IJMPR 2025, 9(9), 08-16

International Journal of Modern Pharmaceutical Research
www.ijmpronline.com

ISSN: 2319-5878
IJMPR
Review Article
SJIF Impact Factor: 6.669

PROSTATE CANCER AND CURRENT SCENARIO: INCIDENCE AND FUTURE OUTLOOK

Anamika Daniel[1]*, Basant Khare[1], Sunayna Kesharwani[2], Shikha Mishra[2], Prateek Kumar Jain[1], Harshita Jain[1]

[*1]Adina College of Pharmacy Sagar, Madhya Pradesh.
[2]Adina Institute of Pharmaceutical Sciences Sagar, Madhya Pradesh.

Article Received on: 02/07/2025
Article Revised on: 22/07/2025
Article Accepted on: 12/08/2025

***Corresponding Author**
Anamika Daniel
Adina College of Pharmacy
Sagar, Madhya Pradesh.

ABSTRACT
An estimated 1.6 million men receive a prostate cancer diagnosis and 366,000 pass away from the disease each year, making it one of the leading causes of illness and death among men. In this review, we go over the current level of evidence supporting particular dietary, lifestyle, and genetic factors linked to risk of prostate cancer. We concentrate on risk factors because of the molecular heterogeneity of this malignancy. For prostate cancer that is deadly or advanced. Initially, we offer descriptive epidemiological data and global trends in the incidence and mortality of prostate cancer. This includes talking about how prostate-specific antigen screening affects the epidemiology of prostate cancer. Additionally we provide an overview of the data supporting a few risk variables for which there is substantial or likely evidence of a correlation: genetics, physical activity, smoking, lycopene and tomatoes, fish, vitamin D and calcium, obesity and weight change, and statins. Lastly, we point out future avenues for the study of prostate cancer epidemiology.

KEYWORDS*:* Prostate Cancer; Malignancy; Incidence & Mortality; Lifestyle.

INTRODUCTION

More than 1.4 million people received a prostate cancer diagnosis in the 2020s, and more than 370,000 people died from the disease worldwide. It is commonly accepted that prostate cancer is a genetic disorder, and that genetic variations are a major factor in determining an individual's risk and susceptibility to developing prostate cancer.[1]

A polygenic risk score that considers more than 269 germline risk variations can be used to accurately estimate the lifetime risk of prostate cancer. Genome-wide investigations have significantly improved our understanding of prostate cancer, but they have had little impact on the development of new diagnostic and therapeutic approaches. Lifestyle factors like food and physical exercise are known to be modifiable risk factors for prostate cancer and its more aggressive forms, in addition to hereditary predispositions.[2] However, more research is still needed to determine the precise relationship between particular lifestyle choices and the risk of prostate cancer. In order to map the entire human genetic landscape—which includes both human and microbial cells that live in and interact with the human body—the Human Microbiome Project is a conceptual extension of the Human Genome Project.[3]

Globally, prostate cancer is a significant health concern. Numerous factors, including lifestyle decisions, genetic predispositions, and the makeup of the microbiome, influence its development and progression. Although the microbiome's impact on general health and illness is widely known, less is known about how specifically it contributes to prostate cancer.[4] The role of the microbiota in this kind of cancer has begun to be investigated in recent years, but it is yet unknown how these results may affect diagnosis and treatment. This emphasizes how important it is to incorporate microbiome research into clinical practice. In this study, we evaluate the potential uses of the microbiome and talk about the limitations of gut and urine microbiomes in relation to several aspects of managing prostate cancer, such as risk assessment, treatment, diagnosis, and prevention.[5,6] The increasing need for more thorough research in this field is supported by our analysis.

Global Incidence

According to the Global Burden of Disease Cancer Collaboration (2016), over 1.6 million new instances of prostate cancer were reported in 2015, making it the most common disease diagnosed in men worldwide. In wealthy countries, this kind of cancer is very common. According to the Global Burden of Disease Cancer Collaboration (2016), the chance of receiving a prostate cancer diagnosis by the age of 79 is one in six in nations with a high sociodemographic index, whereas it is one in 47 in those with a low to moderate sociodemographic index.[7] Prostate cancer is the most prevalent disease in the US, with an estimated 180,890 new cases reported in 2016. There are notable differences in the incidence of prostate cancer worldwide. Men with the highest risk (African-American men in the United States) and those at the lowest risk (Asian men in their native countries) have age-adjusted incidence rates that are a startling 40 times different (There are a number of reasons for this difference in incidence rates. Differences in the severity of diagnostic procedures, especially those related to prostate-specific antigen (PSA) screening, can be associated with variations in population statistics.[8] However, the fact that there were regional differences in the incidence of prostate cancer prior to PSA screening implies that lifestyle factors might potentially play a role in disease risk **(Figure 1).**

Furthermore, results from research on migration support the impact of lifestyle. For example, although their rates are still lower than those of the host countries, men who relocate from low-risk regions, like Asia, to high-risk locations, like the United States, have higher incidence and mortality rates of prostate cancer than their counterparts back home.[9]

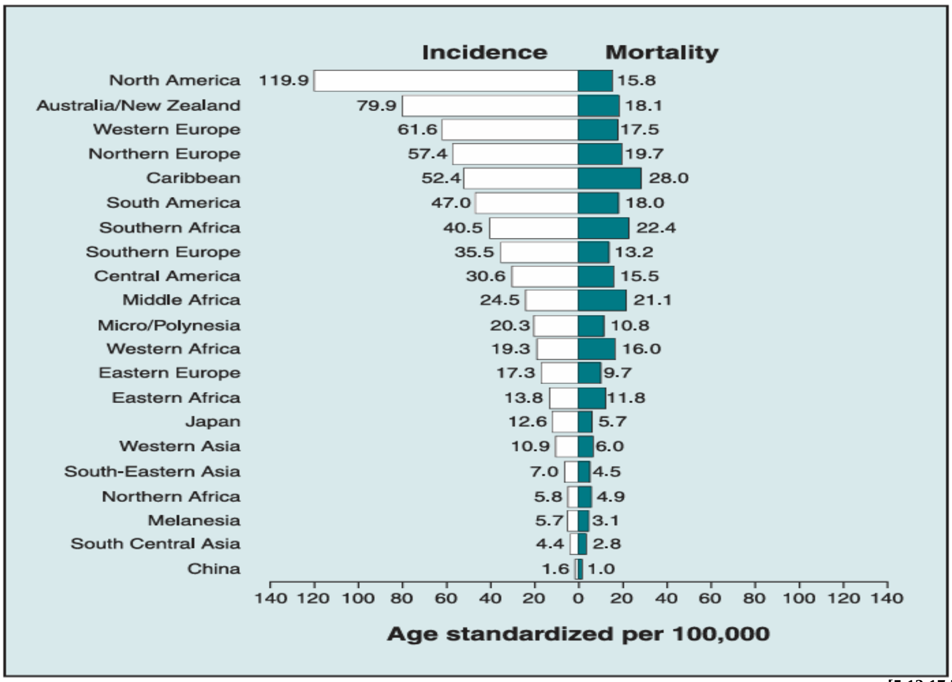

Figure 1: Global Incidence of Prostate cancer and mortality per 100000 persons.[5,12,17,21]

Trends

The use of PSA testing for early disease detection, especially in Western countries, has been strongly associated with recent notable international difference in the incidence and mortality rates of prostate cancer.[10]

Due to the widespread use of PSA testing, incidence rates have decreased in the US, Australia, and Canada after increasing in the 1980s and 1990s. On the other hand, the incidence is still somewhat rising in European nations, which can be ascribed to a combination of variables like dietary practices and UV radiation exposure, as well as an increasing awareness of PSA screening and its slow implementation. Prostate cancer incidence is expected to climb globally by 2040, with an estimated 1,017,712 new cases (+79.7% overall change). Africa is predicted to have the greatest incidence rates (+120.6%), followed by Asia (+100.9%) and Latin America and the Caribbean (+101.1%). Europe, on the other hand, is predicted to experience the least increase in incidence rates (+30.1%).[11-12] Longer life spans are probably linked to this increasing trend in incidence. Improved case reporting and documentation, as well as easier access to healthcare, may be the cause of the increased incidence rates in emerging nations. A change towards Western lifestyle factors, such as obesity, sedentary activity, and nutritional impacts, is also shown by the rise in prevalence in areas where PSA testing is not often done. In many Western countries, notably North America and portions of Western and Northern Europe, the death rate from prostate cancer has been steadily declining. Although the precise reasons for this drop are yet unknown, improvements in early identification and treatment techniques are probably to blame.[13] A recent randomized controlled trial conducted in the United States found no proof that PSA testing significantly lowers the death rate from prostate cancer. However, a different study carried out in Europe found that PSA testing is beneficial. When looking at patterns by ethnicity, it was shown that between 2001 and 2015, African-American men's mortality decreased more than that of White males. According to research this tendency can be explained by the development of better diagnostic methods and therapies, especially for metastatic and resistant prostate cancer.[14]

According to forecasts, world mortality could double between 2018 and 2040, translating into almost 379,005 deaths. With a predicted increase of 124.4%, Africa is predicted to have the greatest death rates, followed by Asia at 116.7%, and Europe at 58.3%. Given the paucity of resources for prostate cancer screening and detection, which frequently results in diagnoses at more advanced stages, this pattern is not surprising. Despite a reduced prevalence of the disease, the high death rates in developing nations may be further exacerbated by the lack of easily accessible medical care.[15]

Mortality

With an estimated 366,000 deaths and 6.3 million disability-adjusted life years lost in 2015, prostate cancer is the fifth most common cause of cancer-related deaths globally (Global Burden of Disease Cancer Collaboration 2016).[16] Prostate cancer mortality rates exhibit less variance, with a roughly tenfold difference seen globally, although incidence rates vary significantly among nations. Furthermore, death trends deviate from incidence rates; for example, prostate cancer mortality is typically higher in less developed regions than in more developed ones **(Table 1).** Populations from the Caribbean and portions of Middle and Southern Africa have the highest death rates, whereas Asia, particularly the Eastern and South-Central regions, have the lowest rates. With an estimated 26,120 fatalities predicted in 2016, prostate cancer ranks as the second most common cause of cancer-related deaths among males in the United States.[17] Although the causes of this trend are yet unknown, prostate cancer mortality has significantly decreased in a number of Western nations, including the US. This decrease has probably been facilitated by early discovery via PSA screening and subsequent therapy. It's interesting to note that prostate cancer mortality is rising in some nations, such those in Africa, where screening systems are either nonexistent or very limited.[18]

Table 1: Global mortality prevalence rate by Prostate cancer (Data as of year 2020-21).[7,13,19,30]

S. No.	Age (Yrs.)	Japan	US (Black)	US (White)	Greece	Hungary
1	21-30	1	10	12	1	2
2	31-40	21	35	34	3	25
3	41-50	15	45	39	4	24
4	51-60	23	50	46	6	27
5	61-70	36	65	59	12	40
6	71-80	43	80	76	26	55
7	81-90	49	2	3	35	70

Survival

Despite the high incidence rate of prostate cancer, the majority of cases are discovered while the cancer is still localized within the prostate. About 98% of men with prostate cancer in the US survive for five years after receiving a diagnosis. The 5-year survival rate was 83%, according to data from the Eurocare study (EUROCARE-5), which followed patients diagnosed between 2003 and 2007.[19] Countries in Eastern Europe reported a survival rate of 76%, but those in Southern and Central Europe reported a percentage of 88%. Interestingly, survival rates have increased over time throughout Europe, with Eastern European countries experiencing the biggest improvements. Prostate cancer is still the second most common cause of cancer-related deaths among men in the US, despite tremendous scientific progress in the last several decades in understanding the molecular pathways and risk factors linked to the disease.[20] In general, early detection is essential for both successful treatment and preserving a disease-free status for all types of cancer. Men may choose active surveillance or watchful waiting over early treatment, however, as most prostate tumors advance slowly and are frequently categorized as "low-risk," avoiding any potential negative effects.[21]

Risk factor

Numerous ways that individual biology and lifestyle decisions impact the risk of getting prostate cancer and surviving it have been revealed by epidemiological studies on the disease. Our present understanding of risk factors enables the identification of people at heightened risk and the application of behavioral modifications to lessen the effect of the disease, even though many parts of its origins are still unknown. Prostate cancer has a wide range of clinical manifestations, as was previously indicated.[22] Most men have slow-growing or indolent types of the disease, but some men have aggressive varieties. The underlying causes of the disease also exhibit this clinical heterogeneity **(Table 2)**. Certain risk variables have distinct associations with fatal and indolent forms of the disease, as will be covered in the sections that follow. Prostate cancer epidemiology must so differentiate between risk variables linked to advanced or fatal stages of the disease and those associated with all incidences of prostate cancer.[23]

Table 2: Elaboration of risk factors associated with the evidence and occurrence of Prostate cancer.

S. No.	Risk Factors	Limit recurrence
1	Obesity	Strong
2	Cholesterol Factor	Probable
3	Gigantism	Strong
4	Dairy consumption	Probable

5	Smoking	Strong
6	Old age	Strong
7	Family history	Strong
8	Genetics	Strong
9.	Vitamin Consumption	Low
10	Nicotine and Caffeine consumption	Probable
11	Physical activities	Low
12	Medicines (drug Abuse)	Strong
13	Statins usage	Probable

The impact of PSA screening in epidemiological studies should be taken into account when evaluating the evidence for prostate cancer risk factors since it may have an impact on the associations between risk factors and prostate cancer that have been observed. Risk factors have the ability to impact the disease at every stage of its development, from onset to metastasis and, eventually, to death. Therefore, depending on clinical features like disease stage or tumor grade, the association between a risk factor and prostate cancer may change.[24] It makes biological sense to believe that the risk factors for aggressive forms of prostate cancer are different from those for prostate cancer in general. Furthermore, PSA testing could introduce confounding variables because men who have regular screenings tend to be healthier generally, which can influence the diagnosis of prostate cancer on its own **(Figure 2)**. Therefore, it is essential to thoroughly examine these characteristics while assessing epidemiological studies pertaining to prostate cancer.[25]

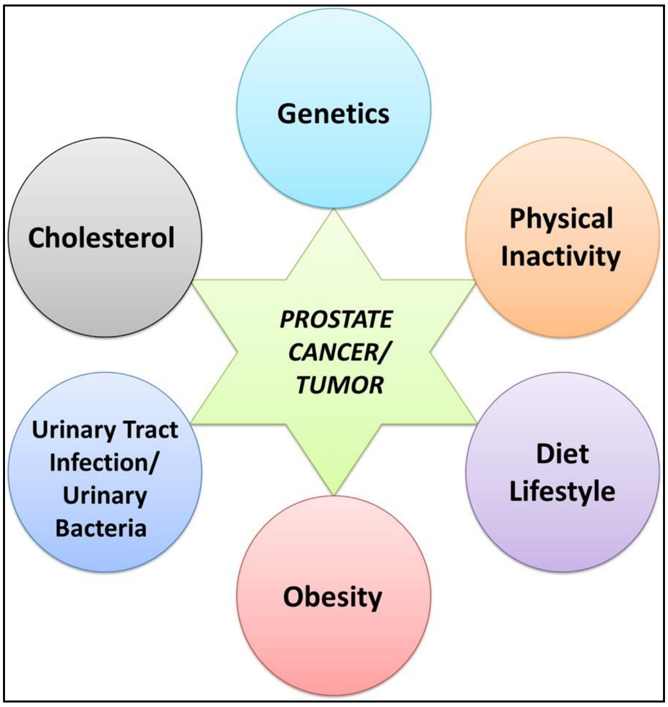

Figure 2: Various Risk factors associated with Prostate cancer and manifestation.

Prevention

A strong approach to prostate cancer prevention might greatly improve public health and provide many benefits for men. This includes the potential to reduce the high lifetime risks of prostate cancer, lessen the side effects of cancer treatments, especially for newly diagnosed patients with biologically indolent prostate cancer, who frequently receive curative treatment rather than choosing active surveillance, and deal with the difficulties in eradicating metastatic prostate cancer, which can be fatal.[26] The substantial impact of lifestyle variables on the beginning of prostate cancer is highlighted by epidemiological research. Because prostate carcinogenesis takes place over a number of decades, changing one's lifestyle may be a useful and affordable way to slow the development of prostate cancer. The majority of studies show that a diet high in fruits, vegetables, and antioxidant-rich micronutrients, and low in saturated fats and well-cooked red meats, can significantly lower the risk of prostate cancer as well as the risk of other diseases common in developed countries, even though results regarding specific lifestyle factors that contribute to prostate cancer risk have occasionally been inconsistent.[27] To open up new preventative options, a better understanding of the etiology of prostate cancer is essential. Numerous dietary supplements and medicinal substances have been studied as possible chemopreventive options. For example, selenium and vitamin E have demonstrated some promise, but conclusive analyses in the SELECT trial showed that neither substance significantly reduced the risk of prostate cancer.[28]

Moreover, laboratory and observational studies have looked at toremifene (a selective estrogen receptor modulator), NSAIDs, and vitamin D analogs. The NSAID rofecoxib, a COX-2 selective inhibitor, was unable to be adequately assessed because the trial was closed after the drug's removal, and vitamin D has not been formally tested in primary preventive trials.[29]

Treatments

Prognostic factors, including the initial PSA level, clinical TNM stage, and Gleason score, have been evaluated alongside additional elements such as baseline urinary function, comorbidities, and age when determining treatment options for prostate cancer. Recent advancements in the diagnosis and treatment of prostate cancer have improved clinicians' ability to categorize patients by risk and recommend therapies tailored to both cancer prognosis and patient preferences.[30]

1. Drug repurposing

Drug repurposing, often referred to as drug repositioning, reprofiling, or retasking, involves discovering new applications for existing approved medications. One significant benefit of drug repurposing compared to traditional drug development is that repurposed candidates have already been subjected to comprehensive research, including animal studies and clinical trials, which assess their safety, optimization, formulation, and pharmacokinetic and pharmacodynamic characteristics. This prior research typically accelerates the development process for new applications of the drug and lowers the likelihood of failure in subsequent efficacy testing during clinical trials.[31]

As a result, these previously evaluated drugs can quickly advance to phase II and phase III human clinical trials, potentially leading to a substantial reduction in drug development costs. Researchers are particularly interested in this approach as it helps address some of the challenges currently encountered in clinical research for new cancer treatments, such as drug shortages.

2. Chemotherapy

Chemotherapy employs anticancer medications to destroy or suppress the proliferation of cancer cells. Significant advancements have been made in the treatment of prostate cancer following years of research into genetics, diagnosis, and therapeutic approaches. Docetaxel (Taxotere) is the most frequently used chemotherapy agent for prostate cancer.[32]

Some of the following examples of drug are often used as first priority treatment of chemotherapy against prostate cancer:

a) Docetaxel is considered the primary treatment option for castration-resistant prostate cancer. This antimicrotubule agent binds to β-tubulin, preventing the depolymerization of microtubules, which in turn hinders mitotic cell division and triggers apoptosis.

b) Cabazitaxel is a new semi-synthetic antineoplastic agent extracted from the needles of different yew tree species (Taxus). It is commonly marketed under the brand name Jevtana. This second-generation treatment is designed to combat resistance to docetaxel. Due to the presence of extra methyl groups, cabazitaxel exhibits a low affinity for P-glycoprotein. Its metabolism occurs primarily in the liver through the enzymes CYP3A4/5 and CYP2C8, accounting for 10–20% of its processing.[33]

3. Combination therapy

Combination therapy has proven to be an effective approach for treating prostate cancer. This strategy was specifically designed to address castration-resistant prostate cancer (CRPC) and other variants of the disease. Currently, there are no medications available that can effectively treat CRPC, and the existing approved treatments, whether used alone or in combination, typically extend a patient's life by only a few months. Presently available treatments for prostate cancer do not offer a cure, and the disease tends to evolve into the castration-resistant form over time.[34] Utilizing combination therapy with existing treatment options may significantly enhance a patient's lifespan and help control tumor growth. Among the various strategies for managing metastatic prostate cancer, androgen deprivation therapy (ADT) shows greater potential for combination treatments compared to other therapeutic methods. Ongoing and approved clinical trials involving ADT include: (i) ADT combined with radiation therapy, which is often used for high-risk patients to delay or prevent progression to CRPC; (ii) ADT paired with chemotherapy, which has been associated with improved survival rates in several studies, although it may lead to adverse side effects and, in some cases, mortality; and (iii) the combination of immunotherapy and ADT, which has been reported to extend patient survival by an average of 8.5 months.[35]

4. Hormonal Therapy

Hormonal therapy, commonly referred to as androgen deprivation therapy (ADT), is utilized in the management of advanced or metastatic prostate cancer. This approach works by inhibiting the production of testosterone and other male hormones, which are known to promote the growth of prostate cancer cells. As a result, the significant reduction in male hormone levels leads to decreased androgen activity on the androgen receptor. This reduction is typically achieved through bilateral orchiectomy or medical castration, which involves the use of luteinizing hormone-releasing hormone (LHRH) analogs or antagonists. LHRH analogs function by increasing levels of luteinizing hormone (LH) and follicle-stimulating hormone (FSH) through stimulation of pituitary receptors. This initial stimulation is followed by a downregulation of these receptors, resulting in lower LH and FSH levels and, consequently, reduced testosterone production. Common LHRH agonists include leuprolide, goserelin, triptorelin, and histrelin. In contrast, LHRH antagonists work by directly blocking pituitary receptors, leading to an immediate decrease in testosterone synthesis. However, ADT is associated with both short-term and long-term side effects, including hyperlipidemia, fatigue, hot flashes, flare effects, osteoporosis, insulin resistance, cardiovascular issues, anemia, and sexual dysfunction.[36]

5. Cryotherapy

This technique entails the surgical placement of cryoprobes into the prostate, guided by ultrasound. It consists of freezing the prostate gland to temperatures ranging from -100 °C to -200 °C for approximately 10 minutes. Nevertheless, there have been reports of complications linked to this procedure, such as urinary incontinence, urinary retention, erectile dysfunction, fistula formation, and rectal discomfort.

6. Radiation

Radiation therapy is considered one of the most effective treatments for eliminating prostate cancer cells through the application of high-energy radiation. This therapy delivers radiation to cancerous cells using various methods, including brachytherapy, which involves placing radioactive seeds within the body, and external beam therapy, where energy is directed through the skin to target the cancerous areas. The primary goal of radiation therapy is to deliver concentrated doses of high-energy rays or particles directly to the prostate while minimizing damage to surrounding healthy tissues. The dosage is tailored according to the severity of the prostate cancer.[37] This treatment option is deemed suitable for patients who are not candidates for surgical interventions. The well-known radiation therapy which are the part of modern day prostate cancer treatment are

a. Brachytherapy
b. External radiation beam therapy
c. Radioactive molecules based radiotherapy (Radium 223).[40]

Application of Nanomedicine in Prostate cancer

The strategic distribution of nanoparticles significantly enhances antitumor responses while minimizing toxicity and associated costs, positioning nanomedicine as a leading approach in cancer treatment. In the context of prostate cancer, various nanoparticles are employed for drug delivery, including liposomes, polymer-based nanoparticles, gold nanoparticles, magnetic nanoparticles, silica nanoparticles, quantum dots, carbon nanotubes, and hybrid particles. These nanoparticles utilize either active or passive targeting to boost immunogenicity. Their high porosity, versatility, and biocompatibility allow them to prevent the self-quenching of photosensitizing agents and facilitate the diffusion of reactive oxygen species (ROS), thereby improving the effectiveness of photodynamic therapy and inducing cytotoxic effects.[38] Additionally, these nanomaterials can be combined with other immunotherapy strategies, such as STING agonists or CpG oligonucleotides, to foster systemic antitumor immunity. Lipophilic anticancer drugs are effectively encapsulated within liposomes, polymer nanoparticles, and other delivery systems, creating a protective barrier between the drug and the body. These systems enable targeted delivery and safeguard against drug degradation in experimental conditions, releasing the drug contents as they degrade. This approach enhances drug accumulation within tumors, thereby increasing therapeutic efficacy. Due to their unique size, surface charge, and ability to be functionalized with specific ligands, nanocarriers are particularly well-suited to overcome the biological barriers that typically limit the effectiveness of treatments for solid tumors.[39] The comprehensive advantages of polymer based nanomedicines in prostate cancer are elaborated in below table 3

Table 3: Illustration of Nanomedicine application in prostate cancer treatment.

S. No.	Nanomedicine Type	Benefits	Target mechanism
1	Dendrimers	Enhanced drug loading and better drug delivery to desired site	Active Targeting/Passive targeting
2	Hydrogel/ Nanogel	Greater biocompatibility, ease in mediation & Patient Compliance	Passive targeting
3	Liposome	Better tumor and cancer targeting potentials & low side effects	Active Targeting/Passive targeting
4	Micelles	Sustained drug release and low toxicity	Active targeting
5	Metallic nanoparticle	High stability, large surface are and sustained release profile	Active Targeting
6	Quantum Dots	High water solubility, and enhance biocompatibility	Active Targeting/Passive targeting
7	Carbon Nanotubes	Enhance drug loading capacity and high stability	Active Targeting/Passive targeting

Future outlook

Numerous modifiable risk factors have the ability to reduce the risk of aggressive prostate cancer or its progression in the future. Molecular profiling of prostate cancer tumors is a promising study topic to improve our understanding of the disease's heterogeneity. The majority of prostate cancers may be divided into seven molecular categories according to the presence of gene fusions or mutations, according to research from The Cancer Genome Atlas. Furthermore, mounting data suggests that prostate cancers with certain genetic characteristics have unique etiologies and risk factors. Strategies for prevention may be greatly impacted by differences in risk factor relationships based on the phenotype of prostate cancer, which is determined by clinical, molecular, or genetic traits.

The goal of future studies should be to expand and incorporate this understanding in order to develop focused treatments that maximize results for people with prostate cancer. Additionally, a better comprehension of the molecular processes at play would make it easier to find biomarkers for early illness detection or susceptibility.[41] This review highlights the particular methodological difficulties that epidemiological studies of prostate cancer encounter. Prostate cancer epidemiology research in the future should focus on clinically relevant instances, especially those that involve high-grade, advanced-stage, or fatal illness. It is also important to consider potential biases unique to this illness when designing epidemiological investigations. For example, correct information about PSA screening is necessary to limit potential detection bias and to make the appropriate adjustments.

CONCLUSION

With a slightly lower incidence than lung cancer, prostate cancer is the most common cancer among men. Understanding the epidemiology of prostate cancer has changed dramatically with the discovery of biomarkers like PSA, which have a favorable correlation with the detection of this illness. In the US, the number of instances of prostate cancer has doubled since PSA testing and the biopsies that followed in the late 1980s. Other countries have also seen this tendency, especially Western countries. However, the hazards of overdiagnosis and serious side effects from treatment have made PSA a risky screening tool, even while it is beneficial in reducing mortality rates specific to prostate cancer. The disparity in incidence and mortality rates between various racial groups—African-American males having the highest prevalence—are among the most startling features of prostate cancer statistics. Both biological and socioeconomic factors could be responsible for this discrepancy, but it is yet unknown which genes are at play and how they interact with environmental circumstances. Research is still ongoing. In 2018, a study called "Research on Prostate Cancer in Men of African Ancestry: Defining the Roles of Genetics, Tumor Markers, and Social Stress" (RESPOND) was funded by the Prostate Cancer Foundation, the National Cancer Institute, and the National Institute on Minority Health and Health Disparities. For the first time, a comprehensive analysis of the genetic and epigenetic changes in human prostate cancer has been made possible by recent developments in genomic technologies. The identification of important signaling pathways that are causally involved in the development and spread of prostate cancer has been made easier by this data as well as focused functional research. These revelations open the door to the creation of novel tailored therapeutic interventions.

REFERENCES

1. Hamalainen EK, Adlercreutz H, Puska P, Pietinen P. Decrease of serum total and free testosterone during a lowfat high-fibre diet. J Steroid Biochem., 1983; 18(3): 369- 370.
2. Rosenthal MB, Barnard RJ, Rose DP, Inkeles S, Hall J, Pritikin N. Effects of a high-complex-carbohydrate, lowfat, low-cholesterol diet on levels of serum lipids and estradiol. Am J Med., 1985; 78(1): 23-27.
3. Lloyd JC, Masko EM, Wu C, Keenan MM, Pilla DM, Aronson WJ, Chi JT, et al. Fish oil slows prostate cancer xenograft growth relative to other dietary fats and is associated with decreased mitochondrial and insulin pathway gene expression. Prostate Cancer Prostatic Dis., 2013; 16(4): 285-291.
4. Berquin IM, Min Y, Wu R, Wu J, Perry D, Cline JM, Thomas MJ, et al. Modulation of prostate cancer genetic risk by omega-3 and omega-6 fatty acids. J Clin Invest, 2007; 117(7): 1866-1875.
5. Gibson TM, Ferrucci LM, Tangrea JA, Schatzkin A. Epidemiological and clinical studies of nutrition. Semin Oncol, 2010; 37(3): 282-296.
6. Rohrmann S, Platz EA, Kavanaugh CJ, Thuita L, Hoffman SC, Helzlsouer KJ. Meat and dairy consumption and subsequent risk of prostate cancer in a US cohort study. Cancer Causes Control, 2007; 18(1): 41-50.
7. Major JM, Cross AJ, Watters JL, Hollenbeck AR, Graubard BI, Sinha R. Patterns of meat intake and risk of prostate cancer among African-Americans in a large prospective study. Cancer Causes Control, 2011; 22(12): 1691-1698.
8. Sinha R, Knize MG, Salmon CP, Brown ED, Rhodes D, Felton JS, Levander OA, et al. Heterocyclic amine content of pork products cooked by different methods and to varying degrees of doneness. Food Chem Toxicol, 1998; 36(4): 289-297.
9. Kazerouni N, Sinha R, Hsu CH, Greenberg A, Rothman N. Analysis of 200 food items for benzo[a]pyrene and estimation of its intake in an epidemiologic study. Food Chem Toxicol, 2001; 39(5): 423-436.
10. Sinha R, Park Y, Graubard BI, Leitzmann MF, Hollenbeck A, Schatzkin A, Cross AJ. Meat and

meat-related compounds and risk of prostate cancer in a large prospective cohort study in the United States. Am J Epidemiol, 2009; 170(9): 1165-1177.
11. Tappel A. Heme of consumed red meat can act as a catalyst of oxidative damage and could initiate colon, breast and prostate cancers, heart disease and other diseases. Med Hypotheses, 2007; 68(3): 562-564.
12. Gao X, LaValley MP, Tucker KL. Prospective studies of dairy product and calcium intakes and prostate cancer risk: a meta-analysis. J Natl Cancer Inst., 2005; 97(23): 1768-1777.
13. Allen NE, Key TJ, Appleby PN, Travis RC, Roddam AW, Tjonneland A, Johnsen NF, et al. Animal foods, protein, calcium and prostate cancer risk: the European Prospective Investigation into Cancer and Nutrition. Br J Cancer, 2008; 98(9): 1574-1581.
14. Park Y, Mitrou PN, Kipnis V, Hollenbeck A, Schatzkin A, Leitzmann MF. Calcium, dairy foods, and risk of incident and fatal prostate cancer: the NIH-AARP Diet and Health Study. Am J Epidemiol, 2007; 166(11): 1270-1279.
15. Koh KA, Sesso HD, Paffenbarger RS, Jr., Lee IM. Dairy products, calcium and prostate cancer risk. Br J Cancer, 2006; 95(11): 1582-1585.
16. Gupta S. Prostate cancer chemoprevention: current status and future prospects. Toxicol Appl Pharmacol, 2007; 224(3): 369-376.
17. Wilson KM, Shui IM, Mucci LA, Giovannucci E. Calcium and phosphorus intake and prostate cancer risk: a 24-y follow-up study. Am J Clin Nutr., 2015; 101(1): 173-183.
18. Singh SV, Srivastava SK, Choi S, Lew KL, Antosiewicz J, Xiao D, Zeng Y, et al. Sulforaphane-induced cell death in human prostate cancer cells is initiated by reactive oxygen species. J Biol Chem.. 2005; 280(20): 19911-19924.
19. Joseph MA, Moysich KB, Freudenheim JL, Shields PG, Bowman ED, Zhang Y, Marshall JR, et al. Cruciferous vegetables, genetic polymorphisms in glutathione Stransferases M1 and T1, and prostate cancer risk. Nutr Cancer, 2004; 50(2): 206-213.
20. Giovannucci E, Rimm EB, Liu Y, Stampfer MJ, Willett WC. A prospective study of cruciferous vegetables and prostate cancer. Cancer Epidemiol Biomarkers Prev, 2003; 12(12): 1403-1409.
21. Hsing AW, Comstock GW, Abbey H, Polk BF. Serologic precursors of cancer. Retinol, carotenoids, and tocopherol and risk of prostate cancer. J Natl Cancer Inst., 1990; 82(11): 941-946.
22. Negoita S, Feuer EJ, Mariotto A, Cronin KA, Petkov VI, Hussey SK, Benard V, et al. Annual Report to the Nation on the Status of Cancer, part II: Recent changes in prostate cancer trends and disease characteristics. Cancer., 2018; 124(13): 2801-2814.
23. Oliver JS. Attitudes and beliefs about prostate cancer and screening among rural African American men. J Cult Divers., 2007; 14(2): 74-80.
24. Taitt HE. Global Trends and Prostate Cancer: A review of incidence, detection, and mortality as influenced by race, ethnicity, and geographic location. Am J Mens Health, 2018; 12(6): 1807-1823.
25. Jemal A, Fedewa SA, Ma J, Siegel R, Lin CC, Brawley O, Ward EM. Prostate cancer incidence and PSA testing patterns in relation to USPSTF screening recommendations. JAMA, 2015; 314(19): 2054-2061.
26. Mottet N, Bellmunt J, Bolla M, Briers E, Cumberbatch MG, De Santis M, Fossati N, et al. EAU-ESTRO-SIOG Guidelines on Prostate Cancer. Part 1: Screening, Diagnosis, and Local Treatment with Curative Intent. Eur Urol, 2017; 71(4): 618-629.
27. Baade PD, Youlden DR, Krnjacki LJ. International epidemiology of prostate cancer: geographical distribution and secular trends. Mol Nutr Food Res., 2009; 53(2): 171-184.
28. Collin SM, Martin RM, Metcalfe C, Gunnell D, Albertsen PC, Neal D, Hamdy F, et al. Prostate-cancer mortality in the USA and UK in 1975-2004: an ecological study. Lancet Oncol, 2008; 9(5): 445-452.
29. Etzioni R, Tsodikov A, Mariotto A, Szabo A, Falcon S, Wegelin J, DiTommaso D, et al. Quantifying the role of PSA screening in the US prostate cancer mortality decline. Cancer Causes Control, 2008; 19(2): 175-181.
30. Lim LS, Sherin K, Committee APP. Screening for prostate cancer in U.S. men ACPM position statement on preventive practice. Am J Prev Med, 2008; 34(2): 164-170.
31. Andriole GL, Crawford ED, Grubb RL, 3rd, Buys SS, Chia D, Church TR, Fouad MN, et al. Mortality results from a randomized prostate-cancer screening trial. N Engl J Med., 2009; 360(13): 1310-1319.
32. Schroder FH, Hugosson J, Roobol MJ, Tammela TL, Ciatto S, Nelen V, Kwiatkowski M, et al. Screening and prostate-cancer mortality in a randomized European study. N Engl J Med., 2009; 360(13): 1320-1328.
33. Bostwick DG, Burke HB, Djakiew D, Euling S, Ho SM, Landolph J, Morrison H, et al. Human prostate cancer risk factors. Cancer, 2004; 101(10Suppl): 2371-2490.
34. Dagnelie PC, Schuurman AG, Goldbohm RA, Van den Brandt PA. Diet, anthropometric measures and prostate cancer risk: a review of prospective cohort and intervention studies. BJU Int., 2004; 93(8): 1139-1150.
35. Pienta KJ, Esper PS. Risk factors for prostate cancer. Ann Intern Med. 1993; 118(10): 793-803.
36. Suzuki K, Kise H, Nishioka J, Hayashi T. The Interaction among Protein C Inhibitor, Prostate-Specific Antigen, and the Semenogelin System. Semin. Thromb. Hemost, 2007; 33: 46–52.
37. Lamy PJ, Allory Y, Gauchez, AS, et al. Prognostic Biomarkers Used for Localised Prostate Cancer Management: A Systematic Review. Eur. Urol. Focus, 2018; 4: 790–803.

38. Meyer AR, Joice GA, Schwen ZR, Partin AW, Allaf ME, Gorin MA. Initial Experience Performing In-office Ultrasound-guided Transperineal Prostate Biopsy Under Local Anesthesia Using the Precision Point Transperineal Access System. Urology, 201(115): 8–13.
39. Niraj LK, MRI in Dentistry—A Future Towards Radiation Free Imaging—Systematic Review. J. Clin. Diagn. Res, 2016; 10: ZE14–ZE19.
40. Chopra S, Foltz WD, Milosevic MF, Toi A, Bristow, RG. Comparing oxygen-sensitive MRI (BOLD R2*) with oxygen electrode measurements: A pilot study in men with prostate cancer. Int. J. Radiat. Biol., 2009; 85: 805–813.
41. Albright F, Stephenson RA, Agarwal N, Teerlink C, Lowrance WT, Farnham JM. Prostate cancer risk prediction based on complete prostate cancer family history. Prostate, 2015; 75: 390–398.