

Tobacco use and prostate cancer in Blacks and Whites in the United States

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Prostate cancer occurs more frequently in Blacks than Whites in the United States. A population-based case-control study which investigated the association between tobacco use and prostate cancer risk was carried out among 981 pathologically confirmed cases (479 Blacks, 502 Whites) of prostate cancer, diagnosed between 1 August 1986 and 30 April 1989, and 1,315 controls (594 Blacks, 721 Whites). Study subjects, aged 40 to 79 years, resided in Atlanta (GA), Detroit (MI), and 10 counties in New Jersey, geographic areas covered by three, population-based, cancer registries. No excesses in risk for prostate cancer were seen for former cigarette smokers, in Blacks (odds ratio [OR] = 1.1, 95 percent confidence interval [CI] = 0.7-1.5) and in Whites (OR = 1.2, CI = 0.9-1.6), or for current cigarette smokers, in Blacks (OR = 1.0, CI = 0.7-1.4) and in Whites (OR = 1.2, CI = 0.8-1.7). Increases in risk were noted for smokers of 40 or more cigarettes per day, among former (OR = 1.4, CI = 1.0-1.5) and current (OR = 1.5, CI = 1.0-2.4) smokers. Duration of cigarette use and cumulative amount of cigarette use (pack-years) were not associated with prostate cancer risk for Blacks or Whites. By age, only the youngest subjects, aged 40 to 59 years, showed excess risk associated with current (OR = 1.5, CI = 1.0-2.3) and former (OR = 1.7, CI = 1.1-2.6) use of cigarettes, but there were no consistent patterns in this group according to amount or duration of smoking. Risks also were not elevated for former or current users of pipes, cigars, or chewing tobacco, but the risk associated with current snuff use was OR = 5.5 (CI = 1.2-26.2). This subgroup finding may have been due to chance. The results of the present study may be consistent with a small excess risk for prostate cancer associated with tobacco use, but the lack of consistent findings in population subgroups and the lack of a clear dose-response relationship argue more strongly that no causal association exists. The data do not indicate that the Black-White difference in prostate cancer risk is related to tobacco use. *Cancer Causes and Control* 1994, 5, 221 - 226

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Introduction

Prostate cancer is the most frequently diagnosed cancer among men in the United States, and the second leading cause of cancer deaths. Incidence rates are substantially higher among Black (163.6 per 100,000 in

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1990) than White men (128.5 per 100,000).¹ Tobacco use is not generally recognized as an etiologic factor for prostate cancer,² but excess risks have been found in some epidemiologic studies,³⁻⁶ suggesting a possible link.⁷

Incidence rates of tobacco-related cancers, such as cancers of the lung, esophagus, larynx, and oral cavity are higher in Black than in White Americans.⁸ However, the contribution of tobacco use to the Black-White differences in occurrence of prostate cancer has not been examined previously in detail. We carried out a large study, which included similar numbers of Black and White prostate-cancer cases and a population-based control group, to examine the reasons for the large racial difference in risk for this disease including the possible role of tobacco use.

Materials and methods

This case-control study of prostate cancer was one component of a multi-center study of cancers of the esophagus, pancreas, prostate, and multiple myeloma in US Blacks and Whites. Study subjects resided in geographic areas covered by three, population-based, cancer registries: Atlanta, GA (the Georgia Center for Cancer Statistics); Detroit, MI (the Metropolitan Detroit Cancer Surveillance System); and 10 counties

of New Jersey (the New Jersey State Cancer Registry).

Cases for this study were men aged 40 to 79 years, identified from pathology and outpatient records at hospitals covered by these registries, newly diagnosed with pathologically confirmed prostate cancer, between 1 August 1986 and 30 April 1989. To ensure an adequate representation of subjects by race and age, we sampled varying proportions of cases for inclusion in the study from among the total number of cases identified in each age-race group. The planned sampling fractions ranged from 100 percent for those younger than age 55 to 20 percent for White males aged 65 to 74, and 17 percent for Black males aged 65 to 74 years.

Population controls were selected in the three geographic areas proportional to the expected age, gender, and race distribution of the combined cases for the four cancer sites. Population controls less than 65 years of age were selected at periodic intervals by random-digit dialing (RDD), using a two-step process involving identification of households with members eligible for study and then selection of potential controls to be contacted.⁹ Older controls were selected systematically (after a random start) from computerized records of the Health Care Financing Administration (HCFA) stratified by age (65-69, 70-74, 75-79), gender, and race (Black, White), for each geographic area.

In-person interviews were conducted with the cases

Table 1. Cigarette use and prostate cancer risk among former and current smokers, by usual number of cigarettes smoked: Atlanta (GA); Detroit (MI); New Jersey; 1986-89

Cigarette use	Black			White			Total	
	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b	OR ^c	(CI) ^b
Never used tobacco ^d	88/116	1.0	—	86/149	1.0	—	1.0	—
Former smokers								
Amount smoked (cigarettes per day)								
Any	189/199	1.1	(0.7-1.5)	243/319	1.2	(0.9-1.6)	1.2	(0.9-1.5)
< 10	45/55	0.9	(0.5-1.5)	21/21	1.6	(0.8-3.2)	1.1	(0.8-1.8)
10-19	41/39	1.2	(0.7-2.1)	46/61	1.2	(0.7-1.9)	1.2	(0.9-1.8)
20-39	74/77	1.1	(0.7-1.7)	103/157	1.0	(0.7-1.5)	1.1	(0.8-1.4)
40+	28/28	1.1	(0.6-2.0)	72/78	1.5	(1.0-2.2)	1.4	(1.0-1.9)
Current smokers								
Amount smoked (cigarettes per day)								
Any	161/221	1.0	(0.7-1.4)	116/177	1.2	(0.8-1.7)	1.1	(0.8-1.4)
< 10	23/32	0.8	(0.4-1.6)	4/7	0.8	(0.2-3.2)	0.9	(0.5-1.6)
10-19	47/59	1.1	(0.6-1.8)	13/29	0.8	(0.4-1.7)	1.0	(0.7-1.6)
20-39	72/115	0.8	(0.6-1.3)	72/104	1.2	(0.8-1.8)	1.0	(0.8-1.4)
40+	19/14	1.9	(0.9-4.2)	27/37	1.3	(0.7-2.4)	1.5	(1.0-2.4)

^a OR = odds ratio adjusted for age (40-49, 50-54, ... 70-74, 75+) and study site.

^b CI = 95% confidence interval.

^c OR = odds ratio adjusted for age, race, and study site.

^d Referent.

Table 2. Prostate cancer risk, by duration and cumulative amount of cigarette use: Atlanta (GA); Detroit (MI); New Jersey; 1986-89

Cigarette use	Black			White			Total	
	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b	OR ^c	(CI) ^b
Never used tobacco ^d	88/116	1.0	—	86/149	1.0	—	1.0	—
Duration (yrs)								
< 20	35/55	0.9	(0.5-1.5)	55/101	1.0	(0.6-1.5)	0.9	(0.7-1.3)
20-39	136/171	1.1	(0.8-1.6)	169/227	1.3	(0.9-1.9)	1.2	(1.0-1.6)
40+	171/180	1.0	(0.7-1.4)	133/153	1.2	(0.8-1.7)	1.1	(0.9-1.5)
Cumulative amount (pack-years)								
< 20	110/136	1.0	(0.7-1.6)	70/120	1.0	(0.7-1.6)	1.1	(0.8-1.4)
20-44	116/161	0.9	(0.6-1.3)	132/161	1.4	(1.0-2.0)	1.1	(0.9-1.5)
45+	115/108	1.2	(0.8-1.8)	155/200	1.2	(0.8-1.7)	1.2	(0.9-1.6)

^a OR = odds ratio adjusted for age (40-49, 50-54, ... 70-74, 75+) and study site.

^b CI = 95% confidence interval.

^c OR = odds ratio adjusted for age, race, and study site.

^d Referent.

and controls, usually in the subjects' homes. Prostate cancer cases and male controls were interviewed concerning demographics, dietary intake, tobacco use, occupational and medical history, sexual activity, and family history of cancer. Medical records of the cases were abstracted for diagnostic confirmation.

Odds ratios (OR) and approximate 95 percent confidence intervals (CI) for prostate cancer were calculated by logistic regression analysis.¹⁰ The ORs were adjusted for age (40-49, 50-54, ... 70-74, 75+) and study site and, where indicated, for race and other factors.

In total, 1,292 cases and 1,767 controls were identified for study. Interviews were obtained for 988 cases (76 percent; Black = 78 percent; White = 75 percent) and 1,336 controls (76 percent; Black = 77 percent, White = 74 percent). After adjustment for non-response in the initial phase of screening for eligibility among RDD contacts, the response rate in controls was 70 percent. Six cases and six controls were dropped from this analysis because of incomplete interviews. Sixteen subjects (15 controls, one case) were excluded because of a prior history of prostate cancer. The subjects for analysis consisted of 981 cases (479 Black, 502 White) and 1,315 controls (594 Black, 721 White).

Results

In Table 1, the ORs associated with cigarette use are shown relative to study subjects who never used tobacco. Risks for prostate cancer were not elevated for former or current cigarette smokers in either Blacks or Whites. When examined by amount, increases in risk

were noted only for smokers of 40 or more cigarettes per day, among former (OR = 1.4, CI = 1.0-1.5) and current (OR = 1.5, CI = 1.0-2.4) smokers. For Blacks, this association was present only for current smokers (OR = 1.9, CI = 0.9-4.2), while for Whites, smaller increases were seen for both former (OR = 1.5, CI = 1.0-2.2) and current smokers (OR = 1.3, CI = 0.7-2.4). Further statistical adjustment for income, education, and marital status did not alter the findings.

As shown in Table 2, duration of cigarette use and cumulative amount of cigarette use in pack-years (PY) were not associated with prostate cancer risk for either Blacks or Whites. ORs were elevated for some categories of cigarette use, but none were statistically significant and there was no trend of increased risk with increased exposure.

Cigarette-use patterns and prostate cancer risk also were examined for subjects according to age (40-59, 60-69, and 70 years or more). Overall risk was not increased for the two older age-groups, but for the youngest age group—*i.e.*, 40-59 years—risks were elevated for both former (OR = 1.7, CI = 1.1, 2.6) and current (OR = 1.5, CI = 1.0-2.3) cigarette smokers. There was, however, no clear pattern of increased risk with increased numbers of cigarettes smoked per day (Table 3), nor with increased duration of use (Table 4). For PYs of cigarette use (Table 4), the associated risks in the youngest age group were: OR = 1.6 (CI = 1.0-2.5) for less than 20 PYs; OR = 1.5 (CI = 1.0-2.4) for 20-44 PYs; and OR = 1.9 (CI = 1.2-3.0) for 45 or more PYs, respectively. The statistical test for trend, however, was not significant ($P > 0.05$). Further analyses in

this age-group for Blacks and Whites separately, for the three study areas, and for the age groups 40-49 and 50-59 years showed no trend of increased prostate cancer risk with increasing PYs of tobacco use.

Selected analyses were carried out with restriction of the case group to subjects with regional or distant disease. The risk patterns were not substantially different for this subgroup.

In Blacks and Whites, risks were not elevated for former or current users of pipe, cigar, or chewing tobacco

(Table 5). Prostate cancer risk was not associated with past snuff use, but increased risks were shown with current snuff use, in both Blacks and Whites. The risk associated with current snuff use, for Blacks and Whites combined, was OR = 5.5 (CI = 1.2-26.2).

Discussion

The overall results of this study suggest that tobacco use probably is not a risk factor for prostate cancer.

Table 3. Cigarette use and prostate cancer risk, by age, among former and current smokers: Atlanta (GA); Detroit (MI); New Jersey; 1986-89

Cigarette use cigarettes per day	Age: 40-59			Age: 60-69			Age: 70 +		
	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b
Never used tobacco ^c	45/134	1.0	—	58/73	1.0	—	71/58	1.0	—
Former smokers									
Any	119/177	1.7	(1.1-2.6)	155/168	1.2	(0.8-1.8)	158/173	0.7	(0.5-1.1)
1-10	13/20	1.6	(0.7-3.7)	31/15	2.3	(1.1-4.8)	22/41	0.5	(0.2-0.8)
10-19	24/33	2.0	(1.0-3.8)	30/31	1.2	(0.7-2.3)	33/36	0.8	(0.4-1.4)
20-39	54/80	1.7	(1.0-2.8)	59/89	0.9	(0.5-1.4)	64/65	0.8	(0.5-1.3)
40 +	27/43	1.6	(0.8-2.9)	35/32	1.5	(0.8-2.7)	38/31	1.0	(0.6-1.8)
Current smokers									
Any	107/206	1.5	(1.0-2.3)	102/115	1.1	(0.7-1.7)	68/77	0.7	(0.5-1.2)
1-10	8/17	1.2	(0.4-3.0)	9/7	1.4	(0.5-4.1)	10/15	0.5	(0.2-1.3)
10-19	22/39	1.8	(0.9-3.5)	22/29	0.9	(0.4-1.7)	16/20	0.7	(0.3-1.4)
20-39	55/116	1.3	(0.8-2.2)	57/67	1.1	(0.6-1.8)	32/36	0.7	(0.4-1.4)
40 +	22/33	1.9	(1.0-3.6)	14/12	1.5	(0.6-3.6)	10/6	1.4	(0.5-4.1)

^a OR = odds ratio adjusted for age (40-49, 50-54, ... 70-74, 75 +), study site, and race.

^b CI = 95% confidence interval.

^c Referent.

Table 4. Cigarette use and prostate cancer risk, by age, duration and cumulative amount smoked: Atlanta (GA); Detroit (MI); New Jersey; 1986-89

Cigarette use	Age: 40-59			Age: 60-69			Age: 70 +		
	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b	Cases/ controls	OR ^a	(CI) ^b
Never used tobacco ^c	45/134	1.0	—	58/73	1.0	—	71/58	1.0	—
Duration (yrs)									
< 20	46/85	1.5	(0.9-2.6)	23/41	0.7	(0.4-1.3)	21/30	0.6	(0.3-1.2)
20-39	145/234	1.8	(1.2-2.6)	100/98	1.3	(0.8-2.0)	60/66	0.7	(0.4-1.2)
40 +	33/48	1.4	(0.8-2.5)	132/135	1.2	(0.8-1.9)	139/150	0.8	(0.5-1.2)
Cumulative amount (pack-years)									
< 20	69/124	1.6	(1.0-2.5)	64/58	1.3	(0.8-2.2)	47/74	0.5	(0.3-0.9)
20-44	83/148	1.5	(1.0-2.4)	95/107	1.1	(0.7-1.7)	70/67	0.9	(0.5-1.4)
45 +	71/94	1.9	(1.2-3.0)	96/109	1.2	(0.7-1.8)	103/105	0.8	(0.5-1.3)

^a OR = odds ratio adjusted for age (40-49, 50-54, ... 70-74, 75 +), study site, and race.

^b CI = 95% confidence interval.

^c Referent.

Table 5. Prostate cancer risk by selected types of tobacco use: Atlanta (GA); Detroit (MI); New Jersey; 1986-89

Tobacco use	Black				White				Total	
	Cases	Controls	OR ^a	(CI) ^b	Cases	Controls	OR ^a	(CI) ^b	OR ^c	(CI) ^b
Never used tobacco ^d	88	116	1.0	—	86	149	1.0	—	1.0	—
Pipe										
Former	52	66	0.9	(0.6-1.5)	110	132	1.2	(0.8-1.7)	1.1	(0.8-1.4)
Current	15	14	1.3	(0.6-2.8)	22	28	1.4	(0.7-2.7)	1.4	(0.8-2.2)
Cigars										
Former	58	79	0.7	(0.5-1.2)	94	104	1.3	(0.8-1.9)	1.0	(0.7-1.3)
Current	18	25	0.8	(0.4-1.6)	29	41	1.0	(0.5-1.7)	0.9	(0.6-1.4)
Chewing tobacco										
Former	29	44	0.8	(0.4-1.4)	27	25	1.2	(0.6-2.3)	1.0	(0.6-1.5)
Current	8	19	0.4	(0.2-1.1)	6	14	0.5	(0.2-1.5)	0.5	(0.2-1.0)
Snuff										
Former	3	9	0.4	(0.1-1.4)	7	8	0.6	(0.2-2.1)	0.6	(0.3-1.4)
Current	7	2	4.7	(0.9-24.7)	3	0	*	—	5.5	(1.2-26.2)

^a OR = odds ratio adjusted for age (40-49, 50-54, ... 70-74, 75+) and study site.

^b CI = 95% confidence interval.

^c OR = odds ratio adjusted for age, race, and study site.

^d Referent.

* Undetermined.

The risks associated with any use of cigarettes were not elevated, for either Blacks or Whites. There was evidence for increased risk in subjects who usually smoked 40 or more cigarettes per day but there was no evidence for increased risk in smokers of less than this amount. Additionally, duration of cigarette use and cumulative amount of cigarette use were not associated with prostate cancer risk. Although 24 percent of cases and 30 percent of controls did not participate in this study, it is unlikely that these findings could have been due to differential tobacco use in the nonrespondents.

Increased risks for prostate cancer were found for men aged 40 to 59 years associated with both former and current cigarette use, but examination of usual daily amount smoked, duration of use, and cumulative amount of cigarettes smoked in this age group, showed no increase in risk with increase in exposure. In particular, the pattern of increased risk with usual use of 40 or more cigarettes per day, found in the total group, was not apparent in the age-specific analyses. The finding of an overall increase in risk for prostate cancer in younger men who smoked, but the lack of a dose-response, may indicate that smoking is associated with an unexamined risk-factor for prostate cancer. A possible selection bias for nonsmokers among younger controls may have occurred, due to differential non-response. Prostate cancer risk also was not associated with pipe or cigar use.

Most previous epidemiologic studies have not shown an association between tobacco use and pros-

tate cancer risk, as recently reviewed by Nomura and Kolonel.¹¹ Excesses were found in two recent cohort mortality studies of US Veterans⁴ and of members of the Lutheran Brotherhood,³ and in two case-control studies.^{5,6} Only one prior study, a case-control study in California (US),¹² examined risk associated with tobacco use according to race, finding no excess in Blacks (OR = 1.1) or Whites (OR = 0.9).

Users of chewing tobacco and former users of snuff showed no excess risk; however, a substantial increased risk was observed for current users of snuff. Given the number of comparisons considered, this may be a chance finding. Nitrosamines, polycyclic aromatic hydrocarbons, and radiation-emitting polonium are found among the numerous compounds in snuff and chewing tobacco. Smokeless tobacco use, and particularly snuff use, have been associated with cancers at sites of direct application in the oral cavity. Constituents of smokeless tobacco can enter the bloodstream, and some are excreted in urine.¹³ A previous study³ showed an excess risk for prostate cancer associated with use of smokeless tobacco, with the greatest risk among regular users. The risks associated specifically with chewing tobacco and snuff use, however, could not be separated, and a further study found no excess risk.⁴

A 1987 survey¹⁴ of tobacco use among US males born between 1921 and 1930—the time period which spans the median birth-year for our study population—showed that about 55 percent of both Black and White

males had ever used cigarettes compared with about 70 percent of both Black and White controls in this study. In the 1987 survey, 30 percent of Blacks and 20 percent of Whites in this age group reported current cigarette use, while in the current study 37 percent of Blacks and 25 percent of Whites reported current use. Our results, showing greater frequency of current and former tobacco use in both Blacks and Whites, may reflect the urban nature of the three geographic areas studied. Both the current study and the 1987 survey find a higher prevalence of current cigarette use in Blacks than Whites. Recency of tobacco use is a major predictor of risk for the tobacco-associated cancers,¹⁵ and probably contributes to the excess risk for several tobacco-associated cancers in Blacks, although, as reported in previous surveys,^{16,17} Blacks who smoked cigarettes reported smoking fewer cigarettes per day than was reported by Whites.

The results of the present study may be consistent with a small excess risk for prostate cancer associated with tobacco use, but the lack of consistent findings in population subgroups and the lack of a clear dose-response relationship argue more strongly that no causal association exists. The risk for prostate cancer is greater in US Blacks than Whites and Blacks may have greater (particularly recently) exposure to tobacco. The data from the present study, however, do not indicate that the Black-White difference in prostate cancer risk is related to tobacco use.

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References

1. Miller BA, Hayes RB, Potosky AL, Brawley O, Kaplan R. Prostate. In: Miller BA, Gloeckler Ries LA, Hankey BF, et al, eds. *Cancer Statistics Review: 1973-1990*. Bethesda, MD, USA: National Cancer Institute 1993, in press.
2. International Agency for Research on Cancer. *Tobacco Smoking*. Lyon, France: IARC, 1986; *IARC Monograph Eval Carcinog Risks Chem Humans*, Vol. 38.
3. Hsing AW, McLaughlin JK, Schuman LM, et al. Diet, tobacco use, and fatal prostate cancer: results from the Lutheran Brotherhood Cohort Study. *Cancer Res* 1990; 50: 6836-40.
4. Hsing AW, McLaughlin JK, Hrubec Z, Blot WJ, Fraumeni JF, Jr. Tobacco use and prostate cancer: 26-year follow-up of US Veterans. *Am J Epidemiol* 1991; 133: 437-41.
5. Honda GD, Bernstein L, Ross RK, et al. Vasectomy, cigarette smoking and age at first sexual intercourse as risk factors for prostate cancer in middle-aged men. *Br J Cancer* 1988; 57: 326-31.
6. Schuman LM, Mandel J, Blackard C, et al. Epidemiologic study of prostatic cancer: preliminary report. *Cancer Treat Rep* 1977; 61: 326-31.
7. Matzkin H, Soloway MS. Cigarette smoking: A review of possible associations with benign prostatic hyperplasia and prostate cancer. *Prostate* 1993; 22: 277-90.
8. Ries LAG, Hankey BF, Edwards BK. *Cancer Statistics Review: 1973-87*. Bethesda, MD, USA: National Cancer Institute, 1990.
9. Waksberg J. Sampling methods for random digit dialing. *J Am Stat Assoc* 1978; 73: 40-6.
10. Breslow NE, Day NE. *Statistical Methods in Cancer Research, Vol I. The Analysis of Case Control Studies*. Lyon, France: International Agency for Research on Cancer 1980; IARC Sci. Pub. No. 82.
11. Nomura AMY, Kolonel LN. Prostate cancer: A current perspective. *Epidemiol Rev* 1991; 13: 200-27.
12. Ross RK, Shimizu H, Paganini-Hill A, Honda G, Henderson BE. Case-control studies of prostate cancer in blacks and whites in southern California. *JNCI* 1987; 78: 869-74.
13. US Department of Health and Human Services. *The Health Consequences of Using Smokeless Tobacco*. Bethesda, MD, USA: DHHS, Public Health Service, National Cancer Institute, 1986; NIH Pub. No. 86-2874.
14. US Department of Health and Human Services. *Strategies to Control Tobacco Use in the United States: a Blueprint for Public Health Action in the 1990's*. Bethesda, MD, USA: DHHS, Public Health Service, National Cancer Institute, 1992; NIH Pub. No. 92-3316.
15. US Department of Health and Human Services. *The Health Benefits of Smoking Cessation. A Report of the Surgeon General, 1990*. Bethesda, MD, USA: DHHS, Public Health Service, Centers for Disease Control, 1990; DHHS Pub. No. (CDC) 90-8416.
16. Novotney TE, Warner KE, Kendrick JS, et al. Smoking by blacks and whites: Socioeconomic and demographic differences. *Am J Public Health* 1988; 78: 1187-9.
17. Kabat GC, Morabia A, Wynder EL. Comparison of smoking habits of blacks and whites in a case-control study. *Am J Public Health* 1991; 81: 1483-6.