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Impact of individual and neighborhood factors on disparities in prostate cancer survival

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Abstract

Background—We addressed the hypothesis that individual-level factors act jointly with social and built environment factors to influence overall survival for men with prostate cancer and contribute to racial/ethnic and socioeconomic (SES) survival disparities.

Methods—We analyzed multi-level data, combining (1) individual-level data from the California Collaborative Prostate Cancer Study, a population-based study of non-Hispanic White (NHW), Hispanic, and African American prostate cancer cases (N = 1800) diagnosed from 1997 to 2003, with (2) data on neighborhood SES (nSES) and social and built environment factors from the California Neighborhoods Data System, and (3) data on tumor characteristics, treatment and follow-up through 2009 from the California Cancer Registry. Multivariable, stage-stratified Cox proportional hazards regression models with cluster adjustments were used to assess education and nSES main and joint effects on overall survival, before and after adjustment for social and built environment factors.

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Author contributions

MCD interpreted the data and led the manuscript writing. CWS designed analyses and interpreted the data. JK, JY, and AH conducted analyses. SSM, MC, and DON interpreted the data and conducted critical review of the manuscript. SAI and EMJ led site-specific case-control studies, interpreted the data and conducted critical review of the manuscript. SLG originated the study, interpreted the data, and assisted with manuscript writing.

Conflict of interest statement

None.

Appendix A. Supplementary data

Supplementary data associated with this article can be found, in the online version, at https://doi.org/10.1016/j.canep.2018.01.003.

Results—African American men had worse survival than NHW men, which was attenuated by nSES. Increased risk of death was associated with residence in lower SES neighborhoods (quintile 1 (lowest nSES) vs. 5: HR = 1.56, 95% CI: 1.11–2.19) and lower education (< high school vs. college: HR = 1.32, 95% CI: 1.05–1.67), and a joint association of low education and low nSES was observed. Adjustment for behavioral, hospital, and restaurant and food environment characteristics only slightly attenuated these associations between SES and survival.

Conclusion—Both individual- and contextual-level SES influence overall survival of men with prostate cancer. Additional research is needed to identify the mechanisms underlying these robust associations.

Keywords

Prostate cancer; Survival; Neighborhood socioeconomic status; Education; Race/ethnicity; Disparities; Built environment

1. Introduction

In the United States (U.S.), African American (AA) men have higher incidence and mortality of prostate cancer, and worse survival compared to White men [1–5]. Many studies have reported independent associations between lower individual- or contextual-level socioeconomic status (SES) and worse survival among men diagnosed with prostate cancer [3–11], and some suggest that contextual-level SES accounts for racial/ethnic disparities in survival [4–6].

Contextual-level SES captures features of the neighborhood environment over-and-above individual-level characteristics of neighborhood residents [12,13]. Negative health consequences of residing in a low SES neighborhood may be determined by aspects of both the social environment (e.g., crowding, ethnic enclave status) and the built environment (e.g., street connectivity, food environment) through negative health behaviors, health-care access, or chronic stress, or cultural factors [8,14–34]. Certain aspects of the neighborhood social environment have been shown to be associated with cancer survival for some racial/ethnic groups and select cancer sites [35], including prostate cancer [32]. Ultimately, survival disparities likely result from complex relationships between these multi-level factors, such that new insights in our understanding of SES survival disparities will require a multilevel approach.

To address the persistent racial/ethnic and SES survival disparities among men with prostate cancer, we conducted the Neighborhoods and Prostate Cancer (NAPC) study that analyzed multi-level data from two multiethnic, population-based case-control studies, combining individual-level data on sociodemographics, family history of prostate cancer, clinical history, health behaviors and tumor characteristics with hospital- and neighborhood-level data on SES and social and built environment factors. Our objective was to assess 1) the contribution of individual- and neighborhood-level SES on racial/ethnic differences in survival after prostate cancer diagnosis, 2) the independent and joint effects of individual- and neighborhood-level SES on survival and, 3) the extent to which specific neighborhood factors contribute to SES survival disparities.

2. Methods

2.1. Study population

The NAPC study, approved by the Institutional Review Board of the Cancer Prevention Institute of California, comprises prostate cancer cases and controls who participated in two population-based case-control studies among AA and non-Hispanic White (NHW) men from the San Francisco Bay Area and AA, Hispanic, and NHW men from Los Angeles county [36–38]. Cases were identified through the population-based cancer registries in the Greater San Francisco Bay Area and Los Angeles County, both part of the California Cancer Registry (CCR). A common questionnaire was utilized at both sites and the survey data were pooled and merged with CCR data and neighborhood data from the California Neighborhoods Data System (CNDS) [39]. Only prostate cancer cases were included in this survival analysis.

Eligible cases from the San Francisco Bay Area site included AA and NHW men aged 40–79 years with a first primary localized prostate cancer diagnosed between October 1, 1997 and September 30, 1998; NHW men with a first primary advanced prostate cancer diagnosed between July 1, 1997 and February 29, 2000; and AA men with a first primary advanced prostate cancer diagnosed between July 1, 1997 and December 31, 2000. The site included random samples of localized cases (60% of AAs, 15% of NHWs) and all cases with advanced prostate cancer [37]. A total of 1334 cases were identified and sampled, 1062 were eligible and contacted, and 776 (191 AAs and 585 NHWs) completed the interview [36,37]. The Los Angeles County site included AA, Hispanic, and NHW men of any age diagnosed with a first primary prostate cancer between January 1, 1999 and December 31, 2003 [36]. A total of 3144 cases were identified, 2402 were contacted, 1870 met the eligibility criteria, and 1232 (376 AAs, 355 Hispanics, and 501 NHWs) completed the interview [36]. In both studies, advanced prostate cancer was defined as a tumor invading and extending beyond the prostatic capsule and/or extending into adjacent tissue or involving regional lymph nodes or distant metastatic sites [37].

2.2. Data collection and follow-up

Trained interviewers conducted in-person interviews in English or Spanish using a structured questionnaire that asked about sociodemographic background, medical history, and lifestyle factors (Table 1). Dietary intake during the calendar year before diagnosis was assessed using the Block Food Frequency questionnaire [40]. Self-reported comorbidities that were associated at $p < 0.05$ with overall survival (i.e., heart disease, diabetes, liver disease, kidney disease) in the base model (adjusted for age at diagnosis and race/ethnicity, stratified by stage at diagnosis, census-block-group adjusted) were used to create a composite measure of any comorbidities (heart disease, diabetes, liver disease, and/or kidney disease). Age at diagnosis, marital status at diagnosis, SEER Summary stage, histology, subsequent (primary) tumors (number of subsequent tumors and time from diagnosis to first subsequent tumor), and first-course treatment, all routinely abstracted from medical records, were obtained from the CCR. The CCR routinely updates vital status and cause of death through linkages with state and national databases. Characteristics of the first reporting hospital were assessed. Hospital NCI Cancer Center designation was coded based on status

as of 2012. The percent of cancer cases in a racial/ethnic group or quintile of nSES among all CCR cases diagnosed from 1997 to 2003 were used to estimate hospital-level race/ethnicity (i.e., percent NHW, percent AA, and percent Hispanic) and nSES, respectively, and scaled into quartiles based on all hospitals statewide [41].

Cases with a residential address at the time of diagnosis were geocoded to latitude/longitude coordinates and then assigned a 2000 Census block group. Batch geocoding was performed using the Texas A &M Geocoder [42] or manually using ArcGIS [43]. Themajority of addresses (99.6%) were geocoded successfully. Of the 1568 block groups represented, 1368 (87%) were represented by a single individual.

2.3. Socioeconomic status

Self-reported education was categorized as high school diploma (or equivalent) or less, vocational/technical degree or some college, and college degree or higher. Neighborhood-level SES (nSES) at the time of diagnosis was measured at the Census block-group level and was based on an index created with principal components analyses that incorporates 2000 Census data on education, occupation, unemployment, household income, house values, rent values, and poverty [44]. The nSES index was scaled into statewide quintiles, low nSES (Q1) to high nSES (Q5). A joint education and nSES variable was created, where low education was defined as high school diploma or less and low nSES included quintiles 1–3.

2.4. Social and built environment factors

Data on several specific social and built environment factors measured at the block group or tract level, or for a residential buffer, were obtained from the CNDS (Table 1). Measures of neighborhood housing, commuting, residential mobility, and population density were at the block group level (2000 Census Summary File 3 [SF-3]) and modeled with statewide quartiles as described previously [21,45]. Census tract-level street connectivity was modeled with quartiles based on the state distribution. Street network-based measures included the gamma measure (ratio of actual number of street segments to the maximum possible given the number of intersections) [21].

Variables measured according to a residential buffer were defined for each case in order to capture access to amenities within a walking distance of 1600 m [46] along a network of pedestrian-accessible pathways (NavStreets) [47]. Information regarding the total number of businesses (quartiles, based on the sample distribution), parks (0, 1–2, 3, or 4), and farmers markets (0, 1, or ≥2) originated from several geocoded data sources for business listings [48], farmers markets (California Department of Food and Agriculture, 2010), and NavStreets [47]. In addition, two previously developed food indices to describe the retail food environment and restaurant environment were included: the Retail Food Environment Index (RFEI) and the Restaurant Environment Index (REI) are ratios of unhealthy to healthy retail food outlets and restaurants within the residential buffer, with higher values indicating a less healthy neighborhood retail or restaurant food environment, respectively; values are presented with categories of “0” (no unhealthy outlets or restaurants) or tertiles based on the sample distribution [21,45,49,50]. The traffic density measure, categorized into quartiles based on the sample distribution, was developed from the database of trafic counts from the

California Department of Transportation for each measured road segment within a residential buffer of 500 m [51,52].

2.5. Survival

Survival, in months, was calculated from date of diagnosis to whichever of the following occurred first: 1) date of death, 2) date of last known contact, or 3) December 31, 2009 (the end of the follow-up period). Of the 1243 patients still alive at the end of the follow-up period, 97.5% had complete follow-up in the last year of the study. Both overall survival and prostate cancer-specific survival were examined.

2.6. Statistical analysis

After excluding 144 cases with a prior malignant tumor, 48 cases with ambiguous tumor staging, and 16 cases with inconsistent dates for survival time calculation, the analyses were based on 1800 prostate cancer cases (686 localized and 1114 advanced). All models were multivariable stage-stratified marginal Cox proportional hazards regression models with robust sandwich estimates minimally adjusted for age at diagnosis, race/ethnicity, and study site, in addition to a cluster adjustment for census block groups (base model) [53]. Stage-stratified models allow the baseline hazards to vary by stage; however, stage effects cannot be estimated.

A series of nested models was created to assess the effect of multilevel factors on SES disparities in survival by first including individual-level covariates, followed by hospital-level covariates, and finally social and built environment factors that were associated with survival at $p < 0.05$ in the base model. Using the nested models, we examined the effect of covariates and social and built environment factors on the association of education and nSES with overall survival, first independently in separate models, then together as independent main effects, and finally as a single combination variable to assess the joint association. We calculated the synergy index in order to also assess whether the nSES/education joint effect on overall survival was additive [54]. The proportional hazards assumption was assessed by including interaction terms between main effects and time and using likelihood ratio tests to check for statistical significance; we observed no significant violations. Interactions between race/ethnicity and the main effects and between nSES and education were checked using likelihood ratio tests; none were statistically significant.

3. Results

Multi-level characteristics for cases and deaths are presented in Table 1 and Supplementary Tables 1 and 2. The majority of cases were NHW (53%), followed by AA (28%) and Hispanic (18%); 37% had high school education or less, 54% resided in high SES neighborhoods; 62% were diagnosed with advanced prostate cancer (reflecting the oversampling of advanced stage cases).

Survival was comparable between NHW and Hispanic men, regardless of model (Table 2). However, compared to NHW men, AA men had greater risk of death in the base model (HR, 1.33; 95% CI, 1.10–1.61). This disparity remained when education was added to the base

model (HR, 1.26; 95% CI, 1.03–1.55), but was attenuated when nSES was added (HR, 1.17; 95% CI, 0.94–1.46).

Individual factors associated with greater risk of death were younger age at diagnosis (40–49 years vs. 60–69 years: HR, 3.47; 95% CI, 1.66–7.25), history of comorbidities (HR, 1.28; 95% CI, 1.08–1.53), obesity (≥30 vs. < 25 kg/m^2: HR, 1.36; 95% CI, 1.07–1.72), and current vs. never smoking (HR, 1.48; 95% CI, 1.16–1.90) (Table 3). Risk of death was lower for foreign-born vs. U.S.-born men (HR, 0.73; 95% CI, 0.54k0.98), history of radical prostatectomy (HR, 0.34; 95% CI, 0.25–0.48), and greater levels of physical activity (≥23 vs. < 2.8 h/week: HR, 0.64; 95% CI, 0.50–0.81). Cases seen at NCI cancer centers had a lower risk of death (HR, 0.70; 95% CI, 0.51–0.97) as did those in hospitals with a greater percentage of patients residing in high SES neighborhoods or with a greater percentage of NHW patients. Social and built environment factors significantly associated with lower risk of death were more household crowding ($p < 0.01$), and a less favorable RFEI (0.04). While the REI did not show a statistically significant association, there was a significant trend between less favorable neighborhood restaurant environments (greater REI ratio value) and greater risk of death ($p = 0.03$).

Both lower nSES and lower education were significantly associated with greater risk of death in Model 1 (Table 4), with significant trends ($p < 0.05$) detected. When nSES and education were included in a single model, their main effects were attenuated yet remained significant when comparing the lowest to highest levels for each SES variable [education: HR = 1.32, (1.05–1.67); nSES: HR = 1.56, (1.11–2.19)]. Education and nSES were jointly associated with survival such that men with the lowest levels of education and living in low nSES areas had the greatest risk of death compared to college graduates living in high nSES areas. However, even among college graduates, low nSES continued to carry a significant excess hazard (HR, 1.39; 95% CI, 1.07–1.80). This joint association of low nSES/less than high school education on overall survival was an additive inverse association, as indicated by a synergy index (SI) of greater than 1 (Model 1: SI, 2.73; 95% CI, 2.42–3.06).

The sequential addition of nativity, comorbidities, health behaviors and hospital SES did not substantially change associations between nSES or education and survival (data not shown), whether modeled separately or jointly, and neither did the inclusion of these factors in a single model (Model 2, Table 4). The further addition of specific neighborhood factors did not change the HRs associated with nSES and education when they were modeled separately; but when included in the same model, the nSES associations were attenuated, although the association between education and survival remained statistically significant. Using these most comprehensive models (Models 3 and 4), relative to men with at least some college education living in high SES neighborhoods, men in all of the lower joint nSES and education combination categories had greater risk of death, as high as 57% (95% CI, 1.18–2.08) for men with less than high school education in low SES neighborhoods.

Results of analyses examining prostate cancer-specific mortality are presented in Supplemental Tables 3 and 4. Due to low numbers of prostate cancer-specific deaths among the study population, analyses of prostate cancer-specific mortality were underpowered. However, we observed similar patterns of association between SES and mortality for

prostate cancer-specific mortality as with overall mortality (Supplemental Table 4). For example, lower nSES is associated with greater prostate cancer-specific mortality, but only the ratio of the lowest nSES quintile, compared to the highest, was statistically significant (HR, 1.85; 95% CI, 1.11–3.07). Lower education (high school or less compared to college graduate or more) was associated with a nearly 20% increased risk of prostate cancer-specific death, but the HR was not statistically significant (HR, 1.19; 95% CI, 0.86–1.66). Like with overall survival; sociodemographic factors, clinical factors, individual behavioral factors, hospital factors, and specific social and built environment factors did not explain associations of nSES with prostate cancer-specific survival (HR, 1.86; 95% CI, 1.06–3.25).

4. Discussion

Our study utilized multi-level data to examine racial/ethnic and SES disparities in overall survival among a diverse, population-based series of California prostate cancer cases. Neighborhood SES, but not individual-level education, attenuated the survival disparity between NHW and AA men, while both education and nSES showed independent associations with overall survival. Sociodemographic, clinical, behavioral, hospital and specific neighborhood factors were associated with overall survival, but explained only a small portion of the independent and joint associations between SES factors and overall survival. Associations between SES and prostate cancer-specific survival were similar.

A multi-level, population-based study of prostate cancer from Taiwan found a joint association of individual- and area-level SES such that only men over age 65 years with lower individual SES residing in lower income areas had worse overall survival [55]. We observed a similar joint effect, as men with lower education and lower nSES had the greatest hazards effect size, and show the joint effect was additive.

Our results indicate that, while sociodemographic factors, clinical factors, individual behavioral factors, hospital factors, and specific social and built environment factors were associated with mortality, they largely do not explain associations of SES with survival, which suggests there remain unmeasured individual factors or unmeasured aspects of the contextual environment mediating the association between lower SES and worse survival for men with prostate cancer. Additional research is needed to identify the factors and mechanisms underlying the robust association between individual and neighborhood SES and survival after prostate cancer diagnosis. Health care access and health insurance status [56] may be important to consider in studies of SES disparities among men with prostate cancer, since individuals of lower SES are more likely to be uninsured [57] or experience other barriers to access (including for follow-up urologic or general healthcare visits). In fact, previous studies have reported shorter survival for uninsured or publicly insured compared to privately insured men diagnosed with prostate cancer, independent of census-tract measures of SES [58,59]. We included information on initial treatment and hospital characteristics, but data on health insurance were not available for the complete case series included in our study [60]. In addition, there may be factors that mediate effects of low SES on mortality that are not directly related to healthcare access. Contextual factors not considered here (e.g., crime, segregation, or social support) may contribute to or modify individuals' experience of chronic stress and overall well-being [61,62] that may ultimately

influence overall survival. Although we assessed several social and built environment characteristics and did not find that these factors mediated the SES associations, this may be due to the lack of specificity of these variables which were based on secondary data and may not capture how patients use and perceive their environments [35].

Data are inconsistent on whether nSES [61] accounts for racial disparities in overall survival among men with prostate cancer [3,4,8,9], although previous studies differ substantially in regards to geographic region, covariates included, and measurement of nSES. Our results indicate that the disparity in survival for AA men compared to NHW men was attenuated by nSES, but not by education. Furthermore, we determined that the independent associations between education and nSES and survival did not differ by race/ethnicity.

Ours is the first multi-level study of mortality among men with prostate cancer to consider individual-, hospital-, and contextual-level data. It combines both interview and secondary data, including specific social and built environment factors, in order to explore potential mediators of SES disparities in survival among men with prostate cancer. However, our study does have some limitations. Education was the only measure of individual-level SES assessed by self-report. The interview questionnaire, developed and administered as part of the individual case-control studies, only assessed the presence of medical conditions relevant to hypotheses of prostate cancer risk examined by those studies (asthma, heart disease, diabetes, kidney disease, liver disease, cataracts, epilepsy, and skin cancer), and thus we did not have the data to use a validated comorbidity instrument (e.g., the Charlson Comorbidity Index). Instead, we created a simple index of comorbidities of conditions from the questionnaire associated ($p < 0.05$) with prostate cancer survival in the Base model (heart disease, diabetes, kidney disease, and liver disease). Compared to the underlying cancer registry population, the study population comprised greater proportions of men 70–79 years of age with localized prostate cancer, men 50–69 years of age with advanced prostate cancer, and AA and Hispanic men with both localized and advanced prostate cancer. The greater proportions of AA and Hispanic men reflect efforts at each site to assure adequate representation and sample size among non-White men with prostate cancer. Neighborhood variables derived from Census data are subject to administrative boundaries that may not accurately portray the real or perceived neighborhood environments experienced by individuals. However, we utilized the smallest level of geography for which robust data were available, census block-group and census-tract, which have been shown to perform well to detect SES gradients in health outcomes [63]. While we conducted analyses for both overall and prostate cancer-specific survival, our analyses of prostate cancer-specific survival were relatively underpowered.

5. Conclusions

Our results demonstrate the importance of individual and neighborhood SES in prostate cancer survival and show that many specific social and built environment characteristics do not account for SES disparities. Additional research is needed to identify the factors and mechanisms underlying the association between neighborhood SES and mortality after prostate cancer diagnosis.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

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Abbreviations

AA	African American
NHW	non-Hispanic White
SES	socioeconomic status
nSES	neighborhood socioeconomic status
NAPC	Neighborhoods and Prostate Cancer Study
CCR	California Cancer Registry
CNDS	California Neighborhoods Data System
SEER	Surveillance Epidemiology and End Results Program
REI	Restaurant Environment Index
RFEI	Retail Food Environment Index

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Table 1

Distribution of multi-level characteristics according to number of total cases and deaths among men diagnosed with prostate cancer, San Francisco Bay Area and Los Angeles County 1997–2003.

	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
Individual-level sociodemographic factors[a]						
Education						
High school degree or less	669	(37.2%)	255	(45.8%)	91	(41.7%)
Some college	516	(28.7%)	154	(27.6%)	62	(28.4%)
College graduate or higher	615	(34.2%)	148	(26.6%)	65	(29.8%)
Study Site						
San Francisco Bay Area	720	(40.0%)	223	(40.0%)	101	(46.3%)
Los Angeles County	1080	(60.0%)	334	(60.0%)	117	(53.7%)
Race/ethnicity						
non-Hispanic White	978	(54.3%)	286	(51.3%)	115	(52.8%)
African-American	505	(28.1%)	177	(31.8%)	69	(31.7%)
Hispanic	317	(17.6%)	94	(16.9%)	34	(15.6%)
Nativity						
U.S.-born	1476	(82.0%)	479	(86.0%)	187	(85.8%)
Foreign-born	324	(18.0%)	78	(14.0%)	31	(14.2%)
Tumor and treatment factors[b]						
Age at diagnosis (years)						
40–49	68	(3.8%)	14	(2.5%)	10	(4.6%)
50–59	444	(24.7%)	82	(14.7%)	50	(22.9%)
60–69	726	(40.3%)	200	(35.9%)	83	(38.1%)
70–79	489	(27.2%)	214	(38.4%)	67	(30.7%)
80+	73	(4.1%)	47	(8.4%)	8	(3.7%)
Marital status at diagnosis						
Single/Never Married	196	(10.9%)	69	(12.4%)	33	(15.1%)
Married	1274	(70.8%)	376	(67.5%)	145	(66.5%)

	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	**(%)**	**n(%)**	**(%)**	**n(%)**	**(%)**
Separated/Divorced	170	(9.4%)	62	(11.1%)	26	(11.9%)
Widowed	82	(4.6%)	33	(5.9%)	7	(3.2%)
Unknown	78	(4.3%)	17	(3.1%)	7	(3.2%)
Stage						
Localized	686	(38.1%)	202	(36.3%)	33	(15.1%)
Advanced	1114	(61.9%)	355	(63.7%)	185	(84.9%)
Histologic Grade						
Grade I–II	1157	(64.3%)	270	(48.5%)	66	(30.3%)
Grade III–IV	604	(33.6%)	261	(46.9%)	138	(63.3%)
Unknown	39	(2.2%)	26	(4.7%)	14	(6.4%)
One or More Subsequent Tumors						
No	1572	(87.3%)	435	(78.1%)	199	(91.3%)
Yes	228	(12.7%)	122	(21.9%)	19	(8.7%)
Surgery						
None	876	(48.7%)	386	(69.3%)	156	(71.6%)
Local or ‘not otherwise specified’	72	(4.0%)	39	(7.0%)	21	(9.6%)
Radical prostatectomy	852	(47.3%)	132	(23.7%)	41	(18.8%)
Radiation						
None	1223	(67.9%)	364	(65.4%)	148	(67.9%)
Given	577	(32.1%)	193	(34.6%)	70	(32.1%)
Medical History[a]						
Family history of prostate cancer						
No	1438	(79.9%)	459	(82.4%)	185	(84.9%)
Yes	361	(20.1%)	98	(17.6%)	33	(15.1%)
Unknown	~	~	~	~	~	~
Benign prostatic hyperplasia						
No	962	(53.4%)	270	(48.5%)	108	(49.5%)
Yes	781	(43.4%)	262	(47.0%)	96	(44.0%)
Unknown	57	(3.2%)	25	(4.5%)	14	(6.4%)

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	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
Prostatitis						
No	1209	(67.2%)	364	(65.4%)	127	(58.3%)
Yes	541	(30.1%)	172	(30.9%)	82	(37.6%)
Unknown	50	(2.8%)	21	(3.8%)	9	(4.1%)
Comorbidities[c]						
No	1230	(68.3%)	335	(60.1%)	157	(72.0%)
Yes	570	(31.7%)	222	(39.9%)	61	(28.0%)
Behavioral factors[a]						
Body mass index (BMI, kg/m^2), year prior to diagnosis						
< 25	457	(25.4%)	142	(25.5%)	66	(30.3%)
25–29	876	(48.7%)	266	(47.8%)	94	(43.1%)
30+	451	(25.1%)	143	(25.7%)	55	(25.2%)
Unknown	16	(0.9%)	~	~	~	~
Average daily caloric intake (kcal, year prior to diagnosis)						
< 1950	395	(21.9%)	132	(23.7%)	47	(21.6%)
1951–2584	403	(22.4%)	121	(21.7%)	45	(20.6%)
2585–3301	374	(20.8%)	110	(19.7%)	44	(20.2%)
3302+	442	(24.6%)	141	(25.3%)	56	(25.7%)
Missing	186	(10.3%)	53	(9.5%)	26	(11.9%)
Average daily alcohol consumption (grams, year prior to diagnosis)						
0	811	(45.1%)	262	(47.0%)	86	(39.4%)
1–5	157	(8.7%)	43	(7.7%)	21	(9.6%)
5–9.9	109	(6.1%)	38	(6.8%)	19	(8.7%)
10–14.9	129	(7.2%)	31	(5.6%)	14	(6.4%)
15+	408	(22.7%)	130	(23.3%)	52	(23.9%)
Unknown	186	(10.3%)	53	(9.5%)	26	(11.9%)
Smoking status, year prior to diagnosis						
Never	515	(28.6%)	141	(25.3%)	59	(27.1%)

	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
Former	929	(51.6%)	289	(51.9%)	106	(48.6%)
Current	343	(19.1%)	121	(21.7%)	50	(22.9%)
Unknown	13	(0.7%)	6	(1.1%)	~	~
Physical Activity (hours/week, previous 5 years, recreational and non-recreational sources)						
< 2.8	435	(24.2%)	173	(31.1%)	53	(24.3%)
2.8–9.2	452	(25.1%)	132	(23.7%)	54	(24.8%)
9.3–22.9	447	(24.8%)	126	(22.6%)	52	(23.9%)
23.0+	445	(24.7%)	118	(21.2%)	59	(27.1%)
Unknown	21	(1.2%)	8	(1.4%)	0	(0.0%)
Hospital-level factors[b]						
NCI designated Cancer Center						
No	1602	(89.0%)	512	(91.9%)	206	(94.5%)
Yes	198	(11.0%)	45	(8.1%)	12	(5.5%)
Hospital race/ethnicity (quartiles)[d]						
Q1	479	(26.6%)	149	(26.8%)	61	(28.0%)
Q2	424	(23.6%)	164	(29.4%)	60	(27.5%)
Q3	424	(23.6%)	128	(23.0%)	52	(23.9%)
Q4	473	(26.3%)	116	(20.8%)	45	(20.6%)
Hospital SES (quartiles)[e]						
Q1	447	(24.8%)	142	(25.5%)	61	(28.0%)
Q2	407	(22.6%)	134	(24.1%)	55	(25.2%)
Q3	489	(27.2%)	162	(29.1%)	46	(21.1%)
Q4	457	(25.4%)	119	(21.4%)	56	(25.7%)
Contextual-level factors[f,g]						
Neighborhood SES (quintiles[h]**)**						
Q1 (lowest)	277	(15.4%)	106	(19.0%)	39	(17.9%)
Q2	269	(14.9%)	95	(17.1%)	45	(20.6%)
Q3	284	(15.8%)	91	(16.3%)	34	(15.6%)

	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
Q4	340	(18.9%)	104	(18.7%)	31	(14.2%)
Q5 (highest)	624	(34.7%)	160	(28.7%)	68	(31.2%)
Percentage of residents traveling 60+ minutes to work (quartiles[h])						
Q1	416	(23.1%)	148	(26.6%)	57	(26.1%)
Q2	469	(26.1%)	144	(25.9%)	59	(27.1%)
Q3	423	(23.5%)	119	(21.4%)	46	(21.1%)
Q4	486	(27.0%)	145	(26.0%)	55	(25.2%)
Percentage of residents traveling to work by car or motorcycle (quartiles[h])						
Q1	430	(23.9%)	149	(26.8%)	66	(30.3%)
Q2	437	(24.3%)	144	(25.9%)	57	(26.1%)
Q3	419	(23.3%)	109	(19.6%)	37	(17.0%)
Q4	508	(28.2%)	154	(27.6%)	57	(26.1%)
Residential mobility[i](quartiles[h])						
Q1	445	(24.7%)	143	(25.7%)	59	(27.1%)
Q2	450	(25.0%)	138	(24.8%)	53	(24.3%)
Q3	450	(25.0%)	157	(28.2%)	61	(28.0%)
Q4	449	(24.9%)	118	(21.2%)	44	(20.2%)
Household crowding (quartiles[h])						
Q1	459	(25.5%)	104	(18.7%)	39	(17.9%)
Q2	419	(23.3%)	130	(23.3%)	45	(20.6%)
Q3	462	(25.7%)	162	(29.1%)	71	(32.6%)
Q4	454	(25.2%)	160	(28.7%)	62	(28.4%)
Percentage of multi-family housing units[j](quartiles[h])						
Q1	447	(24.8%)	127	(22.8%)	49	(22.5%)
Q2	452	(25.1%)	130	(23.3%)	44	(20.2%)
Q3	450	(25.0%)	160	(28.7%)	65	(29.8%)
Q4	445	(24.7%)	139	(25.0%)	59	(27.1%)

	Prostate Cancer Cases N = 1800		All-cause Deaths N = 557		Prostate-specific Deaths N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
Street connectivity (gamma measure[k], quartiles[h])						
Q1	403	(22.4%)	106	(19.0%)	40	(18.3%)
Q2	511	(28.4%)	147	(26.4%)	61	(28.0%)
Q3	397	(22.1%)	132	(23.7%)	51	(23.4%)
Q4	483	(26.8%)	171	(30.7%)	65	(29.8%)
Businesses (total number, quartiles[l])						
Q1	426	(23.7%)	124	(22.3%)	55	(25.2%)
Q2	441	(24.5%)	125	(22.4%)	43	(19.7%)
Q3	453	(25.2%)	143	(25.7%)	53	(24.3%)
Q4	474	(26.3%)	164	(29.4%)	66	(30.3%)
Restaurant Environment Index[l,m]						
0	514	(28.6%)	136	(24.4%)	59	(27.1%)
T1	390	(21.7%)	118	(21.2%)	48	(22.0%)
T2	409	(22.7%)	133	(23.9%)	58	(26.6%)
T3	481	(26.7%)	169	(30.3%)	52	(23.9%)
Food Retail Environment Index[l,n]						
0	333	(18.5%)	83	(14.9%)	35	(16.1%)
T1	472	(26.2%)	161	(28.9%)	68	(31.2%)
T2	534	(29.7%)	180	(32.3%)	68	(31.2%)
T3	455	(25.3%)	132	(23.7%)	46	(21.1%)
Parks (total number)						
0	497	(27.6%)	147	(26.4%)	63	(28.9%)
1–2	907	(50.4%)	291	(52.2%)	103	(47.2%)
3	173	(9.6%)	44	(7.9%)	17	(7.8%)
4	217	(12.1%)	74	(13.3%)	34	(15.6%)
Farmers markets (total number)						
0	1393	(77.4%)	408	(73.2%)	161	(73.9%)
1	287	(15.9%)	108	(19.4%)	39	(17.9%)

	Prostate Cancer Cases		All-cause Deaths		Prostate-specific Deaths	
	N = 1800		N = 557		N = 218	
	n	(%)	n(%)	(%)	n(%)	(%)
2+	114	(6.3%)	40	(7.2%)	17	(7.8%)
Traffic density[l]						
Q1	448	(24.9%)	120	(21.5%)	45	(20.6%)
Q2	449	(24.9%)	137	(24.6%)	53	(24.3%)
Q3	448	(24.9%)	151	(27.1%)	66	(30.3%)
Q4	449	(24.9%)	148	(26.6%)	53	(24.3%)

[~] Censored due to low frequency.

[a] Data obtained from interview.

[b] Data obtained from the California Cancer Registry.

[c] Self-reported comorbidities (asthma, heart disease, diabetes, kidney disease, liver disease, cataracts, epilepsy, and skin cancer) that were associated at $p < 0.05$ with overall survival (heart disease, diabetes, liver disease, kidney disease; data not shown) in the base model (adjusted for age at diagnosis and race/ethnicity, stratified by stage at diagnosis, census-block-group adjusted) were used to create a composite measure of any comorbidities (yes, no), such that a categorization of 'yes' indicates an individual experienced at least one comorbidity among heart disease, diabetes, liver disease, and kidney disease.

[d] Hospital race/ethnicity defined as the percentage of cancer patients that were non-Hispanic White at the time of diagnosis.

[e] Hospital SES defined as the percentage of cancer patients residing in quintile 5 of nSES at the time of diagnosis.

[f] Data obtained from the California Neighborhoods Data System.

[g] Six prostate cancer cases had data missing for contextual-level factors.

[h] Based on the quintile/quartile distribution for block groups/census tracts in California.

[i] Neighborhood residential mobility was measured as the percent of residents who lived in the same location from 1995 to 2000.

[j] Percentage of multi-family housing was defined as the percentage of total housing units that are not single family dwellings (i.e., structures with more than 2 units).

[k] Gamma measure, ratio of actual number of street segments to the maximum possible given the number of intersections.

[l] Based on the quartile/tertile distribution among all study cases.

[m] The Restaurant Environment Index is the ratio of the number of fast food restaurants compared to the number of other restaurants within the residential buffer. Cases with residential buffers with no businesses were included in the '0' category.

[n] The Retail Food Environment Index is the ratio of the number of convenience stores, liquor stores, and fast food restaurants compared to the number of supermarkets and farmers markets within the residential buffer. Cases with residential buffers with no businesses were included in the '0' category.

Table 2

Association of race/ethnicity with all-cause mortality among men with prostate cancer, adjusted for education and neighborhood socioeconomic status (nSES) as indicated, San Francisco Bay Area and Los Angeles County 1997–2003.

	Cases	Deaths	Base model[1]		Base model + Education		Base model + nSES	
	N	N	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)
Race/ethnicity								
Non-Hispanic White	978	286	1.00	reference	1.00	reference	1.00	reference
African American	505	177	**1.33**	**(1.10–1.61)**	**1.26**	**(1.03–1.55)**	1.17	(0.94–1.46)
Hispanic	317	94	1.10	(0.84–1.43)	1.00	(0.76–1.32)	0.98	(0.74–1.31)

[1]Base model adjusted for age, race/ethnicity, and study site; stage stratified; census block group-adjusted

[2]nSES, neighborhood socioeconomic status; HR, hazard ratio; CI, confidence interval

Bold type indicates statistical significance.

Table 3

Associations between individual-, hospital-, and contextual-level factors and all-cause mortality (Base Model[a]) among men diagnosed with prostate cancer, San Francisco Bay Area and Los Angeles County 1997–2003.

	Deaths	Mortality		p-value[b]	p-trend[b]
	n	HR	(95% CI)		
Individual-level sociodemographic factors					
Education					
High School Degree or Less	255	**1.59**	**(1.27–1.99)**		
Some College	154	**1.28**	**(1.01–1.61)**		
College Graduate or Higher	148	1.00	reference	**< 0.01**	**<0.01**
Study Site					
San Francisco Bay Area	223	1.02	(0.84–1.23)		
Los Angeles County	334	1.00	reference		
Race/ethnicity					
Non-Hispanic White	286	1.00	reference		
African American	177	**1.33**	**(1.10–1.61)**		
Hispanic	94	1.10	(0.84–1.43)	**0.01**	
Nativity					
U.S.-born	479	1.00	reference		
Foreign-born	78	**0.73**	**(0.54–0.98)**	**0.03**	
Tumor and treatment factors					
Age at diagnosis (year)					
40–49	14	**3.47**	**(1.66–7.25)**		
50–59	82	1.38	(0.95–2.00)		
60–69	200	1.00	reference		
70–79	214	**0.70**	**(0.49–0.99)**		
80+	47	0.66	(0.34–1.29)	**0.01**	**0.02**
Marital Status (at diagnosis)					
Single/Never Married	69	1.21	(0.93–1.57)		
Married	376	1.00	reference		
Separated/Divorced	62	1.19	(0.92–1.53)		
Widowed	33	1.00	(0.69–1.46)		
Unknown	17	0.67	(0.41–1.12)	0.18	
Stage					
Localized	202	1.00	reference		
Advanced	355	**1.53**	**(1.29–1.83)**	**< 0.01**	
Histologic Grade					
Grade I–II	270	**0.53**	**(0.44–0.63)**		
Grade III–IV	261	1.00	reference		
Unknown	26	1.13	(0.80–1.61)	**< 0.01**	
One or More Subsequent Tumors					
No	435	1.00	reference		

	Deaths	Mortality		p-value[b]	p-trend[b]
	n	HR	(95% CI)		
Yes	122	**4.51**	**(3.09–6.56)**	**< 0.01**	
Surgery					
None	386	1.00	reference		
Local or NOS	39	1.31	(0.94–1.83)		
Radical prostatectomy	132	**0.34**	**(0.25–0.48)**	**< 0.01**	
Radiation					
None	364	1.00	reference		
Given	193	1.09	(0.90–1.30)	0.48	
Medical History					
Family history of prostate cancer					
No	459	1.00	reference		
Yes	98	0.93	(0.75–1.14)	0.55	
Benign prostatic hyperplasia					
No	270	1.00	reference		
Yes	262	0.97	(0.81–1.16)		
Unknown	25	1.26	(0.83–1.93)	0.64	
Prostatitis					
No	364	1.00	reference		
Yes	172	0.96	(0.79–1.18)		
Unknown	21	1.13	(0.69–1.85)	0.66	
Comorbidities					
No	335	1.00	reference		
Yes	222	**1.28**	**(1.08–1.53)**	**< 0.01**	
Behavioral factors					
Body mass index (BMI, kg/m^2), year prior to diagnosis					
< 25	142	1.00	reference		
25–29	266	1.11	(0.90–1.36)		
30+	143	**1.36**	**(1.07–1.72)**		
Unknown	6	1.96	(0.83–4.65)	**0.02**	**0.01**
Average daily caloric intake (kcal, year prior to diagnosis)					
< 1950	132	1.00	reference		
1951–2584	121	0.86	(0.67–1.10)		
2585–3301	110	0.87	(0.67–1.12)		
3302+	141	1.06	(0.83–1.36)		
Missing	53	1.16	(0.82–1.64)	0.20	0.57
Average daily alcohol consumption (grams, year prior to diagnosis)					
0	262	1.00	reference		
1–5	43	0.93	(0.67–1.28)		
5–9.9	38	1.30	(0.94–1.80)		
10–14.9	31	0.92	(0.65–1.30)		
15+	130	0.99	(0.79–1.25)		

	Deaths	Mortality		p-value[b]	p-trend[b]
	n	HR	(95% CI)		
Unknown	53	1.21	(0.89–1.66)	0.32	0.97
Smoking status, year prior to diagnosis					
Never	141	1.00	reference		
Former	289	1.05	(0.85–1.28)		
Current	121	**1.48**	**(1.16–1.90)**		
Unknown	6	**2.61**	**(1.03–6.60)**	**< 0.01**	**< 0.01**
Physical Activity (hours/week, previous 5 years, recreational and non-recreational sources)					
< 2.8	173	1.00	reference		
2.8–9.2	132	**0.75**	**(0.60–0.94)**		
9.3–22.9	126	**0.70**	**(0.55k0.89)**		
23.0+	118	**0.64**	**(0.50–0.81)**		
Unknown	8	0.70	(0.37–1.31)	**< 0.01**	**< 0.01**
Hospital-level factors					
NCI designated Cancer Center[c]					
No	512	1.00	reference		
Yes	45	**0.70**	**(0.51–0.97)**	**0.02**	
Hospital race/ethnicity[d]					
Q1	149	1.00	reference		
Q2	164	1.09	(0.84–1.40)		
Q3	128	0.97	(0.75–1.25)		
Q4	116	**0.74**	**(0.55–0.99)**	**< .01**	**0.02**
Hospital SES[e]					
Q1	142	1.00	reference		
Q2	134	0.91	(0.72–1.17)		
Q3	162	0.88	(0.68–1.15)		
Q4	119	**0.70**	**(0.50–0.99)**	0.11	0.07
Contextual-level factors					
Neighborhood SES[f]					
Q1 (lowest)	106	**1.89**	**(1.38–2.60)**		
Q2	95	**1.45**	**(1.08–1.95)**		
Q3	91	**1.49**	**(1.13–1.96)**		
Q4	104	1.21	(0.95–1.55)		
Q5 (highest)	160	1.00	reference	**< 0.01**	**< 0.01**
Percentage of residents traveling 60+ minutes to work[f]					
Q1	148	1.19	(0.95–1.49)		
Q2	144	1.03	(0.82–1.29)		
Q3	119	0.98	(0.77–1.25)		
Q4	145	1.00	reference	0.41	0.14
Percentage of residents traveling to work by car or motorcycle[f]					

	Deaths	Mortality		p-value[b]	p-trend[b]
	n	HR	(95% CI)		
Q1	149	0.98	(0.77–1.25)		
Q2	144	1.04	(0.83–1.31)		
Q3	109	0.88	(0.69–1.11)		
Q4	154	1.00	reference	0.40	0.83
Residential mobility[f,g]					
Q1	143	1.02	(0.80–1.30)		
Q2	138	1.02	(0.80–1.30)		
Q3	157	**1.30**	**(1.03–1.63)**		
Q4	118	1.00	reference	0.07	0.54
Household crowding[f]					
Q1	104	1.00	reference		
Q2	130	**0.55**	**(0.41–0.73)**		
Q3	162	**0.78**	**(0.61–0.99)**		
Q4	160	0.92	(0.73–1.16)	< **0.01**	< **0.01**
Percentage of multi-family housing units[f,h]					
Q1	127	0.94	(0.74–1.19)		
Q2	130	1.01	(0.79–1.28)		
Q3	160	**1.35**	**(1.07–1.70)**		
Q4	139	1.00	reference	< **0.01**	0.24
Street connectivity (gamma measure[i], quartiles[f])					
Q1	106	1.00	reference		
Q2	147	1.05	(0.82–1.34)		
Q3	132	1.20	(0.93–1.55)		
Q4	171	1.20	(0.93–1.54)	0.08	0.10
Businesses (total number, quartiles[j])					
Q1	124	1.00	reference		
Q2	125	0.90	(0.70–1.15)		
Q3	143	1.05	(0.82–1.36)		
Q4	164	1.10	(0.86–1.39)	0.21	0.23
Restaurant Environment Index[j,k]					
0	136	1.00	reference		
T1	118	1.05	(0.82–1.35)		
T2	133	1.18	(0.93–1.50)		
T3	169	**1.27**	**(1.00–1.62)**	0.06	**0.03**
Food Retail Environment Index[j,l]					
0	83	1.00	reference		
T1	161	**1.32**	**(1.01–1.71)**		
T2	180	**1.31**	**(1.01–1.70)**		
T3	132	1.13	(0.86–1.47)	0.04	0.65

	Deaths	Mortality		p-value[b]	p-trend[b]
	n	HR	(95% CI)		
Parks (total number)					
0	147	1.00	reference		
1–2	291	1.04	(0.85–1.27)		
3	44	0.73	(0.51–1.04)		
4	74	1.01	(0.75–1.37)	0.23	0.56
Farmers markets (total number)					
0	408	1.00	reference		
1	108	**1.27**	**(1.04–1.55)**		
2+	40	0.95	(0.67–1.35)	0.05	0.39
Traffic density[j]					
Q1	120	1.00	reference		
Q2	137	1.08	(0.85–1.38)		
Q3	151	1.25	(0.98–1.60)		
Q4	148	1.20	(0.95–1.52)	0.12	0.07

SES, socioeconomic status; HR, hazard ratio; CI, confidence interval; Bold type indicates statistical significance.

[a]Base Model adjusted for age, race/ethnicity, and study location; stage-stratified; census block group-adjusted.

[b]The reported p-value is for the association of the factor with overall survival, the reported p-trend is for the linear association across categories of the factor.

[c]NCI Cancer Center designation not included in nested models due to low number of deaths associated with the ‘yes’ value.

[d]Hospital race/ethnicity defined as the percentage of cancer patients that were non-Hispanic White at the time of diagnosis. This variable was not included in the nested models due to correlation with Hospital SES (r, 0.6838; *p*-value, < 0.0001).

[e]Hospital SES defined as the percentage of cancer patients residing in nSES Q5 at the time of diagnosis.

[f]Based on the quintile/quartile distribution for block groups/census tracts in California.

[g]Neighborhood residential mobility was measured as the percent of residents who lived in the same location from 1995 to 2000.

[h]Percentage of multi-family housing units was defined as the percentage of total housing units that are not single family dwellings (i.e., structures with more than 2 units).

[i]Measures considered were median block length, median block size, total intersections, total street segments, the alpha measure (ratio of the actual number of complete loops to the maximum possible given the number of intersections), and the gamma measure (ratio of actual number of street segments to the maximum possible given the number of intersections). None of these measures were associated with overall survival at $p < 0.05$.

[j]Based on the quartile/tertile distribution among all study cases.

[k]The Restaurant Environment Index is the ratio of the number of fast food restaurants compared to the number of other restaurants within the residential buffer. Cases with residential buffers with no businesses were included in the ‘0’ category.

[l]The Retail Food Environment Index is the ratio of the number of convenience stores, liquor stores, and fast food restaurants compared to the number of supermarkets and farmers markets within the residential buffer. Cases with residential buffers with no businesses were included in the ‘0’ category.

Table 4

Hazard ratios (95% confidence intervals) from nested models examining associations between nSES, education, and all-cause mortality among men diagnosed with prostate cancer, San Francisco Bay Area and Los Angeles County 1997–2003.

	Cases	Follow-up	Deaths	Model 1[a]: Base Model[1] + tumor and treatment factors		Model 2: Model 1 + nativity, comorbidities, health behaviors[b], and hospital SES		Model 3: Model 2 + Restaurant Environment Index[c]		Model 4: Model 2 + Retail Food Environment Index[d]	
	n	Person-years	n	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)	HR	(95% CI)
Models include nSES variable only (not education)											
nSES quintiles											
Q1, lowest	277	2177	106	**1.75**	**(1.27–2.41)**	**1.64**	**(1.18–2.29)**	**1.60**	**(1.13–2.27)**	**1.56**	**(1.10–2.23)**
Q2	269	2225	95	**1.37**	**(1.02–1.83)**	1.28	(0.95–1.73)	1.25	(0.91–1.72)	1.24	(0.90–1.70)
Q3	284	2377	91	**1.46**	**(1.10–1.93)**	**1.44**	**(1.08–1.91)**	**1.43**	**(1.05 – 1.94)**	**1.43**	**(1.05–1.93)**
Q4	340	3109	104	1.22	(0.95–1.57)	1.17	(0.91–1.51)	1.15	(0.88–1.51)	1.16	(0.89–1.51)
Q5, highest	624	6007	160	1.00	reference	1.00	reference	1.00	reference	1.00	reference
p-trend				**< 0.01**		**< 0.01**		**0.01**		**0.02**	
Models include education variable only (not nSES)											
Education											
High school or less	669	5473	255	**1.46**	**(1.18–1.82)**	**1.44**	**(1.15–1.81)**	**1.42**	**(1.13–1.79)**	**1.41**	**(1.12–1.78)**
Some college	516	4611	154	1.14	(0.90–1.44)	1.12	(0.88–1.43)	1.12	(0.88–1.43)	1.11	(0.87–1.42)
College graduate or more	615	5861	148	1.00	reference	1.00	reference	1.00	reference	1.00	reference
p-trend				**< 0.01**		**< 0.01**		**< 0.01**		**< 0.01**	
Models include separate nSES and education variables											
nSES quintiles											
Q1, lowest	277	2177	106	**1.56**	**(1.11–2.19)**	**1.47**	**(1.04 – 2.08)**	**1.44**	**(1.00–2.06)**	1.41	(0.98–2.03)
Q2	269	2225	95	1.26	(0.93–1.71)	1.18	(0.87 – 1.61)	1.16	(0.83–1.60)	1.16	(0.84–1.59)
Q3	284	2377	91	**1.34**	**(1.01–1.80)**	1.33	(0.99–1.78)	1.32	(0.96–1.80)	1.32	(0.97–1.80)
Q4	340	3109	104	1.16	(0.90–1.50)	1.11	(0.86–1.45)	1.10	(0.83–1.44)	1.11	(0.85–1.44)
Q5, highest	624	6007	160	1.00	reference	1.00	reference	1.00	reference	1.00	reference
p-trend				**0.02**		0.05		0.08		0.10	
Education											
High school or less	669	5473	255	**1.32**	**(1.05–1.67)**	**1.33**	**(1.04–1.68)**	**1.33**	**(1.04–1.69)**	**1.32**	**(1.04–1.68)**
Some college	516	4611	154	1.07	(0.84–1.36)	1.07	(0.83–1.37)	1.08	(0.84–1.38)	1.07	(0.83–1.37)

	Cases	Follow-up	Deaths	Model 1[a]: Base Model[1] + tumor and treatment factors		Model 2: Model 1 + nativity, comorbidities, health behaviors[b], and hospital SES		Model 3: Model 2 + Restaurant Environment Index[c]		Model 4: Model 2 + Retail Food Environment Index[d]	
	n	**Person-years**	**n**	**HR**	**(95% CI)**	**HR**	**(95% CI)**	**HR**	**(95% CI)**	**HR**	**(95% CI)**
College graduate or more	615	5861	148	1.00	reference	1.00	reference	1.00	reference	1.00	reference
p-trend				**0.01**		**0.01**		**0.01**		**0.01**	
Models include joint education/nSES variable											
Education/nSES[e,f]											
High school or less/Low nSES	465	3650	173	**1.64**	**(1.27–2.12)**	**1.60**	**(1.23–2.09)**	**1.58**	**(1.19–2.08)**	**1.57**	**(1.18–2.08)**
High school or less/High nSES	203	1814	82	**1.48**	**(1.14–1.92)**	**1.48**	**(1.14–1.92)**	**1.47**	**(1.13–1.92)**	**1.47**	**(1.12–1.92)**
Some college or more/Low nSES	365	3129	119	**1.39**	**(1.07–1.80)**	**1.37**	**(1.05–1.78)**	**1.36**	**(1.03–1.79)**	**1.35**	**(1.02–1.78)**
Some college or more/ High nSES	761	7302	182	1.00	reference	1.00	reference	1.00	reference	1.00	reference

nSES, neighborhood socioeconomic status; HR, hazard ratio; CI, confidence interval; Body type indicates statistical significance

[1]Base model: adjusted for age, race/ethnicity, and study location; stage-stratified; and census block group-adjusted.

[a]Tumor factors significantly associated with survival in the base model with $p < 0.05$ were 1st subsequent tumor/time to 1st subsequent tumor and histologic grade. Treatment associated with survival in the base model with $p < 0.05$ included surgery.

[b]Health behaviors associated with survival in the base model with $p < 0.05$ were BMI, smoking, and physical activity.

[c]The Restaurant Environment Index is the ratio of the number of fast food restaurants compared to the number of other restaurants within the residential buffer. Cases with residential buffers with no businesses were included in the '0' category.

[d]The Retail Food Environment Index is the ratio of the number of convenience stores, liquor stores, and fast food restaurants compared to the number of supermarkets and farmers markets within the residential buffer. Cases with residential buffers with no businesses were included in the '0' category.

[e]Low nSES (Q1–Q3), high SES (Q4–Q5).

[f]Synergy Index (SI): Model 3 SI, 2.74; 95% CI, 2.42–3.06 and Model 4 SI, 2.58; 95% CI, 2.21–2.94.