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Prostate cancer incidence and survival in relation to education (United States)

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Abstract

Introduction: There are few data on prostate cancer incidence and survival in relation to socioeconomic status in the 1990s, after the introduction of prostate specific Antigen (PSA) testing.
Materials and methods: We studied the relation of education to prostate cancer incidence and survival in the Cancer Prevention Study II (CPS-II) Nutrition Cohort. Participants provided questionnaire data on diet, medical history, education, and PSA testing. We followed 72,449 men from 1992–1999 for cancer incidence (4279 incident cases), and through 2000 for survival (158 prostate cancer deaths among incident cases).
Results: Multivariate-adjusted rate ratios (RRs) were calculated using Cox proportional hazards models. Self-reported (PSA) tests were more common among the more highly educated. Men with at least a college education had a 15–19% higher prostate cancer incidence than those with a high school education or less, but this association was limited to localized cancers and was attenuated by adjustment for PSA testing. Survival analysis among incident prostate cancer cases adjusted for stage and grade at diagnosis showed much lower prostate cancer mortality for men with at least a high school education compared to those with less than a high school education (RR = 0.49, 95% CI = 0.32–0.76).
Conclusions: This study suggests that higher education is associated with slightly increased incidence of prostate cancer, at least partly due to greater use of PSA screening and a greater detection of localized tumors among more highly educated men. The much lower survival rates from prostate cancer among those with less than a high school education cannot be explained by available data and may reflect disparities in treatment. In 1999, 27% of US males over age 55 and older had less than a high school education.

Introduction

Prostate cancer is the most common incident cancer among US men and is second only to lung cancer for cancer deaths [1, 2]. Prostate cancer incidence increased sharply between 1988 and 1992, and then decreased from 1992 to 1995, due to the widespread introduction of prostate specific antigen (PSA) testing in the late 1980s [2]. Virtually all of the continuing gradual increase in incidence during the late 1990s represents localized cancers, presumably detected via PSA screening [3]. The incidence of metastatic cancer has decreased, a phenomenon also presumably due to screening [3]. Death rates from prostate cancer in the US decreased by 4.0% per year from 1994 to 2000 [1], presumably due to earlier diagnosis and/or improved treatment.

Limited evidence suggests that the PSA test is more widely used by men of higher socioeconomic status (SES) [4, 5]. Higher SES was associated with higher prostate cancer incidence in the only published study on this subject after the introduction of the PSA test in 1988–1990 [6]; higher SES groups had higher incidence of localized tumors, but lower rates of distant cancer. Other studies have also reported that higher SES groups have lower stage at presentation [7–10].

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The only published study found by the authors regarding survival among men with prostate cancer in relation to SES considered the time period 1977–1981 (before PSA testing) and reported higher prostate cancer survival associated with higher SES groups, even after adjustment for stage and race [11].

We have further investigated whether prostate cancer incidence and survival were related to level of education in a cohort of 86,000 men enrolled in the American Cancer Society (ACS) Nutrition cohort, which has been followed for incident cases from 1992 to 1999, and for deaths from 1992 to 2000. Given the prior evidence of increased screening, lower stage at diagnosis, and increased incidence among men with higher SES [4–10], we hypothesized that higher education might be associated with higher prostate cancer incidence, and that any such trend in incidence would be attenuated by adjustment for PSA testing. Given the prior study about better survival for higher SES men [11], we also hypothesized that higher education would be associated with better cancer survival even after adjustment for stage at diagnosis.

Methods

We studied the ACS Cancer Prevention Study II (CPS-II) Nutrition Cohort (hereafter called the Nutrition Cohort), which includes 86,404 men enrolled in 1992 at ages 50–74 [12], and residing in 21 states with cancer registries. The Nutrition Cohort was established to investigate the relation between diet and other lifestyle factors and the risk of both cancer incidence and mortality. The Nutrition Cohort is a sub-group of the CPS-II cohort, which was enrolled originally in 1982. This cohort was assembled by volunteers and has a higher SES than the US population [12].

Participants in the Nutrition cohort completed a 10-page self-administered mailed questionnaire in 1992 that included demographic, medical, behavioral, environmental, occupational, and dietary factors. A follow-up questionnaire was sent to cohort members in 1997 and a second one in 1999 to update information and to ascertain newly diagnosed cancers. For living cohort members, the response rate was close to 91% for both questionnaires [12].

We used education as our SES variable. No data on income were available. We also considered occupation as our SES variable but occupational data were limited but it added little to the model including education.

We excluded (in order) from this analysis: men who were lost-to-follow-up from baseline in 1992 through August 31, 1999 (n = 4071), those who reported prevalent prostate cancer (n = 3582), or other cancer (except non-melanoma skin cancer) (n = 5376), at baseline, and those whose self-reported prostate cancer could not be verified (n = 445). Also excluded were men with missing education (n = 481). After these exclusions, the analytic cohort consisted of 72,449 men: a total of 4279 incident prostate cancer cases were identified in this cohort from baseline through August 31, 1999, and 158 prostate cancer deaths occurred among these cases through December 31, 2000.

Incident cases of prostate cancer were identified initially through a self-report of cancer on the 1997–1998 or 1999–2000 follow-up questionnaires and subsequently verified by medical records (n = 3291) or from linkage with state cancer registries (n = 790). Previous work linking cohort members to state cancer registries indicated that the ability of our respondents to accurately report a past diagnosis of cancer is high (sensitivity = 0.93, specificity $>$ 0.99 for report of any cancer) [13]. An additional 93 prostate cancer cases were also identified via the underlying cause of death on a death certificate through December 31, 1999 among cohort members who did not report cancer at enrollment [12]. Seventy of these 93 deaths were subsequently verified via cancer registries or medical records. The remaining 23 decedent cases were included and were assigned a diagnosis date 2.2 years prior to death, the median time between diagnosis and death for other - cancer deaths with known diagnosis date. Finally, 105 cases of prostate cancer were not reported initially as prostate cancer but were identified during confirmation of another reported cancer.

The Nutrition Cohort has been followed for mortality through December 31, 2000 [14]. We conducted survival analyses among incident prostate cancer cases to determine the relationship between education and survival. In this analysis the outcome was death from prostate cancer (n = 158). Follow-up time began at the time of diagnosis and we adjusted for stage and grade at diagnosis.

We used Cox proportional hazards modeling (SAS PHREG [15]) to examine the association between educational attainment and prostate cancer incidence and survival while adjusting for other potential risk factors. Level of education was categorized as less than high school, high school, some college, college graduate, or graduate school. Tests for trends in Cox regression were done by testing the significance of a continuous variable for education, created by assigning scores of 8, 12, 14, 16, and 18 to less than high school education, high school education, some college, college degree, and post-graduate education, respectively. Other tests for trend by education were done via contingency table

analyses and the Mantel extension test [16]. Rates by education directly standardized for age were calculated using the entire Nutrition Cohort as the standard.

Cox models used follow-up time as the time variable, and age at enrollment (single years) was used as a stratification variable (separate models for each age). Age and race are the most important predictors of prostate cancer. While only 1% of the Nutrition Cohort is black, limiting separate analyses by race, we did include a variable for race in all models (white, black, other). Other covariates in the incidence analysis included family history (first degree relatives), quintile of saturated fat intake, marital status (married, not-married, missing) and PSA testing. We tested a variable for body mass index but it was not a predictor of prostate cancer in this cohort and was not included in final models. The proportional hazards assumption for the effect of education was tested (via an interaction term between time and education) and found to hold ($p = 0.87$).

The information on PSA testing was collected in the 1997 follow-up questionnaire and did not distinguish whether PSA testing was conducted for screening or subsequent to a diagnosis. The 1997 questionnaire asked about PSA testing for biannual periods (before 1992, 1992–93, 1994–95, 1995–96, 1997–98). Thus we did not have prospectively collected data on the PSA testing prior to diagnosis for men who developed prostate cancer between 1992 and 1997 (n = 3198, 75% of cases). We therefore created a time-dependent variable for a PSA test presumably done for screening purposes (rather than after diagnosis). For cases (and their corresponding time-matched risk sets in Cox regression) occurring in 1992–1997, this variable (PSA test yes/no) was based on reported PSA testing before 1992 (PSA test = yes if a PSA test was taken before 1992), taken to be the best indicator of pre-diagnostic PSA screening. Our assumption was that 'early adopters', those who had PSA screening before 1992, were more likely to continue to receive PSA screening. For cases (n = 1081, 25% of cases) and risk sets occurring after the 1997 questionnaire, the variable for PSA test was considered 'yes' if any test was reported in the period 1992–1997. We also created a variable for 'missing PSA information,' as 18% of subjects were missing data on PSA testing. We conducted some additional analyses in which missing PSA information was considered equivalent to 'no PSA test'. These analyses were prompted by prior observation of a pattern of missing information for suspected negative responses for other variables in the questionnaire.

Adjustment for PSA testing was not possible in survival analyses because data on this variable derived from the 1997 questionnaire and were not available for the majority of cases who eventually died from prostate cancer.

Information on stage and grade for incident prostate cancer cases, used in survival analyses, was provided by medical records and registries. Stage was divided into localized (intracapsular involvement or invasion into but not beyond capsule), regional (extracapsular extension and invasion into ureter, rectum, regional lymph nodes), or distant (distant lymph nodes, metastases). Staging of prostate cancer can be complex, because the accuracy of staging may depend which treatment is chosen (*e.g.*, cancer in lymph nodes may be discovered during surgery). Grade was categorized into well, moderately, and poorly differentiated. In controlling for grade at diagnosis in survival analysis, we combined well and moderately differentiated into one category (Gleason's score 1–7) due to the small number of deaths in the well-differentiated group. Stage and grade were missing for 4 and 7% of all cases respectively, and 11 and 30% of decedents, and we created separate categories for 'missing' for both these variables.

Results

Table 1 presents data on self-reported PSA testing prior to 1992 and during the period 1992–1997 in relation to level of education (age-adjusted). Men with more education were more likely to report PSA testing. The percentage of men reporting PSA test before 1992 increased from 12% for those with less than high school education to 24% for men who had been to graduate school. The corresponding percentages were 51 and 74% for PSA testing between 1992 and 1997. There was a relatively large amount of missing data (18%) for tests before 1992 or between 1992 and 1997, and missing data were more common for those with less education. Those with missing PSA data were included in the analysis and were assigned to a category of 'missing', or assigned to the category of 'no PSA test' in supplementary analyses.

Table 1 indicates that the percentage of Nutrition Cohort members with less than a high school education was 8%. By comparison, in US males 55 and over in 1999 (approximately the age of the Nutrition cohort), the percentage of the population with less than a high school education was 26% [17]. Similarly, in the cohort those with at least a college education represented 47%, while the corresponding figure in the male US population age 55 or older was 27%.

Table 2 shows that men with at least a college education had a 15–19% higher incidence of prostate cancer than men with a high school education or less (trend test between education and incidence, $p = 0.0001$).

Table 1. Self-reported PSA testing by level of education in the Cancer Prevention Study II Nutrition Cohort 1992–1999[a]

	Education attainment					
	< High school	High school	Some college	College degree	Graduate school	Total
PSA test before 1992	743 (12%)	2194 (16%)	3439 (18%)	3699 (24%)	4374 (24%)	14,449 (20%)
No PSA test before 1992	3705 (62%)	8990 (64%)	11,772 (63%)	9624 (61%)	11,156 (62%)	45,247 (62%)
Missing data on PSA test before 1992	1553 (26%)	2727 (20%)	3591 (19%)	2329 (15%)	2553 (14%)	12,753 (18%)
PSA test 1992–1997	3052 (51%)	8565 (61%)	12,267 (65%)	11,502 (73%)	13,453 (74%)	48,839 (67%)
No PSA test 1992–1997	1396 (23%)	21619 (16%)	2944 (16%)	1821 (12%)	2077 (12%)	10,857 (15%)
Missing data on PSA test 1992–1997	1553 (26%)	2727 (20%)	3591 (19%)	2329 (15%)	2553 (14%)	12,753 (18%)

[a] Data on PSA testing came from the 1997 questionnaire, and was restricted to subjects who survived through 1997.

Table 2. Prostate cancer incidence by education, Cancer Prevention Study II Nutrition Cohort 1992–1999

	# Cases	Rate (per 100,000)[a]	RR (95% CI)[b]	RR (95% CI)[c]	RR (95%CI)[d]
< High school	338	942	1.0	1.0	1.0
High school grad	737	913	0.97 (0.85–1.11)	0.98 (0.86–1.11)	0.95 (0.83–1.08)
Some college	1086	1002	1.06 (0.94–1.20)	1.07 (0.95–1.21)	1.03 (0.91–1.16)
College grad	1008	1117	1.18 (1.04–1.34)	1.19 (1.05–1.35)	1.11 (0.98–1.26)
Graduate School	1110	1069	1.15 (1.02–1.30)	1.15 (1.01–1.30)	1.07 (0.95–1.22)
		p trend < 0.0001	p trend $= 0.0001$	p trend $= 0.01$	
−2 log likelihood		67,923	67,597	67,385	

[a] Directly standardized for age, standard is men in Nutrition cohort.
[b] Adjusted for age and race.
[c] Adjusted for age, race, family history of prostate cancer, quintiles of saturated fat intake, and marital status.
[d] Adjusted for age, race, family history of prostate cancer, quintiles of saturated fat intake, marital status, and PSA testing.

However, the positive relationship between education and prostate cancer incidence was lessened by control for PSA testing (trend test, $p = 0.01$). Additional analyses considering 'missing PSA' to be 'no PSA test' resulting in little change in estimates and are not shown.

Table 3 shows the age-adjusted stage-at-diagnosis by education. Men with higher education have fewer regional/distant cancers at diagnosis (p trend $= 0.006$). Table 4 shows incidence analyses for early and late-stage cancers separately. The trend of higher incidence rates for those with more education was limited to localized cancers, and again was somewhat lessened by adjustment for PSA testing. Conversely, an inverse relationship was seen between education and the incidence of regional or distant prostate cancer.

Table 5 shows results of survival analysis, which assesses the prostate cancer mortality among incident cases. There was a strong trend ($p < 0.0001$) of increased survival among those with higher education, without adjustment for stage and grade. This trend ($p = 0.002$) persisted after adjustment for stage and grade, both of which were important independent predictors for survival (change in log likelihood with their inclusion, 307, 5 df, $p < 0.0001$). Again, the trend was primarily due to the lower death rates among men with at least a high school education compared to those

Table 3. Stage at diagnosis by education for incident prostate cancer, Cancer Prevention Study II Nutrition Cohort 1992–1999

	< High school	High school	Some college	College	Graduate school
Localized	258 (81%)	588 (84%)	902 (86%)	817 (85%)	935 (87%)
Regional/distant	59 (19%)	114 (16%)	147 (14%)	147 (15%)	139 (13%)[a]
Total	317	702	1049	964	1074

[a] Age adjusted linear trend, $p = 0.006$.

Table 4. Prostate cancer incidence by stage at diagnosis and level of education, Cancer Prevention Study II Nutrition Cohort, 1992–1999

	# Cases	Rate (per 100,000)[a]	RR (95% CI)[b]	RR (95%CI)[c]	RR (95%CI)[d]
Localized cancer[e]					
< High School	258	718	1.0	1.0	1.0
High school grad	588	734	1.02 (0.88–1.18)	1.02 (0.88–1.19)	0.99 (0.85–1.14)
Some college	902	837	1.16 (1.01–1.33)	1.16 (1.01–1.34)	1.11 (0.97–1.28)
College grad	817	915	1.26 (1.09–1.44)	1.26 (1.10–1.46)	1.17 (1.02–1.35)
Graduate School	935	910	1.28 (1.11–1.47)	1.27 (1.10–1.46)	1.17 (1.02–1.35)
			p trend < 0.0001	p trend < 0.0001	p trend = 0.0005
Regional/distant cancer[e]					
< High school	59	172	1.0	1.0	1.0
High school grad	114	141	0.83 (0.61–1.14)	0.83 (0.60–1.13)	0.82 (0.60–1.12)
Some college	147	138	0.80 (0.59–1.09)	0.81 (0.60–1.09)	0.79 (0.59–1.08)
College grad	147	162	0.95 (0.70–1.29)	0.95 (0.70–1.29)	0.93 (0.68–1.26)
Graduate school	139	132	0.79 (0.58–1.07)	0.78 (0.57–1.06)	0.76 (0.56–1.04)
			P trend = 0.38	p trend = 0.35	p trend = *0.27*

[a] Directly standardized for age, standard is men in Nutrition cohort.
[b] Adjusted for age and race.
[c] Adjusted for age, race, family history of prostate cancer, quintiles of saturated fat intake, and marital status.
[d] Adjusted for age, race, family history of prostate cancer, quintiles of saturated fat intake, marital status, and PSA testing.
[e] Excludes cancers missing stage (4%).

Table 5. Relative risk of death from prostate cancer among prostate cancer cases by level of education, Cancer Prevention Study II Nutrition Cohort 1992–2000

	# Deaths	Case fatality rate	RR (95% CI)[a]	RR (95% CI)[b]
< High school	31	9.2%	1.0	1.0
≥ High school	127	3.2%	0.39 (0.26–0.59)	0.49 (0.32–0.76)
High school grad	28	3.8%	0.47 (0.28–0.79)	0.51 (0.30–0.89)
Some college	41	3.8%	0.43 (0.26–0.69)	0.55 (0.33–0.92)
College grad	32	3.2%	0.39 (0.23–0.66)	0.48 (0.28–0.83)
Graduate school	26	2.3%	0.29 (0.17–0.50)	0.41 (0.23–0.71)
			p trend < 0.0001	p trend = 0.002

[a] Adjusted for age and race.
[b] Adjusted for age, race, stage, and grade.

who had not completed high school (RR = 0.49, 95% CI = 0.32–0.76, fully adjusted model).

Table 6 presents the relative risks of death by education from the survival analysis stratified by stage of disease. The inverse relationship between education and death from prostate cancer was stronger for localized cancer ($p = 0.002$) than for regional/distant cancers ($p = 0.17$). Trends were again dominated by the low mortality of those with a high school education or greater compared to those with less than a high school education (RR = 0.31, 95% CI = 0.16–0.59 for localized cancer, RR = 0.73, 95% CI = 0.37–1.44 for regional/distant cancer).

We also conducted a supplemental analysis to investigate whether the survival analysis trend among cases with localized tumors might be due to bias from the preferential presence of cases with indolent cancers, which would never result in death, among those with higher education who had had more PSA testing. We first assumed that 29% of the localized non-fatal cancers were indolent cancers detected by PSA screening [18], and then made the assumption that these occurred among educational groups in proportion to the frequency of PSA screening prior to 1992 (Table 1), resulting in relative risks of indolent PSA-detected cancers of 1.0, 1.2, 1.5, 1.9, and 1.9 by increasing education. We then randomly deleted these hypothetically indolent cancers from the non-fatal cases among each education group (18, 22, 27, 35, and 35% of non-fatal localized cancers for less than high school,

Table 6. Relative risk of death from prostate cancer among prostate cancer cases by level of education stratified on stage at diagnosis, Cancer Prevention Study II Nutrition Cohort, 1992–2000

	Localized			Regional or distant		
	# Deaths	Case fatality rate[a]	RR (95% CI)[b]	# Deaths	Case-fatality Rate[a]	RR (95% CI)[b]
< High school	14	5.4%	1.0	12	20.3%	1.0
High school grad	11	1.9%	0.41 (018–0.92)	13	11.4%	0.73 (0.31–1.70)
Some college	14	1.6%	0.29 (0.13–0.63)	24	16.3%	1.02 (0.48–2.21)
College grad	12	1.5%	0.31 (0.14–0.72)	16	10.9%	0.60 (0.26–1.38)
Graduate school	12	1.3%	0.27 (0.12–0.60)	13	9.4%	0.59 (0.26–1.37)
			p trend = 0.002			p trend = 0.17

[a] Percentage incident cases dying from prostate cancer 1992–2000.
[b] Adjusted for age and race.

high school, some college, college and post-college education, respectively). There remained a significant trend of lower mortality with increased education ($p = 0.04$), again due primarily to the high mortality of the least education (rate ratio of all groups vs least educated, 0.37, 95% CI = 0.20–0.70).

Discussion

In our study men with higher education adopted PSA screening tests earlier and continued to have more PSA testing in the 1990s than men with lower education. This finding is consistent with two US reports that PSA screening was significantly more frequent among those with higher education [4, 5].

The incidence of prostate cancer was 15–19% higher among men with college education when PSA testing was not considered, compared to men with less than a high school education. In this generally health-conscious cohort, this trend probably at least partly reflects increased PSA testing by men with higher education. There are two pieces of evidence supporting this line of reasoning. First, the trend is diminished (although still present) after adjustment for PSA testing. Our data on past PSA testing were self-reported and involved inevitable misclassification (one study reports a 74% sensitivity and 65% specificity for self-report of PSA screening [19]). Furthermore, our assumptions about the timing of PSA tests and whether they were done as screening tests or post-diagnosis were another source of misclassification. It is possible that the education-incidence trend would have been further attenuated or eliminated if we had had more accurate information on PSA testing. These are the only published data, to our knowledge, that have been able to adjust for PSA testing in analyses of prostate cancer incidence differences by education. Second, separate examination of incidence by stage at diagnosis revealed that the positive trend with education was apparent only for localized cancer, which would be expected to be most affected by PSA testing.

Our findings regarding incidence and education parallel those of the only other published data on prostate cancer incidence in relation to SES in the 1990s, which was a study of prostate cancer in Los Angeles related to SES defined by area of residence [6]. That study too showed a similar positive gradient of increasing incidence with higher SES for localized cancer and weak or little trend for distant cancer. No data were available on PSA testing in the Los Angeles study.

Survival analysis among the cases has the advantage of being able to adjust for stage at diagnosis, which lessens the effect of differential screening between education groups and focuses on possible differences in treatment. We found the lowest survival for those with the lowest education. This finding is consistent with one earlier study, conducted before PSA screening, which found significantly better survival for higher SES groups after adjustment for race and stage at diagnosis [11].

The trend between survival and education was strongest among those with localized cancer. It may be that treatment differences between groups with different education, which may affect mortality, are less likely to be important among those first diagnosed with regional/distant cancers than among those diagnosed with localized cancers. While there are no clearly optimal treatments for localized prostate cancer [20], it is possible that the quality of care is worse for those with lower SES regardless of which treatment is chosen.

Some caution must be exercised in extrapolating from our results to the general population, as the Nutrition Cohort is more highly educated than the US population, and even within education categories Nutrition Cohort members may be more highly educated than the general US population with the same level of education.

In conclusion, results from this study suggest that higher education is associated with increased incidence of prostate cancer at least partly due to higher use of PSA screening, with increased incidence confined to localized tumors. Men diagnosed with prostate cancer who had less than a high school education had twice the prostate cancer mortality rate as cases with at least a high school education, even after controlling for state and grade at diagnosis, suggesting differences in treatment by education. In 1999 27% of US males age 55 and older had less than a high school education [17]. Future studies might explore further whether increased PSA testing is responsible for increased incidence of prostate cancer among those with higher SES, and how quality of care might differ between different SES groups.

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