Stage at Diagnosis and Survival in a Multiethnic Cohort of Prostate Cancer Patients

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Objectives. We evaluated the effects of socioeconomic status and comorbidity on stage of disease and survival among 1,509 population-based prostate cancer patients.

Methods. We applied logistic regression and Cox proportional hazards regression to data from Whites, African Americans, and Asian Americans who were diagnosed from 1987 to 1991.

Results. Patients with existing comorbid conditions were less likely than those without these conditions to be diagnosed with advanced cancer. Compared with Whites, African Americans (odds ratio [OR]=1.5; 95% confidence interval [CI]=1.1, 2.2) and foreign-born Asian Americans (OR=1.6; 95% CI=1.0, 2.4) were more likely to be diagnosed with advanced cancer. Among men with localized disease, prostate cancer death rates were higher for African Americans than for Whites (death rate ratio=2.3; 95% CI=1.2, 4.7).

Conclusions. These findings support the need for further investigation of factors that affect access to and use of health care among African Americans and Asian Americans. (*Am J Public Health.* 2003;93:1753–1759)

Population-based studies that use cancer registry data consistently show that African Americans with prostate cancer present with more advanced disease[1,2] and that they have poorer survival rates than do US White men with the disease even when diagnosed at the same stage.[3–6] Studies conducted in equal-access health care systems also have found that African Americans present at later stages; however, results regarding survival are mixed.[7–12] In contrast, population-based reports of cancer registry data indicate that Japanese Americans and Chinese Americans (hereafter referred to as Asian Americans) have stage distributions that are similar to those of Whites, with a slightly higher proportion of distant cases,[6,13] and that these men have better 5-year relative survival rates than do Whites.[6,13,14–17] This Asian American survival advantage is especially marked among men with distant disease.[6,13,14]

Although an estimated 28 900 deaths from prostate cancer occurred in the United States in 2001,[18] we still have difficulty determining which localized cases of prostate cancer will progress and subsequently cause death. Advanced stage and high tumor grade are the primary factors linked with poor survival.[4,7–9] These factors may be associated with access to and use of health care, which in turn may be influenced by cultural, economic, and social components that vary by race and by place of birth. Immigrants and minorities often face similar obstacles when trying to obtain adequate health care.[19–24] In particular, recent immigrants may have reduced access to and use of the US and Canadian health care systems because of language barriers, specific cultural practices concerning medical treatment, and the fact that they may be poorer or live in poorer areas than their native-born counterparts and, therefore, have less health care coverage.[25–27]

Socioeconomic status (SES) is related to health care access and usage and health outcomes,[28–36] with lower SES associated with more limited and less frequent use of health care[31,34–36] as well as with higher morbidity and mortality.[31,32,37,38] For example, compared with individuals of high SES, individuals of low SES are less likely to receive aggressive prostate cancer treatment[33]; have altered physician-determined patient profiles for preventive care, disease management, and diagnostic testing costs[34]; have less health care access[35]; perceive greater medical discrimination[36]; and have lower health care utilization.[36] Census-level SES measures also may reflect neighborhood characteristics, such as crime and stress, that are particularly relevant to health status and health outcomes.[29,39–44]

Previous research among Whites and African Americans indicates that SES may account for some or all of the racial differences in disease stage at diagnosis[2,9,12,45,46] and survival.[47–49] However, as individuals age, the risk of developing both prostate cancer and comorbid conditions increases, and perhaps comorbid conditions that require regular medical care increase a man's use of health care. In addition, a number of studies among men with prostate cancer have observed that comorbidity influences survival by altering treatment choices and by contributing to death from other causes.[50–54]

We used 2 proxy measures—SES and comorbidity—to evaluate indirectly whether health and health care on racial differences in stage of disease at diagnosis and survival rates for patients with prostate cancer. We analyzed these SES- and comorbidity-related outcomes in a population-based group of 1638 patients (531 African Americans, 515 Whites, and 592 Asian Americans) who were diagnosed with prostate cancer. These patients lived on the island of Oahu, Hawaii; in the greater metropolitan areas of Los Angeles, Calif; San Francisco, Calif; or Vancouver, British Columbia. We hypothesized that racial differences in prevalence of comorbid conditions and SES would account for later stage at diagnosis and excess mortality among African American prostate cancer patients compared with White prostate cancer patients. In addition, we hypothesized that racial differences in SES and place of birth (United States or Canada vs elsewhere) might account for the higher proportion of distant cases among Asian Americans.

MATERIALS AND METHODS

Patient Ascertainment and Recruitment

Prostate cancer patients who were diagnosed between January 1, 1987, and December 31, 1991, and who were younger than 85 years were identified as part of a multicenter population-based case-control study through cancer registries in Honolulu (HI), Los Angeles (LA), San Francisco (SF), and Vancouver (BC). Patients were eligible to participate if, at the time of diagnosis, they resided in a region that was covered by the tumor registry and if they reported that at least 3 grandparents were of the same race as themselves. Overall, 70% of eligible patients (64% of African Americans, 72% of Whites, 79% of Japanese Americans, and 69% of Chinese Americans) participated in the study. Nonresponders were more likely than responders to have advanced prostate cancer. Additional details regarding the original study can be found in Whittemore et al.[55]

Vital Status Ascertainment

We obtained vital status information, including date and cause of death, from each of the 4 cancer registries through December 31, 1998. We obtained further vital status information from the National Death Index Plus (NDIP) on patients known to be alive before this date, and we obtained additional information on patients known to be deceased but without a known cause of death. NDIP matches were based on social security number, date of birth (within one year), and name (with minor misspellings and suffix differences). If the social security number was not available, we used matches that were exact on date of birth and name. Living subjects with registry follow-up that ended prior to December 31, 1998, and who were not listed in the NDIP, contributed time to the survival analysis until their registry last confirmed their living status. The participating registries and the NDIP code the cause of death as the underlying cause that is listed on the death certificate. We classified as deaths due to prostate cancer those deaths that were listed as codes 185.0–185.9 of the *International Classification of Diseases, Ninth Revision*.[56] All other codes were considered deaths due to other causes.

Other Study Variables

Date of birth, date of diagnosis, and stage at diagnosis information was collected from the registries during the initial study enrollment. *Stage* was defined as localized (confined to the prostate gland), regional (extension through the capsule and/or to regional lymph nodes), or distant (extended beyond regional lymph nodes, to bones, or to other sites).[57] Of 1638 patients, we dropped 129 patients who lacked stage information (59 African Americans, 31 Whites, 24 North America–born Asian Americans, and 15 foreign-born Asian Americans), which left 1509 patients in the analysis. Of the 553 Asian Americans included in the present analysis, 249 (45%) were of Chinese descent and 304 (55%) were of Japanese descent. We obtained census tract identification numbers at diagnosis from the registries or assigned them according to the patient's address at the time of diagnosis. We matched each tract number to data (from the 1990 US Census[58] and the 1991 Canadian Census[59]) on the percentage of individuals in the census tract with a high school education and the percentage of families in the census tract living below the poverty line. We used these census-level SES measures because they have been shown to predict both health status and the use of health services.[28–29] Tertiles of census variables were based on all tracts in the geographic reporting areas of the 4 registries. We also analyzed a measure of personal SES (level of education) and the two comorbidity measures that had been included in the case–control study questionnaire: prevalence of physician-diagnosed hypertension and cardiovascular condition (including myocardial infarction), which were obtained during the original in-person interview. These 2 conditions were included in the original study to evaluate their possible effects on prostate cancer risk.

Statistical Analyses

We used unconditional logistic regression (outcome variable=localized disease vs advanced [regional or distant] disease) to evaluate associations by race between disease stage at diagnosis and specific comorbid conditions adjusted for age, SES, and comorbidity. We also evaluated the risk of advanced disease by comparing African Americans, foreign-born Asian Americans, and North America-born Asian Americans with Whites after adjustment for age, SES and comorbidity. We did not classify African Americans and Whites according to place of birth, because virtually all were born in North America. We used Cox proportional hazards regression to evaluate determinants of time to death from prostate cancer and from other causes. We compared African Americans and Asian Americans with Whites, stratified by study center (LA, HI, SF, BC), and adjusted for SES and comorbidity. In the analysis of time to cause-specific death, men who died from other causes stopped contributing time to the analysis on their date of death.

RESULTS

Characteristics of Study Subjects

Of the 1509 patients in the analysis, 587 were listed as being alive on December 31, 1998, and 731 were listed as being deceased on or before this date. Fifty-one percent of the remaining 191 patients were known to be alive between January 1997 and November 1998, and they stopped contributing time to the analyses on these dates. Table 1 shows the distribution of patient characteristics by race and, among Asian Americans only, by place of birth. Virtually all of the foreign-born Asian Americans were of Chinese descent. A higher proportion of African Americans and foreign-born Asian Americans were diagnosed with distant-stage disease compared with Whites. In contrast, the distribution of stage among North America–born Asian Americans was similar to that of Whites. The proportion of patients with unknown stage and grade varied by race and by place of birth. These proportions were, respectively, 11% and 9% among African Americans, 6% and 5% among North America–born Asian Americans, 9% and 9% among foreign-born Asian Americans, and 6% and 5% among Whites. African Americans were diagnosed at younger ages than were Whites, whereas foreign-born Asian Americans were diagnosed at older ages, and North America–born Asian Americans and Whites were diagnosed at similar ages. Compared with Whites, African Americans and foreign-born Asian Americans

TABLE 1—Characteristics of 1509 Prostate Cancer Patients With Known Stage at Diagnosis, by Race: 4 North American Metropolitan Areas, 1987–1991

Variable	African American, No. (%)	White, No. (%)	Asian American		Total, No. (%)
			North American Born, No. (%)	Foreign Born, No. (%)	
Known stage	472	484	396	157	1509
Local	277 (59)	343 (71)	267 (67)	91 (58)	978 (65)
Regional	89 (19)	85 (18)	83 (21)	34 (22)	291 (19)
Distant	106 (23)*	56 (12)	46 (12)	32 (20)**	240 (16)
Known grade	433	466	375	144	1418
Well differentiated	109 (25)	121 (26)	112 (30)	44 (31)	386 (27)
Moderately differentiated	221 (51)	241 (52)	162 (43)	67 (47)	691 (49)
Poorly differentiated	96 (22)	99 (21)	98 (26)	32 (22)	325 (23)
Undifferentiated	7 (2)	5 (1)	3 (.8)	1 (.7)	16 (1)
Age at diagnosis, y	68.9*	70.5	70.6	72.9*	70.5
Completed high school	234 (50)*	412 (85)	274 (69)*	92 (59)*	1012 (67)
Live in less-educated tract[a]	272 (58)*	43 (9)	81 (21)*	59(38)*	544 (36)
Live in low-income tract[b]	332 (71)*	57 (12)	69 (18)***	75 (49)*	533 (36)
Cardiovascular condition	120 (26)***	155 (32)	86 (22)*	23 (15)*	384 (26)
Hypertension	259 (55)*	187 (39)	169 (43)	61 (39)	676 (45)
Vital status					
Alive	197 (42)*	283 (58)	231 (58)	67 (43)**	778 (52)
Deceased, prostate cancer	122 (26)	77 (16)	69 (17)	34 (22)	302 (20)
Deceased, other causes	153 (32)	124 (26)	96 (24)	56 (36)	429 (28)
Total person-years	2686	3176	2939	1008	9809

Note. Unadjusted comparison of African Americans or Asian Americans with Whites. Means were compared with the Student *t* test. Proportions were compared with the χ^2 test of association.
[a]≤ 70% of eligible individuals in the census tract completed high school.
[b]≥ 11% of families in the census tract live below the poverty line.
*P = .001; **P = .005; ***P = .05; all tests are 2-tailed.

had less education and were more likely to live in census tracts with low educational levels and high poverty. Within each race, census education and census poverty were highly correlated (r=.62–.78); however, personal education was not correlated with census education (r=.23–.25) or census poverty (r=.16–.22). We report analyses based only on census SES measures, because years of personal education contributed little after the addition of the 2 census-level SES measures. A significantly greater proportion of African Americans than of Whites reported hypertension. In contrast, African Americans and foreign-born Asian Americans reported less cardiovascular disease than did Whites. All-cause death rates were higher among African Americans and foreign-born Asian Americans than among Whites or North America–born Asian Americans.

Impact of Comorbidity on Stage at Diagnosis

Table 2 shows the results from race-specific estimates of the odds ratios (ORs) relating to a diagnosis of localized versus advanced disease to the prevalence of a comorbid condition after adjustment for age, census SES, and other comorbid condition. Among African Americans, both a cardiovascular condition (OR=0.6; 95% confidence interval [CI]=0.4, 0.9) and hypertension (OR=0.7; 95% CI=0.5, 1.0) were significantly associated with reduced odds of being diagnosed with advanced-stage disease. Among Whites, the presence of a cardiovascular condition was associated with significantly reduced odds of being diagnosed with advanced-stage disease (OR=0.6; 95% CI=0.4, 1.0). Among Asian Americans, nonsignificant but reduced associations were noted for cardiovascular conditions (OR=0.7; 95% CI=0.4, 1.1) and hypertension (OR=0.7; 95% CI=0.5, 1.1). These results suggest an association between these two comorbid conditions and presentation with localized disease.

Impact of Adjustment for SES and Comorbidity on Racial Differences in Stage at Diagnosis

Table 3 shows the odds ratios that relate stage of disease at diagnosis to race before and after adjustment for census SES and comorbidity. African American patients (OR=1.7; 95% CI=1.3, 2.2) and foreign-born Asian American patients (OR=1.9; 95% CI=1.3, 2.7) were more likely than White patients to be diagnosed with advanced prostate cancer. This disadvantage remained after adjustment for census SES (OR=1.5; 95% CI=1.1, 2.2) and comorbidity (OR=1.6; 95% CI=1.0, 2.4). We found no difference in stage at diagnosis between North America–born Asian Americans and Whites after adjustment for census SES and comorbidity (OR=1.0; 95% CI=0.8, 1.4). We performed separate analyses for foreign-born Chinese Americans, North America–born Japanese Americans, and North America–born Chinese-Americans, comparing them with Whites and finding similar results (data not shown). Thus, this disadvantage appears to be associated with birthplace rather than with ethnicity.

Impact of Adjustment for SES and Comorbidity on Racial Differences in Prostate Cancer Death Rates

Table 4 shows the results of stage-specific analyses that compared prostate cancer death rates among African Americans and Asian Americans with those among Whites. We pooled Asian Americans over place of birth because, compared with Whites, Asian Americans, regardless of place of birth, had similar stage-specific death rates that indicated no survival disadvantage. Table 4 shows that among patients with advanced disease, African Americans had no significant survival disadvantage compared with Whites; however, African American patients who were diagnosed with localized disease had significantly higher rates of death compared with Whites (death rate ratio [DRR]=2.5; 95% CI=1.4,

TABLE 2—Risk of Advanced (Local vs Regional/Distant) Prostate Cancer Diagnosis, by Race and Presence or Absence of Comorbid Conditions: 4 North American Metropolitan Areas, 1987–1991

	African American			White			Asian American		
	Local, No. (%)	Regional/Distant, No. (%)	OR (95% CI)	Local, No. (%)	Regional/Distant, No. (%)	OR (95% CI)	Local, No. (%)	Regional/Distant, No. (%)	OR (95% CI)
Cardiovascular condition[a]									
No	154 (45)	192 (55)	1.0	105 (32)	221 (68)	1.0	162 (37)	275 (63)	1.0
Yes	37 (31)	83(69)	0.6 (0.4, 0.9)	36 (24)	117 (76)	0.6 (0.4, 1.0)	30 (28)	78 (72)	0.7 (0.4, 1.1)
Hypertension[a]									
No	97 (47)	111 (53)	1.0	80 (27)	215 (73)	1.0	122 (38)	197 (62)	1.0
Yes	95 (37)	163 (63)	0.7 (0.5, 1.0)	60 (33)	124 (67)	1.4 (0.9, 2.1)	70 (31)	158 (69)	0.7 (0.5, 1.1)

Note. OR = odds ratio; CI = confidence interval.
[a]Adjusted for age, census education and poverty, and cardiovascular condition or hypertension.

4.5). This disadvantage persisted after adjustment for age and census SES (DRR=2.0; 95% CI=1.0, 4.0). These results did not change when we divided the patients into 2 groups—those aged younger than 70 years and those aged 70 years or older at diagnosis—or when we analyzed men separately within each tertile of poverty (data not shown).

Impact of Adjustment for SES and Comorbidity on Racial Differences in Rates of Death From Other Causes

Table 4 also shows the rate ratios for deaths from other causes. Again, we pooled Asian Americans over place of birth, because we did not see any appreciable differences in other-cause survival by place of birth. African Americans had higher rates of death from other causes than did Whites (DRR=1.5; 95% CI=1.2, 2.0), and this disadvantage persisted after adjustment for census SES and comorbidity (DRR=1.4; 95% CI=1.0, 2.0). In contrast, Asian Americans and Whites had similar rates of death from other causes both before (DRR=1.0; 95% CI=0.8, 1.3) and after (DRR=1.0; 95% CI=0.7, 1.4) adjustment for census SES and comorbidity. These findings were unchanged when we divided patients into groups of those aged younger than 70 years and those aged 70 years or older at diagnosis (data not shown).

DISCUSSION

This multiethnic cohort afforded the opportunity to evaluate inequalities in stage of disease at presentation and death rates from prostate cancer and other causes by race and place of birth. Although the results suggest that the presence of comorbid conditions is associated with earlier prostate cancer diagnosis in all races, adjustment for comorbidity and SES did not eliminate the observed racial disparities in stage of presentation and survival. Patients who did not participate in the original study were likely to present at more advanced stages than did the participants. Therefore, the actual actual stage and survival disadvantages are likely to be even greater than those we observed, because participation rates of African Americans and foreign-born Asian Americans were lower than those of Whites and North American–born Asian Americans.

Other studies also have found that adjustment for SES reduces but does not eliminate the increased likelihood of later stage at diagnosis among African Americans.[1,2,45,48,60,61] Our results among foreign-born Asian Americans confirm previous findings that a greater proportion of Asian Americans than of Whites are diagnosed with distant disease,[6] whereas the results among North America–born Asian Americans confirm other findings of little racial differences in stage at diagnosis.[13]

The higher prostate cancer death rate that has been observed among African American men with localized disease compared with Whites is consistent with results from a Veterans Affairs Medical Center study that did not adjust for SES (risk ratio$_{localized}$=1.34; 95% CI=0.99, 1.83)[62] and from a large population-based study that adjusted for stage and SES (DRR$_{men<65}$=1.41; 95% CI=1.15, 1.72 and DRR$_{men\geq 65}$=1.2; 95%

TABLE 3—Risk of Advanced (Local vs Regional/Distant) Prostate Cancer Diagnosis, by Race, Socioeconomic Status, and Comorbidity: 4 North American Metropolitan Areas, 1987–1991

		Asian American		
Adjustment	African American, OR (95% CI)	North American Born, OR (95% CI)	Foreign Born, OR (95% CI)	Total Asian Americans, OR (95% CI)
Age	1.7 (1.3, 2.2)	1.2 (0.9, 1.6)	1.9 (1.3, 2.7)	1.3 (1.0, 1.7)
Age, SES[a]	1.5 (1.1, 2.2)	1.1 (0.8, 1.5)	1.7 (1.1, 2.6)	1.2 (0.9, 1.6)
Age, SES[a], Comorbidity[b]	1.5 (1.1, 2.2)	1.0 (0.8, 1.4)	1.6 (1.0, 2.4)	1.2 (0.9, 1.5)

Note. OR = odds ratio; CI = confidence interval.
[a]SES = Socioeconomic status defined as census education and census poverty.
[b]Cardiovascular condition and/or hypertension.

TABLE 4—Risk of Death From Prostate Cancer and Other Causes, by Race, Age, Socioeconomic Status, and Comorbidity: 4 North American Metropolitan Areas, 1987–1991

	Death Rate Ratio (95% Confidence Interval)							
	African American				Asian American			
	Prostate Cancer				Prostate Cancer			
Adjustment	Localized (n = 40)[a]	Regional (n = 13)[a]	Distant (n = 69)[a]	Other Causes (n = 153)[a]	Localized (n = 29)[a]	Regional (n = 35)[a]	Distant (n = 39)[a]	Other Causes (n = 152)[a]
Age	2.5 (1.4, 4.5)	0.6 (0.3, 1.1)	1.0 (0.6, 1.7)	1.5 (1.2, 2.0)	0.6 (0.3, 1.1)	0.8 (0.4, 1.5)	1.2 (0.7, 2.0)	1.0 (0.8, 1.3)
Age, SES[b]	2.0 (1.0, 4.0)	0.6 (0.2, 1.4)	1.1 (0.6, 2.0)	1.4 (1.0, 1.9)	0.6 (0.3, 1.1)	0.8 (0.4, 1.5)	1.1 (0.6, 2.0)	0.9 (0.7, 1.2)
Age, SES,[b] Comorbidity[c]	2.3 (1.2, 4.7)	0.6 (0.2, 1.5)	1.1 (0.6, 2.0)	1.4 (1.0, 2.0)	0.6 (0.3, 1.1)	0.8 (0.4, 1.5)	1.1 (0.6, 2.0)	1.0 (0.7, 1.4)

[a]Deaths among Whites: from prostate cancer $n_{local} = 26$, $n_{regional} = 25$, and $n_{distant} = 23$; from other causes n = 124.
[b]Socioeconomic status defined as census education and census poverty.
[c]Cardiovascular condition and/or hypertension.

CI=1.07, 1.35).[49] However, these findings contradict the results of a small (N=391) hospital-based study by Dayal et al.[47] that found no racial differences in prostate cancer death rates after adjustment for SES. At the same time, our results among advanced cases are consistent with those from the Dayal et al. study.[47] In contrast and in disagreement with 1 previous study,[14] we did not find significant differences in prostate cancer death rates among Asian Americans compared with Whites. Our results provide evidence that potential screening bias (earlier and more frequent screenings for Whites that increase survival time) is unlikely to explain the higher prostate cancer death rates among African Americans compared with Whites. If a greater proportion of White patients were screen-detected, we would expect that foreign-born Asian Americans with localized disease also would have higher prostate cancer death rates compared with Whites.

To our knowledge, no studies other than the one by Robbins et al.[49] have specifically examined other-cause deaths rather than all-cause deaths.[4,7–10,47,48] In contrast to our results, Robbins and colleagues found that African Americans and Whites had similar other-cause death rates after adjustment for SES ($DRR_{men<65}$=1.14; 95% CI=0.86, 1.50 and $DRR_{men\geq65}$=0.96; 95% CI=0.85, 1.08). The larger sample size in the Robbins et al. study (N=23 334 vs N=956 [African Americans and Whites only] in our study) suggests that our results for other-cause deaths could be attenuated in a larger sample.

The influence of comorbidity on treatment choices and on death rates is particularly relevant in prostate cancer survival studies.[50,53,54,63] Comorbidity has been found to elevate the likelihood of death from other causes[51,52] and to elevate[51,52] or reduce[54,63] the likelihood of death from prostate cancer. In comparison with men in watchful-waiting or other treatment groups, men who undergo aggressive prostate cancer treatment (surgery or radiation with or without another therapy) are more likely to have their underlying cause of death listed as a cancer other than prostate cancer.[54] Because some data suggest that aggressive treatment is occurring disproportionately among Whites,[64–67] White patients who die may be less likely to have their cause of death listed as prostate cancer. Treatment information and medical records, including Gleason scores, were not collected in our original etiological study.[55] Our study did not include active follow-up, current medical record releases, or death certificate collection. Without this information, our ability to estimate the potential effect of misidentified cause of death on the observed African American and White survival differences is limited.

This type of study has limitations that deserve mention. First, tumor stage is better measured in surgically treated cases when compared with nonsurgically treated cases.[68] Given that patterns of care vary by race,[64–67] a higher proportion of African American patients with localized disease may be understaged (i.e., the diagnosed stage may underestimate the extent of disease progression) compared with White patients. If African Americans with understaged disease contribute disproportionately to the observed African American prostate cancer death rate among localized cases, adjustment for understaging could attenuate our results. This possible effect is difficult to quantify, partly because of a lack of treatment data. Second, although the literature suggests that ecological SES measures may capture factors specifically related to health that are independent of personal SES measures,[29,39–42] we cannot reject the possibility that different or additional measures of SES would further modify our results.

Although disease patterns are often described by race, observed racial disparities may reflect a number of factors for which race may be a proxy, including SES, comorbidity, acculturation, health care access and use, treatment, environment, and lifestyle factors such as diet. Our study extends previous research by including foreign-born and North America–born Asian Americans, and the results suggest that birthplace also may serve as a proxy for a number of factors related to stage at diagnosis. These results should reinforce the important contribution of acculturation and race/ethnicity to health care access and use.

SES and comorbidity are limited indicators of cultural, economic, and social risk factors that may be associated with later stage at diagnosis and with poor survival. Adjustment for them reduces but does not eliminate the observed racial disparities. The observation that African Americans and foreign-born

Asian Americans are more likely than Whites to be diagnosed with advanced disease, and the persistent survival disadvantage among African Americans in contrast to the lack of a survival disadvantage among foreign-born Asian Americans, are important findings. They support the need for further study of biological and social factors, including the impact of racial/ethnic bias on health care, in a large multiethnic population–based group of patients with prostate cancer. ■

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Contributors

I. Oakley-Girvan contributed to the analysis and directed all vital status follow-up. L.N. Kolonel, R.P. Gallagher, and A.H. Wu provided the original data. A. Felberg performed the statistical analysis. A.S. Whittemore provided the original data and directed the analysis.

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Human Participant Protection

Stanford's institutional review board reviewed and approved the human subjects protocol for this study. Human subjects were not contacted as part of this study.

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