, 3F*). Together, these data suggest that DHT

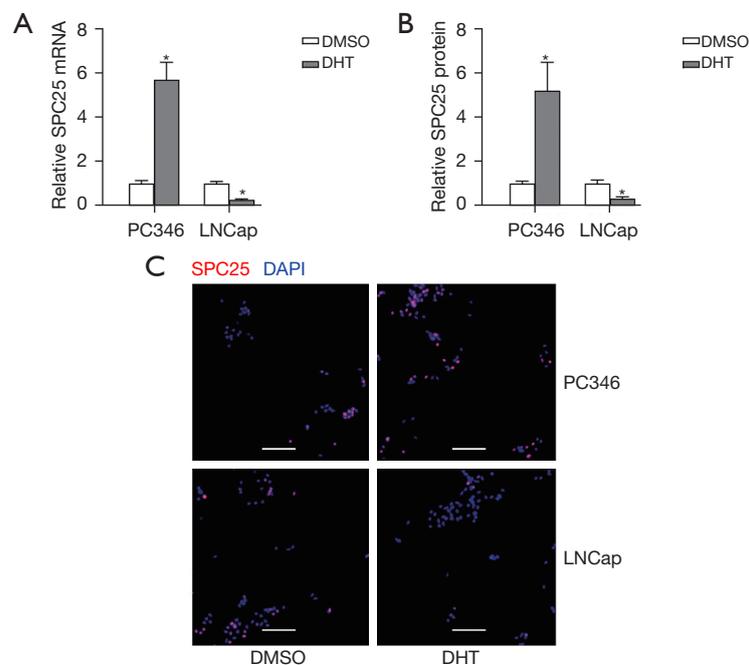


Figure 2 DHT treatment transcriptionally inhibits SPC25 in LNCap cells. Next, 2 different prostate cancer cell lines were treated with DHT. The PC346 cells were used as the controls, as they express high levels of normal ARs and are very sensitive to androgen treatment. The LNCap cells were used to examine SPC25 expression in response to androgen, as they have an abnormal AR with broad steroid binding specificity and represent castration-resistant prostate cancer. (A) qRT-PCR results for SPC25; (B) ELISA results for SPC25; (C) immunocytochemistry results for SPC25 (red) and DAPI (blue). *, $P < 0.05$. $N = 5$. Scale bar: 100 μm . DHT, dihydrotestosterone; SPC25, spindle pole body component 25; AR, androgen receptor; ELISA, enzyme-linked immunosorbent assay; DAPI, diamidino-2-phenylindole; DMSO, dimethyl sulfoxide.

treatment inhibits LNCap cell growth, invasion, and migration.

Preparation of SPC25-modified LNCap cells to evaluate the roles of SPC25 in relation to the DHT effects

As DHT treatment not only decreases SPC25 levels but also inhibits cell growth, invasion, and migration in LNCap cells, we also sought to determine the alteration of SPC25 that contributes to the effects of DHT in relation to cell growth, invasion, and migration in LNCap cells. Thus, siRNAs were used to prepare overexpressed and depleted SPC25 plasmids. As PC346 cells increased cell growth, invasion, and migration in response to DHT, they were transfected with siRNA for SPC25 (si-SPC25) to knockdown SPC25, and then examined to determine the effects on growth, invasion, and migration. Additionally, as the LNCap cells decreased cell growth, invasion, and

migration in response to DHT, they were transfected with SPC25 plasmid to overexpress SPC25, and the effects on growth, invasion, and migration were then examined.

The qRT-PCR results showed that si-SPC25 decreased SPC25 mRNA in PC346 cells by about 85%, while SPC25 plasmid increased SPC25 mRNA in LNCap cells by about 7-fold (Figure 4A). Additionally, the SPC25 mRNA was decreased in the si-SPC25-transfected PC346 cells treated with DHT to a level similar to that of the Scr-transfected PC346 cells treated with DMSO (Figure 4B), while the SPC25 mRNA was increased in the SPC25-transfected PC346 cells treated with DHT to a level similar to that of the Scr-transfected LNCap cells treated with DMSO (Figure 4C). As for this analysis, the SPC25 levels in the si-SPC25-treated PC346 cells exposed to DHT should have decreased to levels similar to those of the control PC346 cells (Scr-treated) exposed to control DMSO to be qualified as a loss-of-function experiment to examine the effects on

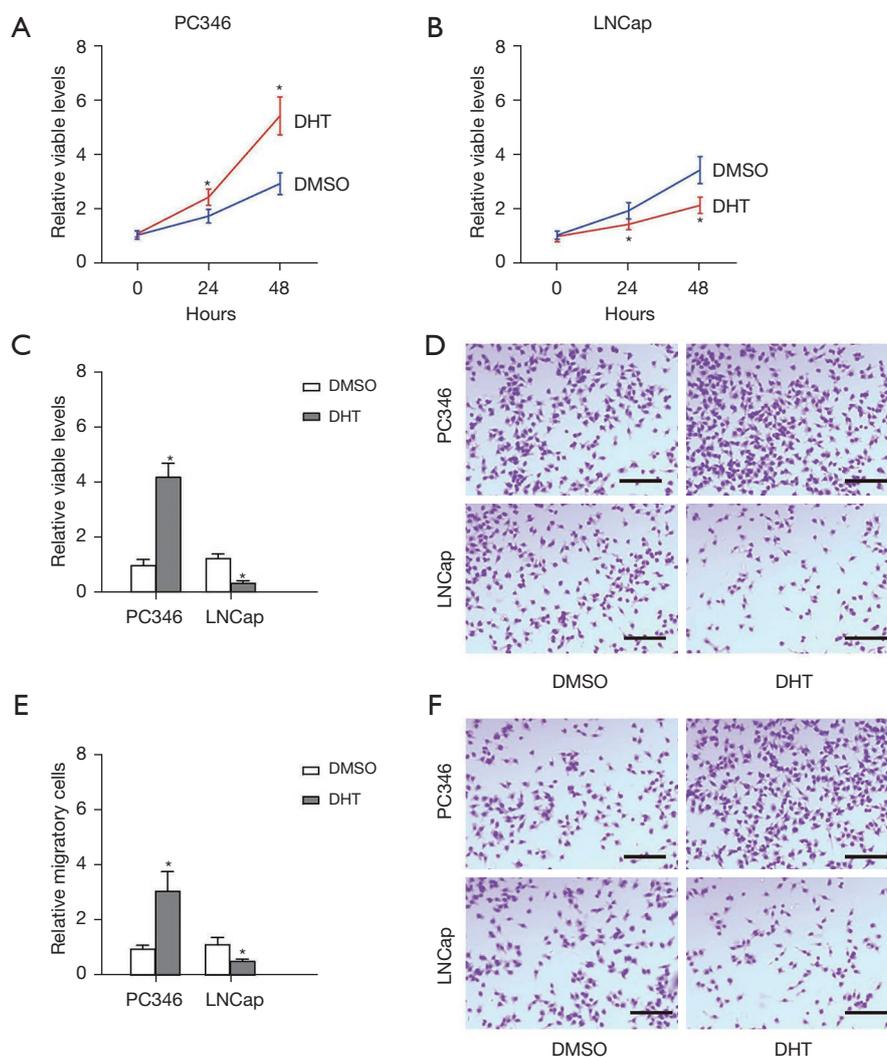


Figure 3 DHT treatment inhibits LNCap cell growth, invasion, and migration. The effects of androgen treatment on LNCap cell growth, invasion, and migration were assessed. (A,B) CCK-8 assay results for PC346 cells (A) and LNCap cells (B). (C,D) Transwell cell invasion assay results shown by quantification (C) and representative images stained with 0.1% crystal violet (D). (E,F) Transwell cell migration assay results shown by quantification (E) and representative images stained with 0.1% crystal violet (F). *, $P < 0.05$. $N = 5$. Scale bar: 100 μm . DHT, dihydrotestosterone; DMSO, dimethyl sulfoxide.

cell growth, invasion, and migration.

DHT treatment inhibits LNCap cell growth, invasion, and migration through SPC25

Finally, we examined whether SPC25 is important to the inhibition of LNCap cell growth, invasion, and migration by DHT. First, we found that SPC25 depletion significantly attenuated the promoting effects of DHT on cell growth in the PC346 cells (Figure 5A), and SPC25 re-expression

significantly attenuated the suppressive effects of DHT on cell growth in the LNCap cells (Figure 5B). Additionally, SPC25 depletion significantly attenuated the promoting effects of DHT on cell invasion in the PC346 cells (Figure 5C,5D), and SPC25 re-expression significantly attenuated the suppressive effects of DHT on cell invasion in the LNCap cells (Figure 5C,5D). Further, SPC25 depletion significantly attenuated the promoting effects of DHT on cell migration in the PC346 cells (Figure 5E,5F), and SPC25 re-expression significantly attenuated the

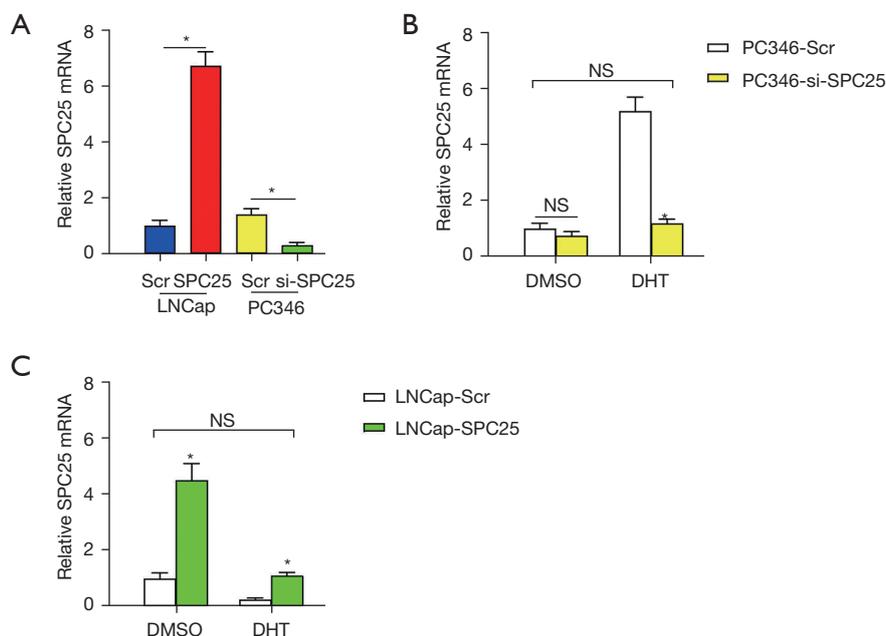


Figure 4 Preparation of SPC25-modified LNCap cells to evaluate the roles of SPC25 in DHT effects. The PC346 cells were transfected with siRNA for SPC25 (si-SPC25) to knockdown SPC25, and the LNCap cells were transfected with SPC25 plasmid to overexpress SPC25. (A) qRT-PCR results for SPC25 in normal cells. (B,C) qRT-PCR results for SPC25 in PC346 cells (B) or LNCap cells (C) treated with DMSO or DHT. *, $P < 0.05$. $N = 5$. NS, non-significant; DMSO, dimethyl sulfoxide; DHT, dihydrotestosterone; SPC25, spindle pole body component 25.

suppressive effects of DHT on cell migration in the LNCap cells (Figure 5E, 5F). Together, these data suggest that DHT treatment inhibits LNCap cell growth, invasion, and migration at least partially through SPC25.

Discussion

The AR is the most important regulator of normal and abnormal prostate growth and maintenance. In response to androgen, AR directly or indirectly activates a number of genes associated with cell-cycle regulation and metabolism in the prostate system (3). Given the specific function of the AR in prostate and PCa, it is not surprising that the AR acts as a primary oncogenic driver of PCa and is a major therapeutic target (5). The suppression of AR activity by inhibitors in the AR signaling pathway is effective in almost all patients at the beginning, but PCa patients have begun to develop resistance to this treatment, which can result in PCa progressing to CRPC (5). Different mechanisms (e.g., AR mutation, amplification of the AR, alternative splicing, and altered AR functionality) have been shown to be responsible for this cancer growth and metastasis resistant

to AR signaling inhibitors (6); however, the final effective molecules must be regulators of the cell cycle. Thus, our findings that SPC25 appears to be a critical in cellular growth were not at all surprising.

We showed that SPC25 was upregulated in AR-normal PCa cells in response to androgen, but downregulated in AR-insensitive PCa cells. This is consistent with a previous report that showed that androgen treatment reduces cancer growth and metastasis in some CRPCs (8). Notably, in addition to the regulation of SPC25 observed in this study, another study suggested that the crosstalk between ARs and 6-phosphogluconate dehydrogenase may play a role in PCa growth (12). Further, the androgen-induced suppression of CRPC growth could result from the transactivation of abnormal ARs in CRPC cells, which in turn could lead to the activation of some alternative pathways that drive the cell-cycle arrest (13). It is highly possible that SPC25 is a downstream factor of these altered pathways, as the correction of its level attenuated the effects of androgen induction in the LNCap cells.

To the best of our knowledge, this is the first study to describe the role of SPC25 in the molecular mechanisms

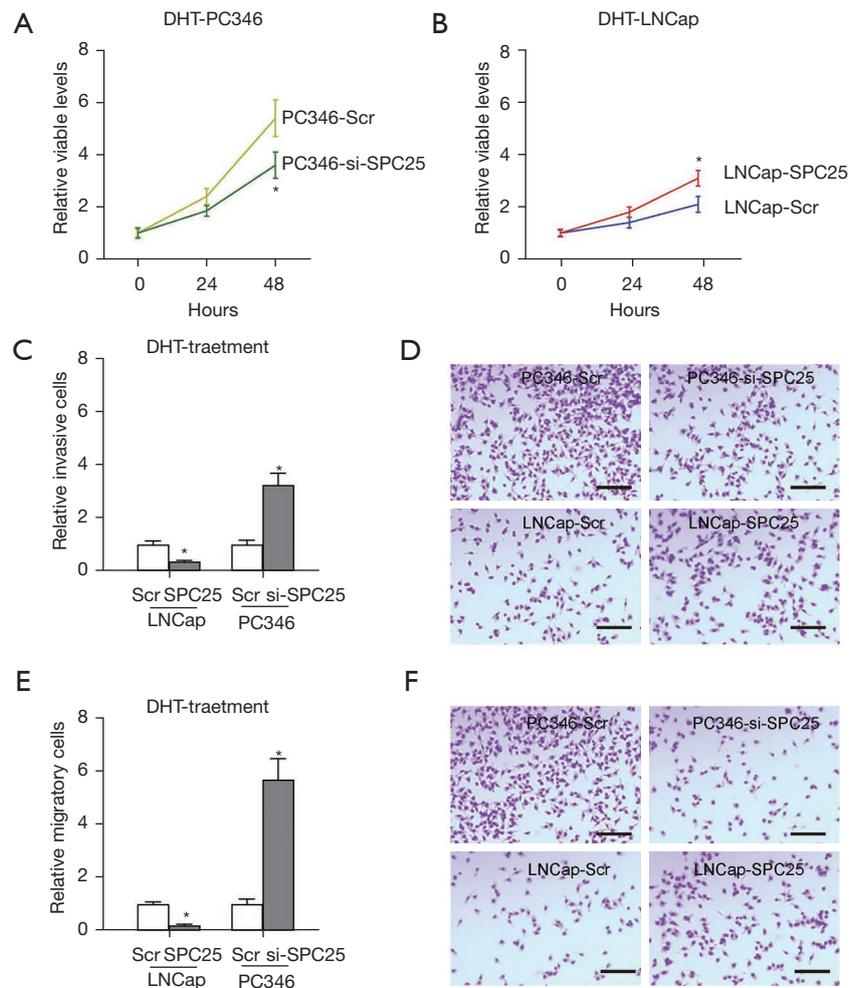


Figure 5 DHT treatment inhibits LNCap cell growth, invasion, and migration through SPC25. We examined whether SPC25 is important in the inhibition of LNCap cell growth, invasion, and migration by DHT. (A,B) Viability assay results for PC346 cells (A) and LNCap cells (B). (C,D) Transwell cell invasion assay results shown by quantification (C) and representative images stained with 0.1% crystal violet (D). (E,F) Transwell cell migration assay results shown by quantification (E) and representative images stained with 0.1% crystal violet (F). *, $P < 0.05$. $N = 5$. Scale bar: 100 μm . DHT, dihydrotestosterone; SPC25, spindle pole body component 25.

underlying castration resistance and androgen-induced cancer growth arrest in androgen-insensitive PCa; however, further research needs to be conducted using human primary CRPC. Collectively, our results provide evidence that SPC25 represents an interesting target for CRPC therapy.

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Footnote

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Data Sharing Statement: Available at <https://tau.amegroups.com/article/view/10.21037/tau-22-214/dss>

Conflicts of Interest: All authors have completed the ICMJE uniform disclosure form (available at <https://tau.amegroups.com/article/view/10.21037/tau-22-214/coif>). The authors

have no conflicts of interest to declare.

Ethical Statement: The authors are accountable for all aspects of the work in ensuring that questions related to the accuracy or integrity of any part of the work are appropriately investigated and resolved.

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