Family History, Hispanic Ethnicity, and Prostate Cancer Risk

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Family history is known to be a prostate cancer (CaP) risk factor for non-Hispanic Whites (NHWs) and African Americans, but little data are available on the risk for Hispanics (Hs). This population-based case-control study used mailed surveys to assess the effects of ethnicity and family history of CaP on CaP risk in Hs and NHWs. Cases ($N=351$) were those identified by the New Mexico Tumor Registry as having been newly diagnosed with CaP from October 1, 1994 to October 31, 1995. Controls ($N=618$) were randomly selected and frequency-matched to cases by ethnicity and 5-year age groups. Multivariate analyses were conducted using conditional logistic regression. After controlling for age, education, and income in the models, positive family history increased risk for both Hispanics (H) (OR 2.7, 95% CI 1.5–4.7) and non-Hispanic Whites (NHW) (OR 2.0, 95% CI 1.3–3.1), suggesting that having a family history of CaP is a risk factor for both ethnic groups. (*Ethn Dis*. 2003; 13:233–239)

Key Words: Case-Control Studies, Family Health, Male, Prostatic Neoplasms/Epidemiology, Registries, Risk Factors

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Introduction

Although incidence rates have declined over the past 5 years, prostate cancer is still the second leading cause of cancer death and is the number one incident cancer for men in the United States.[1] In 2002, an estimated 189,000 American men will have been diagnosed with prostate cancer, and approximately 30,200 men will have died from the disease.[2] As in the rest of the United States, the prostate is a leading site for cancer incidence, and prostate cancer is a major cause of cancer mortality among New Mexican men.[3,4] Overall age-adjusted 1994–1998 prostate cancer incidence was 142.0 cases per 100,000 men for the United States, and 124.1 per 100,000 men for New Mexico.[5]

Although Hispanics (Hs) are the fastest growing ethnic population in the United States, national cancer data are often reported in terms of "White" and "Black," and are, therefore, of limited utility to analyses requiring data on H ethnicity. The New Mexico Tumor Registry (NMTR), a population-based cancer registry covering the state of New Mexico, and American Indians in Arizona, participates in the National Cancer Institute's Surveillance, Epidemiology, and End Results (SEER) program and is a unique resource in the state, allowing researchers to break down cancer incidence and mortality data in terms of H ethnicity. New Mexico's heterogeneous population consists primarily of 4 ethnic/racial groups including NHWs (50%), Hs (38%), American Indians (9%), and African Americans (2%).[4]

According to NMTR data, average annual age-adjusted prostate cancer incidence in New Mexico increased for Hs from 51.3 cases per 100,000 men in 1972, to 76.4 cases per 100,000 men in 1982, and to 125.0 per 100,000 men in 1992.[3,6] Since 1992, the incidence of prostate cancer has declined slightly for Hs, although rates remain higher than those reported during the 1970s. Prostate cancer mortality rates have also increased for H men in New Mexico, with the average annual age-adjusted mortality rate increasing from 17.1 deaths per 100,000 men in 1972, to 20.0 per 100,000 men in 1982, and to 22.6 per 100,000 men in 1992.[3,6] Several studies of NMTR data have suggested that ethnic differences found in prostate and colon cancer incidence and survival in New Mexico may be explained by late detection or treatment differentials related to healthcare access. These studies have also documented the need to identify and describe risk factors in the different racial/ethnic groups represented in New Mexico.[7,8]

The major causes of prostate cancer are not known, although several risk factors have been suggested. These include: age, having a family history of prostate cancer, being African-American, being employed within certain farming and chemical occupations in which hormonal changes due to chemical exposures may be present, consuming a high fat diet, and being genetically predisposed.[9–14] Having a family history and being African-American are 2 of the most accepted risk factors for the disease and are often used by physicians to assess the need for early screening (at 40 years of age) using the prostate specific antigen test (PSA).[14] A cross-sectional study,[15] several hospital-based studies,[14,16–22] and population based case-control studies[11–13,23–27] have all investigated the role of familial aggregation

and risk for prostate cancer, and found that family history of prostate cancer is associated with a 2- to 3-fold increased risk. Despite the fact that race/ethnicity and family history are strong risk factors for prostate cancer, there are no reports describing systematic research focusing on H men with family history of the disease. To date, research investigating the role of race/ethnicity in family aggregation of prostate cancer is scarce, and has not considered Hs, concentrating instead on African-American or Asian men.[25,28] Therefore, the unique composition of New Mexico's population, and the existence of the NMTR, present an opportunity to consider prostate cancer risk in relation to family history and ethnicity.

The purpose of this project was to assess the effects of family history and ethnicity on prostate cancer risk in H and NHW men. We examined the following hypotheses: 1) a positive family history of one or more first-degree relatives with prostate cancer increases risk of prostate cancer for both H and NHW men; 2) the relative risk of prostate cancer increases monotonically with the number of first-degree relatives diagnosed with the disease for both H and NHW men.

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METHODS

Study Population

New Mexico cases were selected through the NMTR for the Prostate Cancer Outcomes Study (PCOS). The PCOS was initiated by the National Cancer Institute in 1994 to measure practice patterns and assess health-related quality of life among men diagnosed with prostate cancer in the United States. Methods for this multi-site, longitudinal project are described elsewhere.[29–31] Potential cases included men diagnosed with prostate cancer (ICD-O code C61.9) from October 1, 1994 through October 31, 1995, as identified by the NMTR. The NMTR utilizes both patient self-report and the GUESS program (Generally Useful Ethnic Search System), which calculated probable ethnicity based on Hispanic surname, to identify possible H and NHW cancer patients. Although the program was used to identify a likely ethnicity for both cases and controls in this project, all analyses were based on self-reported ethnicity. Of 753 NHW NMTR patients, we randomly chose 294 (39.0%) to participate. A total of 246 (38.4%) participants were randomly selected from 640 H NMTR patients. American Indians were not included in this project. Therefore, the total number of potential cases identified for the study was 540. Of these, 50 (9.3%) were later found to be ineligible due to inappropriate diagnosis date, or a diagnosis of a primary cancer other than prostate cancer. Physicians refused to give us permission to contact 4 potential case subjects, and the physicians for 8 subjects determined that their patients did not have prostate cancer. Eleven (2.0%) cases died before we could contact them, and 27 (5.0%) responded to the questionnaire by saying that they had not been diagnosed with prostate cancer. Thus, 425 (78.7%) of the originally selected subjects were presumed eligible to be cases, of which 351 (82.6%) participated by responding to the first survey.

In addition to participating in the PCOS, the NMTR conducted a parallel study in order to identify and follow a population-based, matched cohort of men who were not diagnosed with prostate cancer. The objective of the parallel project was to validate the PCOS case questionnaire and tracking methods, and to gather family history information from the general population.[32] Controls were randomly selected from 2 sources: those under age 65 years were from the New Mexico Motor Vehicles Department (MVD) data files, and those over age 65 years were from the Health Care Finance Administration (HCFA) claims files. These men were frequency matched by ethnicity and 5-year age groups to the prostate cancer cases, and were selected based on the expectation that they would exhibit baseline health status similar to that of cases, excluding prostate cancer. The 1400 controls selected comprised 795 NHW men, and 605 H men. As with cases, we used the GUESS program to select controls based on probable ethnicity. Of the potential controls we selected, 265 (18.9%) were later found ineligible due to our inability to ascertain place of residence and preliminary contact information; 41 (2.9%) died prior to enrollment; and 14 (1.0%) were found ineligible for other reasons: incorrect gender assigned in the HCFA/MVD files, or not being New Mexico residents. Therefore, 1,080 (77.1%) subjects were presumed eligible to be controls, of which 618 (57.2%) participated by responding to the first survey.

Data Collection

A self-administered survey questionnaire was mailed to all sampled cases approximately 6 months after the initial diagnosis of prostate cancer, and was mailed to the frequency-matched controls during the same time period. The questionnaire included information on demographics (age, race/ethnicity, geographic region, marital status); socioeconomic factors (education level, employment status, insurance status, and in-

come level); health status (urinary, bowel, sexual function; selected scales from the Medical Outcomes Study short form health survey [SF-36]), clinical symptoms (urinary, systemic); co-morbidity; and family history of prostate cancer.[29,33] The survey questionnaire for controls consisted of the same items used for the PCOS case survey, but with most references to cancer removed, except for those relating to family history.

Survey Methods

The survey questionnaire was sent as "Priority Mail" to draw greater attention to the package, and to stimulate prompt replies. The packet consisted of a survey questionnaire, cover letter, contact form, and a self-addressed, postage-paid, return envelope. Mailings were scheduled by a computer program with internal system checks to ensure timeliness. Trained interviewers using pre-approved scripts conducted telephone follow-up calls. Telephone follow-up calls were also scheduled by a computer tracking system with internal checks, which identified the individuals to be called based on variables such as questionnaire mailing date, status codes, and previous call dispositions.

If a questionnaire was not returned in 1.5 weeks (10 business days) from the date of the initial mailing, several attempts were made, at different times of day, to contact the respondent by telephone. The primary objective of the first phone call was to prompt the respondent to complete and return the questionnaire. If the respondent had not completed the questionnaire or been interviewed by 3 months from the date the survey was first sent, and after at least 10 attempts were made to reach the respondent over a period of several weeks, we ended attempts to contact the respondent.

Statistical Analyses

Descriptive statistics were calculated for the following categorical variables: ethnicity/race, age, stage at diagnosis, education level, marital status, employment status, and income level. Chi-square tests were used to identify statistically significant differences between cases and controls, as well as between those subjects who did and did not know their family history of prostate cancer. A two-tailed P value of .05 was considered statistically significant. After considering results of the univariate analyses, as well as the current literature, the following variables were selected to be included in the multivariate models: 1) age (5-year age groups); 2) education level (<high school, >high school); and 3) income level (total house hold income, <$20,000 per year, >$20,000 per year). Odds ratios and 95% confidence intervals from conditional logistic regression were used to estimate the relative risk of prostate cancer associated with family history of prostate cancer for H and NHW men. The conditional logistic regression models were fit using the SAS PHREG procedure (SAS Institute, Cary, NC).

RESULTS

Although cases and controls were originally matched by ethnicity and 5-year age groups, we found that more controls than cases described themselves in the survey as being NHW. A significant difference was also observed in the age distribution of cases and controls, when stratified by five-year age groups (Table 1). A higher proportion of the cases were over the age of 65, though the mean ages were the same. Additionally, more controls than cases reported having more than a high school education, as well as having higher incomes, although these findings were not statistically significant. Controls also were more likely than cases to be employed at the time of the interview. The response rate for controls was 57.2%, and 82.6% for cases. The response rate for Hs was lower than for NHWs, for both cases and controls, with younger men being more likely to participate than older men.

Univariate analyses concerning family history of prostate cancer are summarized in Table 2. Cases (24%) were more likely than controls (15%) to report a family history of prostate cancer, and were also more likely not to know their family history of prostate cancer (30% vs 12%). Compared to cases, controls reported fewer total number of relatives diagnosed with prostate cancer, with 6% of cases having 2 or more positive relatives, vs 1% of controls.

Since almost one fifth of all subjects did not know their family's history of prostate cancer (18.6%), we decided to include these subjects in our analyses, though current research often excludes such cases. We then compared the responses of those who did and did not know their family history, stratified by case/control status, in order to explore potential biases. The only statistically significant difference observed among case respondents was that those who did not know their family history tended to be younger than those who did. Among controls, we detected a difference in family history knowledge when we considered marital status: controls who were married were more likely to know their family history than those who were unmarried.

After controlling for age, education, and income in multivariate models, we found that positive family history increased risk for both Hs (OR=2.7, 95% confidence interval [CI]: 1.5, 4.7) and NHWs (OR=2.0, 95% CI: 1.3, 3.1) (Table 3). Interestingly, the risk of prostate cancer increased for both H men (OR=2.3, 95% CI: 1.2, 4.3) and NHW men (OR=3.5, 95% CI: 2.2, 5.6) who did not know their family history, when compared to those who did. When the type of relative was considered, there was an increased risk of prostate cancer for H men with a positive father (OR=2.7, 95% CI: 1.1, 6.9) or brother (OR=3.1, 95% CI: 1.4, 6.9). Non-Hispanic White (NHW) men also

Table 1. Characteristics of cases and controls by ethnicity, age and stage at diagnosis, education, marital status, employment, and income, New Mexico, 1994–1995

	Cases (*N*=351)		Controls (*N*=618)		*P* value
Ethnicity					
Non-Hispanic White	189	53.8%	393	63.6%	.001
Hispanic	141	40.2%	212	34.3%	
Missing	21	6.0%	13	2.1%	
Age (years)					
<49	9	2.6%	10	1.6%	.001
50–64	135	38.5%	284	46.0%	
65–74	138	39.3%	205	33.2%	
75+	69	19.7%	119	19.3%	
Mean age (SD)	66.1	(9.0 years)	65.6	(9.2 years)	.406
Stage at diagnosis					
T1	59	16.8%	N/A	N/A	N/A
T1/2	125	35.6%	N/A	N/A	
T2	115	32.8%	N/A	N/A	
T3	4	1.1%	N/A	N/A	
T4	42	12.0%	N/A	N/A	
Unspecified	6	1.7%	N/A	N/A	
Education					
<High school	157	44.7%	262	42.4%	.154
>High school	174	49.6%	353	57.1%	
Missing	20	5.7%	3	0.5%	
Marital status					
Married	262	74.6%	482	78.0%	.001
Not married	68	19.4%	133	21.5%	
Missing/refused	21	6.0%	3	0.5%	
Employment					
Working	88	25.1%	238	38.5%	.001
Not working	241	68.7%	377	61.0%	
Missing/refused	22	6.3%	3	0.5%	
Yearly income					
<20 K	121	34.5%	184	29.8%	.130
>20 K	230	65.5%	434	70.2%	

Table 2. Frequencies of parents, siblings, and other family members with prostate cancer by case-control status, New Mexico, 1994–1995

	Cases (*N*=351)		Controls (*N*=618)		*P* value
Family history					
Unknown	107	30%	74	12%	.001
Negative history	160	46%	452	73%	
Positive history	84	24%	92	15%	
Relationship					
Father	36	10%	50	8%	.016
Brother	32	9%	31	5%	
Other	16	5%	11	2%	
Number of relatives					
None	248	71%	526	85%	.011
1 relative	56	16%	67	11%	
≥2 relatives	22	6%	8	1%	

demonstrated an increased risk when a positive father (OR=1.5, 95% CI: 0.8, 2.8) or brother (OR=2.1, 95% CI: 1.0, 4.3) was reported, although the associations were not as strong as for H men. We also found that risk of prostate cancer diagnosis for both Hs and NHWs increased 2-fold when comparing men with one positive relative to those with a negative family history, and the risk continued to increase 6- to 8-times for subjects with two or more positive relatives.

DISCUSSION

Although race/ethnicity is often considered in epidemiologic studies, research on risk factors for prostate cancer has primarily focused on NHW, Asian- and African-American men. Our study confirms previous findings associating a family history of prostate cancer with an increased risk of prostate cancer for NHWs. We also present new information that family history of prostate cancer is also a risk factor for H men. For both ethnic groups, not only was an increased risk of prostate cancer associated with a reported family history of prostate cancer, but also with the number of affected first degree relatives. In addition, we found that cases were more likely to be diagnosed if they had a positive first-degree relative (father or brother), compared to more distant relatives.

Several previous case-control projects have shown an increase in prostate cancer risk for men reporting a positive family history. Andersson et al examined associations between lifestyle factors and subsequent risk of prostate cancer in a population-based case-control study. Men with a father who had prostate cancer were at a 2-fold increased risk of prostate cancer, whereas those with an affected brother exhibited an approximately 5-fold greater risk.[27] Fincham et al also conducted a population-based case-control study of prostate cancer

Table 3. Prostate cancer risk for first-degree relatives of cases compared with relatives of controls by kinship and number of affected relatives, stratified by ethnicity, and adjusted for age, education, and income, New Mexico, 1994–1995

	Non-Hispanic White		Hispanic	
	OR*	95% CI†	OR*	95% CI†
Model 1: Family history				
Negative	1.0		1.0	
Positive	2.0	(1.3–3.1)	2.7	(1.5–4.7)
Unknown family history	3.5	(2.2–5.6)	2.3	(1.2–4.3)
Model 2: Relationship				
Negative	1.0		1.0	
Father	1.5	(0.8–2.8)	2.7	(1.1–6.9)
Brother	2.1	(1.0–4.3)	3.1	(1.4–6.9)
Other	3.6	(1.5–8.9)	1.8	(0.6–5.5)
Unknown family history	3.5	(2.3–5.6)	2.3	(1.3–4.3)
Model 3: Number of relatives				
None	1.0		1.0	
1 relative	2.2	(1.3–3.7)	2.3	(1.2–4.9)
>2 relatives	6.8	(2.0–23.6)	8.7	(2.6–28.2)
Unknown family history	4.0	(2.5–6.4)	2.7	(1.5–5.1)

* Odds ratio.
† 95% CI=95 percent confidence interval.

A positive familiy history of prostate cancer increased risk of the disease for both Hispanics and non-Hispanic Whites.

and found that factors significantly related to the risk of developing prostate cancer included ethnic group (British, Ukrainian), and family history.[11] A case-control study was performed by Steinberg et al to estimate the relative risk of developing prostate cancer for men with a positive family history. Men with an affected father or brother were twice as likely to develop prostate cancer as were men with no affected relatives. In addition, researchers observed a trend of increasing risk with increasing number of affected family members, such that men with 2 or 3 first-degree relatives affected had a 5- and 11-fold increased risk of developing prostate cancer, results which coincide with those presented in this paper.[19] Lesko et al reported similar results, with an increased risk for prostate cancer among men reporting a history of this cancer in either their fathers or brothers (OR=2.3, 95% CI: 1.7, 3.3). As is the case with this study, risk varied with the number of relatives affected, and by their relationship to the case.[13]

Our results are consistent with previous studies of prostate cancer and family history for other racial/ethnic groups. Whittemore et al conducted a population-based case-control study of familial aggregation of prostate cancer[25] among Blacks, Whites, and Asian Americans in the United States and Canada. Their results were similar to ours in that positive family history was associated with a statistically significant 2- to 3-fold increase in risk for each of the 3 ethnic groups. For all 3 ethnicities, the overall odds ratio associated with a family history, adjusted for age, was 2.5 (95% CI: 1.9,3.3). Hayes et al also compared African Americans to Whites in a population-based case-control study investigating the association of prostate cancer with family history of cancer.[26] Prostate cancer risk was significantly elevated among those reporting a history of prostate cancer in first-degree relatives (OR=3.2; 95% CI: 2.0, 5.0), with Blacks and Whites having similarly elevated risks. Overall, the odds ratios associated with history of prostate cancer in fathers and brothers were 2.5 (95% CI: 1.5, 4.2) and 5.3 (95% CI: 2.3, 12.5), respectively. Kolonel et al also considered race/ethnicity in a population-based case-control study in Hawaii concentrating on the possible role diet plays in the development of prostate cancer, and interviewed subjects from 5 different ethnic groups, including Japanese, Chinese, Filipino, Hawaiian, and Caucasian.[12]

Surprisingly, we found that subjects who did not know their family history status were also at increased risk for prostate cancer. Hispanic (H) men were at a 2-fold increased risk for prostate cancer when they did not know their family history, and NHW men had a 3-fold increased risk. There are several possible reasons for the large proportion of men who did not know their family's history of prostate cancer. In general, men may be reluctant to discuss their health problems with male relatives, especially a genito-urinary problem, due to the stigma surrounding symptoms.[34–36] Also, the PSA test has only become widely used during the last decade. Men who were screened more recently may have presumed that their older relatives did not have access to this screening test, and therefore have never raised the issue. We believe this is a fertile area for future research. Many physicians, when faced with a patient who does not know his family history, may assume that there is no increased risk, and therefore may not recommend screening for prostate cancer with the PSA test.

We recognize some important limitations of the study, several of which are due to the case-control design. As with other studies of this type, recall bias is always a concern when relying on self-reported information. Other cancer

family history projects have found that cases are more likely to remember and report a family member having had prostate cancer, which can lead to recall bias.[36–39] Our findings appear to describe a different phenomenon. Cases were actually more likely to report that they did not know their family history status for prostate cancer. Without access to data concerning recall accuracy of family history information for prostate cancer in the general population, we can only postulate about the reasons for these unusual results. It is possible that cases may have recalled conditions that their family members have had with symptoms similar to those of prostate cancer, including benign prostate hyperplasia. If so, the cases may have been more aware than controls that they could not distinguish between prostate cancer and benign prostate hyperplasia, whereas controls may have confused benign prostate hyperplasia with prostate cancer.

Selection bias may also be an issue. Those subjects with affected relatives may be more likely to be screened due to increased knowledge of the disease, therefore increasing their opportunity for diagnosis. Although over 900 men participated in the project, the sample size is still fairly small, especially when considering the question of increased risk with increasing number of affected relatives. In addition, response rates for cases and controls differed (82.6% and 57.2%, respectively).

In conclusion, our results provide strong evidence that family history of prostate cancer is a risk factor for prostate cancer for both NHW and H men. In addition, for both ethnicities considered in this research, men who do not know their family history of prostate cancer appear to be at increased risk for the disease, when compared to those with a negative family history. These findings have important implications for prostate cancer screening policies, and practical meaning for medical practitioners. We suggest that prostate cancer screening become more directed toward men at increased risk because of family history information. In addition, practitioners need to encourage their patients to discuss their medical history with relatives and in order to improve the assessment of prostate cancer risk.

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