

Risk Factors, Prevalence and Epidemiology of Prostate Cancer

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Abstract: This article presents information from foreign sources about the factors and epidemiology of prostate cancer. Information on the importance and influence of risk factors for prostate cancer, including genetic factors, poor-quality diet, chemical drug exposure, infections, sexual activity, and ethnicity, was obtained from the National Cancer Institute (NCI) database.

Keywords: Prostate cancer genetics. Risk factors for prostate cancer. Chemical exposure and drugs. Epidemiology. Introduction is the main part.

Globally, prostate tumors are the most commonly diagnosed malignancy in men and the fifth leading cause of cancer death in men. [1] [2] It accounted for 1,414,249 new diagnoses and 375,000 deaths from the disease worldwide in 2020. [1] Globally, prostate cancer is the most commonly diagnosed malignancy in more than fifty percent of countries (112 out of 185). [6]. Most cases have no initial or early symptoms, but late symptoms may include fatigue due to anemia, bone pain, paralysis due to spinal metastases, and renal failure due to bilateral ureteral obstruction. Diagnosis is primarily based on the prostate specific antigen (PSA) test and biopsy of prostate tissue using transrectal ultrasound (TRUS), but PSA testing for screening remains controversial. [8] [9]. New diagnostic methods include free and total PSA levels, PCA3 urine test, Prostate Health Index (PHI) assessment, 4K test, exosome test, genomic analysis, MRI, PIRADS assessment and MRI-TRUS-guided biopsy. [10]. If the disease has spread to the bones or beyond the prostate, pain relievers, bisphosphonates, selective ligand inhibitors, hormonal therapy, chemotherapy, radiopharmaceuticals, immunotherapy, focused radiation, and other targeted therapies may be used. Outcomes depend on age, underlying medical conditions, tumor histology, and prostate tumor grade. [12]

Etiology: The major known risk factors are age, ethnicity, obesity, and family history. [13]. The general incidence increases with age. [14]. Risk factors for prostate cancer include male gender, older age, increased height, obesity, hypertension, lack of exercise, persistently high testosterone levels, exposure to Agent Orange, and ethnicity. [15] [16] [17]. 5 alpha reductase inhibitors. Inhibitors such as finasteride and dutasteride can shrink low-grade prostate tumors, but they do not affect the risk of high-grade cancers and therefore do not significantly improve survival. These drugs reduce the PSA level by about 50%, which should be taken into account when comparing prostate specific antigen (PSA) serial values. [14] Taking 5-alpha reductase inhibitors does not affect the risk of prostate cancer. [12] A follow-up study by health professionals examined 5-alpha reductase use and prostate cancer in 38,000 men followed over 20 years. Men who took the drug had more PSA tests, prostate exams, and biopsies, but no association was found with death, overall survival, or cancer-specific survival. At the same time, the frequency of general and local diseases was reduced in men who received 5-alpha reductase drugs. [11] [13]

Genetics: The cause of prostate cancer is not clear, but genetics certainly plays a role. Genetics, ethnicity, and family history are known to contribute to prostate cancer risk. [14] In general, patients with genetic or hereditary prostate cancer develop malignancies at an earlier age, progress more rapidly, are more likely to have locally advanced disease, and have a higher risk of recurrence after surgery. [15] Hereditary prostate cancer has the highest heritability of all major cancers in men. [26] A family history of hereditary breast and ovarian cancer or Lynch syndrome increases the risk of prostate cancer, suggesting a genetic link. [17] [18] Men in the high-risk 1% group have a nearly 6-fold increased incidence of prostate cancer compared to controls. Men with a first-degree relative (father or brother) with prostate cancer are twice as likely as the general population. p. The risk for an affected sibling is greater than for the father. [thirty]The risk is further increased if a first-degree relative has early-onset disease (<55 years). Men with two first-degree relatives have a five-fold higher risk. Patients with a family history of prostate cancer have a younger onset of cancer (2.9 years) develops and has locally advanced diseases. [11]. They also have a higher risk of biochemical recurrence after radical prostatectomy. In the United States, black men are more affected than white or Hispanic men, and the disease is more fatal for blacks. [12]

Hispanic men have a one-third lower prostate cancer incidence and mortality rate than non-Hispanic white men. [13]. No single gene is responsible for prostate cancer, but many genes are now known. [14]BRCA1 and especially BRCA2 mutations are associated with breast and prostate cancer. [34]P53 mutations are relatively rare in localized prostate cancer and more frequent in metastatic disease. P53 is usually a tumor suppressor gene. Its activity produces the protein p21, which slows down cell division. Loss of p53 activity reduces tumor sensitivity to androgens, increases prostate cancer cell proliferation, and promotes tumor growth. Thus, p53 mutations are usually a late and ominous manifestation of prostate cancer. [15]. More than 100 single nucleotide polymorphisms (SNPs) and other genes are associated with an increased risk of prostate cancer. These include: hereditary prostate cancer gene 1, various androgen and vitamin D receptors, HPC1, HPC2, HPCX, CAPB, mutL homolog 1 (MLH1), mutS homologs 2 and 6 (MSH2 and MSH6, respectively), increased post-meiotic segregation 2 (PMS2).), homeobox B13 (HOXB13), checkpoint kinase 2 (CHEK2), nibrin (NBN), BRCA1-interacting protein C-terminal helicase 1 (BRIP1), ataxia telangiectasia mutated (ATM), TMPRSS2- ETS gene family; Cancer cell growth promoters TMPRSS2-ERG and TMPRSS2-ETV1/4. [16][14][16] (Note: This is only a partial list. Clinically significant gene mutations are discussed later. Genetic Risk Score (GRS), including high-risk genetic markers and SNPs, can help stratify prostate cancer risk, especially in families. but this type of test is not yet ready for individual patient diagnosis [17].

Diet: There is very little data showing a link between trans fat, saturated fat, or carbohydrate intake and prostate cancer. [39]However, a lard diet (high in unsaturated fat) has been shown to significantly accelerate the development of prostate cancer in a mouse model. [40]Alcohol consumption has little or no effect on prostate cancer risk. [11] [12] But there is some evidence that moderate consumption of red wine may be beneficial. [13]Vitamin supplements do not reduce the risk, and some vitamins may increase it. [11]High calcium intake is associated with prostate cancer. [12]Diets high in saturated fat and dairy products increase the risk of cancer. [14]Consuming whole milk after a prostate cancer diagnosis increases the risk of recurrence, especially in overweight men. [15]Low levels of vitamin D in the blood may increase the risk of developing prostate cancer. [46] Patients with prostate cancer and vitamin D deficiency have higher overall and cancer-related mortality. [17] [48]This suggests that vitamin D supplementation may be beneficial for prostate cancer patients who are vitamin deficient. Red meat and processed meat in general are also not significantly affected, but some studies suggest that a higher intake of meat is associated with a higher risk. [11]Fish consumption may reduce prostate cancer mortality but not incidence. [10]However, high levels of omega-3 fatty acids in fish oil are associated with an increased risk of clinically significant high-grade prostate cancer. [11] [12]. Some evidence supports the idea that a vegetarian diet reduces the incidence of

prostate cancer, but this is not considered a reliable or significant effect. [13]. Increasing the amount of soy products in the diet reduces the risk of prostate cancer. These products contain phytoestrogens that have a direct estrogenic effect or by inhibiting 5-alpha reductase. [54] [15]. Folic acid supplementation also had no significant effect on prostate cancer risk. Lycopene in tomatoes has a protective effect against prostate cancer. In general, the Mediterranean diet (rich in antioxidants from olive oil, tomatoes, etc.) may help reduce the risk of prostate cancer to some extent. [10] It has also been shown to slow Gleason grade progression in patients on active surveillance for low-grade prostate cancer. [11]

Chemical exposure and medications: Prostate cancer is associated with certain medications, surgical procedures, and medical conditions. [12] The use of statins, metformin, and NSAIDs, especially those with anti-COX-2 activity, may reduce the risk of prostate cancer. [13] Metformin inhibits the COX2/PGE2 axis, which blocks prostate cancer progression by suppressing tumor-associated macrophages. This effect is enhanced in patients on androgen deprivation therapy. Regular use of aspirin, which is currently taken by approximately 23.7 million men, reduces the risk of prostate cancer. [15] This effect may be due to both anti-inflammatory activity and decreased angiogenesis. [66] The beneficial effects of aspirin and NSAIDs appear to be more important in aggressive prostate cancer and prostatitis. Veterans exposed to Agent Orange develop prostate cancer at a younger age and at a higher clinical stage than veterans not exposed. However, the overall results were similar. Exposure to Agent Orange may increase the risk of prostate cancer, especially after surgery.

Sexual activity: Having multiple sexual partners in a lifetime or starting sexual activity at an early age increases the risk of prostate cancer. Frequent ejaculation may reduce the overall risk of prostate cancer, but a reduction in ejaculation frequency is not associated with a corresponding increase in the incidence of advanced disease. [10] [11]

Infections: Infections may be associated with the onset and progression of prostate cancer. [72] Infections with chlamydia, gonorrhea, or syphilis increase the risk of developing prostate cancer. [73] Human papillomavirus (HPV) has been suggested to play a role in prostate cancer, but the evidence is inconclusive. [14]

Vasectomy and prostate cancer: There was once thought to be a link between vasectomy and prostate cancer, but larger observational studies have failed to confirm such a link. [15] However, a recent meta-analysis again suggested an association, so the question remains unresolved. [15] [16]

Epidemiology: Prostate cancer is the most commonly diagnosed malignancy in men and the second leading cause of cancer death among men in the United States. [17] Lung cancer ranks first. [11] [12] In the United States, prostate cancer is more common in African Americans, at twice the rate of the general population. [84]. The disease is less common in Asian and Hispanic men than in white men. [85] The World Health Organization (WHO) lists Guadeloupe, Martinique, Ireland, Barbados, Saint Lucia, Estonia, Puerto Rico, France, Sweden and the Bahamas as countries with high rates of prostate cancer. . The incidence is highest in Guadeloupe at 184 per 100,000, followed by the Bahamas at 98 per 100,000, with the United States in 14th place. The least incidence is observed in Asian countries. The highest WHO prostate cancer death rates are reported in Grenada, Zimbabwe, Barbados, Haiti, Zambia, Jamaica, Trinidad/Tobago, Bahamas, Dominican Republic, Saint Lucia and Côte d'Ivoire. Mortality rates within this group range from 80/100,000 in Grenada to 30/100,000 in Côte d'Ivoire. By comparison, the global average death rate is 7.7 per 100,000. The US death rate is 11.46 per 100,000, ranking 126th. The lowest prostate cancer death rates are reported in Nepal and Yemen, <1 per 100,000.

Prostate cancer is more common in developed countries and less common in Asian men living in Asia. When Asians immigrate to the United States, their incidence of prostate cancer increases, but remains below the overall risk for the general population of American men. [16]. In Europe,

prostate cancer is the third most common cancer after breast and colon cancer. [17] In the United Kingdom, as in the United States, it is the second leading cause of cancer death among men after lung cancer. [17] According to the World Health Organization, the death rate in Sweden, which performs very few PSA tests and is less aggressive in treating prostate cancer, is 2.5 times higher than in the United States. This makes prostate cancer the number one cause of cancer death among Swedish men, even surpassing lung cancer. By age 80, more than 80% of men will have prostate cancer. However, it can be slow-growing, low-grade, relatively harmless, and has little effect on their survival in this age group. In 2015, there were approximately 3 million prostate cancer survivors in the United States. This number is expected to reach 4 million by 2025. [88] Approximately 20% of men diagnosed with prostate cancer will eventually die from cardiovascular disease. Androgen deprivation therapy increases cardiovascular risk. Kentucky has the highest prostate cancer incidence and mortality rates in the United States. National Cancer Institute (NCI) and Surveillance, Epidemiology, and End Results (SEER) databases per 100,000 Prostate cancer mortality rates, calculated as deaths, are as follows: 42.0: Blacks 20.1: General population 19.4: American Indians 18.7: Whites (Caucasians) 16.5: Hispanics 8.8: Asians.

The prostate is primarily a glandular tissue that produces 25% to 30% of spermatozoa. This part of the prostate gland nourishes the sperm and provides alkalinity, which helps maintain a high pH level. (The seminal vesicles produce the rest of the seminal fluid.) Androgen (testosterone) is required for optimal prostate function. Therefore, hormone therapy (testosterone deficiency) is very effective. Castration-resistant tumors are thought to produce androgens intracellularly.

Cancer begins with mutations in the normal glandular cells of the prostate, usually in the peripheral basal cells. Prostate cancer often occurs in the peripheral zone, primarily in the part of the prostate that can be palpated during a digital rectal examination (DRE). Prostate cancer is an adenocarcinoma because it develops mainly from the glandular part of the organ and shows typical glandular structures on microscopic examination. Cancer cells begin to grow and multiply, initially spread to nearby prostate tissue and form a tumor nodule. Such a tumor can grow outside the prostate (extracapsular spread) or be localized within the prostate for decades. Prostate cancer usually metastasizes to bones and lymph nodes. Bone metastases are thought to arise in part from drainage of the prostatic venous plexus into the vertebral veins. The prostate accumulates zinc and produces citrate. However, increasing zinc and citrate in the diet or in supplements does not appear to affect prostate health or the development of prostate cancer.

Conclusions.

Prostate cancer is the most commonly diagnosed malignancy in men and the second leading cause of cancer death among men in the United States. In the United States, prostate cancer is more common in African Americans. Risk factors for prostate cancer include male sex, older age, increased height, obesity, hypertension, lack of exercise, persistently high testosterone levels, exposure to Agent Orange, and ethnicity. Risk factors for prostate cancer include genetic factors, poor diet, exposure to chemical drugs, infections, sexual activity, and ethnicity. Prostate cancer mortality rates, calculated as deaths per 100,000 population in the National Cancer Institute (NCI) and Surveillance, Epidemiology, and End Results (SEER) databases, are: 42.0: Black 20.1: General population 19.4: American Indian 18.7: White (Caucasian)) 16.5: Spaniards 8.8: Asians.

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