

NIH Public Access Author Manuscript

J Urol. Author manuscript; available in PMC 2014 June 10.

Published in final edited form as:

J Urol. 2014 April; 191(4): 952–956. doi:10.1016/j.juro.2013.10.075.

Prostate Cancer Risk Profiles in Asian Americans: Disentangling the Effects of Immigration Status and Race/Ethnicity

Daphne Y. Lichtensztajn^a, Scarlett Lin Gomez^{a,b}, Weiva Sieh^b, Benjamin I. Chung^c, Iona Cheng^a, and James D. Brooks^c

^aCancer Prevention Institute of California, Fremont, CA

^bDivision of Epidemiology, Department of Health Research and Policy, Stanford University School of Medicine, Stanford, CA

^cDepartment of Urology, Stanford University School of Medicine, Stanford, CA

Abstract

Purpose—Asian-American men with prostate cancer have been reported to present with higher grade and later stage disease than White Americans. However, Asian Americans comprise a heterogeneous population with distinct health outcomes. We compared prostate cancer risk profiles among the diverse racial and ethnic groups in California.

Materials and Methods—We used data from the California Cancer Registry for 90,845 Non-Hispanic White, Non-Hispanic Black, and Asian-American men diagnosed with prostate cancer between 2004 and 2010. Patients were categorized into low, intermediate, or high-risk groups based on clinical stage, Gleason score, and PSA value at diagnosis. Using polytomous logistic regression, we estimated adjusted odds ratios for the association of race/ethnicity and nativity with risk group.

Results—In addition to Non-Hispanic Blacks, six Asian-American groups (US-born Chinese, foreign-born Chinese, US-born Japanese, foreign-born Japanese, foreign-born Filipino, and foreign-born Vietnamese) were more likely to have an unfavorable risk profile compared to Non-Hispanic Whites. The odds ratios for high vs. intermediate-risk disease ranged from 1.23 (95% CI, 1.02–1.49) for US-born Japanese to 1.45 (95% CI, 1.31–1.60) for foreign-born Filipinos. These associations appeared to be driven by higher grade and PSA values, rather than advanced clinical stage at diagnosis.

Conclusions—In this large, ethnically diverse population-based cohort, we found that Asian-American men were more likely to have unfavorable risk profiles at diagnosis. This association varied by racial/ethnic group and nativity, and was not attributable to later stage at diagnosis, suggesting that Asian men may have biological differences that predispose to the development of more severe disease.

Keywords

Asian Americans; prostatic neoplasms; epidemiology; SEER Program

Corresponding Author: Daphne Lichtensztajn, Cancer Prevention Institute of California, 2201 Walnut Ave, Ste 300, Fremont, CA 94538, Daphne.Lichtensztajn@cpic.org, Tel: 510- 608-5131, Fax: 510-608-5085.

INTRODUCTION

In men with prostate cancer (PCa), it is clinically important to distinguish between low risk disease, where treatment-related morbidity could be minimized through active surveillance, and high-risk disease, where more aggressive treatment may be indicated. Risk stratification tools based on prognostic factors such as Gleason score (GS), serum prostate specific antigen (PSA), and stage are widely used to categorize men into pretreatment risk groups that predict disease progression^{1, 2}.

PCa clinical characteristics vary by race/ethnicity^{3–6} and birthplace^{7, 8}. Studies have suggested that Asian-Americans have proportionally more advanced stage^{9–11} and high grade^{9, 10, 12–14} disease compared to Whites, which could have significant implications for treatment and prognosis in this population. However, previous studies were unable to disaggregate Asian subpopulations by ethnicity and birthplace, or were based upon data collected prior to the widespread adoption of PSA screening or in selected clinical populations.

Using a pretreatment risk stratification approach, we compared clinical risk profiles among Asian-American populations, defined by ethnicity and birthplace, to those of Non-Hispanic (NH) Whites and NH Blacks in a large population-based cohort.

PATIENTS AND METHODS

The California Cancer Registry (CCR) comprises four registries from the NCI's Surveillance, Epidemiology and End Results (SEER) program. We used tumor and demographic data collected through the CCR for men diagnosed with adenocarcinoma of the prostate during the years 2004–2010. These years were selected because GS and PSA were unavailable prior to 2004. We limited our study to men with first primary tumors, not diagnosed solely on death certificate or autopsy, and further restricted to NH Whites, NH Blacks, or members of one of the six largest Asian racial/ethnic groups in California: Chinese, Japanese, Filipino, Korean, Vietnamese, and South Asian (n=102,824). Using a previously described validated algorithm based on age at issue of social security number¹⁵, we imputed nativity for the 38% of Asian-American men whose place of birth was unknown in the registry data. US born Koreans (n=13), Vietnamese (n=19), and South Asians (n=61) were excluded because of small numbers that limited meaningful analyses. We further excluded cases with clinical stage T0 (n=23) or unknown (n=2510), unknown GS (n=2806), or unknown PSA value (n=6547). The final cohort consisted of 90,845 men.

Cases were geocoded to their census block group of residence at the time of diagnosis. Using a previously described composite measure of neighborhood socioeconomic status (SES) based on block group at diagnosis¹⁶, we assigned men to an SES quintile based on the statewide distribution.

Using a modification of the original D'Amico risk groups¹⁷, we categorized men into three clinical risk groups. Men with a highest pre-treatment PSA > 20 ng/ml, GS 8, or stage cT3 or higher were classified as having high-risk disease (n=19,648). Men with stage cT1-cT2a,

PSA 10 ng/ml, and GS 6 were classified as having low-risk disease (n=26,918). The remainder were considered to have intermediate-risk disease (n=44,279).

Statistical Analyses

Differences in distributions of disease and sociodemographic characteristics among groups were compared using chi-square tests. Medians were compared using the Kruskal-Wallis test. Comparisons to NH Whites were performed for each racial/ethnic group and p-values were adjusted for multiple comparisons using the step-down Bonferroni method¹⁸ as implemented in the SAS MULTTEST procedure.

Because high and low risk disease carry well-defined, but distinctly different, treatment recommendations, we used polytomous logistic regression to estimate adjusted odds ratios (ORs) and 95% confidence intervals (CIs) for the association of race/ethnicity with these risk categories compared to intermediate risk. To further analyze the nature of this relationship, we deconstructed the overall risk category and modeled the association of race/ethnicity with each component prognostic factor using polytomous logistic regression. Each factor was categorized into high, intermediate, and low-risk categories using the same cutpoints that were used to define overall risk groups.

Statistical analyses were performed with SAS 9.3 (SAS Institute, Cary, NC).

This study was approved by the Institutional Review Board of the Cancer Prevention Institute of California.

RESULTS

The sociodemographic and clinical characteristics of the cohort are described in Table 1. Median age at diagnosis was older for foreign-born (FB) Chinese, FB Japanese, FB Filipinos, FB Koreans, FB Vietnamese, and US-born (USB) Japanese, and younger for USB Filipinos and NH Blacks compared to NH Whites. The majority of the Asian groups (FB Chinese, USB Japanese, FB Japanese, FB Filipino, FB Korean, and FB Vietnamese) had higher proportions of men with high GS, and all groups except USB Chinese and USB Filipinos had lower proportions of men with low PSA compared to NH Whites. The proportion of patients diagnosed with high-risk disease was higher among FB Chinese, USB and FB Japanese, FB Filipinos, FB Vietnamese, and NH Blacks compared to NH Whites. Interestingly, several groups (NH Black, USB Chinese and FB Chinese) presented with favorable clinical stage distribution relative to NH Whites.

The odds of presenting with an adverse risk profile compared unfavorably to NH Whites for the majority of the racial/ethnic groups, after adjusting for age, SES, marital status, and year of diagnosis (Table 2). The effect sizes observed in the Asian groups were similar to those in NH Blacks. However, the pattern of prognostic factors conferring this increased risk differed among the groups (Table 3).

USB Chinese were more likely than NH Whites to be diagnosed with high vs. intermediaterisk disease (OR, 1.34; 95% CI, 1.05–1.71). This resulted from higher odds of high vs.

Lichtensztajn et al.

intermediate GS (OR, 1.58; 95% CI, 1.19–2.11), and lower odds of low vs. intermediate PSA category (OR, 0.76; 95% CI, 0.58–0.98) compared to NH Whites. FB Chinese had a similar profile as their US-born counterparts, with increased odds of high vs. intermediate-risk disease relative to NH Whites (OR 1.35; 95% CI, 1.20–1.52), apparently also attributable to an increased likelihood of high vs. intermediate GS (OR, 1.36; 95% CI, 1.18–1.57) and decreased likelihood of low vs. intermediate PSA (OR, 0.67; 95% CI, 0.59–0.76). Interestingly, USB and FB Chinese were more likely than NH Whites to be diagnosed with early vs. intermediate stage (OR, 1.52; 95% CI 1.19–1.93 and OR, 1.46; 95% CI, 1.29–1.64, respectively) but not advanced stage disease.

USB Japanese were more likely to have high-risk (OR, 1.23; 95% CI 1.02–1.49) and less likely to have low-risk disease (OR, 0.74; 95% CI 0.60–0.92) vs. intermediate-risk disease compared to NH Whites. The unfavorable risk profile in USB Japanese was attributable to decreased odds of low vs. intermediate PSA levels at diagnosis (OR, 0.79; 95% CI 0.64–0.97). FB Japanese also had a decreased likelihood of low vs. intermediate-risk disease (OR, 0.61; 95% CI 0.42–0.90), but their likelihood of high vs. intermediate-risk disease did not differ from NH White men. In addition to decreased odds of low vs. intermediate PSA levels (OR, 0.59; 95% CI, 0.42–0.84), they also had decreased odds of low vs. intermediate GS (OR, 0.67; 95% CI 0.48–0.93) at diagnosis.

While USB Filipinos did not differ from NH Whites in any risk categories, FB Filipino men had the highest odds of being diagnosed with high vs. intermediate-risk disease of any group (OR, 1.45; 95% CI, 1.31–1.60). FB Filipinos were more likely to have high vs. intermediate GS (OR, 1.27; 95% CI, 1.12–1.43), high vs. intermediate PSA (OR, 1.35; 95% CI, 1.17–1.56), and less likely to have low vs. intermediate PSA (OR, 0.72; 95% CI, 0.65–0.81). However, like Chinese men, they were more likely to be diagnosed at an early vs. intermediate stage (OR, 1.18; 95% CI, 1.07–1.30).

Compared to NH Whites, FB Vietnamese had higher odds of high vs. intermediate-risk disease (OR, 1.39; 95% CI, 1.13–1.72), resulting from higher odds of high vs. intermediate GS (1.37; 95% CI, 1.05–1.77) and lower odds of low vs. intermediate PSA (OR, 0.59; 95% CI, 0.48–0.74). They also had a higher likelihood of being diagnosed at an early vs. intermediate stage (OR, 1.39; 95% CI, 1.13–1.71).

FB South Asians and FB Koreans did not differ from NH Whites in overall risk category, but compared unfavorably to NH Whites for at least one prognostic factor.

Older age, lower SES, lack of health insurance at diagnosis, and unmarried and unknown marital status were associated with increased odds of high vs. intermediate-risk disease. Similarly, older age, lack of health insurance, and the lowest vs. highest SES quintile were associated with decreased odds of having low vs. intermediate-risk disease.

To assess the effect of excluding men with incomplete prognostic factor data, we performed a sensitivity analysis, imputing risk group. Men who had at least one prognostic factor in the high category were assigned to the high-risk group, and men with two known prognostic factors were assigned to the highest of the two known categories. Using this method, we

were able to assign a risk category to 98% of our cases. The results using imputed risk category were similar to the complete case analysis (Supplementary Table 1).

DISCUSSION

To our knowledge, no prior studies have examined proportional differences in PCa clinical characteristics jointly by birthplace and specific Asian ethnicity. In a large, multiethnic population-based study we found six specific Asian-American groups were more likely to have unfavorable clinical risk profiles than NH Whites. With the exception of USB Filipinos, all Asian groups were more likely to have at least one unfavorable prognostic factor at diagnosis. These findings have important clinical implications as categorization into a higher risk stratum limits treatment choices and predicts disease progression.

The unfavorable risk profile seen in many of the Asian-American groups could partly be explained by differences in screening behavior. Asian-Americans report less PSA screening than NH White men in California¹⁹. In our study, all Asian-American groups except USB Filipinos were less likely than NH Whites to have low PSA at diagnosis. In the National Cancer Database, Asian and Pacific Islander race/ethnicity was also associated with higher PSA at diagnosis¹⁴, whereas this association was not observed in a military-insured population with mandatory PSA screening¹³, supporting the notion that elevated PSA at diagnosis may be related to screening behavior.

Earlier studies using pre- and early-PSA era data found that, consistent with decreased screening behavior, Asian-Americans were more likely than NH Whites to be diagnosed at a more advanced stage^{9–11}. However, neither we nor Fedewa et al.¹⁴ found such an association using contemporary data. Instead, we found that FB Chinese, FB Vietnamese, FB Filipinos, and USB Chinese were more likely to present with early stage disease, similar to findings in a population with mandatory PSA screening¹³.

The association of Asian-American race/ethnicity with high GS that we and others observed^{9, 10, 12–14} is also not likely to be explained by screening behavior, as higher GS has not clearly been linked to the timing of diagnosis²⁰. Furthermore, in populations with low screening penetrance, Zlotta et al. found that while Japanese and Caucasian men had similar prevalence of latent PCa at autopsy, Japanese men were more likely to harbor high grade disease¹².

While differences in screening behavior may account for higher PSA at diagnosis, the association with high grade and the lack of association with advanced stage at diagnosis suggests that the differences we observed in risk profile cannot fully be explained by screening behaviors or delayed access to care, and may be attributable to an underlying intrinsic biological mechanism.

Androgens play an important role in prostatic carcinogenesis, and racial differences in androgen metabolism have been documented²¹. Studies have noted that Asians have lower 5α -reductase activity^{21–23}, and an association between reduced 5α -reductase activity and high grade is supported by evidence from the Prostate Cancer Prevention Trial (PCPT) and Reduction by Dutasteride of Prostate Cancer Events (REDUCE) trial, where an increased

Lichtensztajn et al.

risk of high grade cancers was observed despite a reduction in the overall incidence of PCa in men taking 5α -reductase inhibitors^{24, 25}. It has been hypothesized that the low intraprostatic androgen levels resulting from 5α -reductase inhibition may selectively inhibit low grade cancers and favor the selection of poorly differentiated cancer cells resulting in higher grade tumors. Alternatively, reduction in prostate size from decreased 5α -reductase activity may facilitate increased detection of high grade disease on biopsy²⁵. Whether through preferential selection or increased detection of higher grade tumors, lower 5α -reductase activity may be one factor contributing to the increased likelihood of categorization of Asian PCa patients into higher clinical risk categories.

In our study, birthplace was associated with a strikingly different risk profile in Filipino men but less so in Chinese and Japanese men, which may reflect differences in acculturation, environmental exposures, or lifestyle factors among these groups. Our findings reinforce the importance of considering both detailed ethnicity and nativity when studying cancer outcomes among the heterogeneous Asian-American population¹⁵.

While our findings are provocative, a greater understanding of the outcomes of high risk prostate cancer in Asian men is necessary before screening and treatment practices are changed. Some studies have found that Asian-American men, despite their poorer prognostic features at diagnosis, have better survival and biochemical recurrence outcomes than NH White men^{9, 10, 13}, suggesting that the traditional criteria used to determine clinical risk may be inadequate to assess the risk of disease progression in Asian populations.

Like all observational, registry-based studies, this study has some limitations. Our results may be affected by potential misclassification of race/ethnicity and/or nativity. While registry data on birthplace has been shown to be highly accurate at the level of US or foreign birth²⁶, there may be some misclassification associated with our imputation method. Further, misclassification of specific Asian race/ethnicity²⁷ may have biased the results toward the null, as the extent of misclassification is most likely non-differential with regard to the outcomes of interest. Another limitation is that data on tumor volume, and the number and involvement of biopsy cores were not available. However, the available data were sufficient to stratify men into the broad recurrence risk groups that guide the initial management choices in the National Comprehensive Cancer Network guidelines²⁸. Finally, we were unable to control for prostate size or body mass index, which may be associated with grade^{13, 25, 29}.

Our study has several strengths. California is home to the largest and most diverse Asian-American population in the United States, accounting for approximately one third of all U.S. Asian-Americans³⁰. We therefore had sufficient numbers to be able to disaggregate the Asian-American population by ethnicity and birthplace, and were able to demonstrate the heterogeneity that exists among these groups. By using data from a state-mandated registry that captures all cancer cases in California, the results are less susceptible to reporting and selection bias than studies that rely on voluntary reporting or specific health care systems and patient populations, and are widely applicable to the general population.

CONCLUSIONS

Given the indolent nature of the majority of prostate cancers currently being diagnosed, determining the risk of disease progression is important in guiding treatment decisions. We found that most Asian-American groups present with higher risk disease than NH Whites. Further research is necessary to assess whether the poorer risk profiles we observed in this study translate to worse clinical outcomes in these Asian-American populations.

Supplementary Material

Refer to Web version on PubMed Central for supplementary material.

Acknowledgments

This research was supported by the National Cancer Institute's Surveillance, Epidemiology and End Results Program under contract HHSN261201000140C awarded to the Cancer Prevention Institute of California and NCI grant K07CA143047 awarded to Weiva Sieh. The collection of cancer incidence data used in this study was supported by the California Department of Health Services as part of the statewide cancer reporting program mandated by California Health and Safety Code Section 103885; the National Cancer Institute's Surveillance, Epidemiology, and End Results Program under contract HHSN261201000140C awarded to the Cancer Prevention Institute of California, contract HHSN261201000035C awarded to the University of Southern California, and contract HHSN261201000034C awarded to the Public Health Institute; and the Centers for Disease Control and Prevention's National Program of Cancer Registries, under agreement #1U58 DP000807-01 awarded to the Public Health Institute. The ideas and opinions expressed herein are those of the authors, and endorsement by the State of California, the California Department of Health Services, the National Cancer Institute, or the Centers for Disease Control and Prevention or their contractors and subcontractors is not intended nor should be inferred.

REFERENCES

- 1. Freedland SJ. Screening, risk assessment, and the approach to therapy in patients with prostate cancer. Cancer. 2011; 117:1123. [PubMed: 20960523]
- 2. National Comprehensive Cancer Network: Prostate Cancer. 2011
- 3. Howlader, N.; Noone, AM.; Krapcho, M., et al. SEER Cancer Statistics Review, 1975–2009 (Vintage 2009 Populations). Bethesda, MD: National Cancer Institute;
- 4. Latini DM, Elkin EP, Cooperberg MR, et al. Differences in clinical characteristics and disease-free survival for Latino, African American, and non-Latino white men with localized prostate cancer: data from CaPSURE. Cancer. 2006; 106:789. [PubMed: 16400651]
- Dall'era MA, Hosang N, Konety B, et al. Sociodemographic predictors of prostate cancer risk category at diagnosis: unique patterns of significant and insignificant disease. J Urol. 2009; 181:1622. [PubMed: 19230923]
- Hoffman RM, Gilliland FD, Eley JW, et al. Racial and ethnic differences in advanced-stage prostate cancer: the Prostate Cancer Outcomes Study. J Natl Cancer Inst. 2001; 93:388. [PubMed: 11238701]
- Cook LS, Goldoft M, Schwartz SM, et al. Incidence of adenocarcinoma of the prostate in Asian immigrants to the United States and their descendants. J Urol. 1999; 161:152. [PubMed: 10037388]
- 8. Watanabe M, Nakayama T, Shiraishi T, et al. Comparative studies of prostate cancer in Japan versus the United States. A review. Urol Oncol. 2000; 5:274. [PubMed: 11008096]
- Robbins AS, Koppie TM, Gomez SL, et al. Differences in prognostic factors and survival among white and Asian men with prostate cancer, California, 1995–2004. Cancer. 2007; 110:1255. [PubMed: 17701951]
- Man A, Pickles T, Chi KN. Asian race and impact on outcomes after radical radiotherapy for localized prostate cancer. J Urol. 2003; 170:901. [PubMed: 12913726]
- Oakley-Girvan I, Kolonel LN, Gallagher RP, et al. Stage at diagnosis and survival in a multiethnic cohort of prostate cancer patients. Am J Public Health. 2003; 93:1753. [PubMed: 14534233]

- 12. Zlotta AR, Egawa S, Pushkar D, et al. Prevalence of Prostate Cancer on Autopsy: Cross-Sectional Study on Unscreened Caucasian and Asian Men. J Natl Cancer Inst. 2013
- Raymundo EM, Rice KR, Chen Y, et al. Prostate cancer in Asian Americans: incidence, management and outcomes in an equal access healthcare system. BJU Int. 2011; 107:1216. [PubMed: 21040364]
- Fedewa SA, Etzioni R, Flanders WD, et al. Association of insurance and race/ethnicity with disease severity among men diagnosed with prostate cancer, National Cancer Database 2004– 2006. Cancer Epidemiol Biomarkers Prev. 2010; 19:2437. [PubMed: 20705937]
- Gomez SL, Quach T, Horn-Ross PL, et al. Hidden breast cancer disparities in Asian women: disaggregating incidence rates by ethnicity and migrant status. Am J Public Health. 2010; 100(Suppl 1):S125. [PubMed: 20147696]
- Yost K, Perkins C, Cohen R, et al. Socioeconomic status and breast cancer incidence in California for different race/ethnic groups. Cancer Causes Control. 2001; 12:703. [PubMed: 11562110]
- D'Amico AV, Whittington R, Malkowicz SB, et al. Biochemical outcome after radical prostatectomy, external beam radiation therapy, or interstitial radiation therapy for clinically localized prostate cancer. JAMA. 1998; 280:969. [PubMed: 9749478]
- Holm S. A Simple Sequentially Rejective Bonferroni Test Procedure. Scandinavian Journal of Statistics. 1979; 6:65.
- 19. California Health Interview Survey (CHIS). 2003,2005,2009.
- 20. Lavery HJ, Droller MJ. Do Gleason patterns 3 and 4 prostate cancer represent separate disease states? J Urol. 2012; 188:1667. [PubMed: 22998919]
- 21. Crawford ED. Understanding the epidemiology, natural history, and key pathways involved in prostate cancer. Urology. 2009; 73:S4. [PubMed: 19375626]
- 22. Wu AH, Whittemore AS, Kolonel LN, et al. Serum androgens and sex hormone-binding globulins in relation to lifestyle factors in older African-American, white, and Asian men in the United States and Canada. Cancer Epidemiol Biomarkers Prev. 1995; 4:735. [PubMed: 8672990]
- Ross RK, Bernstein L, Lobo RA, et al. 5-alpha-reductase activity and risk of prostate cancer among Japanese and US white and black males. Lancet. 1992; 339:887. [PubMed: 1348296]
- 24. Thompson IM, Goodman PJ, Tangen CM, et al. The influence of finasteride on the development of prostate cancer. N Engl J Med. 2003; 349:215. [PubMed: 12824459]
- 25. Andriole GL, Bostwick DG, Brawley OW, et al. Effect of dutasteride on the risk of prostate cancer. N Engl J Med. 2010; 362:1192. [PubMed: 20357281]
- 26. Gomez SL, Glaser SL, Kelsey JL, et al. Bias in completeness of birthplace data for Asian groups in a population-based cancer registry (United States). Cancer Causes Control. 2004; 15:243. [PubMed: 15090719]
- 27. Gomez SL, Glaser SL. Misclassification of race/ethnicity in a population-based cancer registry (United States). Cancer Causes Control. 2006; 17:771. [PubMed: 16783605]
- 28. National Comprehensive Cancer Network: Prostate Cancer. 2012
- Amling CL, Kane CJ, Riffenburgh RH, et al. Relationship between obesity and race in predicting adverse pathologic variables in patients undergoing radical prostatectomy. Urology. 2001; 58:723. [PubMed: 11711349]
- 30. US Census Bureau: 2010 Census Brief. US Census Bureau; 2012. The Asian Population: 2010.

NIH-PA Author Manuscript

NIH-PA Author Manuscript

Lichtensztajn et al.

Table 1

Characteristics by race/ethnicity, and nativity among Asian-Americans, of men diagnosed with adenocarcinoma of the prostate, California, 2004–2010

						Race	Race/ethnicity & nativity	nativity				
	All N=90845	NH White N=73040	NH Black N=10886	US born Chinese N=414	Foreign born Chinese N=1645	US born Japanese N=604	Foreign born Japanese N=186	US born Filipino N=246	Foreign born Filipino N=2333	Foreign born Korean N=412	Foreign born Vietnamese N=522	Foreign born South Asian N=557
	%	%	%	%	%	%	%	%	%	%	%	%
Age												
<50	3%	3%	6%	3%	1%	1%		5%	2%	%0	2%	2%
50-59	22%	21%	29%	25%	14%	11%	19%	29%	17%	5%	15%	21%
60–69	41%	41%	41%	34%	35%	33%	36%	46%	42%	44%	46%	44%
70–79	27%	27%	20%	32%	39%	39%	31%	19%	33%	45%	31%	28%
80+	7%	8%	4%	7%	11%	16%	14%	2%	7%	5%	7%	5%
p value*			<.0001	0.0290	<.0001	<.0001	0.0060	<.0001	<.0001	<.0001	0.0018	0.0674
Median age	66.0	66.0	63.0	66.0	0.69	70.0	68.0	62.0	67.0	69.5	67.0	66.0
p value*			<.0001	1.000	<.0001	<.0001	.0005	<.0001	<.0001	<.0001	0.0074	1.000
Marital status												
single, divorced, separated, widowed	23%	22%	37%	16%	10%	18%	18%	12%	12%	%6	14%	11%
married	71%	72%	56%	76%	82%	76%	73%	80%	83%	%6L	78%	82%
unknown	6%	%9	%L	%6	8%	6%	%6	8%	5%	12%	8%	7%
p value*			<.0001	0.0110	<.0001	0.0787	0.1923	0.0030	<.0001	<.0001	0.0004	<.0001
Block group SES (1=lowest)												
1	%6	7%	23%	4%	7%	6%	8%	8%	11%	16%	10%	8%
2	15%	14%	23%	6%	12%	11%	10%	15%	18%	15%	23%	12%
ω	20%	20%	22%	11%	17%	18%	16%	25%	25%	17%	24%	19%
4	24%	25%	19%	26%	25%	30%	30%	28%	25%	19%	21%	19%
Ŋ	32%	34%	13%	53%	39%	35%	37%	23%	22%	33%	23%	42%
p value*			<.0001	<.0001	0.0013	0.0472	0.1466	0.0244	<.0001	<.0001	<.0001	0.0018
Insurance status (Payer source at diagnosis)												

_
~
~
_
_
_
0
~
- C
~
<u> </u>
-
_
-
0
<u> </u>
uthor
_
~
\geq
Mar
a
=
-
C
77
S
Ö
0
-
uscrip
9
-

_
_
_
_
_
_
U
~
~
utho
<u> </u>
-
_
\cap
_
2
2
2
2
2
2
2
2
2
2
2
2
2
2
r Manusc
r Manusc
2
r Manusc
r Manusc
r Manusc

Race/ethnicity & nativity

Lichtensztajn et al.

	All N=90845	NH White N=73040	NH Black N=10886	US born Chinese N=414	Foreign born Chinese N=1645	US born Japanese N=604	Foreign born Japanese N=186	US born Filipino N=246	Foreign born Filipino N=2333	Foreign born Korean N=412	Foreign born Vietnamese N=522	Foreign born South Asian N=557
	%	%	%	%	%	%	%	%	%	%	%	%
no known insurance	3.5%	3.5%	3.7%	1.4%	2.6%	3.1%	2.2%	4.5%	3.4%	5.6%	3.8%	3.4%
insurance	96.5%	96.5%	96.3%	98.6%	97.4%	96.9%	97.8%	95.5%	96.6%	94.4%	96.2%	96.6%
p value*			1.000	0.2324	0.5203	1.000	1.000	1.000	1.000	0.1833	1.000	1.000
Clinical T stage												
TI	62%	61%	66%	71%	67%	63%	58%	66%	63%	61%	67%	62%
Τ2	35%	36%	30%	26%	29%	33%	39%	31%	34%	35%	29%	34%
T3	3%	3%	2%	3%	3%	3%	2%	2%	3%	3%	3%	2%
Τ4	1%	1%	1%	0%	1%	1%	1%	%0	1%	1%	%0	2%
p value *			<.0001	0.0029	<.0001	1.000	1.000	1.000	0.6062	1.0000	0.0588	0.1163
Gleason score (from largest specimen)												
Gleason score 2–6	46%	46%	44%	44%	41%	39%	34%	53%	41%	37%	47%	46%
Gleason score 7	40%	39%	41%	37%	38%	41%	47%	36%	39%	43%	34%	39%
Gleason score 8–10	15%	14%	15%	19%	21%	20%	19%	11%	20%	19%	18%	15%
p value *			<.0001	0.0667	<.0001	0.0002	0.0213	0.1451	<.0001	0.0015	0.0414	0.9178
PSA (highest value before treatment)												
0–10.0 ng/ml	74%	76%	66%	72%	65%	67%	64%	79%	66%	67%	65%	%69
10.1–20.0 ng/ml	16%	15%	19%	18%	21%	20%	25%	13%	19%	23%	22%	20%
>20 ng/ml	10%	10%	15%	10%	13%	13%	11%	8%	16%	10%	13%	11%
p value*			<.0001	0.4293	<.0001	<.0001	0.0013	0.4293	<.0001	<.0001	<.0001	0.0025
Risk Group												
Low risk	30%	30%	27%	30%	26%	21%	19%	35%	25%	25%	28%	28%
Intermediate risk	49%	49%	48%	46%	46%	49%	53%	49%	46%	50%	45%	50%
High risk	22%	21%	24%	24%	28%	30%	27%	16%	28%	25%	27%	22%
p value*			<.0001	0.3451	<.0001	<.0001	0.0118	0.3109	<.0001	0.0753	0.0117	0.5423
Year of diagnosis												
2004	15%	15%	13%	15%	14%	20%	18%	%6	16%	12%	15%	13%

								6				
	All N=90845	NH White N=73040	NH Black N=10886	US born Chinese N=414	Foreign born Chinese N=1645	US born Japanese N=604	Foreign born Japanese N=186	US born Filipino N=246	Foreign born Filipino N=2333	Foreign born Korean N=412	Foreign born Vietnamese N=522	Foreign born South Asian N=557
	%	%	%	%	%	%	%	%	%	%	%	%
2005	13%	13%	13%	11%	14%	15%	13%	7%	14%	%6	12%	13%
2006	15%	15%	14%	13%	14%	16%	13%	13%	13%	16%	15%	12%
2007	16%	16%	15%	17%	16%	13%	16%	21%	16%	18%	14%	14%
2008	14%	14%	15%	15%	15%	14%	10%	17%	14%	15%	16%	16%
2009	14%	14%	15%	14%	15%	13%	13%	17%	14%	15%	15%	16%
2010	13%	13%	14%	14%	13%	%6	16%	16%	14%	14%	13%	17%
p value*			<.0001	1.000	1.000	0.0428	1.000	0.0062	0.8159	0.8159	1.000	0.1478

5, controls the ranking was ented the (right renormed on an the comparisons) at an e-exert the septerown memory as who will be a the control interior) as the simple Bonferoni correction; it adjusts sequentially from the lowest to the highest p value, using different (weighted) significance levels for observed p values.

		High Risk Ris	High Risk vs Intermediate Risk Group	Low Risk Rie	Low Risk vs Intermediate Risk Group
		OR	(95% CI)	OR	(95% CI)
Race/ethnicity	NH White	1.00		1.00	
	NH Black	1.26	(1.19 - 1.33)	0.86	(0.82 - 0.90)
	US born Chinese	1.34	(1.05 - 1.71)	1.02	(0.82 - 1.28)
	Foreign born Chinese	1.35	(1.20 - 1.52)	0.96	(0.85 - 1.09)
	US born Japanese	1.23	(1.02 - 1.49)	0.74	(0.60 - 0.92)
	Foreign born Japanese	1.12	(0.79 - 1.58)	0.61	(0.42 - 0.90)
	US born Filipino	06.0	(0.63 - 1.30)	1.07	(0.81 - 1.41)
	Foreign born Filipino	1.45	(1.31 - 1.60)	0.91	(0.82 - 1.01)
	Foreign born Korean	1.09	(0.86 - 1.38)	0.89	(0.70 - 1.13)
	Foreign born Vietnamese	1.39	(1.13–1.72)	1.04	(0.85 - 1.28)
	Foreign born South Asian	1.09	(0.87 - 1.34)	06.0	(0.74 - 1.09)
Age at diagnosis	per year	1.05	(1.04 - 1.05)	0.98	(0.98-0.98)
Quintile of block group SES	1 (lowest)	1.21	(1.13 - 1.29)	06.0	(0.84 - 0.95)
	2	1.19	(1.13 - 1.26)	0.95	(0.91 - 1.00)
	3	1.13	(1.08 - 1.19)	0.99	(0.95 - 1.03)
	4	1.06	(1.01 - 1.12)	1.01	(0.97 - 1.05)
	5 (highest)	1.00		1.00	
Insurance status (Payer source at diagnosis)	Insurance	1.00		1.00	
	No known insurance	1.22	(1.12 - 1.33)	0.82	(0.75-0.90)
Marital Status	Married	1.00		1.00	
	Single, divorced, separated, widowed	1.34	(1.28 - 1.39)	0.98	(0.94 - 1.02)
	Unknown	1.10	(1.03 - 1.18)	1.20	(1.12–1.27)
Year of diagnosis	per year	1.00	(0.99 - 1.01)	0.99	(0.98 - 1.00)

J Urol. Author manuscript; available in PMC 2014 June 10.

OR=odds ratio; CI=confidence interval

Lichtensztajn et al.

Table 3

Association of prognostic factors with race/ethnicity, and nativity among Asian-Americans.

		Gleason Category ^a	ategory	a		Clinical T Stage Category ^b	ge Categ	goryb		PSA Ca	PSA Category ^c	
	Gleas	Gleason score 8–10 vs 7	Gleas	Gleason score 2–6 vs 7	Adva (T3+) v	Advanced stage (T3+) vs T2b,c,NOS	Ea T2	Early stage (T1–T2a) vs T2b,c,NOS	>2 10.1-	>20ng/ml vs 10.1–19.9 ng/ml	0-1 10.1-	0–10 ng/ml vs 10.1–19.9 ng/ml
	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)	OR	(95% CI)
NH White	1.00		1.00		1.00		1.00		1.00		1.00	
NH Black	0.97	(0.91 - 1.04)	0.87	(0.83-0.91)	0.89	(0.79–1.01)	1.23	(1.17–1.29)	1.20	(1.11 - 1.30)	0.64	(0.61 - 0.68)
US born Chinese	1.58	(1.19–2.11)	1.00	(0.81–1.25)	1.12	(0.61–2.04)	1.51	(1.18–1.92)	0.84	(0.57–1.25)	0.76	(0.58–0.98)
Foreign born Chinese	1.36	(1.18–1.57)	0.96	(0.86–1.08)	1.11	(0.84–1.47)	1.45	(1.29–1.63)	0.96	(0.81–1.15)	0.67	(0.59–0.76)
US born Japanese	1.11	(0.88 - 1.40)	0.84	(0.70 - 1.00)	1.05	(0.67–1.64)	1.15	(0.95–1.37)	0.99	(0.74–1.33)	0.79	(0.64–0.97)
Foreign born Japanese	0.97	(0.64–1.46)	0.67	(0.48–0.93)	0.76	(0.32 - 1.80)	0.87	(0.64–1.19)	0.68	(0.40–1.15)	0.59	(0.42–0.84)
US born Filipino	0.98	(0.62–1.55)	1.23	(0.94–1.63)	1.16	(0.51–2.59)	1.12	(0.83 - 1.50)	1.04	(0.58–1.86)	0.97	(0.66–1.42)
Foreign born Filipino	1.27	(1.12–1.43)	0.93	(0.85–1.02)	0.79	(0.61–1.01)	1.18	(1.07 - 1.30)	1.35	(1.17–1.56)	0.72	(0.65 - 0.81)
Foreign born Korean	1.13	(0.85–1.49)	0.77	(0.62–0.96)	1.22	(0.73–2.06)	1.17	(0.94–1.47)	0.63	(0.43-0.91)	0.66	(0.52 - 0.84)
Foreign born Vietnamese	1.37	(1.05–1.77)	1.19	(0.97–1.44)	1.14	(0.69 - 1.89)	1.39	(1.13–1.71)	0.93	(0.68–1.27)	0.59	(0.48 - 0.74)
Foreign born South Asian	1.01	(0.78–1.32)	1.08	(0.90 - 1.30)	1.04	(0.65–1.67)	0.95	(0.79–1.15)	06.0	(0.65–1.24)	0.62	(0.50-0.77)
^a Model additionally adjusted for age, block group SES, insurance status, marital status, clinical T stage, PSA category, and year of diagnosis	or age, h	olock group SES	, insura	nce status, mari	tal status	s, clinical T stag	e, PSA	category, and y	ear of d	iagnosis		
b Model additionally adjusted for age, block group SES, insurance status, marital status, Gleason score, PSA category, and year of diagnosis	or age, h	olock group SES	, insurai	nce status, mari	tal status	s, Gleason score	, PSA c	ategory, and ye	ar of di	agnosis		
^C Model additionally adjusted for age, block group SES, insurance status, marital status, Gleason score, clinical T stage, and year of diagnosis	or age, b	olock group SES	, insuraı	ice status, mari	tal status	s, Gleason score	, clinica	l T stage, and y	'ear of d	iagnosis		
OR=odds ratio; CI=confidence interval	interva	_										