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David A. Burks

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# The Epidemiology of Prostate Cancer in Black Men

David A. Burks, MD,\* and Ray H. Littleton, MD\*

*Data on the epidemiology of prostate cancer from the 1930s to the present document a dramatic racial difference in incidence, survival, and mortality rates in American men. American black men have the highest incidence and mortality rates of prostate cancer in the world. Survival data have been related to access to medical care, genetic and environmental factors, and cultural differences, including diet and social habits. Most reports present conflicting data with no clear positive correlations, and conclusions are often speculative. Better controlled, prospective studies of epidemiologic variables and a comprehensive genetic evaluation of black families with prostate cancer are needed to better understand the racial disparity affecting American black men and the biology of this disease in all men. (Henry Ford Hosp Med J 1992;40:89-92)*

The incidence and mortality rate of prostate cancer in black men has been increasing since the 1950s and is higher than that of any other group in America. Three national cancer surveys, four registries in the Surveillance, Epidemiology and End Results (SEER) of the National Cancer Institute, and surveys done by the American College of Surgeons provide a data base from 1949 to the present. Black men have an 85% greater chance of being diagnosed with prostate cancer and a 114% greater chance of dying from it than white men (1). The increased mortality rate is explained in part by the increased incidence, but a survival disadvantage is strongly suggested. Despite increased efforts and advances in early detection and screening, blacks still present with advanced disease 40% more often than whites.

Numerous investigators have evaluated multiple factors which include socioeconomic status, diet, hormones, occupational exposure, and infectious agents in an attempt to identify risk factors. Results are variable and often conflicting. We review the epidemiological data and explore new concepts regarding genetic and environmental factors that may shed some light on this malignant epidemic in the black community.

## Temporal Trends

Three national cancer surveys, conducted in 1937-1939, 1947-1948, and 1969-1971, show a marked increase in the annual incidence of prostate cancer in blacks when compared to whites. This racial disparity occurs in all age groups and parallels mortality rate trends. From the 1937-1939 survey to the 1969-1971 survey, the incidence of prostate cancer increased 53.3% for whites and 151.6% for blacks for all ages, adjusted to the 1950 U.S. population figures (2). This dramatic racial disparity is emphasized in the third national cancer survey (TNCS), from 1969-1971, which found that for the first time the incidence of prostate cancer in blacks is higher than the incidence of lung cancer (94.9 versus 89.9 per 100,000 population) (2).

More recent evaluations continue to show these trends. From 1973 to 1981 the SEER program evaluated data on black patients, principally from Detroit, Atlanta, and the San Francisco-Oakland area. From 1978 to 1981, the average annual age-adjusted incidence of prostate cancer was 120.3 per 100,000, up from 94.9, a 26.8% increase in 10 years. This compares with the increase in white men from 57.7 to 75.1 per 100,000 during the same period (3).

Data since 1981 continue to document the disparity. The SEER 1985 report, adjusted to 1970 census standards, shows an incidence of 512.2 per 100,000 population for all cancer sites in black males versus 422.4 for white males, a 21% higher incidence. For prostate cancer only, the rate was 127.6 for black males and 84.9 for white males, a 50% higher incidence for blacks (4). The gap, therefore, is widening with time.

## Age at presentation

Using the TNCS data, in each age group, except for the youngest and oldest patients, incidence rates for blacks are higher than for whites. On average for blacks, the incidence curve by age was shifted 5 years earlier than for whites. Occurrence of prostate cancer for both groups increased with age but the rate of increase occurs earlier for blacks (2).

## Stage at presentation

Jackson et al (5) reported on 231 black patients with prostate cancer studied in Washington, DC, from 1973 to 1978. A total of 51% had stage 1 or 2 (localized) disease. Only 4.4% had stage 3 disease (regional spread), and 44.9% had metastatic disease at presentation.

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\*Department of Urology, Henry Ford Hospital.

Address correspondence to Dr. Burks, Department of Urology, Henry Ford Hospital, 2799 W Grand Blvd, Detroit, MI 48202.

SEER data from 1974-1985 for San Francisco-Oakland, Atlanta, Detroit, and Connecticut showed that, for both races, the percentage of patients presenting at each disease stage remained constant over time, but blacks were consistently 40% more likely to present with distant metastasis (27.5%) than were whites (19.4%) (4). This disparity was confirmed by the American College of Surgeons study (1) which evaluated more than 1,000 patients in 1974 and 1983. The study concluded that metastatic disease presented initially in black males 60% more frequently than in white males for both years. This is the most important fact in translating incidence data into mortality data, but it cannot account for the entire increase in mortality.

### Geographic distribution

Geographic differences in the incidence of prostate cancer are difficult to evaluate because standards of medical care, demographic features, and accuracy of reporting vary substantially from region to region. Higher incidence rates for blacks have been reported in the Southwest, Midwest, and Northeast when the analysis was done by counties which are more homogeneous than are states. By contrast, whites had a higher incidence rate in the upper Midwest states and in the New England areas. There was no reported significant difference between urban and rural areas, with the exception of farmers (2).

The TNCS, 1969-1971, showed no statistically significant age-adjusted difference in incidence by geographic area for blacks, although the survey did not cover every state or city (6). Colorado, the Dallas-Fort Worth area, the San Francisco-Oakland area, and Detroit had the highest incidence.

### Mortality data

Age-adjusted mortality rates for prostate cancer in U.S. black men have also risen dramatically since 1955. The racial disparity with U.S. white men has become an epidemiologic characteristic of the disease and forms the rationale for studies on environmental or life-style factors. The time period seems too short for genetic mechanisms to be entirely responsible.

American blacks have the highest age-adjusted mortality rate for prostate cancer on earth. From 1930-1974, there was a 33.6% mortality increase among whites but a 322% increase (adjusted to the 1950 census) among nonwhites (predominantly blacks). Most of this increase occurred in blacks over age 75, whereas the mortality rates in whites leveled off after age 75 (2). In 1985, SEER data from San Francisco-Oakland, Detroit, Atlanta, and Connecticut, adjusted to the 1970 U.S. population census, revealed a death rate of 21.9 per 100,000 white males and 45.8 for black males, a twofold difference. This racial mortality disparity is greater than the incidence disparity. In 1969, the rate was 21.6 per 100,000 population for white males and 33.6 for black males, demonstrating stability over 15 years in the age-adjusted mortality for whites, while the rate increased 36% for blacks (4).

### Survival Data

Mortality rates are a product of the incidence rate in previous years and the survival rate. Survival rates depend on the stage of

disease at diagnosis, treatment, and response to treatment, all highly variable. Survival data, therefore, are not accurately reflected in mortality data and should be analyzed independently.

Prostate cancer accounts for nearly 11% of cancer deaths in males (7). The five-year survival rate for prostate cancer, adjusted for normal life expectancy, is 70% for localized disease but only 35% for patients with distant metastases (8). Numerous studies show that survival is related to tumor aggressiveness, the extent and stage at which the tumor is detected, the timing and type of treatment, as well as the willingness of the patient to follow the prescribed treatment regimen.

The SEER five-year survival rates for cases diagnosed from 1978 through 1982 for black and white males by age and stage of disease indicate nearly a 10% poorer overall survival rate for black males than for white males. The survival rate for white males of all ages and disease stages is 69.2% versus 63.3% for black males (2).

Blacks tend to have more advanced disease at the time of discovery, indicating a delay in diagnosis and treatment. However, survival rates for blacks are lower even when diagnosed at the same stage of disease. A number of factors have been suggested as influencing survival: 1) access to medical care, 2) behavioral patterns that delay seeking treatment, 3) genetic susceptibility, 4) cultural differences, and 5) socioeconomic status (SES).

Access to medical care is related to both SES and availability of health care. Dayal and Chiu (8) showed that apparent survival differences attributed to race are probably due to SES differences and that the biology of the disease is apparently the same for the two races. A study by Austin et al (9) not only revealed a poorer survival in blacks caused by delay in seeking medical attention but also showed that young blacks had a worse survival rate than older black or young white males. A median survival rate of 3.9 years versus 6.0 years was found when young black males with cancer of the prostate were compared to young white males. The young black male delayed seeking medical attention more than three months 72% of the time, but there was no such delay among white males. Many black men avoid doctors because of fear of having cancer and because they wish to avoid a possibly unpleasant cross-cultural encounter.

### Epidemiologic Factors

Black American males have the highest known incidence of prostate cancer in the world (10). Epidemiologists have sought clues to the causes of these racial disparities (Table) but have found no correlation between prostatic cancer and variables such as venereal disease, smoking, alcohol, sexual habits, or occupational exposure.

High testosterone levels have been proposed as a risk factor, as well as composition of the diet, exposure to heavy metals, and levels of vitamin A. However, the most consistent risk factor is age. Carcinoma of the prostate is a disease of old men. A Washington, DC, survey of prostate cancer in black men revealed that the incidence was essentially nil in the age group 30 to 39, 5.3/100,000 in the age group 40 to 49, but 953.5/100,000 in the age group of 80 years old and older. The peculiar age distribution of prostate cancer may be attributed either to an unusually long la-

tent period or to the fact that important risk factors begin to operate late in life.

Access to medical care is a factor related more to survival than to the incidence of prostate cancer. SES is associated with race only in that a higher proportion of blacks have lower SES levels. Occupational exposure may be important. Brownson et al (11) found increased risk for farmers, mechanics, sheet metal workers, separating machine operators, and men employed in several manufacturing industries. The greatest risk occurs in workers exposed to cadmium as in welding, electroplating, and alkaline battery production. No definite relationship is reported between prostate cancer and drinking alcohol. In fact, Jackson et al (5) have suggested that the cirrhotic liver, unable to metabolize estrogen normally, could be protective against prostate cancer. Only one study showed any association between smoking and prostate cancer.

The association between sexual activity and prostate cancer is not clear. Reportedly the disease is more frequent and mortality rates are higher among married men than among those who were never married (5). Also, incidence is higher among the "common law" group than among those who are widowed, divorced, or separated. However, other studies did not demonstrate any relationship between marital status, fertility, and prostate cancer. A recent article suggests a relationship between vasectomy and prostate cancer, but the numbers are small, controls poorly chosen, and no known biologic reason was offered for the cause and effect (12). Steele et al (13) reported a positive association between prostatic cancer and sexual activity, and Jackson et al (5) showed that prostate cancer patients were more active sexually than were controls.

The heavy metals zinc and cadmium have long been suspected as etiologic factors. The prostate gland has a high concentration of zinc, and sexual activity lowers its zinc content. Reportedly prostate glands with cancer or prostatitis contain lower levels of zinc than nonneoplastic glands. Cadmium is considered a zinc antagonist, and several studies have associated prostatic cancer with consumption of diets rich in cadmium. Oysters are an important source of this element. Increased cadmium intake may also be related to consumption of soft drinking water that has passed through metal pipes (14,15).

A family history of cancer is commonly elicited from patients with prostatic carcinoma, and familial aggregation has been observed repeatedly. Steele et al (13) reported that fathers and brothers of index patients had a history of prostatic disease more often than did relatives in control groups. Family aggregations of the disease suggest not only a genetic relationship but also that family members are exposed to the same environments and diets.

In an editorial, Kovi (16) stated that the mortality rate of prostate cancer correlates positively with consumption of fats, coffee, meats, milk, and sugar. A negative correlation has been shown with consumption of vitamin A, rich green and yellow vegetables, cereals, pulses, and rice.

Based on mortality data and dietary patterns, Wynder et al (17) postulated that ingestion of a diet high in fat is associated with an increased death rate from prostatic cancer. Blair and Fraumeni (18) reported an association between high fat intake

**Table**  
**Environmental Factors and Theories Suggested**  
**as Related Causes of Prostate Cancer in Black Men**

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Environmental Factors/Theories:
Urban/Rural
Socioeconomic status
Educational attainment
Occupation (Exposure to gas or exhaust)
Alcohol
Smoking
Other Environmental Factors:
Air pollution
SO <sub>3</sub>
Dustfall soiling
BPH
Cadmium and zinc
Sexual Factors/Practices:
Marital status
Age of first ejaculation
Frequency of coitus
Number of sexual partners
Utilization of prostitutes
Abstinence > 3 months
Intercourse without orgasm
Frequency of masturbation
Oral genital intercourse
Homosexual experience
Venereal disease

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and the incidence of the disease. On the other hand, Mettlin (19), studying dietary habits of blacks and whites, found data that do not support the hypothesis that increased intake of lipid-rich foods is related to the more frequent development of prostate cancer.

## Hormones

Much current evidence supports the concept that malignant prostatic disorders result from androgenic stimulation. The absence of prostatic cancer in castrated men, the responsiveness of prostatic cancer to hormone manipulation, and the evidence that androgenic steroids can induce prostatic cancer in animal models all suggest the important role of hormones in development of prostate cancer in men.

Because testosterone and its metabolite dihydrotestosterone are the principal hormones affecting growth of prostate epithelial tissue, Ross et al (20) proposed that the risk of prostate cancer is determined by the cumulative exposure to circulating testosterone. Evaluating black and white male students showed that mean testosterone levels in blacks were 15% higher and free testosterone levels were 13% higher than in whites. They suggested that the 15% difference in circulating testosterone levels could readily explain the twofold difference in prostate risk. When hormone levels of American blacks with prostate cancer were compared to that of Nigerians with prostate cancer, significantly higher levels of both testosterone and estrogens were seen in the Americans. Ahluwalia et al (21) concluded that differences in hormone profiles of the two populations were caused by factors other than genetic constitution alone.

Environmental factors in North America, which modify hormone metabolism, may be important in the etiology of this disease. There is a tenfold greater incidence of prostate cancer in black North Americans than in black South Africans. This fact supports the theory that life-style and dietary differences are significant factors. Studies have compared the effect of the African vegetarian diet and the typical Western diet on the excretion of androgens and estrogens. With the vegetarian diet, excretion of androgens and estrogens was decreased in black North American men, while use of the Western diet increased the excretion of estrogens and androgens in black South African men (22-25).

While the hormonal milieu and its modification by dietary factors apparently influence the developing prostate cancer, genetic factors may also be involved. Recent experiments have shown that the wild type P53 gene may have a role in suppressing prostatic tumorigenesis. Other studies have demonstrated the loss of chromosome 17P in nearly one-fifth of clinically resected tumors, suggesting a mechanism for inactivation of the P53 gene in human prostatic tumorigenesis. In vitro studies on human prostatic cancer cells by Isaacs et al (26) showed that the wild type P53 was inhibitory to DNA synthesis when compared to action of the mutant gene. P53 mutations can be detected in primary cancer cells as well as metastatic deposits, suggesting a functional role for P53 as a suppressor of prostatic tumorigenesis.

## Conclusions

Prostate cancer is now the leading cause of cancer deaths among American males, affecting one of every 11 men. For black American males, the incidence is 1 in 7. Because its incidence is increasing, there have been numerous efforts to identify epidemiological factors. A striking feature of the epidemiology of prostate cancer is the extreme range in incidence among racial and ethnic groups (50% greater in blacks than in whites and 3 to 4 times greater in blacks than in Chinese or Japanese). The reasons for such differences are unknown but are thought not to be entirely caused by genetic predisposition.

The study of external factors in high-risk patients, specifically black males, may lead to the discovery of genetic and environmental interactions that cause prostate cancer.

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