



REVIEW ARTICLE

Non-dietary environmental risk factors in prostate cancer[☆]

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KEYWORDS

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factors;
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Abstract

Introduction: The aim is to update and disclose the main environmental risk factors, excluding dietary factors, involved in the etiopathology of prostate cancer.

Materials and methods: Bibliographic review of the last 25 years of non-dietary environmental risk factors associated with prostate cancer between 1985 and 2010, obtained from *MedLine*, *CancerLit*, *Science Citation Index* and *Embase*. The search profiles were *Environmental Risk Factors/Tobacco/Infectious-Inflammatory Factors/Pesticides/Vasectomy/Occupational Exposures/Chemoprevention Agents/Radiation and Prostate Cancer*.

Results: While some non-dietary environmental risk factors increase the risk of acquiring the disease, others decrease it. Of the former, it is worth mentioning exposure to tobacco smoke, chronic infectious-inflammatory prostatic processes and occupational exposure to cadmium, herbicides and pesticides. The first factors that reduce the risk are the use of chemopreventive drugs (Finasteride, Dutasteride) and exposure to ultraviolet solar radiation. With the current data, a vasectomy does not influence the risk of developing the disease.

Conclusions: The slow process of prostate carcinogenesis is the final result of the interaction of constitutional risk and environmental factors. Non-dietary environmental factors play an important role in the etiopathology of this disease. To appropriately assess the risk factors, extensive case studies that include all the possible variables must be analyzed.

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PALABRAS CLAVE

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Factores de riesgo ambientales no dietéticos en el cáncer de próstata**Resumen**

Introducción: Se pretende actualizar y divulgar los principales factores de riesgo ambientales, excluyendo los dietéticos, implicados en la etiopatogenia del cáncer de próstata.

Material y método: Revisión bibliográfica de los últimos 25 años de los factores de riesgo ambientales no dietéticos asociados a cáncer de próstata entre 1985 y 2010, obtenida del *MedLine*, *CancerLit*, *Science Citation Index* y *Embase*. Los perfiles de búsqueda han sido *Environmental Risk Factors/Tobacco/Infectious-Inflammation Factors/Pesticides/Vasectomy/Occupational Exposures/Chemoprevention Agents/Radiation* y «Cáncer de Próstata».

Resultados: Mientras que algunos factores de riesgo ambientales no dietéticos incrementan el riesgo de padecer la enfermedad, otros lo disminuyen. Entre los primeros destacan la exposición al humo del tabaco; los procesos prostáticos infecto-inflamatorios crónicos y la exposición profesional al cadmio, herbicidas y pesticidas. Los principales factores que reducen el riesgo es el empleo de fármacos quimiopreventivos (finasterida, dutasterida) y la exposición a la radiación ultravioleta solar. La vasectomía, con los datos actuales, no influye en el riesgo de desarrollar la enfermedad.

Conclusiones: El lento proceso de carcinogénesis prostática es el resultado final de la interacción de factores de riesgo constitucionales y ambientales. Los factores ambientales no dietéticos desempeñan un papel importante en la etiopatogenia de esta enfermedad. Para valorar apropiadamente los factores de riesgo deben analizarse grandes casuísticas que incluyan todas las variables posibles.

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Introduction

In Western countries prostate cancer is a major medical problem due to its high incidence and significant mortality.¹ For these reasons, primary prevention reduces not only the considerable economic burden of diagnosis and treatment but it also the enormous emotional stress of patients and their family and social environment.^{1,2} To this end, we must progress in our current understanding of the risk factors involved in its etiopathogenesis.¹

In this paper, we carry out an updated review of the main non-dietary environmental risk factors associated with prostate cancer, with varying degrees of scientific consistency, in accordance with our bibliographic research.

Smoking

Active and passive exposures to smoke from burning tobacco are considered a carcinogen for many human cancers.³ In spite of this, the process for establishing its causal association with prostate cancer has been slow. With respect to its incidence, most epidemiological studies^{4,5} have not established a causal relationship between tobacco and the disease. Case-control studies have not found a relationship between the various variables of smoking and the incidence of the disease. However, some cohort studies documented a 2–3 times higher risk in smokers of more than a pack a day compared with nonsmokers. However, these studies have not demonstrated a convincing dose-response relationship, neither have they evaluated the influence of possible dietary risk factors that are confusing.⁴

Regarding mortality, the association has been more consistently documented.^{4–6} Several cohort studies have

established a direct relationship between smoking and higher mortality due to prostate cancer. Smoking patients double the risk of dying from the disease compared to non-smokers. Most authors found a gradual risk in relation to the number of cigarettes smoked daily and years one has been smoking. However, a convincing article documented a dose-response relationship between the number of packs per year of the smoker 10 years before diagnosis.⁶ Although in several studies ex-smokers did not seem to have a higher risk of dying from prostate cancer, one study documented adverse effects on mortality until a decade after cessation of smoking.⁶

A recently published meta-analysis provides convincing data and statistically significant causal association between exposure to tobacco smoke and the increased incidence and mortality of prostate cancer.⁷ Analyzing 24 epidemiological cohort studies including 21,579 patients, it was found that when stratified by number of cigarettes smoked, current smokers had a higher risk of between 11% and 22% compared to those who had never smoked, and of 9% compared to ex-smokers; these differences were statistically significant. Mortality in active smokers showed statistically significant differences, with an increased risk of 14% compared to those who had never smoked, which increased to 24% and 30% for the most addicted. The authors commented that probably because of their lower case material, studies carried out previously underestimated the true influence of smoking on the incidence and mortality of prostate cancer. They also recommended the need to design studies to review prognosis variables (tumor stage and grade) and medical history of smoking, both in smokers and former smokers.

The causal association with prostate cancer is biologically plausible because smoke carcinogens from tobacco act directly, causing DNA mutations, and indirectly, causing

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