Letter to Editor
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Prostate Cancer and PSA Test

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Letter to Editor

Prostate Cancer (PCa) is the second most diagnosed cancer after lung cancer in men worldwide from 2018 to 2018. Race, age and positive family history of PCa are the three confirmed risk factors for PCa. The incidence of PCA is increasing in Asia but is still significantly lower than in Western countries. The incidence of PCa increases sharply with age. The risk of PCa increases sharply after age 55 and peaks at age 70-74 and then decreases slightly. Recent studies have shown that the incidence of PCa has increased in older adolescents and young men. This may be due to the process of obesity, physical inactivity, HPV infection, exposure to carcinogens and substances in the environment. Standard PCa treatments include surgical removal, radiation therapy, and hormone therapy, and in recent years, immunotherapy has become increasingly popular as an alternative to androgen deprivation (ADT), using surgical or chemical castration that blocks testosterone synthesis [1]. Blocks or blocks the androgen receptor, a standard treatment used in all stages of recurrent PCA. The management of metastatic hormone-sensitive prostate cancer (mHSPC) has changed dramatically in the last few years.

The data supported the survival benefit by adding four different factors (dutaxel, abiraturon acetate, enzalotamide, and apalotamide) to androgen deprivation in men with mHSPC, both chemotherapy and androgen receptor signaling inhibitors (ARSi) when compared with ADT. Combined with ADT alone for patients with mHSPC, they show a significant survival. Almost all patients eventually progress to refractory prostate cancer (CRPC) after about 18 months of ADT. The mechanism of CRPC remains unclear. Chromosomal rearrangement and the number/achievements of copy number and gene change in the androgen axis and kinase-dependent signal pathways are thought to be important mechanisms for the emergence and development of CRPC. They may be involved in the development of CRPC. Prostate cancer stem cells (PCSCs) are a small population of PCa cells that exhibit unpleasant regenerative capacity and indefinite tumor initiation capacity. Studies have shown that ADT leads to a time-dependent increase in PCSC population and PCSC is associated with high risk of disease, metastasis, biochemical failure, and po survival without progression PCSCs can survive chemotherapy or radiotherapy and are offered responsibility for developing CRPCs [2]. Expression of prostate-specific antigen (PSA), as a marker of differentiation, in PCa is positively correlated with its overall degree of differentiation PCa contains differentiated (PSA+) and undifferentiated tumor cells (PSA–/ lo). PSA–/ lo cells have been reported to preferentially express stem cell genes, can initiate tumor growth, have long-term tumor proliferative capacity, and are highly tumorigenic castor-resistant PCA cells that can utilize ALDH + CD44 to be enriched +β2β1+phenotype. Conversely, PSA+PCa cells have a more limited tumor proliferative capacity, are symmetrically divided, and are susceptible to castration. Thus, PSA–/ lo cells may be a vital source of CRPC cells. New treatment strategies targeting PSA/lo cells may overcome and prevent recurrence of PCa. Peptide therapy is a promising and new approach to treat many diseases, including cancer [3]. It has several unique advantages over proteins or antibodies, including ease of synthesis, high target specificity, and low selectivity and toxicity in natural tissues. Phage display is a powerful technology for screening and isolating specific peptides.

Despite significant advances in CRPC therapeutic development, CRPC remains a deadly malignancy. Although the origin of CRPC cells is still controversial, several studies clearly show the presence of PCSCs in CRPCs. Most treatment regimens only target proliferative tumor cells. Thus, targeting the PCSC to reduce the spread of the CSC population may lead to new strategies for overcoming chemical resistance [4]. Peptide therapy has received a great deal of attention due to its benefits such as low molecular weight, ability to specifically target tumor cells, and low toxicity in normal tissues. Peptide-based therapies have shown promising results in *in vitro* and *in vivo* studies. For example, a bifunctional peptide designed by binding a cancer-detecting peptide to a toxic peptide demonstrates toxicity to breast cancer, PCa, and neuroblastoma cells. Han et al. They have produced PCa for targeting and imaging. Wada et al. have identified peptides that specifically target xenografted PCa cells and suppress cell growth. In this study, using phage display library screening, we identified a TAP1 peptide that specifically targets PSA–/lo PCa cells and inhibits cell growth both *in vitro* and *in vivo*. In addition, our findings suggest that this selective targeting peptide may be a very promising factor that prevents adverse effects on non-target cells and can be used alone or in combination with standard therapies. Maintaining telomere length is critical for the regeneration and unlimited proliferation of stem cells.

Pluripotent stem cells need to maintain telomere length and homeostasis to maintain their ability to regenerate and multiply. We found that ALDH+ CD44+ CXCR4+ CD24+ cells showed shorter telomeres after TAP1 treatment. These findings suggest that TAP1 may inhibit telomere proliferation, disrupt telomere, inhibit PCSC multiplication, and ultimately delay cell proliferation and tumor initiation. Studies show that CSC renewal causes tumor recurrence, and therapeutic differentiation may be a strategy for sensitizing CSCs to chemotherapy. TAP1 may also increase the sensitivity of ALDH+ CD44+ CXCR4+ CD24+ cells to chemotherapy agents by inducing CSC differentiation. HOXB9 is highly expressed in different types of cancer and causes PCa invasion and metastasis [5]. TGF-roles signaling has different roles in regulating tumor proliferation. TGF-β1 suppresses cell proliferation in early-stage lesions, while promoting late-stage tumor progression. High levels of TGF-1 were associated with a poor prognosis in patients with PCa. Targeting the mechanism associated with TGF-could2 could provide new opportunities to prevent lethal PCa metastasis. Aberrant TGF-can signaling can direct CRPC. In this study, we observed that the expression of HOXB9 and TGF-2, but not TGF-1, was significantly decreased in ALDH+ CD44+ CXCR4+ CD24+ cells after TAP1 treatment. This may be due to the presence of HOX binding sites in the TGF-β2 promoter regions but not in the TGF-40140 [6,7].

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