

FACTORS IN THE FORMATION OF PROSTATE CANCER

Умуров Бобиржон Файзиллоевич

Бухарский государственный медицинский институт

Abstract

Prostate cancer is one of the most common forms of malignant tumors among men, and its development is associated with a variety of factors, including genetic, hormonal, environmental, and lifestyle-associated risks. The article discusses the main factors contributing to the occurrence of prostate cancer, such as age, family history, ethnicity, as well as the influence of diet and environment hormones and gene mutations associated with tumor progression. In addition, modern approaches to prevention and diagnosis that can help reduce morbidity and improve prognosis for patients are analyzed.

Keywords: Prostate cancer, risk factors, genetic mutations, hormone therapy, diet, ecology, prevention, diagnostics, androgenic hormones.

Introduction

Prostate cancer (PCa) is the most common malignancy in men (excluding skin cancer) and the second leading cause of cancer mortality among men in the United States. The risk of developing PCa is determined by a variety of factors, including age, genetics, lifestyle, and environmental exposure. Most cases of PCa are diagnosed in older adults using a variety of evaluation methods, such as prostate biopsy, prostate-specific antigen (PSA) testing, digital rectal examination, magnetic resonance imaging (MRI), or preventive screenings.

Prostate cancer is the second leading cause of cancer mortality in men in the United States. Advanced stages of prostate cancer are the transition from an intraprostatic form to an invasive and metastatic form. Bone metastases are the most common site of metastasis, and they are predominantly osteoblastic and lead to osteoskeletal complications, significantly worsening the quality of life of patients. The mechanisms by which prostate cancer causes bone formation remain unclear; however, they depend on the interaction of cancer cells and the microenvironment of the bone.

Methods

There are various treatment options for PCa, including radical prostatectomy, radiation therapy, hormone therapy, androgen deprivation therapy (ADT), and chemotherapy, with ADT being the main treatment for PCa. PCa is classified into androgen-sensitive and androgen-insensitive types based on its response to testosterone and ADT stimulation. While localized PCa can be successfully treated, the disease eventually progresses to an aggressive stage in which cancer cells develop resistance to treatment, making the disease incurable. Thus, there is an urgent need to develop new therapeutics for the treatment of progressive PCa.





The aim of this work is to investigate the role and mechanism of action of DKK-1 in the progression of prostate cancer and osteoblastic bone metastases. Dogs are the only animals that spontaneously develop prostate cancer with osteoblastic bone metastases similar to those in humans. This study used several canine prostate cancer cell lines as models. Evaluation methods based on mouse calvaria, intraosseous and intracardiac injections, and transcriptomic RNA sequencing were used to investigate the ability of prostate cancer cells to induce bone formation and resorption in vitro, the effect of DKK-1 on bone metastases in vivo, and the overall role of DKK-1 in the progression of prostate cancer.

Results

To test this hypothesis, we created stable cell lines with suppression or enhancement of NRP2 or PlexinD1 expression in a number of PCa cell lines. Inhibition of NRP2 or PlexinD1 significantly reduced cell proliferation, colony formation, sphere formation, cell migration, and cell invasion. In contrast, enhanced expression of NRP2 or PlexinD1 increased these phenotypes. Moreover, NRP2 and PlexinD1 also promote cell differentiation towards stem-like, basal, and neuroendocrine phenotypes.

Conclusion

In conclusion, these studies highlighted the value of the canine prostate cancer model for bone metastasis research, confirmed the tumor-promoting role of DKK-1 in prostate cancer, and pointed to possible therapeutic targets in DKK-1-activated signaling pathways for future research.² Overall, the data presented highlight the important role of CdGAP in prostate cancer, confirming it as a novel biomarker and a potential molecular target for developing therapeutic strategies in the treatment of patients with this disease.³

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